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Neural Indicators of Error Processing in Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, and Major Depressive Disorder

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The ability to detect and respond to errors is critical to successful adaptation to a changing environment, and variation in error-related brain activity has been linked to psychopathology. The error-related negativity (ERN), an event-related potential component, represents a unique neural response to errors and is generated in the anterior cingulate cortex (ACC). In the present study, we measured the ERN in a sample of individuals with Generalized Anxiety Disorder (GAD), Obsessive Compulsive Disorder (OCD), Major Depressive Disorder (MDD), or some combination of the 3. Also included were 56 healthy control participants. Consistent with previous research, a diagnosis of GAD, only in the absence of a comorbid diagnosis of depression, was characterized by a larger ERN than controls. No such enhancement was evident in the depressed group, or the comorbid group, suggesting comorbid depression may eliminate the relationship between the ERN and GAD. Across all groups, symptoms of checking were associated with a larger ERN, whereas symptoms of psychomotor retardation were associated with a smaller ERN. The results of the present study indicate that interactions among transdiagnostic dimensions will likely need to be considered in the creation of neurobiologically informed classification schemes.

Keywords: error-related negativity, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Major Depressive Disorder

The ability to detect and respond to errors is critical to successful adaptation to a changing environment, and plays an essential role in the governance of goal-directed behavior (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Holroyd & Coles, 2002). Moreover, variation in both the ability to monitor errors and sensitivity to the commission of errors is evident across the population, and abnormalities in error monitoring have been implicated in multiple forms of psychopathology (e.g., Foti, Kotov, Bromet, & Hajcak, 2012; Gehring, Himle, & Nisenson, 2000; Hall, Bernat, & Patrick, 2007; Weinberg, Olvet, & Hajcak, 2010). Therefore, neural indices of error monitoring will likely be useful metrics in Research Domain Criteria (RDoC)-inspired research that seeks to link psychopathology to the dysfunction of core neural systems (Cuthbert, 2014; Cuthbert & Insel, 2010, 2013; Cuthbert & Kozak, 2013; Insel et al., 2010; Sanislow et al., 2010).

The error-related negativity (ERN) is an event-related potential that represents a neural response to errors. The ERN is maximal at central-frontal sites ~50 ms after the commission of an error, and reflects the early error-processing activity of the anterior cingulate cortex (ACC; Carter et al., 1998; Fitzgerald et al., 2005; van Veen & Carter, 2002). The ACC coordinates information across limbic, association, and motor cortices to effectively regulate behavior; one important role of the ACC involves the integration of information about punishment to guide behavior (Shackman et al., 2011). In particular, the ACC is richly innervated by dopaminergic neurons (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001), and a prominent model of the ERN suggests that it reflects dopaminergic disinhibition of neurons in the ACC when events are worse than anticipated (Holroyd & Coles, 2002). Consistent with this model, there is evidence that dopamine (DA) agonists enhance the ERN (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004), whereas DA antagonists attenuate it (de Bruijn et al., 2004; Zirnheld et al., 2004).

In addition, research on the psychometric properties of the ERN suggest that it is relatively trait-like, in that it is a stable (Foti, Kotov, & Hajcak, 2013; Meyer, Riesel, & Hajcak Proudfit, 2013; Olvet & Hajcak, 2009b) and reliable neural signal (Olvet & Hajcak, 2009a; Segalowitz et al., 2010; Weinberg & Hajcak, 2011) with excellent internal consistency (Riesel, Weinberg, Endrass, Meyer, & Hajcak, 2013). The magnitude of the ERN is also significantly heritable (Anokhin, Golosheykin, & Heath, 2008). Because it is a stable, heritable individual difference measure with direct referents in both neurobiology and behavior, some have suggested that the ERN will provide a basis for better understanding individual differences in cognition, personality, and psychopathology (Hajcak, 2012; Patrick & Bernat, 2010; Vaidyanathan, Nelson, & Patrick, 2012; Weinberg, Riesel, & Hajcak, 2012).

In particular, there is a well-documented association between an enhanced ERN and some forms of anxiety (e.g., Carrasco, Harbin, et al., 2013; Endrass, Klawohn, Schuster, & Kathmann, 2008; Fitzgerald et al., 2005; Gehring et al., 2000; Hajcak, McDonald, & Simons, 2003; Meyer, Weinberg, Klein, & Hajcak, 2012; Riesel,

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Endrass, Kaufmann, & Kathmann, 2011; Weinberg, Klein, & Hajcak, 2012; Weinberg et al., 2011). An enhanced ERN was first observed in Obsessive Compulsive Disorder (OCD; Gehring et al., 2000), a result which has since been replicated over 15 times (e.g., Carrasco, Harbin, et al., 2013; Endrass et al., 2008; Endrass et al., 2010; Hajcak, Franklin, Foa, & Simons, 2008; Riesel et al., 2011). Indeed, this finding is so robust that an enhanced ERN has been proposed as a viable endophenotype for OCD (Hajcak et al., 2008; Manoach & Agam, 2013; Olvet & Hajcak, 2008; Riesel et al., 2011).

However, an enhanced ERN has also been observed in Generalized Anxiety Disorder (GAD; Carrasco, Hong, et al., 2013; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; Weinberg, Klein, et al., 2012; Weinberg et al., 2011; Xiao et al., 2011). Though GAD and OCD are frequently comorbid with one another (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), and share multiple features (e.g., worry, obsessions, and intolerance of uncertainty; Holaway, Heimberg, & Coles, 2006; Nitschke et al., 2009), there are also important clinical distinctions (Watson, 2005); indeed, OCD is no longer classified as an anxiety disorder in the *Diagnostic and Statistical Manual for Mental Disorders-Fifth Edition* (*DSM-5*; APA, 2013), though GAD is. Thus, it is not yet clear what psychological phenotype might be common to both GAD and OCD and be associated with an increased ERN.

On the other hand, depression is also frequently comorbid with both OCD and GAD (Brown et al., 2001) and shares multiple clinical features (e.g., worry, checking; Fresco, Frankel, Mennin, Turk, & Heimberg, 2002; Segerstrom, Tsao, Alden, & Craske, 2000; Watson, Gamez, & Simms, 2005; Wu & Watson, 2005). In fact, some have proposed that GAD and MDD are essentially redundant constructs and would be better represented by a single dimension (e.g., Watson, 2005). However, despite compelling evidence for an enhanced ERN in OCD and GAD, the literature regarding the ERN in depression has been mixed. Whereas some studies have reported an enhanced ERN associated with a diagnosis of depression (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008, 2010), others have reported either no relation, or an attenuated ERN (Compton et al., 2008; Olvet, Klein, & Hajcak, 2010; Ruchsow et al., 2006; Schrijvers et al., 2008; 2009). However, the majority of these studies have not adequately considered the influence of comorbid or concurrent anxiety. In a recent report, we documented that whereas GAD alone was associated with an enhanced ERN, a comorbid diagnosis of depression appeared to eliminate this association (Weinberg, Klein, et al., 2012).

In summary, the extant research on the ERN points toward similarities (e.g., between GAD and OCD) and distinctions (e.g., between GAD and MDD) that are not readily apparent in many nosological models. Moreover, variation in the ERN may relate to phenotypes that cut across multiple disorders; however, data supporting this notion are limited. Some have proposed that an enhanced ERN reflects excessive error signaling in the ACC, which might be responsible for the increased self-monitoring and compulsive compensatory behaviors seen in OCD and to a lesser extent GAD (e.g., obsessions, checking; Gehring et al., 2000). On the other hand, a recent meta-analysis of 37 studies examining the association between anxiety and the ERN proposed that anxious apprehension (i.e., worry) is the dimension of anxiety most closely associated with error monitoring, and found effect sizes of $r = -0.35$ for this dimension relative to nonspecific measures of anxiety ($r = -0.09$; Moser, Moran, Schroder, Donnellan, & Yeung, 2013). We have proposed that the magnitude of the ERN likely tracks multiple phenotypes (Weinberg, Riesel, et al., 2012). For instance, given the role the DA system plays in the processing of errors (Holroyd & Coles, 2002), dopaminergic dysfunction associated with depression and anhedonia (Martinot et al., 2001; Nestler & Carlezon, 2006) might interfere with the extent to which performance monitoring, and the enhanced ERN, is engaged at all (Weinberg, Klein, et al., 2012; Weinberg, Riesel, et al., 2012). In fact, symptom profiles related to lower dopamine functioning (Martinot et al., 2001), such as high psychomotor retardation (Schrijvers et al., 2008), and psychomotor poverty (Bates et al., 2002; Foti et al., 2012) have also been associated with a blunted ERN. In general, given the heterogeneity of these psychopathological conditions, it is informative to consider specific dimensions of dysfunction and not only the disorder categories (Mataix-Cols, do Rosario-Campos, & Leckman, 2005; Watson, 2009; Watson et al., 2007); moreover, it seems increasingly important to look simultaneously at multiple phenotypic dimensions (e.g., Foti et al., 2012; Weinberg et al., 2012). However, this approach has rarely been used to investigate clinical correlates of the ERN.

Through the present study, we sought to specify psychological phenotypes that might relate to performance monitoring and the ERN, as well as to further clarify how these phenotypes might interact to determine the magnitude of the ERN. The sample was drawn from a large and diverse psychiatric outpatient sample, and consisted of individuals with a diagnosis of either GAD, OCD, or MDD, or some combination of the three diagnoses, as well as a group of healthy controls (i.e., no past or current Axis I disorders). In keeping with the principles of RDoC, comorbid diagnoses were not ruled out (Sanislow et al., 2010). Consistent with our previous work, we hypothesized that diagnoses of GAD and OCD would be associated with an enhanced ERN relative to the controls, but not when a comorbid diagnosis of depression was also present. Further, we did not expect individuals with pure depression (i.e., MDD without GAD or OCD) to differ from the controls in terms of the ERN. In more exploratory analyses, we then sought to link variation in the ERN to transdiagnostic symptom dimensions. Based on previous evidence, we expected that worry, obsessions, and checking would be associated with an enhanced ERN (Gehring et al., 2000; Moser et al., 2013), whereas anhedonia and psychomotor retardation would be associated with a decreased ERN (Bates et al., 2002; Foti et al., 2012; Schrijvers et al., 2008).

Method

Participants

Psychiatric patients ($N = 316$) were recruited from outpatient Psychology and Psychiatry clinics at Stony Brook University, local community mental health centers, and assisted-living facilities and community programs for the mentally ill. We focus the current analyses on 106 individuals out of this sample who met diagnostic criteria for any of the following diagnoses: GAD, OCD, or MDD. Patients without one or more of these diagnoses, or any individuals who met criteria for current psychosis or a current manic episode, were excluded from the analysis. Healthy controls were recruited in one of two ways: one group ($N = 26$) were patients who presented at Stony Brook University Medical Center for treatment of chronic

medical conditions (e.g., diabetes mellitus, ischemic heart disease, chronic obstructive pulmonary disease, or autoimmune disorders) and had no current or lifetime psychiatric disorders. Another group (N = 34) was recruited from the community via electronic and print advertisements, and similarly had no current or lifetime psychiatric disorders. In total, 187 participants were eligible for the current analyses. Of these, 18 participants were excluded because they committed fewer than six errors (per Olvet & Hajcak, 2009b), or were correct less than 45% of the time (this calculation included no-response trials as well as errors), and 11 were excluded as a result of excessive noise in the ERP data. The study was approved annually by the institutional review board of Stony Brook University.

The final sample was 63% female (n = 102), primarily White American (76%, n = 124), and middle-aged (Age M = 42.01, SD = 14.01). Overall, 38% (n = 62) of the sample had a current diagnosis of MDD; 36% had a current diagnosis of GAD (n = 58), 16% had a current diagnosis of OCD (n = 26), and 30% of the sample did not meet current or past criteria for any Axis I disorder (n = 56). Table 1 shows descriptive data associated with each diagnosis (allowing for diagnostic overlap).

Table 1
Demographic and Clinical Characteristics of Healthy Comparison Participants and Each of the Three Clinical Groups

Characteristic	MDD (N = 62)	GAD (N = 57)	OCD (N = 26)	Healthy controls (N = 56)
Demographics				
Female N (%)	39 (63%)	42 (72%)	18 (69%)	31 (55%)
Age M (SD)	41.55 (13.09)	43.02 (13.52)	42.96 (11.69)	41.38 (15.94)
White N (%)	48 (77%)	47 (81%)	20 (77%)	39 (70%)
Functioning				
Employed N (%)	13 (21%)	21 (36%)	13 (50%)	36 (64%)
On disability N (%)	21 (34%)	23 (39 %)	14 (54%)	4 (7%)
GAF M (SD)	49.29 (6.25)	52.69 (8.80)	50.88 (11.58)	79.97 (9.38)
				Healthy controls (N = 29)
IMAS Scales				
Dysphoria	7.29 (2.38)	6.05 (3.65)	5.08 (3.53)	.43 (1.35)
Lassitude	8.65 (2.25)	7.58 (3.24)	6.54 (3.47)	.97 (1.82)
Anhedonia	8.66 (2.64)	7.53 (3.89)	8.27 (3.34)	1.24 (2.55)
Insomnia	5.77 (2.27)	4.70 (2.95)	5.42 (2.66)	1.72 (2.37)
Suicidality	2.56 (2.22)	2.19 (2.22)	2.69 (2.59)	.45 (.83)
Appetite loss	4.03 (2.10)	3.35 (2.53)	3.96 (2.57)	.86 (1.75)
Agitation	5.66 (3.58)	5.28 (3.22)	5.38 (3.20)	.86 (2.03)
Retardation	3.61 (2.99)	3.40 (3.29)	3.00 (2.83)	.38 (1.15)
Excessive worry	3.85 (2.03)	3.79 (2.27)	3.85 (2.01)	.28 (.88)
GAD symptoms	9.48 (2.07)	8.39 (3.45)	7.77 (3.65)	1.24 (2.03)
Cleaning	.92 (2.03)	1.26 (2.57)	4.42 (3.83)	.04 (.27)
Rituals	1.10 (1.69)	1.93 (2.80)	4.81 (4.23)	.13 (.66)
Checking	2.48 (2.89)	2.88 (2.97)	4.96 (3.16)	.11 (.37)
Obsessions	4.08 (3.52)	4.05 (3.42)	5.04 (3.89)	.38 (1.08)
Irritability	7.48 (3.93)	6.56 (4.28)	6.65 (4.71)	1.41 (2.73)
Current disorders no. (%)				
MDD	62 (100%)	27 (47%)	4 (15%)	0
GAD	27 (44%)	57 (100%)	12 (46%)	0
OCD	4 (6.5%)	12 (21%)	26 (100%)	0
Agoraphobia	13 (21%)	14 (24%)	9 (34%)	0
Social Anxiety Disorder	14 (23%)	20 (35%)	9 (34%)	0
PTSD	14 (23%)	10 (17%)	7 (27%)	0
Specific phobia	25 (40%)	26 (45%)	15 (58%)	0
Panic disorder	23 (37%)	18 (31%)	8 (31%)	0
Lifetime disorders no. (%)				
MDD	62 (100%)	41 (71%)	9 (35%)	0
Panic	26 (42%)	22 (38%)	13 (50%)	0
Agoraphobia	15 (24%)	15 (26%)	10 (39%)	0
Social Anxiety Disorder	17(27%)	22 (38%)	4 (29%)	0
Specific phobia	25 (40%)	26 (45%)	15 (58%)	0
OCD	4 (7%)	12 (21%)	26 (100%)	0
PTSD	14 (23%)	11 (19%)	8 (31%)	0
ERPs (μV)				
ERN	−.66 (5.57)	−2.32 (6.62)	−.21 (5.88)	.38 (5.43)
CRN	3.26 (5.73)	3.62 (5.56)	5.07 (7.00)	4.94 (4.50)
ΔERN	−3.92 (4.97)	−5.94 (6.11)	−5.28 (7.02)	−4.56 (5.42)

Note. GAF = Global Assessment of Functioning; MDD = Major Depressive Disorder; OCD = Obsessive-Compulsive Disorder; GAD = Generalized Anxiety Disorder; PTSD = posttraumatic stress disorder; ERN = error-related negativity; CRN = correct-related negativity; ΔERN = ERN minus CRN. We also examined levels of checking in GAD and MDD when a diagnosis of OCD was not present. The means were as follows: GAD without OCD (M = 2.22, SD = 2.70), MDD without OCD (M = 2.29 SD = 2.84).

Measures

Once in the lab, all participants were administered the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (DSM) fourth edition (SCID; Spitzer, Williams, Gibbon, & First, 1992). The SCID is a well-validated semistructured interview for current and past *DSM–IV* Axis I diagnoses. The SCID was administered by one of five master's-level clinicians. Interrater reliability was assessed based on 21 participants in this study; the second randomly selected clinician rated video-recorded interviews blind to original ratings. For each of the three diagnoses of interest (i.e., MDD, GAD, and OCD) κs were 1.00. All diagnostic-level information reported here is based on the SCID results.

In addition, participants were administered the Interview for Mood and Anxiety Symptoms (IMAS; Kotov, Perlman, Gamez, & Watson, 2014; Watson et al., 2012). Like the SCID, the IMAS provides information on symptoms in the past month. However, every question is asked of every participant (i.e., there are no skip-out rules), permitting dimensional assessment of phenotypes. The IMAS was designed to cover all *DSM–IV* mood and anxiety disorder symptom criteria. Based on data from prior studies, the interview was revised to improve its symptom coverage (Watson et al., 2012). The IMAS includes 10 primary scales corresponding to major *DSM–IV* anxiety and mood syndromes, plus irritability. It also includes 32 subscales that assess lower-order dimensions. We report here on 13 subscales within the GAD, OCD, and depression domains: dysphoria (5 items; $\alpha = .79$), lassitude (5 items; $\alpha = .82$), anhedonia (6 items; $\alpha = .81$), insomnia (4 items; $\alpha = .73$), suicidality (4 items; $\alpha = .63$), appetite loss (3 items; $\alpha = .84$), agitation (5 items; $\alpha = .77$), retardation (5 items; $\alpha = .72$), excessive worry (5 items; $\alpha = .84$), additional GAD symptoms (7 items; $\alpha = .81$), cleaning (5 items; $\alpha = .79$), rituals (6 items; $\alpha = .81$), and checking (4 items; $\alpha = .83$). The IMAS was administered by lay interviewers, each of whom completed a 20-hr training program and was certified based on observation of interviews and reliability of ratings. The training included modules on establishing rapport, distinguishing between clinically significant and normative responses, probing techniques, and the specific content of the IMAS. Individual items are scored on a 3-point rating scale (*absent*, *subthreshold*, and *above threshold*). All interviews in the present study were recorded; a randomly selected interviewer blindly rescored 34 tapes. Interrater reliability was excellent, with ICCs (one-way random model, absolute agreement, and single measure) ranging from .93 to .99 across the scales included here. In controls, IMAS data were only available for 27 participants.

Task and Materials

An arrow version of the flanker task (Eriksen & Eriksen, 1974) was administered on a Pentium D class computer, using Presentation software (Neurobehavioral Systems, Inc., Albany, CA) to control the presentation and timing of all stimuli. Each stimulus was displayed on a 19 in (48.3 cm) monitor. On each trial, five horizontally aligned arrowheads were presented. Half of all trials were compatible ("< < < < <" or "> > > > >") and half were incompatible ("< < > < <" or "> > < > >"). The order of compatible and incompatible trials was random. Each set of arrowheads occupied approximately 1.3 degrees of visual angle vertically and 9.2 degrees horizontally. All stimuli were presented for 200 ms followed by an intertribal interval (ITI) that varied randomly from 2,300 to 2,800 ms.

Procedure

After informed consent and a brief description of the experiment, EEG electrodes were attached and the subject was given detailed task instructions. All participants performed multiple tasks during the experiment. The order of the tasks was counterbalanced across participants and the results of other tasks will be reported elsewhere. Participants were seated at a viewing distance of approximately 24 in (61 cm) and were instructed to press the right mouse button if the center arrow was facing to the right and to press the left mouse button if the center arrow was facing to the left. Information about each response (e.g., reaction time [RT], accuracy), was recorded. Participants performed a practice block containing 30 trials during which they were instructed to be both as accurate and fast as possible. The actual task consisted of 11 blocks of 30 trials (330 trials total) with each block initiated by the participant. Participants received feedback based on their performance at the end of each block. If performance was 75% correct or lower, the message "Please try to be more accurate" was displayed. Performance above 90% correct was followed by "Please try to respond faster." If performance was between 75 and 90% correct, the message "You're doing a great job" was displayed.

Psychophysiological Recording, Data Reduction, and Analysis

Continuous EEG recordings were collected using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). Thirty-four electrode sites were used, based on the 10/20 system, as well as two electrodes on the right and left mastoids. Electrooculogram (EOG) generated from eye movements and eyeblinks was recorded using four facial electrodes: horizontal eye movements (HEM) were measured via two electrodes located approximately 1 cm outside the outer edge of the right and left eyes. Vertical eye movements (VEM) and blinks were measured via two electrodes placed approximately 1 cm above and below the right eye. The EEG signal was preamplified at the electrode to improve the signal-to-noise ratio by a BioSemi ActiveTwo system (BioSemi, Amsterdam). The data were digitized at 24-bit resolution with a LSB value of 31.25 nV and a sampling rate of 1,024 Hz, using a low-pass fifth order sinc filter with −3dB cutoff point at 208 Hz. Each active electrode was measured online with respect to a common mode sense (CMS) active electrode, located between PO3 and POz, producing a monopolar (nondifferential) channel. CMS forms a feedback loop with a paired driven right leg (DRL) electrode. Offline, all data were referenced to the average of the left and right mastoids, and band-pass filtered with low and high cutoffs of 0.1 and 30 Hz, respectively. Eyeblink and ocular corrections were conducted using both VEM and HEM channels per a modification of the original algorithm published in Gratton, Coles, and Donchin (1983).

A semiautomatic procedure was used to detect and reject artifacts. Data from individual channels were rejected if a voltage step of more than 50.0 μV between sample points or a voltage difference of 300.0 μV within a trial existed. In addition, data were

identified as artifacts if a voltage difference of less than .50 μV within 100 ms intervals was present. Visual inspection of the data was then conducted to detect and reject any remaining artifacts.

The EEG was segmented for each trial beginning 300 ms before response onset and continuing for 1,300 ms (i.e., 1,000 ms after the response); a 200 ms window from −300 to −100 ms before the response onset served as the baseline. Correct and error trials were averaged separately. Because this sample was characterized by greater variability in behavioral and ERP latencies than is typically observed in less diverse samples (e.g., Weinberg, Klein, & Hajcak, 2012), and because the ERN can begin before the registration of the motoric response, defining the ERN and CRN as the average activity in a preset time-window did not adequately capture activity associated with these components. However, peak measures tend not to adequately or accurately capture the underlying components (Luck, 2014). Therefore, for each subject, the most negative peak in a time window from 50 ms before response onset to 100 ms after the response was detected on error trials. Because the width of the ERN component is typically between 80 ms and 100 ms, the ERN was then quantified as the average activity in the 100 ms around this peak (i.e., 50 ms on either side of the peak) on error trials at Cz, where error-related brain activity was maximal. In addition, the correct response negativity (CRN) was evaluated in the same time window and electrode on correct trials. Measuring the area around a peak has the advantage of utilizing an individually based ERN latency, while also being an area measure that is not biased in favor of a single value (Luck, 2014).

Because the CRN appears to measure generic response monitoring (e.g., Simons, 2010), and a negative deflection is typically present on both error and correct trials (e.g., Burle, Roger, Allain, Vidal, & Hasbroucq, 2008), it is often critical to examine the difference between the ERN and CRN (i.e., ΔERN) to quantify neural activity unique to error-processing. Difference scores for error minus correct trials (ΔERN) were calculated for each subject subtracting CRN from ERN (Riesel, Weinberg, Endrass, et al., 2013).

Behavioral measures included both the number of error trials for each subject, and accuracy expressed as a percentage of trials with correct responses. Average RTs on error and correct trials were also calculated separately. Number of errors, accuracy on trials after errors, and posterror RT were also evaluated to determine if there were group differences in posterror behavior. Trials were removed from analysis if RTs were faster than 200 ms or slower than 1,000 ms.

Results

Behavioral Data

Accuracy, RT data, posterror accuracy, and posterror RT data are presented in Table 2. Results of a repeated-measures analysis of variance (ANOVA) indicate that RT varied as a function of accuracy $F(1, 160) = 223.05$, $p < .0001$, such that participants were faster on error than correct trials. Similarly, participants were slower on trials that occurred after an error than on trials occurring after a correct response $F(1, 160) = 6.75$, $p = .01$.

Table 2 also displays Pearson's correlations between behavioral measures and clinical variables. As indicated, a diagnosis of depression was associated with increased RTs and decreased accuracy. Similarly, multiple symptom scales typically linked to depression were associated with increased RTs and decreased accuracy (i.e., psychomotor retardation, dysphoria, anhedonia, insomnia, appetite loss, and irritability). Symptoms of GAD were also associated with decreased accuracy, and a diagnosis of GAD was associated with increased posterror slowing. A diagnosis of OCD was not significantly related to any behavioral measures, nor were any OCD symptom scales on the IMAS, with the exception of self-reported cleaning, that was associated with slower responses on correct trials only.

Associations Between Error-Related Brain Activity and Diagnostic-Level Data

Bivariate associations between the ERN, CRN, ΔERN, and clinical variables are also presented in Table 2. A hierarchical multiple regression analysis was calculated to examine effects of diagnosis on the ERN. To isolate variance specific to error processing, and not generic performance monitoring, the CRN was entered in Step 1, followed by diagnoses (i.e., MDD, GAD, and OCD) in Step 2 and interactions between diagnoses to examine comorbidity (i.e., OCD × GAD, GAD × MDD, and MDD × OCD) in Step 3.[1] Results are presented in Table 3.

The effect of the CRN predicting the ERN was significant, $t(160) = 6.72$, $p < .0001$. In the first step, there was also a significant main effect of GAD $t(160) = 2.35$, $p = .02$, such that a diagnosis of GAD was associated with a larger (more negative) ERN. Additionally, there was a trend-level effect of MDD in the first step $t(160) = 1.72$, $p = .08$, such that a diagnosis of MDD was associated with a blunted (less negative) ERN. The effect of OCD was not significant. The effect of GAD remained significant in the final model, $t(160) = 3.54$, $p = .001$, but was qualified by a significant interaction between GAD and MDD, $t(160) = 2.38$, $p = .02$.

To interpret the MDD by GAD interaction, the model was calculated for each level of current MDD. For individuals with no current depression, a diagnosis of GAD was associated with an increased ERN, $\beta = -.27$, $t(98) = 3.00$, $p = .003$. For those individuals with a current diagnosis of depression, however, the effect of GAD was not significant, $\beta = .03$, $t(98) = .26$, $p = .80$.[2]

Figure 1 displays grand average response-locked ERPs at Cz by diagnosis, displaying the ERPs in individuals with no current diagnoses (HC), individuals with MDD but no GAD or OCD (MDD), individuals with GAD but no MDD (regardless of comorbid diagnosis of OCD; labeled GAD), individuals with comorbid GAD and MDD (regardless of comorbid diagnosis of OCD; labeled GAD/MDD) and individuals with OCD but no MDD (regardless of comorbid diagnosis of GAD; labeled OCD) and individuals with comorbid OCD and MDD (regardless of comorbid diagnosis of GAD; labeled MDD/OCD). Topographic maps are presented in Figure 2 for individuals who fell into the HC, GAD,

[1] Because the individuals in this report also had a number of comorbid diagnoses, we tested independent effects of social anxiety disorder, agoraphobia, panic disorder, specific phobia, and posttraumatic stress disorder (PTSD). There were no independent effects of any of these disorders (all $ps > .22$).

[2] Among individuals with current GAD and no current MDD, neither the ERN $t(29) = .69$, $p = .49$ nor the ΔERN $t(29) = .25$, $p = .80$ differentiated those with a history of MDD and those without.

Table 2
Descriptive Statistics and Bivariate Associations Between Clinical Variables and Behavioral and Neural Measures of Error-processing

		Diagnoses			IMAS Scales														
	Mean (*SD*)	MDD	OCD	GAD	Check	PR	Dys	Lass	Anh	Ins	Suic	App	Agit	Worry	GAD Symp	Clean	Rituals	Obs	Irrit
Reaction time (ms)																			
Error trials	390.46 (104.60)	.22**	−.02	.05	−.05	.13	.18*	.09	.12	.13	.01	.18*	−.02	.07	.11	.01	−.02	−.09	.08
Correct trials	492.50 (100.60)	.21**	−.03	.14	−.01	.10	.22*	.14	.13	.10	.06	.18*	.02	.11	.09	.19*	.004	.02	.10
Accuracy																			
No. of errors	35.98 (31.34)	.16*	.12	.01	−.05	.29**	.09	.12	.17	.21*	.01	.18*	.07	.05	.19*	−.004	.05	−.06	.17
No. of correct trials	290.52 (35.77)	−.17*	−.11	−.03	.01	−.32**	−.09	−.13	−.19*	−.17*	−.01	−.21*	−.11	−.08	−.19*	−.01	−.08	.03	−.20*
Post-trial reaction time (ms)																			
Post-error trials	504.99 (124.90)	.29**	−.03	.16*	.03	.11	.30**	.17	.15	.14	.07	.21*	.05	.10	.11	.22**	.02	.04	.13
Post-error accuracy																			
No. of errors	7.44 (15.93)	.12	.04	.04	−.07	.24**	.08	.13	.12	.20*	−.03	.17	.05	.05	.18*	−.03	.03	−.10	.19*
% correct	88.35 (13.64)	−.09	−.03	−.08	.04	−.26**	−.03	−.11	−.12	−.16	.01	−.16	−.08	−.04	−.14	.03	−.08	.07	−.17
ERPS (μV)																			
ERN	−.89 (6.02)	.03	.05	−.18*	−.04	.13	.001	−.004	−.04	.03	.12	.04	.04	.05	.05	−.14	−.05	−.07	.05
CRN	4.14 (5.42)	−.13	.08	−.07	.09	−.14	−.04	−.10	−.09	−.08	−.006	−.12	−.10	−.05	−.07	−.15	.04	−.01	−.12
ΔERN	−5.03 (5.93)	.15	−.02	−.11	−.13	.29*	.04	.10	.05	.11	.13	.13	.14	.11	.13	.001	−.09	−.06	.17
ERN (partial *r* controlling for CRN)	—	.10	.02	−.16*	−.11	.25**	.12	.06	.02	.08	.14	.13	.11	.10	.11	−.05	−.06	−.08	.10

Note. IMAS = Interview for Mood and Anxiety Symptoms; ERP = Event-Related Potentials; ERN = error-related negativity; CRN = Correct-Related Negativity; ΔERN = ERN minus CRN; MDD = Major Depressive Disorder; OCD = Obsessive-Compulsive Disorder; GAD = Generalized Anxiety Disorder; Check = checking; PR = psychomotor retardation; Dys = dysphoria; Lass = lassitude; Anh = anhedonia; Ins = insomnia; Suic = suicidality; App = appetite loss; Agit = agitation; GAD symp = GAD symptoms; Clean = cleaning; Rit = rituals; Obs = obsessions; Irrit = irritability.
* $p < .05$. ** $p < .01$.

Table 3
Hierarchical Regression With the Correct-Related Negativity (CRN), SCID Diagnoses of Obsessive Compulsive Disorder (OCD), Major Depressive Disorder (MDD), and Generalized Anxiety Disorder (GAD), and Interactions Between Diagnoses Predicting the Error-Related Negativity (ERN)

Predictor	b (*SE*)	Entry β	Final β
1. Neural response			
CRN	.52 (.08)	.47**	.46**
2. Diagnoses			
OCD	.91 (1.16)	.06	−.06
GAD	−2.06 (.88)	−.17*	−.37**
MDD	1.52 (.88)	.12†	−.03
3. Interactions between diagnoses			
OCD × GAD	4.00 (2.65)	—	.17
GAD × MDD	4.52 (1.90)	—	.28*
OCD × MDD	−.87 (3.54)	—	−.02
	Total model R^2 = .29		

† $p < .10$. * $p < .05$. ** $p < .01$.

OCD, MDD, GAD/MDD, and MDD/OCD groups. These maps depict voltage differences (in μV) across the scalp for error minus correct responses in the time window of the ERN.

Associations With Transdiagnostic Dimensions

Partial bivariate associations between the ERN and IMAS scales, controlling for the CRN, were examined across all groups and are reported in Table 2. As indicated in Table 2, only increased psychomotor retardation was significantly associated with the magnitude of the ERN, such that greater psychomotor retardation was associated with a blunted (i.e., less negative) ERN. However, dysphoria, suicidality, appetite loss, agitation, excessive worry, GAD symptoms, and checking were each related to the ERN at a trend level. All correlations with an absolute value exceeding .10 were entered into a multiple regression as simultaneous predictors, along with the CRN (see Table 4). This model significantly predicted the magnitude of the ERN $R^2 = .37$, $F(10, 123) = 7.18$, $p < .001$. However, among the IMAS scales, only symptoms of psychomotor retardation β = .21, $t(123) = 2.28$, $p = .03$ and checking β = −.18, $t(123) = 2.15$, $p = .03$ remained significant *unique* predictors, with increased retardation related to diminished ERN and increased checking related to enhanced ERN (see Figure 3). Additionally, we conducted 36 hierarchical regressions with all nine IMAS scales included in the first step of the regression, and each of the two-way interaction terms between the scales included in the second step of the regression (i.e., each interaction term was entered on its own in each of the regressions), and found no interactions that were significant after correction for multiple tests.

Finally, we also examined whether elevated checking was unique to a diagnosis of OCD. Although individuals with OCD endorsed higher levels of checking behavior relative to those with no diagnosis of OCD $F(1, 143) = 47.42$, $p < .001$ (see Table 1 for means), individuals with a diagnosis of GAD but no OCD $F(1, 112) = 7.85$, $p < .01$, and MDD but no OCD $F(1, 112) = 13.67$, $p < .001$ also reported increased levels of checking behavior relative to those without these diagnoses. Self-reported levels of checking in Healthy Controls also differed significantly from zero $t(28) = 2.27$, $p < .05$.

Discussion

In the present study, we sought to examine the ERN across diagnostic boundaries by extending previous work on GAD, OCD, and MDD, as well as to examine whether the ERN relates to dimensional phenotypes that cut across these disorders. In the traditional diagnosis-based analyses, we replicated evidence for an enhanced ERN in individuals with GAD (Ladouceur et al., 2006; Weinberg, Klein, et al., 2012; Weinberg et al., 2011; Xiao et al., 2011), as well as evidence that the association between GAD and the ERN is affected by the presence of comorbid depression

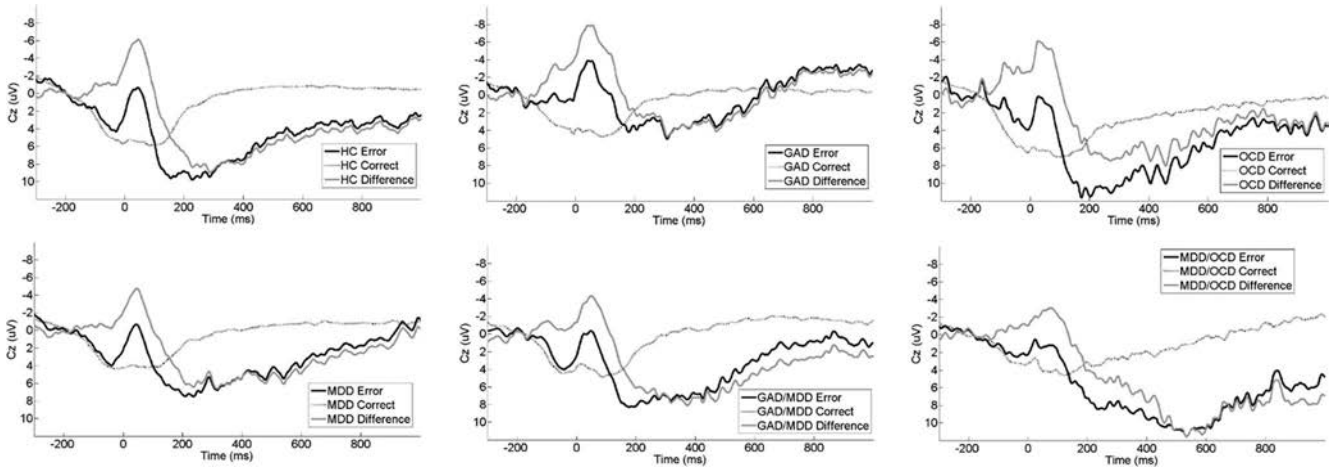

Figure 1. Response-locked Event-Related Potential (ERP) waveforms at electrode site Cz for Healthy Controls (HC), Generalized Anxiety Disorder (GAD), Obsessive-Compulsive Disorder (OCD), Major Depressive Disorder (MDD), Comorbid GAD and MDD (GAD/MDD), and Comorbid OCD and MDD (OCD/MDD). For each panel, response onset occurred at 0 ms. In addition to raw waveforms for correct and error responses, each panel also depicts the error-correct difference (solid gray line). Per ERP convention, negative voltages are plotted up.

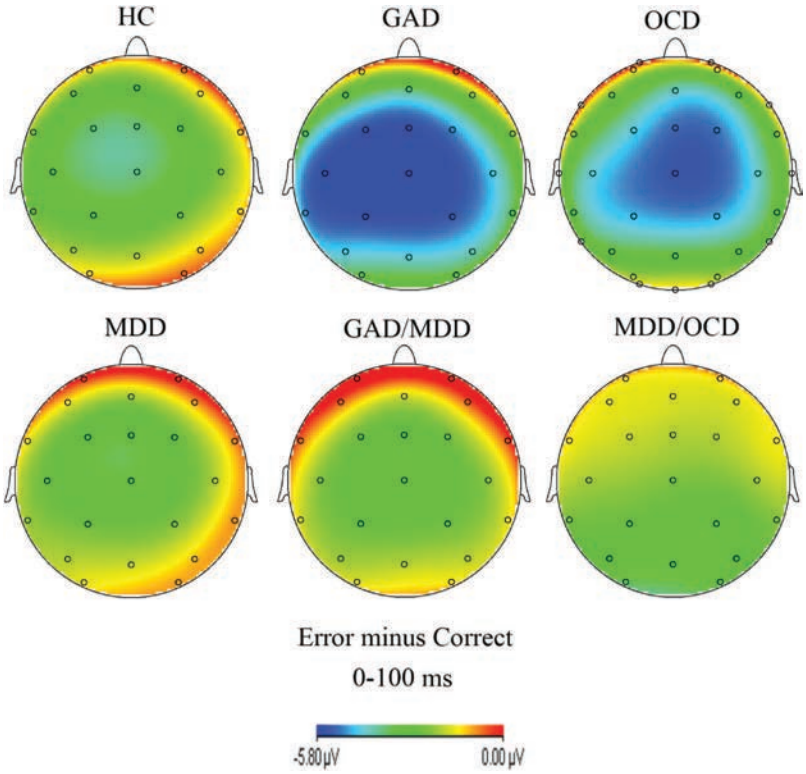

Figure 2. Scalp topographies representing the error-related negativity (ERN). These maps are derived from the average difference (error minus correct response) for Healthy Controls (HC), Generalized Anxiety Disorder (GAD), Obsessive-Compulsive Disorder (OCD), Major Depressive Disorder (MDD), Comorbid GAD and MDD (GAD/MDD), and Comorbid OCD and MDD (OCD/MDD) groups. See the online article for the color version of this figure.

(Weinberg, Klein, et al., 2012). By fully crossing diagnoses of GAD and MDD, these results indicate that the ERN is enhanced in some forms of anxiety, but not anxiety with comorbid depression, and not depression alone. This was the case despite that fact that MDD and GAD were both associated with elevated scores on symptom measures of depression and generalized anxiety. Although GAD and MDD are sometimes thought to be redundant constructs, they appear to differ in terms of neural response to errors. On the other hand, a diagnosis of OCD was not associated with significantly enhanced error processing in the current sample, though this diagnostic group had a larger difference between the ERN and CRN than controls or individuals with depression. It is plausible that the failure to detect a significant effect in the sample is due in part to the relatively small number of individuals with OCD but no GAD or MDD ($N = 14$).

Table 4
Simultaneous Regression With Interview for Mood and Anxiety Symptoms (IMAS) Scales Predicting the Error-Related Negativity (ERN)

Predictor	b (*SE*)	β	*t*
CRN	.64 (.08)	.61	8.11**
Retardation	.41 (.18)	.21	2.28*
Checking	−.37 (.17)	−.18	2.15*
Appetite loss	.19 (.22)	.08	.85
Agitation	.13 (.16)	.08	.81
GAD symptoms	−.13 (.19)	−.09	.70
Irritability	.08 (.13)	.06	.62
Dysphoria	−.09 (.19)	−.06	.48
Suicidality	.23 (.25)	.08	.92
Excessive worry	.05 (.27)	.02	.19
	Total model $R^2 = .37$		

Note. CRN = Correct-Related Negativity; GAD = Generalized Anxiety Disorder.
* $p < .05$. ** $p < .01$.

Perhaps more importantly, each of these diagnostic groups represents a heterogeneous collection of individuals. Thus, we also examined the ERN in relationship to dimensional phenotypes that cut across diagnostic categories. First, contrary to our expectations, obsessions and worry did not relate to the magnitude of the ERN (cf. Moser et al., 2013). On the other hand, more checking behaviors were associated with an enhanced ERN across all groups, consistent with theories that excessive error signals generated by the ACC underlie the constant self-monitoring and compulsive compensatory behaviors often seen in OCD (Gehring et al., 2000; Pitman, 1987). However, checking behaviors are not unique to OCD. Indeed, in comparison to other compulsive behaviors (e.g., cleaning, symmetry/orderliness compulsions), checking appears more closely related to symptoms like worry or obsessive thoughts (Schut, Castonguay, & Borkovec, 2001; Summerfeldt, Richter,

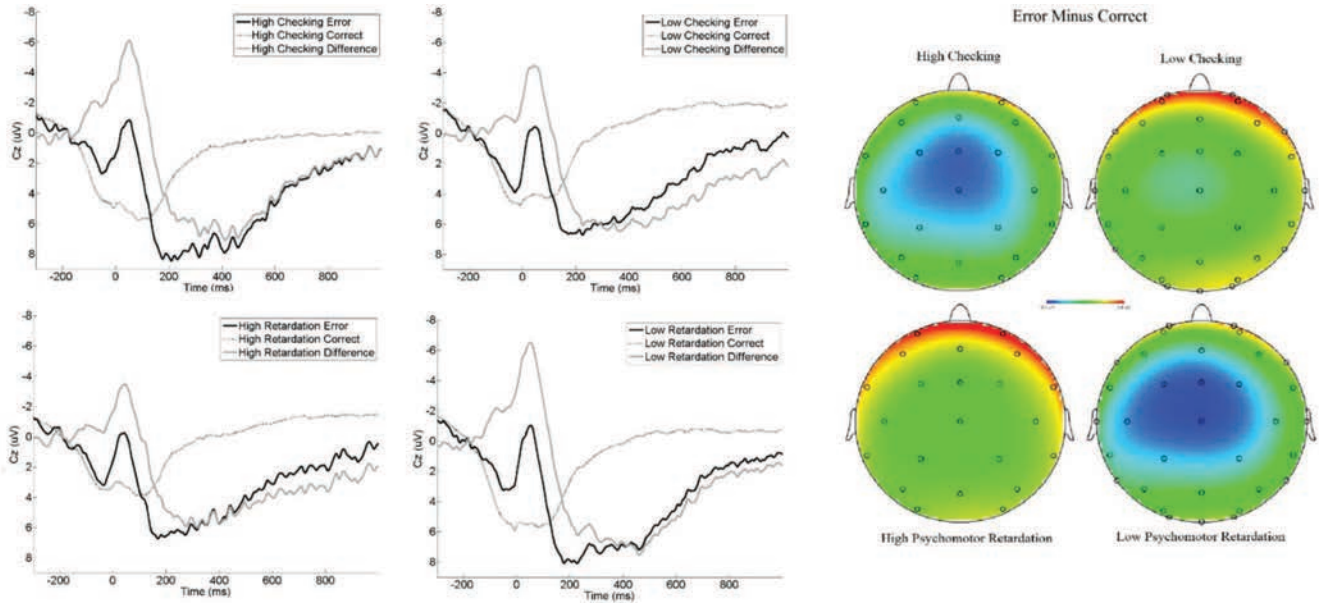

Figure 3. Response-locked Event-Related Potential (ERP) waveforms at electrode site Cz for individuals high and low on checking (top) and high and low on psychomotor retardation (bottom). For each panel, response onset occurred at 0 ms. In addition to raw waveforms for correct and error responses, each panel also depicts the error-correct difference (solid gray line). Per ERP convention, negative voltages are plotted up. Also shown are scalp topographies representing the error-related negativity (ERN). These maps are derived from the average difference (error minus correct response; Δ ERN) and represent the Δ ERN for individuals high and low on checking (top) and high and low on psychomotor retardation (bottom). See the online article for the color version of this figure.

Antony, & Swinson, 1999). Furthermore, compared with other symptoms typically associated with OCD, checking appears to be relatively nonspecific: checking demonstrates greater overlap with other forms of psychopathology, including GAD (Schut et al., 2001) and depression (Watson, 2005; Wu & Watson, 2005), as well as stronger associations with the personality dimensions of neuroticism and negative affectivity (Watson et al., 2005). Consistent with these data, patterns of self-report in the current study showed elevated checking associated with a diagnosis of OCD. However, checking was also elevated in individuals with GAD and MDD without a comorbid diagnosis of OCD, and the mean for checking was nonzero even in the healthy controls. Additionally, a *reduced* ERN has consistently been demonstrated in studies examining traits and disorders (e.g., psychosis, externalizing spectrum disorders; Foti et al., 2012; Hall et al., 2007; Mathalon, Jorgensen, Roach, & Ford, 2009) and states (e.g., alcohol consumption; Bartholow, Henry, Lust, Saults, & Wood, 2012) associated with deficient error-checking and failures in performance monitoring. It is possible that previously observed associations between the ERN and anxiety disorders may reflect, at least in part, this link between the ERN and checking behaviors.

In contrast, symptoms of psychomotor retardation, which tend to be related to hypoactivation of the dopaminergic system (Martinot et al., 2001), were associated with a decreased ERN, consistent with previous studies in depression and psychosis (Bates et al., 2002; Foti et al., 2012; Schrijvers et al., 2008). There is accumulating evidence that the magnitude of the ERN is sensitive to activity of the neural system responsible for the production and regulation of DA (de Bruijn, et al., 2004; Holroyd & Coles, 2002; Zirnheld et al., 2004). Moreover, this system appears to be critically involved in both reward processing and clinical depression (Foti et al., 2014; Forbes et al., 2009; Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; Pizzagalli et al., 2009; Smoski et al., 2009). In fact, DA-linked deficits in reward processing appear to play an important role in both current depression (Pizzagalli et al., 2008) and risk for depression (e.g., Bress et al., 2013; Kujawa, Proudfit, & Klein, 2014; Pizzagalli et al., 2008). Depression-related reductions in reward circuits and DA deficiencies might therefore attenuate error processing (Holroyd & Coles, 2002; Holroyd, Yeung, Coles, & Cohen, 2005). However, it should be noted that, contrary to our expectations, the IMAS Anhedonia scale did not show the same pattern of results. This may be because the IMAS Anhedonia scale captures both lack of pleasure *and* interest, yet DA functioning may relate more specifically to decreased pleasure and motivation than to loss of interest (e.g., Treadway & Zald, 2011). Thus, further research is necessary to clarify how mechanisms involved with performance monitoring and reward processing interact—for instance via the examination of interactions between the ERN and more direct ERP markers of reward (e.g., the feedback negativity, or FN; e.g., Foti, Weinberg, Dien, & Hajcak, 2011) in relation to dimensions and diagnosis. Additionally, future studies using tasks that can more effectively examine competing cognitive mechanisms associated with reward processing (e.g., Pizzagalli et al., 2008) will be helpful in clarifying these effects.

Examination of these effects across diverse tasks is particularly important in light of the mixed findings regarding en-

hancement or attenuation of the ERN in clinical depression (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008, 2010; Olvet, Klein, & Hajcak, 2010; Schrijvers et al., 2008; Schrijvers et al., 2009). Though a well-validated behavioral task was used in the current study (Foti, Kotov, & Hajcak, 2013; Riesel, Weinberg, et al., 2013), other tasks which manipulate the value and salience of errors, as well as intrinsic versus extrinsic monitoring of errors, may be necessary to fully understand this phenomenon. For example, studies that have failed to find an enhancement of the ERN in depression have often used a simple flankers task devoid of trial-to-trial feedback (e.g., Weinberg, Klein, & Hajcak, 2012; Olvet, Klein, & Hajcak, 2010). In contrast, in some studies identifying an enhanced ERN in depression, participants' responses were followed by immediate performance feedback (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008). There is evidence that the presentation of evaluative feedback after responses may alter or eliminate the relationship between the ERN and anxiety (Gründler, Cavanagh, Figueroa, Frank, & Allen, 2009; Olvet & Hajcak, 2009c). Likewise, manipulations of incentive salience (e.g., associating errors with monetary gains or losses) appear to impact the magnitude of the ERN (e.g., Hajcak et al., 2005) as well as the relationship of the ERN with depression (e.g., Chiu & Deldin, 2007; Holmes & Pizzagalli, 2010). Combined, these data suggest that future studies should explore the differential relationships between performance monitoring and depressive symptoms in a variety of tasks designed to elicit errors.

Evidence for links between self-reported checking behaviors and psychomotor retardation and the neural response to errors is also interesting given the observed associations between self-reported phenotypes and behavior. Many models of performance monitoring suggest that variation in the magnitude of the ERN should relate to variation in behavior (Holroyd & Coles, 2002; Holroyd et al., 2005; Holroyd & Yeung, 2012). In particular, exaggerated processing of errors should result in increased behavioral regulation. However, a substantial body of clinical literature suggests at least a partial dissociation between ERN magnitude and behavioral measures (for a review, see: Weinberg, Riesel, & Hajcak, 2012). This is also true of the present study: despite evidence for a link between checking and an enhanced ERN, checking was not associated with improved performance on the task. On the other hand, multiple symptom dimensions linked to depression were associated with poorer performance on the task, including psychomotor retardation, which was also associated with a decreased ERN. These data are consistent with previous evidence that studies demonstrating blunted ERN amplitude in psychopathology also reveal poorer behavioral performance in these groups (for a review, see: Weinberg, Riesel, & Hajcak, 2012). However, the source of this covariation is not clear; further research will be necessary to understand whether this association is direct and causal, or whether it is driven by other factors, like working memory, cognitive control, or motivational factors. Additionally, the task we used in this study was designed to balance accuracy and speed through feedback to our participants. Use of other tasks in which behavior was not constrained might be useful in clarifying the association between behavior, the ERN, and performance monitoring.

Though these data provide evidence of a neural marker that is enhanced with increased checking and diminished in association with psychomotor retardation, it is not clear whether these findings indicate the ERN is a stable, trait-like marker, or if it is sensitive to state-linked variation. For instance, for individuals with GAD, past diagnoses of depression did not appear to influence the ERN, but current depression did—a finding that is consistent with past research (Weinberg, Klein, et al., 2012). These data collectively suggest that *state*-related characteristics of depression may suppress the relationship between error-processing and *trait* anxiety. Future studies might move beyond the cross-sectional design of the current study and utilize the ERN to track symptomatic individuals over time, as anxiety and depression severity fluctuate, to clarify the association between anxious and depressive phenotypes and the ERN.

Combined, these data indicate that the ERN may be useful for ongoing efforts to link psychopathology symptoms to integrative neuroscience (i.e., RDoC; Cuthbert & Insel, 2013; Insel et al., 2010; Sanislow et al., 2010). Though we rely upon diagnostic categories as one level of analysis, we further demonstrate that the ERN has transdiagnostic properties (i.e., in linking the ERN to checking and psychomotor retardation, agnostic to diagnostic category). Moreover, the results of the present study suggest that the ERN is linked to multiple phenotypes.

The link to multiple phenotypes is particularly important to note in light of RDoC, as the ERN currently appears as a potential unit of measurement in three RDoC constructs: Positive Valence Systems (Reward Learning), Negative Valence Systems (Sustained Threat), and Cognitive Systems (Cognitive Control: Performance Monitoring). We have previously argued that variation in the ERN reflects individual differences in response to internal threats (i.e., mistakes; Hajcak & Foti, 2008; Proudfit, Inzlicht, & Mennin, 2013; Riesel, Weinberg, Moran, & Hajcak, 2013; Weinberg, Riesel, & Hajcak, 2012), consistent with the inclusion of the ERN as a potential unit of measurement in the sustained threat construct. The current results suggest that at the behavioral level, this may be manifested by increased checking. The results of this study demonstrate an association between a pathological behavioral response (i.e., checking) and a conceptually proximal neural process (i.e., the ERN). Moreover, this behavioral response cuts across multiple diagnoses, suggesting it might one day be assessed and treated outside the context of the diagnostic categories with which it is more or less associated.

At the same time, these results highlight how multiple phenotypes relate to the ERN, and how future work may be needed to clarify the relation between reward and error processing and the ERN. It is possible that the ERN reflects the result of a rather complex computation—integrating information about threat, reward, and punishment to increase cognitive control (e.g., Shackman et al., 2011)—and it may therefore be appropriate for inclusion in multiple RDoC domains. Moreover, the present results suggest the value of the ERN in tracking the ways in which dysfunction of multiple core neural systems might interact to influence psychological functioning. Indeed, in relating specific behaviors (i.e., checking) and dysfunctions (i.e., psychomotor retardation) to well-studied cognitive neural processes using a reliable paradigm, evidence from this study has the potential to fill in cells within the RDoC matrix. Future

RDoC-inspired studies looking across multiple neural responses, multiple paradigms, and a broader range of dysfunction, might more ably consider the ways in which reward, threat, and cognitive control combine to influence abnormal behaviors.

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