

Abnormally Enhanced Midfrontal Theta-Band Activity During Response Monitoring in Youth  
with Obsessive-Compulsive Disorder

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### Abstract

**Background:** Response monitoring, as reflected in the electroencephalogram (EEG) recording after commission of errors, has been consistently shown to be abnormally enhanced in individuals with obsessive-compulsive disorder (OCD). This has been traditionally quantified as error-related negativity (ERN) and may reflect abnormal neurophysiological mechanisms underlying OCD. However, the ERN reflects the increase in phase-locked activities, particularly in the theta-band (4-8 Hz), and does not reflect non-phase-locked activities. To more broadly interrogate midfrontal theta-band activity in a brain region essential for complex cognition, this study investigated theta-band abnormalities during response monitoring in OCD participants for a better understanding of the mechanism underlying the ERN. **Methods:** EEG data were recorded from 99 participants with pediatric OCD and 99 sex- and age-matched healthy control (HC) participants while they completed the arrow flanker task. Effects of Group (OCD, HC) and Response type (Error, Correct) on post-response theta-band total power and inter-trial phase coherence (ITPC) were examined using mixed analysis of covariance and Bayesian analyses controlling for sex and accuracy. **Results:** Theta-band total power was larger on Error than Correct trials and larger in OCD than HC participants, but there was no effect of response type between groups. Theta-band ITPC was larger on Error than Correct trials, but there were no group difference nor response type difference between groups. Correlations of theta-band total power and ITPC with clinical measures were overall small. **Conclusions:** Abnormally enhanced midfrontal theta-band total power, but not ITPC, may reflect ineffective heightened response monitoring or compensatory activity in pediatric OCD.

## Introduction

Obsessive-compulsive disorder (OCD) is characterized by recurrent intrusive thoughts (obsessions) and repetitive behaviors or mental acts (compulsions) (1). Its lifetime prevalence is estimated to be approximately 2%, and it often emerges in pre-pubertal children, with a median age of onset of 18 years old (2,3). Importantly, of individuals with OCD, approximately 88% experience impairments in role or social functioning, and 65% experience severe dysfunction (3). Randomized controlled studies have found that cognitive-behavioral therapy using exposure and response prevention and serotonin reuptake inhibitors (SSRIs) are effective treatments for pediatric OCD (4). However, some youth do not respond to either treatment, and a large proportion has clinically significant residual symptoms (5). Understanding the etiology and neural underpinnings of OCD is crucial for improving existing treatments, and in developing new treatments and preventative strategies for OCD.

The error-related negativity (ERN) is an electrophysiological marker consistently shown to be abnormally enhanced in individuals with OCD (6). The ERN is a response-locked event-related potential (ERP) characterized by a larger negative deflection in the electroencephalogram (EEG) recording after a commission of an error compared to making a correct response (7,8). This negative deflection is maximal in the midfrontal region, occurs within 100 ms after the response, and can be elicited by various speeded response tasks (9). Neuroimaging studies indicate that the ERN originates from the anterior cingulate cortex, posterior medial prefrontal cortex, and bilateral anterior insula (10–12). The ERN can be modulated by affective and motivational contextual manipulations, indicating the relevance of the system underlying the ERN in affective processes and psychopathology (13,14). Although the exact mechanism is still debated (15–17), evidence indicates that the ERN exhibits characteristics as a potential

endophenotype of OCD (18–20). The ERN is observed in children as young as three years old and amplitude increases stabilize around 18 years old (21,22).

The ERN has a rich literature in psychopathology research as well as in basic human research (23). Despite the robust findings of abnormally enhanced ERN in OCD, only a handful of studies has investigated ERN-analogs in animal models (24–26), which limits the translation of insights only possible in animal research (e.g., gene knockout) (27) to human research (28). This limitation of the ERN can be addressed by examining the time-frequency aspect of the EEG data, which reflects neural oscillatory activity (29). Research from animal models and computational modeling suggest that oscillatory activities are the building blocks of various brain activities, ranging from basic neuronal cell and synaptic level activities to distal brain region communications in complex cognitions (30). Therefore, understanding the oscillatory activities underlying the ERN can leverage and integrate findings from a broader range of basic research than is possible with the ERN, which in turn could inform the development of biologically-based interventions targeting specific neural mechanisms (28).

Three components of oscillatory activity can be extracted using time-frequency decomposition: Frequency, power, and phase (29). The frequency is the rate of the oscillation (per second) and ranges from delta ( $< 4$  Hz) to gamma waves ( $> 30$  Hz). Power reflects the magnitude of the oscillations and the level of increased or suppressed brain activities. Phase reflects the point in the cycle ( $0^\circ$  to  $360^\circ$ ) that the extracted frequency is at in a particular moment. The ERPs are the sum of these activities and could arise from (a) an increase in total power/amplitude, (b) an increase in inter-trial phase coherence (ITPC; phase resetting), or (c) increases in both (31). Further ERP reflects only the phase-locked power, as non-phase-locked power cancels each other out, while time-frequency decomposition allows the extraction of

power even if the phases are not aligned, thus providing information (total power) that is not available in ERP form.

Past empirical and simulation studies indicate that the ERN primarily reflects part of the change in theta-band (4-8Hz) activities (31,32). Specifically, the ERN appears to result primarily from theta-band total power increase and some increase in ITPC, but only reflects the phase-locked power. Midfrontal theta-band is of particular interest to psychopathology researchers, as it appears to reflect the cognitive control hub activity that regulates downstream affective and cognitive processes (33–35). A small number of recent studies have investigated the relevance of theta-band activities in OCD. For example, Peris and colleagues (36) found that post-stimulus theta-band power in arrow flanker task to be larger in pediatric OCD participants across all trials, suggesting abnormal conflict monitoring. Carmi and colleagues (37) investigated the perceived error-related theta activity in a mathematical task and found that increased response-locked theta-band ITPC, but not power, underlies the enhanced N1 in adult OCD participants. Riesel and colleagues (38) investigated oscillatory activities underlying the ERN in adults with OCD and found theta-band power to be enhanced. These findings suggest abnormal theta-band activity in OCD.

Despite the robust findings of the enhanced ERN in individuals with OCD and the likely theta-band activity underlying it, no research to date has investigated the response-monitoring theta-band activity abnormality in youth with early-onset OCD. This study investigated the theta-band total power and ITPC abnormalities in a large sample of youth with OCD compared to age- and sex-matched healthy control (HC) participants. The following hypotheses were made: (a) error responses have larger theta-band total power and ITPC than correct responses; (b) OCD participants have larger theta-band total power and ITPC than HC participants; and (c) OCD

participants have larger differences between error and correct responses in theta-band total power and ITPC than HC participants.

## **Methods and Materials**

### *Participants*

Participants were recruited from the University of Michigan Department of Psychiatry and the surrounding community. Written consent was obtained from the participant or one parent. All procedures and tasks were approved by the University of Michigan Medical School Institutional Review Board. The final sample consisted of 58% female, ranged in age from 8 to 19 years, and had an ethnic distribution of 92.4% White, 1% African American, 1% Asian, 5.5% two or more races, and 4% Hispanic.

All 99 OCD participants in the final analysis had a lifetime diagnosis of OCD with an age of onset ranging from 3 to 16 years. Current and lifetime Children's Yale-Brown Obsessive Compulsive Disorder Scale (CY-BOCS) (39) scores in the OCD participants ranged from 0 to 37 and 9 to 38, respectively. Of the OCD participants, 70 had a current diagnosis and 70 had a history of at least one other DSM-5 disorder. Patients were excluded if they had a lifetime diagnosis of autism spectrum disorder, schizophrenia, other psychotic disorder, bipolar disorder, substance-related disorder, or anorexia nervosa. All HC participants had no history of any specific DSM-5 disorder. Participants were excluded if they had a history of intellectual disability, head injury with a loss of consciousness, or chronic neurological disorder other than tics. All participants lived with at least one English-speaking biological parent to participate in research. Lifetime and current diagnoses were made independently by two clinicians using all sources of information according to *DSM-5* criteria (1).

### *Clinical and Behavioral Measures*

All participants were interviewed with the Schedule for Schizophrenia and Affective Disorders for School-Aged Children-Present and Lifetime Version (40) and Schedule for Obsessive-Compulsive and Other Behavioral Syndromes (41). Lifetime and current severity of OCD were assessed in patients with a modified version of the CY-BOCS, with patients and their parents providing item scores retrospectively for the most severe episode of OCD along with item scores for current severity. Participants completed the Tower of London (42) to assess planning deficits. Parents completed the Child Behavior Checklist (CBCL) (43,44) to assess behavioral and emotional problems.

#### *Modified arrow flanker task*

In this task (45), five arrows appeared on the center of a computer screen with four equiprobable conditions: congruent ( $\rightarrow\rightarrow\rightarrow\rightarrow\rightarrow$ ,  $\leftarrow\leftarrow\leftarrow\leftarrow\leftarrow$ ) and incongruent ( $\rightarrow\rightarrow\leftarrow\rightarrow\rightarrow$ ,  $\leftarrow\leftarrow\rightarrow\leftarrow\leftarrow$ ). Participants' task was to identify the direction of the arrow in the center and to press one of two keyboard buttons corresponding to the direction while ignoring the remaining arrows. Participants were seated approximately 0.65 meters from the computer monitor and were instructed to respond as quickly and accurately as possible. The stimuli remained on the screen for 250 ms with a response-stimulus interval of 1500ms. Participants completed a 32-trial practice block, then up to eight of 64-trial blocks (up to 512 trials). Feedback on performance was provided after every block to maintain error rates of approximately 10%. If there were fewer than four errors in a block, participants were encouraged to respond quicker, and if there were more than 10 errors, they were encouraged to respond more accurately.

#### *EEG Data Acquisition*

While participants completed the modified flanker task, EEG was recorded using the BioSemi Active 2 System with 64 Ag/AgCl electrodes, two mastoid electrodes, and two vertical

and two horizontal electro-oculogram electrodes. Data were collected at 512 Hz and online referenced to a ground using a common mode sense active electrode and a driven right leg passive electrode.

### *EEG Preprocessing*

Data from 109 participants who met criteria for OCD and 109 HC (no past or current psychiatric conditions) participants matched on sex and age were used. All preprocessing was conducted in MATLAB (version R2019b) (46) and EEGLAB (version 2019.1) (47) (See Supplemental Procedure for details). Preprocessing resulted in retaining 99 participants in each group.

### *Time-Frequency Decomposition*

All time-frequency decomposition was conducted in MATLAB using a customized script based on examples provided in Cohen (29). This study focused on the Cz electrode, which was found to be the maximal point for ERN in previous studies using this data, and because the purpose of this study was to investigate the underlying theta-band activities (50,51). Complex Morlet wavelet convolutions were created for frequencies ranging from 2 Hz to 80 Hz at 30 points on a logarithmically spaced scale with cycles ranging from 3 (at 2 Hz) to 10 cycles (at 80 Hz) on a logarithmically spaced scale. For total power, using the -3000 to 2500 ms epochs, mean power from pre-stimulus -300 to -50 ms window was used as the baseline (Supplemental Figure S1). Stimulus-locked baseline, rather than response-locked baseline, was used to avoid any confound that may be introduced by stimulus presentation and cognitive processes prior to response. A decibel (dB) conversion was then applied to the entire response-locked epoch, such that the activities at all frequencies and time points were quantified as relative to the baseline total power. All trials within each Response Type (Error, Correct) were averaged for each



participant, and difference scores at each frequency and time point were calculated to investigate the activities that differ specifically between the two Response Types by removing activities reflecting common processes (e.g., response preparation). Then, the post-response maximal decibel point was identified using a combined sample of OCD and HC participant data blind to group (89.84 ms and 7.14 Hz; Figure 1). Using this point as a reference, a rectangular window with a width of  $\pm 100$ ms and height of  $\pm 2$  Hz (but bounded to 4 Hz and 8 Hz to match traditional theta-band definition) was created. This resulted in a window of -10.16 to 189.84ms and 5.14 to 8 Hz that was used to extract the means of both theta-band total power and ITPC (See Supplemental Figure S2 for scalp distribution). ITPC values range from 0 (completely random phases across trials) to 1 (always the same phase across trials). This approach maximizes the power differences between Error and Correct responses, but remains orthogonal to OCD and HC participant comparison, which was the primary effect of interest.

#### *Event-Related Potential Extraction*

Using the exact same trials, ERPs corresponding to the ERN and correct-related negativity (CRN) were extracted to investigate the consistency of the results with the literature. To extract the ERPs using the same trials, data were re-referenced to TP7/TP8 electrode average and baseline corrected to pre-response -200 to -50 ms window. ERPs were defined as the average voltage between 0 to 80 ms post-response at Cz electrode (50,51). The difference between ERN and CRN ( $\Delta$ ERN) was calculated for each participant.

#### **Statistical Analyses**

All statistical analyses were conducted in R (Version 4.1.0) (52) using `psych` package (version 2.1.6) (53) for descriptive statistics and correlational analyses; `lmerTest` package (version 3.1-3) (54) for linear mixed analysis to conduct analysis of covariance (ANCOVA);

base stats package for chi-square, *t*-test, and ANCOVA information extraction, **R2jags** (version 0.6-1) (55) linked to JAGS (version 4.3.0) (56) and **lattice** (version 0.20-44) (57) for hierarchical Bayesian analysis; and **ggplot2** for plotting (version 3.3.5) (58). First, chi-square test and *t*-tests were conducted to ensure matching of sex and age in the final sample. Second, *t*-tests comparing RT and accuracy, common covariates used in error-related EEG studies, between groups were conducted (50). Third, ANCOVAs, including age and accuracy as covariates, were conducted to test the main and interaction effects of Group (OCD vs. HC) and Response Type (Error vs. Correct) on theta-band total power and ITPC. Fourth, Bayesian analysis analogues of the ANCOVAs using uninformative priors were also conducted to provide more context to the ANCOVA results (See Supplemental Script for a sample R script). The same set of analyses was conducted for ERPs.

Two sensitivity analyses investigating the effects of trial selections and an analysis on delta-band activities were conducted using the same procedure (Supplemental Analyses). As exploratory analyses to investigate the external relevance of total power, ITPC, and ERPs, correlations to clinical and behavioral measures (CBCL and ToL) were conducted. Equations used to calculate post-error behavioral measures are presented in Supplemental Table S6. Further, differences in EEG indicators in OCD participants with present compared to past OCD (51) and those with current compared to no current SSRI treatment were investigated. Cohen's guidelines (59) were used when interpreting the effect sizes (e.g., for *d*, .20 = small and .50 = medium effects; for *r*, .10 = small and .30 = medium effects).

## Results

All descriptive statistics are presented in Tables 1 and 2, and ANCOVA and Bayesian analysis results are presented in Supplemental Table S1. Figurative representations of the time-

frequency decomposition for total power and ITPC are presented in Figures 2 and 3, respectively. Testing of the matching variables indicated successful matching ( $p > .05$  for both age and sex). Overall RT was not different across the two groups ( $p > .05$ ), but HC (90.5%) were more accurate than OCD participants (88.9%;  $p = .037$ ). ANCOVAs on theta-band total power indicated main effects of both Group (larger in OCD) and Response Type (larger in Error, as expected), but no Group  $\times$  Response interaction. Of the covariates, older age was related to larger total power, but accuracy was not statistically significantly related to total power. ANCOVAs on ITPC indicated main effect of Response Type (larger in Error), but no main effect of Group or Group  $\times$  Response interaction. Older age and higher accuracy (both covariates) were associated with larger ITPC. Similar results were obtained with ANCOVAs on ERPs (Figure 4): there were main effects of both Group (larger in OCD) and Response Type (larger in Error), but no Group  $\times$  Response interaction, although a Group  $\times$  Response interaction was found when all correct trials were included (Supplemental Results, Table S2). Older age and higher accuracy (both covariates) were related to larger ERP amplitude. Hierarchical Bayesian analyses concurred with these results. Particularly, there was a 98.3% posterior probability that OCD participants had larger theta-band total power than HC participants and 44.3% posterior probability that OCD participants had larger theta-band ITPC than HC participants.

Correlations of the EEG measures (power, ITPC, and ERP) with each other are presented in Supplemental Table S5. The theta-band power ( $r = .62$ ) and ERP ( $r = .63$ ) correlations between the Error and Correct Responses were large while correlations of ITPC ( $r = .22$ ) between the Error and Correct Responses were small. Overall, EEG measures from Error Responses had medium to large correlations with each other ( $|r|$  ranging from .43 to .55), while EEG measures from Correct responses had small correlations with each other ( $|r|$  ranging from

.15 to .25). Correlations of the EEG measures with clinical and behavioral measures (CBCL and ToL) are presented in Supplemental Table S7. Given the exploratory nature and the number of correlations, statistical significance was not interpreted. Nonetheless, this table shows that the correlations of the EEG with clinical and behavioral measures are small ( $|r|$  less than .30). Comparison of conditions within OCD participants is presented in Supplemental Table S8. ANCOVAs in OCD participants indicated that participants with current and past OCD diagnoses were comparable in power, ITPC, and ERPs. SSRI usage had no main effect, but had an interaction effect on power, such that the difference between Error and Correct Power was smaller in those with current SSRI treatment. SSRI treatment had a main effect on ERP amplitudes, such that they were more negative. No SSRI effect was found for ITPC.

### **Discussion**

To our knowledge, this was the first study investigating the response monitoring theta-band activity abnormality in youth with early-onset OCD. Across OCD and age- and sex-matched HC participants, both theta-band total power and ITPC were larger in Error than Correct Responses, consistent with previous studies finding that theta-band activities underlie the ERN (31,32). Effect sizes indicated a large effect of power and a small effect of ITPC, also consistent with previous research indicating a larger role of power than ITPC in underlying the ERN (31). In addition to these aggregate response effects in all participants (within-subject effects), correlation of EEG measures with each other also indicated stronger relation of individual differences in ERPs to power than to ITPC (between-subject effects). Overall, these results indicate the stronger relevance of theta-band power than ITPC to response monitoring ERP.

Across response types, OCD participants had larger theta-band total power compared to HC participants, but ITPC was comparable across the two groups. This lack of ITPC difference

between the two groups (between-subject effect) is despite the finding that both power and ITPC are larger in Error responses (within-subject effect). These findings together indicate that abnormal ERN amplitudes consistently observed in OCD are only due to part of the mechanism (only power) that generates the ERN (both power and ITPC). Differential abnormalities in theta power and ITPC may elucidate the shared and unique underlying mechanisms across forms of psychopathology. For example, Groom and colleagues (60) found theta-band ITPC, but not power, from a Go/No-Go task was reduced in adolescents with attention-deficit/hyperactivity disorder. This difference likely indicates different mechanisms underlie abnormalities in response monitoring in OCD and ADHD. While the exact implications of the differences in power and ITPC are still not well known, abnormal ERN activity has been implicated in several forms of psychopathology and multiple domains of the National Institute of Mental Health Research Domain Criteria (RDoC) (15,61,62). For example, the ERN is implicated in Sustained Threat and Performance Monitoring, but power may be more relevant in managing threatening information (e.g., making an error) while phase may be more relevant in monitoring to ensure correct responses (63). Further characterization of neural oscillatory activities underlying different forms of psychopathology and RDoC domains could inform mechanisms and treatment.

The response effect (i.e., differences between Error and Correct Responses) in power, ITPC, and ERP (i.e.,  $\Delta$ ERN) were comparable across OCD and HC participants (i.e., no response type by group interaction), except for when all correct trials after preprocessing were included (i.e., no matching with error trials). This general lack of interaction effect is contrary to a previous study finding theta-band power response type by group interaction (38; they did not investigate ITPC) and with some studies finding larger  $\Delta$ ERN amplitudes in OCD participants (6,64). When all correct trials were used, an interaction effect on the ERPs was observed, though

there was still no interaction effect for power or ITPC. This likely reflects the higher reliability of the CRN from using more trials. Since there was an age effect and the ERN is known to develop into adulthood (21,22), another possibility is that the interaction effect may not be (as clearly) evident in youth with early-onset OCD, a group that has a higher genetic influence than individuals with later onset OCD (65,66). Time-frequency analyses of previously published data, particularly with both children and adults, would provide a more definite conclusion on whether only altered power underlies the ERN abnormality in OCD. Nonetheless, this study suggested that patients with OCD exhibit overall heightened response monitoring compared to HC participants. Since OCD participants with current and past diagnoses had comparable EEG measures, the findings provide further evidence for these indicators as possible endophenotypes (19,51). Further, oscillation phase is a key element of coordination of intra- and inter-region brain communications (30). Since behavioral performance was broadly comparable between OCD and HC participants, with lower accuracy in the OCD group, the increased theta power but lack of ITPC increase in OCD participants, might indicate inefficient response monitoring or increased effort not resulting in improved performance. This inefficient effort, in turn, could reflect the difficulty in deciding when to check (e.g., realistic concerns, when an error is made) and when not to check (e.g., non-realistic concerns, when a response is completed correctly) that may be unique to OCD compared to anxiety disorders (1,19). This could underlie, for example, the urge to check even when patients with OCD *know* they locked the door (see below for interpretation as compensatory mechanism).

Correlational results were mostly consistent with previous research. Error theta-band power and ERN were related more strongly than correct theta-band power of correct and CRN (67,68). These likely reflect more consistent individual differences in engagement of response

monitoring during error than correct trials, providing support for the practice of focusing on the ERN in psychopathology research. The correlations between EEG measures (power, ITPC, ERPs) and clinical measures were small, at best, which are not surprising and are consistent with previous research. For example, Riesel and colleagues (19) found that ERN/CRN amplitudes or their changes were associated neither with OCD symptoms nor treatment response. This is also consistent more broadly with research on other psychological constructs (e.g., risk-taking) finding generally small cross-methods effect sizes (e.g., functional imaging, questionnaires) (69–71). One explanation is that the relations between EEG measures and symptoms are more complex than the linear statistical relations often tested (72,73). It is also possible that abnormally increased ERN and theta-band power in OCD reflects compensatory mechanisms rather than symptom expression (64). For example, Carmi and colleagues found that *increase* in ERN amplitude from deep transcranial magnetic stimulation was associated with *improved* OCD symptoms, suggesting enhanced ERN may reflect adaptive attempts to manage behaviors and symptoms (74). Future research using brain stimulation methods experimentally modulating theta-band activity (e.g., theta burst stimulation (75)) examining their effects on symptoms and functioning could elucidate the role of theta-band activity in OCD.

Some limitations of this study need to be noted. First, the sample was racially homogenous with more than 92% of the participants identifying as White. Future studies using diverse samples are essential to determine the generalizability of the present findings. Second, medication was not controlled, but was associated with some EEG measures. While some studies found unaffected family members of participants with OCD showing enhanced ERN (76), suggesting a genetic predisposition, some experimental studies have found SSRI to increase

ERN and CRN (77,78). Further investigation of the effects of SSRI is needed to elucidate its role in response monitoring EEG measures.

This study provided further evidence that increased theta-band total power and ITPC underlie the ERN. However, only abnormality in theta-band power, and not ITPC, underlies the abnormally enhanced ERN in pediatric OCD participants. Oscillatory activities also underlie other ERP components (e.g., N2, P300) that have been associated with psychopathology (63,79). Time-frequency analysis could shed light on the mechanisms giving rise to different observed ERP abnormalities and inform the pathophysiology of some forms of psychopathology. For example, increased theta-band power appears to underlie both response monitoring (ERN) and conflict monitoring (N2) (36) abnormalities in OCD patients, and thus may be a biomarker and neural target for OCD (80). Elucidating the neural oscillatory characteristics underlying multiple ERP abnormalities observed in psychopathology has great potential to provide a physiologically plausible and parsimonious framework to uncover etiology and guide treatment research.



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### **Disclosure**

The authors reported no biomedical financial interests or potential conflicts of interest

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Table 1. Descriptive statistics of demographic, clinical measures, and cognitive data in participants with obsessive-compulsive disorder (OCD) and healthy controls (HC).

Variable	OCD					HC						
	N	Mean	SD	Min	Max	N	Mean	SD	Min	Max	<i>t</i>	<i>p</i>
<b>Demographics</b>												
Sex (% Female)	99	60.6				99	54.6					
Age (year)	99	13.8	3.0	8.1	18.8	99	13.7	3.1	8.1	19.3	0.15	0.881
<b>OCD Symptoms</b>												
Lifetime CY-BOCS total score	99	26.5	7.2	9.0	38.0							
Present CY-BOCS total score	99	16.2	9.0	0.0	37.0							
Age at onset of OCD symptoms	99	7.9	3.3	3.0	16.0							
Current diagnosis of OCD (%)	99	70.7										
<b>Child Behavior Checklist (CBCL)</b>												
Total score	98	38.1	23.9	3.0	95.0	97	9.7	9.5	0.0	51.0	10.92	<0.001
Internalizing score	98	14.7	9.3	0.0	41.0	97	3.2	3.3	0.0	15.0	11.59	<0.001
Externalizing score	98	7.2	7.2	0.0	32.0	97	2.7	3.8	0.0	18.0	5.46	<0.001
Obsessive-Compulsive scale	98	6.3	3.6	0.0	15.0	97	0.9	1.0	0.0	4.0	14.06	<0.001
Anxious/Depressed scale	98	8.3	5.3	0.0	20.0	97	1.6	1.9	0.0	9.0	11.74	<0.001
Withdrawn/Depressed scale	98	3.0	2.5	0.0	12.0	97	0.6	1.0	0.0	4.0	8.53	<0.001
Somatic Complaints scale	98	3.5	3.4	0.0	18.0	97	0.9	1.6	0.0	8.0	6.79	<0.001
Social Problems scale	98	3.1	3.2	0.0	17.0	97	0.7	1.2	0.0	6.0	6.92	<0.001
Thought Problems scale	98	5.5	3.6	0.0	15.0	97	0.6	0.9	0.0	4.0	13.25	<0.001
Attention Problems scale	98	4.4	4.3	0.0	16.0	97	1.3	1.9	0.0	8.0	6.41	<0.001
Rule-Breaking behavior scale	98	1.6	2.3	0.0	13.0	97	0.8	1.4	0.0	6.0	2.89	<0.001
Aggressive Behavior scale	98	5.6	5.8	0.0	25.0	97	1.8	2.7	0.0	13.0	5.86	<0.001
<b>Tower of London</b>												

Total move score	59	33.8	14.7	4.0	71.0	70	28.1	13.7	4.0	64.0	2.27	0.025
Total correct score	59	3.5	1.8	0.0	9.0	70	4.1	1.9	0.0	8.0	-2.15	0.034
Total rule violation score	59	0.1	0.4	0.0	2.0	70	0.1	0.4	0.0	2.0	0.07	0.947

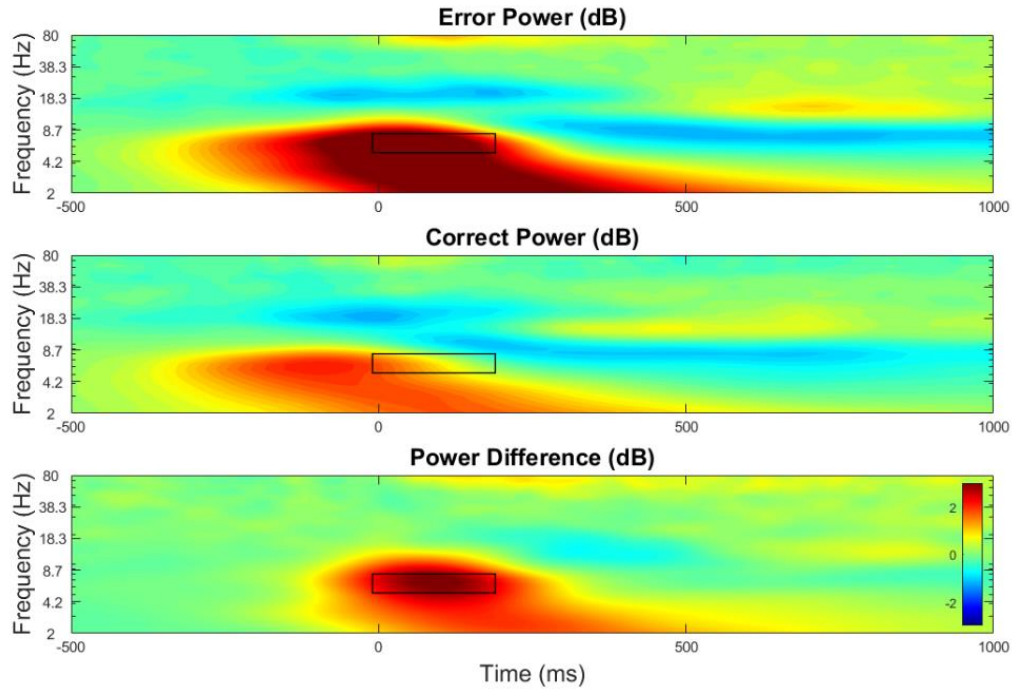
Notes. N = Sample size; SD = Standard deviation; Min = Minimum value; Max = Maximum value; CY-BOCS = Children's Yale-Brown Obsessive-Compulsive Scale.

Table 2. *Descriptive statistics of electroencephalogram indicators and behavioral data in obsessive-compulsive disorder (OCD) and healthy control (HC) participants.*

Variable	OCD					HC					<i>t</i>	<i>p</i>
	N	Mean	SD	Min	Max	N	Mean	SD	Min	Max		
Power (dB)												
Error trials	99	3.79	2.43	-2.62	9.39	99	3.18	2.27	-2.19	8.97	1.82	.070
Correct trials	99	0.99	1.45	-2.38	4.52	99	0.65	1.43	-2.21	7.11	1.69	.093
ITPC												
Error trials	99	0.32	0.14	0.09	0.72	99	0.32	0.12	0.07	0.64	0.22	.827
Correct trials	99	0.27	0.10	0.10	0.69	99	0.28	0.12	0.06	0.67	-1.24	.218
ERP ( $\mu$ V)												
ERN	99	-2.58	5.81	-18.65	14.82	99	0.05	6.01	-14.93	13.05	-3.13	.002
CRN	99	0.46	4.52	-10.88	9.91	99	2.22	4.80	-8.61	15.45	-2.67	.008
Accuracy (%)												
Overall	99	88.9	6.1	65.6	97.5	99	90.5	4.8	70.4	97.7	-2.11	.037
Congruent trials	99	95.9	3.8	78.1	100.0	99	96.6	3.4	79.7	100.0	-1.45	.150
Incongruent trials	99	81.9	9.8	51.2	96.5	99	84.4	7.3	60.9	95.7	-2.05	.041
Post-error improvement	99	0.4	3.6	-19.6	29.6	99	0.8	2.2	-7.4	7.1	-0.88	.381
Error trials used in analyses (not %)	99	36.7	22.7	8	123	99	33.1	16.5	8	97	1.27	.206
Reaction time (ms)												
Overall	99	519.0	172.7	351.4	1172.7	99	503.0	124.3	363.6	1189.4	0.75	.456
Congruent trials	99	489.3	160.5	335.4	1125.9	99	472.7	107.7	344.4	955.9	0.86	.393
Incongruent trials	99	548.9	186.6	358.9	1278.8	99	533.4	144.2	380.8	1429.1	0.65	.515
Correct trials	99	520.9	168.0	353.8	1161.6	99	506.0	122.8	364.9	1162.4	0.71	.478
Error trials	99	501.3	237.4	328.3	1554.4	99	475.8	159.8	340.2	1482.5	0.88	.378
Post-Correct Correct Trials	99	520.2	167.2	352.5	1159.9	99	505.5	122.6	363.8	1153.8	0.70	.482
Post-Correct Error Trials	99	501.2	238.9	328.0	1559.2	99	474.5	159.4	340.2	1482.5	0.93	.356
Post-Error Correct Trials	99	532.9	182.3	359.4	1277.8	99	512.2	121.6	370.4	1064.6	0.94	.349

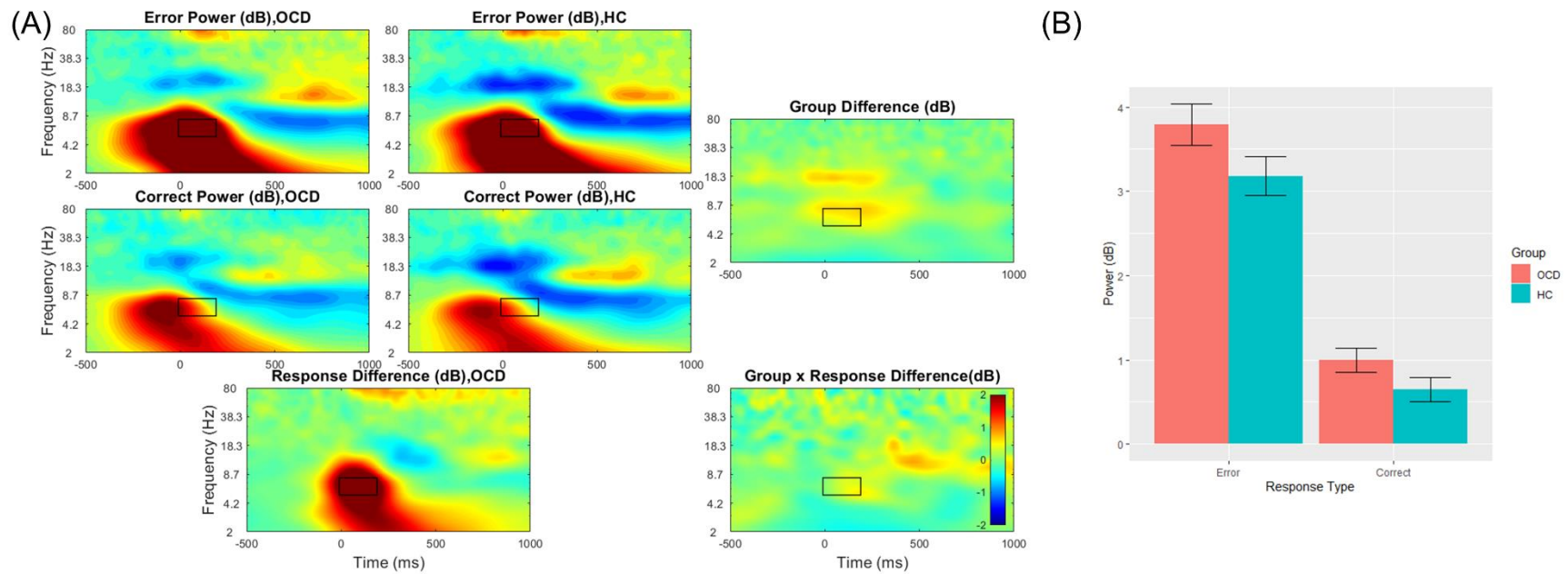
Post-Error Slowing, Traditional	99	12.7	52.5	-202.1	317.8	99	6.7	43.1	-101.2	239.7	0.88	.379
Post-Error Slowing, Robust	99	24.0	47.2	-133.6	242.6	99	24.2	43.5	-143.4	231.8	-0.03	.973
Post-Error reduction in interference	99	15.9	117.5	-225.1	949.8	99	-4.1	89.0	-444.8	256.0	1.35	.178

Notes. N = Sample size; SD = Standard deviation; Min = Minimum value; Max = Maximum value; dB = decibel; ITPC = Inter-trial phase coherence; ERP = Event-related potential;  $\mu$ V = microvolts; ERN = Error-related negativity; CRN = Correct-related negativity; ms = Milliseconds.

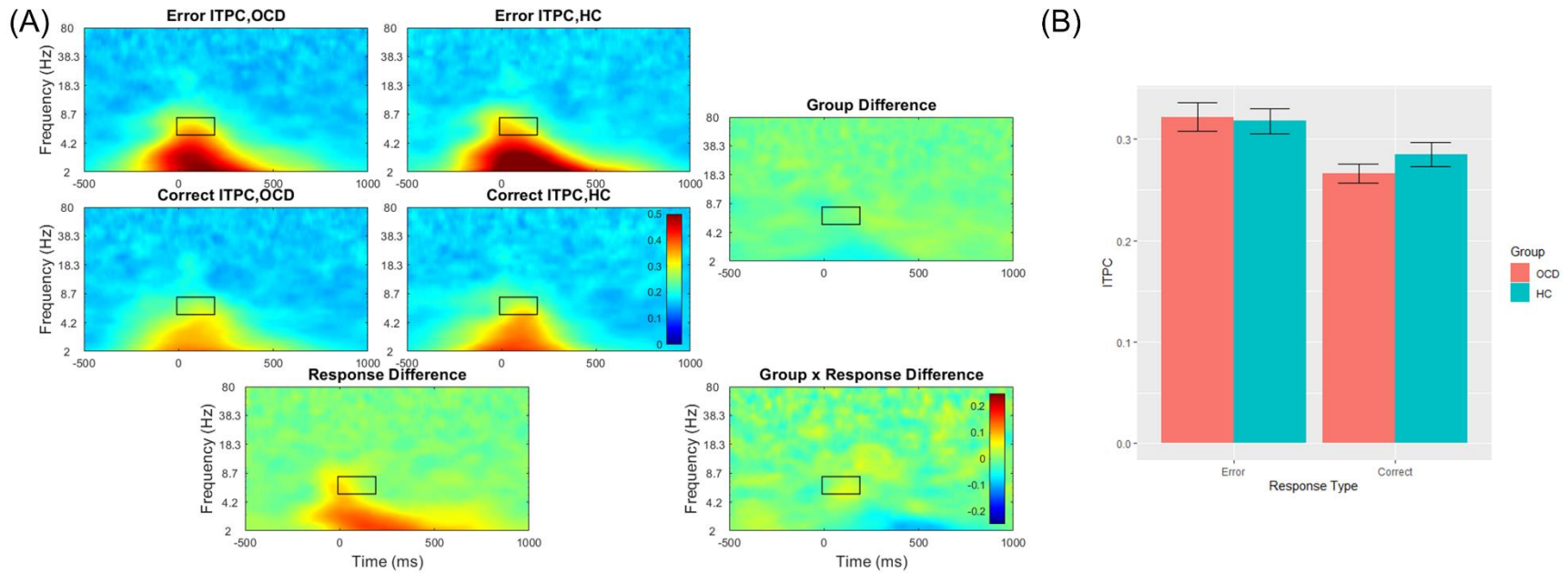


*Figure 1.* Time-frequency decomposition results on total power of Error responses (top), Correct responses (center), and Error minus Correct (bottom) averaged across all participants. Y-axis reflects frequency in Hz (ranging from 2 to 80; logarithmically spaced) and x-axis reflects time in milliseconds (ms; ranging from -500 to 1000ms relative to response). The mean activity in the boxed area was used as theta-band activity data. All figures are on the same scale ( $\pm 3$  dB), with darker red reflecting more positive values and darker blue reflecting more negative values.



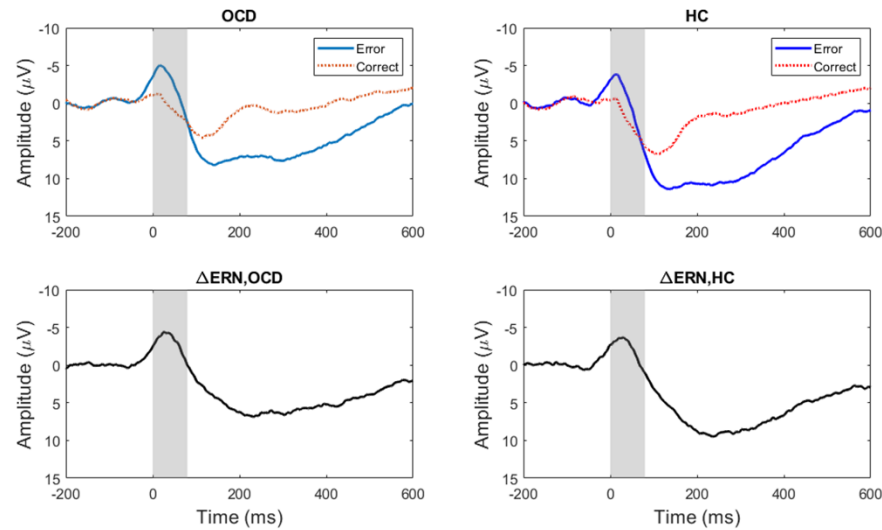


*Figure 2.* (A) Power of Error and Correct responses from obsessive-compulsive disorder (OCD; left top and left middle, respectively) and healthy control (HC) participants (center top and center middle respectively). Also, within-participant differences (bottom left; essentially Figure 1; reflects main effect of responses), between-participant differences (top right; reflects main effect of group differences), and group differences in response differences (bottom right; reflects interaction effect). Y-axis reflects frequency in Hz (ranging from 2 to 80; logarithmically spaced), and x-axis reflects time in milliseconds (ms; ranging from -500 to 1000ms relative to response). All figures are on the same scale ( $\pm 2$  dB), with darker red reflecting more positive values and darker blue reflecting more negative values. The boxed area reflects the area used to extract theta-band activity used in the analyses. (B) Bar chart showing mean theta-band power values for each response type and group with  $\pm 1$  standard error bars. OCD group is in red and HC group is in blue.

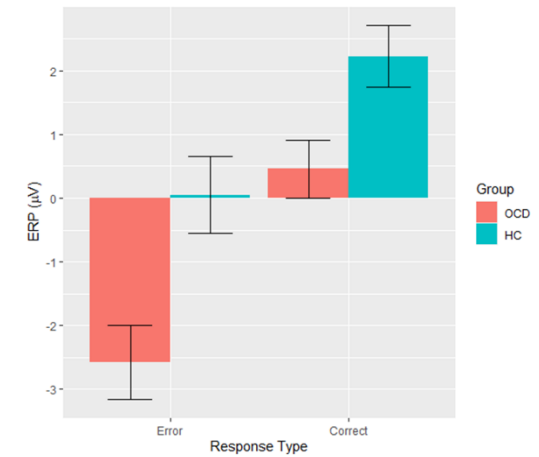


*Figure 3.* (A) Inter-trial phase coherence of Error and Correct responses from children with obsessive-compulsive disorder (OCD; left top and left middle, respectively) and healthy control (HC) participants (center top and center middle respectively). These four figures are on the same scale. Also, within-participant differences (bottom left; essentially Figure 1; reflects main effect of responses), between-participant differences (top right; reflects main effect of group differences), and group differences in response differences (bottom right; reflects interaction effect). Y-axis reflects frequency in Hz (ranging from 2 to 80; logarithmically spaced), and x-axis reflects time in milliseconds (ms; ranging from -500 to 1000ms relative to response). The differences figures are on a narrower scale ( $\pm 0.25$  instead of 0 to .50) to make the differences starker. Darker red reflect more positive values and darker blue reflect more negative or smaller values. The boxed area reflects the area used to extract theta-band activity used in the analyses. (B) Bar chart showing mean theta-band ITPC values for each response type and group with  $\pm 1$  standard error bars. OCD group is in red and HC group is in blue.

(A)



(B)



*Figure 4.* (A) The error-related negativity (ERN; navy blue and blue lines of top figures) and correct-related negativity (orange and red lines of bottom figures) for obsessive-compulsive disorder (OCD; left) and healthy control (HC; right) participants. Error minus correct ( $\Delta$ ERN) for OCD (bottom left) and HC participants (bottom right). Y-axis reflects ERP amplitudes (in microvolts), and X-axis reflects time in milliseconds (ms; ranging from -200 to 600ms relative to response). Shaded areas indicate the 0 to 80 ms post-response time window that the ERPs were extracted from. (B) Bar chart showing mean ERP values for each response type and group with +/- 1 standard error bars. OCD group is in red and HC group is in blue.