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Reduced occurrence of alpha waves during resting state predicts high attention-deficit/hyperactivity disorder traits in young adults

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Abstract

Background: Attention-deficit/hyperactivity disorder (ADHD) is a neuro-developmental condition with significant cognitive and social impacts. Identifying reliable biomarkers for ADHD is crucial for developing personalised therapies. Electroencephalography (EEG) alpha oscillations (8–12 Hz) have been suggested as a potential biomarker, but findings have been inconsistent.

Methods: This study aimed to investigate whether alpha oscillations in young adulthood are associated with high ADHD traits using EEG data from a large twin sample ($N = 556$) enriched with participants with ADHD and autism. We assessed whether alpha oscillations during rest were associated with high ADHD traits. In addition, we used twin modelling to estimate the heritability of EEG alpha measures and their relationship with ADHD traits.

Results: Results showed that relative alpha peak amplitude was a significant predictor of ADHD traits when controlling for other factors such as age, sex and autistic traits. Specifically, we found that for each unit decrease in relative alpha peak amplitude (z-scored), the likelihood of being in the high ADHD trait group increased by approximately 26%. Further analysis suggested that group differences were due to a reduced occurrence (but not amplitude) of oscillatory bursts in the alpha range. Finally, our twin modelling results suggested that although these alpha measures are heritable, the genetic factors contributing to individual differences in alpha measures and ADHD traits were largely independent.

Conclusion: Together, these findings suggest that reduced alpha oscillations, particularly the occurrence of alpha bursts, may serve as a potential biomarker for ADHD. Our results may have implications for neuromodulation therapies targeting alpha rhythms in ADHD, such as neurofeedback and transcranial alternating current stimulation.

KEYWORDS
ADHD, alpha oscillations, biomarkers, EEG, twin study

INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a neuro-developmental condition characterised by pervasive patterns of inattention, hyperactivity, and impulsivity that is estimated to affect approximately 5% of adults worldwide (McCarthy et al., 2012; Polanczyk et al., 2007). ADHD has a significant impact on individuals' quality of life, mental health and social relationships (Adamou et al., 2013; Aduen et al., 2018; Aydin et al., 2022a; Capp et al., 2023). While psychopharmacological treatments, particularly stimulant medications, are considered the most effective interventions for managing ADHD symptoms (Cortese et al., 2018; Crescenzo et al., 2017; Skirrow et al., 2014), their use is frequently accompanied by side effects such as loss of appetite, sleep problems, and mood disturbances (Cascade et al., 2010). Although non-pharmacological treatments show high promise, the evidence of their success is still limited (Nimmo-Smith et al., 2020).

Recent advancements in precision psychiatry and neuro-modulation have paved the way for developing personalised therapies for ADHD (Arns, 2012; Voetterl et al., 2023). In this regard, neuromodulation techniques, such as neurofeedback and non-invasive brain stimulation, seem promising alternative or complementary treatments (Arns et al., 2014; Boetzel & Herrmann, 2021; Frohlich & Riddle, 2021). These approaches aim to directly modulate brain activity to improve symptomatology by promoting neuroplastic changes (Arns et al., 2014; Veniero et al., 2015; Vossen et al., 2015). In this context, EEG signatures can act as different classes of bio-markers that are relevant for neuromodulation (FDA-NIH Biomarker Working Group, 2016; Michelini et al., 2022). For instance, EEG metrics could serve as a monitoring and/or response biomarkers that would allow to track within-person changes after neuromodulation that co-vary with symptoms (Klooster et al., 2024; Riddle & Frohlich, 2023).

The study of brain oscillations in ADHD has emerged as a particularly promising approach for identifying biomarkers relevant for neuromodulation (Günther et al., 2022; McLoughlin, Makeig, et al., 2014; Riddle & Frohlich, 2021). Neural oscillations, as measured by electroencephalography (EEG), reflect the coordinated firing of neuronal populations and have been implicated in various cognitive and behavioural processes (Klimesch, 1999b; von Stein & Sarnthein, 2000). The most widely studied oscillatory measure in ADHD is the theta/beta ratio, with previous literature suggesting a higher ratio between theta (4–7 Hz) and beta (13–30 Hz) power in ADHD (Arns et al., 2013; McLoughlin et al., 2022). However, recent evidence has challenged the reliability and specificity of this finding (for a review, see McLoughlin et al., 2022). As an alternative, alpha oscillations (8–12 Hz) have emerged as an EEG characteristic of interest in ADHD research, with several studies reporting significant reductions in individuals with ADHD (Cañigueral et al., 2022; Deiber et al., 2020; Loo et al., 2009; Voetterl et al., 2023; Woltering et al., 2012).

Alpha oscillations fulfil several conditions to become a relevant ADHD biomarker. First, alpha oscillations have the highest test-retest reliability (Gudmundsson et al., 2007; Mathewson et al., 2015; Näpflin et al., 2007) and signal-to-noise ratio (Klimesch, 1999a, 2012) when compared to other brain rhythms. Secondly, alpha oscillations could offer a mechanistic explanation for

Key Points

What's known:
- ADHD has been linked to atypical brain activity, with Electroencephalography (EEG) alpha oscillations (8–12 Hz) suggested as a potential biomarker. However, most of previous studies have relied on small samples and yielded inconsistent results.

What's new:
- This study demonstrates that young adults with high ADHD traits show a significant reduction in the occurrence (but not the amplitude) of alpha waves independent of autistic traits.

What's relevant:
- Findings support the use of EEG alpha dynamics—especially wave occurrence—as a potential biomarker for ADHD. This has important implications for the development of personalised neuromodulation therapies targeting alpha rhythms such as neurofeedback or transcranial alternating current stimulation.

cognitive deficits in ADHD (Pievsky & McGrath, 2018), given their essential role in inhibitory control and arousal regulation (Bazanova & Vernon, 2014; Codispoti et al., 2023; Klimesch et al., 2007). Lastly, alpha oscillations can be modified by neuromodulation techniques such as transcranial alternating current stimulation (tACS) (Vossen et al., 2015), transcranial magnetic stimulation (TMS) (Gregorio et al., 2022) and neurofeedback (Deiber et al., 2020), thereby emphasising its potential for treatment development (Holland, 2016).

In the present study, we investigated whether alpha oscillations are associated with high ADHD traits in a large EEG sample of twins (N = 556). Specifically, we compared relative alpha peak amplitude between individuals with high (N = 193) and low ADHD traits (N = 343) using a logistic regression analysis that controlled for covariates including age, sex and autistic traits. In addition, we assess whether putative differences in alpha peak amplitude are actually due to oscillatory activity (Donoghue et al., 2021) using a recently developed algorithm that detects oscillatory bursts in EEG (Rodriguez-Larios et al., 2024; Rodriguez-Larios & Haegens, 2023). Finally, we used twin modelling to assess the genetic and environmental contributions to variance in alpha oscillatory dynamics, allowing us to estimate the heritability of the relevant alpha measures and their relationship with ADHD traits.

METHODS

Participants

Ethical approval for the study was received from King's College London Psychiatry, Nursing and Midwifery Research Ethics Subcommittee (RESCMR-16/17–2673). Data were collected as part of the Individual Differences in EEG in young Adults Study (IDEAS), which is a subsample of the Twins Early Development Study (TEDS) (Rimfeld et al., 2019). The sample consisted of 556 participants (267

males) with an average age of 22.43 (SD = 0.96 years) (119 Monozygotic (MZ) and 164 Dizygotic (DZ) pairs) (see full sample description at (Aydin et al., 2022a)).

Questionnaires and trait characterisation

Participants were classified into high and low ADHD trait groups using both the Diagnostic Interview for ADHD in Adults (DIVA 2.0) and the Barkley Adult ADHD Rating Scale (BAARS-IV). The BAARS provided a dimensional self-report measure of symptom severity across daily contexts, whereas the DIVA is a structured clinical interview to assess onset, chronicity, and functional impairment according to DSM-5 diagnostic criteria. Participants were classified as having high ADHD traits if they met DSM-5 diagnostic criteria for adult ADHD based on DIVA 2.0 or if their BAARS-IV total ADHD score was 39 or higher, reflecting mild to significant ADHD traits and related difficulties (Capp et al., 2023). This approach was followed to increase our sensitivity to detect ADHD traits. This resulted in 205 participants in the high ADHD traits group and 351 in the low ADHD traits group.

A similar approach was followed to control for autistic traits. In this way, the Autism Diagnostic Observation schedule 2 (ADOS-2) and Social Responsiveness Scale-2 (SRS-2) were used to classify participants into those with high (N = 127) and low (N = 429) autistic traits. Individuals with high autistic traits were the ones that scored four or higher on the ADOS-2 CSS (indicating behaviours consistent with an autism diagnosis) or had an SRS-2 raw score of 68 or above (indicating mild to severe difficulties with social behaviour related to autism). Those who did not meet these criteria were classified as having low autistic traits (Capp et al., 2023).

EEG recording and analysis

EEG was recorded with a mobile wireless 64-channel (10-10 montage) system (Cognionics, San Diego, CA; Ag/AgCl electrodes, with a sampling rate of 500 Hz). The dataset contained both resting state (eyes open and eyes closed) and task EEG data (Aydin, Gyurkovics, et al., 2023). Three minutes long eyes closed resting state EEG data was used in this study. This choice was based on previous literature showing higher reproducibility of eyes closed resting state EEG (Duan et al., 2021).

EEG analysis was performed in MATLAB R2023a using custom scripts and EEGLAB (Delorme & Makeig, 2004). Data cleaning was performed using an automatic pre-processing pipeline based on EEGLAB functions. First, data were resampled to 250 Hz, re-referenced to common average and filtered between 1 and 30 Hz. Abrupt noise in the data was removed using the Artefact Subspace Reconstruction method with a cut-off value of 20 SD (Chang et al., 2019). Noisy channels were detected automatically with the EEGLAB function clean_channels using a threshold of 0.6 and were later interpolated. The average number of interpolated channels was 9.5 (SD = 3.6) and no subject was excluded based on the number of interpolated channels. Independent component analysis (ICA) and an automatic component rejection algorithm (Pion-Tonachini et al., 2019) were used to discard components associated with muscle activity, eye

movements, heart activity or channel noise (threshold = 0.8). The average number of rejected components was 2.5 (SD = 2.3). Continuous data was divided into 2 s epochs (50% overlap) and epochs that exceeded an amplitude of ±100 mV were removed (average number of rejected epochs = 18). Subjects with more than 50% of rejected epochs were excluded from subsequent analysis (N = 20).

For the estimation of relative alpha peak amplitude and the theta/beta ratio, the spectrum between 1 and 30 Hz (in steps of 0.5 Hz) was estimated using a short-time Fourier transform as implemented through the MATLAB function *spectrogram* (sliding window of 2 s with 50% overlap). Individual alpha peak was estimated as the local maximum between 7 and 14 Hz using the *findpeaks* MATLAB function. Subjects that did not show an alpha peak in a specific electrode were excluded from the statistical analysis. In this regard, 23 subjects did not show an alpha peak in at least one electrode. Relative alpha peak amplitude was defined as the amplitude of the individual alpha peak of the normalised spectrum (i.e., z-scored over the frequency dimension). The theta/beta ratio was estimated from the Cz electrode as the average power in the 4–7 Hz range (theta) divided by the power in the 13–30 Hz range (beta) (Arns et al., 2013).

In order to assess whether putative differences in relative alpha peak amplitude were due to oscillatory activity, a recently developed algorithm was adapted (Rodriguez-Larios et al., 2024; Rodriguez-Larios & Haegens, 2023). In short, EEG data is first transformed to the time-frequency domain using Morlet wavelets (6 cycles width) as implemented in (Caplan et al., 2015). The frequency resolution was 1 Hz (between 1 and 30 Hz). Oscillatory bursts are defined as time points in which the amplitude at a specific frequency exceeds estimate of aperiodic activity (e.g., 1/f trend) for at least one full cycle. The 1/f trend is estimated by fitting a straight line to the spectrum in the log-log space per electrode across all time points and epochs. Only oscillatory bursts that had their highest spectral peak in the alpha range (7–14 Hz) were selected. This algorithm allows for disentangling the quantity of time in which oscillatory activity is present (i.e., oscillatory burst coverage) and the amplitude of the burst (which is estimated as the oscillation maximum peak prominence in the time domain).

Statistical analysis

A logistic regression model was employed to investigate the association between EEG alpha oscillations and the likelihood of having high ADHD traits. The analysis was conducted using a generalised linear mixed-effects model (GLMM) with a logit link function in MATLAB 2023a (*fitglme* function). To control for potential confounding factors, the model included additional fixed effects for age, sex and autistic traits (binary: high vs. low). To account for clustering within the sample due to relatedness (i.e., twins), random intercepts were included in the model for each pair of twins (Aydin, Cañigueral, et al., 2023; Malone et al., 2014). Only the association between alpha metrics and ADHD traits will be discussed as the rest of the predictors were only included as controls. In line with previous literature (Chu et al., 2020), effect sizes were estimated through the odds ratio, which was calculated by exponentiating the logistic regression coefficients (β). In order to assess significance, t-values for fixed effects

were calculated as the ratio of the estimated coefficient to its standard error.

In order to test the relationship between ADHD symptomatology and EEG metrics, we performed a linear mixed model as implemented in MATLAB (fitlme function). For this purpose, we used the total score of the BAARS questionnaire as a dependent variable. Like in the logistic regression analysis, the model included additional fixed effects for age, sex and autistic traits (binary: high vs. low) and random intercepts for each pair of twins. Continuous variables (BAARS, alpha amplitude, age) were normalised (z-scored) prior to analysis to facilitate interpretation of regression coefficients in terms of effect size (i.e., the change in the dependent variable in standard deviations associated with a one standard deviation change in the predictor).

Correlations were computed through Pearson correlation coefficient and multiple comparisons correction was implemented through the False Discovery Rate (Benjamini & Hochberg, 1995). Outliers were detected and rejected before all statistical analyses. Outliers were defined as elements more than three scaled median absolute deviations from the median (see *isoutlier* function in MATLAB 2023a).

Twin modelling

We applied genetic multivariate liability threshold models based on the principles that monozygotic (MZ) twins share 100% of their genetic influences, while dizygotic (DZ) twins share 50%, and both groups share common environmental factors equally (Neale & Maes, 2004). These models treat high ADHD traits and high autistic traits as dichotomous variables and use the MZ:DZ ratio of cross-twin within-trait correlations to partition the variation in EEG traits into standardised additive genetic variance (a^2), common environmental variance (c^2), and unique environmental variance, including measurement error (e^2). The phenotypic correlation (Rph) between traits is calculated as the zero-order correlation, while the MZ:DZ ratio of cross-twin cross-trait correlations is used to partition the covariation between traits (e.g., ADHD and relative alpha peak

amplitude) to report genetic (Ra) correlations. Since the sample had a higher proportion of participants meeting ADHD or ASD criteria compared to the general population, we applied corrections to the standard twin model, as described in (Aydin et al., 2022b). Twin modelling was conducted using structural equation modelling in OpenMx (Boker et al., 2011), with likelihood-based asymmetric 95% confidence intervals (CIs) estimated for all parameters. To examine associations between ADHD or autistic traits and specific EEG metrics, we ran bivariate models for relative alpha peak amplitude, burst alpha amplitude, and burst alpha coverage.

RESULTS

Relative alpha peak amplitude predicts ADHD traits and symptoms

Logistic regression analysis revealed that relative alpha peak amplitude was a significant predictor of high ADHD traits (p-values after FDR correction <0.05 in 55 out of 64 electrodes) (see Figure 1A). Specifically, participants with high ADHD traits showed a significant reduction in relative alpha peak amplitude when compared to the low ADHD trait group (see Figure 1B,C). The odds ratio in the electrode with the lowest p-value (AF8) was 0.74, which means that for every unit of relative alpha amplitude (in z-scores) the odds of being in the high ADHD trait group decreased by approximately 26% (t-value (497) = −3.02; p-value = 0.0026) (see Table 1). Subsequent analyses were performed in the electrode with the lowest p-value (AF8; marked in Figure 1A).

A dimensional analysis using the BAARS questionnaire (i.e., total ADHD score) showed that ADHD traits were negatively associated with relative alpha peak amplitude. Specifically, 1 SD increase in relative peak alpha amplitude in electrode AF8 was associated with a 0.09 SD decrease in the total ADHD score (t-value (497) = −2.11; p-value = 0.035) (see Table 2).

In order to assess the specificity of our findings, we performed the same analyses with a commonly used EEG metric in the ADHD
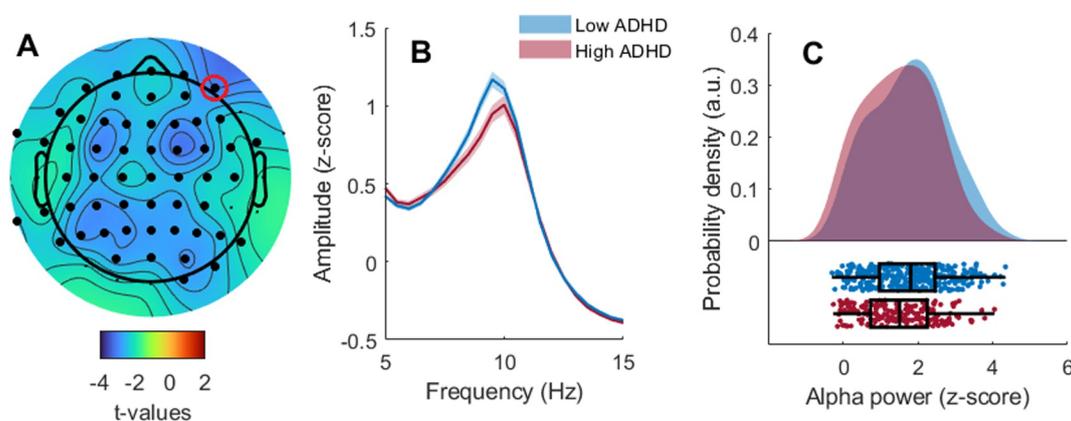

FIGURE 1 Association between relative alpha peak amplitude and ADHD traits. (A) Topographical plot depicting t-values obtained when using relative alpha peak amplitude as predictor in the specified model. Black dots indicate significant electrodes (p-value <0.05) after FDR correction and the red circle marks AF8, the most significant electrode (i.e., highest absolute t-value). (B) Frequency spectrum (z-scored) averaged across significant electrodes for low (blue) and high (red) ADHD trait groups. The shaded area depicts standard error. (C) Relative alpha amplitude for the low (blue) and high (red) ADHD trait groups. Each point depicts relative alpha amplitude for one subject across significant electrodes. Boxplots indicate the median and interquartile range per group. ADHD, attention-deficit/hyperactivity disorder.

TABLE 1 Logistic regression model for the prediction of ADHD traits (0 = low, 1 = high) from autistic traits (0 = low, 1 = high), age, sex and relative alpha peak amplitude at electrode AF8.

Predictor	β	SE	t-value	p-value	Odds ratio
Intercept	6.18	2.59	2.38	0.0172	487.23
Autistic traits	1.02	0.23	4.28	<0.001	2.78
Age	−0.29	0.11	−2.63	0.0087	0.74
Sex	0.03	0.21	0.17	0.8610	1.04
Relative alpha peak amplitude	−0.323	0.10	−3.02	0.0026	0.72

TABLE 2 Linear mixed model for the prediction of ADHD traits (BAARS total score) from autistic traits (0 = low, 1 = high), age, sex and relative alpha amplitude at electrode AF8.

Predictor	B	SE	t-value	p-value
Intercept	0.18	0.05	3.41	0.0007
Autistic traits	−0.33	0.05	−6.19	<0.001
Age	−0.06	0.04	−1.37	0.1715
Sex	0.02	0.04	0.50	0.6158
Relative alpha peak amplitude	−0.09	0.04	−2.11	0.0355

TABLE 3 Logistic regression model for the prediction of ADHD trait (0 = low, 1 = high) from autistic trait (0 = low, 1 = high), age, sex, mean oscillatory burst alpha amplitude (i.e., amplitude) and mean oscillatory burst alpha coverage (coverage) at electrode AF8.

Predictor	β	SE	t-value	p-value	Odds ratio
Intercept	4.90	2.46	1.98	0.0472	134.93
Autistic traits	1.04	0.23	4.48	<0.001	2.84
Age	−0.23	0.10	−2.15	0.0316	0.79
Sex	0.04	0.20	0.22	0.8245	1.04
Amplitude	0.002	0.01	0.13	0.8901	1.00
Coverage	−0.06	0.02	−2.28	0.0228	0.94

literature (i.e., theta/beta ratio; TBR). Unlike relative alpha amplitude, TBR was not a significant predictor of either high ADHD traits whether using the binary grouping derived from BAARS and DIVA (t-value (497) = −0.52; p-value = 0.60) or the continuous BAARS total score (t-value (497) = −0.86; p-value = 0.38).

Assessing the putative oscillatory nature of alpha amplitude modulations

In order to assess whether the reported modulations of alpha amplitude in relation to ADHD traits were due to oscillatory activity, we employed a recently developed algorithm that estimates the occurrence and amplitude of oscillatory bursts (Rodriguez-Larios et al., 2024; Rodriguez-Larios & Haegens, 2023). For this analysis, we selected the frontal EEG electrode with the highest group difference as quantified by its t-value (i.e., electrode AF8; see red circle in Figure 1).

On the one hand, logistic regression analysis revealed a significant effect of oscillatory burst coverage (t-value (499) = −2.28, p-value = 0.0228) but not of oscillatory burst amplitude (t-value (499) = 0.13, p-value = 0.89) (see Table 3 for full model details and Figure 2A,B for depiction of results). The odds ratio for burst coverage was 0.94, which means that for every second of alpha oscillatory bursts in the resting state EEG, the odds of being in the high ADHD trait group decreased by approximately 6%.

On the other hand, a dimensional analysis (i.e., linear mixed model) showed that ADHD traits (as assessed with BAARS total score) were significantly associated with alpha burst coverage but not amplitude (see Table 4 for full model). Specifically, 1 SD increase in burst coverage was associated with a 0.10 SD decrease in BAARS score (t-value (497) = −2.11; p-value = 0.035) (see Table 4).

Together, these results suggest that relative alpha amplitude modulations in relation to ADHD were mostly due to the occurrence of oscillatory bursts rather than their amplitude. In line with this idea, inter-individual differences in relative alpha amplitude showed a higher correlation to burst coverage (r-value = 0.83; p-value <0.001; Figure 2C) than to burst amplitude (r-value = 0.33; p-value <0.001; Figure 2D).

Genetic contributions of alpha parameters

We found statistically significant and high genetic contributions (a^2) to the total variance for relative alpha peak amplitude and burst alpha coverage but not for burst alpha amplitude (Table 5). Common environment contributions (c^2) were not significant. In terms of associations between ADHD traits and alpha measures, we observed significant negative phenotypic correlations (Rph) for relative alpha amplitude (−0.15) and burst alpha coverage (−0.12) but in both cases, the genetic correlations (Ra) were not significant (Table 5). Phenotypic and genetic correlations between autistic traits and the alpha measures were not statistically significant (see Table 6).

Taken together, these findings suggest that while relative alpha peak amplitude and alpha burst coverage are highly heritable, their modest phenotypic associations with ADHD traits are not explained by common genetic influences.

DISCUSSION

The aim of this study was to investigate whether adults with high ADHD traits show alterations in alpha oscillations during resting state. For this purpose, we analysed a large twin EEG data set (N = 556) enriched for ADHD and autistic traits (Aydin et al., 2022a;
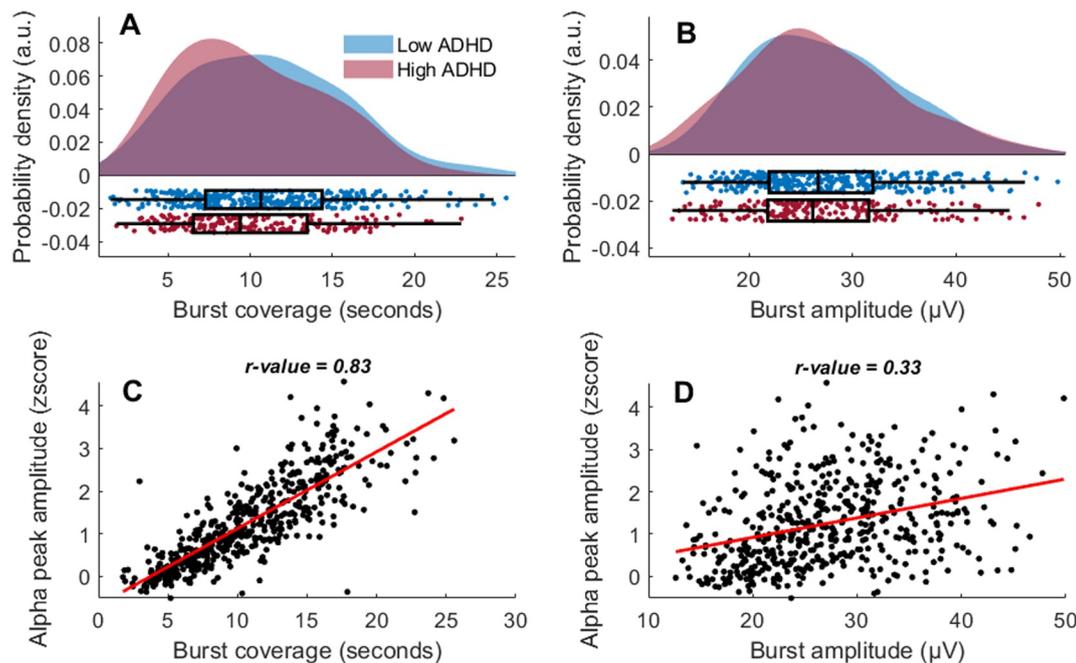
FIGURE 2 Alpha oscillatory burst differences between groups with high and low ADHD traits and their correlation with relative alpha amplitude. (A) Burst coverage for the low (blue) and high (red) ADHD trait groups. Each point depicts relative alpha amplitude for one subject for the selected frontal electrode. Boxplots indicate median and interquartile range per group. (B) Same as A but for burst amplitude. (C, D) Correlation between burst parameters (coverage and amplitude) and individual alpha peak amplitude (each point depicts data of one subject for the selected electrode). ADHD, attention-deficit/hyperactivity disorder.

TABLE 4 Linear mixed model for the prediction of ADHD traits (BAARS total score) from autistic traits (0 = low, 1 = high), age, sex, mean oscillatory burst alpha amplitude (i.e., amplitude) and mean oscillatory burst alpha coverage (coverage) at electrode AF8.

Predictor	β	SE	t-value	p-value
Intercept	0.18	0.05	3.38	0.0007
Autistic traits	−0.33	0.05	−6.02	
Age	−0.06	0.04	−1.33	0.1821
Sex	0.002	0.04	0.05	0.9580
Amplitude	0.036	0.04	0.74	0.4553
Coverage	−0.10	0.04	−2.07	0.0387

McLoughlin, Palmer, et al., 2014). Our results showed that participants with high ADHD traits have significantly reduced relative alpha peak amplitude at rest across EEG electrodes. Specifically, logistic regression analysis revealed that for every unit of relative alpha peak amplitude (in z-scores), the odds of being in the high ADHD trait group decreased by approximately 26%. Further analysis suggested that group differences in relative alpha amplitude were due to changes in the occurrence of oscillatory bursts in the alpha range rather than their amplitude. Crucially, unlike alpha metrics, the theta/beta ratio was not a significant predictor of ADHD traits, which is in line with recent reports (Arns et al., 2013; McLoughlin et al., 2022). Finally, our twin modelling analysis showed that although the relevant alpha metrics for ADHD are highly heritable (i.e., relative peak amplitude and burst coverage), their associations with ADHD traits are not explained by common genetic influences.

Although previous studies have shown that relative alpha amplitude during resting state is significantly reduced in populations with high ADHD traits (Deiber et al., 2020; Loo et al., 2009; Ponomarev et al., 2014; Woltering et al., 2012), this finding was not completely consistent with the literature (Bresnahan et al., 2006; Koehler et al., 2009; Poil et al., 2014; van Dongen-Boomsma et al., 2010; Voetterl et al., 2023). These inconsistencies could be due to small sample sizes and the lack of control variables in previous studies. Our large and well-characterised sample allowed us to clarify these inconsistencies by demonstrating that reduced alpha amplitude remains significantly associated with ADHD even when controlling for autistic traits, age and sex. In addition, by employing a recently developed algorithm (Rodriguez-Larios et al., 2024; Rodriguez-Larios & Haegens, 2023) we further characterise the origin of the reported alpha differences and control for the possible influence of aperiodic activity. This analysis suggested that alpha amplitude differences between high and low ADHD groups were more influenced by the occurrence of alpha bursts than by their amplitude. Nonetheless, it is important to highlight that relative alpha peak amplitude was a better predictor of ADHD traits than alpha burst coverage (i.e., greater effect size as quantified through the odds ratio). This could be because relative alpha peak amplitude is also influenced by other EEG parameters such as aperiodic activity and/or band power at other frequency bands (Donoghue et al., 2021) that are also affected in ADHD (Chen et al., 2024; Dakwar-Kawar et al., 2024). Although the odds ratio for burst coverage indicates a smaller effect size than that for relative alpha peak amplitude, its identification as a potential ADHD biomarker remains important from a translational perspective. For example, in the context of neurofeedback, the use of oscillatory burst metrics (such as coverage) might be more effective for ADHD than general alpha amplitude estimates (Deiber et al., 2020) due to their higher degree of specificity (Enriquez-Geppert et al., 2017). In support of this idea, one previous study

TABLE 5 Phenotypic (Rph) and genetic (Ra) correlations between ADHD trait and relative alpha amplitude, mean oscillatory burst alpha amplitude, and **mean oscillatory burst alpha coverage**.

	Rph	Ra	a²	c²	e²
Relative alpha peak amplitude	**−0.15** (−0.25, −0.05)	−0.14 (−0.31, 0.03)	**0.59** (0.48, 0.69)	0.00 (0.00, 0.28)	**0.41** (0.31, 0.52)
Burst alpha amplitude	−0.04 (−0.14, 0.05)	−0.05 (−1.00, 1.00)	0.38 (0.00, 0.61)	0.11 (0.00, 0.44)	**0.51** (0.39, 0.65)
Burst alpha coverage	**−0.12** (−0.22, −0.03)	−0.13 (−0.31, 0.05)	**0.59** (0.46, 0.69)	0.00 (0.00, 0.29)	**0.41** (0.31, 0.54)

Note: Standardised estimates of genetic (a^2), common (c^2) and nonshared (e^2) environment contributions to variance of the EEG measures. 95% confidence intervals are given in parentheses, and significant estimates are written in bold.

TABLE 6 Phenotypic (Rph) and genetic (Ra) correlations between autistic trait and relative alpha amplitude, mean oscillatory burst alpha amplitude, and **mean oscillatory burst alpha coverage**.

	Rph	Ra	a²	c²	e²
Relative alpha peak amplitude	−0.07 (−0.19, 0.05)	−0.09 (−0.30, 0.12)	**0.59** (0.26, 0.69)	0.00 (0.00, 0.28)	**0.41** (0.31, 0.52)
Burst alpha amplitude	−0.08 (−0.05, 0.20)	0.27 (−0.02, 1.00)	0.35 (0.00, 0.61)	0.13 (0.00, 0.44)	**0.51** (0.39, 0.65)
Burst alpha coverage	−0.11 (−0.23, 0.01)	−0.20 (−0.41, 0.02)	**0.59** (0.46, 0.69)	0.00 (0.00, 0.27)	**0.41** (0.31, 0.54)

Note: Standardised estimates of genetic (a^2), common (c^2) and nonshared (e^2) environment contributions to variance of the EEG measures. 95% confidence intervals are given in parentheses, and significant estimates are written in bold.

suggests that alpha neurofeedback protocols actually modulate alpha burst occurrence rather than alpha amplitude per se (Ossadtchi et al., 2017).

Our twin modelling results confirmed the high heritability of EEG alpha measures, with particularly strong genetic contributions to relative alpha amplitude and burst alpha coverage. This finding aligns with previous reports indicating the high heritability of alpha oscillations' characteristics (Malone et al., 2014; Smit et al., 2006; van Beijsterveldt & van Baal, 2002). Despite the observed phenotypic correlations between ADHD traits and alpha oscillations, we did not detect significant genetic correlations between these measures. This suggests that the genetic factors influencing alpha dynamics may be at least partly independent from those contributing to ADHD liability, although limited power cannot be excluded. Importantly, such independence would not diminish the relevance of alpha oscillations as a mechanistic target; on the contrary, it could indicate that modulating alpha activity might benefit individuals regardless of their underlying genetic risk profile.

The identification of alpha oscillations as a possible biomarker for ADHD has important implications for therapy. If reduced alpha oscillations are present in ADHD, neuromodulation protocols that enhance alpha oscillations (such as tACS and neurofeedback) (Soriano et al., 2024; Vossen et al., 2015) may improve ADHD symptoms. Recent evidence suggests that specific alpha oscillatory patterns may also help guide treatment selection, potentially improving clinical outcomes through personalised approaches (Voetterl et al., 2023). In this direction, it has been shown that a neurofeedback protocol that increased alpha amplitude improved ADHD symptoms and attentional performance (Deiber et al., 2020). Similarly, a recent alpha tACS study has shown that repeated sessions of alpha tACS can improve ADHD symptoms (although in this case changes in EEG were not investigated) (Farokhzadi et al., 2020). Together, although more evidence is needed, neuromodulation protocols aimed at enhancing alpha oscillations in ADHD show significant promise. An important question for future research is whether individuals with ADHD who exhibit reduced alpha amplitude at rest would derive greater benefits from these therapies.

Our study has several limitations. First, we did not systematically compare alpha metrics with a myriad of other metrics that can be obtained from EEG as this was beyond the scope of this study. Therefore, we cannot claim that metrics based on alpha oscillations are the best EEG predictor of ADHD traits. Secondly, the here proposed link between ADHD traits and resting state alpha wave activity is purely correlational. Future studies using neuromodulation techniques capable of modulating alpha wave activity (Neurofeedback, tACS, TMS) are needed to establish a causal relationship. Third, it is unclear how exactly resting state alpha waves are mechanistically related to ADHD symptomatology. Although alpha oscillations have been shown to play a key role in arousal regulation and attentional control (Bazanova & Vernon, 2014; Codispoti et al., 2023; Klimesch et al., 2007), we did not assess whether these processes are correlated with alpha metrics during rest. In fact, the observed differences in the high ADHD trait group could be due to several factors known to affect alpha waves, ranging from anatomical differences (Kumral et al., 2022; Valdés-Hernández et al., 2010) to modulations in resting-state cognition (e.g., mind wandering levels) (Compton et al., 2019; Rodriguez-Larios & Alaerts, 2020; Rodriguez-Larios et al., 2021). In order to answer these questions, future studies would highly benefit from a combination of resting state EEG, questionnaires and a comprehensive battery of cognitive tests in well-characterised clinical samples. Lastly, the relationship between alpha metrics and ADHD traits was modest for both the categorical and dimensional analyses. Therefore, rather than serving as a standalone diagnostic biomarker, alpha activity may be more appropriately conceptualised as a monitoring and/or response biomarker relevant for neuromodulation. In this regard, EEG alpha metrics could be potentially useful for tracking treatment-related changes in ADHD. Nonetheless, future longitudinal and interventional studies are needed to determine whether changes in alpha activity reliably parallel clinical improvement.

In conclusion, this study shows that adults with high ADHD traits have reduced relative alpha amplitude at rest, primarily due to a decrease in the occurrence of alpha oscillatory bursts. While our findings support the idea that alpha oscillations could be a potential

biomarker for ADHD, its consistency across different populations and usefulness for therapy is still to be tested. Future research should investigate the underlying mechanisms contributing to these differences and determine if individuals with lower alpha amplitude benefit more from targeted neuromodulation therapies.

AUTHOR CONTRIBUTIONS

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the author G.M. upon reasonable request.

ETHICAL CONSIDERATIONS

Ethical approval for the study was received from King's College London Psychiatry, Nursing and Midwifery Research Ethics Subcommittee on 21/04/2016 (RESCMR-16/17–2673). Informed consent was obtained from all participants.

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REFERENCES

Adamou, M., Arif, M., Asherson, P., Aw, T.-C., Bolea, B., Coghill, D., Guðjónsson, G., Halmøy, A., Hodgkins, P., Müller, U., Pitts, M., Trakoli, A., Williams, N., & Young, S. (2013). Occupational issues of adults with ADHD. *BMC Psychiatry*, *13*(1), 59. https://doi.org/10.1186/1471-244X-13-59

Aduen, P. A., Day, T. N., Kofler, M. J., Harmon, S. L., Wells, E. L., & Sarver, D. E. (2018). Social problems in ADHD: Is it a skills acquisition or performance problem? *Journal of Psychopathology and Behavioral Assessment*, *40*(3), 440–451. https://doi.org/10.1007/s10862-018-9649-7

Arns, M. (2012). EEG-based personalized medicine in ADHD: Individual alpha peak frequency as an endophenotype associated with nonresponse. *Journal of Neurotherapy*, *16*(2), 123–141. https://doi.org/10.1080/10874208.2012.677664

Arns, M., Conners, C. K., & Kraemer, H. C. (2013). A decade of EEG theta/beta ratio research in ADHD: A meta-analysis. *Journal of Attention Disorders*, *17*(5), 374–383. https://doi.org/10.1177/1087054712460087

Arns, M., Heinrich, H., & Strehl, U. (2014). Evaluation of neurofeedback in ADHD: The long and winding road. *Biological Psychology*, *95*, 108–115. https://doi.org/10.1016/j.biopsycho.2013.11.013

Aydin, Ü. Cañigueral, R., Tye, C., & McLoughlin, G. (2023a). Face processing in young adults with autism and ADHD: An event related potentials study. *Frontiers in Psychiatry*, *14*, 1080681. https://www.frontiersin.org/articles/10.3389/fpsyt.2023.1080681

Aydin, Ü., Capp, S. J., Tye, C., Colvert, E., Lau-Zhu, A., Rijsdijk, F., Palmer, J., & McLoughlin, G. (2022a). Quality of life, functional impairment and continuous performance task event-related potentials (ERPs) in young adults with ADHD and autism: A twin study. *JCPP Advances*, *2*(3), e12090. https://doi.org/10.1002/jcv2.12090

Aydin, Ü., Capp, S. J., Tye, C., Colvert, E., Lau-Zhu, A., Rijsdijk, F., Palmer, J., & McLoughlin, G. (2022b). Quality of life, functional impairment and continuous performance task event-related potentials (ERPs) in young adults with ADHD and autism: A twin study. *JCPP Advances*, *2*(3), e12090. https://doi.org/10.1002/jcv2.12090

Aydin, Ü., Gyurkovics, M., Ginestet, C., Capp, S., Greven, C. U., Palmer, J., & McLoughlin, G. (2023b). Genetic overlap between midfrontal theta signals and attention-Deficit/Hyperactivity disorder and autism spectrum disorder in a longitudinal twin cohort. *Biological Psychiatry*, *94*(10), 823–832. https://doi.org/10.1016/j.biopsych.2023.05.006

Bazanova, O. M., & Vernon, D. (2014). Interpreting EEG alpha activity. *Neuroscience & Biobehavioral Reviews*, *44*, 94–110. https://doi.org/10.1016/j.neubiorev.2013.05.007

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society: Series B*, *57*(1), 289–300. https://doi.org/10.1111/j.2517-6161.1995.tb02031.x

Boetzel, C., & Herrmann, C. S. (2021). Potential targets for the treatment of ADHD using transcranial electrical current stimulation. *Progress in Brain Research*, *264*, 151–170. https://doi.org/10.1016/BS.PBR.2021.01.011

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., Spies, J., Estabrook, R., Kenny, S., Bates, T., Mehta, P., & Fox, J. (2011). OpenMx: An open source extended structural equation modeling framework. *Psychometrika*, *76*(2), 306–317. https://doi.org/10.1007/s11336-010-9200-6

Bresnahan, S. M., Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2006). Quantitative EEG analysis in dexamphetamine-responsive adults with attention-deficit/hyperactivity disorder. *Psychiatry Research*, *141*(2), 151–159. https://doi.org/10.1016/j.psychres.2005.09.002

Cañigueral, R., Palmer, J., Ashwood, K. L., Azadi, B., Asherson, P., Bolton, P. F., McLoughlin, G., & Tye, C. (2022). Alpha oscillatory activity during attentional control in children with autism spectrum disorder (ASD), attention-Deficit/Hyperactivity disorder (ADHD), and ASD+ADHD. *Journal of Child Psychology and Psychiatry*, *63*(7), 745–761. https://doi.org/10.1111/jcpp.13514

Caplan, J. B., Bottomley, M., Kang, P., & Dixon, R. A. (2015). Distinguishing rhythmic from non-rhythmic brain activity during rest in healthy neurocognitive aging. *NeuroImage*, *112*, 341–352. https://doi.org/10.1016/J.NEUROIMAGE.2015.03.001

Capp, S. J., Agnew-Blais, J., Lau-Zhu, A., Colvert, E., Tye, C., Aydin, Ü., Lautarescu, A., Ellis, C., Saunders, T., O'Brien, L., Ronald, A., Happé, F., & McLoughlin, G. (2023). Is quality of life related to high autistic traits, high ADHD traits and their interaction? Evidence from a young-adult community-based twin sample. *Journal of Autism and Developmental Disorders*, *53*(9), 3493–3508. https://doi.org/10.1007/s10803-022-05640-w

Cascade, E., Kalali, A. H., & Wigal, S. B. (2010). Real-world data on: Attention deficit hyperactivity disorder medication side effects. *Psychiatry*, *7*(4), 13–15.

Chang, C.-Y., Hsu, S.-H., Pion-Tonachini, L., & Jung, T.-P. (2019). Evaluation of artifact subspace reconstruction for automatic artifact components removal in multi-channel EEG recordings. *IEEE Transactions on Biomedical Engineering*, *67*(4), 1–1121. https://doi.org/10.1109/tbme.2019.2930186

Chen, R., Liu, W., Wang, J.-J., Zhou, D.-D., & Wang, Y. (2024). Aperiodic components and aperiodic-adjusted alpha-band oscillations in children with ADHD. *Journal of Psychiatric Research*, *173*, 225–231. https://doi.org/10.1016/j.jpsychires.2024.03.042

Chu, B., Liu, M., Leas, E. C., Althouse, B. M., & Ayers, J. W. (2020). Effect size reporting among prominent health journals: A case study of odds ratios. *BMJ Evidence-Based Medicine*, *26*(4), 184. https://doi.org/10.1136/bmjebm-2020-111569

Codispoti, M., De Cesarei, A., & Ferrari, V. (2023). Alpha-band oscillations and emotion: A review of studies on picture perception. *Psychophysiology*, 60(12), e14438. https://doi.org/10.1111/psyp.14438

Compton, R. J., Gearinger, D., & Wild, H. (2019). The wandering mind oscillates: EEG alpha power is enhanced during moments of mind-wandering. *Cognitive, Affective, and Behavioral Neuroscience*, 19(5), 1–8. https://doi.org/10.3758/s13415-019-00745-9

Cortese, S., Adamo, N., Giovane, C. D., Mohr-Jensen, C., Hayes, A. J., Carucci, S., Atkinson, L. Z., Tessari, L., Banaschewski, T., Coghill, D., Hollis, C., Simonoff, E., Zuddas, A., Barbui, C., Purgato, M., Steinhausen, H.-C., Shokraneh, F., Xia, J., & Cipriani, A. (2018). Comparative efficacy and tolerability of medications for attention-deficit hyperactivity disorder in children, adolescents, and adults: A systematic review and network meta-analysis. *The Lancet Psychiatry*, 5(9), 727–738. https://doi.org/10.1016/S2215-0366(18)30269-4

Crescenzo, F. D., Cortese, S., Adamo, N., & Janiri, L. (2017). Pharmacological and non-pharmacological treatment of adults with ADHD: A meta-review. *BMJ Ment Health*, 20(1), 4–11. https://doi.org/10.1136/eb-2016-102415

Dakwar-Kawar, O., Mentch-Lifshits, T., Hochman, S., Mairon, N., Cohen, R., Balasubramani, P., Mishra, J., Jordan, J., Cohen Kadosh, R., Berger, I., & Nahum, M. (2024). Aperiodic and periodic components of oscillatory brain activity in relation to cognition and symptoms in pediatric ADHD. *Cerebral Cortex*, 34(6), bhae236. https://doi.org/10.1093/cercor/bhae236

Deiber, M.-P., Hasler, R., Colin, J., Dayer, A., Aubry, J.-M., Baggio, S., Perroud, N., & Ros, T. (2020). Linking alpha oscillations, attention and inhibitory control in adult ADHD with EEG neurofeedback. *NeuroImage: Clinical*, 25, 102145. https://doi.org/10.1016/j.nicl.2019.102145

Delorme, A., & Makeig, S. (2004). EEGLAB: An open sorce toolbox for analysis of single-trail EEG dynamics including independent component anlaysis. *Journal of Neuroscience Methods*, 134(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Donoghue, T., Schaworonkow, N., & Voytek, B. (2021). Methodological considerations for studying neural oscillations. *European Journal of Neuroscience*, 55(11–12), 3502–3527. https://doi.org/10.1111/EJN.15361

Duan, W., Chen, X., Wang, Y.-J., Zhao, W., Yuan, H., & Lei, X. (2021). Reproducibility of power spectrum, functional connectivity and network construction in resting-state EEG. *Journal of Neuroscience Methods*, 348, 108985. https://doi.org/10.1016/j.jneumeth.2020.108985

Enriquez-Geppert, S., Huster, R. J., & Herrmann, C. S. (2017). EEG-neurofeedback as a tool to modulate cognition and behavior: A review tutorial. *Frontiers in Human Neuroscience*, 11, 51. https://doi.org/10.3389/fnhum.2017.00051

Farokhzadi, F., Mohamadi, M. R., Khosli, A. K., Akbarfahimi, M., Beigi, N. A., & Torabi, P. (2020). Comparing the effectiveness of the transcranial alternating current stimulation (TACS) and ritalin on symptoms of attention deficit hyperactivity disorder in 7-14-Year-Old children. *Acta Medica Iranica*, 637–648. https://doi.org/10.18502/acta.v58i12.5156

FDA-NIH Biomarker Working Group. (2016). BEST (biomarkers, EndpointS, and other tools) resource. *Food and Drug Administration (US)*. Retrieved from http://www.ncbi.nlm.nih.gov/books/NBK326791/

Frohlich, F., & Riddle, J. (2021). Conducting double-blind placebo-controlled clinical trials of transcranial alternating current stimulation (tACS). *Translational Psychiatry*, 11(1), 1–12. https://doi.org/10.1038/s41398-021-01391-x

Gregorio, F. D., Trajkovic, J., Roperti, C., Avenanti, A., Thut, G., Correspondence, V. R., Gregorio, D., Marcantoni, E., Luzio, P. D., & Romei, V. (2022). Tuning alpha rhythms to shape conscious visual perception. *Current Biology*. https://doi.org/10.1016/J.CUB.2022.01.003

Gudmundsson, S., Runarsson, T. P., Sigurdsson, S., Eiriksdottir, G., & Johnsen, K. (2007). Reliability of quantitative EEG features. *Clinical Neurophysiology*, 118(10), 2162–2171. https://doi.org/10.1016/j.clinph.2007.06.018

Günther, A., Hanganu-opatz, I. L., & Robinson, J. (2022). Neuronal oscillations: Early biomarkers of psychiatric disease? December, 1–9. https://doi.org/10.3389/fnbeh.2022.1038981

Holland, R. L. (2016). What makes a good biomarker? *Advances in Precision Medicine*, 1(1), 66. https://doi.org/10.18063/APM.2016.01.007

Klimesch, W. (1999a). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews*, 29(2–3), 169–195. https://doi.org/10.1016/s0165-0173(98)00056-3

Klimesch, W. (1999b). EEG alpha and theta oscillations reflect cognitive and memory performance: A rKlimesch, W. (1999). EEG alpha and theta oscillations reflect cognitive and memory performance: A review and analysis. *Brain Research Reviews*, 29(2–3), 169–195. https://doi.org/10.1016/S0165-0173(98)00056-3

Klimesch, W. (2012). Alpha-band oscillations, attention, and controlled access to stored information. *Trends in Cognitive Sciences*, 16(12), 606–617. https://doi.org/10.1016/j.tics.2012.10.007

Klimesch, W., Sauseng, P., & Hanslmayr, S. (2007). EEG alpha oscillations: The inhibition-timing hypothesis. *Brain Research*, 53(1), 63–88. https://doi.org/10.1016/j.brainresrev.2006.06.003

Klooster, D., Voetterl, H., Baeken, C., & Arns, M. (2024). Evaluating robustness of brain stimulation biomarkers for depression: A systematic review of magnetic resonance imaging and electroencephalography studies. *Biological Psychiatry*, 95(6), 553–563. https://doi.org/10.1016/j.biopsych.2023.09.009

Koehler, S., Lauer, P., Schreppel, T., Jacob, C., Heine, M., Boreatti-Hümmer, A., Fallgatter, A. J., & Herrmann, M. J. (2009). Increased EEG power density in alpha and theta bands in adult ADHD patients. *Journal of Neural Transmission*, 116(1), 97–104. https://doi.org/10.1007/s00702-008-0157-x

Kumral, D., Cesnaite, E., Beyer, F., Hofmann, S. M., Hensch, T., Sander, C., Hegerl, U., Haufe, S., Villringer, A., Witte, A. V., & Nikulin, V. V. (2022). Relationship between regional white matter hyperintensities and alpha oscillations in older adults. *Neurobiology of Aging*, 112, 1–11. https://doi.org/10.1016/j.neurobiolaging.2021.10.006

Loo, S. K., Hale, T. S., Macion, J., Hanada, G., McGough, J. J., McCracken, J. T., & Smalley, S. L. (2009). Cortical activity patterns in ADHD during arousal, activation and sustained attention. *Neuropsychologia*, 47(10), 2114–2119. https://doi.org/10.1016/j.neuropsychologia.2009.04.013

Malone, S. M., Burwell, S. J., Vaidyanathan, U., Miller, M. B., McGue, M., & Iacono, W. G. (2014). Heritability and molecular-genetic basis of resting EEG activity: A genome-wide association study. *Psychophysiology*, 51(12), 1225–1245. https://doi.org/10.1111/psyp.12344

Mathewson, K. J., Hashemi, A., Sheng, B., Sekuler, A. B., Bennett, P. J., & Schmidt, L. A. (2015). Regional electroencephalogram (EEG) alpha power and asymmetry in older adults: A study of short-term test–retest reliability. *Frontiers in Aging Neuroscience*, 7, 177. https://doi.org/10.3389/fnagi.2015.00177

McCarthy, S., Wilton, L., Murray, M. L., Hodgkins, P., Asherson, P., & Wong, I. C. (2012). The epidemiology of pharmacologically treated attention deficit hyperactivity disorder (ADHD) in children, adolescents and adults in UK primary care. *BMC Pediatrics*, 12(1), 78. https://doi.org/10.1186/1471-2431-12-78

McLoughlin, G., Gyurkovics, M., & Aydin, Ü. (2022). What has been learned from using EEG methods in research of ADHD? In S. C. Stanford & E. Sciberras (Eds.), *New discoveries in the behavioral neuroscience of attention-deficit hyperactivity disorder* (pp. 415–444). Springer International Publishing. https://doi.org/10.1007/7854_2022_344

McLoughlin, G., Makeig, S., & Tsuang, M. T. (2014a). In search of biomarkers in psychiatry: EEG-based measures of brain function. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 165(2), 111–121. https://doi.org/10.1002/ajmg.b.32208

McLoughlin, G., Palmer, J. A., Rijsdijk, F., & Makeig, S. (2014b). Genetic overlap between evoked frontocentral theta-band phase variability, reaction time variability, and attention-Deficit/Hyperactivity disorder symptoms in a twin study. *Biological Psychiatry*, 75(3), 238–247. https://doi.org/10.1016/J.BIOPSYCH.2013.07.020

Michelini, G., Norman, L. J., Shaw, P., & Loo, S. K. (2022). Treatment biomarkers for ADHD: Taking stock and moving forward. *Translational Psychiatry*, 12(1), 444. https://doi.org/10.1038/s41398-022-02207-2

Näpflin, M., Wildi, M., & Sarnthein, J. (2007). Test–retest reliability of resting EEG spectra validates a statistical signature of persons. *Clinical Neurophysiology*, 118(11), 2519–2524. https://doi.org/10.1016/j.clinph.2007.07.022

Neale, M. C., & Maes, H. H. M. (2004). *Methodology for genetic studies of twins and families*. Kluwer Academic Publishers B.V.

Nimmo-Smith, V., Merwood, A., Hank, D., Brandling, J., Greenwood, R., Skinner, L., Law, S., Patel, V., & Rai, D. (2020). Non-pharmacological interventions for adult ADHD: A systematic review. *Psychological Medicine*, 50(4), 529–541. https://doi.org/10.1017/S0033291720000069

Ossadtchi, A., Shamaeva, T., Okorokova, E., Moiseeva, V., & Lebedev, M. A. (2017). Neurofeedback learning modifies the incidence rate of alpha spindles, but not their duration and amplitude. *Scientific Reports*, 7(1), 3772. https://doi.org/10.1038/s41598-017-04012-0

Pievsky, M. A., & McGrath, R. E. (2018). The neurocognitive profile of attention-Deficit/Hyperactivity disorder: A review of meta-analyses. *Archives of Clinical Neuropsychology*, 33(2), 143–157. https://doi.org/10.1093/arclin/acx055

Pion-Tonachini, L., Kreutz-Delgado, K., & Makeig, S. (2019). ICLabel: An automated electroencephalographic independent component classifier, dataset, and website. *NeuroImage*, 198, 181–197. https://doi.org/10.1016/j.neuroimage.2019.05.026

Poil, S.-S., Bollmann, S., Ghisleni, C., O'Gorman, R. L., Klaver, P., Ball, J., Eich-Höchli, D., Brandeis, D., & Michels, L. (2014). Age dependent electroencephalographic changes in attention-deficit/hyperactivity disorder (ADHD). *Clinical Neurophysiology*, 125(8), 1626–1638. https://doi.org/10.1016/j.clinph.2013.12.118

Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry*, 164(6), 942–948. https://doi.org/10.1176/ajp.2007.164.6.942

Ponomarev, V. A., Mueller, A., Candrian, G., Grin-Yatsenko, V. A., & Kropotov, J. D. (2014). Group independent Component analysis (gICA) and current source density (CSD) in the study of EEG in ADHD adults. *Clinical Neurophysiology*, 125(1), 83–97. https://doi.org/10.1016/j.clinph.2013.06.015

Riddle, J., & Frohlich, F. (2021). Targeting neural oscillations with transcranial alternating current stimulation. *Brain Research*, 1765, 147491. https://doi.org/10.1016/j.brainres.2021.147491

Riddle, J., & Frohlich, F. (2023). Mental activity as the bridge between neural biomarkers and symptoms of psychiatric illness. *Clinical EEG and Neuroscience*, 54(4), 399–408. https://doi.org/10.1177/15500594221112417

Rimfeld, K., Malanchini, M., Spargo, T., Spickernell, G., Selzam, S., McMillan, A., Dale, P. S., Eley, T. C., & Plomin, R. (2019). Twins early development study: A genetically sensitive investigation into behavioral and cognitive development from infancy to emerging adulthood. *Twin Research and Human Genetics*, 22(6), 508–513. https://doi.org/10.1017/thg.2019.56

Rodriguez-Larios, J., & Alaerts, K. (2020). EEG alpha-theta dynamics during mind wandering in the context of breath focus meditation: An experience sampling approach with novice meditation practitioners. *European Journal of Neuroscience*, 53(May), 1–14. https://doi.org/10.1111/ejn.15073

Rodriguez-Larios, J., Bracho Montes de Oca, E. A., & Alaerts, K. (2021). The EEG spectral properties of meditation and mind wandering differ between experienced meditators and novices. *NeuroImage*, 245, 118669. https://doi.org/10.1016/J.NEUROIMAGE.2021.118669

Rodriguez-Larios, J., & Haegens, S. (2023). Genuine beta bursts in human working memory: Controlling for the influence of lower-frequency rhythms. *Advances.in/Psychology*, 1, 1–17. https://doi.org/10.56296/aip00006

Rodriguez-Larios, J., Wong, K. F., & Lim, J. (2024). Assessing the effects of an 8-week mindfulness training program on neural oscillations and self-reports during meditation practice. *PLoS One*, 19(6), e0299275. https://doi.org/10.1371/journal.pone.0299275

Skirrow, C., Mcloughlin, G., Banaschewski, T., Brandeis, D., Kuntsi, J., & Asherson, P. (2014). Normalisation of frontal theta activity following methylphenidate treatment in adult attention-deficit/hyperactivity disorder. *European Neuropsychopharmacology*, 25(1), 85–94. https://doi.org/10.1016/j.euroneuro.2014.09.015

Smit, C. M., Wright, M. J., Hansell, N. K., Geffen, G. M., & Martin, N. G. (2006). Genetic variation of individual alpha frequency (IAF) and alpha power in a large adolescent twin sample. *International Journal of Psychophysiology*, 61(2), 235–243. https://doi.org/10.1016/j.ijpsycho.2005.10.004

Soriano, J. R., Oca, E. B. M. de, Karaiskou, A.-I., Vuyst, H.-J. D., Rodriguez-Larios, J., Feng, N., Varon, C., & Alaerts, K. (2024). Bidirectional alpha power EEG neurofeedback during a focused attention meditation practice in novices. *NeuroRegulation*, 11(3), 230–241. Article 3. https://doi.org/10.15540/nr.11.3.230

Valdés-Hernández, P. A., Ojeda-González, A., Martínez-Montes, E., Lage-Castellanos, A., Virués-Alba, T., Valdés-Urrutia, L., & Valdes-Sosa, P. A. (2010). White matter architecture rather than cortical surface area correlates with the EEG alpha rhythm. *NeuroImage*, 49(3), 2328–2339. https://doi.org/10.1016/j.neuroimage.2009.10.030

van Beijsterveldt, C. E. M., & van Baal, G. C. M. (2002). Twin and family studies of the human electroencephalogram: A review and a meta-analysis. *Biological Psychology*, 61(1), 111–138. https://doi.org/10.1016/S0301-0511(02)00055-8

van Dongen-Boomsma, M., Lansbergen, M. M., Bekker, E. M., Sandra Kooij, J. J., van der Molen, M., Kenemans, J. L., & Buitelaar, J. K. (2010). Relation between resting EEG to cognitive performance and clinical symptoms in adults with attention-deficit/hyperactivity disorder. *Neuroscience Letters*, 469(1), 102–106. https://doi.org/10.1016/j.neulet.2009.11.053

Veniero, D., Vossen, A., Gross, J., & Thut, G. (2015). Lasting EEG/MEG aftereffects of rhythmic transcranial brain stimulation: Level of control over oscillatory network activity. *Frontiers in Cellular Neuroscience*, 9, 477. https://doi.org/10.3389/fncel.2015.00477

Voetterl, H. T. S., Sack, A. T., Olbrich, S., Stuiver, S., Rouwhorst, R., Prentice, A., Pizzagalli, D. A., van der Vinne, N., van Waarde, J. A., Brunovsky, M., van Oostrom, I., Reitsma, B., Fekkes, J., van Dijk, H., & Arns, M. (2023). Alpha peak frequency-based Brainmarker-I as a method to stratify to pharmacotherapy and brain stimulation treatments in depression. *Nature Mental Health*, 1(12), 1023–1032. https://doi.org/10.1038/s44220-023-00160-7

von Stein, A., & Sarnthein, J. (2000). Different frequencies for different scales of cortical integration: From local gamma to long range alpha/theta synchronization. *International Journal of Psychophysiology*, 38(3), 301–313. https://doi.org/10.1016/S0167-8760(00)00172-0

Vossen, A., Gross, J., & Thut, G. (2015). Alpha power increase after transcranial alternating current stimulation at alpha frequency (α-tACS) reflects plastic changes rather than entrainment. *Brain Stimulation*, 8(3), 499–508. https://doi.org/10.1016/J.BRS.2014.12.004

Woltering, S., Jung, J., Liu, Z., & Tannock, R. (2012). Resting state EEG oscillatory power differences in ADHD college students and their peers. *Behavioral and Brain Functions*, 8(1), 60. https://doi.org/10.1186/1744-9081-8-60