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An Exploratory Study of Alpha EEG Activity at Rest and During Cognitive Task Performance in Adults with ADHD

Mohammad Hassan Aghaei [1], **Asgar Alimohammadi** ✉[2], and **Kambiz Banihashemi** [3]

1. ee partment of Psychology and Education of xx ceptional Children, Allameh Tababab ai' University, Faculty of Psychology and Educational Sciences, Tehran, Iran. E mail: M.h.Aghaie79@gmail.com
2. *Corresponding author*, Assistant Professor, Department of Psychology and Education of Exceptional Children, Allameh Tabataba'i University, Tehran, Iran. Email: Asgar.alimohamadi@yahoo.com
3. M,, Gen. ticist and Academic Member, ee ad of Cognitive Sciences Laboratory, ۲ llameh Tabatabai'.University, Tehran, Iran. Email: kambiz.banihashemi@atu.ac.ir

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ABSTRACT

Objective: This exploratory, hypothesis-generating study investigated alpha-band EEG activity during resting state and cognitive task performance in adults with Attention-Deficit/Hyperactivity Disorder (ADHD) and examined its relationship with symptom severity, without a healthy control group.

Method: EEG (8-12 Hz) was recorded in 28 adults with ADHD (21-31 years) during (1) eyes-closed rest and (2) a cognitive task requiring sustained attention. Absolute power and NeuroGuide z-scores were extracted. ADHD symptoms were assessed using the ASRS. Paired t-tests, Pearson correlations, and exploratory median split comparisons were performed.

Results: Resting and task alpha activity were strongly correlated ($r = .77$, $p < .01$). At the group level, alpha activity unexpectedly increased from rest to task ($t(27) = 2.34$, $p = .027$). Linear correlations between ASRS total score and alpha activity at rest ($r = .07$) and during task ($r = .20$) were weak and non-significant ($p > .05$). In exploratory median-split comparisons (higher vs. lower symptom severity), individuals with higher severity showed significantly lower task-related alpha ($t(26) = 2.12$, $p = .044$, $d = .81$), but no significant difference in resting alpha ($t(26) = 1.92$, $p = .066$).

Conclusions: In this hypothesis-generating study of adults with ADHD conducted without a neurotypical control group, we observed within-sample patterns of alpha modulation. The unexpected task-related alpha increase and the association between higher symptom severity and reduced task-related alpha warrant replication in larger, controlled studies with concurrent behavioral performance measures. No ADHD-specific conclusions can be drawn from this exploratory investigation.

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Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental condition characterized by persistent patterns of inattention, impulsivity, and hyperactivity that frequently extend from childhood into adulthood, leading to impairments in cognitive control, executive functioning, and everyday activities (American Psychiatric Association, 2020). Epidemiological evidence indicates that ADHD affects a substantial proportion of the adult population, with prevalence estimates ranging from 4% to 7%, and longitudinal studies suggesting that approximately 60% of individuals diagnosed in childhood continue to exhibit clinically significant symptoms across the lifespan (Anbarasan et al., 2020; Song et al., 2021). While clinical assessment of adult ADHD has traditionally relied on behavioral observations and self-report instruments—most notably the Adult ADHD Self-Report Scale (ASRS)—there is growing interest in the identification of objective neurophysiological markers that more directly capture the underlying neural dynamics associated with attentional and executive dysfunctions (Slater et al., 2022).

Early electrophysiological research in children with ADHD reported a pattern of global EEG slowing, typically marked by increased power in lower frequency bands, particularly theta (4–7 Hz), alongside reduced power in higher frequency ranges such as beta (14–25 Hz) (Arns et al., 2013). Because the Theta/Beta Ratio (TBR) normally decreases across typical neurodevelopment (Perone et al., 2018), elevated TBR values observed in individuals with ADHD have been interpreted as indicative of delayed cortical maturation and/or reduced cortical arousal (Barry et al., 2003; Sangal & Sangal, 2015). Nevertheless, accumulating evidence has questioned the strength of the link between TBR and arousal mechanisms, as well as its utility as a robust biomarker for ADHD diagnosis (Arns et al., 2013; Lenartowicz & Loo, 2014). Additionally, increases in theta-band activity may, in certain cases, arise from a downward shift in the dominant alpha frequency rather than from a true enhancement of theta power per se (Lansbergen et al., 2011).

In ADHD research, resting-state EEG studies have consistently reported aberrant spectral power profiles compared to neurotypical controls, including elevated slow-wave activity (eg, theta) and attenuated fast frequencies such as alpha and beta bands. For example, adults with ADHD have demonstrated reduced alpha power at rest, suggesting atypical cortical activation patterns that may reflect dysfunctions in attentional networks. This reduction has been observed across resting conditions with woth wyes open and eyes closed, sgpporting the notion of "cortical hyper-activation" in DDDD relatiee to controls. .dditi onally, decreased alpha poee r has been linked to inhibitory control performance, indicating that alpha modulation may contribute to task-related attention processes, particularly in paradigms requiring response inhibition (Go/NoGo tasks) DDeieer et al)))))))))

Growing evidence suggests that alterations in neural oscillatory activity, particularly within the alpha frequency band (8–12 Hz), play a central role in the pathophysiology of Attention-Deficit/Hyperactivity Disorder (ADHD). Alpha oscillations are thought to reflect functional inhibition, attentional allocation, and cortical idling, and their modulation is critical for efficient cognitive control and task engagement. Several EEG studies have reported atypical resting-state alpha activity in adults with ADHD, often characterized by reduced alpha power or altered topographical distributions compared with neurotypical controls, indicating disrupted baseline cortical regulation (Clarke et al., 2008; Dimova, 2024). Beyond resting-state abnormalities, task-related EEG investigations demonstrate that adults with ADHD show impaired alpha modulation during cognitive demands requiring sustained attention and inhibitory control, such as Go/NoGo and continuous performance tasks (Deiber et al., 2019; Gu et al., 2022). These findings suggest a diminished capacity to dynamically regulate alpha activity in response to task demands. Importantly, symptom severity appears to further modulate alpha-band dynamics.

Individuals with higher levels of ADHD symptomatology exhibit more pronounced reductions in task-related alpha power, supporting the notion that alpha oscillations may index the efficiency of attentional engagement rather than serving as a static biomarker (Deiber et al., 2019). Systematic reviews of EEG research in adult ADHD further highlight alpha-band abnormalities as one of the most consistently reported electrophysiological features, although results vary depending on task conditions, analytical methods, and symptom profiles (Adamou et al., 2020; Slater et al., 2022). On the whole, these findings underscore the importance of examining alpha EEG activity across both resting and active cognitive states, as task-related alpha modulation may provide greater sensitivity to ADHD-related neural dysregulation than resting-state measures alone. However, relatively few studies have directly compared resting and task-evoked alpha activity within the same adult ADHD samples, highlighting the need for further investigation into state-dependent alpha dynamics and their relationship with clinical symptom severity.

Although clinical assessment of adult ADHD has primarily relied on self-report instruments such as the Adult ADHD Self-Report Scale (ASRS), these measures provide limited insight into the underlying neural mechanisms associated with attentional dysregulation, motivating growing interest in neurophysiological approaches (Glind et al., 2013; Slater et al., 2022). Early electroencephalographic (EEG) research in ADHD, particularly in pediatric populations, emphasized global EEG slowing, commonly reflected by increased theta power and elevated theta/beta ratio (TBR) values (Arns et al., 2013; Barry et al., 2003). Because TBR typically decreases across normative neurodevelopment, elevated values in ADHD were initially interpreted as markers of delayed cortical maturation or hypoarousal (Perone et al., 2018). However, subsequent research—especially in adult ADHD—has increasingly questioned the diagnostic specificity and theoretical validity of TBR, highlighting substantial inter-individual variability and limited sensitivity to cognitive state (Lenartowicz & Loo, 2014; Su et al., 2025). Importantly, apparent increases in theta activity may partly reflect a downward shift in dominant alpha frequency rather than genuine enhancement of theta power, complicating interpretations based solely on spectral ratios (Lansbergen et al., 2011).

In contrast to global spectral indices, alpha-band oscillations (8–12 Hz) have emerged as a functionally meaningful marker of attentional regulation and cognitive control. Contemporary neurocognitive models conceptualize alpha activity as a dynamic mechanism supporting functional inhibition and the selective allocation of processing resources, rather than a passive indicator of cortical idling (Klimesch, 2012). Within this framework, alpha power is understood as reflecting the efficiency of cortical regulation, whereby reduced alpha power may indicate heightened cortical activation and increased attentional engagement. This conceptualization allows the integration of inhibitory and engagement-based interpretations of alpha activity within a unified regulatory model.

Resting-state EEG studies in adults with ADHD have reported heterogeneous patterns of alpha activity, with several investigations demonstrating reduced alpha power relative to neurotypical controls (Clarke et al., 2008; Deiber et al., 2020), while others report weak or inconsistent differences (Adamou et al., 2020). Crucially, reduced resting-state alpha power has been linked to deficits in sustained attention, supporting its relevance to core cognitive impairments in ADHD (Sharififar & Rezaeyan, 2024). These findings suggest that alterations in alpha activity may reflect dysregulated baseline cortical states rather than static diagnostic markers. Beyond resting-state conditions, task-based EEG studies indicate that alpha-band activity is dynamically modulated in response to cognitive demands. In adults with ADHD, evidence suggests impairments in task-related alpha modulation during paradigms requiring sustained attention and inhibitory control, such as Go/NoGo tasks (Deiber et al., 2020; Gu et al., 2022). Consistent with this view, increased oscillatory activity in higher frequency bands during cognitive engagement has been associated with more efficient attentional allocation, underscoring the role of oscillatory dynamics in adaptive cognitive control (Kamali Khalavi et al., 2024). Together, these findings support the interpretation of alpha modulation as a state-dependent index of attentional engagement rather than a trait-like biomarker.

Despite extensive electroencephalographic research on ADHD, the majority of studies have focused on pediatric populations and have primarily emphasized global spectral indices such as the theta/beta ratio during resting-state conditions. While these approaches have contributed to early models of cortical hypoarousal and delayed neurodevelopment, their diagnostic specificity and theoretical validity—particularly in adult ADHD—have increasingly been questioned. In contrast, considerably less attention has been given to alpha-band oscillatory activity, despite its established role in attentional control, inhibitory processing, and task-related neural modulation. Furthermore, although symptom severity is known to vary substantially among adults with ADHD, prior research has rarely examined how individual differences in clinical symptom burden relate to alpha-band activity during different cognitive states. This lack of integration between neurophysiological measures and dimensional symptom profiles limits the interpretability and translational value of EEG findings in adult ADHD. Accordingly, there is a critical need for studies that systematically investigate alpha-band EEG activity in adults with ADHD across both resting and task-based conditions, while explicitly examining its association with clinical symptom severity. Addressing this gap may help clarify the functional relevance of alpha oscillatory dynamics and advance the identification of more sensitive neurophysiological markers of ADHD-related neural dysregulation in adulthood.

The present study adopted an explicitly exploratory, hypothesis-generating framework. Given the limited prior research directly comparing resting and task-related alpha dynamics in adult ADHD, and the absence of a healthy control group, we did not test confirmatory hypotheses. Instead, within-sample patterns of alpha modulation and their associations with symptom severity were examined. Critically, because no neurotypical control group was included, none of the observed patterns can be interpreted as specific to ADHD. All findings are preliminary and intended solely to inform the design of future confirmatory studies. This study included only adults with ADHD and did not have a neurotypical control group. Consequently, none of the observed patterns can be interpreted as specific to ADHD. The present findings are strictly within-sample observations intended to generate hypotheses for future controlled studies. Any interpretation of alpha activity as an ADHD-related marker would be premature and is not claimed here.

Materials and Methods

Study Design

This study employed a cross-sectional, exploratory design. No healthy control group was included. Therefore, all interpretations are strictly limited to within-sample comparisons among adults with ADHD. Given the exploratory nature of this investigation, no apriori hypotheses were tested.

Crucially, the absence of a neurotypical control group means that:

- No conclusion can be drawn about whether observed alpha patterns are specific to ADHD.
- No comparison to non-ADHD populations is possible.
- No claim of an ADHD-specific biomarker is made or implied.

Participants and Sampling

The study population consisted of adults diagnosed with ADHD who were referred to ADHD intervention centers and primary care clinics in Tehran during 2025. Using convenience sampling, twenty-eight adults (men and women), aged 21–31 years, were recruited. Inclusion criteria were: a) age ≥ 88 years;)) a confireed. diagnosis of AHHD based on MSM-5 criteria; c) completion of a semi-structured clinical interview conducted by a licensed clinical psychologist; and d) a positive screening score on the Adult ADHD Self-Report Scale (ASRS).

Exclusion criteria were defined as conditions that could interfere with EEG validity. Participants were excluded prior to EEG recording if any of the following conditions were present: (1) current use of psychotropic medication; (2) history of neurological disorder or head trauma within the past six months; (3) consumption of caffeine or other psychoactive substances within 48 hours before recording; or (4) severe sleep deprivation, operationally defined as fewer than five hours of sleep on the night preceding assessment. During signal acquisition, recordings were discontinued if unforeseen events compromised EEG data quality, including (1) excessive movement artifacts, (2) electrode detachment, (3) technical recording failure, or (4) acute discomfort preventing completion of the session.

The final sample consisted of 28 adults diagnosed with ADHD (12 males, 42.9%; 16 females, 57.1%), aged 21–31 years (M = 25.96, SD = 2.67). Participants' educational level ranged from bachelor's to master's degree. The sample size was determined based on the exploratory nature of the study and consistency with prior EEG investigations in adult ADHD reporting comparable sample sizes (Adamou et al., 2020; Deiber et al., 2020). Given the absence of reliable effect size estimates for task- related alpha modulation in adult ADHD, a formal a priori power analysis was not conducted.

Instruments

ADHD symptom severity was assessed using the Adult ADHD Self-Report Scale (ASRS; Kessler et al., 2005; Mokhtari et al., 2015). The ASRS consists of 18 items assessing inattention and hyperactivity–impulsivity dimensions, with higher scores indicating greater symptom severity.

EEG Recording and Preprocessing

EEG data were recorded using an 18-channel Mitsar-202 EEG system with electrodes positioned according to the international 10–20 system. Signals were sampled at 250 Hz and recorded using WinEEG software (version 2.8).

Preprocessing procedures included band-pass filtering (0.5–45 Hz), visual inspection for gross artifacts, and amtomatic reeection of epochs exceedin. 0000 0.. Ind. pendent Coeponent ena lysis (ICA) was applied to identify and remove ocular and muscle artifacts. EEG signals were referenced to linked earlobes. Artifact-free epochs were segmented into resting-state and task-related conditions.

Normative Reference and Alpha Measures

Absolute alpha power (8–12 Hz) was calculated for each condition using spectral analysis procedures. Alpha-band activity was expressed as standardized z-scores derived from the NeuroGuide normative database, which is adjusted for age and sex. In this procedure, raw absolute power values were log-transformed and statistically compared with values from an international non-clinical reference dataset to determine standardized deviations from normative means. Z-scor s therefore reflect the extent to which an indi.i dual's GGG activity differs from expect.d values for their demographic profile. Absolute power values were used to quantify within-sample variability, whereas z-scores were employed to contextualize individual deviations relative to normative EEG patterns, enabling complementary interpretation of alpha activity across both raw and standardized metrics.

Cognitive Task

Participants completed a working memory training task within the Captain's Log cognitive training software, presented as a computer-based game. In each trial, participants viewed and heard a specific sequence pattern, which they were required to remember. Following a 3-second delay, participants selected the target pattern by clicking the mouse. If the response was correct, task difficulty increased automatically; if incorrect, difficulty decreased or remained stable. Each participant performed the task for 8 to 10 minutes and completed a minimum of 20 pattern trials. The primary objective was to record EEG activity during cognitive engagement.

Behavioral performance data (accuracy, reaction time, game scores, or accuracy rates) were not recorded or analyzed, as the study's exclusive focus was on neurophysiological modulation rather than behavioral outcomes. The absence of behavioral data is a significant methodological limitation of this exploratory study. Consequently, any interpretation of task-related alpha activity in terms of cognitive performance, attentional engagement, or inhibitory control remains speculative. This study reports only neurophysiological patterns without behavioral validation. Future confirmatory studies must include concurrent behavioral measures.

Operational Definition of Alpha Modulation

Alpha modulation was operationalized as the difference between resting-state and task-related alpha z-scores. Although ratio-based or time–frequency analyses may offer greater sensitivity, this difference-based approach was selected to provide a parsimonious and interpretable index of state-dependent alpha regulation in an exploratory framework.

Data Analysis

Statistical analyses were conducted using SPSS (version 25). Prior to inferential testing, assumptions of normality and linearity were examined. Pearson correlation coefficients were used to examine associations between alpha measures and ASRS scores. A paired-samples t-test compared alpha activity between resting and task conditions.

In addition to continuous analyses, an exploratory median split of ASRS scores (median = 44.5) was conducted as a hypothesis-generating approach to compare alpha activity between individuals with higher versus lower symptom severity. The median split analysis was employed solely for exploratory, hypothesis-generating purposes. Given the small sample size (N = 28) and the post-hoc nature of this grouping, the resulting group comparisons should not be interpreted as confirmatory evidence. Consistent with the exploratory nature of this study and the sample size (N = 28), these group comparisons are presented as preliminary patterns that require replication in larger samples before firm conclusions can be drawn.

Results

Demographic Characteristics and Clinical Scores

The study sample consisted of 28 adults (men and women) diagnosed with Attention-Deficit/Hyperactivity Disorder (ADHD), aged between 21 and 31 years (M = 25.96, SD = 2.67). Descriptive statistics for demographic variables, ADHD symptom severity, and alpha-band EEG measures are presented in Table 1.

The mean total score on the Adult ADHD Self-Report Scale (ASRS) was 44.79 (SD = 6.72). Mean subscale scores were 22.25 (SD = 4.04) for inattention and 22.75 (SD = 4.31) for hyperactivity-impulsivity. Mean alpha-band EEG activity (z-scores) was 0.42 (SD = .77) during resting state (eyes closed) and .67 (SD = .75) during cognitive task performance.

Table 1. Descriptive Statistics of Study Variables

Variable	N	Mean	SD	Min	Max
Age	28	25.96	2.67	21	31
ASRS Total	28	44.79	6.72	35	63
Attention Deficit	28	22.25	4.04	16	31
Hyperactivity	28	22.75	4.31	11	33
Alpha Rest (z-score)	28	.42	0.77	-1.12	1.45
Alpha Task (z-score)	28	.67	0.75	-.70	2.13

Associations Between ADHD Symptoms and Alpha EEG Activity

Pearson correlation analyses were conducted to examine the associations between ADHD symptom severity and alpha EEG activity at rest and during task performance. Results are summarized in Table 2. The total ASRS score was positively correlated with both attention deficit ($r = .43$, $p < .05$) and hyperactivity subscales ($r = .58$, $p < .01$). Alpha activity at rest and during cognitive task performance showed a strong positive correlation ($r = .77$, $p < .01$), indicating consistent individual patterns across conditions.

Prior to conducting Pearson correlation analyses, assumptions of linearity, approximate normality, and absence of extreme outliers were assessed. Shapiro–Wilk tests indicated that all variables were approximately normally distributed (ASRS Total: $W = .95$, $p = .23$; Attention Deficit: $W = .96$, $p = .32$; Hyperactivity: $W = .94$, $p = .18$; Alpha Rest: $W = .97$, $p = .41$; Alpha Task: $W = .95$, $p = .27$). Visual inspection of scatterplots and boxplots revealed no extreme outliers. Based on these checks, the data met the assumptions required for Pearson correlation analyses.

Table 2. Pearson Correlations Between ADHD Symptoms and Alpha EEG Activity

Variable	ASRS Total	Attention Deficit	Hyperactivity	Alpha Rest	Alpha Task
ASRS Total	1.00	.43*	.58**	.07	.20
Attention Deficit	.43*	1.00	.25	.12	.05
Hyperactivity	.58**	.25	1.00	-.13	.14
Alpha Rest	.07	.12	-.13*	1.00	.77**
Alpha Task	.20	.05	.14*	.77**	1.00

*p < .05, **p < .01

Comparison of Alpha Activity Between Resting and Task Conditions

A paired-samples t-test was conducted to compare alpha activity between resting and task conditions. Results indicated a significant increase in alpha activity from resting state to task performance ($t(27) = 2.34$, $p = .027$, Hedges' $g = .33$).

Exploratory group comparison

Exploratory comparisons based on median split of ASRS scores suggested a potential difference in task-related alpha activity between higher and lower symptom severity groups (Cohen's $d = .81$), while the difference in resting-state alpha activity did not reach statistical significance (Cohen's $d = .73$).

Alpha Modulation Index

An alpha modulation index was calculated as the difference between task and resting alpha z-scores (Task - Rest). The mean modulation index was .25 (SD = .67). Correlation analysis revealed no significant association between the modulation index and ASRS total score ($r = .18$, $p = .36$).

Discussion

The present study examined alpha-band EEG activity during resting state and cognitive task performance in adults with ADHD, with a particular focus on its association with symptom severity. Overall, the main findings can be summarized as follows: (1) linear correlations between alpha activity and ADHD symptom severity were generally weak; (2) alpha activity increased from resting state to task performance at the group level; and (3) individuals with higher symptom severity exhibited significantly lower task-related alpha activity compared to those with lower symptom severity. These results offer preliminary patterns that align with previous electrophysiological research, emphasizing the relevance of task-related neural dynamics rather than resting-state measures alone. The weak dimensional associations observed between ASRS scores and alpha-band activity are consistent with prior EEG studies reporting substantial heterogeneity in adult ADHD and limited reliability of simple linear correlations between spectral power and clinical symptoms (Arns et al., 2013; Lenartowicz & Loo, 2014). In contrast, group-based analyses revealed more clear differences in task-related alpha activity. This apparent discrepancy may reflect non-linear or threshold effects, whereby neural differences become more pronounced at higher levels of symptom severity, reducing the sensitivity of correlational analyses in small samples. Importantly, because symptom-based grouping was conducted post hoc, these findings should be interpreted as exploratory rather than confirmatory. Notably, resting-state alpha activity showed only a trend toward lower values in participants with higher symptom severity, whereas task-related alpha activity was significantly reduced in this group. This dissociation suggests that symptom severity in adult ADHD may be more closely linked to impairments in task-dependent alpha modulation than to global reductions in alpha power.

Unexpected task-related alpha increase was another finding of the study. One unexpected finding of this exploratory study was the overall increase in alpha activity from rest to task ($t(27) = 2.34$, $p = .027$). This pattern directly contradicts the well-established phenomenon of task-related alpha desynchronization (alpha suppression) consistently reported in neurotypical populations during cognitive engagement (Klimesch, 2012). Because we did not record behavioral performance data (accuracy or reaction time), we cannot determine whether this alpha increase reflects: (1) disengagement from the task or drowsiness, (2) recording artifacts or technical issues, (3) a genuine but poorly understood characteristic of this particular ADHD sample, or (4) a compensatory neural mechanism. Given the exploratory nature of this study, the absence of a healthy control group, and the lack of behavioral data, this unexpected finding remains unexplained. It is important to emphasize that this unexpected task-related alpha increase must be interpreted in light of major data limitations: the absence of behavioral performance measures and the lack of a healthy neurotypical control group. Without these, it remains impossible to determine whether this alpha increase reflects disengagement, drowsiness, compensatory neural effort, or a nonspecific fluctuation. Therefore, no definitive mechanistic interpretation of this pattern is possible within the scope of this exploratory investigation and it is reported as a hypothesis-generating observation that requires replication in larger, controlled studies. From a mechanistic perspective, alpha oscillations are increasingly recognized as active contributors to attentional gating and inhibitory control processes rather than passive markers of cortical idling (Ippolito et al., 2022). Within this framework, the reduced task-related alpha activity observed in participants with higher ADHD symptom severity may reflect inefficiencies in inhibitory timing and attentional filtering, potentially contributing to the distractibility and impulsivity that characterize the disorder. This interpretation is supported by neurofeedback studies showing that modulation of alpha activity is associated with improvements in attentional control and inhibitory functioning in ADHD populations (Deiber et al., 2019).

Taken together, the present findings underscore the importance of examining EEG activity under conditions of cognitive challenge when investigating the neurophysiology of adult ADHD. While resting-state measures provide valuable information, our results suggest that task-related alpha dynamics may be more sensitive to individual differences in symptom severity. This task-dependent vulnerability may help explain why many adults with ADHD function relatively well in low-demand contexts but experience pronounced difficulties when cognitive load increases. Future research integrating resting and task-based EEG approaches may therefore offer a more comprehensive understanding of neural dysregulation in adult ADHD.

Conclusion

The present study adopted an exploratory approach to investigate alpha-band EEG activity in adults with Attention-Deficit/Hyperactivity Disorder (ADHD) under both resting-state and cognitive task conditions. The findings revealed that while no significant linear correlations emerged between symptom severity and alpha activity, group-based comparisons indicated that participants with higher symptom severity exhibited significantly lower task-related alpha activity compared to those with lower symptom severity. This pattern did not reach statistical significance for resting-state alpha activity. An unexpected finding was the overall increase in alpha activity from rest to task conditions across the sample, contrasting with the typical alpha desynchronization observed in neurotypical populations during cognitive engagement. This observation could reflect compensatory neural strategies or alternative processing patterns, but this interpretation remains speculative and requires confirmation with behavioral data and a healthy control group.

The present study used NeuroGuide Z-scores to benchmark alpha activity against an international normative database. While useful for standardization, this approach has several limitations. First, the normative sample may not fully represent the demographic and cultural characteristics of the Iranian population, potentially introducing bias. Second, Z-scores assume normally distributed EEG metrics in the reference population, which may not hold across all age groups or recording conditions. Third, the database does not provide separate norms for clinical populations, limiting its specificity for ADHD-related interpretations. Therefore, the Z-score-based findings reported here should be considered approximate. Future studies should validate these patterns using locally collected control data rather than relying exclusively on commercial normative databases.

Several limitations should be considered when interpreting the findings of the present study. First, the relatively small sample size (N = 28) limits statistical power and restricts the generalizability of the results. Consequently, it cannot be determined whether the observed alpha patterns are specific to ADHD or reflect broader individual differences in attentional functioning. Within the limitations of this exploratory study, task-related alpha modulation differed by symptom severity, suggesting that cognitive state may be relevant when investigating neural dynamics in adult ADHD. Second, the cross-sectional design prevents causal conclusions regarding the relationship between alpha-band activity and ADHD symptom severity. Third, the absence of a neurotypical control group precludes determining whether the observed alpha-band patterns are specific to ADHD or reflect broader variability in attentional functioning. Fourth, the present study did not systematically assess or control for ADHD subtype (e.g., predominantly inattentive vs. combined presentation), overall clinical severity beyond ASRS total score, or potential psychiatric comorbidities. These uncontrolled clinical variables may contribute to sample heterogeneity and could influence EEG findings. Most critically, the absence of behavioral performance data (accuracy and reaction time) severely limits any interpretation of task-related alpha activity. It was not possible for us to determine whether the observed alpha patterns reflect successful cognitive engagement, disengagement, drowsiness, or task-irrelevant processes. This limitation is particularly problematic given the unexpected task-related alpha increase.

Future studies must include concurrent behavioral measures to validate the functional significance of alpha modulation. In addition, medication status was not systematically controlled. Although participants using psychotropic medications were excluded, prior medication exposure and individual differences in treatment history may have influenced EEG measures. Finally, the cognitive task employed—while designed to engage sustained attention and inhibitory control—represents only a single type of cognitive demand. Different task paradigms may elicit distinct patterns of alpha modulation. Future research should aim to address these limitations by employing larger samples and longitudinal designs, incorporating neurotypical control groups, and systematically accounting for medication history and treatment effects. Examining alpha-band dynamics across multiple cognitive domains and task conditions may further clarify the functional role of alpha oscillations in ADHD. Moreover, investigating the specificity of alpha-band alterations in ADHD relative to other neurodevelopmental or psychiatric conditions will be essential for evaluating the potential diagnostic and clinical utility of EEG-based biomarkers. Future research employing larger samples, healthy control groups, multiple task paradigms, and comprehensive behavioral assessments will be essential to clarify the functional significance of alpha-band oscillations and their potential relevance to ADHD symptomatology.

Ethical considerations

The study was approved by the Ethics Committee of the University of ABCD (Ethical code: FR.AMU.REC.2022.500). The authors avoided from data fabrication and falsification.

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Conflict of interest

The authors declare no conflict of interest.

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Supplementary Material S1: Exploratory group comparison based on median split

These exploratory analyses are presented for hypothesis-generating purposes. Given the sample size (N=28), these findings require independent replication in larger samples before confirmatory conclusions can be drawn.

Condition	Group	n	Mean	SD	t (26)	p	Cohen's d
Rest	Low Symptom	14	.68	.85	1.92	.066	.73
	High Symptom	14	.17	.61			
Task	Low Symptom	14	.95	.83	2.12	.044	.81
	High Symptom	14	.38	.57			