Understanding the Error in Psychopathology:
Notable Intraindividual Differences in Neural Variability of Performance Monitoring

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Abstract

Background: Abnormal performance monitoring is a possible transdiagnostic marker of psychopathology. Research on neural indices of performance monitoring, including the error-related negativity (ERN), typically examines group and interindividual (between-person) differences in mean/average scores. Intraindividual (within-person) variability in activity captures the capacity to dynamically adjust from moment to moment, and excessive variability appears maladaptive. Intraindividual variability in ERN represents a unique and largely unexamined dimension that might impact functioning. We tested whether psychopathology group differences (major depressive disorder [MDD], generalized anxiety disorder [GAD], obsessive-compulsive disorder [OCD]) or corresponding psychiatric symptoms account for intraindividual variability in single-trial ERN scores.

Methods: High-density electroencephalogram (Electrical Geodesics, Inc.) was recorded during a semantic flanker task in 51 participants with MDD, 44 participants with GAD, 31 participants with OCD, and 56 psychiatrically-healthy participants. Mean ERN amplitude was scored 0-125ms following participant response across four fronto-central sites. Multilevel location-scale models were used to simultaneously examine interindividual and intraindividual differences in ERN.

Results: Analyses indicated considerable intraindividual variability in ERN that was common across groups. However, we did not find strong evidence to support relationships between ERN and psychopathology groups or transdiagnostic symptoms.

Conclusions: These findings point to important methodological implications for studies of performance monitoring in healthy and clinical populations—the common assumption of fixed intraindividual variability (i.e., residual variance) may be inappropriate for ERN studies.

Implementation of multilevel location-scale models in future research can leverage between-person differences in intraindividual variability in performance monitoring to gain a rich understanding of trial-to-trial performance monitoring dynamics.

Keywords: performance monitoring; individual differences; event-related potentials (ERPs); error-related negativity (ERN); within-person variability; ERP psychometric reliability

Introduction

The ability to accurately monitor and adapt performance is critical for healthy functioning. Impairment in performance monitoring is observed across various forms of psychopathology and contributes to poor functional outcome (1, 2). Basic and applied researchers have focused on differences in mean/average scores between conditions, persons, and groups and have typically treated response variability within persons as “noise”. However, neural variability can capture the capacity to dynamically adjust from moment to moment within a person (i.e., intraindividual variability; 3, 4), and failing to consider within-person variability might miss important differences in performance monitoring that contribute to maladaptive behaviors. Therefore, the present study tested whether distinct patterns of intraindividual variability in neural measures of performance monitoring are observed in people with psychopathology.

Neural activity in spatial and temporal domains is variable across neuroimaging modalities (3), and excessive neural variability appears maladaptive (5, 6). For example, participants with autism spectrum disorders exhibit excessive neural variability despite intact mean responses and excessive neural variability might contribute to an unstable perception of social and emotional information (7-9). Variability in blood-oxygenation level-dependent (BOLD) signal is larger in patients with schizophrenia than in healthy controls, which could indicate disrupted neuronal communication, and variability in BOLD signal distinguishes patients with schizophrenia from those with bipolar disorder (10). Variability of responses in the auditory midbrain also distinguished people with schizophrenia from healthy controls (11), and these responses related to speech discriminability in the context of pro-cognitive therapeutics

(12). Findings suggest converging evidence for testing clinical differences in neural variability to understand the role that disruption in biological mechanisms play in psychopathology.

The error-related negativity (ERN) is a scalp-recorded event-related brain potential (ERP) that represents an index of performance monitoring, and ERN is abnormal in multiple psychopathology groups (13-15). Prominent explanations of ERN suggest it represents conflict between the mental representation of a correct response and an observed error response (13, 16, 17) or an emotional response to errors that are viewed as aversive (18, 19). ERN studies in psychopathology provide some support for its use as a biomarker for anxiety (20, 21), depression (22, 23), and obsessive-compulsive disorder (OCD; 24, 25), but it remains unclear whether differences in intraindividual variability of ERN might be uniquely related to traditional diagnostic categories or psychiatric symptoms. Intraindividual variability in ERN is often treated as noise (26), but the present study focused on intraindividual variability as a signal of interest.

The purpose of the present study was to use single-trial ERN amplitudes to determine whether psychopathology group differences account for intraindividual variability in performance monitoring. Based on findings of excessive neural variability in other clinical disorders (5, 6), we predicted that intraindividual/within-person variability in ERN scores would be higher in three psychopathology groups (generalized anxiety disorder [GAD], major depressive disorder [MDD], and OCD) than in healthy controls and that this relationship would be observed independent of group differences in mean ERN scores. Given the heterogeneous nature of psychopathological presentations (27, 28), relationships between measures of psychiatric symptoms and ERN were examined.

Methods and Materials

Participants

The Brigham Young University Institutional Review Board approved all study procedures, and participants provided written informed consent. Participants with MDD, GAD, and OCD were recruited via flyers and referrals from university and community mental health centers. All psychopathology participants self-reported a primary diagnosis of MDD, GAD, or OCD made by a psychiatrist, psychologist, or physician. Healthy control participants were recruited from undergraduate courses and advertisements. Exclusion criteria for all participants included medication changes in the last two months, neuroleptic medications, a psychotic or bipolar disorder, a history of any substance use disorder of any severity, self-reported learning disabilities or attention deficit/hyperactivity disorder, neurological disease, left handedness, and a status as a minor (age < 18 years old). Healthy controls were excluded if they self-reported any psychiatric diagnosis. Final study enrollment included 182 participants (see Table 1).

We primarily aimed to test an ecologically valid sample presenting from the community with self-reported diagnoses. We also sought to ensure the pattern of results would be similar in a group with confirmed diagnoses. Thus, psychiatric diagnoses were confirmed using the Mini-International Neuropsychiatric Inventory (MINI; 29), which was administered to all participants by clinical psychology students trained and supervised by a licensed clinical psychologist (MJL). The MINI screener was given first and modules endorsed on the screener were subsequently administered. The MINI was chosen because of its good concordance with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) and with diagnoses made by physicians and psychiatrists (kappas range from .31 to .86), but the MINI requires less time to administer than the SCID (29-33). Summary information for the subset of participants with MINI-confirmed diagnoses (GAD $n = 18$, MDD $n = 20$, OCD $n = 15$) is provided in Supplementary Table 1.

Clinical Ratings

Participants completed the Beck Depression Inventory, 2nd Edition (BDI-II; 34) as a measure of depression symptoms, the Penn State Worry Questionnaire (PSWQ; 35) as a measure of trait worry, and the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; 36, 37) as a measure of obsessive-compulsive symptoms. Corresponding coefficient alphas for the current sample were .94 (95% CI: .93, .95) for BDI-II, .95 (95% CI: .94, .96) for PSWQ, and .92 (95% CI: .91, .94) for Y-BOCS. Trait worry (i.e., anxious apprehension), rather than broad trait anxiety, was used due to meta-analytic work showing ERN more strongly relates to worry than to broad anxiety (38).

Experimental Task

The semantic flanker task was identical to the one in Larson et al. (39). Full details are provided in the supplementary material. Three identical distractor words (“Left”, “Right”, “Up”, or “Down”) appeared at the center of the screen for 133 ms. Next, after a 33 ms blank screen, the target word (“Left”, “Right”, “Up”, or “Down”) appeared for 133 ms. Participants were instructed to identify the target word as quickly and accurately as possible by making a spatially compatible button press. The distracter and target stimuli were combined to create pooled congruent trials (left-left, right-right, up-up, down-down) and pooled incongruent trials (left-right, right-left, up-down, down-up). Participants completed eight 96-trial blocks.

Electrophysiological Data Recording and Reduction

A brief description of the data recording and reduction procedures are described here, and full details are in the online supplement. Electroencephalogram (EEG) was recorded from 128 scalp sites using a 129-channel hydrocel geodesic sensor net and Electrical Geodesics, Inc. (EGI; Eugene, OR) amplifier system (20K nominal gain, bandpass = .10-100Hz). EEG was digitized continuously at 250Hz, and impedances were maintained below 50k Ω .

EEG data were bandpass filtered (.05 and 20 Hz) in ERPLab v6.1.4 (40). Response-locked epochs were then extracted (400 ms prior to participant response to 800 ms following response). Consistent with our work (e.g., 41), eye blinks and saccadic eye movement were removed from EEG using independent components analysis (ICA). Then, channels were marked bad using automated procedures. Bad channels were interpolated using spherical splines (42). EEG data were then re-referenced to the average reference. The period from 400 ms to 200 ms prior to participant response was baseline adjusted. ERN was extracted as the average activity over four frontocentral *a priori* selected sites from an *a priori* selected time window: 0 to 125 ms following participant response. Internal consistency was estimated using the ERP Reliability Analysis (ERA) Toolbox v 0.5.3 (43-46). Internal consistency estimates across groups ranged from .98 to .99 for correct-trial scores and from .67 to .86 for error-trial scores.

Data Analysis

We used the location-scale generalization of multilevel models to address our aims (47). Briefly, location-scale multilevel models expand the multilevel structure into the scale part of the model (see the supplementary material for a detailed description). Although standard multilevel models include fixed and random effects for the conditional means (i.e., the location), they leave the residual variance (i.e., the scale) unmodeled (i.e., homogeneous variance structure). Location-scale models relax the assumption of homogenous variance by allowing the residual variance to be a function of predictors (continuous or categorical). Further, the multilevel location-scale model allows a multilevel structure for the residual variance, including both fixed and random effects. Thus, the residual variance (i.e., scale) can be defined by fixed effects capturing the overall variance and random effects that capture the person-specific deviations

from the fixed effects. In short, the residual variance is not constant, as in the standard multilevel model, but is allowed to differ among participants and change across measurement occasions.

Multilevel models are well suited to examining single-trial ERP scores (41, 48-50). Location-scale models can examine the impact of covariates on outcome means (location) and variances (scale). The multilevel location-scale generalization allows variance components and the residual variance to vary as a function of covariates, including interactions.¹ Consequently, the multilevel location-scale model is an excellent tool for examining whether psychopathology groups and psychiatric symptoms are related to ERN score averages (location) and within-person/intraindividual variability (scale).

Initially, we fit three multilevel location-only models that are a standard multilevel model with constant error variance. *Model 1* included predictors for event type (correct, error). *Model 2* included event type, diagnostic group (controls, GAD, MDD, OCD), each interaction between event and group, the interaction between GAD and MDD, and the three-way interaction: Event x GAD x MDD. Diagnostic group was dummy coded, with controls as the reference level. *Model 3* included event type, psychiatric symptoms (BDI-II, PSWQ, YBOCS), and each interaction.

Next, to determine whether psychopathology group differences account for intraindividual variability in ERN, we repeated Models 1-3 but this time with covariates added to the scale portion of the model (i.e., within-person variability). *Model 2*, the model with covariates for psychopathology groups, tested the hypothesis that psychopathology group status would predict intraindividual variability in ERN scores. *Model 3*, the model with covariates for psychiatric symptoms, tested whether symptoms accounted for intraindividual variability. The covariates for the scale portion were identical to the location portion. This approach examines

¹ Covariates for the location and scale portions of the model do not need to be the same, though they often are.

whether simultaneously modeling average scores and within-person variance improved fit over the location-only models and whether psychopathology group or psychiatric symptoms predicted between- or within-person variation. For the location and scale portions of the model (when applicable), we suppressed the overall intercept and estimated event type specific random intercepts for participants.

The models were fit within *R* v4.0.2 (51) using the package *brms* v2.14.4 (52, 53), which is a front end wrapper for *Stan* (54). The priors for parameter estimates and *R* code are described in the online supplement. Leave-one-out cross-validation (LOO-CV) was used to compare model fit (55).

Results

Summary demographic, psychiatric symptom, accuracy, and correct- and error-trial ERN data are presented in Table 1. Grand average waveforms and topographical maps are shown in Figure 1.

Model Comparison

Location-Only vs. Location-Scale Models. *Model 1* only included event type (correct, error; see Table 2). *Model 2* included event type, diagnostic group, and their interactions (see Table 3). *Model 3* included event type, psychiatric symptoms, and their interactions (see Table 4). Each location-only model (i.e., only mean structure) was compared to the corresponding location-scale model that included identical predictors on the scale portion (i.e., variance structure). For example, *Model 1* was separately estimated using location-only and location-scale approaches.

Each location-scale model showed substantial improvement in model fit over its corresponding location-only model based on the expected log predictive density ($\widehat{elpd}_{loo}$).

Differences in $\widehat{elpd}_{loo}$ between location-scale and location-only models were above 6,212 (*SEs* of difference in $\widehat{elpd}_{loo} < 174$). These comparisons indicated that permitting a heterogeneous variance structure across participants (i.e., random effects) improved model fits over a fixed homogeneous variance structure. Although the strength of the predictors for the location portion of the location-scale models were similar to the strength of the predictors for the location-only models, the location-scale model included predictors of within-person variability. There was significant variability in person-specific estimates of within-person variability, and the assumption of a fixed within-person variance from the location-only model risked mischaracterizing variance of some participant data.

Location-Scale Models. We next determined which location-scale model showed the best fit. The models showed comparable fit (differences in $\widehat{elpd}_{loo} < 4.0$, *SEs* of difference in $\widehat{elpd}_{loo} < 2.6$). Therefore, including predictors for diagnostic group or for psychiatric symptoms did not improve fit over a model that only included event type. Therefore, *Model 1* was selected as the best fitting model due to parsimony.

Event-Only Location-Scale Model (*Model 1*)

Model 1 was the focus of interpretation, because the inclusion of diagnostic category (*Model 2*) or psychiatric symptoms (*Model 3*) did not improve prediction of ERN amplitude (see Tables 2-4).

The intraclass correlation coefficient (ICC) was estimated using the between-person variance estimates and group-level residual variance estimate. The group-level ICC for the correct trials was .11 (95% CI: .09 to .14), which indicates that 11% of the variance in correct-trial amplitudes was accounted for by between-person variability. The group-level ICC for error-

trial amplitude was .09 (95% CI: .06 to .12), indicating that 9% of the variance in error-trial amplitudes was accounted for by between-person variability.

The location portion of *Model 1* indicated that error-trial amplitude (95% CI: -1.24 to -.82) was more negative than correct-trial amplitude (95% CI: 0.14 to 0.52). For the scale portion of the model, error-trial standard deviation on the log scale (95% CI: 1.30 to 1.38) was somewhat larger than the correct-trial standard deviation (95% CI: 1.27 to 1.34), but there was overlap in the credible intervals. These estimates were back transformed (via exponentiation of the estimates) to reflect the within-person variance for visualization (see Figure 2).

The location-scale model also described the correlation between means and variances because the random effects parameters are estimated jointly from a multivariate normal distribution. The estimated correlation between correct-trial average amplitude and variation in amplitude was .27 (95% CI: .13 to .40) indicating that smaller (i.e., less negative) correct-trial amplitude was associated with greater variation therein (see Figure 2). The estimated correlation between error-trial ERN scores and within-person ERN variation was -.19 (95% CI: -.36 to -.02), which suggests that larger (i.e., more negative) ERN amplitudes were related to larger variation therein.

Subject-Level Estimates. Person-specific estimates of within-person variance were estimated within the location-scale model, and these point estimates and their 95% CI are shown in Figure 3. When 95% CIs did not include group-level estimates, the CIs of the subject-level estimates are shown in blue, and these participants' within-person variances were considered mischaracterized by the group-level estimate (56). Based on visual inspection of Figure 3, it appears that the CIs for correct-trials are narrower than for error-trials. This behavior in the model would be expected because many more correct trials than error trials were recorded,

which should lead to narrower CIs for correct-trial variances. Notably, the group-level within-person variance mischaracterized correct-trial within-person variances for 148 participants (81.3%) and error-trial within-person variances for 90 participants (49.4%). Point estimates of the subject-level within-person variances ranged from 4.0 to 34.4 for correct trials and from 4.9 to 38.5 for error trials. Therefore, there was about an eight-fold difference between the smallest and largest within-person variances for correct and error trials.

An implication of the heterogeneous within-person variance is that a group-level ICC risks mischaracterizing ICCs at the subject level. Subject-level ICCs were estimated using the person-specific estimates of variance (see 56), and these are shown in Figure 4, respectively. It is clear from Figure 4 that many participants were not well characterized by the group-level ICC.

The group-level ICC for correct-trial ERN mischaracterized subject-level ICCs for 104 participants (57.1%), and the group-level ICC for error-trial ERN mischaracterized 59 participants (32.4%). The range of point estimates for subject-level ICCs was from .05 to .30 for correct-trial ERN and from .04 to .23 for error-trial ERN. Therefore, there was approximately a six-fold increase from the smallest to largest subject-level ICCs.

Based on visual inspection of Figure 4, the proportion of participants with mischaracterized subject-level ICCs was similar across diagnostic categories. Therefore, the assumption of fixed within-person variance seems inappropriate for healthy and clinical groups.

MINI-Confirmed Diagnoses

A subsample of participants with MINI-confirmed diagnoses was examined using the same approach as was used for the overall sample. An identical pattern of effects was observed on the subsample, and these findings are shown in the supplementary material.

Exploratory Analyses

Additional location-scale models were examined that included covariates for medication status and sex (and their interactions) in the location and scale portions of the model. None of the models with the additional covariates showed *improved* fit over the event-only location-scale model, *Model 1* (differences in $\widehat{elpd}_{loo} < 0.4$, *SEs* of difference in $\widehat{elpd}_{loo} < 3.8$), suggesting that these potential confounding variables did not meaningfully improve the prediction of study outcomes.

Discussion

We tested whether intraindividual variability in performance monitoring indexed by single-trial amplitude scores of ERN related to psychopathology. Results of the present analyses failed to find strong evidence in support of clinical differences in correct- or error-trial ERN scores when examining multiple traditional diagnostic categories (GAD, MDD, OCD; *Model 2*) or when examining transdiagnostic symptom relationships (*Model 3*). This conclusion is based on *Models 2* and *3* failing to show improvement in model fit over an event-only model (*Model 1*). Nonetheless, findings indicated that between-person differences in intraindividual variability of correct- and error-trial ERN scores are common across healthy and psychopathology groups. Therefore, neural variability might represent an important dimension for understanding performance monitoring for future research, and the findings points to important methodological implications.

Clinical variables were largely unrelated to intraindividual variability in ERN amplitude or to mean ERN scores, suggesting that performance monitoring was intact in the psychopathology groups studied. This conclusion is supported by the failure of models with the covariates for clinical variables to show improvements in model fit over an event-only model. The factors that contribute to intraindividual variability in ERN are poorly understood. Studies of

related processes suggest arousal/fatigue (57), cognitive function (58-60), or task-unrelated global activity (3, 4) might contribute to neural variability. Arousal (61-63), cognitive function (64-66), and attention-related manipulations (15, 67-69) have all been related to enhanced mean ERN scores, and although speculative, a similar pattern of effects might be observed for intraindividual variability in ERN scores.

Intact performance monitoring in psychopathology was unexpected, but these findings reflect the somewhat mixed state of a large literature. Although meta-analytic evidence supports the relationship between ERN and depression (22, 23), this relationship might be due to a failure to consider the impact of comorbid anxiety symptoms (70, 71). However, the present study did not support this dissociation. Similarly, although the ERN-anxiety relationship appears unique to women (72, 73), the present study did not observe an improvement in model fit when including biological sex and interactions with biological sex as covariates. The disparate findings of relationships might represent differences in tasks used for recording ERN (26) or in approaches to analyzing ERN (74) and suggest a need to move ERN studies toward standardization to rule out nuisance factors (e.g., paradigm characteristics) that could obscure relationships with clinical characteristics. The present study used a semantic version of the flanker paradigm² that has been used in behavioral (75) and EEG research (39), but it is only one of many instantiations of the flanker task. It is unknown which task or data processing features yield the most robust psychopathology vs. healthy control differences in ERN or dimensional relationships between ERN and clinical characteristics (76).

² An analysis of response times is presented in the supplementary materials. These analyses support that congruency effects were observed in the semantic version of the flanker paradigm. However, there was not strong support for clinical differences in response times.

The present analysis provided an avenue for understanding mean-variance relationships (see 77). Larger (i.e., more negative) ERN was related to more variability in ERN scores for error trials, suggesting heightened conflict processing (13, 16, 17) or exaggerated emotional responding is associated with more variability (18, 19), but an opposite pattern was observed for correct trials. The functional significance of ERN following correct trials (i.e., correct-related negativity; CRN) is poorly understood, but is generally considered to reflect overall response monitoring with larger amplitude reflecting greater monitoring (78). Therefore, the finding of larger correct-trial ERN relating to less amplitude variability suggests that individuals with heightened response monitoring consistently engaged monitoring processes throughout the task.

It appears that intraindividual variability is the norm, rather than the exception, for ERN. The present results show notable between-person differences in intraindividual variability, indicating that the assumption of a fixed within-person variance (i.e., residual variance) is inappropriate for ERN studies. Failing to account for individual differences in within-person variance leads to highly biased parameter estimates and inflated Type I errors in traditional (location-only) multilevel models (see 47, 79, 80). Although speculative, there may be systematic patterns and individual differences that account for intraindividual variability of correct- and error-trial ERN amplitude scores, and the use of location-scale multilevel models provide a powerful framework for examining the possibility. Furthermore, the intraindividual variability of many neural measurements represents a largely unexamined area of investigation that might lead to a contribute to a better understanding of psychopathology, as it has in autism spectrum disorders (7-9) and schizophrenia (10).

Another consequence of the between-person differences in intraindividual variability is that group-based estimates of psychometric internal consistency risk masking low reliability in

some participants (44, 56, 77, 81). The assumption of fixed within-person variance in group-based estimates can be relaxed so persons have their own estimate of “error variance” for calculating person-specific reliability (44, 56). The use of person-specific reliability allows for the identification of persons with low reliability, and such data with low reliability can increase the likelihood of finding results that do not replicate and the likelihood of missing true phenomena. More specifically, low psychometric internal consistency can contaminate correlations with clinical variables and between-group differences (82), which hinder the replicability of findings. Multilevel location-scale models can examine the simultaneous impact of covariates on the means and within-person variances to understand the factors that lead to high estimates of psychometric reliability. This approach can be used to optimize other ERPs and paradigms for clinical application by identifying parameters and situations that lead to robust ERP effects, which could move ERP studies toward standardization and support normative databases (26, 83).

The present study had some limitations. First, an ecologically valid sample of participants with diagnoses made by community practitioners was used, and this could have led to the misclassification of some participants. However, study findings were consistent in a subsample of participants with MINI-confirmed diagnoses, and psychiatric symptoms were not meaningfully related to ERN. Second, most participants with clinical diagnoses were receiving medication which might have normalized ERN, but exploratory analyses of medication effects did not improve model fits. Third, specific underlying symptoms, such as checking symptoms or psychomotor retardation (e.g., 70), have been shown to account for the relationship with ERN and might account for intraindividual variability in ERN, but each specific symptom feature was not rigorously assessed as they were not part of the specific hypotheses. Fourth, the present

examination only focused on time-window mean amplitudes, but future research could consider other aspects of ERN (e.g., latency measurements), other error-related ERPs, or even oscillatory phenomena to understand intraindividual variability in performance monitoring. Strengths of the study include use of a unique technique to model intraindividual variability, relatively large samples of participants with psychopathology for the literature, and multiple pathology groups for comparison and assessment of transdiagnostic variables.

Taken together, intraindividual variability in ERN scores is a largely unexplored dimension of performance monitoring. Although psychopathology groups did not systematically differ in within-person variances, there was substantial between-person differences in intraindividual variability. Future research might consider examining intraindividual variability in ERN scores to understand how neural variability contributes to performance monitoring and impacts functional outcome across psychopathology groups. The present study also provides a novel application of multilevel location-scale models to examining neural variability of ERPs that can be readily applied to novel measures and paradigms.

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Table 1

Summary Demographic, Response Time (RT), and Event-Related Brain Potential (ERP) Data

	Controls <i>n</i> = 56		GAD <i>n</i> = 44		MDD <i>n</i> = 51		OCD <i>n</i> = 31	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Female	35	63	37	84	39	77	13	42
Medicated	0	0	28	64	36	71	17	55
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
Age (years)	21.4	4.4	21.6	5.2	22.2	4.4	21.2	2.3
Years of Education	13.6	1.4	14.1	1.5	14.3	1.5	14.0	1.4
BDI-II	2.9	3.2	15.2	8.7	19.1	11.1	13.6	11.8
PSWQ	37.8	10.6	60.8	9.9	56.4	12.9	55.1	12.4
Y-BOCS	1.7	2.9	9.1	8.3	6.6	6.9	18.4	9.3
Accuracy	95%	5%	90%	11%	94%	5%	93%	7%
CRN (μ V)	0.7	1.6	0.2	1.2	0.3	1.1	-.1	1.2
ERN (μ V)	-1.5	2.0	-1.1	1.2	-.9	1.5	-1.3	1.6
	<i>Mean</i>	<i>95% CI</i>	<i>Mean</i>	<i>95% CI</i>	<i>Mean</i>	<i>95% CI</i>	<i>Mean</i>	<i>95% CI</i>
CRN ϕ	.99	.99, .99	.99	.98, .99	.98	.97, .99	.98	.97, .99
ERN ϕ	.86	.79, .91	.68	.50, .82	.76	.62, .86	.74	.54, .87

Note: A total of 12 participants with a primary diagnosis of GAD also had a comorbid MDD diagnosis, and a total of 19 participants with a primary diagnosis of MDD also had a comorbid diagnosis of GAD. Participants with OCD did not have any identified comorbidities. GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive compulsive disorder; BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; CRN = correct-related negativity; ERN = error-related negativity; ϕ = group-level dependability estimates (internal consistency)

Table 2

Estimates from Multilevel Models for Event-Only Models

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Location Portion						
Correct Trials						
Intercept	0.34	0.10	0.15, 0.53	0.33	0.10	0.14, 0.52
Error Trials						
Intercept	-1.06	0.11	-1.28, -0.85	-1.03	0.11	-1.24, -0.82
Residual (<i>SD</i>)	1.36	.002	1.36, 1.36			
Scale Portion (<i>SD</i>)						
Correct Trials						
Intercept				1.31	0.02	1.27, 1.34
Error Trials						
Intercept				1.34	0.02	1.30, 1.38
Random Effects						
Mean Structure						
Correct Intercept	1.72	0.18	1.40, 2.11	1.71	0.18	1.39, 2.11
Error Intercept	1.58	0.24	1.15, 2.11	1.43	0.24	1.01, 1.95
Covariance	0.66	0.16	0.37, 1.00	0.71	0.16	0.41, 1.05
Variance Structure (<i>SD</i>)						
Correct Intercept				0.23	0.01	0.21, 0.25
Error Intercept				0.23	0.02	0.20, 0.27
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{loo}$	-373,259.1			-367,042.7		
$SE(\widehat{elpd}_{loo})$	403.0			418.6		

Note: The residual estimate corresponds to the residual error term of the location-only model. Parameters in standard deviations (*SD*) units are shown as log odds. *SE* = standard error; 95% CI = 95% credible interval; $\widehat{elpd}_{loo}$ = expected log predictive density; $SE(\widehat{elpd}_{loo})$ = the standard error of the expected log predictive density.

Table 3

Estimates from Multilevel Models with Predictors for Psychopathology Groups

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Mean Portion						
Correct Trials						
Intercept	0.70	0.17	0.36, 1.04	0.69	0.17	0.35, 1.03
GAD	-0.28	0.31	-0.89, 0.34	-0.27	0.31	-0.89, 0.34
MDD	-0.17	0.28	-0.73, 0.37	-0.16	0.28	-0.71, 0.40
OCD	-0.81	0.29	-1.37, -0.25	-0.80	0.29	-1.35, -0.22
GAD x MDD	-0.27	0.43	-1.12, 0.58	-0.29	0.43	-1.13, 0.56
Error Trials						
Intercept	-1.24	0.20	-1.64, -0.84	-1.20	0.20	-1.58, -0.81
GAD	0.36	0.35	-0.33, 1.06	0.37	0.34	-0.29, 1.03
MDD	0.40	0.33	-0.25, 1.03	0.43	0.32	-0.19, 1.06
OCD	0.07	0.34	-0.59, 0.73	0.06	0.33	-0.58, 0.69
GAD x MDD	-0.59	0.49	-1.55, 0.38	-0.67	0.47	-1.60, 0.25
Residual (SD)	1.36	.002	1.36, 1.36			
Scale Portion (SD)						
Correct Trials						
Intercept				1.34	0.03	1.28, 1.40
GAD				-0.09	0.06	-0.20, 0.02
MDD				-0.05	0.05	-0.15, 0.05
OCD				0.02	0.05	-0.08, 0.12
GAD x MDD				0.07	0.08	-0.08, 0.23
Error Trials						
Intercept				1.37	0.04	1.30, 1.45
GAD				-0.08	0.07	-0.21, 0.05
MDD				-0.04	0.06	-0.16, 0.08
OCD				-0.02	0.06	-0.14, 0.10
GAD x MDD				0.05	0.09	-0.13, 0.23
Random Effects Variance						
Mean Structure						
Correct Intercept	1.64	0.18	1.33, 2.03	1.63	0.18	1.32, 2.01
Error Intercept	1.60	0.25	1.17, 2.14	1.45	0.25	1.03, 1.99
Covariance	0.67	0.16	0.37, 1.00	0.71	0.16	0.41, 1.04
Variance Structure (SD)						
Correct Intercept				0.23	0.01	0.20, 0.25
Error Intercept				0.24	0.02	0.20, 0.27
Covariance				0.05	0.01	0.04, 0.06

Model Diagnostics

$\widehat{elpd}_{loo}$	-373,258.6	-367,046.2
$SE(\widehat{elpd}_{loo})$	403.0	418.7

Note: The residual estimate corresponds to the residual error term of the location-only model.

Parameters in standard deviations (*SD*) units are shown as log odds. *SE* = standard error; 95% CI = 95% credible interval; GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive compulsive disorder; $\widehat{elpd}_{loo}$ = expected log predictive density; $SE(\widehat{elpd}_{loo})$ = the standard error of the expected log predictive density.

Table 4

Estimates from Multilevel Models with Predictors for Psychiatric Symptoms

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Mean Structure						
Correct Trials						
Intercept	0.33	0.09	0.15, 0.51	0.33	0.09	0.15, 0.52
BDI	-0.08	0.12	-0.32, 0.16	-0.08	0.12	-0.32, 0.16
PSWQ	-0.22	0.13	-0.48, 0.04	-0.22	0.13	-0.47, 0.03
YBOCS	-0.16	0.11	-0.38, 0.05	-0.16	0.11	-0.37, 0.04
Error Trials						
Intercept	-1.08	0.11	-1.29, -0.86	-1.04	0.11	-1.25, -0.83
BDI-II	0.14	0.14	-0.14, 0.41	0.11	0.14	-0.16, 0.38
PSWQ	-0.15	0.15	-0.44, 0.14	-0.15	0.14	-0.43, 0.14
YBOCS	0.05	0.12	-0.20, 0.29	0.03	0.12	-0.20, 0.26
Residual (SD)	1.36	.002	1.36, 1.36			
Scale Portion (SD)						
Correct Trials						
Intercept				1.31	0.02	1.27, 1.34
BDI				-0.02	0.02	-0.06, 0.02
PSWQ				-0.02	0.02	-0.07, 0.03
YBCOS				0.03	0.02	-0.01, 0.07
Error Trials						
Intercept				1.34	0.02	1.30, 1.38
BDI-II				-0.01	0.03	-0.06, 0.04
PSWQ				-0.02	0.03	-0.07, 0.04
YBCOS				0.01	0.02	-0.03, 0.05
Random Effects Variance						
Mean Structure						
Correct Intercept	1.59	0.17	1.29, 1.97	1.59	0.17	1.28, 1.96
Error Intercept	1.61	0.25	1.18, 2.14	1.47	0.25	1.05, 2.00
Covariance	0.67	0.16	0.38, 1.00	0.72	0.16	0.42, 1.06
Variance Structure (SD)						
Correct Intercept				0.23	0.01	0.21, 0.25
Error Intercept				0.24	0.02	0.20, 0.27
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{loo}$	-373,258.9			-367,042.4		
$SE(\widehat{elpd}_{loo})$	403.0			418.2		

Note: The residual estimate corresponds to the residual error term of the location-only model. Parameters in standard deviations (*SD*) units are shown as log odds. *SE* = standard error; 95% CI = 95% credible interval; BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; $\widehat{elpd}_{loo}$ = expected log predictive density; $SE(\widehat{elpd}_{loo})$ = the standard error of the expected log predictive density.

Figure Captions

Figure 1. Grand average waveforms for the error-related negativity (ERN) shown separately for each diagnostic group. Waveforms are shown for fronto-central activity average across four sites, and activity corresponding to ERN is shown in gray. Topographical maps showing the fronto-central scalp distribution of ERN for each group are shown in the top-right corner of each plot, and the maps represent the average Δ ERN difference activity (error minus correct) from 0 to 125 ms. Grand average waveforms for Δ ERN are shown separately for each group in the far-right panel. GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive compulsive disorder

Figure 2. The top row shows the estimated posterior density curves and their associated uncertainty intervals, with the 95% credible intervals shaded around the posterior mean. The estimated fixed effects means for correct and error trials are shown in the top left, and the estimated fixed effects variances for correct and error trials are shown in the top right. The bottom row shows the relationships between person-average error-related negativity (ERN) scores and within-person standard deviations (bottom left: correct-trial ERN or correct-related negativity [CRN]; bottom right: error-trial ERN), and these scores were estimated within the multilevel location-scale models. Note different x axes across plots.

Figure 3. Plots show the subject-level within-person variance for correct-trial (top) and error-trial amplitudes with their respective 95% credible intervals (CIs) in ascending order from smallest to largest point estimate for all participants. The dotted line corresponds to the group-level within-

person variance. When the subject-level CIs do not include the group-level ICC, the CIs are shown in blue.

Figure 4. Plots show the subject-level intraclass correlation coefficients (ICCs) for correct-trial (top) and error-trial (bottom) amplitudes with their respective 95% credible intervals (CIs) in ascending order from smallest to largest point estimate for all participants (left) and separately for each diagnostic group (right). The dotted line corresponds to the group-level ICC. When the CIs do not include the group-level ICC, the CIs are shown in blue.

Figure 1

Figure 2

Figure 3

Figure 4

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Supplementary Method

Experimental Task

Stimuli were presented on a 17-inch computer monitor located approximately 60 cm from the participant's head. Each block began with a fixation cross (white, Arial 50-point font), which was presented for 2000 ms on a black background. Three horizontally-oriented, identical distractor words ("Left", "Right", "Up", or "Down"; white, Arial 48-point font) – one just above fixation, one at fixation, and one just below fixation – appeared at the center of the screen for 133 ms. Next, after a 33 ms blank screen, one of four possible target words ("Left", "Right", "Up", or "Down"; white, Arial 77-point font) appeared for 133 ms at the center of the screen. After 133 ms, the target word was replaced by a fixation cross. Participants were instructed to identify the target word as quickly and accurately as possible by making a spatially compatible button press on a computer keyboard. Participants were instructed to press the "F" key with the left middle finger to indicate the target word was "Left", and "G" key with the left index finger for the target word "Right", the "J" key with the right middle finger for the target word "Up", or the "N" key with the right index finger for the target word "Down". The word "Error" replaced the fixation cross for 200 ms, between 1500 and 1700 ms after target onset, if a participant responded incorrectly or not at all within 1500 ms of target onset. The inter-trial interval (ITI) varied randomly (without replacement) between 1850 ms and 2150 ms in steps of three ms around an average duration of 2000 ms.

The distracter and target stimuli described above were combined to create four pooled congruent distracter-target pairs (left-left, right-right, up-up, down-down) and four pooled incongruent distracter-target pairs (left-right, right-left, up-down, down-up). Distracter-target pairs involving the words "Left" and/or "Right" were presented in odd trials while distracter-

target pairs involving the words “Up” and/or “Down” were presented in even trials. Each congruent and incongruent distracter-target pair was randomly presented twelve times within a block. Participants completed a 48-trial practice block and eight 96-trial test blocks.

Electrophysiological Data Recording and Reduction

Electroencephalogram (EEG) was recorded from 128 scalp sites using a 129-channel hydrocel geodesic sensor net and Electrical Geodesics, Inc. (EGI; Eugene, OR) amplifier system (20K nominal gain, bandpass = .10-100Hz). EEG was online referenced to the vertex electrode; a ground sensor was placed at location PCz. EEG was digitized continuously at 250Hz with a 24-bit analog-to-digital converter. Impedances were maintained below 50k Ω .

EEG data were filtered offline using a sixth-order (36 db/oct) infinite impulse response Butterworth filter with half-amplitude cutoffs at .05 and 20 Hz implemented in ERPLab v6.1.4 (Lopez-Calderon & Luck, 2014). Response-locked epochs were then extracted separately for each participant using a temporal window from 400 ms prior to participant response to 800 ms following participant response. Consistent with our previous work (e.g., Clayson & Larson, 2019), eye blinks and horizontal and vertical saccadic eye movement were removed from the segmented waveforms using independent components analysis (ICA). For the ICA procedure, epoched EEG data from all channels were processed through a binary version of EEGLab’s *runica* function called *binica* (Delorme & Makeig, 2004). ICA components that correlated .9 or above with the scalp topography of a blink template and .8 or above with the scalp topography of vertical and horizontal saccade templates were removed. The templates that were used for artifact correction included those that were automatically generated by the ERP PCA Toolkit and two created from the current dataset.

Then, channels with more than a 100 μV step within 100 ms intervals, a voltage difference of 300 μV through the duration of the epoch, or an absolute correlation with the nearest six neighboring channels that fell below .4 were marked as a bad. Channels marked as bad for more than 20% of epochs were considered globally bad. Bad channels were interpolated using spherical splines (Perrin et al., 1989), but if more than 10% of channels were marked bad for an epoch, the entire epoch was rejected. EEG data were then re-referenced to the average reference. The period from 400 ms to 200 ms prior to participant response was used for baseline adjustment.

Response-locked ERPs were then analyzed; sensors for analysis were chosen *a priori* and were identical to studies from our lab (Clayson et al., 2012; Clayson & Larson, 2013). To reduce the biasing effects of background noise on ERP measurements, ERN was scored using a time-window mean amplitude (Clayson et al., 2013; Luck, 2014). Regions of interest (ROIs), which are created by averaging data across multiple electrodes, were used to improve signal-to-noise ratio over data from a single electrode (Baldwin et al., 2015; Huffmeijer et al., 2010). ERN was extracted as the average activity over four *a priori* selected frontocentral sites (6 [FCz], 7, 106, 129 [Cz]; for electrode configuration, see Clayson & Larson, 2013) from an *a priori* selected time window: 0 to 125 ms following participant response.

Internal Consistency

Generalizability theory was used to calculate dependability (ϕ), which is a measure of internal consistency (see Baldwin et al., 2015; Brennan, 2001; Clayson, Baldwin, et al., 2021; Shavelson & Webb, 1991), but participants were not excluded based on dependability estimates due to the emphasis on within-person variance. Dependability was examined using the ERP Reliability Analysis (ERA) Toolbox v 0.5.3 (Clayson, Brush, et al., 2021; Clayson, Carbine, et

al., 2021; Clayson & Miller, 2017a, 2017b). The ERA Toolbox used CmdStan v 2.24.1 (Stan Development Team, 2017) to implement the analyses in Stan (Carpenter et al., 2017). To estimate variance components, the Markov chain Monte Carlo estimation procedures used 4 chains and 10,000 iterations each. Dependability point estimates across groups ranged from .98 to .99 for correct-trial scores and from .67 to .86 for error-trial scores.

Multilevel Location-Scale Model

The modeling approach used here relaxes the assumption of a constant residual variance as it is defined in the standard multilevel model. Typically, the residual variance is fixed across all observations implying homoskedasticity. Scale models, in general, relax this assumption and allow variances to be a function of model parameters and predictors. Multilevel location-scale models represent a specific class of scale models that include a random effects structure not only for the conditional means, the location, but also the residual variances or the scale. Originally these models were introduced as double hierarchical generalized linear models (Lee & Nelder, 2006) and were expanded into multilevel or mixed effects location-scale models based on maximum likelihood (Hedeker et al., 2008) as well as Bayesian estimation methods (Rast et al., 2012).

Briefly, the standard multilevel model assumes that the response y_{ij} for person $i=1, \dots, N$ at occasion $j=1, \dots, K_i$ is a linear function of within- and between person predictors plus a residual error. For example, with a person level predictor x_{ij} and one between-person predictor Z_i we could define

$$\begin{aligned}
 \text{Level 1:} \quad & y_{ij} = \hat{\mu}_{ij} + e_{ij} \\
 & \hat{\mu}_{ij} = \beta_{0i} + \beta_{1i}x_{ij} \\
 \text{Level 2:} \quad & \beta_{0i} = \tau_{00} + \tau_{01}Z_i + u_{0i}
 \end{aligned} \tag{1}$$

$$\beta_{1i} = \tau_{10} + \tau_{11}Z_i + u_{1i}$$

$\hat{\mu}_{ij}$ represents the expected conditional mean, or location, for each person and each time point given the linear model defined in the next lines. The intercept β_{0i} and the slope β_{1i} represent the person level effects while the τ 's capture the between-person effects. The u 's are the random effects that define the deviation of each individual about the fixed τ parameters. The u 's are assumed to follow a multivariate normal distribution $u \sim N(0, \Sigma)$, where Σ represents a covariance matrix that captures the variances and covariances of the random effects. e_{ij} are the residuals that are assumed to follow a Normal distribution with mean = 0 and constant variance of σ^2 , such that $e_{ij} \sim N(0, \sigma^2)$. Critically, the assumption, of a constant residual variance, σ^2 , will be relaxed in the location-scale model. The standard model can be rewritten concisely as $y_{ij} \sim N(\hat{\mu}_{ij}, \sigma^2)$.

We can now relax the assumption that each residual is sampled from a distribution with the same scale. For one, we could imagine that different individuals are inherently different in their tendency to consistently respond to a task. Some might be consistent in their responses, whereas others tend to be more error prone and less consistent overall. These differences can be expressed in the model by allowing the residual variances to be person specific. In terms of notation, we can add a subscript to σ_i^2 that indicates that the residual variance is now person-specific. Following the same logic, we can further assume that the residual variance changes over time or conditions. For example, one might hypothesize that fatigue reduces the consistency of responses as participants start committing more errors toward the end of a long task. Accordingly, we can add a j subscript to the error variance term, σ_{ij}^2 , to indicate that the residual variance is allowed to not only change across individuals but also across time (or other

conditions). In terms of notation, we can simply re-express the model above to indicate that residual variance is not constant anymore: $y_{ij} \sim N(\hat{\mu}_{ij}, \sigma_{ij}^2)$. Note that the only change has been done in the scale part of the model, the σ_{ij}^2 , while the location, $\hat{\mu}_{ij}$, remained the same. Now that we allow σ_{ij}^2 to vary across i and j , we can write a model that explains these differences. Given that we are now modeling a variance, we need to ensure positivity of the outcome. This can be obtained by defining our model on the log-scale such that $\log(\sigma_{ij}^2)$.

The multilevel location-scale model extends the multilevel structure from the location part in Equation (1) to the scale part – hence, multilevel location-scale models. We can now write the multilevel model part for the scale as

$$\text{Level 1:} \quad \sigma_{ij} = \exp(\gamma_{0i} + \gamma_{1i}x_{ij})$$

$$\text{Level 2:} \quad \gamma_{0i} = \pi_0 + \pi_1 Z_i + v_{0i}$$

$$\gamma_{1i} = \pi_1 + \pi_1 Z_i + v_{1i}$$

Again, the π 's are the fixed effects and the v 's are the random effects. Here we chose to use the same within- (x) and between-person (Z) level predictors as for the location part in Equation (1) -- this is not necessary and one could use different predictors for the location and the scale. It's important to note here that the location and the scale part in Equation (1) and (2) respectively, constitute one model. Consequently, the distribution of the random effect comprises all, u 's for the location and all v 's for the scale. That is, if we stack the random effect vectors $[\mathbf{u}, \mathbf{v}]$ (in this case we have 4 random effects; 2 for the location and 2 for the scale) we can assume that $[\mathbf{u}, \mathbf{v}]' \sim N(\mathbf{0}, \Phi)$. Here Φ is a 4 x 4 covariance matrix for the random effect variances in its diagonal and the random effect covariances in its off diagonal. The top left 2 x 2 quadrant captures the (co)variance of the location, the lower right 2 x 2 quadrant captures the scale (co)variances and the lower left 2 x 2 quadrant captures the four covariances among the location and the scale. This

is a unique feature of multilevel location-scale models as they allow the estimation and quantification of random effects across the location and the scale. For example, in a longitudinal study a positive covariance among the random location intercept and the random scale intercept indicates that those who start out at higher levels also tend to be more variable in their responses.

Another consequence of relaxing the constant error variance assumption affects the computation of indices that involve estimates of the error variance. One of those indices is the intraclass correlation coefficient (ICC). In an unconditional means model the ICC relates the between-person variance (σ_{bp}^2) to the total variance so that $\sigma_{bp}^2 / (\sigma_{bp}^2 + \sigma_e^2)$. This estimate again assumes a constant error variance (σ_e^2) yielding an ICC that is constant across the sample. By relaxing that assumption we can obtain an $ICC_{ij} = \sigma_{u_i}^2 / (\sigma_{u_i}^2 + \sigma_{e_{ij}}^2)$ that varies across individuals (Brunton-Smith et al., 2017). For example, Figure 5 in the main text illustrates the effect of allowing ICC's to vary across individuals.

R Code

The multilevel location-only and location-scale models were fit within *R* v4.0.2 (R Development Core Team, 2020) using the package *brms* v2.14.4 (Bürkner, 2017, 2018). The basic syntax used to run these models within *R* is shown and briefly described below.

The priors for fixed effect estimates, including the event-type intercepts, used a normal distribution with a mean of 0 and a standard deviation of 3. All variance components were estimated using a Student's *t* prior distribution with 10 degrees of freedom, a location parameter of 0, and a scale parameter of 3. An LKJ prior distribution with a shape parameter of 2 was used as the prior for the correlation between the varying intercepts and slopes effects of event type (correct, error) within persons (Lewandowski et al., 2009). A shape = 2 parameter can be

considered mildly informative, putting more weight on an identity matrix. Leave-one-out cross-validation (LOO-CV) was used to compare model fit (Vehtari et al., 2016).

Priors:

```
prior_location      <- c(set_prior("normal(0,3)", class = "b"),
                          set_prior("lkj(2)", class = "L"),
                          set_prior("student_t(10, 0, 2)", class = "sd"),
                          set_prior("student_t(10, 0, 2)", class = "sigma"))

prior_locationscale  <- c(set_prior("normal(0,3)", class = "b"),
                          set_prior("lkj(2)", class = "L"),
                          set_prior("student_t(10, 0, 2)", class = "sd"),
                          set_prior("student_t(10, 0, 2)", class = "sd", dpar = "sigma"))
```

For RT models only, a prior of "normal(600,100)" was used for class = "b".

Model Syntax (*Model 1*)

```
brm_location_eventonly <- brm(bf(ern ~ 0 + event +
                                (0 + event|p|subjid)),
                              data = df,
                              prior = prior_location,
                              family = gaussian(),
                              chains = 4,
                              cores = 4,
                              iter = 10000,
                              seed = 042221,
```

```

save_pars = save_pars(all = TRUE),
sample_prior = TRUE)

brm_locationscale_eventonly <- brm(bf(ern ~ 0 + event +
                                     (0 + event|p|subjid),
                                     sigma ~ 0 + event +
                                     (0 + event|p|subjid)),
data = df,
prior = prior_locationscale,
family = gaussian(),
chains = 4,
cores = 4,
iter = 10000,
seed = 042221,
save_pars = save_pars(all = TRUE),
sample_prior = TRUE)

```

Covariates were added to the model by only altering the `bf` portion of the syntax.

Model 2

Location-Only

```
bf(ern ~ 0 + event + event:gad + event:mdd + event:ocd + event:gad:mdd + (0 + event|p|subjid))
```

Location-Scale

```
bf(ern ~ 0 + event + event: gad + event:mdd + event:ocd + event:gad: mdd + (0 + event|p|subjid),
```

$\sigma \sim 0 + \text{event} + \text{event:gad} + \text{event:mdd} + \text{event:ocd} + \text{event:gad:mdd} + (0 + \text{event|p|subjid}))$

Model 3

Location-Only

$\text{bf}(\text{ern} \sim 0 + \text{event} + \text{event:bdi} + \text{event:pswq} + \text{event:ybocs} + (0 + \text{event|p|subjid}))$

Location-Scale

$\text{bf}(\text{ern} \sim 0 + \text{event} + \text{event:bdi} + \text{event:pswq} + \text{event:ybocs} + (0 + \text{event|p|subjid}),$

$\sigma \sim 0 + \text{event} + \text{event:bdi} + \text{event:pswq} + \text{event:ybocs} + (0 + \text{event|p|subjid}))$

Variable Descriptions

ern = single-trial time-window mean ERN amplitude scores

event = correct, error

gad = dummy code for participants with a GAD diagnosis

mdd = dummy code for participants with a MDD diagnosis

ocd = dummy code for participants with a OCD diagnosis

bdi = mean-centered BDI scores

pswq = mean-centered PSWQ scores

ybocs = mean-centered YBOCS scores

Additional resources for fitting location-scale models can be found in the following articles and in their associated supplementary materials: Williams et al. (2021) and Williams et al. (2019).

Results

We sought to investigate the impact of psychopathology and psychiatric symptoms on response times (RTs). The same set of models that was used to examine ERN was used to examine RTs. *Model 1* only included event type (correct, error; see Table 2). *Model 2* included event type, diagnostic group, and their interactions (see Table 3). *Model 3* included event type, psychiatric symptoms, and their interactions (see Table 4). Each location-only model (i.e., only mean structure) was compared to the corresponding location-scale model that included identical predictors on the scale portion (i.e., variance structure).

Similar to ERN findings, each location-scale model for RTs showed substantial improvement in model fit over its corresponding location-only model based on the expected log predictive density ($\widehat{elpd}_{loo}$). Differences in $\widehat{elpd}_{loo}$ between location-scale and location-only models were above 6,565 (*SEs* of difference in $\widehat{elpd}_{loo} < 189$). We next examined which location-scale model showed the best fit for RTs. The models showed comparable fit (differences in $\widehat{elpd}_{loo} < 0.5$, *SEs* of difference in $\widehat{elpd}_{loo} < 1.8$). Therefore, including predictors for diagnostic group or for psychiatric symptoms did not improve fit over a model that only included event type. Therefore, *Model 1* was considered as the best fitting model for RTs due to parsimony, and these findings (*Models 1-3*) are summarized in supplementary Tables 5-7.

The location portion of *Model 1* indicated that congruent-trial RTs (95% CI: 547.35 to 567.91) was faster than incongruent-trial RTs (95% CI: 655.38 to 678.46). Therefore, the RTs should show the typical congruency effect. For the scale portion of the model, congruent-trial standard deviations on the log scale (95% CI: 4.91 to 4.98) were somewhat lower than incongruent-trial standard deviations (95% CI: 4.96 to 5.04), but there was overlap in the credible intervals.

The estimated correlation between congruent-trial RTs and incongruent-trial RTs was .91 (95% CI: .89 to .94). The estimated correlation between mean congruent-trial RTs and variation

in congruent-trial RTs was .72 (95% CI: .65 to .79) indicating that longer RTs were associated with greater variation in RTs. A similar pattern was observed for incongruent-trial RTs. The estimated correlation between mean incongruent-trial RTs and variation in incongruent-trial RTs was .73 (95% CI: .65 to .79).

Table 1

Summary Demographic, Response Time (RT), and Event-Related Brain Potential (ERP) Data for Psychopathology Groups with MINI-Confirmed Diagnoses

	Controls <i>n</i> = 56		GAD <i>n</i> = 18		MDD <i>n</i> = 20		OCD <i>n</i> = 15	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Female	35	63	15	79	17	81	6	30
Medicated	0	0	13	72	15	75	6	40
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>
Age (years)	21.4	4.4	22.8	7.6	22.0	3.7	21.5	2.3
Years of Education	13.6	1.4	14.7	1.5	14.5	1.3	14.1	1.2
BDI-II	2.9	3.2	15.2	8.7	19.1	11.1	13.6	11.8
PSWQ	37.8	10.6	60.8	9.9	56.4	12.9	55.1	12.4
Y-BOCS	1.7	2.9	9.1	8.3	6.6	6.9	18.4	9.3
Accuracy	95%	5%	91%	5%	94%	3%	92%	6%
CRN (μ V)	0.7	1.6	0.3	0.9	0.2	0.8	-.3	1.3
ERN (μ V)	-1.5	2.0	-.9	0.8	-1.1	1.1	-1.9	1.4
	<i>Mean</i>	<i>95% CI</i>	<i>Mean</i>	<i>95% CI</i>	<i>Mean</i>	<i>95% CI</i>	<i>Mean</i>	<i>95% CI</i>
CRN ϕ	.99	.99, .99	.99	.98, .99	.98	.97, .99	.98	.97, .99
ERN ϕ	.86	.79, .91	.49	.15, .75	.56	.21, .79	.69	.43, .87

Note: A total of 10 participants with a primary diagnosis of GAD also had a comorbid MDD diagnosis, and a total of 4 participants with a primary diagnosis of MDD also had a comorbid diagnosis of GAD. MINI = Mini-International Neuropsychiatric Inventory; GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive compulsive disorder; BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; CRN = correct-related negativity; ERN = error-related negativity

Table 2

Estimates from Multilevel Models for Event-Only Models for Psychopathology Groups with MINI-Confirmed Diagnoses

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Location Portion						
Correct Trials						
Intercept	0.28	0.12	0.04, 0.50	0.27	0.12	0.04, 0.50
Error Trials						
Intercept	-1.18	0.12	-1.42, -0.94	-1.14	0.12	-1.38, -0.91
Residual (SD)	1.37	0.002	1.36, 1.37			
Scale Portion (SD)						
Correct Trials				1.31	0.02	1.28, 1.35
Intercept						
Error Trials				1.35	0.02	1.31, 1.39
Intercept						
Random Effects						
Mean Structure						
Correct Intercept	1.72	0.18	1.40, 2.11	1.71	0.18	1.39, 2.11
Error Intercept	1.58	0.24	1.15, 2.11	1.43	0.24	1.01, 1.95
Covariance	0.66	0.16	0.37, 1.00	0.71	0.16	0.41, 1.05
Variance Structure (SD)						
Correct Intercept				0.23	0.01	0.20, 0.26
Error Intercept				0.23	0.02	0.20, 0.27
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{100}$	-279,675.3			-274,895.8		
$SE(\widehat{elpd}_{100})$	331.3			320.9		

Note: Parameters in standard deviations (SD) units are shown as log odds. MINI = Mini-International Neuropsychiatric Inventory; SE = standard error; 95% CI = 95% credible interval; $\widehat{elpd}_{100}$ = expected log predictive density.

Table 3

Estimates from Multilevel Models With Predictors for Psychopathology Groups for Psychopathology Groups with MINI-Confirmed Diagnoses

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Mean Portion						
Correct Trials						
Intercept	0.68	0.18	0.33, 1.04	0.69	0.17	0.35, 1.03
GAD	-0.68	0.39	-1.43, 0.07	-0.69	0.38	-1.44, 0.05
MDD	-0.53	0.31	-1.14, 0.09	-0.54	0.31	-1.14, 0.07
OCD	-0.96	0.32	-1.60, -0.34	-0.96	0.32	-1.59, -0.33
GAD x MDD	0.59	0.59	-0.54, 1.74	0.61	0.58	-0.51, 1.74
Error Trials						
Intercept	-1.26	0.20	-1.65, -0.86	-1.21	0.2	-1.59, -0.83
GAD	0.28	0.40	-0.52, 1.07	0.25	0.39	-0.52, 1.02
MDD	0.21	0.33	-0.44, 0.86	0.21	0.33	-0.43, 0.85
OCD	-0.25	0.35	-0.94, 0.44	-0.23	0.34	-0.91, 0.43
GAD x MDD	-0.17	0.61	-1.36, 1.02	-0.19	0.58	-1.34, 0.96
Residual (SD)	1.37	0.002	1.36, 1.37			
Scale Portion (SD)						
Correct Trials						
Intercept				1.34	0.03	1.28, 1.40
GAD				-0.09	0.07	-0.22, 0.04
MDD				-0.06	0.05	-0.17, 0.05
OCD				0.04	0.06	-0.07, 0.15
GAD x MDD				0.05	0.10	-0.15, 0.25
Error Trials						
Intercept				1.37	0.04	1.30, 1.44
GAD				-0.04	0.08	-0.19, 0.11
MDD				-0.07	0.06	-0.20, 0.05
OCD				0.01	0.06	-0.12, 0.13
GAD x MDD				0.02	0.12	-0.20, 0.26
Random Effects Variance						
Mean Structure						
Correct Intercept	1.64	0.18	1.33, 2.03	1.63	0.18	1.32, 2.01
Error Intercept	1.60	0.25	1.17, 2.14	1.45	0.25	1.03, 1.99
Covariance	0.67	0.16	0.37, 1.00	0.71	0.16	0.41, 1.04
Variance Structure (SD)						
Correct Intercept				0.23	0.01	0.20, 0.26
Error Intercept				0.23	0.02	0.20, 0.27
Covariance				0.05	0.01	0.04, 0.06

Model Diagnostics

$\widehat{elpd}_{loo}$	-279,674.8	-274,897.8
$SE(\widehat{elpd}_{loo})$	331.3	320.8

Note: Parameters in standard deviations (*SD*) units are shown as log odds. MINI = Mini-International Neuropsychiatric Inventory; *SE* = standard error; 95% CI = 95% credible interval; GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive compulsive disorder; $\widehat{elpd}_{loo}$ = expected log predictive density.

Table 4

Estimates from Multilevel Models With Predictors for Psychiatric Symptoms for Psychopathology Groups with MINI-Confirmed Diagnoses

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Mean Structure						
Correct Trial						
Intercept	0.27	0.11	0.04, 0.49	0.27	0.11	0.05, 0.49
BDI	-0.07	0.14	-0.34, 0.21	-0.07	0.14	-0.34, 0.21
PSWQ	-0.25	0.15	-0.55, 0.05	-0.25	0.15	-0.56, 0.05
YBCOS	-0.18	0.12	-0.42, 0.06	-0.18	0.12	-0.42, 0.06
Error Trial						
Intercept	-1.22	0.12	-1.46, -0.97	-1.17	0.12	-1.42, -0.93
BDI-II	0.22	0.15	-0.08, 0.51	0.20	0.15	-0.09, 0.48
PSWQ	-0.25	0.16	-0.58, 0.07	-0.26	0.16	-0.57, 0.06
YBCOS	0.06	0.13	-0.19, 0.31	0.07	0.13	-0.18, 0.31
Residual Variance (<i>SD</i>)	1.37	0.002	1.36, 1.37			
Variance Structure (<i>SD</i>)						
Correct Trials						
Intercept				1.31	0.02	1.27, 1.35
BDI				-0.02	0.02	-0.07, 0.03
PSWQ				-0.02	0.03	-0.07, 0.03
YBCOS				0.03	0.02	-0.01, 0.07
Error Trials						
Intercept				1.35	0.02	1.30, 1.40
BDI-II				-0.02	0.03	-0.08, 0.03
PSWQ				-0.01	0.03	-0.07, 0.05
YBCOS				0.02	0.02	-0.03, 0.06
Random Effects Variance						
Mean Structure						
Correct Intercept	1.59	0.17	1.29, 1.97	1.59	0.17	1.28, 1.96
Error Intercept	1.61	0.25	1.18, 2.14	1.47	0.25	1.05, 2.00
Covariance	0.67	0.16	0.38, 1.00	0.72	0.16	0.42, 1.06
Variance Structure (<i>SD</i>)						
Correct Intercept				0.23	0.01	0.20, 0.26
Error Intercept				0.23	0.02	0.20, 0.27
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{loo}$	-279,675.1			-274,897.1		
$SE(\widehat{elpd}_{loo})$	331.3			320.9		

Note: Parameters in standard deviations (*SD*) units are shown as log odds. MINI = Mini-International Neuropsychiatric Inventory; *SE* = standard error; 95% CI = 95% credible interval; BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; $\widehat{elpd}_{loo}$ = expected log predictive density

Table 5

Estimates from Multilevel Models Predicting Response Times for Event-Only Models (Model 1)

Predictor	Estimate	Location-Only		Estimate	Location-Scale	
		SE	95% CI		SE	95% CI
Location Portion						
Congruent Trials						
Intercept	557.51	5.24	547.35, 567.91	557.57	5.43	546.79, 568.09
Incongruent Trials						
Intercept	666.84	5.87	655.38, 678.46	666.77	5.98	654.97, 678.34
Residual (<i>SD</i>)	5.02	0.002	5.01, 5.02			
Scale Portion (<i>SD</i>)						
Congruent Trials				4.94	0.02	4.91, 4.98
Intercept						
Incongruent Trials				5.00	0.02	4.96, 5.04
Intercept						
Random Effects						
Mean Structure						
Congruent Intercept	5237.89	533.98	4294.11, 6390.36	5108.73	514.79	4188.38, 6216.26
Incongruent Intercept	6542.85	669.27	5363.64, 7989.94	6302.56	627.83	5183.96, 7650.42
Covariance	5363.24	570.05	4361.11, 6592.28	5190.29	539.69	4234.80, 6339.71
Variance Structure (<i>SD</i>)						
Congruent Intercept				0.24	0.01	0.22, 0.27
Incongruent Intercept				0.25	0.01	0.22, 0.27
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{loo}$	-783,577.9			-777,011.3		
$SE(\widehat{elpd}_{loo})$	423.0			440.7		

Note: Parameters in standard deviations (*SD*) units are shown as log odds. MINI = Mini-International Neuropsychiatric Inventory; *SE* = standard error; 95% CI = 95% credible interval; $\widehat{elpd}_{loo}$ = expected log predictive density.

Table 6

Estimates from Multilevel Models (Model 2) Predicting Response Times with Predictors for Psychopathology Groups

Predictor	Estimate	Location-Only		Estimate	Location-Scale	
		SE	95% CI		SE	95% CI
Mean Portion						
Congruent Trials						
Intercept	531.23	9.70	512.35, 550.46	532.08	9.63	512.94, 550.69
GAD	42.18	17.45	8.46, 76.88	42.25	17.13	8.61, 76.13
MDD	23.79	15.74	-7.38, 54.37	23.83	15.32	-5.46, 53.99
OCD	54.39	16.65	21.43, 87.39	52.82	16.15	21.51, 85.28
GAD x MDD	-31.56	23.73	-78.58, 14.82	-32.87	23.29	-78.56, 12.69
Incongruent Trials						
Intercept	632.72	10.71	611.78, 654.14	633.55	10.67	612.56, 654.08
GAD	54.95	19.38	17.39, 93.81	54.17	18.93	17.43, 91.94
MDD	33.88	17.45	0.14, 67.91	33.97	17.06	1.34, 67.47
OCD	64.91	18.46	28.51, 101.40	63.47	17.85	29.49, 98.99
GAD x MDD	-42.36	26.39	-93.96, 9.33	-43.06	25.63	-93.54, 7.07
Residual (<i>SD</i>)	5.02	0.002	5.01, 5.02			
Scale Portion (<i>SD</i>)						
Congruent Trials						
Intercept				4.82	0.03	4.76, 4.89
GAD				0.18	0.06	0.07, 0.30
MDD				0.13	0.05	0.03, 0.23
OCD				0.22	0.05	0.11, 0.32
GAD x MDD				-0.15	0.08	-0.31, 0.01
Incongruent Trials						
Intercept				4.87	0.03	4.80, 4.93
GAD				0.21	0.06	0.09, 0.32
MDD				0.15	0.05	0.04, 0.25

OCD				0.23	0.05	0.12, 0.33
GAD x MDD				-0.16	0.08	-0.32, -0.00
Random Effects						
Variance						
Mean Structure						
Congruent Intercept	5482.88	569.00	4467.95, 6700.26	5322.48	554.34	4349.99, 6527.05
Incongruent						
Intercept	6828.98	717.37	5555.18, 8360.75	6533.13	683.27	5326.66, 8006.04
Covariance	5621.18	609.01	4537.66, 6930.40	5405.06	586.73	4366.12, 6673.97
Variance Structure						
(SD)						
Congruent Intercept				0.24	0.01	0.22, 0.27
Incongruent						
Intercept				0.24	0.01	0.22, 0.27
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{loo}$	-783,578.3			-777,012.4		
$SE(\widehat{elpd}_{loo})$	423.0			440.7		

Note: Parameters in standard deviations (SD) units are shown as log odds. MINI = Mini-International Neuropsychiatric Inventory; *SE* = standard error; 95% CI = 95% credible interval; GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive compulsive disorder; $\widehat{elpd}_{loo}$ = expected log predictive density.

Table 8

Estimates from Multilevel Models (Model 3) Predicting Response Times from Psychiatric

Predictor	Location-Only			Location-Scale		
	Estimate	SE	95% CI	Estimate	SE	95% CI
Mean Structure						
Correct Trial						
Intercept	557.46	5.36	546.94, 568.01	557.75	5.23	547.49, 568.05
BDI	-13.22	6.80	-26.54, 0.18	-12.98	6.85	-26.43, 0.65
PSWQ	17.89	7.20	3.94, 32.25	17.48	7.25	3.33, 31.80
YBCOS	6.05	6.11	-5.71, 18.25	6.02	6.08	-5.74, 18.07
Error Trial						
Intercept	666.77	5.97	655.09, 678.42	667.00	5.81	655.64, 678.42
BDI-II	-6.71	7.58	-21.59, 8.07	-6.65	7.56	-21.41, 8.42
PSWQ	19.41	8.02	3.79, 35.18	19.01	8.00	3.26, 34.70
YBCOS	5.32	6.84	-7.92, 18.88	5.39	6.77	-7.74, 18.78
Residual Variance						
(SD)	5.02	0.002	5.01, 5.02			
Variance Structure (SD)						
Correct Trials						
Intercept				4.94	0.02	4.91, 4.98
BDI				-0.03	0.02	-0.07, 0.02
PSWQ				0.08	0.02	0.03, 0.13
YBCOS				0.03	0.02	-0.01, 0.07
Error Trials						
Intercept				5.00	0.02	4.97, 5.03
BDI-II				-0.01	0.02	-0.06, 0.03
PSWQ				0.07	0.02	0.02, 0.12
YBCOS				0.04	0.02	0.00, 0.08

Random Effects						
Variance						
Mean Structure						
Correct Intercept			4194.93,			4118.25,
	5125.69	533.13	6277.75	5004.54	504.72	6105.69
Error Intercept			5270.44,			5098.44,
	6437.49	664.34	7863.51	6188.97	621.2	7524.80
Covariance			4272.67,			4155.66,
	5268.97	567.71	6494.55	5094.03	532.13	6245.89
Variance Structure						
(SD)						
Correct Intercept				0.23	0.01	0.21, 0.26
Error Intercept				0.23	0.01	0.21, 0.26
Covariance				0.05	0.01	0.04, 0.06
Model Diagnostics						
$\widehat{elpd}_{loo}$	-783,577.7			-777,009.9		
$SE(\widehat{elpd}_{loo})$	423.0			440.7		

Note: Parameters in standard deviations (SD) units are shown as log odds. MINI = Mini-International Neuropsychiatric Inventory; *SE* = standard error; 95% CI = 95% credible interval; BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; $\widehat{elpd}_{loo}$ = expected log predictive density

Figure 1

Waveforms and Topographical Maps for Controls and Psychopathology Groups with MINI-Confirmed Diagnoses

Note: see Supplementary Table 1 for summary characteristics of each group.

Figure 2

Scatterplots for the Relationship Between Error-Related Negativity and Psychiatric Symptom Measurements

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