

ORIGINAL ARTICLE

Associations of neural error-processing with symptoms and traits in a dimensional sample recruited across the obsessive–compulsive spectrum

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Funding information

German Research Foundation, Grant/Award Number: KA 815/7-1; HUMBOLDT-UNIVERSITAET ZU BERLIN

Abstract

The error-related negativity (ERN), a neural response to errors, has been associated with several forms of psychopathology and assumed to represent a neural risk marker for obsessive–compulsive disorder (OCD) and anxiety disorders. Yet, it is still unknown which specific symptoms or traits best explain ERN variation. This study investigated performance-monitoring in participants ($N = 100$) recruited across a spectrum of obsessive–compulsive characteristics ($n = 26$ patients with OCD; $n = 74$ healthy participants including $n = 24$ with low, $n = 24$ with medium, and $n = 26$ with high OC-characteristics). Several compulsivity- and anxiety-associated characteristics were assessed and submitted to exploratory principal axis factor analysis. Associations of raw measures and derived factors with ERN and correct-related negativity (CRN) were examined. Patients with OCD showed increased ERN amplitudes compared to healthy participants. The ERN was associated with a variety of traits related to anxiety and negative affect. Factor analysis results revealed a most prominent association of the ERN with a composite measure of anxiety and neuroticism, whereas the CRN was specifically associated with compulsivity. Results support differential associations for the ERN and CRN and demonstrate that a dimensional recruitment approach and use of composite measures can improve our understanding of characteristics underlying variation in neural performance monitoring.

KEYWORDS

anxiety, compulsivity, error-related negativity ERN, event-related potentials ERP, obsessive–compulsive disorder, transdiagnostic symptoms

1 | INTRODUCTION

Obsessive–compulsive disorder (OCD) is a debilitating neuropsychiatric disorder with a 2%–3% lifetime

prevalence (Ruscio et al., 2010). It often follows a chronic course (Eisen et al., 2013) and results in severe burden of illness (Mendlowicz & Stein, 2000). Key symptoms are centered on the fear of committing errors (e.g., not

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turning off the oven) that could lead to catastrophic consequences and harm (e.g., setting the house on fire). In response, compulsions often are aimed to avoid such errors and harm for oneself and others, and to neutralize respective anxiety. Given these symptoms, it has long been proposed that persistent hyperactive neural error signals are a central process underlying the pathophysiology of OCD (Pitman, 1987). Consistent with this, increased neural error signaling in OCD has been shown by EEG (Riesel, 2019) and fMRI (Norman et al., 2019) studies. A well-validated EEG-marker to study the neural underpinnings of error processing is the error-related negativity (ERN; Falkenstein et al., 1991; Gehring et al., 1993), a fronto-central negativity after errors. After correct trials, a similar but smaller negativity can be observed, the correct-response negativity (CRN, Vidal et al., 2000). The mid-cingulate cortex, particularly the anterior cingulate cortex (ACC), has been suggested as a main neural generator of these negativities (e.g., Debener et al., 2005; Norman et al., 2019; Ridderinkhof et al., 2004), working in concert with other areas such as the presupplementary motor area (Grützmann et al., 2016), lateral prefrontal cortex (Gehring & Knight, 2000; Kiehl et al., 2000), basal ganglia (Holroyd & Coles, 2002), and amygdala (Pourtois et al., 2010) to promote adaptive responses and avoid harm.

Over 35 studies have examined the ERN in individuals with OCD or subclinical OC-symptoms (e.g., Endrass & Ullsperger, 2014; Gehring et al., 2000). A recent meta-analysis aggregates these findings and illustrates that in response-conflict tasks, the ERN was robustly increased in both pediatric and adult OCD patients as well as in subclinical samples (Riesel, 2019). Further, results showing that increased ERN amplitudes in OCD persist after cognitive behavioral therapy (Hajcak et al., 2008; Riesel et al., 2015) and can be seen in healthy at-risk participants (Carrasco et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019) suggest that increased neural error signals are not a mere correlate of symptoms but a promising neural risk-marker for OCD underlying symptom development. However, not only OCD and OC-characteristics have been linked to increased ERN amplitudes but also other anxiety disorders, such as social anxiety (e.g., Endrass et al., 2014), generalized anxiety (e.g., Weinberg et al., 2010), and health anxiety (Riesel, Goldhahn, & Kathmann, 2017). As with OCD, the increased ERN in other anxiety disorders does not seem to vary with symptom reduction via treatment (Kujawa et al., 2016; Ladouceur et al., 2018) and is already evident in healthy at-risk participants (e.g., Riesel, Klawohn, et al., 2019). Moreover, increased ERN amplitudes have been shown to predict the development of anxiety symptoms (Lahat et al., 2014; Lamm et al., 2014; Meyer et al., 2015, 2018; Riesel et al., 2021) further highlighting the utility of the ERN as a neural risk marker in

clinical research (Hajcak et al., 2019). The uniform increase in ERN across several disorders has been assumed to stem from transdiagnostically shared psychological and neurobiological processes involved in the pathophysiology of OCD and anxiety disorders. Consistent with this, disorders characterized by hyperactive error monitoring are frequently comorbid (e.g., Kessler et al., 2008; Krueger & Markon, 2006; Ruscio et al., 2010) and share clinical features. Furthermore, recent research suggests common genetic risk factors between different disorders (Anttila et al., 2018) as well as common etiological factors, such as structural abnormalities in the ACC and disruptions in cognitive control (e.g., Goodkind et al., 2015; McTeague et al., 2017). Together, these results highlight the need to take a focus on transdiagnostic cognitive and neurobiological mechanisms underlying psychopathology, as proposed by the *Research Domain Criteria Initiative* (RDoC; Cuthbert, 2014; Insel et al., 2010; Kozak & Cuthbert, 2016). The ERN has been identified as a relevant dimension for RDoC-inspired research (Weinberg, Dieterich, & Riesel, 2015) and has been suggested a promising transdiagnostic neural risk marker for obsessive-compulsive and anxiety disorders (Riesel, Klawohn, et al., 2019).

Despite this substantial body of research on the ERN, there is still an ongoing debate about which specific psychological process, trait, or dimension is reflected in increased ERN amplitudes. A variety of traits and symptoms have been identified and discussed to be associated with ERN amplitude variation (see Cavanagh & Shackman, 2015; Weinberg, Dieterich, & Riesel, 2015). Relevant traits include behavioral inhibition (Lahat et al., 2014; McDermott et al., 2009), harm avoidance (Riesel, Klawohn, et al., 2019), perfectionism (Meyer & Wissemann, 2020; Perrone-McGovern et al., 2017; Stahl et al., 2015), impulsiveness (Ruchow et al., 2005; Taylor et al., 2018), threat or error sensitivity (Chong & Meyer, 2019; Weinberg et al., 2012), and neuroticism (Olivet & Hajcak, 2012). Furthermore, associations with measures of symptoms and states have been observed for worry and anxious apprehension (Hajcak et al., 2003; Moser et al., 2013; Zambrano-Vazquez & Allen, 2014), uncertainty (Cavanagh & Shackman, 2015), negative affect (Hajcak et al., 2004), checking symptoms (Weinberg, Kotov, & Proudfit, 2015) and subclinical OC symptoms (Gründler et al., 2009; Hajcak & Simons, 2002). The association of the ERN with worry or anxious apprehension received increased attention after a meta-analysis (Moser et al., 2013) which suggested that the ERN might be specifically related to measures of anxious apprehension ($r = -.35$), but not to other mixed anxiety measures ($r = -.09$). An association with anxious apprehension was further supported by a study that compared groups with symptom profiles of OCD, worry, and anxiety symptoms and linked increased ERN amplitudes specifically to worry (Zambrano-Vazquez & Allen, 2014). However, other

studies failed to replicate the association between anxious apprehension and ERN (Härpfer et al., 2020; Muir et al., 2020) and it has been suggested that the anxiety-ERN relationship might be stronger or even specific to clinical samples (Härpfer et al., 2022; Saunders & Inzlicht, 2020; Seow et al., 2020).

Moreover, most psychophysiological studies that examined the association of ERN with individual differences have focused on a single or a limited number of features and symptoms. Looking at the individual differences previously associated with ERN, they overlap considerably, and it is likely that they share a significant amount of variance in their association with ERN. This calls for an attempt to integrate ERN-associations across a variety of individual difference measures. Finally, in contrast to the abundance of studies concerning the ERN, the CRN and its associations to individual differences have often been disregarded and alterations in psychopathology have less consistently been reported (e.g., Endrass & Ullsperger, 2014). However, a more complete picture of the processes associated with heightened ERN and CRN is critical to improve our understanding of common etiological processes in anxiety and OCD and to enable targeted prevention and intervention approaches.

The present study examined the association between ERN and CRN and a relatively large set of traits and symptoms in a sample recruited across the spectrum of obsessive-compulsive characteristics ranging from low, medium, and high subclinical to clinical symptom severity. The sampling strategy ensured that we covered the full range of obsessive-compulsive symptoms to provide an optimal basis for examining the associations of ERN and CRN with individual differences and symptoms. We then explored the latent dimensions underlying the traits and symptoms studied. In a next step, we analyzed the associations of these dimensions with ERN and CRN. It should be noted that although the analyses, especially those on the ERN, are based on previous findings and we expect to observe a relationship between the ERN and measures of anxiety and worry, they are exploratory in nature and serve to provide new insights into the phenotype underlying ERN and CRN variations.

2 | METHOD

2.1 | Sample recruitment and clinical assessment

Participants ($N = 139$) were recruited across the spectrum of obsessive-compulsive symptom severity including one clinical and three subclinical groups. Patients with OCD were recruited from the outpatient clinic at Humboldt-Universität zu Berlin, where they were seeking

or receiving cognitive-behavioral therapy. Subclinical participants were recruited from a large screening sample of 1145 individuals recruited via locally targeted online advertisements to complete the Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al., 2002) online. Using pre-defined cut-offs for OCI-R total scores (Gonner et al., 2008), participants were recruited from the screening sample to form three groups: with low (OCI-R range 0 to 8), medium (OCI-R range 9 to 16), and high (OCI-R > 16) OC-characteristics. This recruitment strategy ensured to represent the full spectrum of OC severity in a balanced way and optimized the variance distribution, setting ideal conditions for dimensional analyses. However, it clearly did not lead to distinct groups and we thus analyzed the data dimensionally. After exclusion of participants with excessive EEG artifacts ($n = 13$), missing questionnaire data ($n = 3$), and less than six artifact free error trials ($n = 23$; Olvet & Hajcak, 2009), we retained a final sample of 100 participants, 26 patients with OCD, and 74 healthy comparison participants including 24 participants with low, 24 with medium, and 26 with high OC-characteristics, matched with regard to age, self-reported gender, and years of education. The mean number of included error trials was 20.05 ($SD = 13.76$) and did not differ between patients and healthy participants, $t(98) = 3.50$, $p = .27$. Table 1 shows demographical, clinical and electrophysiological measures for patients with OCD and healthy participants. In the data supplement accompanying this article, this information is also provided for the four groups we used for recruitment (Table S1, Figure S1). All participants were free of neurological disorders and had normal or corrected-to-normal vision. Presence of current or past psychiatric diagnoses was assessed in all participants using the Structural Clinical Interview for DSM-IV (SCID). Exclusion criteria were a lifetime diagnosis of any psychotic or substance use disorder, neuroleptic medication or use of benzodiazepines in the previous 4 weeks. In addition, healthy comparison participants were free of any current or past psychiatric disorders.

Thirteen participants with OCD had one or more current comorbid diagnoses, namely major depressive disorder ($n = 8$), panic disorder ($n = 2$), social anxiety disorder ($n = 2$), generalized anxiety disorder ($n = 1$), and somatic symptom disorder ($n = 1$). Twelve patients were currently medicated with either selective serotonin reuptake inhibitors (SSRI; $n = 11$) or tricyclic antidepressants ($n = 1$). The study was approved by the local institutional review board and conducted in accordance with the ethical guidelines of the declaration of Helsinki. Written informed consent was obtained from all participants before data collection. Note, the current study was part of a two-day study protocol that included several EEG paradigms and results of

TABLE 1 Group-specific means and standard deviations of demographical, questionnaire, and ERP data

	HC (<i>n</i> = 74)			OCD (<i>n</i> = 26)			Group comparison	
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>F</i> / χ^2	<i>p</i>
Demographics								
Reported Gender (f/m)	37/35	–	–	13/14	–	–	.082	.774
Age (years)	67	30.76	8.28	26	30.81	7.49	–0.02	.980
Questionnaires								
BIS	67	70.73	9.46	26	73.38	7.62	–1.28	.205
BDI-II	74	5.85	6.81	26	13.65	9.19	–3.96	<.001
BIS/BAS inhibition	67	19.18	2.83	26	22.35	2.86	–4.83	<.001
BIS/BAS activation	67	40.09	4.64	26	38.92	5.12	1.06	.293
FMPS perfection	67	73.33	17.83	26	86.69	16.99	–3.29	.001
NEO-FFI conscient.	69	31.71	7.55	25	31.32	7.06	0.23	.822
NEO-FFI neurot.	69	17.87	7.49	25	29.16	8.35	–6.26	<.001
OCI-R	74	11.04	9.39	26	27.58	12.90	–6.00	<.001
PSWQ	69	41.03	10.77	26	58.19	13.64	–5.77	<.001
PANAS positive	67	31.48	6.67	26	26.88	7.30	2.90	.005
PANAS negative	67	13.31	4.44	26	21.81	8.21	–5.00	<.001
RS-13	69	71.38	9.29	26	58.31	12.53	4.84	<.001
STAI-T	73	38.29	9.49	26	51.38	11.78	–5.66	<.001
YBOCS	28	8.11	8.98	26	22.08	5.38	–6.99	<.001
ERPs								
ERN (μ V)	74	–4.58	3.75	26	–7.21	4.37	2.94	.004
CRN (μ V)	74	0.63	3.07	26	0.08	2.43	0.82	.411

Note: Group comparisons for ERN and CRN report results of an univariate ANOVA for ERN and CRN separately, including group (patients with OCD vs. healthy participants) as between-subjects factor; *p* < .05 are printed in bold.

Abbreviations: BDI-II, Beck Depression Inventory–II; BIS, Barratt Impulsiveness Scale; BIS/BAS, behavioral inhibition and activation system; CRN, correct-response negativity; ERN, error-related negativity; ERP, event-related potential; FMPS, frost multidimensional perfectionism scale; Gender (f, female; m, male); NEO-FFI, NEO Five-Factor Inventory (conscient., conscientiousness scale; neurot., neuroticism scale); OCI-R, obsessive–compulsive inventory-revised; PANAS, positive and negative affect schedule; PSWQ, Penn State Worry Questionnaire; RS-13, resilience scale; STAI-T, Spielberger State–trait anxiety inventory (trait anxiety scale); YBOCS, Yale-Brown obsessive compulsive scale.

partly overlapping subsamples have been published elsewhere (Klawohn, Hajcak, et al., 2020; Riesel, Kathmann, & Klawohn, 2019).

2.2 | Measures

Based on previous findings of associations with the ERN, a broad battery of questionnaires was assessed (Table 2). Symptom severity in patients with OCD was evaluated using the Yale-Brown Obsessive Compulsive Scale (YBOCS; Goodman et al., 1989; Hand & Büttner-Westphal, 1991).

2.3 | Task and procedure

Participants were seated in a dimly lit, electrically shielded room. They completed a flanker task presented using

Presentation software (Neurobehavioral Systems, Albany, California, USA) on a 19-inch LCD monitor (resolution 1280 × 1024 pixels, refresh rate 60 Hz, viewing distance approx. 65 cm). On each trial, five vertically aligned arrows were presented (i.e., one target, four flankers; set size 2.5° × 2.5° visual angle) and participants were asked to respond with their right or left index finger to the direction of the central arrow using zero-buffer response buttons. Instruction emphasized to respond as accurately and quickly as possible. Half of the stimuli were compatible (i.e., all arrows pointing in the same direction), the other half incompatible (i.e., flankers pointing in the opposite direction), presented in pseudo-randomized order. Stimuli were presented for 100 ms, followed by a 1000 ms response window and an ITI (random variation between 200 and 1200 ms) during which a fixation cross was presented. The task encompassed 480 trials (with additional 20 practice trials), administered in 6 blocks with short breaks in between.

TABLE 2 Battery of questionnaires utilized in the present study

Symptom or trait	Questionnaire	References	Items	Scale	Cronbach's α
Impulsiveness	Barratt Impulsiveness Scale (BIS-10)	Patton et al. (1995), Preuss et al. (2008)	34	1–4	.73
Depressive symptoms	Beck Depression Inventory–II (BDI-II)	Beck et al. (1996), Kühner et al. (2007)	21	0–3	.92
Behavioral inhibition	Behavioral Inhibition System and Behavioral Activation Systems Scales: Inhibition (BIS)	Carver and White (1994), Strobel et al. (2001)	7	1–4	.63
Behavioral activation	Behavioral Inhibition System and Behavioral Activation Systems Scales: Activation (BAS)	Carver and White (1994), Strobel et al. (2001)	13	1–4	.77
Perfectionism	Frost Multidimensional Perfectionism Scale (FMPS)	Stöber (1998)	35	1–5	.90
Conscientiousness	NEO Five-Factor Inventory (NEO-FFI): Conscientiousness	Borkenau and Ostendorf (2008), Costa and McCrae (1992)	12	1–5	.87
Neuroticism	NEO Five-Factor Inventory (NEO-FFI): Neuroticism	Borkenau and Ostendorf (2008), Costa and McCrae (1992)	12	1–5	.90
Obsessive–compulsive symptoms	Obsessive–Compulsive Inventory–Revised (OCI-R)	Foa et al. (2002), Gönner et al. (2008)	18	0–4	.91
worry	Penn State Worry Questionnaire (PSWQ)	Glöckner-Rist and Rist (2014), Meyer et al. (1990)	16	1–5	.95
Positive affect	Positive and Negative Affect Schedule (PANAS): Positive Affect	Breyer and Bluemke (2016), Watson et al. (1988)	10	1–5	.79
Negative affect	Positive and Negative Affect Schedule (PANAS): Negative Affect	reyer and Bluemke (2016), Watson et al. (1988)	10	1–5	.81
Resilience	Resilience Scale (RS-13)	Schumacher et al. (2005), Wagnild and Young (1993)	13	1–7	.89
Trait anxiety	Spielberger State–Trait Anxiety Inventory (STAI): Trait	Laux et al. (1981), Spielberger et al. (1970)	20	1–4	.95

Note: Scale refers to number of steps on respective Likert scale; Cronbach's α calculated from the present sample.

2.4 | Psychophysiological recording, data reduction, and statistical analysis

The continuous EEG was recorded from 64 Ag/AgCl-sintered electrodes using an electrode cap with equidistant electrode locations (EASYCAP GmbH, Hersching, Germany). External electrodes were mounted below the eyes, on the nasion and the cheek (ground), Cz served as recording reference. All impedances were below 5 k Ω . The EEG was digitized at a sampling rate of 1000 Hz and amplified with a band pass filter of 0.01–250 Hz. Brain Vision Analyzer hard- and software (Brain Products, Gilching, Germany) were used for recording and analysis. Off-line, data were filtered with a bandpass from .1 to 30 Hz (4th order Butterworth) and re-referenced to an average reference. Response locked epochs of 1500 ms including a 500 ms pre-response interval were extracted. Eye movements were corrected using the Gratton and Coles procedure (Gratton et al., 1983). Artifacts were automatically excluded using the following criteria: voltage step >50 μ V between consecutive datapoints, absolute voltage difference >200 μ V within a segment, or low activity <0.5 μ V over 100 ms. After averaging the segments separately for correct and incorrect responses, baseline correction was applied using the –500 to –300 ms pre-response interval. ERN and CRN were quantified as the mean amplitudes from 0 to 100 ms at electrode FCz (Klawohn, Meyer, et al., 2020).

Statistical analyses were conducted using SPSS, version 25.0 (IBM, Armonk, NY). Univariate ANOVAs or Chi-Square tests were applied to examine differences in error rates, post-error-slowing, demographical, and clinical variables. Response-locked ERPs and response times were analyzed using mixed measures ANOVA, including the within-subjects factor response type (correct, error) and the between-subjects factor group (healthy controls and patients with OCD). The significance level was $\alpha = .05$, two-tailed. Regression analyses with bootstrapping (1000 samples) were used to examine the impact of OC-symptom severity on ERN and CRN, respectively while controlling for the influence of age and gender, 95% confidence intervals for the regression coefficients will be reported. In a next model, we included the interaction between OCI-R and gender. In the supplemental Table S2 the correlations (Pearson's r) between ERN and CRN with all clinical and trait measures and the associations between traits and symptoms are shown. Reliability estimates for all measures were calculated, including Spearman-Brown corrected split-half reliability for ERP data and Cronbach's alpha for questionnaire data. Reliability estimates for questionnaire data are presented in Table 2. Questionnaire data were integrated using exploratory principal axis factor analysis to examine underlying latent dimensions. The

exploratory principal axis factoring was conducted with all subscales of the measures used. The Kaiser–Meyer–Olkin measure confirmed the sample adequacy for this analysis, $KMO = .81$. This is similarly reflected in a significant Bartlett's test of sphericity ($\chi^2(253) = 1675.85$, $p < .001$) that supports that the correlation matrix diverges significantly from the identity matrix supporting suitability of the data set for dimension reduction. Factors were extracted based on visual inspection of the screeplot and the Kaiser's criterion (eigenvalue >1). The screeplot is presented in the data supplement in Figure S2. A Promax rotation was performed to allow correlations between the resulting factors in order to realistically depict the complex relationships of the traits and symptoms under study. Regression-based factor scores were used to extract composite scores across all questionnaires. These composite scores were then used for regression analyses. Again, age and gender were included to control for their influence. In addition to the derived factor scores, we also included ERN or CRN in the regression model to examine the specificity of the associations for ERN and CRN, respectively, and to correct for their shared variance.

3 | RESULTS

3.1 | Demographical and clinical characteristics

Means and standard deviations for demographic, clinical, and ERP data for patients with OCD and healthy participants, including group comparisons are displayed in Table 1. Consistent with matching procedures, groups did not differ in terms of gender, age, or years of education. As expected, OC symptom severity differed between groups. Groups also differed with respect to the other clinical measures such as depressive symptoms, behavioral inhibition, perfectionism, neuroticism, positive and negative affect, worry, resilience, and trait anxiety. No group differences were found for impulsiveness, behavioral activation, or conscientiousness.

3.2 | Event-related potentials

3.2.1 | Group comparisons

Grand average waveforms at electrode FCz and topographies are shown in Figure 1. Both ERN and CRN showed excellent reliability (i.e., Spearman-Brown corrected split-half reliability: ERN = .85, CRN = .99). As expected, errors elicited a stronger negativity compared to correct responses ($F(1, 98) = 210.746$, $p < .001$, $\eta_p^2 = 0.683$). A main

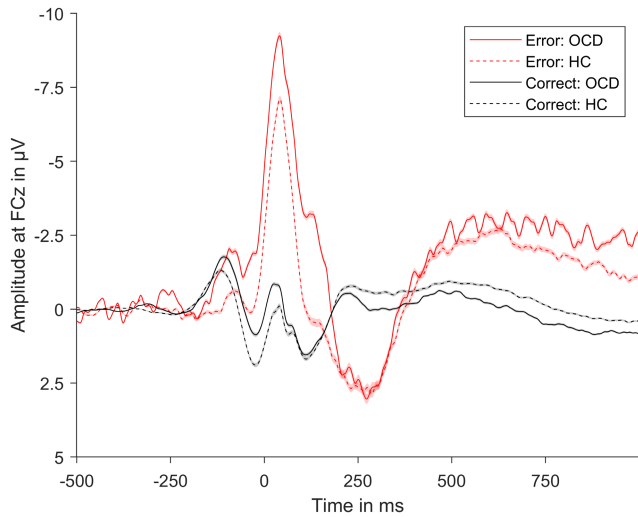


FIGURE 1 Grand average waveforms of the error-related negativity (ERN, red) and correct-response negativity (CRN, black) in patients with OCD (solid line) and healthy participants (dotted line). Error area around the ERP curve indicates one standard error of the mean at each timepoint.

effect of group was present ($F(1, 98) = 5.801, p = .018, \eta^2_p = 0.056$), further specified by a significant interaction between group and response ($F(1, 98) = 5.803, p = .018, \eta^2_p = 0.056$). Patients with OCD showed a larger ERN ($t(98) = 2.94, p = .004$) but did not differ in CRN amplitude ($t(98) = 0.83, p = .41$) when compared to all healthy participants.

3.2.2 | Dimensional analyses

Results of the regression analysis examining the impact of OC-symptom severity on ERN and CRN while accounting for age and gender are shown in Table 3. Confirming the group comparisons, dimensional analyses for ERN and CRN show that OCI-R significantly predicts ERP variations, with higher OC-symptom severity being associated with larger (i.e., more negative) ERN and CRN magnitude. Gender did not moderate this relationship. Correlations between ERN and CRN to all symptom and trait measures are shown in the supplemental material in Table S2. The correlation patterns descriptively show that the ERN was negatively correlated with many traits and symptom measures, especially in the domain of anxiety and negative affect, while the CRN was specifically associated with higher OC symptom severity. High intercorrelations between the used symptom and trait measures further indicate a large degree of shared variance and underline the suitability of using symptom reduction methods. The exploratory principal axis analysis yielded six factors, extracted based on visual inspection of the

TABLE 3 Results of regression analyses using gender, age, and obsessive-compulsive symptom severity to predict CRN and ERN

Variable	CRN					ERN								
	b	SE	p	Lower 95% CI	Upper 95% CI	F	R ²	b	SE	p	Lower 95% CI	Upper 95% CI	F	R ²
<i>Basic model</i>														
Gender	-.25	.62	.704	-1.41	0.99	2.13	.067	.33	.80	.662	-1.21	1.94	4.70*	.137
Age	-.02	.03	.593	-0.08	0.05			.13	.05	.007	0.04	0.24		
OCI-R	-.06	.02	.008	-0.10	-0.02			-.09	.03	.002	-0.14	-0.04		
<i>Interaction model</i>														
Gender	-.25	.61	.675	-1.43	0.98	1.58	.067	.32	.79	.675	-1.29	1.82	3.52*	.138
Age	-.02	.03	.573	-0.08	0.05			.13	.05	.009	0.05	0.23		
OCI-R	-.06	.08	.443	-0.21	0.08			-.06	.09	.489	-0.23	0.11		
OCI-R × Gender	<0.1	.04	.998	-0.08	0.09			-.02	.06	.704	-0.13	0.08		

Note: Error-related negativity (ERN) and correct-response negativity (CRN) quantified as mean amplitude (0–100 ms) at electrode FCz; gender (0 = female, 1 = male); age in years. $N = 101$.

Abbreviation: OCI-R, obsessive-compulsive inventory-revised (mean centered).

*Total model $p < .005$.

Bold indicates significance level at $p < .05$

TABLE 4 Results of regression analyses to examine the associations between the derived latent dimensions and the ERN and CRN, controlling for age, gender and the respective other ERP component

Variable	<i>b</i>	<i>SE</i>	<i>p</i>	Lower 95% CI	Upper 95% CI	<i>F</i>	<i>R</i> ²
<i>CRN</i>						4.15*	.310
Gender	−0.44	0.58	.462	−1.52	0.70		
Age	−0.07	0.03	.055	−0.13	<0.01		
ERN	0.34	0.07	<.001	0.19	0.48		
Factor 1 anxious misery	0.53	0.37	.148	−0.21	1.22		
Factor 2 conscientiousness	−0.45	0.33	.176	−1.14	0.18		
Factor 3 compulsivity	−0.94	0.36	.012	−1.69	−0.30		
Factor 4 positive affect	−0.13	0.38	.749	−0.91	0.59		
Factor 5 fun seeking	0.03	0.35	.925	−0.65	0.75		
Factor 6 standards	−0.65	0.31	.034	−1.20	0.04		
<i>ERN</i>						5.00*	.354
Gender	0.07	0.75	.942	−1.46	1.39		
Age	0.13	0.05	.004	0.04	0.22		
CRN	0.63	0.15	<.001	0.32	0.92		
Factor 1 anxious misery	−1.66	0.61	.004	−2.95	−0.60		
Factor 2 conscientiousness	0.07	0.47	.859	−0.80	1.12		
Factor 3 compulsivity	0.82	0.50	.103	−0.08	1.88		
Factor 4 positive affect	−0.08	0.46	.872	−1.02	0.76		
Factor 5 fun seeking	0.37	0.44	.412	−0.52	1.20		
Factor 6 standards	0.20	0.53	.724	−0.84	1.27		

Note: Error-related negativity (ERN) and correct-response negativity (CRN) quantified as mean amplitude (0–100 ms) at electrode FCz; gender (0 = female, 1 = male); age in years. *N* = 101.

Abbreviations: CI, confidence interval, OCI-R, obsessive–compulsive inventory-revised (mean centered).

**p* < .005.

Bold indicates significance level at *p* < .05

screplot and the Kaiser's criterion (eigenvalue >1). The factor loadings are presented in Table 5 and the screplot is presented in Figure S2 in the supplement. Factor 1 primarily represents anxiety and negative affect and will be referred to as the anxious misery factor. The highest loadings were observed for OCI-R, STAI trait anxiety, PSWQ and NEO-FFI Neuroticism. Factor 2, called conscientiousness, showed the highest loadings in subscales focusing on conscientiousness and was negatively related to attentional impulsiveness. The OCI-R subscales except for the obsessing and washing subscale showed high loadings on Factor 3, which thus seemed closely related to compulsivity and behavioral OCD symptoms and was named compulsivity. Factor 4 predominantly reflected positive affect measures and resilience and was called positive affect. Variance in fun seeking and motor impulsiveness is captured in Factor 5, labeled fun seeking. Finally, on Factor 6 the highest loadings were obtained for Personal Standards and Parental Expectations, and the factor was labeled standards. In a next step, regression analyses

(Table 4) were used to examine the associations between the derived latent dimensions and the ERN and CRN, controlling for age, gender and the respective other ERP component. For the ERN we observed a significant association to age, CRN and the anxious misery factor. For the CRN model, the ERN, the compulsivity, and the personal standards factors emerged as significant predictors. Note that these associations hold after correction for the shared variance between ERN and CRN, which supports the specificity of the observed associations. The respective scatterplots are shown in Figure 2.

4 | DISCUSSION

Although increased ERN amplitude has repeatedly been considered as a neural correlate and risk marker for obsessive–compulsive disorder and anxiety (e.g., Riesel, Klawohn, et al., 2019), the specific psychological correlate of ERN variation is still debated. The present article

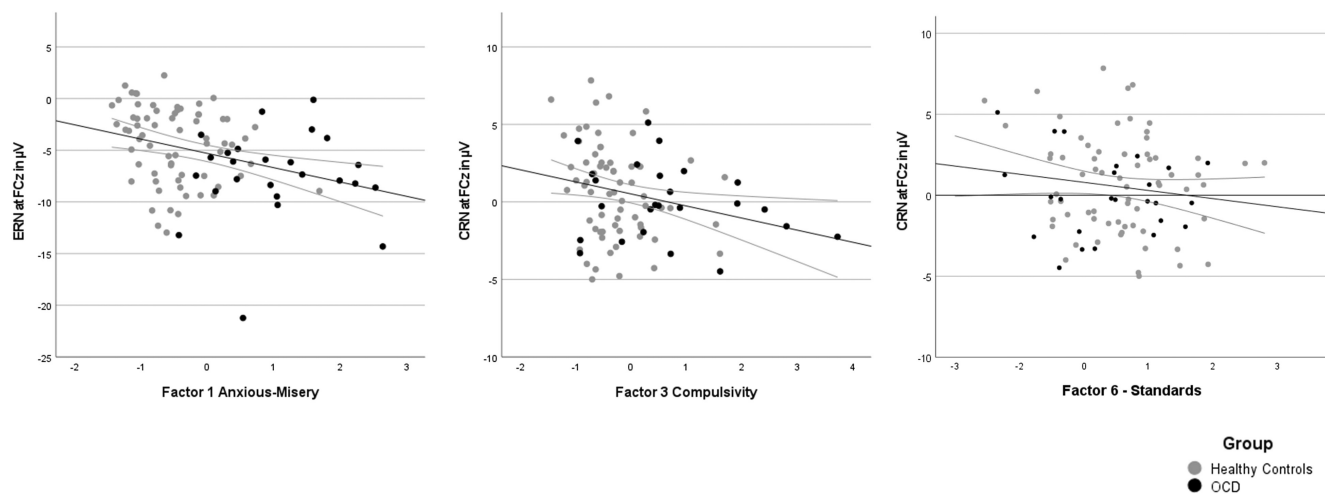


FIGURE 2 Scatterplots depicting associations between the ERN (error-related negativity) and Factor 1—Anxious-Misery (left), between CRN (correct-response negativity) and Factor 3—Compulsivity (middle), and between CRN (correct-response negativity) and Factor 6—Standards (right) in patients with OCD (black) and healthy participants (gray).

examined associations between the ERN and CRN and various transdiagnostic traits and symptoms in a large sample ($N = 101$) recruited across the whole spectrum of OC symptom severity, including 26 patients with OCD. Key findings were: (1) a replication of increased ERN amplitudes in patients with OCD compared to healthy individuals; (2) a negative association between ERN and CRN with OC symptom severity; (3) using factor analysis to explore the associations between ERN and CRN and underlying symptom dimensions, we showed that ERN was specifically associated with anxious misery symptoms, while tCRN variations were related to compulsivity and personal standards.

The present results confirmed that OCD patients were characterized by increased ERN amplitudes, adding to a growing number of studies that collectively support that increased neural error signals can robustly be found in patients with OCD (Riesel, 2019). This result is complemented by dimensional analyses, which also show an association between OC symptom severity and ERN, extending the results and showing a similar association for CRN. Beyond clinical variation in the ERN, a main objective of the present study was to examine associations to transdiagnostic traits and symptoms to further clarify which psychological phenotypes, if any, might best explain ERN variations observed across a range of anxiety-related traits and symptoms. Notably, many of these previously proposed transdiagnostic phenotypes can be assumed to overlap considerably, and the substantial intercorrelations between several measures observed in the current study support this assumption. Moreover, these overlapping phenotypes also share variance in explaining ERN variations—a fact possibly often overlooked since simultaneous associations have not been explored systematically.

The results of the present study support an association of the ERN with a broad phenotypic factor composed of several traits and symptoms from the anxiety-neuroticism spectrum reflecting anxious-misery.

Higher scores in this composite measure were moderately related to more negative ERN amplitudes, while correcting for age gender and variance shared with the CRN. This supports the notion that not *one* specific symptom or trait of anxiety is associated with increased ERN amplitudes but rather a broader, shared latent dimension reflecting several anxiety and negative emotionality measures. This corresponds well to results pointing to a transdiagnostic increase in ERN amplitudes across anxiety disorders and OCD (Riesel, Klawohn, et al., 2019). Moreover, it is in line with results suggesting that variation in psychopathology can be related to a limited number of dimensions, suggesting that negative emotionality represents a core vulnerability factor for psychopathology (Caspi et al., 2014; Kotov et al., 2017; Van den Bergh et al., 2021). Our results suggest that individuals high on an underlying anxious-misery trait are likely to show an increased neural response to errors, which may indicate increased alertness to potential harm and negative outcomes (Proudfit et al., 2013), irrespective of the specific phenotypic appearance of the anxiety-proneness. This relatively broad anxious-misery composite trait could also be seen as a measure of general distress. Thus, an increased ERN might render individuals more prone to experience higher distress in general, across specific phenotypes and symptoms. This notion is well in line with recent findings showing an association of the ERN with stress reactivity and increased risk for symptom increase under conditions of stress (Riesel et al., 2021; Weinberg et al., 2022). Together with results supporting that

TABLE 5 Factor loadings of the exploratory factor analysis

	Factor					
	1 Anxious-Misery	2 Conscientiousness	3 Compulsivity	4 Positive Affect	5 Fun seeking	6 Standards
<i>Measure</i>						
OCI-R obsessions	1.039					
STAI trait anxiety	.847					
PSWQ	.830					
NEO-FFI neuroticism	.818					
BIS attentional impulsiveness	.751					
BDI-2	.641					
PANAS negative affect	.636					
BIS/BAS inhibition	.611					
OCI-R obsessing	.585		.404			
FMPS concern over mistakes, doubts about actions	.539					.463
OCI-R washing	.515					
FMPS organization		.896				
NEO-FFI conscientiousness		.725				
BIS nonplanning impulsiveness		-.645				
OCI-R hoarding		-.424	.409			
OCI-R neutralizing			.866			
OCI-R ordering			.730			
BIS/BAS activation reward responsiveness				.637		
PANAS positive affect	-.507			.535		
BIS/BAS activation drive		.491		.531		
RS13 resilience	-.503			.510		
FMPS personal standards						.604
FMPS parental expectations and criticism						.422
BIS/BAS activation fun seeking					.655	
BIS motor impulsiveness					.575	

Note: Only Factor loading above .40 are shown; associations to ERPs as Pearson's *r*.

Abbreviations: BDI-II, Beck Depression Inventory–II; BIS, Barratt Impulsiveness Scale; BIS/BAS, behavioral inhibition and activation system; FMPS, frost multidimensional perfectionism scale; NEO-FFI, NEO Five-Factor Inventory; OCI-R, obsessive–compulsive inventory-revised; PANAS, positive and negative affect schedule; PSWQ, Penn State Worry Questionnaire; RS-13, resilience scale; STAI-T, Spielberger State–trait anxiety inventory.

increased ERN amplitudes precede development of OCD and anxiety symptoms (Meyer et al., 2015, 2018; Riesel, Klawohn, et al., 2019), this may suggest that increased ERN amplitudes represent a rather unspecific neural risk marker related to an increased risk to react to stressors with distress and for the development of internalizing psychoathology in general. The specific clinical outcome and trajectory to symptoms could then be shaped by additional genetic and environmental factors contributing to increased risk or resilience. This interpretation

can be complemented by a current conceptualization that describes dispositional negativity as a common vulnerability factor for internalizing psychopathology using a predictive-coding framework (Van den Bergh et al., 2021). Specifically, the authors argue that individuals with higher negative emotionality are characterized by an information processing style that follows a *better safe than sorry* rational and leads to an oversimplified input, allowing a greater speed in categorizing input as threat at the expense of detail and, in the long run, also

resulting in poor updating of prior beliefs (Van den Bergh et al., 2021). This framework can account for a variety of different symptoms and neurocognitive alterations, including increased neural error signals as a neuronal equivalent of a low-threshold alarm system (Proudfit et al., 2013) as a common vulnerability factor for internalizing psychopathology (Pasion & Barbosa, 2019).

In addition to OCD-group specific increases in ERN amplitude, CRN amplitudes were associated with OC symptom severity. An amplification in monitoring after correct responses has been reported before in OCD (e.g., Endrass & Ullsperger, 2014; Klawohn et al., 2014; Riesel et al., 2011; Riesel, Klawohn, et al., 2019), whereas the CRN was previously found unaltered in most other anxiety disorders and depression (Endrass & Ullsperger, 2014; Riesel, Goldhahn, & Kathmann, 2017; Weinberg, Kotov, & Proudfit, 2015). Thus, variation in the CRN might be associated with phenotypic features more specific to OCD, a notion that is also supported by the current factor analysis showing that the CRN was related to the compulsivity and personal standards factors, both of which represent central characteristics of OCD psychopathology. In line with this current finding, an increase in CRN has previously been linked to a general increase in performance monitoring irrespective of response type (Klawohn et al., 2014) and together with studies showing increased neural activity in low-conflict trials (Riesel, Klawohn, et al., 2017) and impaired flexibility of performance monitoring in OCD (Endrass et al., 2010; Riesel, Kathmann, & Klawohn, 2019) these findings may indicate that OCD is characterized by overactive monitoring that seems to operate more independently of the actual performance monitoring demands. In this view, increased CRN amplitudes in OCD might be linked to a more conservative response strategy and doubt, which triggers compulsive behavior. Collectively, these results suggest that increases in CRN amplitudes may be more closely and specifically related to OC symptoms and might ultimately be useful to differentiate OCD and anxiety.

The present results should be considered in consideration of several limitations. Results of the factor analysis as well as derived associations with ERN and CRN need replication in independent samples. Moreover, it should be pointed out that the selection of self-report measures was based on a narrative literature review at the time of study implementation, but not a systematic review. Thus, the selection of measures is not exhaustive, other relevant phenotypes and symptoms might have been included. Finally, the study design and sample recruitment were optimized to disentangle the effects of often overlapping phenotypes on error processing in the context of variations in OC symptoms. While we think that this allows for better

insight into some associations between OCD and anxiety related traits and symptoms by ensuring adequate variability in self-reported characteristics, it might bias results towards such traits related to the OCD/anxiety-spectrum. In addition, the comorbidity and medication rates in our clinical sample are comparatively low, likely due to the admission restrictions of the outpatient clinic through which the patients were recruited which may exclude particularly severely distressed and comorbid patients. However, the low medication rates also reflect guideline recommendations in Germany, where the recommended first line treatment of OCD is cognitive behavioral therapy including exposure and response prevention. For future studies in this context, naturalistic and truly transdiagnostic samples across the spectrum of relevant disorders and severity levels are needed.

In summary, the results of the present study confirm that OCD is characterized by increased neural activity in error and correct trials. Moreover, adopting a dimensional analytic approach results showed that increases in the ERN were associated with higher severity in several anxiety and neuroticism associated traits, which were represented by an underlying rather broad anxious-misery dimension. These results suggest that altered ERN amplitudes are related to a broad anxiety-neuroticism dimension relevant for a range of disorders and their development, including OCD and anxiety. In contrast, the negativity after correct responses was related to dimensions of compulsivity and high standards, both striking as more closely related to OCD symptomatology. Taken together, the current study identified different trait/symptom dimensions associated with variation in ERN and CRN, respectively, thus extending our knowledge of the differential functionalities of both ERPs and their involvement in psychopathology. While elevated ERN amplitudes may indicate a low-threshold alarm signal, in line with a “better safe than sorry” approach shared by individuals with anxiety and OCD, CRN alterations may be more specifically linked to OCD-specific cognitions and behavior.

AUTHOR CONTRIBUTIONS

Anja Riesel: Conceptualization; data curation; formal analysis; investigation; methodology; writing – original draft; writing – review and editing. **Kai Härpfer:** Formal analysis; visualization; writing – original draft. **Lars Thoma:** Data curation; formal analysis; project administration. **Norbert Kathmann:** Funding acquisition; investigation; resources; supervision; writing – review and editing. **Julia Klawohn:** Conceptualization; data curation; formal analysis; funding acquisition; investigation; project administration; supervision; writing – original draft; writing – review and editing.

ACKNOWLEDGEMENTS

The authors thank Dr. Benedikt Reuter and Dr. Eva Kischkel for clinical supervision, Rainer Kniesche and Thomas Pinkpank for lab and technical support, and Ulrike Bunzenthall, Sarah Dressel, and Verena Wüllhorst for their assistance in data acquisition. Conflicts of interests: none to declare. Open Access funding enabled and organized by Projekt DEAL.

FUNDING INFORMATION

This research was supported by the German Research Foundation (DFG; Grant KA 815/7-1 awarded to N. Kathmann)

DATA AVAILABILITY STATEMENT


Data are available from the authors upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

TABLE S1 Group specific means and standard deviations of demographical, questionnaire, and ERP data

TABLE S2 Correlation matrix for associations between questionnaire and ERP data. Reliability measures are presented in the diagonal. Reported *p* values are not corrected for multiple comparisons.

FIGURE S1 Grand average waveforms of the error-related negativity (ERN, left) and correct-response negativity

(CRN, right) in patients with OCD (red) and healthy participants with varying subclinical OC symptom severity (from gray to black). Error area around the ERP curve indicates one standard error of the mean at each timepoint.

FIGURE S2 Screeplot of the exploratory principal axis analysis

How to cite this article: Riesel, A., Härpfer, K., Thoma, L., Kathmann, N., & Klawohn, J. (2023). Associations of neural error-processing with symptoms and traits in a dimensional sample recruited across the obsessive–compulsive spectrum. *Psychophysiology*, 60, e14164. <https://doi.org/10.1111/psyp.14164>