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Divergent Associations of Anxiety and Comorbid Depression with Midfrontal Theta in a Large Transdiagnostic Sample

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Running Title: *Midfrontal Theta in the Obsessive-Compulsive and Anxiety Spectrum*

Keywords: anxiety; depression; transdiagnostic; midfrontal theta; performance monitoring; cognitive control

Abstract

Background: Error monitoring is reliably elevated in obsessive-compulsive and anxiety spectrum (OCA) disorders, yet the transdiagnostic neural mechanisms remain unclear. We investigated whether response-related midfrontal theta oscillations (4–7 Hz) could serve as a transdiagnostic marker of altered cognitive control across OCA disorders, and whether comorbid depression modulates this effect. We further examined associations between dimensional measures of obsessive-compulsive, worry, and depressive symptoms and theta alterations.

Methods: EEG was recorded from 310 adults (153 patients with OCA disorders without comorbid depression, 60 with comorbid depression; 97 healthy controls) during a speeded arrowhead flanker task. We compared midfrontal theta power (0–200 ms post-response) and behavioral performance-monitoring indices (error rate, post-error slowing) across groups and examined relationships with symptom dimensions.

Results: Relative to controls, patients with OCA disorders without comorbid depression exhibited elevated theta power following both errors and correct responses. Comorbid depression blunted this effect. Symptom dimensions were not significantly associated with theta. However, by combining symptom dimensions (obsessive-compulsive symptoms and worry) and neurobehavioral measures (error rate, post-error slowing, prefrontal theta coherence), we could predict changes in behavioral performance monitoring indices.

Conclusions: Elevated response-related theta power represents a potential transdiagnostic neural marker of OCA disorders, consistent with prior error-related negativity findings. Since theta was enhanced after both correct and incorrect responses, our results extend previous accounts of altered error processing toward generalized cognitive control hyperactivity. Comorbid depression may attenuate this neural signature, highlighting the importance of psychiatric comorbidity in transdiagnostic neurobiological research.

1. Introduction

For about three decades, researchers have intensively investigated the error-related negativity (ERN) in human electroencephalography (EEG) as an indicator of cognitive control (Gehring et al., 2018). Clinical studies not only found strong associations of elevated ERN amplitudes with obsessive-compulsive disorder (OCD) and related symptoms (Mathews et al., 2012; Riesel, 2019), but also with anxiety disorders and traits, such as social anxiety disorders (Endrass et al., 2014; Kujawa et al., 2016), generalized anxiety disorder (GAD; Xie et al., 2025), and anxious apprehension (Moser et al., 2013; Saunders & Inzlicht, 2020). Regarding the relation of major depressive disorder (MDD) and the ERN, findings are inconsistent, with some reporting an increased ERN (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008, 2010), a reduced ERN (Ladouceur et al., 2012; Weinberg et al., 2012), or alterations depending on symptom severity (Olvet et al., 2010). ERN reduction has been attributed particularly to anhedonia within MDD (Weinberg et al., 2016). However, meta-analyses suggest either publication bias (Moran et al., 2017) or null findings (Lu et al., 2025; Pasion & Barbosa, 2019; Riesel et al., 2025), with substantial inconsistencies that may mirror the heterogeneous symptomatology of MDD. Consequently, an altered ERN amplitude has been proposed as a transdiagnostic risk marker cutting across various diagnostic entities within the anxiety and obsessive-compulsive spectrum (Härpfer & Riesel, 2026).

In recent years, oscillatory, error-related theta activity (4–7 Hz) has also attracted increasing attention in the research field as it shares the same cortical origin, i.e., the anterior cingulate cortex (ACC), and bursts within a comparable time window (0–200 ms) as the ERN (0–100 ms) following error commission (Luu et al., 2004; Trujillo & Allen, 2007). Consistent with the ERN, midfrontal, error-related theta activity is thought to reflect a core neural mechanism of cognitive control (Cavanagh & Frank, 2014; Meyer & Hajcak, 2019). Focusing on oscillatory activity, such as theta, offers a major advantage over ERPs: it provides additional insight into how brain regions communicate and, in the case of theta, how cognitive control is implemented. Error-related theta has been shown to coordinate communication between the ACC and lateral prefrontal regions, enabling adaptive behavioral adjustment (Cavanagh & Frank, 2014; Pagnotta et al., 2024). Based on these insights, theta coherence quantifies this inter-regional communication by capturing the degree of phase synchronization between distinct cortical areas, complementing theta power as an index of prefrontal network coordination (Cavanagh et al., 2009; Lachaux et al., 1999). Altogether, error-related theta appears to represent a promising, clinically useful marker (McLoughlin et al., 2022). The so-far unresolved question is whether error-related theta power is increased across different disorders from the obsessive-compulsive and anxiety spectrum (OCA) to underscore its potential as a transdiagnostic marker of anxiety (Cavanagh & Shackman, 2015).

Initial evidence for a transdiagnostic anxiety account of theta mainly stems from primary studies on patients with OCD and GAD. Several studies have shown that OCD is accompanied by increased error-related theta power (Dell'Acqua et al., 2023a; Riesel et al., 2014; Suzuki et al., 2023) or also by heightened theta activation during rest (Lee et al., 2023; Perera et al., 2023), assumed to reflect error-related and broader trait-like ACC hyperactivation. Findings regarding GAD are mixed: Cavanagh et al. (2017) reported increased error-related theta power, whereas Muir et al. (2020) found no significant associations. Beyond anxiety, MDD represents a

clinically relevant factor concerning theta, as it appears to blunt rather than enhance error-related theta activity (Dell'Acqua et al., 2023b), similar to some aforementioned ERN findings (Ladouceur et al., 2012; Weinberg et al., 2012). Taken together, initial studies support that OCA disorders appear to be associated with increased error-related midfrontal theta activity, accompanied by a potential attenuating role of comorbid depression. So far, prior work has largely relied on categorical diagnoses and single-disorder samples, limiting insights into shared vs. specific dimensional mechanisms across OCA disorders. Furthermore, neural indices of cognitive control have rarely been examined alongside behavioral correlates, such as post-error slowing (PES), an adaptive behavioral adjustment following errors (Danielmeier & Ullsperger, 2011; Rabbitt, 1966).

Consistent with the Research Domain Criteria (RDoC; Insel et al., 2010) and the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017), the present study adopts a transdiagnostic approach to examine response-related theta activity and behavioral indices of adaptive control. By combining dimensional and categorical, diagnosis-based perspectives, we focus on obsessive-compulsive, worry, and depressive symptom dimensions and complement these by grouping OCA disorders and MDD as internalizing disorders (Kotov et al., 2017) and testing the role of comorbid depression within this group. We expect (1a) increased error-related theta power in the combined group of patients with internalizing disorders compared to healthy controls. Exploratorily, we further investigate (1b) the modulating role of comorbid depression and whether the group of patients with OCA disorders without comorbid depression exhibits, contrary to the OCA with comorbid depression group, a significant increase in error-related theta power. At the dimensional level, we expect that (2a) greater obsessive-compulsive and worry symptoms are associated with an increase, and that (2b) more severe depressive symptoms are linked to a decrease in error-related theta power. In addition, we hypothesize that across all participants, behavioral indices of adaptive control (error rate, PES) are associated with obsessive-compulsive, worry, or depressive symptom dimensions; error-related theta power or coherence; or the respective other behavioral measure. We expect to find (3a) that higher symptom severity across the three dimensions and (3b) that increases in neural indices of cognitive control, i.e., theta power or coherence, are linked to improved accuracy and prolonged PES.

2. Results

Demographic and Clinical Data

The two main categorical groups (HC and INT) did not differ significantly in age, gender, or educational level (see Table 1.1). As expected, the INT group showed significant differences in clinical symptom measures, i.e., greater obsessive-compulsive, worry, and depressive symptoms (all *p*s <.001). When comparing the HC group with the two anxiety subgroups (ANX and ANX & MDD; both including patients with OCD), we again observed group differences across all clinical measures (see Table 1.2). In this case, the two clinical groups differed significantly from the controls and from each other. The ANX & MDD group showed increased symptom severity in all dimensional measures, including obsessive-compulsive (p = .024), worry (p = <.001), and depressive symptoms (p = <.001) compared to the ANX group.

Table 1.1

Demographic and questionnaire data across main categorical groups.

***N* = 310**	**HC (*n* = 97)**		**INT (*n* = 213)**		**Group comparison**			
	M	***SD***	***M***	***SD***	χ^2/t	***df***	***g***	***p***
Demographics								
Gender	74/	–	171/	–				
(f/m/d)	22/1		42/0		2.61	2	–	.271
Age	29.68	9.55	29.31	9.10	0.33	308	0.04	.744
Edu.	12.29	0.84	12.19	1.03	0.84	308	0.10	.400
Questionnaires								
OCI-R	5.85_a	8.55	15.56_b	12.57	-6.91	307	-0.85	**<.001**
PSWQ	39.33_a	11.95	56.66_b	12.33	-11.59	308	-1.42	**<.001**
BDI-II	3.96_a	5.04	15.20_b	11.52	-9.21	308	-1.13	**<.001**

Note. HC = healthy controls; INT = patients with current internalizing disorders; gender (f = female, m = male, d = diverse); age and education (Edu.) in years; OCI-R = Obsessive-Compulsive Inventory-Revised; PSWQ = Penn State Worry Questionnaire; BDI-II = Beck Depression Inventory-II. Within rows, values not sharing a subscript differ significantly (p < .05). *p*s < .05 are printed in bold.

Table 1.2

Demographic, diagnosis, medication, and questionnaire data across HC and anxiety subgroups (with and without MDD).

***N* = 310**	**HC (*n* = 97)**		**ANX (*n* = 153)**		**ANX & MDD (*n* = 60)**		**Group comparison**			
	M	***SD***	***M***	***SD***	***M***	***SD***	χ^2/F	*df*	η_p^2	*p*
Demographics										
Gender	74/	–	124/	–	47/13	–				
(f/m/d)	22/1		29/0		/0		2.81	4	–	.591
Age	29.68	9.55	28.59	8.59	31.13	10.16	1.69	2, 307	.01	.186
Edu.	12.29	0.84	12.20	1.05	12.15	1.01	0.42	2, 307	<.01	.660
Primary Diagnoses										
OCD	0_a	–	27_b	–	12_b	–	–	–	–	–
SAD	0_a	–	26_b	–	13_b	–	–	–	–	–
PHOB	0_a	–	31_b	–	6_b	–	–	–	–	–
GAD	0_a	–	22_b	–	11_b	–	–	–	–	–
PD	0_a	–	19_b	–	13_b	–	–	–	–	–
AG	0_a	–	28_b	–	5_b	–	–	–	–	–
Medication										
Psychotropics	1_a	–	14_b	–	17_c	–	39.05	6	–	**<.001**
SSRIs	0_a	–	8_a	–	14_b	–	–	–	–	–
Other †	1_a	–	5_a	–	1_a	–	–	–	–	–
Mix	0_a	–	1_a	–	2_a	–	–	–	–	–
Questionnaires										
OCI-R	5.85_a	8.55	14.12_b	12.39	19.20_c	12.37	28.80	2, 306	.16	**<.001**
PSWQ	39.33_a	11.95	54.25_b	12.69	62.80_c	8.83	83.13	2, 307	.35	**<.001**
BDI-II	3.96_a	5.04	11.68_b	9.04	24.18_c	12.32	97.68	2, 307	.39	**<.001**

Note. HC = healthy controls; ANX = patients with current OCA disorder and without current depressive disorder; ANX & MDD = patients with current OCA disorder and depressive disorder; gender (f = female, m = male, d = diverse); age and education (Edu.) in years; OCI-R = Obsessive-Compulsive Inventory–Revised; PSWQ = Penn State Worry Questionnaire; BDI-II = Beck Depression Inventory-II. SSRIs = selective serotonin reuptake inhibitors. † = serotonin–norepinephrine reuptake inhibitors (SNRIs), other antidepressants, anxiolytics, stimulants, and beta-blockers. Within rows, values not sharing a subscript differ significantly ($p < .05$). *p*s < .05 are printed in bold.

Behavioral Data

When comparing the HC with the INT group, we found no significant effects on error rates or PES, either for the robust or the congruency-corrected robust version (see Table 2.2, Supplement B). With respect to RT, a significant response effect was observed, indicating that incorrect responses were associated with shorter RT than correct responses, $F(1, 308) = 998.73$, $p < .001$, $\eta_p^2 = .76$. In addition, a significant group (HC vs. INT) effect, $F(1, 308) = 4.19$, $p =$

.042, η_p^2 = .01, revealed that the INT group (*M* = 388.42 ms, *SD* = 47.27) generally responded faster than the HC group (*M* = 400.46 ms, *SD* = 49.71). This significant group effect on RT also held for the comparison between the HC group and the anxiety subgroups, *F*(2, 307) = 4.41, *p* = .013, η_p^2 = .03 (see Table 2.1). Post hoc comparisons indicated that the HC group (*M* = 400.46 ms, *SD* = 49.71) and the ANX group (*M* = 384.03 ms, *SD* = 3.74) differed significantly in overall response speed, *t*(248) = 2.70, *p* = .025, *g* = .34, 95% CI [0.09, 0.60]. The differences between the ANX & MDD group (*M* = 399.61 ms, *SD* = 48.23) and both the HC group and the ANX group were not significant.

Table 2.1

Behavioral Performance-monitoring Measures by Group.

Measure	HC (*n*=97)	ANX (*n*=153)	ANX & MDD (*n*=60)	Test Statistic
Error rate (%)	14.21 (7.77)	15.14 (7.98)	13.94 (7.58)	*F*(2,307) = 0.69, *p* = .502
RT (ms)	400.46 (49.71)	384.03 (46.31)	399.61 (48.23)	*F*(2,307) = 4.41, ***p* = .013**
RT, in correct trials (ms)	432.12 (47.66)	418.47 (46.07)	433.03 (44.72)	Group × Response Type: *F*(2,307) = 0.79, *p* = .454
RT, in error trials (ms)	368.80 (58.05)	349.59 (51.56)	366.18 (57.11)	*(see above)*
PES_a (ms)	50.18 (31.63)	48.63 (26.76)	56.09 (29.70)	*F*(2,307) = 1.44, *p* = .238
PES_b (ms)	46.30 (31.45)	44.51 (26.43)	53.72 (37.79)	*F*(2,307) = 1.99, *p* = .138

Note. Values are *M* (*SD*), unless otherwise indicated.
RT = Reaction Time; PES_a = Post-error Slowing, robust version; PES_b = congruency-corrected robust version. *p*s < .05 are printed in bold.

Response-related Theta Power

Categorical Analyses

Regarding response-related theta power, see Table 3.1 for ANOVA results of two-group (HC, INT) and three-group comparisons (HC, ANX, ANX & MDD), and Tables 3.2 and 3.3 for results of direct group comparisons (Supplement B). A significant main effect of response type emerged, *F*(1, 308) = 871.99, *p* <.001, η_p^2 = .74, indicating that theta power was clearly increased following errors compared to correct responses. Furthermore, we found a significant group effect for the comparison of the HC vs. INT group, *F*(1, 308) = 12.20, *p* <.001, η_p^2 = .04. The INT group (*M* = 110.32 %, *SD* = 40.30) showed increased overall theta power (averaged across response types; percentage given relative to baseline) compared to the HC group (*M* = 93.42 %, *SD* = 37.69). However, the response × group interaction did not reach significance, *F*(1, 308) = 3.04, *p* = .082, η_p^2 = .01, supporting a general, not error-specific increase in theta power in internalizing disorders. Accordingly, when comparing response-specific theta activity between groups, a significant group difference emerged both in error-related theta, *t*(308) = -

2.76, p = .006, g = -0.31, 95% CI [-0.54, -0.09], and in theta following correct responses, t(308) = -3.49, p <.001, g = -0.40, 95% CI [-0.62, -0.17], underscoring the overall heightened level of response-related theta power in the INT group. Theta results (HC vs. INT) are depicted in Figures 1.3 and 1.4 (see Supplement B).

The group main effect on theta power was also significant for the model including three groups, i.e., between the HC, ANX and ANX & MDD groups, F(2, 307) = 6.55, p = .002, η_p^2 = .04. Interestingly, the post hoc comparisons revealed that overall theta power (averaged across response types) only differed between the HC and the ANX group, t(248) = -3.67, p <.001, g = -0.48, 95% CI [-0.73, -0.22], and not between the HC and the ANX & MDD group, t(155) = -1.97, p = .143, g = -0.32, 95% CI [-0.64, 0.00]. Similarly, error-related theta differed only between HC and the ANX group, t(248) = -2.74, p = .019, g = -0.31, 95% CI [-0.54, -0.09], and not between HC and the ANX & MDD group, t(155) = -1.77, p = .216, g = -0.20, 95% CI [-0.43, 0.02]. Also, theta power following correct responses was increased only in the ANX group, t(248) = -4.00, p <.001, g = -0.46, 95% CI [-0.68, -0.23], but not significantly in the ANX & MDD group, t(155) = -1.23, p = .521, g = -0.14, 95% CI [-0.36, 0.08]. Figures 1.1 and 1.2 depict theta results (HC vs. ANX vs. ANX & MDD).

To test whether age, gender, or medication influenced the observed group effects on theta power, we conducted additional control analyses (see Supplement B). These revealed no significant interactions, and the aforementioned group effects remained significant, supporting the robustness of results across age, gender, and medication.

Table 3.1

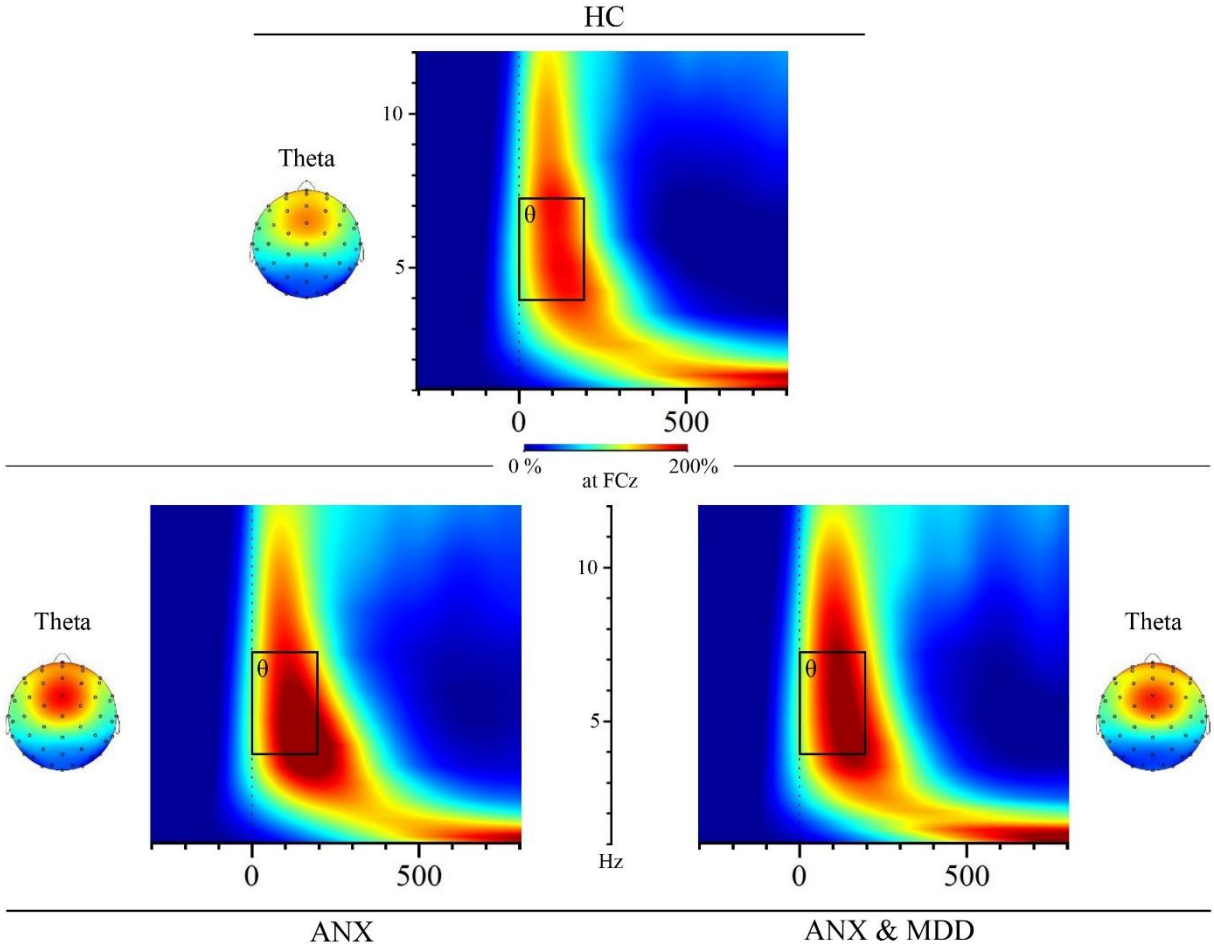
Results of the mixed-effects ANOVAs (2 x 2 or 2 x 3, respectively) for theta power across the two main categorical groups (HC, INT) and across three groups: the control and the two anxiety subgroups (HC, ANX, ANX & MDD).

	F	df	η_p^2	p
Theta Power *(HC, INT)*				
Response	872.00	1, 308	.74	**<.001**
Group	12.20	1, 308	.04	**<.001**
Response × Group	3.04	1, 308	.01	.082
Theta Power *(HC, ANX, ANX & MDD)*				
Response	914.18	1, 307	.75	**<.001**
Group	6.55	2, 307	.04	**.002**
Response × Group	1.53	2, 307	.01	.218

Note. Response refers to the response type (i.e., incorrect or correct). ps < .05 are printed in bold.

Figure 1.1

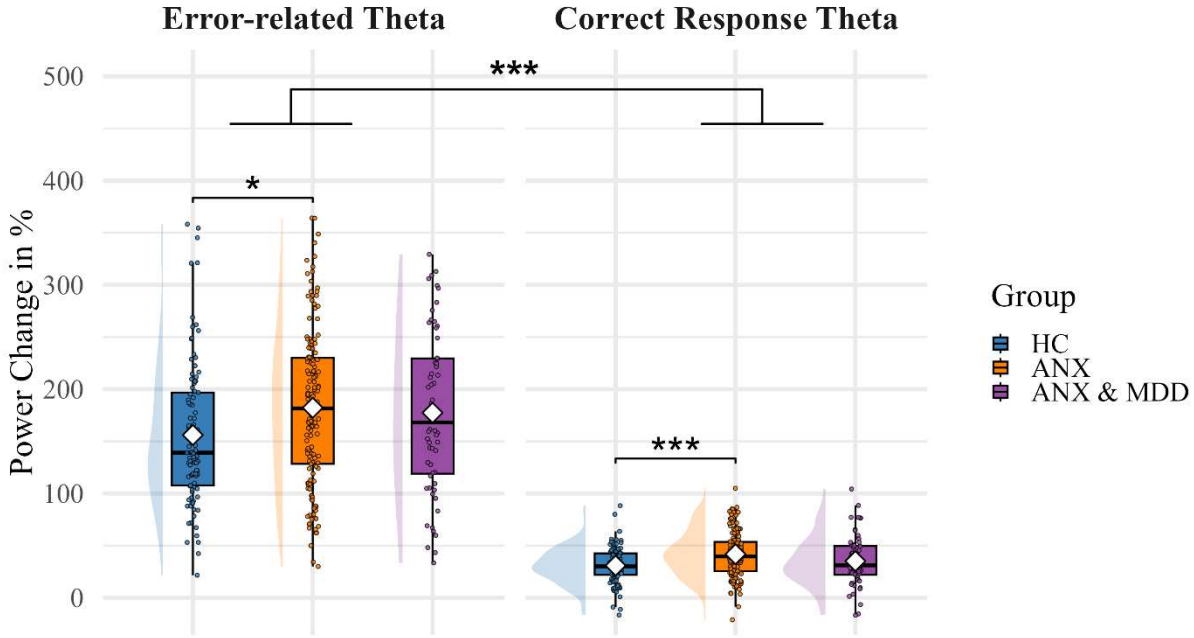
Time-Frequency heatmaps of error-related theta power across HC, ANX, and ANX & MDD groups at the channel FCz.



Note. On the x-axis, the time span from -300 to 800 ms and on the y-axis, the frequency range from 1 to 12 Hz are shown. '0' on the x-axis marks the time point of error commission. Power ranges from 0% change (dark blue) relative to the -300 to 0 ms baseline to 200% increase (dark red). Topographic plots are shown on the left and right sides, directly next to corresponding subplots for Theta (3.97–7.24 Hz). They show the averaged topographical activation of Theta (0-200 ms) after error commission.

Figure 1.2

Boxplots of response-related theta power at channel FCz across HC and the two anxiety subgroups.



Note. Response type is shown above the panels, and mean power change relative to baseline (-300 to 0 ms) in % at the FCz channel is shown on the y-axis. Boxplots depict the median, interquartile range, and whiskers; individual data points are overlaid; white diamonds indicate group means. For all groups, theta power is significantly increased after errors compared to after correct responses. The response effect and the group difference in theta after correct responses ($p < .001$) are marked by]*** and the group difference in theta following errors ($p < .05$) by]*. Analysis: mixed-effects ANCOVA, with response (error/correct) as within-subject factor, group (HC/ANX/ANX & MDD) as between-subject factor. $N = 310$; n (HC) = 97, n (ANX) = 153, n (ANX & MDD) = 60.

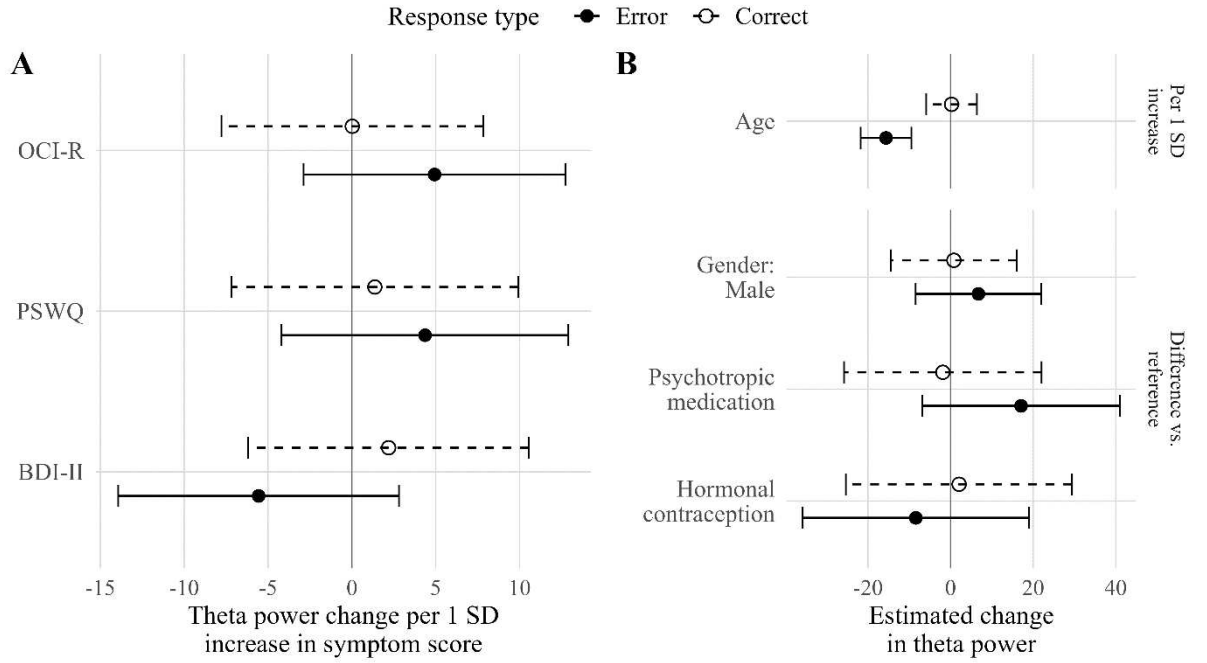
Dimensional Analysis

The dimensional LMM analysis of response-related theta power (see Table 4, Supplement B) replicated the previously reported response effect, indicating that theta power was significantly increased following errors compared to correct responses, $F(1, 300) = 22.69$, $p < .001$, $\eta_p^2 = .07$. Also, we found an overall effect of age, $F(1, 300) = 11.07$, $p < .001$, $\eta_p^2 = .04$, on theta. The age main effect was further specified by a significant response type × age interaction, $F(1, 300) = 14.11$, $p < .001$, $\eta_p^2 = .04$. Post-hoc comparisons revealed that increasing age led to a decline in theta power, but only following errors, $b = -15.62$, $t(595) = -4.99$, $p < .001$, and not after correct responses, $b = 0.24$, $t(595) = 0.08$, $p = .940$. Given our hypotheses on the effects of symptom dimensions (OCI-R, PSWQ, BDI-II), we examined their associations with theta overall and separately for correct and incorrect responses using post-hoc simple slopes, despite non-significant omnibus effects. Post-hoc trends of dimensional effects on overall theta were not significant (all ps > .37). Likewise, response-specific trends revealed no significant effects for the three symptom dimensions on correct response theta (all ps > .60) or error-related theta (all ps > .19). Response-specific effects of symptom dimensions and control variables on theta are

presented in Figure 2. Results remained unchanged when the model was estimated without control variables (age, gender, medication).

Figure 2

Forest Plot of the LMM effects of the three symptom dimensions and the control variables (age, gender, medication) on response-related theta power.



Note. **A**. OCI-R = Obsessive-Compulsive Inventory–Revised; PSWQ = Penn State Worry Questionnaire; BDI-II = Beck Depression Inventory-II. **B**. Effect of male gender with "female gender" as reference; effect of psychotropic medication and hormonal contraception with "no medication" as reference.

Multiple Linear Regressions of Error-related Behavior

The results of the correlation matrix (see Table 5, Supplement B) showed that all questionnaire sum scores (OCI-R, PSWQ, BDI-II) were strongly correlated (all *r*s = .59–.70, all *p*s <.001) with no further significant correlations among the remaining variables.

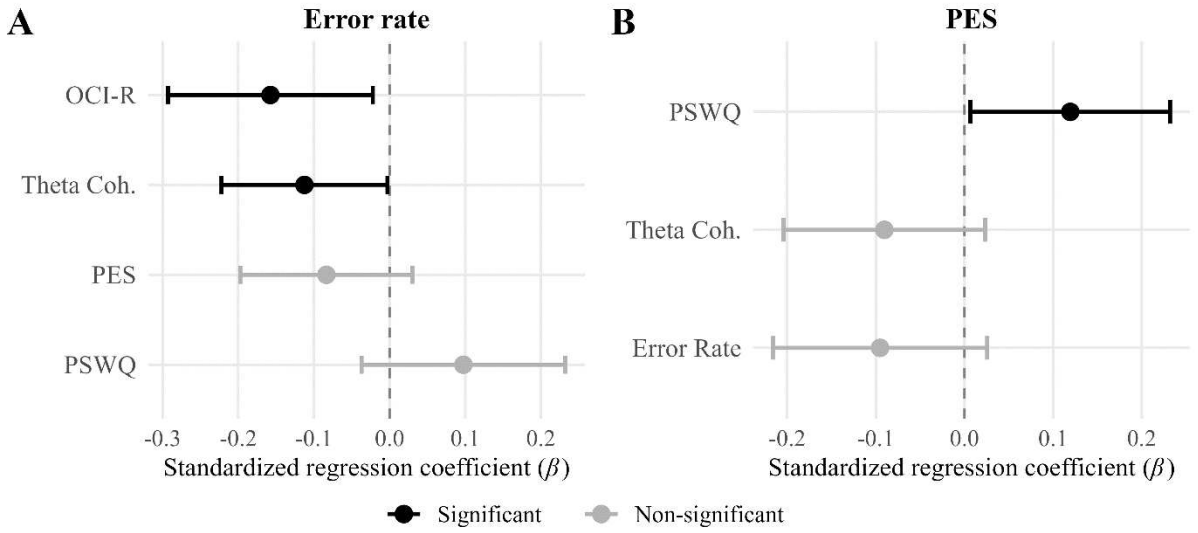
The final model explaining the error rate (see Table 6, Supplement B) was significant, $F(4, 277) = 2.96$, $p = .020$. It explained 2.7% of the variance in the error rate, *adj.* $R^2 = .027$. The predictors of the final model were (from strongest to weakest): OCI-R, error-related prefrontal theta coherence, PES, and PSWQ. Notably, the OCI-R sum score, $\beta = -0.16$, $t(277) = -2.29$, $p = .023$, and the prefrontal theta coherence, $\beta = -0.11$, $t(277) = -2.02$, $p = .044$, were significant predictors within the model. At the same time, the other two were not significant (see Figure 3). Both the OCI-R and theta coherence were negatively associated with the error rate, indicating that greater obsessive-compulsive symptom load and higher prefrontal theta coherence following errors went along with increased accuracy. For a visualization of significant MLR predictors for error rate, see Figure 4.1 (Supplement B).

Likewise, the final model explaining PES (see Table 6, Supplement B) was significant, $F(3, 278) = 3.02$, $p = .030$. It explained 2.1% of the variance of PES, *adj.* $R^2 = .021$. The predictors

of the final model were (from strongest to weakest): PSWQ, error-related prefrontal theta coherence, and the error rate. Of these three, only PSWQ was a significant predictor (see Figure 3), $\beta = 0.12$, $t(278) = 2.09$, $p = .038$, indicating a positive association with PES. This suggests that greater worry symptoms went along with increased reaction time slowing following errors. For a visualization of the significant MLR predictor for PES, see Figure 4.2 (Supplement B).

Figure 3

Predictors of the final MLR models for the error rate and PES.



Note. **A**. MLR model with error rate as the dependent variable; OCI-R = Obsessive-Compulsive Inventory–Revised; Theta Coh. = Error-related Prefrontal Theta Coherence; PES = Post-error Slowing; PSWQ = Penn State Worry Questionnaire. **B**. MLR model with PES as the dependent variable.

3. Discussion

This study investigated response-related midfrontal theta power as a transdiagnostic marker of cognitive control across obsessive-compulsive and anxiety spectrum (OCA) disorders with and without comorbid depression, combining categorical and dimensional approaches to capture both diagnostic and symptom-level alterations. Three main results emerged regarding midfrontal theta power: First, patients with internalizing disorders (OCA disorders and MDD) showed increased theta power compared to healthy controls. Second, contrary to our error-specific hypotheses, this increase was not limited to errors but also emerged for correct responses. Third, an additional analysis showed that this effect was primarily attributable to patients with OCA disorders without comorbid depression. The dimensional analysis revealed no significant symptom-specific effects on theta power. Multiple linear regressions indicated that across all participants, higher accuracy was associated with increased obsessive-compulsive symptoms and elevated prefrontal error-related theta coherence, and that prolonged PES was associated with greater worry symptoms, highlighting OCA-related and symptom-specific alterations in behavioral indices of adaptive control.

In line with our first hypothesis, we found increased theta activity, i.e., elevated cognitive control, in patients with internalizing disorders. We identified a modulating role of comorbid depression related to this effect, as patients with OCA disorders without MDD but not those with MDD showed a significant increase in theta compared with healthy controls. However, contrary to our expectations and previous findings (Cavanagh et al., 2017; Dell'Acqua et al., 2023a), this pattern was not error-specific but also occurred after correct responses. Previous studies identified MDD as a potential blunting factor of the ERN (Ladouceur et al., 2012; Weinberg et al., 2012) and of error-related theta (Dell'Acqua et al., 2023b). In our results, only patients with OCA disorders without comorbid MDD showed significantly increased theta power compared with controls, which may point in a similar direction. However, the OCA groups with and without MDD did not differ significantly from each other, and a dimensional association between theta and depressive symptoms did not reach significance. Our findings confirm the linkage between OCA disorders and enhanced prefrontal engagement of cognitive control (Cavanagh et al., 2017; Dell'Acqua et al., 2023a), and are in line with the view of theta as a signal of the need for control under uncertainty or threat (Cavanagh & Shackman, 2015). This also fits the notion of threat-related increases in cognitive control, which, however, do not improve performance but rather help maintain adequate performance levels (Eysenck et al., 2007). Interestingly, our dimensional analyses did not reveal significant associations between OCA symptoms and theta, but were similar in direction. Overall, our results support midfrontal theta power as a transdiagnostic marker of OCA-related alterations in cognitive control, which may be modulated by comorbid depression (Cavanagh & Shackman, 2015; Dell'Acqua et al., 2023b; McLoughlin et al., 2022).

An important question is whether this cognitive control marker specifically reflects alterations in relation to error processing. As aforementioned, patients with OCA disorders showed increased theta power not only after errors but also after correct responses. This pattern may reflect a general, sustained hyperengagement of cognitive control in midfrontal areas rather than an error-specific alteration. Accordingly, enhanced theta power may serve as a

transdiagnostic marker not only for error monitoring but, more broadly, for overactive performance monitoring, extending beyond earlier, more error-focused conceptualizations (McLoughlin et al., 2022). This interpretation aligns with ERP literature, where increases have been reported not only for the ERN but also for the correct-response negativity (CRN), such as in OCD (Endrass et al., 2008; Klawohn et al., 2014; Riesel et al., 2023). More recently, an aggregated group of patients with present and past internalizing disorders showed increases in ERN and CRN, likewise challenging the notion of error- and disorder-specific neural markers and instead indicating enhanced performance monitoring as a broad, transdiagnostic marker (Härpfer et al., 2025). Given the close correspondence between the ERN and error-related theta power outlined earlier (Luu et al., 2004; Trujillo & Allen, 2007), a similarly general rather than error-specific increase in oscillatory activity in OCA disorders seems plausible. From a clinical perspective, this general, response-independent over-engagement of cognitive control may manifest as hypervigilance toward potential conflicts or errors and increased monitoring of all types of responses. This is consistent with findings of increased error monitoring or cognitive control in situations where errors are not particularly relevant (Dolge et al., 2026; Riesel et al., 2019). Such hypervigilance, in turn, could contribute to transdiagnostic symptoms such as persistent doubt and uncertainty and may prompt checking behavior.

However, the categorical and dimensional effects of OCA disorders did not show a consistent pattern. Specifically, in the categorical analysis, we found an effect of OCA disorders on theta power that was not observable in the three symptom dimensions across the whole sample. Several underlying reasons might be possible. First, the potential effects of psychopathology on error-related theta activity may be enhanced above a certain clinical threshold or specific to that threshold. Second, dimensional effects may partly reflect the distribution of symptom scores in our sample: both OCI-R and BDI-II scores were notably right-skewed. Such skewness represents a common challenge in samples that include both clinical and healthy participants (Arrindell et al., 2026) and can restrict the effective range of symptom severity available for detecting linear associations. Third, categorical contrasts capture variance shared across symptom domains, whereas our dimensional models estimated each symptom dimension's unique contribution while controlling for the others. Effects of psychopathology on theta power may thus be driven by variance shared across the internalizing spectrum (Krueger & Markon, 2006), rather than by specific symptom dimensions, in line with evidence linking midfrontal theta to anxiety more broadly (Cavanagh & Shackman, 2015).

In contrast to theta power, symptom-specific associations emerged at the behavioral level. In the MLR on the error rate, increased obsessive-compulsive symptoms were associated with enhanced accuracy. This fits the notion that OCD is often characterized by traits and symptoms such as perfectionism, concerns about mistakes, and doubts about actions (Callaghan et al., 2024), which may facilitate high accuracy. The association between increased prefrontal theta coherence and enhanced accuracy aligns with the notion that midfrontal theta coordinates and supports cognitive control (Cavanagh & Frank, 2014), a process captured by coherence (Cavanagh et al., 2009; Lachaux et al., 1999), leading to improved performance. Furthermore, increased worry symptoms were associated with prolonged PES, which is consistent with the finding that PES appears to be specifically increased in anxiety patients (Rueppel et al., 2022). This over-engagement of adaptive control after errors is not accompanied by improved accuracy

and mirrors our categorical findings, where increased theta activity likewise was not connected to improved accuracy. Previous single-trial analyses point in a similar direction: midfrontal theta predicted divergent behavioral adaptation across groups, such that in patients with OCD, increased theta was followed by response slowing without a corresponding gain in accuracy, whereas in unaffected controls theta increase was associated with faster and comparably more accurate subsequent responses (Dolge et al., under review). Taken together, these findings suggest that patients with OCA disorders differ not simply in magnitude of cognitive control engagement but rather in how effectively it is coupled with behavioral benefit. This can be interpreted through Eysenck's Attentional Control Theory (Eysenck et al., 2007), assuming anxiety primarily compromises processing efficiency. Accordingly, anxious individuals compensate for increased threat-related interference by recruiting additional cognitive control resources to maintain adequate performance levels despite reduced efficiency. This is also in line with meta-analytic evidence of shared structural alterations in the dorsal ACC across psychopathology, including OCD and anxiety (Goodkind et al., 2015), and with transdiagnostic deficits in cognitive control at both the neuropsychological (McTeague et al., 2016) and neural level (McTeague et al., 2017). Together with our findings, this suggests that shared structural changes in the ACC may influence less the recruitment of cognitive control itself than the translation into adaptive behavior.

Strengths and Limitations

This study benefits from a large, transdiagnostic sample with heterogeneous disorders across the whole anxiety spectrum in combination with MDD, allowing for a comprehensive investigation of theta as a promising transdiagnostic marker of cognitive control. However, a few limitations are important to note: First, our MLR models explain only a relatively modest proportion of variance. Similarly, in the LMM, a rather small proportion of variance in theta power was explained by differences between participants (adj. ICC = .09), limiting the variance that person-level predictors such as symptom dimensions could account for. Second, the comparison between patients with OCA disorders with and without comorbid depression was based on a post hoc subdivision of the sample rather than on an a priori targeted sampling strategy of an MDD group. This may have resulted in the observed pattern partly reflecting overall symptom burden rather than depression-specific effects. Lastly, our cross-sectional design precludes conclusions about the temporal or causal nature of observed group differences or associations between symptom measures and theta activity or behavioral outcomes. Thus, it remains unclear whether alterations in cognitive control or adaptive behavior represent a vulnerability marker preceding symptom onset or a state-dependent correlate of current symptom expression, which longitudinal designs could disentangle. Future studies should extend this approach to externalizing disorders (e.g., ADHD, substance use disorder) to examine whether theta represents a broader marker of psychopathology.

Conclusion

This study provides empirical evidence for midfrontal theta power as a transdiagnostic marker of cognitive control across internalizing disorders during performance monitoring. Increases in theta power following both errors and correct responses were found in patients with OCA

disorders without comorbid depression but not in those with comorbid depression, suggesting that anxiety and OCD seem to be associated with sustained hyperengagement of cognitive control, which appears to be attenuated in the presence of comorbid depression. Enhanced accuracy was associated with greater obsessive-compulsive symptoms and increased prefrontal theta coherence, and prolonged PES, in turn, with greater worry symptoms. Taken together, these results suggest that patients with OCA disorders overly engage cognitive and adaptive control (i.e., theta and PES) without improving their performance, but rather to maintain a performance level comparable to unaffected individuals, albeit at higher cognitive cost, consistent with a transdiagnostic structural alteration of the cognitive control system. Thus, our findings support the notion that alterations in cognitive control are shared across internalizing disorders, with comorbid depression modulating their neural expression.

4. Methods

Before data analysis, research hypotheses and methodological decisions were preregistered (osf.io/8mhnq). An ERN study based on a subset of the sample presented in this study (156 participants; data from 2021–2022) has already been published (doi.org/10.1155/da/9505414) and pre-registered (osf.io/7dcm3).

Participants

In total, 363 participants were recruited between 2021 and 2025 through the University of Hamburg's outpatient unit and local advertisements. They gave written informed consent. Recruitment groups were based on a categorical, diagnosis-based assignment: obsessive-compulsive disorder (OCD; n = 40), social anxiety disorder (SAD; n = 40), specific phobia (PHOB; n = 40), generalized anxiety disorder (GAD; n = 43), agoraphobia with or without panic disorder (AG; n = 41), and panic disorder (PD; n = 39), as well as 120 control participants. Before laboratory testing, participants were screened using a structured clinical interview for DSM-5 (SCID-5-CV; Beesdo-Baum et al., 2019; First et al., 2016) conducted by trained personnel. Participants meeting DSM-5 criteria for a current obsessive-compulsive and anxiety spectrum (OCA) disorder were assigned to respective clinical groups. If multiple current OCA disorders were present, the personnel administering the SCID-5-CV identified a primary diagnosis that caused the most psychological distress and impairment in daily life. We imposed no restrictions on comorbidities among clinical participants, except for predefined clinical exclusion criteria, which applied to all groups: a history of schizophrenia, a history of substance use disorder, a current bipolar disorder, a current manic episode, or a current post-traumatic stress disorder (further exclusion criteria are listed in Supplement A). Control participants had no current or lifetime OCA disorder. The University of Hamburg's local ethics committee approved all study procedures (AZ: 2021_351; KA_2023_011), and we conducted the study in accordance with the Declaration of Helsinki (World Medical Association, 2013).

We excluded 53 participants before data analysis. We excluded 8 participants due to insufficient accuracy (< 60%). We further excluded 10 participants based on data quality: 8 due to excessive artifact contamination, and 2 due to outlying theta power values (identified via visual inspection of the theta power distribution across the sample). A further 35 participants were excluded because their comorbidity profile did not allow unambiguous post hoc group assignment: control participants who met criteria for a current mental disorder (n = 15) and therefore did not qualify as healthy controls, and patients who met criteria for both a current internalizing and a current externalizing disorder (n = 20). Thus, we defined healthy controls (HC) as control participants without any current mental disorder. Lifetime mental disorders were permitted unless they met criteria for an OCA disorder or a clinical exclusion criterion.

The final sample comprised N = 310 participants. Post hoc group assignment followed the main categorical comparison of the study: patients with internalizing disorders (INT; i.e., OCA disorders, with or without depression; n = 213) versus HC (n = 97). Exploratorily, the INT group was further divided into an anxiety group with comorbid depression (ANX & MDD; n = 60) and without comorbid depression (ANX; n = 153). Tables 1.1 and 1.2 present detailed demographic and clinical characteristics by diagnostic subgroup. Sensitivity power analyses are reported in Supplement A.

Task and Procedure

Participants performed an arrowhead version of the Eriksen flanker task (Eriksen & Eriksen, 1974), preceded by a resting measurement, embedded as the first task, and followed by three other tasks within the experimental session (passive picture viewing task, NPU threat task, fear conditioning paradigm). The flanker task included 20 practice trials and five blocks of 80 trials (400 total), with an equal number of congruent and incongruent trials pseudo-randomly presented. Participants indicated the direction of the central arrow via left/right keypress. They were instructed to respond as quickly as possible from the break after the first block onward (based on Riesel et al., 2019). However, this instruction did not correspond to actual performance. Each trial began with a fixation cross (200–1200 ms, jittered), followed by stimulus presentation (100 ms), and a response window (max. 800 ms). The display parameters, viewing angle, randomization details, and complete task sequence are listed in Supplement A.

Clinical and Behavioral Measures

Three symptom dimensions were included in the present study: obsessive-compulsive symptoms via the Obsessive-Compulsive Inventory–Revised (OCI-R; 20 items, 5-point Likert scale 0-4; Foa et al., 2002; Gönner et al., 2007; $\alpha = .91$), worry or anxious apprehension via the Penn-State Worry Questionnaire (PSWQ; 16 items, 5-point Likert scale 1–5; Glöckner-Rist & Rist, 2014; Meyer et al., 1990; $\alpha = .95$), and depressive symptoms via the Beck Depression Inventory-II (BDI-II; 21 items, 4-point Likert scale 0–3; Beck et al., 1996; Hautzinger et al., 2006; $\alpha = .94$).

Behavioral measures included reaction time (RT), error rate, and post-error slowing (PES). We computed PES following the classical robust version (Dutilh et al., 2012) and the congruency-corrected robust version (Derrfuss et al., 2022). RT showed an excellent Spearman-Brown corrected split-half correlation ($r = .99$). PES showed a moderate split-half correlation: $r = .445$ for the robust version and $r = .422$ for the congruency-corrected robust version. The limited reliability observed for PES is likely attributable to the relatively small number of error trials available per participant for its computation, combined with high intra-individual variability.

EEG Recording and Data Processing

EEG was recorded from 61 Ag/AgCl electrodes (equidistant montage, Easycap) using two 32-channel BrainAmp amplifiers (Brain Products GmbH), recorded at a sampling rate of 1,000 Hz with a band-pass filter of 0.01–250 Hz. For preprocessing, data were band-pass (0.1–30 Hz, 24 dB/oct) and notch-filtered (50 Hz), corrected for ocular artifacts via semi-automatic ICA (Jung et al., 2000), re-referenced to the average of all electrodes, and segmented into response-locked epochs (-1500 to 2500 ms) for correct and incorrect responses. Artifact-contaminated segments (absolute voltage range exceeding 200 µV, voltage step exceeding 50 µV between consecutive data points, or maximum voltage difference of less than 0.5 µV within 100 ms intervals) were removed. A current source density (CSD) transformation was applied to reduce volume conduction effects (Cohen, 2014). A complex Morlet wavelet transformation ($c = 4$ cycles) generated 20 logarithmically spaced frequency layers (1–30 Hz), normalized to the 300 ms preceding the response. Total (induced + evoked) theta power was extracted from two layers (3.97–7.24 Hz) at the electrode FCz, from 0–200 ms post-response. Magnitude-squared

coherence (MSC) between medial (FCz) and dorsolateral (F5/F6) prefrontal electrodes was computed from single-trial wavelet data, following Cavanagh et al. (2009). Full extraction and trial-matching details are provided in Supplement A.

Theta Power showed a very good spearman-brown corrected split-half correlation (r = .843). Group-specific split-half correlations were comparable, $r_{(HC)}$ = .808, $r_{(ANX)}$ = .828, and $r_{(ANX\ \&\ MDD)}$ = .862.

Statistical Analysis

Analyses were performed in R (4.6.0; R Core Team, 2026) and RStudio (2026.06.0; Posit Team, 2026), with α = .05 (two-sided). The frequencies of gender, diagnosis, and medication use across groups were analyzed using a chi-square test. All other demographic and clinical measures, and error rate and PES, were tested with independent-samples *t*-tests for the binary HC vs. INT comparison and with univariate ANOVAs (*1 x 3*) for the exploratory comparison with HC, ANX, and ANX & MDD groups. RT and oscillatory measures were compared using mixed-effects ANOVAs (*2 x 2* or *2 x 3*, respectively) with response type (incorrect/correct) as a within-subject factor. Significant interactions were resolved using post hoc *t*-tests (Sidak-corrected). Effect sizes, i.e., partial Eta squared (η_p^2) or Hedges' *g* with 95% confidence intervals (CI), are reported throughout. To assess whether potential group effects in theta power were confounded by age, gender, or medication, we additionally tested the respective effects as covariates in separate models (see Supplement B).

To examine dimensional effects on response-related theta power, oscillatory measures were analyzed with linear mixed models (LMMs) using the *lme4* package (Bates et al., 2015) in R. Each model included response type (correct vs. incorrect), OCI-R, PSWQ, and BDI-II scores as predictors of interest, with age, gender, and medication as control variables. Continuous predictors were *z*-standardized. Random intercepts for participants were included to account for the repeated-measures structure. After accounting for response type, the intraclass correlation was low (adjusted ICC = .09), indicating that most variance in theta power was located within rather than between participants. Multicollinearity among predictors was low (all VIFs < 2.1). Models were estimated using restricted maximum likelihood (REML). Fixed effects were tested with Type III *F*-tests and Satterthwaite-approximated degrees of freedom (*lmerTest*; Kuznetsova et al., 2017). Significant interactions were further investigated using the *emmeans* package (Lenth & Piaskowski, 2026): simple slopes of continuous predictors were estimated separately for correct and incorrect responses (emtrends), and categorical effects were examined with pairwise comparisons of estimated marginal means. *P*-values of post-hoc comparisons were Sidak-adjusted.

DV ~ Response Type * (OCI-R + PSWQ + BDI-II + Age + Gender + Medication) + (1 | Participant)

Spearman correlations were computed among clinical and error-related behavioral and neural measures. Two multiple linear regression models (MLRs) predicted error rate and PES (congruency-corrected robust version) from clinical and error-related neural and behavioral predictors, using stepwise backward elimination with Akaike Information Criterion (AIC) for predictor selection. All predictors met multicollinearity criteria (tolerance > 0.2; VIF < 5).

5. Disclosures

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5.2 Conflict of Interest

The authors declare no conflicts of interest.

5.3 Ethics Approval

The local ethics committee of the University of Hamburg approved the study with the approval numbers AZ: 2021_351 and KA_2023_011.

5.4 Consent to Participate

All participants provided written informed consent to participate in this study.

5.5 Consent for Publication

We obtained written informed consent for publication from all participants included in the study.

5.6 Availability of Data and Data Statement

The complete dataset used (without demographics) is made available with restricted access at Zenodo (https://doi.org/10.5281/zenodo.23695100). Data are not made fully available to the public, as clinical data are subject to certain regulations, so they cannot be shared with everyone but can be shared upon request.

5.7 Code Availability

EEG analyses were performed with Brain Vision Analyzer (Brain Products GmbH, Gilching, Germany, Version 2.3), and the code is not shared. Code from R (4.6.0; R Core Team, 2026) and RStudio (2026.06.0; Posit Team, 2026) used for the statistical analyses and figure creation is available with restricted access at Zenodo (https://doi.org/10.5281/zenodo.23695100).

5.8 Open Practices Statement

The dataset and code for statistical analyses and figure creation are available at Zenodo.

The study was preregistered on the Open Science Framework (OSF; DOI: osf.io/8mhnq). To improve focus and readability, not all preregistered primary analyses are reported in the main manuscript; delta power and theta/delta coherence analyses are presented only in Supplement C and excluded from the correlation analysis. We changed the originally planned group *clinical* to the group with *internalizing disorders*. Delta power and coherence, but not theta coherence, are excluded from the multiple linear regression analyses. We simplified the previously planned

LMM formula and focused on OCI-R, PSWQ, and BDI-II as symptom measures. We added medication as an additional control variable. In addition, we excluded age and gender from the potential regression predictors to increase statistical power and focus on the most theoretically relevant variables (symptom dimensions and neural measures).

5.9 Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used Claude (Claude Sonnet 5.5 and Opus 5.5, Anthropic, 2026) to adapt R scripts for statistical analyses and figure creation, and to refine the manuscript language. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

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Author Contributions (CRediT)

Alexander Dolge: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Data Curation, Writing – Original Draft, Writing – Review & Editing, Visualization; **Kai Härpfer:** Conceptualization, Software, Investigation, Data Curation, Writing – Review & Editing, Project Administration; **Hannes P. Carsten:** Data Curation, Writing – Review & Editing, Project Administration; **Franziska M. Kausche:** Data Curation, Writing – Review & Editing, Project Administration; **Anja Riesel:** Conceptualization, Methodology, Validation, Investigation, Resources, Writing - Review & Editing, Supervision, Project Administration, Funding Acquisition

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