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CLINICAL TRIAL REPORT

Effects of Mobile Neurofeedback on Theta–Gamma Coupling and Clinical Outcomes in Children with ADHD: A Double-Blind, Sham-Controlled Randomized Clinical Trial

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Objective: This double-blinded randomized trial investigated the therapeutic effects of theta/beta mobile neurofeedback (MNF) in 8–15-year-old ADHD and neurotypical children (randomized N = 139; analyzed N = 120).

Methods: Participants were divided into three groups: ADHD Combination (MNF + Medication), ADHD MNF alone, and Neurotypical, and each group underwent 3 months of MNF intervention, either active or sham, the latter using randomly generated training results. Pre- and post-intervention electroencephalography tests were conducted, including an analysis of theta-gamma coupling (TGC), along with assessments of clinical variables.

Results: Regarding clinical outcomes, MNF was not superior to sham in alleviating ADHD symptoms based on self-reports or in cognitive test performance, as indicated by limited changes in clinical variables. Before the intervention, the Neurotypical group exhibited higher TGC scores than the ADHD groups, reaffirming TGC as a neurophysiological marker for attention and working memory. Post-intervention, the ADHD Combination group with active MNF showed increased TGC in Fp2, F3, F4, F8, P4, O1, and O2, while the ADHD MNF alone group had mixed results. Notably, active MNF had no significant effect on TGC in the Neurotypical group, while sham MNF reduced TGC.

Conclusion: MNF produced measurable neurophysiological modulation—reflected in enhanced TGC—when used as an adjunct to medication. Although clinical improvements were modest, these findings support TGC as a sensitive biomarker of MNF-induced brain regulation in ADHD.

Keywords: ADHD, mobile neurofeedback, double-blind randomized controlled trial, theta-gamma coupling

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a type of neurodevelopmental disorder characterized by inattention, impulsivity, and hyperactivity, associated with adverse long-term functional outcomes.[1] ADHD is a common childhood-onset neurodevelopmental disorder associated with functional impairments across academic and social domains. Early diagnosis and continuous management are essential for favorable long-term outcomes.[2]

Currently, pharmacological therapy is widely recognized as the first-line treatment for ADHD.[3] Stimulant medications, in particular, are effective in reducing core symptoms, with meta-analyses reporting large effect sizes.[4] However, a review of adverse drug reactions associated with ADHD medications in children identified decreased appetite, gastrointestinal discomfort, and headaches as common side effects, underscoring the need for extended safety monitoring given the chronic treatment course of ADHD.[5] The Multimodal Treatment Study of Children With ADHD (MTA) demonstrated that stimulant medication showed relative superiority for symptom reduction during the first two years of treatment, but this

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benefit appeared to diminish by the third year, highlighting the limited durability of pharmacological effects over time.[6] Furthermore, the 8-year follow-up of the MTA found that the type or intensity of the initial 14-month ADHD treatment did not predict functional outcomes in adolescence. Instead, long-term prognosis was best explained by the trajectory of symptom change in the early stages, regardless of treatment modality. This finding suggests that early and sustained symptom improvement, rather than the specific form of treatment, is the most critical determinant of long-term outcomes.[7]

For these reasons, neurofeedback training as an add-on to medication therapy has been proposed. Neurofeedback training has received ongoing interest from researchers due to its direct relationship with deviances in electroencephalography (EEG). Neurofeedback was developed based on the research results of Lubar et al, which involved a training method aimed at decreasing theta in the frontal and parietal lobes and increasing beta.[8] With the development of neurofeedback practices, mobile neurofeedback devices were introduced. In the case of neurofeedback training for ADHD children, sensorimotor rhythm (SMR), theta/beta ratio (TBR) and slow cortical potential (SCP) neurofeedback are considered as standard protocols, and the reward children receive is improved performance or positive feedback through visual cues on the computer screen or the sound of a success signal.[9] A meta-analysis reported large effect sizes for reducing inattention and impulsivity and a moderate effect for hyperactivity, supporting its clinical utility.[10] Another systematic review further showed that neurofeedback had sustained effects in reducing parent-rated ADHD behaviors compared with non-active control conditions.[11]

In addition to clinical conditions, research focusing on "optimal" or "performance enhancement" is also being conducted on healthy participants. One study introduced a novel method for enhancing golf performance using personalized real-life neurofeedback during golf putting.[12] Another study utilized neurofeedback training using EEG to optimize attention in expert rifle shooters, and results indicated improved shooting performance correlated with specific EEG patterns, suggesting that certain brain states are optimal for achieving "good" shots and that neurofeedback training can influence these states.[13] However, research on the effects of neurofeedback in healthy children and adolescents is still scarce. The period of childhood and adolescence is characterized by significant neuroplasticity of the brain compared to adulthood, making it an opportune time to maximize the effects of neuroregulatory therapies such as neurofeedback.[14] In addition, it is anticipated that manipulative conditioning processes and motivation would be easier in healthy children and adolescents who possess superior attention and self-regulation abilities compared to those with ADHD. However, to validate these assumptions, scientific verification such as double-blind experiments and sham control groups are necessary.

Recently, there has been an increasing emphasis on using objective and quantifiable neurophysiological data in both diagnostic and therapeutic strategies for ADHD, with quantitative electroencephalography (QEEG) receiving growing attention. QEEG studies have reported characteristic alterations in brain activity among children with ADHD, although findings are not always consistent.[15] To address these limitations, more advanced approaches such as EEG-based deep learning models have been developed, demonstrating robust predictive power for ADHD diagnosis.[16] Beyond diagnostic applications, current research has focused on cross-frequency interactions, particularly theta–gamma coupling (TGC), as a candidate neurophysiological marker of ADHD. TGC was first described in the rat hippocampus,[17] where gamma oscillations preferentially aligned with specific phases of the theta rhythm during task performance, and later confirmed in the human cortex as a mechanism supporting efficient cognitive processing.[18] Impairments in functional connectivity and neuronal communication have been proposed as a key pathophysiological mechanism of ADHD, and TGC analysis has shown superior accuracy in differentiating ADHD from typically developing children compared to traditional QEEG parameters such as the theta/beta ratio.[19] Furthermore, while typically developing children show stable TGC during cognitive tasks, children with ADHD exhibit decreased synchronization, supporting the notion of impaired attentional shifting.[20] Taken together, these findings suggest that TGC is closely related to working memory and attentional control, both of which are core deficits in ADHD, and justify its use as a promising neurophysiological marker in our study. Because the MNF protocol targets the TBR, which modulates attentional control via frontal cortical oscillations, it is mechanistically linked to TGC, a cross-frequency interaction underlying the integration of attentional and executive networks.

Our group has previously reported the effects of mobile neurofeedback on internet addiction and neurocognitive function in neurotypical children in a single-center trial.[21] Building on this work, the present multi-center study included both ADHD and neurotypical children to examine neurophysiological changes and clinical outcomes. Despite the growing evidence for neurofeedback, clinical results remain variable, suggesting a gap between behavioral improvement

and underlying neural modulation. This study aimed to address this gap by examining whether mobile neurofeedback (MNF) enhances TGC, a sensitive marker of cortical synchronization, compared with sham feedback. TGC was defined as the primary neurophysiological outcome, whereas clinical and cognitive measures were treated as secondary exploratory outcomes. The inclusion of neurotypical and medication subgroups allowed evaluation of normative baselines and potential additive effects. We hypothesized that active MNF would increase TGC, especially in frontal regions related to attention control, and that this effect would be most pronounced in children receiving MNF with medication.

Methods

Participants

The participants were children aged 8 to 15 years, and were enrolled from the Department of Psychiatry, Daegu Catholic University Medical Center, Seoul National University Hospital and Hanyang University Seoul Hospital between 2019 and 2021. After given detailed information of the study, written informed consent for the medical use of test results and participation of the children in this study were received from all of the children and their parents. The ADHD diagnosis was based on the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) using the Kiddie Schedule for Affective Disorders and Schizophrenia Present and Lifetime Version Korean Version (K-SADS-PL-K), which is a semi-structured clinical interview.

Children were excluded from the study if they had a history of congenital genetic disease, brain damage, neurological disorder, psychiatric disorder such as schizophrenia spectrum disorder, autism spectrum disorder, obsessive-compulsive disorder, major depressive disorder, bipolar disorder. Those with an IQ below 70 according to the Korean-Wechsler Intelligence Scale for Children-Fourth Edition (K-WISC-IV) were excluded from the study also.

The ADHD group included children who were diagnosed as ADHD based on the K-SADS-PL-K. Among the ADHD group, those who were receiving medication therapy already were assigned to the ADHD Combination group, while the others were assigned to the ADHD MNF alone group. The Neurotypical group included children who exhibited no abnormalities based on DSM-5 and had no personal history of any psychological disorder or accompanying disease. This study protocol was approved by the Institutional Review Boards of the Daegu Catholic University Medical Center (CR-19-064), Seoul National University Hospital (H-1905-145-1035) and Hanyang University Seoul Hospital (HYUH 2020–02-025-005), and complied with the Declaration of Helsinki (World Medical Association: Ethical Principles for Medical Research Involving Human Subjects, 1964). This study was also registered in the ClinicalTrials.gov with registration number NCT04469335 (14/07/2020).

Trial Design

This study employed a double-blinded randomized sham-controlled trial design to investigate the effect of MNF in ADHD and neurotypical children. Within each group, participants were randomly assigned to either the active or sham MNF intervention, carried out by a web-based randomization program of the Medical Research Collaborating Center (MRCC) at Seoul National University Hospital's Biomedical Research Institute (https://mrcc.snuh.org/), a center audited and recognized for its high-quality data management services in controlled clinical trials, as per the ECRIN Data Management Centre Certification program. Random allocation was maintained in a blinded state for the researchers, clinicians, children, and their parents until the end of the study. Prior to randomization, all participants underwent assessments which included clinical evaluations, and EEG which were repeated after the completion of 3-month intervention. Regarding medication usage, participants in the combination group continued their medication at the same dosage throughout the assessment period, ensuring consistency in treatment conditions and allowing for a clearer assessment of the MNF intervention's effects.

Intervention

The neurofeedback headsets, developed by OmniCNS (https://brain.omnifit.co.kr/en/index) and utilizing a dry EEG method, consisted of 2 channels at prefrontal Fp1 and Fp2 electrodes as per the 10–20 system, with a reference electrode located at A2 using conductive silicone earphone tip inserted into the ear canal. To treat artifacts electromyography in the

neurofeedback group, a band-pass filter using infinite impulse response Butterworth filters (High-pass filter: 1st order with fc = 2.6 Hz; Low-pass filter: 8th order with fc = 43 Hz) was applied, allowing a frequency range of 3–43 Hz. EEG signals were recorded at a rate of 250Hz and transformed into the frequency domain through Fourier transform, which assessed the power of a wide range of frequencies from theta (4–8Hz) to gamma (30–51Hz). EEG power was collected at 2-second intervals, and based on the level of attention (determined by Low beta + Middle beta / Theta Power), feedback was given in a game format, manifesting as various activities (levitation, running, turning a fan, lifting weights, bursting balloons). Each level of the game required a certain level of Low beta + Middle beta / Theta Power, with the game being divided into three levels. The sham neurofeedback was realized by displaying randomized game results, generated irrespective of actual TBR measurements. The distribution of the training result ratings was carefully adjusted to avoid extremes and maintain consistency in user experience, with the score range of 0 to 100 favoring median scores over the highest and lowest levels.

Each participant was provided with a MNF application and equipment and received approximately an hour of training on how to use them. During this training, the research team concluded that the participants were able to grasp the concept and adequately learned the neurofeedback processes involved in the study. Following the training, participants played neurofeedback games in the Omnifit Brain application using headsets provided and their own smartphones for a duration of 12 weeks. The neurofeedback games were conducted three times a week for 10–20 minutes each session, with two sessions per day. The game consisted of four different types, utilizing the theta/beta ratio. Participants were free to choose the game they found most interesting, and there were no restrictions on the type of game selected.

The implementation of the neurofeedback intervention could be confirmed through device connection and usage logs. If the participants encountered difficulties in using the application, they had the option to seek assistance from their parents. Every two weeks, the actual usage time was evaluated, and if the participation rate was less than 75%, encouragement was provided through telephone interviews to promote engagement. Additionally, an assessment of any discomfort experienced during usage was conducted.

Measures

Kiddie Schedule for Affective Disorders and Schizophrenia Present and Lifetime Version (K-SADS-PL)

K-SADS-PL is a tool developed by Kaufman et al and is a semi-structured interview designed to assess the current and lifetime diagnostic status of 32 child and adolescent psychiatric disorders, as well as the severity of symptoms, based on the DSM-IV diagnostic criteria.[22] The Korean version of K-SADS-PL was translated by Kim et al and has been validated for reliability and validity in assessing disorders such as ADHD, tic disorders, oppositional defiant disorder, depressive disorders, and anxiety disorders.[23]

Clinical Global Impression-Severity (CGI-S)

The Clinical Global Impression (CGI) rating scales were developed by Guy and are commonly used to assess symptom severity, treatment response, and the effectiveness of interventions for individuals with mental disorders.[24] CGI-S scale is a measure in which the clinician assesses the overall severity of a mental disorder, regardless of specific diagnosis. It is a single-item scale consisting of seven levels ranging from 1 normal to 7 most severe.

Korean ADHD Rating Scale (K-ARS)

The ADHD Rating Scale (ARS) is a behavioral assessment tool developed by DuPaul for evaluating ADHD symptoms in school-age children.[25] The scale consists of a total of 18 items based on the diagnostic criteria for ADHD in DSM-IV. The odd-numbered items measure inattention symptoms, while the even-numbered items assess hyperactivity-impulsivity symptoms. K-ARS was translated by So et al and has been shown to have good reliability and validity.[26]

Korean Wechsler Intelligence Scale for Children-Fourth Edition (K-WISC-IV)

The K-WISC-IV is a standardized tool designed to evaluate the intellectual abilities of children aged 6 to 16 years and 11 months.[27] It provides a comprehensive profile that includes an overall measure of intellectual functioning, known as the FSIQ.

Advanced Test of Attention (ATA)

ATA is a tool developed by Hong et al that evaluates sustained and selective attention abilities and impulse control in children older than 5 years old.[28] It serves as an evaluation tool to differentiate children with attention disorders such as ADHD. The ATA consists of visual and auditory tasks where stimuli are presented with a mix of target and non-target stimuli at regular intervals. The participants are instructed to respond only to the target stimuli using a keyboard or mouse. The assessment measures several variables, including omission errors to assess attentional lapses, commission errors to measure impulsivity, response time to evaluate information processing speed, and standard deviation of response time to assess consistency in attentional focus.

Stroop Test

The Stroop Test is a tool used to evaluate the efficiency of inhibitory processes in the frontal lobe.[29] It consists of three conditions. The first condition, known as the Word score, assesses the ability to read words as quickly as possible within a time limit of 45 seconds. The second condition, called the Color score, evaluates the ability to name the colors of color patches quickly. Lastly, the Color-Word score measures the ability to inhibit automatic responses and instead say the color of the ink while the word and color mismatch. The Korean version of the Stroop test has been standardized by Shin and Park.[30]

Children's Color Trails Test (CCTT)

CCTT evaluates functions related to the frontal lobe, such as visual-motor coordination, attention, visual scanning, and cognitive flexibility.[31] It is comprised of two parts: CCTT-1, where participants are required to connect numbers in ascending order like 1-2-3, and CCTT-2, where participants need to connect numbers in sequence while alternating colors like Pink1-Yello2-Pink3. The Korean version of the assessment was developed by Koo and Shin.[32]

EEG Recording and Pre-Processing

The EEG data were acquired using four different measuring systems: Compumedics Grael-4K System (https://www.compumedics.com.au/en/product-category/neurology-diagnostics/, Australia), Nihon Kohden Corporation Neurofax EEG-1200K System (https://us.nihonkohden.com/products, Japan), Grass Technologies Comet-Plus System (www.grasstechnologies.com, USA), and Ybrain MINDD-SCAN system (https://www.ybrain.com/en/product/professional/, Republic of Korea). The EEG data were recorded from 19 channels based on the international 10–20 system (Fp1, Fp2, F3, F4, F7, F8, Fz, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, O2) at a sampling rate of 500Hz. During the EEG assessment, participants were seated comfortably in a chair with their eyes closed, and the recording was performed for a duration of 5 minutes.

The EEG data underwent the following preprocessing steps. Firstly, the data were detrended to remove the DC component and normalization was done for comparability. Secondly, the data were resampled to 250Hz to adjust the sampling rate ensuring consistency across the data. Then, to achieve montage consistency in the EEG recordings, the data were re-referenced to an average reference. A 1–100Hz Bandpass filter and a 60Hz notch filter were applied and independent component analysis were performed to remove artifacts such as eye blinks, muscle and heart noises. The decomposed signals were labeled using the ICLabel algorithm and components which were most likely not from the brain such as muscle, eye, heart, line and channel noise were excluded.[33] Finally, clinical psychiatrists and EEG experts visually inspected the corrected EEG data for any remaining artifacts. All pre-processing steps were performed using the SciPy and MNE-Python software in Python. More than two minutes of artifact-free EEG were selected for the analysis.

Power-Spectrum Analysis of the EEG Recordings

Following five frequency bands were defined for spectral analysis: delta (1–4Hz), theta (4–8Hz), alpha (8–12Hz), beta (12–30Hz), gamma (30–51Hz). The power spectral analysis was performed by Welch's method using 1000ms time window, 800ms overlap, and Hamming window in MNE-Python software. The absolute powers of each frequency band were averaged over all time windows and frequencies.

Theta-Gamma Coupling (TGC) Analysis

The CFPAC between theta and gamma oscillations was assessed to investigate cross-frequency interactions. Phase of the theta signal (4–8Hz) and amplitude of the gamma signals (30–33Hz, 33–36Hz, 36–39Hz, 39–42Hz, 42–45Hz, 45–48Hz, 48–51Hz) were decomposed from the signals through the Hilbert transform. The phase-amplitude coupling between the

theta phase and each gamma amplitude were extracted using the Phase-Locking Value method. All calculations were conducted using the Tensorpac toolbox.[34]

Statistical Analysis

The demographic characteristics within each group were analyzed using independent *t*-tests and chi-square tests. To compare clinical variables within each ADHD group before and after the intervention, repeated-measures ANOVA was performed. Comparisons of TGC values among the three groups were conducted using one-way ANOVA, and pre–post differences within each group were assessed using paired *t*-tests. Statistical significance was defined as $p < 0.05$. Statistical analyses of EEG data were performed using SciPy in Python, while other data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 25.0 (SPSS Inc., Chicago, IL, USA).

Theta–gamma coupling (TGC) was designated a priori as the primary neurophysiological outcome, whereas clinical and cognitive measures were treated as secondary exploratory outcomes. The use of repeated-measures ANOVA and paired *t*-tests was chosen for consistency with prior neurofeedback studies and to preserve interpretability given the modest sample size and attrition. Future studies with larger and more balanced samples may benefit from employing mixed-effects modeling and corrections for multiple comparisons.

Results

Participants Characteristics

In the ADHD Combination group, initially 81 children were randomized into active and sham groups, with 52 (42 males, 10 females) eventually participating and maintaining their medication on average dose of atomoxetine 38.3mg or methylphenidate 31.6mg. Over the 3-month intervention period, there were 29 dropouts. The ADHD MNF Alone group started with 42 children randomized, with 26 (19 males, 7 females) completing the study, and 16 dropouts. In the Neurotypical group, from an initial 65 children randomized, 61 (30 males, 31 females) completed the intervention, with 4 dropouts (Figure 1). No significant differences in the sex ratio, age and Full Scale Intelligence Quotient (FSIQ) were found between the active MNF group and sham MNF group within each group (Table 1).

Comparisons Within Each Group

The repeated measure analysis of variance (ANOVA) for the ADHD Combination group revealed significant main effect of group for the Korean ADHD Rating Scale (K-ARS) inattention subscale ($F(1,49)=4.998$, $p=0.030$), indicating differences between the active MNF and sham MNF within the ADHD Combination group. However, no significant interaction effect between time and group was observed for any of the assessed clinical variables, suggesting that the differences between the active MNF and sham MNF did not vary across time points within the ADHD Combination group (Table 2).

In the ADHD MNF alone group, significant main effects of group were observed for the Advanced Test of Attention (ATA) auditory Omission error ($F(1, 24)=10.840$, $p=0.003$), suggesting that there was a significant difference between the active MNF and sham MNF in terms of the ATA auditory omission errors. However, none of the clinical variables analyzed demonstrated a significant interaction between time and group. This implies that the changes observed over time did not differ significantly between the active MNF and sham MNF in the ADHD MNF alone group (Table 3).

In summary, while some significant effects were observed in both ADHD groups, the lack of a significant interaction effect between time and group suggests that the active MNF did not have a superior impact compared to the sham MNF for the clinical variables.

In the Neurotypical group, significant main effects of group were observed for the K-ARS total ($F(1,59)=5.178$, $p=0.027$), K-ARS hyperactive/impulsivity subscale ($F(1,59)=5.191$, $p=0.026$), ATA visual commission error ($F(1,59)=6.184$, $p=0.016$), ATA auditory commission error ($F(1,59)=4.954$, $p=0.030$), ATA auditory response time standard deviation ($F(1,59)=5.140$, $p=0.027$), indicating differences between the active MNF and sham MNF within the Neurotypical group. Also, there were significant interactions between time and group for the ATA visual response time ($F(1,59)=4.727$, $p=0.034$), ATA auditory commission error ($F(1,59)=11.053$, $p=0.002$), which implied that the differences between the active MNF and sham MNF vary across time points within the Neurotypical group.

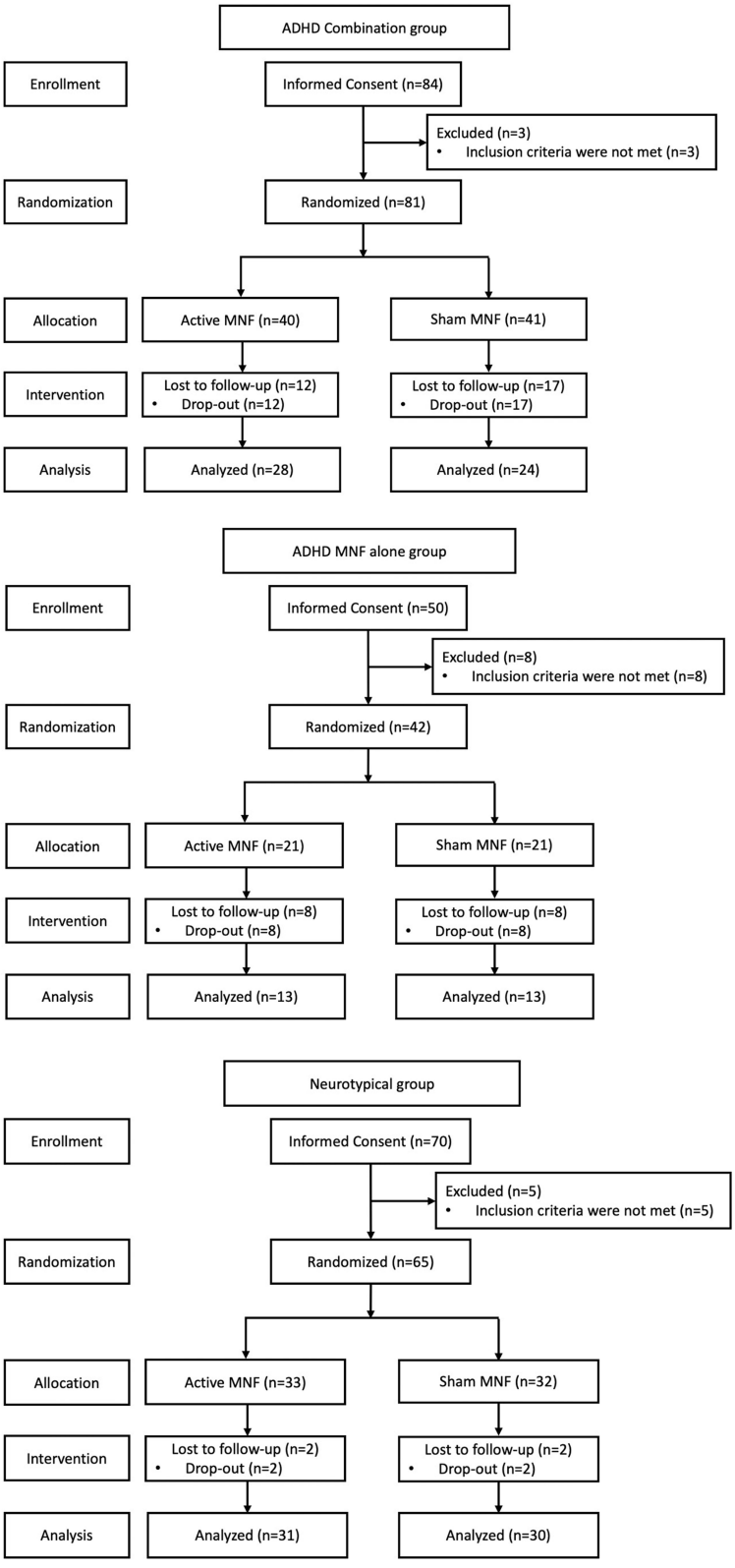

Figure 1 Flow diagram of study.
Abbreviations: ADHD, Attention-Deficit/Hyperactivity Disorder; MNF, Mobile Neurofeedback.

Table 1 Demographic Characteristics of ADHD and Neurotypical Children

	ADHD Combination			ADHD MNF Alone			Neurotypical		
	Active (n=28)	Sham (n=24)	p	Active (n=13)	Sham (n=13)	p	Active (n=31)	Sham (n=30)	p
Age	10.07 (1.90)	10.67 (1.71)	0.25	9.54 (1.81)	10.31 (1.80)	0.70	10.61 (1.80)	10.93 (1.84)	0.49
Sex			0.74			0.67			0.90
Male	22 (79%)	20 (83%)		9 (69%)	10 (77%)		15 (48%)	15 (50%)	
Female	6 (21%)	4 (17%)		4 (31%)	3 (23%)		16 (52%)	15 (50%)	
FSIQ	97.04 (14.36)	97.92 (17.69)	0.16	104.15 (13.78)	94.00 (14.07)	0.86	95.39 (15.87)	96.20 (12.21)	0.82

Note: Data are mean (SD), n (%).
Abbreviations: ADHD, Attention-deficit/hyperactivity disorder; MNF, Mobile neurofeedback; FSIQ, Full Scale Intelligence Quotient.

Table 2 Comparisons of Clinical Variables in ADHD Combination Group

	Active (n=28)		Sham (n=24)		F(group)	F(time)	F(g X t)
	Before	After	Before	After			
CGI-S	4.11 (0.32)	3.61 (0.83)	4.00 (0.00)	3.75 (0.44)	0.03	16.91*	1.88
K-ARS [a]							
Total	23.11 (9.97)	17.37 (8.17)	18.87 (11.07)	14.54 (8.78)	2.25	15.74*	0.31
Inatt	14.22 (5.47)	11.07 (4.54)	10.71 (5.85)	8.88 (5.27)	5.00*	11.27*	0.79
H/I	8.89 (6.00)	6.30 (4.82)	8.17 (5.78)	5.67 (4.71)	0.25	16.27*	0.01
ATA visual[†]							
Omission error	61.57 (17.35)	60.25 (20.08)	53.62 (11.94)	52.29 (15.66)	3.75	0.37	0.00
Commission error	61.32 (17.98)	58.96 (14.89)	60.04 (18.83)	59.50 (16.27)	0.01	0.42	0.16
Response time	67.04 (13.55)	66.68 (14.87)	63.13 (14.40)	64.63 (13.30)	0.80	0.08	0.21
Response time SD	63.89 (23.28)	60.64 (17.40)	54.42 (17.61)	55.83 (20.68)	2.04	0.15	0.94
ATA auditory[†]							
Omission error	63.11 (21.67)	61.25 (20.65)	70.58 (25.66)	64.21 (22.08)	0.81	3.10	0.93
Commission error	65.71 (20.04)	61.61 (18.26)	74.21 (24.01)	66.83 (22.40)	1.53	8.64*	0.70
Response time	57.29 (10.81)	57.75 (11.47)	56.25 (9.95)	59.25 (12.47)	0.01	1.63	0.87
Response time SD	48.68 (14.14)	46.04 (13.40)	48.71 (13.04)	47.63 (13.11)	0.05	1.85	0.34
Stroop[†]							
Word	41.75 (10.32)	45.93 (10.73)	42.75 (11.57)	50.42 (11.79)	0.90	31.23*	2.71
Color	44.82 (12.46)	47.75 (11.88)	49.38 (10.71)	54.00 (12.10)	3.05	11.36*	0.57
Color-Word	45.32 (12.54)	48.86 (13.38)	50.46 (11.10)	54.38 (13.34)	2.83	5.91*	0.02
CCTT[† a]							
CCTT-1	51.48 (12.39)	53.48 (12.93)	52.50 (11.35)	57.58 (7.03)	0.90	4.64*	0.88
CCTT-2	49.48 (12.98)	52.41 (9.65)	51.67 (9.10)	55.63 (8.93)	1.06	7.43*	0.17

Note: Data are mean (SD), *p<0.05, [†]T-score, [a]1 participant data were missing.
Abbreviations: ADHD, Attention-deficit/hyperactivity disorder; MNF, Mobile neurofeedback; CGI-S, Clinical Global Impression severity scale; K-ARS, Korean ADHD Rating Scale; Inatt, Inattention subscale; H/I, Hyperactivity/Impulsivity subscale; ATA, Advanced Test of Attention; SD, Standard deviation; CCTT, Children's Color Trails Test.

Table 3 Comparisons of Clinical Variables in ADHD MNF Alone Group

	Active (n=13)		Sham (n=13)		F(group)	F(time)	F(g X t)
	Before	After	Before	After			
CGI-S [a]	4.62 (0.51)	4.00 (0.71)	4.42 (0.52)	4.17 (0.58)	0.01	11.52*	2.05
K-ARS							
Total	28.23 (11.66)	24.08 (15.65)	28.08 (13.08)	22.23 (9.99)	0.05	8.18*	0.23
Inatt	15.92 (6.25)	13.85 (7.89)	15.38 (6.58)	12.92 (5.55)	0.09	5.88*	0.04
H/I	12.31 (6.20)	10.23 (8.39)	12.69 (6.64)	9.31 (4.94)	0.01	8.95*	0.51

(*Continued*)

Table 3 (Continued).

	Active (n=13)		Sham (n=13)		F(group)	F(time)	F(g X t)
	Before	After	Before	After			
ATA visual†							
Omission error	54.92 (13.31)	35.15 (21.52)	67.85 (21.27)	68.92 (23.16)	2.19	0.94	0.51
Commission error	60.15 (20.47)	61.08 (21.79)	64.23 (20.22)	62.85 (14.25)	0.18	0.01	0.12
Response time	66.77 (9.64)	65.23 (9.72)	72.92 (16.32)	72.15 (12.77)	2.36	0.24	0.03
Response time SD	62.92 (16.83)	62.77 (19.02)	70.15 (18.96)	69.92 (20.63)	1.30	0.00	0.00
ATA auditory†							
Omission error	55.31 (12.02)	56.00 (11.30)	75.38 (19.05)	70.77 (21.96)	10.84*	0.26	0.47
Commission error	66.77 (20.73)	61.92 (20.45)	75.00 (21.61)	71.08 (18.03)	1.36	2.53	0.03
Response time	53.69 (15.01)	52.15 (13.04)	58.77 (9.79)	56.69 (9.91)	1.18	1.06	0.02
Response time SD	49.31 (10.88)	49.63 (14.54)	57.23 (10.31)	53.62 (11.18)	2.29	0.45	0.64
Stroop†							
Word	43.00 (16.46)	47.69 (10.00)	43.15 (13.11)	44.54 (10.71)	0.10	2.68	0.80
Color	49.38 (10.15)	51.08 (12.78)	43.46 (8.78)	47.08 (11.73)	1.52	3.08	0.40
Color-Word	47.62 (11.86)	50.62 (12.49)	41.31 (8.17)	46.08 (11.37)	2.09	3.11	0.16
CCTT†							
CCTT-1	48.46 (13.21)	55.69 (8.99)	52.77 (9.26)	54.31 (7.32)	0.18	6.27*	2.64
CCTT-2	53.15 (8.84)	54.08 (8.95)	51.69 (14.05)	23.77 (9.90)	0.07	0.41	0.06

Note: Data are mean (SD), *p<0.05, †T-score, [a]1 participant data were missing.
Abbreviations: ADHD, Attention-deficit/hyperactivity disorder; MNF, Mobile neurofeedback; CGI-S, Clinical Global Impression severity scale; K-ARS, Korean ADHD Rating Scale; Inatt, Inattention subscale; H/I, Hyperactivity/Impulsivity subscale; ATA, Advanced Test of Attention; SD, Standard deviation; CCTT, Children's Color Trails Test.

Comparisons Between Groups and Within Each Group: TGC Values

When comparing the mean absolute power value for every frequency band between groups and within groups, significant differences were observed in TGC values. Before the MNF intervention, the Neurotypical group had higher TGC values in almost all brain regions than the other two ADHD groups (Figure 2). After the 3 months of MNF intervention, within the ADHD Combination group, the active MNF increased TGC values of Fp2, F3, F4, F8, P4, O1 and O2; whereas the sham MNF decreased TGC values of C4 and Cz. Within the ADHD MNF alone group, both active and sham MNF showed variability in outcomes, evidenced by the lack of a consistent trend across different brain regions and frequency

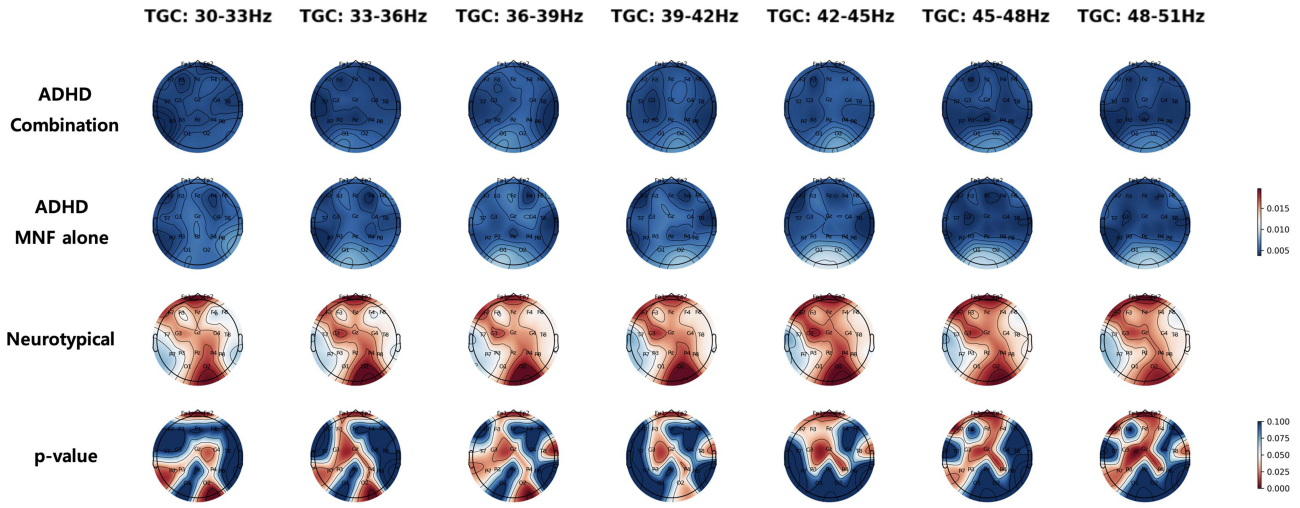

Figure 2 EEG analysis collected before MNF intervention in ADHD and Neurotypical group.
Abbreviations: EEG, Electroencephalography; MNF, Mobile Neurofeedback; ADHD, Attention-Deficit/Hyperactivity Disorder; TGC, Theta-gamma coupling.

https://doi.org/10.2147/NDT.S554756

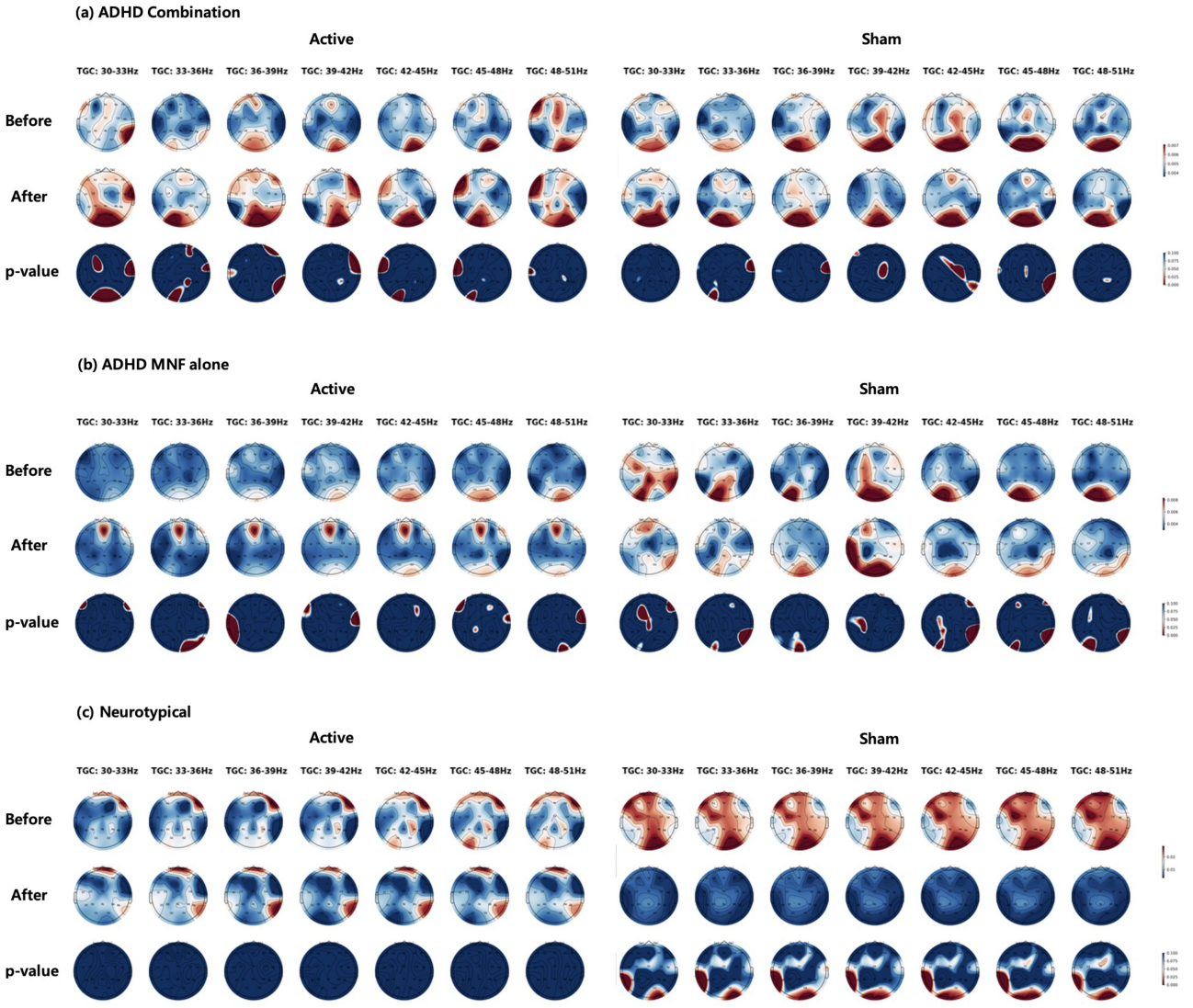

Figure 3 Comparisons of EEG analysis before and after the intervention in each group. (**a**) ADHD Combination (medication + MNF), (**b**) ADHD MNF alone, and (**c**) Neurotypical. For each panel, results are shown separately for Active MNF and Sham MNF at Before (baseline) and After (post-intervention, 12 weeks).
Abbreviations: EEG, Electroencephalography; MNF, Mobile Neurofeedback; ADHD, Attention-Deficit/Hyperactivity Disorder; TGC, Theta-gamma coupling.

bands. Due to such mixed results, no definitive pattern or direction could be confirmed. Lastly, within the Neurotypical group, the active MNF did not produce any significant changes in TGC values, while sham MNF decreased TGC values of P7, Cz and O2 (Figure 3).

Discussion

The present study aimed to investigate the therapeutic effect of MNF in ADHD and neurotypical children compared to the sham MNF. The study design involved three groups: ADHD Combination group, ADHD MNF alone group, and Neurotypical group. Each group underwent a 3-month of MNF intervention, either with active MNF or sham MNF intervention. The impact of this intervention was assessed via EEG TGC values and clinical variables before and after the intervention. Comparing the TGC values change between two ADHD groups, the ADHD Combination group showed increases of TGC values in various channels by the active MNF but ADHD MNF alone group did not show a significant effect compared to the sham MNF. In the Neurotypical group, the active MNF did not result in any significant alterations in TGC values, whereas the sham mobile neurofeedback led to a decrease of TGC values at some regions.

For clinical variables, the ADHD Combination group showed no significant interaction between time and group for any of those evaluated, indicating that the differences between active and sham MNF did not vary across time points within the ADHD Combination group. These findings are consistent with a prior randomized controlled trial (RCT), which found that neurofeedback was not superior to sham neurofeedback in reducing symptoms of ADHD for 15 weeks in children who were already taking medication.[35] Other RCTs have indicated that a combination of neurofeedback and medication significantly alleviated ADHD symptoms, as reported by parents or self-assessments for 10 weeks of follow-up. Interestingly, this combination did not show a significant difference in effectiveness when compared to either neurofeedback or medication used alone also.[36,37] However, with the 6-month follow-up assessment reported by parents, teachers and oneself, combination of neurofeedback and medication showed significant improvements in inattention symptoms compared to the either alone, showing mixed results compared to the previous studies.[38]

In the ADHD MNF alone group, none of the clinical variables analyzed showed a significant interaction between time and group meaning that the changes observed over time were similar for both groups. In a double-blind RCT involving children with ADHD, neurofeedback did not show a specific effect in improving inattention compared to a sham condition at the end of 14-week period treatment, which finding is consistent with the present study.[39]

Within the Neurotypical group, significant interaction between time and group were observed for the ATA visual Response time, ATA auditory Commission error, suggesting that the active MNF did not demonstrate better results than sham. In a study of double-blind single-session neurofeedback training for healthy subjects, despite some improvements in specific cognitive tasks by the neurofeedback training group, many behavioral assessments, including the Stroop test, showed no significant difference between the neurofeedback and control group.[40] Also in a single-blind, sham-controlled study involving healthy participants, investigated the impact of alpha down-regulation neurofeedback training on implicit motor learning in a complex motor task, finding that while there was no significant enhancement of motor learning.[41] Consistent with previous research, this study also could not identify any benefits of MNF in healthy participants.

This study has similarities to previous studies, and the overall conclusions did not reveal significant differences between the active and sham MNF in terms of clinical variables. However, when conducting TGC analysis after only 3 months, differences were observed, specifically in the ADHD Combination group. After the 3-month MNF intervention, the active MNF increased TGC values in specific brain regions (Fp2, F3, F4, F8, P4, O1 and O2) within the ADHD Combination group. These findings suggest that the observed enhancement in theta–gamma coupling (TGC) reflects early neural plasticity or engagement with neurofeedback training rather than direct evidence of clinical improvement. TGC modulation may thus represent a mechanistic biomarker indicating early neurophysiological adaptation that precedes measurable behavioral changes. Within the ADHD MNF Alone group, both active and sham MNF yielded mixed results with no definitive pattern.

In a previous RCT with a 6-month follow-up which is longer than the present study, three different treatment approaches were examined: a combination of medication and neurofeedback, medication alone, and neurofeedback alone. Ratings provided by parents and teachers in all three treatment groups demonstrated significant improvements in inattention symptoms, with the combination group achieving higher scores. These clinical effects were sustained at the 6-month follow-up assessment.[38] Combination treatment may offer several advantages, such as the potential for reducing medication dosage and minimizing the time parents and teachers need to spend on behavior monitoring. It should be noted that non-specific factors, including parental support and cognitive training, may contribute to the positive behavioral effects observed during neurofeedback treatment. In contrast to the previous study, this current study conducted a reevaluation after only 3 months, and no significant differences were observed in clinical variables. However, there were some changes observed within the 3-month timeframe, specifically in the TGC values obtained from EEG. TGC has been known to be more accurate than QEEG in differentiating between ADHD and typically developing children,[19] and it is also recognized as reflecting attentional shifting in ADHD children.[20] Additionally, since neurofeedback itself operates through changes in EEG, it is possible that the initial assessment of neurofeedback's effects was observed through TGC. The neurophysiological changes induced by neurofeedback may indeed be reflected in TGC.[42] If a follow-up period of 3 months were conducted, differences may have been observed in other clinical indicators such as ATA. This suggests the need for further research. However, the fact that we were able to observe neurophysiological changes induced by neurofeedback through TGC during the short period of 3 months highlights the value of this study.

Additionally, regarding the point in time before the MNF intervention, the Neurotypical group exhibited higher TGC values in nearly all brain regions compared to the other two ADHD groups. TGC was identified in the human neural cortex, which serves as physiological evidence of communication and interaction between groups of neurons.[18] One research which compared the mean absolute power value of specific frequency bands in QEEG, no difference was found between individuals with ADHD and the normal control. However, a difference in TGC was observed between the two groups suggesting that children with ADHD exhibit reduced interactions within the functional neuronal system, particularly in frontal-subcortical interactions, during cognitive processes compared to individuals without ADHD.[20] The present study supports previous research by demonstrating that the Neurotypical group exhibited higher TGC values compared to individuals with ADHD.

This study acknowledges several limitations. First, the 3-month intervention period may have been too short to detect sustained clinical improvements in ADHD, even though neurophysiological markers such as theta–gamma coupling (TGC) may respond earlier. Second, the relatively high dropout rates, particularly in the ADHD groups, could have reduced statistical power and introduced potential selection bias. Third, repeated-measures ANOVA and paired *t*-tests were used for comparability and clarity; however, future research employing mixed-effects modeling and corrections for multiple comparisons would strengthen analytic robustness. Finally, TGC was designated as the primary neurophysiological outcome, while clinical and cognitive measures were treated as secondary exploratory outcomes. Adherence and attrition were monitored throughout the study using device usage logs, and missing data were handled using a complete-case analysis approach.

In conclusion, in the ADHD MNF alone group, active MNF did not show a significant effect. However, as an additive therapy for ADHD medication, MNF has been shown to induce neurophysiological changes, which could be confirmed by TGC values. Additionally, when observing a decrease in TGC values in the Neurotypical group after sham MNF, caution is required when conducting neurofeedback without appropriate feedback, as it may cause unexpected results. Several advantages of this study include the use of mobile devices for neurofeedback training, which differs from traditional neurofeedback methods. Additionally, efforts were made to ensure compliance with the intervention by monitoring device connections and usage logs. Another strength is the inclusion of not only children with ADHD but also neurotypical children, allowing for a comprehensive interpretation of the results. Furthermore, the inclusion of a sham design enhances the study by enabling a more distinct assessment of neurofeedback effects. Addressing the study's limitations—such as the relatively short 3-month period, attrition, and limited statistical power—in future research will enhance the validity and clinical relevance of the findings. A larger sample size, longer follow-up periods, and replication across multiple sites using more robust statistical models are needed to confirm the stability of these results and to explore the predictive validity of TGC as a biomarker. These efforts will contribute to a more precise understanding of the neurophysiological mechanisms of mobile neurofeedback and its potential clinical applications in ADHD.

Data Sharing Statement

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

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Disclosure

The authors report no conflicts of interest in this work.

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