Error-related brain activity: A time-domain and time-frequency investigation in pediatric obsessive-compulsive disorder

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Abstract

Increased error-related negativity (ERN), a measure of error monitoring, has been suggested as a biomarker of obsessive-compulsive disorder (OCD). Additional insight into error monitoring is possible using time-frequency decomposition of electroencephalographic (EEG) data, as it allows disentangling the brain's parallel processing of information. Greater error-related theta is thought to reflect an error detection signal, while delta activity may reflect more elaborative post-detection processes (i.e., strategic adjustments). Recent investigations show that decreased error-related alpha may index attentional engagement following errors; additionally, increases and decreases in error-related beta could reflect motor inhibition and motor preparation, respectively. However, time-frequency dynamics of error monitoring in OCD are largely unknown. The present study examined time-frequency theta, delta, alpha and beta power in early adolescents with OCD using a data-driven, cluster-based approach. The aim was to explore electrocortical measures of error monitoring in early adolescents with (n = 27, 15 females) and without OCD (n = 27, 14 females) during an arrowhead version of the flanker task while EEG activity was recorded. Results indicated that the OCD group was characterized by increased ERN and theta, as well as reduced error-related beta power compared to participants without OCD. Greater error-related beta explained variance in OCD over and above the ERN and error-related theta. By examining separate time-frequency measures, the present study provides novel insights into the dynamics of error monitoring, suggesting that pediatric OCD may be characterized by enhanced error monitoring (i.e., greater theta power) and post-error inhibition (i.e., reduced beta power decrease).

Keywords: OCD; error monitoring; EEG; ERN; Time-Frequency

1. Introduction

Obsessive-compulsive disorder (OCD) is a highly debilitating psychiatric disorder characterized by the presence of intrusive recurrent unwanted thoughts and repetitive behaviors that lead to significant functional impairment (American Psychiatric Association, 2013). The peak onset of OCD occurs in late childhood and early adolescence (Tanidir et al., 2015) affecting approximately 3% of the pediatric population (Thomsen, 2013). Children usually show a pre-pubertal onset of the symptoms, but the disorder may go unrecognized for some time because thoughts and rituals are not recognized as irrational, and limited verbal skills prevent children from articulating their obsessions (Geller, Homayoun, & Johnson, 2021). When left untreated, OCD follows a chronic course (Skoog & Skoog, 1999) resulting in an increased risk for long-term psychiatric comorbidities (Stewart et al., 2004). Despite this burden, the pathophysiology of OCD is still unclear. Recent research has focused on neural signatures of OCD to enhance our understanding of its biological correlates and to inform early identification and the design of novel interventions (Perera, Bailey, Herring, & Fitzgerald, 2019).

Obsessions and compulsions are often related to preventing a feared outcome that may arise following the commission of an error that gives rise to feelings of "wrongness" (Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005). This relates to neurocognitive impairments that characterize both adult and pediatric OCD, such as hyperactive error monitoring (e.g., De Wit et al., 2012; Liu, Gehring, Weissman, Taylor, & Fitzgerald, 2012). Neurobiological models of OCD consistently suggest that the activity of cortico-striatal circuits connecting several areas with the anterior cingulate cortex (ACC), a region associated with the detection of cognitive conflict and error monitoring, has a role in the etiology of this disorder (Milad & Rauch, 2012). Several studies showed reduced gray matter volume and increased brain activity following the commission of an error in the ACC and other areas

within error-processing networks in both adult (Grüzmann et al., 2022; Norman et al., 2019; Rotge et al., 2009) and pediatric samples with OCD (Gilbert, Barclay, Tillman, Barch, & Luby, 2018).

To explore error monitoring, studies using electroencephalography (EEG) have predominantly focused on an event-related potential (ERP) called error-related negativity (ERN; Endrass & Ullsperger, 2014; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN is a fronto-central response-locked event-related potential (ERP) characterized by a negative deflection that peaks approximately 50 ms following the commission of an error during speeded response conflict tasks (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). The primary generator of the ERN has been suggested to be the ACC (Debener et al., 2005; Fitzgerald et al., 2005). The ERN may function as an early indicator signaling the need for increased cognitive control to correct behavior (Weinberg, Riesel, & Hajcak, 2012; Weinberg et al., 2016). Individual differences in the amplitude of the ERN are thought to reflect threat sensitivity; namely, variation in the degree to which making mistakes is experienced as aversive to an individual (Hajcak & Foti, 2008; Meyer, Bress. & Hajcak Proudfit, 2014; Meyer, 2017; Weinberg et al., 2016).

There is consistent evidence showing enhanced (i.e., more negative) ERN amplitudes in adults and children with OCD compared to controls with no history of psychopathology (e.g., Carrasco et al., 2013; Endrass, Riesel, Kathmann, & Buhlmann, 2014; Hajcak, Franklin, Foa, & Simons, 2008; Mathews et al., 2016; Nawani et al., 2018; Perera et al., 2019). This is supported by a meta-analysis of 40 studies that found an increased ERN across various populations with OCD (adults, children, subthreshold OCD), collectively suggesting that hyperactive neural error signals might represent part of the pathophysiology of OCD (Riesel, 2019). Taken together, the presence of an enhanced ERN in pediatric OCD suggests that this measure is a promising neural correlate of OCD that is evident across development.

Recently, there has been a growing interest in integrating the time-frequency dynamics of error monitoring, in addition to the ERN. Indeed, time-frequency analysis of EEG activity within specific frequency bands allows the extrapolation of information that is not available using ERP analysis and fully leverage multiple dimensions of the signal, reflecting distinctive aspects of information processing (Cohen, 2014; Morales et al., 2022; Munneke, Nap, Schippers, & Cohen, 2015). Specifically, error monitoring can be examined in terms of power across several frequency bands. Greater power in the delta and theta bands are observed following error relative to correct responses, indicating that these frequency bands are both involved in error monitoring processes (Beatty, Buzzell, Roberts, & McDonald, 2020; Cavanagh, Cohen, & Allen, 2009; Cavanagh, Meyer, & Hajcak, 2017; Luu, Tucker, & Makeig, 2004; Muir, Hedges-Muncy, Clawson, Carbine, & Larson, 2020; Munneke et al., 2015; Sandre & Weinberg, 2019; Trujillo & Allen, 2007). In particular, error-related theta is the most prominent oscillatory activity underlying the ERN and has been interpreted as reflecting an early error-detection signal that leads to the recruitment of greater cognitive control (Ullsperger, Fischer, Nigbur, & Endrass, 2014). Increased error-related delta activity may reflect post-detection processes, such as strategic adjustments in behavior (Sandre et al., 2019).

Additionally, alpha power has been investigated in the context of error processing (Carp et al., 2009; Li et al., 2021; van Driel, Ridderinkhof, & Cohen, 2012). Alpha power represents an inverse index of cortical arousal such that task-related alpha suppression is thought to reflect greater attentional engagement (Freeman & Quiroga, 2012; van Ede, De Lange, Jensen, & Maris, 2011). A decrease in alpha power following error relative to correct responses has been found, suggesting that error-related alpha increases or decreases may reflect increased attention following error detection (Carp et al., 2009; Li et al., 2021). Relatively fewer studies have examined the contributions of the beta frequency band, which

is generated in sensorimotor areas (Tzagarakis, West, & Pellizzer, 2015), and may enable motor-action preparation (Wilhelm, Threadgill, & Gable, 2021, 2022). Beta oscillations are suppressed during action preparation (McFarland, Miner, Vaughan, & Wolpaw, 2000; Pfurtscheller, Pregenzer, & Neuper, 1994; Yang, Leung, Plank, Snider, & Poizner, 2015), while increased beta power reflects inhibition of prepared actions in multiple tasks also related to error monitoring (Li et al., 2021; Rosin et al., 2011; Swann et al., 2012; Wach et al., 2013; Wessel et al., 2016).

Time-frequency dynamics of error monitoring in OCD remain largely unexplored, especially in pediatric samples. Recently, increased error-related theta power during a flanker task was found in pediatric OCD (Suzuki et al., 2022). Adults with OCD were characterized by enhanced ERN and CRN as well as increased delta and theta power for both error and correct trials (Riesel, Kathmann, & Endrass, 2014). However, error-related alpha and beta power have not been assessed in OCD. Considering that adults and children with OCD are characterized by post-error slowing and difficulties in making trial-by-trial adjustments (e.g., Fitzgerald et al., 2005; Liu et al., 2012), OCD may be associated with greater error-related beta.

Another question is whether the combination of the ERN and time-frequency measures could be used together to improve the identification of OCD. Thus, it is important to determine whether ERN and spectral measures of error processing explain unique or overlapping variance in OCD. For instance, Riesel and colleagues (2014) found that ERN, CRN, delta, and theta share substantial common variance, although they did not test whether various error-related neural measures explained independent variance in OCD status.

The main goal of the current work was to examine error monitoring in early adolescence, among individuals with and without OCD. To this end, we analyzed ERN as well as time-frequency power within the delta, theta, alpha, and beta band. Consistent with

previous literature (e.g., Hajcak et al., 2008; Riesel, 2019), the OCD group was expected to show larger ERN amplitude compared to controls. Regarding time-frequency measures, we hypothesized that participants with OCD would show greater error-related theta and delta power relative to control participants (Riesel et al., 2014). Considering the exploratory nature of this work, no a priori hypotheses were formulated regarding alpha and beta power in OCD. Lastly, to provide further information on the pathophysiology of OCD, an exploratory aim of this study was to examine whether using ERN and time-frequency measures explain unique variance in OCD status.

2. Method

2.1 Participants

Participants were part of a large, multi-site longitudinal study that was funded by the U.S. National Institute of Mental Health (NIMH, MH106477) and aimed to examine the effectiveness of a computerized adaptive attention bias modification training in modifying neural activity associated with errors and anxiety symptoms in adolescence. The present study included data from a subset of participants collected at the baseline visit: 54 participants (29 females) between the ages of 11 and 14 years (M = 12.4 years; SD = 1.09). For the larger project, families were recruited via a commercial mailing list, referrals, and other advertisements. Families were eligible to participate if they had a daughter or a son aged between 11 and 14 years with no known medical or developmental disability, a biological parent willing to participate, and the ability to read and write English.

Participants were divided into an OCD group (n = 27, 15 females) and a healthy control group (n = 27, 14 females) matched for age and gender. The presence of OCD was determined by a trained clinical interviewer using the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS, Birmaher et al., 2009). Table 1 includes

the characteristics of the sample. Both groups were characterized by mostly Caucasian participants (HC: *n* = 18; OCD: *n* = 18), while few participants were Hispanic (HC: *n* = 5; OCD: *n* = 1), African American (HC: *n* = 1; OCD: *n* = 5), Asian (HC: *n* = 2; OCD: *n* = 0) or belonged to other ethnic groups (HC: *n* = 1; OCD: *n* = 3). Exclusion criteria included a current and history of cardiovascular, neurological diseases, and other psychiatric conditions. Three participants with OCD were taking psychotropic medications (i.e., antidepressants, stimulants, mood stabilizers)[1]. All participants had a normal or corrected-to-normal vision and were naive to the purpose of the experiment. Participants were compensated for their participation ($20 per hour). Parents/guardians and participants provided consent and assent prior to participating. The present study was conducted in accordance with the Declaration of Helsinki and was approved by the local Ethics Committee of Florida State University and San Diego State University.

2.2 Procedure

Participants completed a lab visit that lasted approximately 4–5 hours that consisted of multiple tasks, including self-report questionnaires, and psychophysiological and neuroimaging tasks. Moreover, the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime Version (KSADS; Kaufman et al., 1997) was used to assess current and lifetime psychiatric history in the adolescent participants. The KSADS is a semi-structured clinical interview with good psychometric properties (Birmaher et al., 2009). The KSADS was conducted with parents and participants, separately, by trained interviewers under supervision of experienced, Ph.D.-level clinical psychologists (Amir et al., 2022). Relevant to the current study, participants completed a flanker task while continuous EEG data were collected. Stimulus-locked ERP data from the

flanker task as a part of this study has been previously published elsewhere (see Santopetro, Kallen, Threadgill, Amir, & Hajcak, 2022).

2.3 Flanker Task and behavioral data reduction

The continuous EEG was recorded while participants completed an arrow version of the flanker task administered through the Presentation software (Neurobehavioral Systems, Inc., Albancy, CA). On each trial, five horizontally aligned arrowheads were presented for 200 ms, followed by an ITI that varied between 2300 and 2800 ms. Half of the trials were compatible ("<<<<<" or ">>>>>") and half were incompatible ("<<><<" or ">><>>"); the order of trials was randomly determined. Participants were instructed to respond as quickly and as accurately as possible using their right hand on the mouse by pressing the right mouse button if the central arrow was pointing to the right, and the left mouse button if the central arrow was pointing left. Participants completed a practice block of 30 trials to ensure adequate performance. The task consisted of 11 blocks of 30 trials (330 trials total). At the end of each block, participants received feedback based on their performance. If performance was 75% correct or lower, the message "Please try to be more accurate" was presented; if performance was above 90% correct, the message "Please try to respond faster" was displayed; otherwise, the message "You're doing a great job" was shown.

The first trial of each block and trials with no response were excluded from the analysis. Due to their skewness (skewness before transformation = 1.65; skewness after transformation = 0.33), RTs were log-transformed to produce the normal distribution required for analyses.

2.4 Electroencephalogram recording

Continuous EEG was recorded during the flanker task using a 34-channel system (ActiCHamp system, Brain Products) placed according to the 10/20 system and two electrodes on the left and right mastoid, and Cz was used as the online reference. Fpz served as the ground electrode. Electrooculogram was recorded from electrodes placed above and below the right eye and two placed on the outer canthus of both eyes. The EEG was digitized with a sampling rate of 1000 Hz, with a low-pass fifth-order sinc filter with a half-power cutoff set at 100 Hz.

2.5 EEG data processing

2.5.1 Time-domain analysis

Data were processed offline with Brain Vision Analyzer (Brain Products, Gilching, Germany). EEG data were referenced to the average mastoid electrodes and filtered with low and high filter cutoffs set at 0.01 Hz and 30 Hz, respectively. For analyses of response-related ERPs (i.e., ERN, CRN, and Pe), EEG segments of 1,500 ms were extracted from the continuous EEG, beginning 500 ms prior to responses. Data were then corrected for eye-movements and blinks (Gratton, Coles, & Donchin, 1983). Segments containing voltage steps >50 µV between sample points, a voltage difference of 300 µV within a single trial, or a maximum voltage difference of <0.5 µV within 100-ms intervals were automatically rejected and additional artifacts were identified and removed based on visual inspection. ERP averages were created for error and correct trials and a baseline of the average activity from −500 to–300 ms prior to the response was subtracted from each data point. Only participants with at least six usable error trials were included (Olvet & Hajcak, 2009). Based on previous research (e.g., Klawohn, Hajcak, Amir, Kathmann, & Riesel 2020; Meyer & Klein 2018), the error-related negativity (ERN) and correct-related negativity (CRN) were scored as the average voltage in the window between 0 ms and 100 ms after response commission on error

and correct trials, respectively; the CRN and ERN were quantified at electrode site FCz, where error-related brain activity was maximal. The error positivity (Pe) was scored at electrode site Pz as the mean amplitude from 200 to 400 ms after errors, and the respective mean amplitude after correct responses was analyzed as the correct positivity (Pc).

2.5.2 Time-frequency analysis

To conduct time-frequency analyses, EEG data were processed offline in Brainstorm (Tadel, Baillet, Mosher, Pantazis, & Leahy, 2011). The signal was filtered offline with low and high filter cutoffs set at 0.3 Hz and 30 Hz, respectively, to minimize slow drifts that could have adverse effects on time-frequency decomposition. Blink artifacts were corrected using independent component analysis (ICA) and segmented for each trial 500 ms before stimulus onset to 1000 ms after onset. Each epoch was baseline-corrected by subtracting the mean pre-stimulus voltage between −250 ms and −50 ms. Then, segments containing residual artifacts exceeding ±70 μV (peak-to-peak) were excluded. Four participants were not included in the time-frequency analysis as their brain activity was recorded from a limited number of electrodes.

Time-frequency analysis was conducted using Morlet wavelet transformation on individual trials for each 1-Hz frequency bin between 1 and 30 Hz, using a mother wavelet at 1 Hz with 3-s time resolution (as calculated by the full width at half maximum, FWHM). Time-frequency decompositions were then averaged for each participant and condition (error and correct trials), and the event-related spectral perturbation (ERSP) was computed as the change in power expressed in decibels (dB) relative to the baseline (−300 to −100 ms) in each frequency bin at each time point. Then, data were grand averaged across each participant for each condition.

2.6 Statistical analyses

Statistical analyses were conducted using Rstudio (version 1.2.5001; R Core Team, 2012), Jamovi (The Jamovi Project, 2021), and Matlab using a two-tailed $\alpha = .05$.

Behavioral performance analyses were conducted using the *lmer* function from the *lme4* (Bates, Mächler, Bolker, & Walker, 2015) and *lmerTest* packages in R (Kuznetsova, Brockhoff, & Christensen, 2017). Two models that included RT as the dependent variable, trial type (correct, error or post-error, post-correct) and group (OCD, HC) and their interaction as fixed effects and participant as a random effect were conducted. The models were re-fitted after excluding outliers, which were identified as observations with absolute standardized residual greater than 3 (0.01% of all trials; e.g., Ambrosini, Pezzulo, & Costantini, 2015). The *p*-values obtained through the Satterthwaite approximation and model parameters were estimated using restricted maximum likelihood estimation (REML). Significant effects followed by Tukey HSD post-hoc tests to correct for multiple comparisons.

For the time-domain data, residualized differences scores were computed to isolate variance specific to each measure by saving the unstandardized residuals in linear regressions predicting values on error trials from values on correct trials (Meyer, Lerner, De Los Reyes, Laird, & Hajcak, 2017). Since we were interested in examining error-related brain activity differences between the two groups, two separate one-way ANOVAs were used compare the ERN_{res} and the Pe_{resid} across the two groups.

Since the time-frequency dynamics related to error monitoring are not well-established, a data-driven approach was selected for time-frequency data. A cluster-based permutation approach was conducted to identify trial type (error and correct trials) differences in event-related delta (1–3 Hz), theta (4–8 Hz), alpha (9–14 Hz) and beta (15–20 Hz) as implemented by the FieldTrip toolbox (Oostenveld, Fries, Maris, & Schoffelen, 2011).

This method adequately controls type I error rate arising from conducting multiple statistical tests across electrodes and time points (Maris & Oostenveld, 2007). With this approach, by iteratively shuffling the condition labels over trials or over subjects and recomputing the statistics, a theoretical underlying distribution of the test statistics under the null hypothesis is generated by the data itself. Whenever the test statistic associated with the non-shuffled data falls within the distribution of the null-hypothesis test statistic values, the null hypothesis cannot be rejected and this would indicate that the observed data could have been randomly generated (Cohen, 2014; Luck, 2014). With cluster-based correction, at each iteration of the null-hypothesis distribution generation, the outcome is units of clusters instead of single pixels (i.e., electrodes; Cohen, 2014). In the present study, the differences within conditions (correct and error trials) were shuffled pseudo-randomly 2000 times. To obtain a 'null' distribution of effect sizes, the maximal cluster-level statistics (i.e., the sum of values across contiguously significant electrodes and time points at the threshold level) were extracted for each shuffle. For each significant cluster in the (non-shuffled) data, the cluster-corrected *p*-value was computed as the statistics of the proportion of clusters in the null distribution that exceeded the one obtained for the cluster in question. Clusters with a $p_{corr} < .05$ were considered statistically significant. Cluster-based repeated measures ANOVAs were conducted to test within-subjects differences in event-related power changes between conditions (error vs. correct) and two-tailed independent samples *t*-tests were conducted to test between-groups (i.e., OCD versus HC) differences within each condition.

Finally, to obtain time-frequency components with specific timing and location, power was extracted according to the significant frequency band, window, and location (i.e., sensors) that emerged from the between-group differences. For logistic regression and correlational analyses, residualized difference scores for time-domain and time-frequency measures were determined by saving the unstandardized residuals in linear regressions

predicting values on error trials from values on correct trials. Pearson and point-biserial correlations between each significant time-frequency cluster measure (i.e., significantly different between the two groups from the cluster analysis), the ERN_{resid}, the Pe_{resid}, behavioral RTs, and group status were conducted across the whole sample. Then, a logistic regression was conducted to examine the amount of shared variance explained by each significant time-frequency and time-domain measure in determining the likelihood of OCD diagnosis. The collinearity was tested by calculating the Variance Inflation Factors (VIF) with the *vif* function of the *car* package (Fox, Weisberg, & Price, 2019).

3. Results

3.1 Behavioral data

The two groups did not differ in terms of number of errors (HC: M_{err} = 45.6, SD_{err} = 23.2; OCD: M_{err} = 47.2, SD_{err} = 19.3; $t(52) = -0.27$, $p = .80$). Descriptive statistics of response times for the two groups are shown in Table 2. The first model showed that all participants were faster on error trials compared to correct trials ($F_{1, 17393} = 2408.64$, $p < .001$). Overall, participants with OCD were faster than healthy controls ($F_{1,53} = 7.33$, $p = .009$); however, this was qualified by a significant interaction between group and trial type ($F_{1, 17393} = 18.75$, $p < .001$), where the OCD group was faster than the HC group in error trials (Tukey post-hoc: $p = .006$) but not in correct trials ($p = .171$).

Additionally, the second model showed that participants were slower to generate a correct response on trials that occurred after an error compared to trials that occurred after a correct response ($F_{1, 17415} = 72.30$, $p < .001$), but no group differences emerged in post-error slowing. No other behavioral differences emerged.

3.2 ERPs

As shown in Figure 1, the OCD group showed a larger (more negative) ERN_{resid} compared to the HC group ($F_{1,52} = 4.11$, $p = .048$, Cohen's $d = 0.55$), while the two groups did not differ in the Pe_{resid} ($F_{1,52} = 0.85$, $p = .360$). The ERN was larger (more negative) than the CRN ($F_{1,53} = 39{,}4$, $p < .001$) and the Pe was larger than the Pc (more positive) ($F_{1,53} = 217$, $p < .001$) across the whole sample. Table 2 illustrates descriptive statistics of all EEG measures.

3.3 Time-frequency differences between error and correct trials

5.3.1 Delta power (1- 3 Hz)

The cluster-based analysis on event-related delta power showed a significantly greater delta power to error trials relative to correct trials (electrodes = FP1, FZ, F3, F7, FCZ, FC5, FC1, C3, T7, CP5, CP1, PZ, P3, P7, O1, OZ, O2, P4, P8, CP6, CP2, C4, T8, FC6, FC2, F4, F8, FP2, CZ; cluster *F*-valuemax =342479.74, *p*corr < .001, time window 0 to 1000 ms; Cohen's $d = 1.18$; Figure 2, panel a and b).

3.3.2 Theta power (4 - 8 Hz)

The cluster-based analysis on event-related theta power showed a significantly greater theta power to error trials relative to correct trials (electrodes = FP1, FZ, F3, F7, FCZ, FC5, FC1, C3, T7, CP5, CP1, PZ, P3, P7, O1, OZ, O2, P4, P8, CP6, CP2, C4, T8, FC6, FC2, F4, F8, FP2, CZ; cluster *F*-valuemax =154576.14, *p*corr < .001, time window 0 to 490 ms; Cohen's $d = 0.84$; Figure 2, panel c and d).

3.3.3 Alpha power (9 – 14 Hz)

The cluster-based analysis on event-related alpha power showed a significantly greater alpha suppression (i.e., decreased power) to error relative to correct trials (electrodes

= FP1, FZ, F3, F7, FCZ, FC5, FC1, C3, T7, CP5, CP1, PZ, P3, P7, O1, OZ, O2, P4, P8, CP6, CP2, C4, T8, FC6, FC2, F4, F8, FP2, CZ; cluster *F*-valuemax =165354.29, *p*corr < .001, time window 0 to 1000 ms; Cohen's *d* = 0.75; Figure 2, panel e and f).

3.3.4 Beta power (15 – 20 Hz)

The cluster-based analysis on event-related beta power showed a significantly greater beta suppression (i.e., decreased power) to error relative to correct trials (electrodes = FP1, FZ, F3, F7, FCZ, FC5, FC1, C3, T7, CP5, CP1, PZ, P3, P7, O1, OZ, O2, P4, P8, CP6, CP2, C4, T8, FC6, FC2, F4, F8, FP2, CZ; cluster *F*-valuemax =63632.68, *p*corr < .001, time window 0 to 538 ms; Cohen's *d* = 0.72, Figure 2, panel g and h).

3.4 Time-frequency differences between groups

Cluster-based analyses on event-related theta power revealed a significantly greater theta power to error trials in the OCD group relative to the HC group (electrodes = FP1, FZ, F3, F7, FCZ, FC5, FC1, C3, T7, CP5, CP1, PZ, P3, P4, CP2, C4, FC6, FC2, F4, F8, FP2, CZ; cluster *t*-valuemax = 7658.27; p_{corr} = .030, time window = 0 to 346 ms; Cohen's *d* = 0.67; Figure 3, panel a, b and c). There were no group differences in theta power to correct trials.

Similarly, cluster-based analyses on event-related beta power revealed a significantly greater beta power to error trials in the OCD group relative to the HC group (electrodes = FP1, FZ, F3, F7, FCZ, FC5, FC1, C3, T7, CP1, PZ, P3, OZ, O2, P4, P8, CP6, CP2, C4, FC6, FC2, F4, F8, FP2, CZ; cluster *t*-valuemax = 7310.13; *p*corr = .010, time window = 0 to 488 ms; Cohen's *d* = 0.78; Figure 3, panel d, e and f). There were no group differences in beta power to correct trials. No other group differences in time-frequency power were observed.

3.5 Correlations

Correlations between study variables are shown in Table 3. Increased $Beta_{resid}$ was associated with slower RTs on trials that occurred after an error (i.e., post-error) and error trials. Greater $Theta_{resid}$ was associated with faster RTs in trials that occurred after an error (i.e., post-error), post-correct, and correct trials, and, only marginally, on error trials. The ERN_{resid} was negatively correlated with $Theta_{resid}$ and associated with faster RTs on correct trials, error trials, post-error, and post-correct trials. $Beta_{resid}$ was significantly correlated with $Alpha_{resid,}$ and $Delta_{resid}$ was significantly correlated with $Theta_{resid.}$ OCD group status related to ERN and $Theta_{resid}$ and $Beta_{resid}$. There were no other significant correlations among significant time-frequency and time-domain or behavioral measures.

3.6 Logistic regression

Results of the logistic regressions are shown in Table 4. The first regression model revealed that $Theta_{resid}$, but not the ERN_{resid}, was related to increased likelihood of being diagnosed with OCD. That is, ERN_{resid} accounted for overlapping variance in OCD status with $Theta_{resid}$, and only the latter remained a significant and independent predictor of OCD status. The second multiple logistic regression that included all significant measures that emerged from the cluster-based analyses showed that greater $Beta_{resid}$, but not $Theta_{resid}$ or ERN_{resid}, was independently related to increased likelihood of being diagnosed with OCD. Of note, there was a trend evident for $Theta_{resid}$, such that greater $Theta_{resid}$ was associated with a diagnosis of OCD. VIF values were all < 1.30, indicating low multicollinearity.

4. Discussion

The main goal of the present study was to explore error-related time-frequency brain dynamics in a sample of early adolescents with OCD relative to a healthy control group. Regarding within-groups patterns, consistent with previous literature (e.g., Cavanagh et al.,

2017; Muir et al., 2020; Munneke et al., 2015; Sandre & Weinberg, 2019), the cluster-based analysis revealed increased theta and delta power on error relative to correct trials. Moreover, these two frequency bands had different time courses and they may reflect different processes relevant to error monitoring. For example, theta activity may index an initial evaluation of the primary response outcome (e.g., Cavanagh et al., 2009; Cavanagh & Frank, 2014), whereas delta power to errors may reflect more elaborative process during error monitoring (Bernat, Nelson, & Baskin-Sommers, 2015; Watts & Bernat, 2018), such as the processing of higher-level aspects of outcomes (e.g., outcome magnitude, expectancy). Along similar lines, recent work examining time-frequency dynamics of error monitoring in uncertain contexts has shown that theta power may reflect the signaling of increased need for control, whereas delta may reflect subsequent elaborative processing aimed at strategically adjusting behavior (Sandre & Weinberg, 2019).

Moreover, in line with previous work (Li et al., 2021), participants showed a decrease of alpha power (i.e., desynchronization) on error relative to correct trials. Considering that alpha activity desynchronization is thought to index cortical arousal (van Ede et al., 2011), this pattern might reflect increased engagement and attentional resources needed to adjust subsequent behavior following the commission of an error (Carp et al., 2009; Li et al., 2021). Also, a beta power decrease on error relative to correct trials was observed across the whole sample. Considering previous work showing that beta band activity is associated with motor processing during a flanker task (Li et al., 2021; Wilhelm et al., 2022), this pattern is consistent with the possibility that beta activity may be related to motor preparation for subsequent trials. Indeed, individuals with greater error-related beta also had longer RTs on trials that followed the commission of an error (i.e., post-error trials). Future studies should be designed to explore the link between beta power and other ERPs associated with action preparation, such as the lateralized readiness potential (Dayan, Berger, & Anholt, 2014;

Dayan, Berger, & Anholt, 2017; Frota Lisbôa Pereira de Souza, 2021; Morand-Beaulieu, Aardema, O'Connor, & Lavoie, 2021; Schurger, Pak, & Roskies, 2021).

Consistent with previous work on the ERN-OCD relationship (e.g., Carrasco et al., 2013; Endrass et al., 2014; Hajcak et al., 2008), the current study found that the ERN was potentiated among those with OCD compared to controls. In addition to a larger ERN, the OCD group was characterized by enhanced theta power to error but not correct trials; ERN amplitude and theta power were negatively correlated with each other across the whole sample (i.e., greater error-related theta power was associated with greater error-related negativity). On the other hand, ERN was not associated with delta, alpha or beta activity. These results are generally consistent with previous work on ERN and theta activity in OCD (Riesel et al., 2014; Suzuki et al., 2022), as well as the potentially shared functional role of the ERN and error-related theta (e.g., Cavanagh et al., 2009). Consistent with the ERN topography, the theta power cluster that differed between the two groups was maximal at fronto-central scalp sites, providing support that these electrophysiological signatures may be similarly generated by the ACC (e.g., Debener et al., 2005).

Unique to the current study, the OCD group was characterized by increased beta power on error (but not correct) trials, relative to the control group. Based on previous literature (e.g., Li et al., 2021; Wilhelm et al., 2022), this finding could indicate that participants with OCD are characterized by reduced motor preparation following errors (e.g., greater motor inhibition). Consistent with this possibility, increased error-related beta was associated with slower post-error RTs, linking increased beta power on error trials to increased behavioral inhibition on subsequent trials. Additionally, the significant beta cluster was maximal in parietal scalp sites, providing further support for this functional interpretation of error-related beta power and for the link between alterations in motor-related regions and OCD (de Wit et al., 2012).

Similar to a recent study (Suzuki et al., 2022), the present study did not find increased error-related delta in OCD (cf. Riesel et al., 2014). The contrast between the present findings and the work by Riesel et al. (2014) might lie in key methodological differences, for example, Riesel and colleagues extracted time-frequency measures at one scalp position in a priori time window, preventing the exploration of error-related effects across multiple locations and time points. Moreover, Riesel et al. (2014) assessed adults with OCD, while the present study and a recent investigation with similar results (Suzuki et al., 2022) focused on a pediatric sample; therefore, it could be that error-related delta dysfunctions in OCD develop later with increasing duration and chronicity.

Another aim of the present work was to examine whether leveraging a combination of time domain (i.e., ERN) and time-frequency measures would explain unique variance in OCD status. In a logistic regression, greater error-related beta emerged as a significant predictor of OCD over and above the ERN and theta. These analyses indicate that ERN and error-related theta reflect overlapping variance with error-related beta, and that once the shared variance among these measures is accounted for, only error-related beta predicts OCD. Given the relatively low sample size, future studies should further explore error-related beta as a correlate of OCD in youth.

On a methodological note, an advantage of the present investigation in comparison to previous studies is that we selected time-frequency measures (in both time and space) using a cluster-based analysis—which is an ad hoc data-driven method that avoids selection biases (e.g., selecting windows based on a priori intuition; Cohen, 2014; Luck, 2014).

The present study has limitations worth noting. First, the sample was homogeneous in ethnicity; consequently, future studies will need to determine whether the current results generalize to more diverse samples. Second, the present study had a relatively low sample size, and future studies with larger samples are required to confirm these results and better

clarify the time-frequency dynamics of error monitoring in OCD. In addition, epochs for the time-frequency decomposition began 500 ms before stimulus onset and this might have influenced the interpretation of 1-Hz delta power. Lastly, consistent with the U.S. NIMH's Research Domain Criteria (RDoC, Insel et al., 2010) framework, future studies should better examine associations between these neural measures and symptom dimensions (Riesel et al., 2014) to further examine whether error-related brain activity is associated with specific OCD symptom dimensions.

In conclusion, by examining separate time-frequency measures, the present study provided novel insights into the dynamics of error monitoring in OCD, suggesting that OCD may be characterized by enhanced error monitoring (i.e., greater theta power) and post-error inhibition (i.e., greater beta power). These results are promising as examining error-related theta and beta power could enhance clinical utility, both in terms of identification and for informing potential targets for treatment.

DATA AVAILABILITY STATEMENT

Data or other materials are available through correspondence with the first author.

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footnotes:

[1]The results of the statistical analyses did not differ based on the inclusion of these three participants taking psychotropic medications.

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Conflict of interest statement

The authors declare no competing interests.

Tables

Table 1. Demographic variables for the group with obsessive-compulsive disorder (OCD) and the healthy control group (HC).

	HC group (*n* = 27)	OCD group (*n* = 27)	*p*
Age	12.3 (1.10)	12.6 (1.03)	.31
Sex (% female)	51.85	55.56	.79
Ethnicity % of Caucasian participants	66.67	66.67	.77

Note. Age is expressed in mean (standard deviation).

Table 2. Descriptive statistics of behavioral and EEG measures for the group with obsessive-compulsive disorder (OCD) and the healthy control group (HC).

	HC group (*n* = 27)	**OCD group (*n* = 27)**
RTs correct (ms)	463 (78.3)	434 (60.5)
RTs error (ms)	348 (67.7)	328 (54.0)
RTs post-error (ms)	471 (84.8)	442 (70.0)
RTs post-correct (ms)	447 (76.3)	416 (59.1)
ERN at FCz (μV)	1.58 (8.15)	-0.36 (9.23)
CRN at FCz (μV)	5.30 (5.62)	7.00 (7.40)
Pe at Pz (μV)	16.2 (7.94)	15.8 (10.6)
Pc at Pz (μV)	-0.98 (4.81)	0.73 (4.98)
Theta error trials (between-groups cluster) (dB)	-0.17 (0.80)	0.38 (0.89)
Theta correct trials (between-groups cluster) (dB)	-0.73 (0.39)	-0.70 (0.37)
Beta error trials (between-groups cluster) (dB)	-0.39 (0.70)	0.38 (1.21)
Beta correct trials (between-groups cluster) (dB)	0.56 (0.62)	0.65 (0.53)
Time-frequency within-subjects clusters	**Error trials**	**Correct trials**
Delta (dB)	0.17 (0.34)	-0.15 (0.19)
Theta (dB)	-0.34 (0.76)	-0.86 (0.43)
Alpha (dB)	-0.57 (0.84)	0.05 (0.78)
Beta (dB)	0.09 (1.0)	0.66 (0.56)

Table 3. Bivariate and point-biserial correlations of EEG measures and behavioral data.

	Delta	Theta	Alpha	Beta	ERN	Pe	RTs correct	RTs error	RTs post-error	RTs post-correct	Group
Delta	-										
Theta	0.29*	-									
Alpha	0.24	0.22	-								
Beta	-0.17	0.05	0.46*	-							
ERN	-0.20	-0.42*	0.16	0.05	-						
Pe	-0.03	-0.04	-0.08	-0.25	0.12	-					
RTs correct	-0.08	-0.38*	0.05	0.23	0.37*	-0.05	-				
RTs error	-0.27	-0.26	0.31*	0.46*	0.44*	-0.21	-.73*	-			
RTs post-error	-0.09	0.39*	0.16	0.28*	0.34*	-0.08	0.92*	0.77*	-		
RTs post-correct	-0.11	-0.39*	0.04	0.26	0.35*	-0.09	0.99*	0.71*	0.90*	-	
Group	0.07	0.32*	0.20	0.36*	-0.27*	-0.10	-0.26	-0.19	-0.28*	-0.23	-

Note. All EEG measures are reported in residualized difference scores and reflect error-related activity. ERN = error-related negativity; Pe = error positivity; RTs = response times; Group, 0 = HC, 1 = OCD. *p <.05.

Table 4. Results of the logistic regression analysis predicting diagnostic status (OCD, HC) from the ERN and theta and beta power to error trials.

Measure	Prediction of diagnostic status (OCD, HC)				
	R^2	$\chi 2$	OR	95% CI_{OR}	*p*
Model 1					
	.19	7.90			
ERN_{resid}			.98	-.13-.10	.72
$Theta_{resid}$			2.74	.08-1.90	.03
Model 2					
	.41	18.1			
ERN_{resid}			0.96	0.84-1.09	.48
$Theta_{resid}$			2.40	0.91-6.31	.08
$Beta_{resid}$			4.83	1.38-16.83	.01

Note. Logistic regression was used to predict the dichotomous dependent variable diagnosis of OCD (0 = absent, 1 = present); The Nagelkerke R^2 and χ^2 statistics are reported for the logistic regression models. CI = confidence intervals; OR = odds ratio. *Model 1*. logistic regression predicting diagnostic status from the ERN and theta power; *Model 2*. logistic regression predicting diagnostic status from both time-frequency measures that emerged as significant from the cluster-based analyses (beta and theta power) and the ERN.

Figure captions

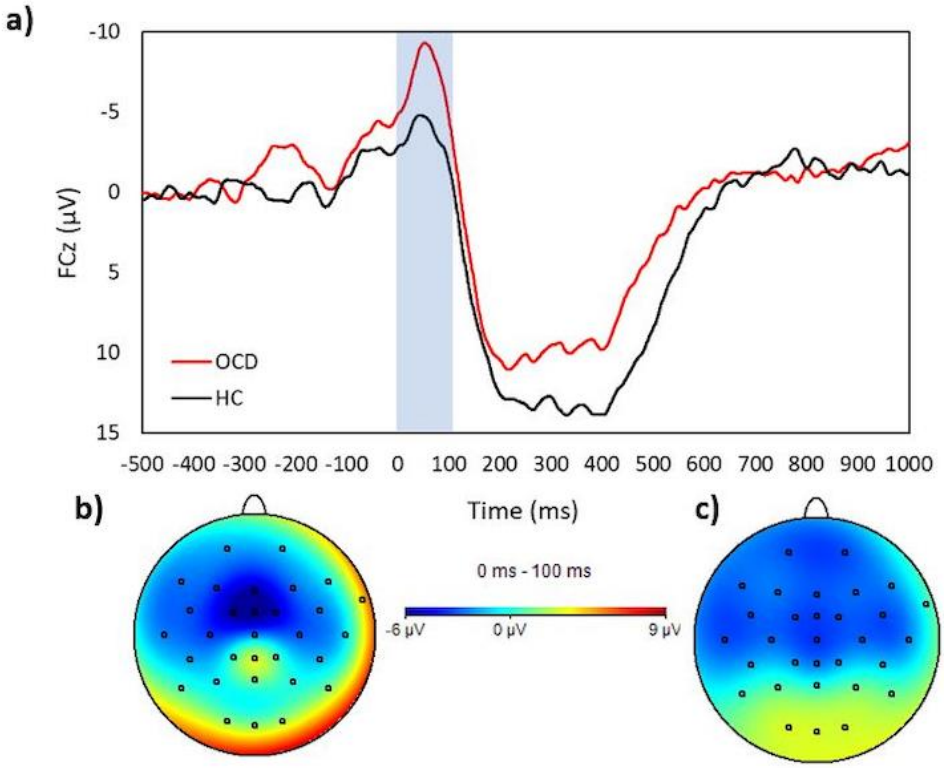

Figure 1. (Panel a) Response-locked event-related potential (ERP) waveforms for the difference between error and correct trials (ΔERN) in the OCD group (red line) and HC group (black line). (Panel b) Topographic map of activity (error minus correct) in the ERN time-window (i.e., 0-100 ms) in the OCD group. (Panel c) Topographic map of activity (error minus correct) in the ERN time-window (i.e., 0-100 ms) in the HC group.

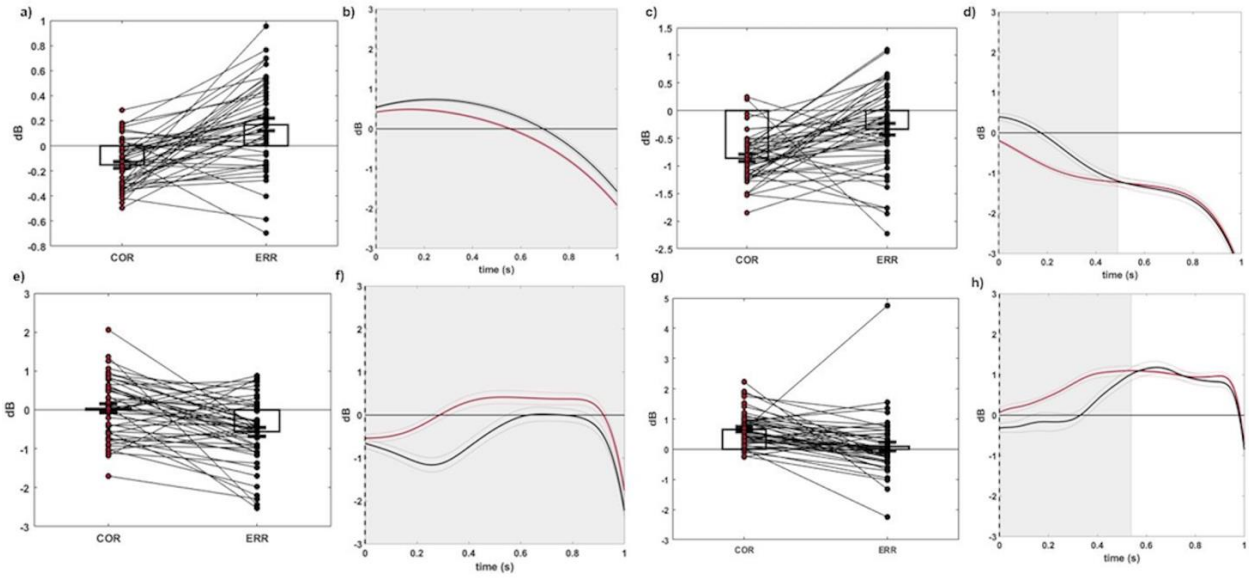

Figure 2. (Panel a, c, e, g) Mean event-related time-frequency power (a: delta; c: theta, e: alpha; g: beta) of each participant averaged over the significant electrodes and time points for correct and error trials. Each circle represents one participant. (Panel b, d, f, h) Time course of grand-average event-related time-frequency power (b: delta; d: theta, f: alpha; h: beta) of participants averaged over the marginally significant electrodes for correct (red line) and error (black line) trials. Shaded areas represent ± standard error of the mean (SEM) and the gray box represents the significant time window.

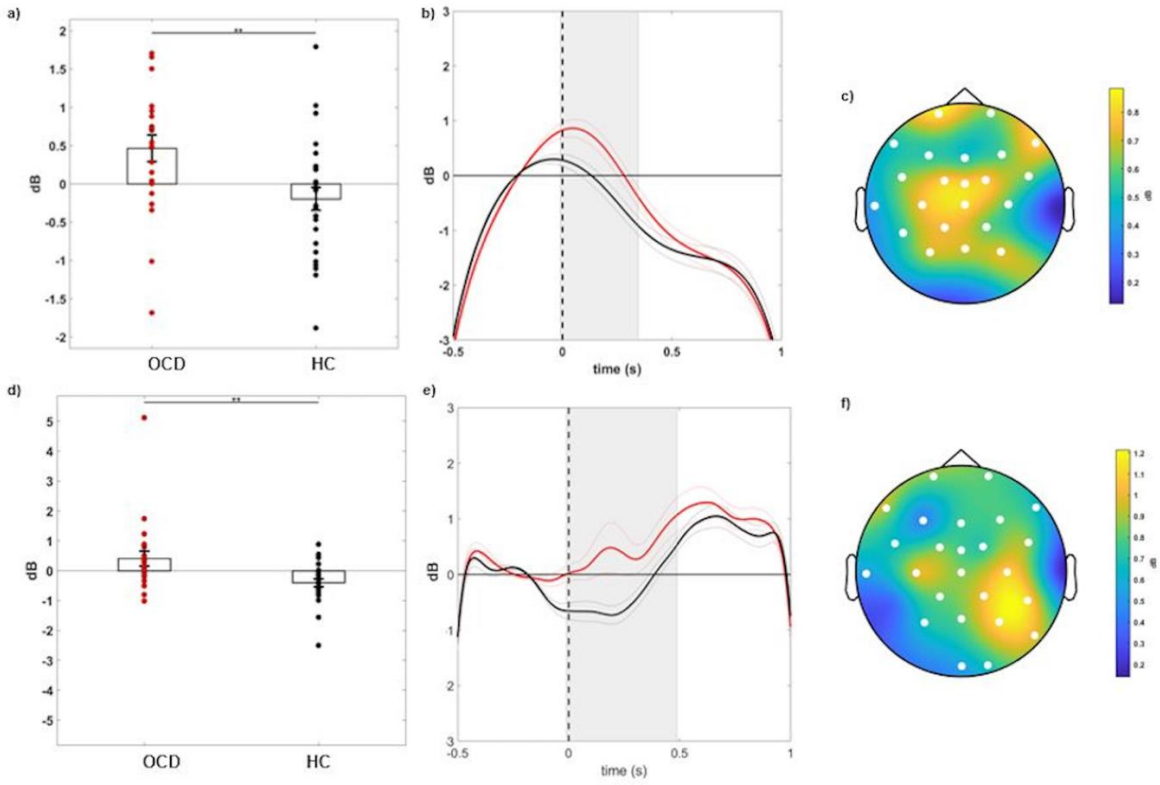

Figure 3. (Panel a and d) Mean event-related theta (panel a) and beta (panel d) power of each participant in the OCD group and the HC group averaged over the significant electrodes and time points for error trials. Each circle represents one participant (Panel b and e) Time course of grand-average event-related theta (panel b) and beta (panel e) power averaged over the significant electrodes for error trials in the OCD group (red line) and the HC group (black line). Shaded areas represent ± standard error of the mean (SEM); the gray box represents the significant time window. (Panel c and f) Topography of the mean difference between groups in event-related theta (panel c) and beta (panel f) power (dB; OCD group minus HC group) averaged over the significant time points for error trials. $^{**}p < .01$.