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Rumination is Associated With Diminished Performance Monitoring

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Rumination is a construct that cuts across a variety of disorders, including anxiety and depression. It has been associated with deficits in cognitive control thought to confer risk for psychopathology. One aspect of cognitive control that is especially relevant to the content of ruminative thoughts is error processing. We examined the relation of rumination and 2 electrophysiological indices of error processing, error-related negativity (ERN), an early index of error detection, and error positivity (Pe), a later index of error awareness. Consistent with prior work, ERN was negatively correlated with anxiety (i.e., more anxious individuals were characterized by larger ERNs). After controlling for anxiety, rumination—but not worry—predicted ERN attenuation. No significant relation between rumination and Pe emerged. Findings suggest that rumination may diminish resources early in the processes of performance monitoring and the recruitment of cognitive control.

Keywords: rumination, performance monitoring, error-related negativity (ERN), error processing, cognitive control

Rumination is a style of repetitive thinking about negative emotional content (Nolen-Hoeksema, 1991, 2000; Nolen-Hoeksema & Morrow, 1991). Those who ruminate do so ostensibly to understand the causes and consequences of their feelings with the hope that rumination will solve their problems (Nolen-Hoeksema, 1991; Watkins & Baracaia, 2001). In reality, ruminating most often has negative consequences. Experimental work has shown that rumination augments negative cognition (e.g., Ciesla & Roberts, 2007). Rather than unlocking solutions to problems or bringing relief from emotional turmoil, rumination prolongs negative mood states and has been shown to predict depressive symptoms prospectively (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Nolen-Hoeksema, Parker, & Larson, 1994).

Rumination was originally conceptualized as a vulnerability factor for depressed mood and hypothesized to affect the onset and course of depression (Nolen-Hoeksema, 1991). It has also been associated with a number of other *DSM* disorders, such as generalized anxiety disorder and bulimia nervosa (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007; Ruscio et al., 2015). Of interest,

Nolen-Hoeksema (2000) found that rumination predicts the chronicity of anxiety symptoms and suggested that it may be particularly characteristic of mixed anxiety and depressive symptoms. For these reasons, rumination has been characterized as a transdiagnostic construct (Nolen-Hoeksema & Watkins, 2011).

Researchers have suggested that rumination can be explained by two factors: brooding and reflective pondering (Nolen-Hoeksema & Morrow, 1991). Brooding features abstract negative thoughts ("What am I doing to deserve this?") and focuses on obstacles to problems ("Why can't I handle problems better?"). Reflective pondering features self-reflection ("I go someplace alone to think about my feelings") and focuses on problem solving ("I analyze recent events to try to understand why I am depressed;" Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Brooding has been positively correlated with both concurrent and longitudinal depressive symptoms, while reflective pondering has been positively correlated with concurrent depressive symptoms but negatively correlated with longitudinal depressive symptoms (Treynor et al., 2003). Brooding may be more maladaptive overall, while reflective pondering may confer some long-term benefits (Joormann, Dkane, & Gotlib, 2006; Nolen-Hoeksema et al., 2008).

Recently, researchers have directed empirical effort toward understanding the cognitive and the neural mechanisms of rumination. Some have hypothesized that rumination reflects a failure to exert cognitive control over negative emotional material. In this view, rumination involves the repeated retrieval of negative content kept active in working memory. Enticed by a false hope of relieving distress, those who ruminate have difficulty shifting their thinking away from negative content and get caught in a loop that sustains distress with no relief (see Joormann & Tanovic, 2015). Recent work has indeed found that deficits in inhibition, shifting, and updating are associated with trait rumination in both clinical

and nonclinical samples (De Lissnyder, Derakshan, De Raedt, & Koster, 2011; Joormann & Gotlib, 2010; Pe, Raes, & Kuppens, 2013).

Event-related potentials (ERPs) associated with error processing are relevant to the cognitive and neural processes involved with rumination because ruminative thoughts focus on past failures and mistakes (Nolen-Hoeksema & Morrow, 1991). It therefore seems likely that ruminators would exhibit aberrant electrophysiological responses to errors. In the present study, errors were elicited by using a flanker task, a paradigm that requires participants to overcome response conflict and inhibit prepotent responses. This approach allowed building upon previous findings of impaired inhibition in rumination (De Lissnyder et al., 2011; Joormann & Gotlib, 2010). Aberrant error processing as indexed by ERP components has been documented in psychopathology commonly associated with rumination, such as anxiety and depression (e.g., Endrass, Klawohn, Schuster, & Kathmann, 2008; Holmes & Pizzagalli, 2008). Additionally, the temporal resolution of certain ERP waveforms could shed further light on how rumination relates to neural indices of distinct stages of cognition, ranging from more automatic (i.e., error detection) to more reflective (i.e., error awareness) processes.

Error-related negativity (ERN) is an early index of error detection that manifests as a negative deflection in the ERP waveform occurring approximately 50 ms after a mistake is made. It is maximal at frontocentral electrode sites (Simons, 2010), and neuroimaging work has shown that it emanates from the midregion of the anterior cingulate cortex (ACC), an area in the medial frontal lobe associated with monitoring performance, evaluating response conflict, and signaling the need for increased cognitive control (Shackman et al., 2011). In contrast, a small negative deflection, termed correct response negativity (CRN), is evident following trials where a correct response is made. The CRN component occurs in the same temporal interval and scalp topography as ERN but is smaller in amplitude (Simons, 2010).

Differences in ERN have been associated with psychopathology that is characterized by rumination. An abnormally enhanced ERN has been observed in clinically anxious populations. ERN amplitude has been correlated with scores on anxiety measures in both clinical and analog groups (for a review, see Moser, Moran, Schroder, Donnellan, & Yeung, 2013). For example, enhanced ERN has been observed following errors on a flanker task in patients with GAD compared with healthy controls (Weinberg, Olvet, & Hajcak, 2010) and in patients with obsessive–compulsive disorder (OCD) compared with healthy controls (Endrass et al., 2010). Similar results have been obtained with analog groups, where ERN enhancement has been correlated with measures of obsessive–compulsive symptoms (Grundler, Cavanagh, Figueroa, Frank, & Allen, 2009; Santesso, Segalowitz, & Schmidt, 2006), state anxiety (Compton et al., 2007; Vocat, Pourtois, & Vuilleumier, 2008), trait anxiety (Aarts & Pourtois, 2010; Meyer, Weinberg, Klein, & Hajcak, 2012; Olvet & Hajcak, 2009), worry (Hajcak, McDonald, & Simons, 2003; Moser, Moran, & Jendrusina, 2012), and behavioral inhibition (McDermott et al., 2009).

In contrast to anxiety, findings in depression are mixed. Some studies on depression report ERN enhancement (Aarts, Vanderhasselt, Otte, Baeken, & Pourtois, 2013; Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; Tang et al., 2013), some report ERN attenuation (Ladouceur et al., 2012; Ruchsow et al., 2006; Ruchsow et al., 2004; Weinberg, Liu, & Shankman, 2016), and others have reported no difference compared to control participants (Olvet, Klein, & Hajcak, 2010; Schrijvers et al., 2010, 2009). Recent work by Weinberg and colleagues (Weinberg, Klein, & Hajcak, 2012; Weinberg, Kotov, & Proudfit, 2015) has shown that ERN is enhanced in patients with generalized anxiety disorder (GAD) but not in those with comorbid GAD and major depressive disorder (MDD) or those with MDD alone. Their results showed further that, across all groups, ERN enhancement was associated with symptoms of checking behaviors and that ERN attenuation was associated with psychomotor retardation (Weinberg et al., 2015). Different but frequently related phenotypes associated with *DSM* disorders relate to ERN, highlighting the value of examining the relation of ERN with a transdiagnostic construct such as rumination. Further, correlated phenotypes may have opposite or opposing (i.e., suppressor-like) effects (see Weinberg, Dieterich, & Riesel, 2015, for further discussion).

Researchers have attempted to clarify what specific processes of anxiety and depression are most closely related to ERN. Moser, Moran, Schroder, Donnellan, and Yeung (2013) have suggested that the enhanced ERN amplitude seen in relation to anxiety reflects worry; they argue that ERN enhancement represents an effort to compensate for the distracting effects of worry on task performance. In light of similarities between worry and rumination as forms of repetitive negative thinking (e.g., McEvoy, Watson, Watkins, & Nathan, 2013; Watkins & Baracaia, 2001), it is possible that rumination could also be associated with ERN enhancement.

Error positivity (Pe) is an index of error processing that occurs after ERN. The Pe component is characterized as a positive deflection and occurs approximately 200 ms–500 ms after an incorrect response is made. It is maximal at centroparietal electrode sites (Simons, 2010) and has been associated with the rostral ACC (van Veen & Carter, 2002). Because it transpires later in the error monitoring process when conscious reflection on performance is possible, Pe is thought to capture awareness that a mistake has been made (Murphy, Robertson, Allen, Hester, & O'Connell, 2012; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005).

Like ERN, Pe has been examined in relation to psychopathology characterized by rumination. However, reports of abnormalities in Pe in relation to both anxiety and depression are mixed. Some researchers have found no relationship between Pe and OCD (Endrass et al., 2010; Ruchsow et al., 2005; Xiao et al., 2011), GAD (Xiao et al., 2011), MDD (Holmes & Pizzagalli, 2008), anxiety about math (Suarez-Pellicioni, Nunez-Pena, & Colome, 2013), induced mood (Larson, Gray, Clayson, Jones, & Kirwan, 2013), helplessness (Pfabigan et al., 2013), or dysphoria in young adults (Compton et al., 2008) and healthy adults (Chang, Davies, & Gavin, 2010). In contrast, others have found attenuation of Pe in MDD (Aarts, Vanderhasselt, Otte, Baeken, & Pourtois, 2013; Olvet, Klein, & Hajcak, 2010; Schrijvers et al., 2008; Schrijvers et al., 2009), that it is associated with higher levels of negative affect (Hajcak, McDonald, & Simons, 2004), and correlated with depressive symptoms in undergraduates (Schroder, Moran, Infantolino, & Moser, 2013). Enhanced Pe has also been reported in relation to anxiety in healthy adults (Chang, Davies, & Gavin, 2010). While it appears that Pe may be broadly related to internalizing psychopathology, the exact nature of this relation remains to be clarified.

To investigate the relation between error processing and rumination, we examined both ERN and Pe. We aimed to clarify when in the error monitoring process rumination may become important. Specifically, we examined whether rumination is related to the early stages of error monitoring (indexed by ERN), before there is reflective awareness of committing an error, or the later stages of error monitoring (indexed by Pe), when reflective awareness is possible. We included the construct worry because of past findings linking it to anxiety and ERN, and because of conceptual similarities to rumination. In addition to ERN and Pe, we were interested in CRN following correct trials to determine whether neural patterns were specific to error commission.

Method

Participants

Fifty-two university students (38 females) were recruited via electronic and print advertisements. Seven participants were excluded from analyses due to difficulties performing the tasks ($n = 1$), problems during electroencephalogram (EEG) recording that compromised the quality of the data ($n = 4$), experimenter error ($n = 1$), and making fewer than six errors ($n = 1$). The final sample consisted of 45 participants (34 females) with a mean age of 20.20 ($SD = 1.25$). Of these participants, 51.1% identified as White, 35.6% as Asian, 2.2% as American Indian, 2.2% as Black, and 6.7% as more than one race; 2.2% of participants did not respond. Hispanic participants comprised 8.9% of the final sample.

The university institutional review board approved the study, and informed consent was obtained from each participant prior to beginning the procedure. Participants were compensated $20.00 for their time. No participants chose to discontinue participation.

Flanker Task

A version of the flanker task (Eriksen & Eriksen, 1974) as modified by Weinberg, Olvet, and Hajcak (2010) was used to elicit ERN, CRN and Pe. The task was administered using Presentation software (Neurobehavioral Systems, Inc., Albany, CA) on a Pentium 4 class computer with a 19-in. monitor. Each trial consisted of the presentation of five horizontal arrowheads; 50% of the trials were compatible, with all of the arrowheads facing in the same direction:

> > > > > or < < < < <

and 50% were incompatible, with the middle arrowhead facing the opposite direction of the flanking arrows:

> > < > > or < < > < <

Participants were instructed to respond to the direction that the middle arrow was pointing. Following a practice run consisting of 30 trials, compatible and incompatible trials were presented randomly in 11 blocks of 30 trials each, for a total of 330 trials. Between each block, participants received one of three types of feedback based on their performance: (a) "You're doing a great job!" if they made an adequate number of errors; (b) "Please try to respond faster" if they did not make enough errors; and (c) "Please try to be more accurate" if they made too many errors. The intertrial interval randomly varied from 2,300 ms to 2,800 ms, and each stimulus was presented for 200 ms.

Measures

Ruminative Responses Scale. The Ruminative Responses Scale (RRS) of the Response Style Questionnaire (Nolen-Hoeksema & Morrow, 1991) was used to assess rumination. The RRS consists of 22 items that assess ruminative responses to depressed mood states; items examine rumination that is focused on the self (e.g., "I think back to other times I have been depressed"), on symptoms (e.g., "I think about how hard it is to concentrate"), or on the possible causes and consequences of the individual's mood (e.g., "I go away by myself and think about why I feel this way"; Nolen-Hoeksema & Morrow, 1991). The RRS has been used to examine rumination in clinical (e.g., Carter et al., 2009; Nolen-Hoeksema, 2000) and analog (e.g., Roberts, Gilboa, & Gotlib, 1998; Roelofs et al., 2006) samples ($\alpha = .90$; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). The total RRS score is computed by summing the individual's ratings (on a scale of 1 to 4) of the 22 items. In addition to the overall RRS score, analyses were done with the brooding and reflective pondering subscales; Items 5, 10, 13, 15, and 16 were summed for the brooding subscale, and Items 7, 11, 12, 20, and 21 for the reflective pondering subscale (Treynor et al., 2003).

Penn State Worry Questionnaire. The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) was used to assess worry. The PSWQ consists of 16 items that examine the degree to which an individual's worry is excessive, generalized, and uncontrollable (Fresco, Mennin, Heimberg, & Turk, 2003). It has been used with both clinical (e.g., Esbjorn et al., 2013; Starcevic, 1995) and analog (e.g., Compton et al., 2008; Hajcak, McDonald, & Simons, 2004) samples ($\alpha = .93$; Brown et al., 1992). The total score is the sum of ratings (on a scale of 1 to 5) of Items 2–7, 9, and 12–16, in addition to the sum of Items 1, 3, 8, 10, and 11 after reverse scoring (these items reflected the absence of pathological worry—e.g., Item 3: "I do not tend to worry about things").

Personality Assessment Inventory. The Personality Assessment Inventory (PAI; Morey, 1991) is a measure of personality and psychopathology. The PAI consists of multiple clinical scales that may be administered individually; in the present study, the anxiety (PAI-ANX) and depression (PAI-DEP) scales were used to assess anxiety and depression symptoms, respectively.

The scale ranges from 1 to 4, and scores of 1 are recoded into 0, 2 into 1, 3 into 2, and 4 into 3. On the PAI-ANX, seven items are reverse scored because they reflect the absence of anxiety (Items 10, 13, 14, 15, 17, 19, and 24; 0 is rescored as 3, 1 as 2, 2 as 1, and 3 as 0). On the PAI-DEP, eight items are reverse scored in the same way (Items 6, 9, 17, 18, 19, 20, 22, and 23). Total and subscale raw scores are converted into t scores, which reference a participant's raw score to the average scores of a sample of 1,000 adults living in the community in the United States (Morey, 2003, p. 28). Internal consistencies of the anxiety and depression subscales are high ($\alpha = .90$ and $\alpha = .87$, respectively; Morey, 1991).

Electrophysiological recording and data processing. During the flanker task, EEG activity was recorded a 64-channel cap with electrode sites arranged based on the 10–20 System. Electrodes were placed on the left and right mastoids; during offline processing, all data were referenced to the average of these channels. Eye movements and blinks were recorded from four electrodes placed around the right and left eyes. One electrode was

placed one centimeter outside of each eye to record horizontal eye movements. Electrodes were also placed above and below the left eye to record vertical eye movements. Recordings were collected using the BioSemi Active Two system (BioSemi, Amsterdam, Netherlands).

EEG data were obtained with a sampling rate of 1,024 Hz and filtered with a low-pass 100 Hz filter and a high-pass 0.16 Hz filter. At each electrode, the signal was amplified by a gain of one. Data were processed offline using BrainVision Analyzer (Brain Products GmbH, Munich, Germany). After being rereferenced to the average of the left and right mastoid electrodes, data were filtered with Butterworth zero phase filters with a low cutoff of 0.1 Hz, a high cutoff of 30 Hz, and a maximal slope of 24 dB/oct. Data were then segmented into response-locked epochs that include 500 ms before the behavioral response and 1,000 ms after. Ocular corrections were performed using the Gratton and Coles algorithm (Gratton, Coles, & Donchin, 1983). Artifacts were detected and rejected through semiautomatic inspection. Segments falling outside of the following parameters were automatically marked for rejection: a maximal voltage step of 50 μV/ms, a maximal difference of 300 μV between the highest and lowest points in an interval of 200 ms, and activity below 0.5 μV for 100 ms. A trained research assistant also visually inspected the data to remove any additional artifacts. Trials were then segmented based on whether they were correct or incorrect trials. Each channel for each trial type was averaged across all of the trials, to yield one average for each channel for each trial type. The 200-ms interval between 500 ms and 300 ms before the onset of the response served as a baseline.

Specific ERP components were calculated from the averaged data. The ERN was quantified as the average area of activity on incorrect trials from 0 ms to 100 ms (where 0 ms was the onset of the motor response) at frontocentral sites (i.e., FCz and Cz) where ERN was maximal. The Pe was calculated as the average area of activity on incorrect trials from 200 ms to 400 ms at Pz, where the Pe was maximal. For both ERN and Pe, a difference score was also computed to examine the difference in average voltage for incorrect trials versus correct trials.

Analysis Overview

Descriptive statistics for the clinical measures (RRS, PSWQ, PAI-ANX, and PAI-DEP) and behavioral data from the flanker task were calculated. Correlations among the clinical measures were obtained, as were correlations among clinical measures and behavioral data. Means of the ERP components and the correlations between them were calculated. Then, correlations between the clinical measures and the ERP components were examined. Finally, multiple regression analyses were performed using the clinical measures as predictors of ERP components as dependent variables.

Results

Self-Report Data

Means, ranges, and standard deviations for the clinical measures are reported in Table 1, as are the range of possible scores for each measure. Scores on all measures were normally distributed. On the

Table 1

Descriptive Statistics and Possible Score Ranges of Clinical Measures (n = 45)

	Mean (SD)	Range	Possible range
RRS	43.59 (10.75)	24–63	22–88
PSWQ	49.51 (14.48)	23–76	16–80
PAI-ANX	57.16 (13.50)	35–89	20–120
PAI-DEP	52.44 (10.85)	38–92	20–120

Note. RRS = Ruminative Responses Scale; PSWQ = Penn State Worry Questionnaire; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; PAI-DEP = Personality Assessment Inventory—Depression Scale.

PAI-ANX and PAI-DEP, scores above 70T are considered as meriting clinical attention (Morey, 1991, p. 28). In the present sample, 13.3% of participants had scores in the clinical range for anxiety, and 4.4% had scores in the clinical range for depression.

RRS, PSWQ, PAI-ANX, and PAI-DEP data were analyzed by computing a total score for each measure for each participant. Scores for the subscales of the RRS, brooding and reflective pondering, were also computed and analyses were done with the brooding and reflective pondering subscales individually, in addition to the overall RRS score. Three participants did not answer one or two questions on the RRS, PAI-ANX, or the PAI-DEP. For those participants, mean substitutions were used in order to obtain total scores. Gender differences were not examined because of the proportion of males in the sample (24.4%).

Correlations among the clinical measures are reported in Table 2. Brooding was significantly correlated with worry, $r(43) = .57$, $p < .001$; anxiety, $r(43) = .54$, $p < .001$; depression, $r(43) = .46$, $p = .002$; and reflective pondering, $r(43) = .31$, $p = .041$. Reflective pondering was not significantly correlated with any of these variables except for brooding.

Behavioral Data

Performance accuracy on the flanker task was high ($M = 89.86\%$, $SD = 4.84$). When response time (reaction time [RT]) was examined, there was a main effect of congruency, $F(1, 37) = 95.10$, $p < .001$, $\eta_p^2 = .72$, where RTs on congruent trials were faster ($M = 333.12$, CI [323.74, 342.74], $SD = 39.06$) than on incongruent trials ($M = 375.98$, CI [365.98, 385.25], $SD = 34.25$).[1] There was also a significant main effect of accuracy, $F(1, 37) = 345.2$, $p < .001$, $\eta_p^2 = .90$, such that correct trials were slower ($M = 399.69$, CI [388.37, 411.63], $SD = 38.67$) than error trials ($M = 309.41$, CI [301.17, 317.86], $SD = 34.64$). As well, there was an interaction between congruency and accuracy, $F(1, 37) = 38.5$, $p < .001$, $\eta_p^2 = .51$, such that RTs were slower on incongruent than congruent trials but less so on error trials, $F(1, 37) = 4.72$, $p = .036$, $\eta_p^2 = .11$, than correct trials, $F(1, 44) = 407.03$, $p < .001$, $\eta_p^2 = .90$ (see Figure 1). This confirmed that, as expected in speeded response time tasks, errors tended to be impulsive (Simons, 2010).

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[1] Seven participants did not make any errors on congruent trials, so only 38 cases were used for analyses of congruent trial response time for both incorrect and correct trials.

Table 2
Correlations Among Clinical Measures (n = 45)

	RRS	RRS-B	RRS-R	PSWQ	PAI-ANX	PAI-DEP
RRS	—					
RRS-B	.73**	—				
RRS-R	.73**	.31*	—			
PSWQ	.52**	.57**	.18	—		
PAI-ANX	.52**	.54**	.17	.79**	—	
PAI-DEP	.52**	.46**	.11	.60**	.74**	—

Note. RRS = Ruminative Responses Scale; RRS-B = Ruminative Responses Scale-brooding subscale; RRS-R = Ruminative Responses Scale-reflective pondering subscale; PSWQ = Penn State Worry Questionnaire; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; PAI-DEP = Personality Assessment Inventory—Depression Scale.
* $p < .05$. ** $p < .01$.

Electrophysiological Data

As predicted, incorrect responses in the flanker task were followed by a larger negative deflection within 100 ms than correct responses. ERN ($M = 2.59$, $SD = 5.79$ μV) was more negative than CRN ($M = 10.91$, $SD = 5.95$ μV; $t(44) = -10.35$, $p < .001$; see Figure 2).

In the interval of 200 ms to 400 ms after a response, error trials were characterized by a larger positive deflection, the Pe ($M = 16.66$, $SD = 6.71$ μV), than correct trials ($M = 3.41$, $SD = 3.67$ μV; $t(44) = 15.59$, $p < .001$).

Correlations among the ERP components are presented in Table 3. Following correct trials, Pe was significantly correlated with ERN, $r = .33$, $p = .026$ and CRN, $r = .54$, $p < .001$. ERN and CRN were significantly correlated, $r = .58$, $p < .001$. No other correlations between the ERP variables were significant.

Correlations of Behavioral Data With ERP Components and Clinical Measures

Accuracy was positively correlated with Pe on error trials, $r = .32$, $p = .035$. There were no significant correlations between ERP components and RT. None of the correlations of clinical measures with RT (correct or incorrect responses) for either trial type (congruent, incongruent) were significant. There was a trend for the correlation between anxiety RT on correct incongruent trials, $r = .28$, $p = .065$.

Correlations of Clinical Measures and ERP Components

Correlations among the clinical measures and ERP components are presented in Table 4. Neither brooding, $r(43) = .14$, $p = .345$, nor reflective pondering, $r(43) = .07$, $p = .654$, were significantly correlated with ERN. However, ERN was significantly negatively correlated with anxiety, $r(43) = -.30$, $p = .048$; because ERN is a negative deflection, this means that greater anxiety scores were associated with enhanced ERNs. A similar pattern emerged for CRN and anxiety, $r(43) = -.30$, $p = .046$. No correlations with Pe, either after error trials or correct trials, were significant.

Regression of Clinical Measures Predicting ERP Components

To examine the ability of the clinical measures to predict the amplitude of ERN, CRN, and Pe, multiple linear regression analyses were conducted. The regressions were done hierarchically in order to examine the incremental predictive value of adding each predictor to the model. In the first analysis, shown in Table 5, ERN was examined. Because of the well-established relationship between ERN and anxiety, anxiety was included in the first step. The total score on the RRS, the primary variable of interest, was included in the second step. To examine whether the relation between the ERN and rumination varied based on levels of anxiety, the third step featured the interaction between rumination and anxiety. The final step examined if there was an additional influence of worry or depression on ERN. All predictor variables were mean-centered prior to analysis. The same multiple regressions were repeated with CRN (see Table 6), with Pe following error trials, and then with Pe following correct trials.

The results indicated that anxiety explained significant variance in ERN. After accounting for the effect of anxiety, rumination explained additional variance in ERN. The effects of anxiety and rumination were approximately equal in magnitude but in different directions: Anxiety was associated with ERN enhancement, while rumination was associated with ERN attenuation. This regression was repeated for each of the RRS subscales—brooding and reflective pondering—separately. After controlling for anxiety ($B = -0.13$, $SE = .06$, $t = -2.04$, $p = .048$, $\Delta R^2 = .09$, CI [−.25, −.001]), reflective pondering did not predict significant variance in ERN, while brooding did ($B = 0.84$, $SE = .31$, $t = 2.67$, $p = .011$, $\Delta R^2 = .13$, CI [.21, 1.47]). The effects of anxiety and brooding remained significant even when worry, depression, and the anxiety by rumination interaction term were included in the model, which is shown in Table 7.[2]

The same multiple regressions were performed for CRN. The regression using total RRS scores in shown in Table 6, and the regression using the brooding subscale is shown in Table 8. Although total RRS scores did not predict significant variance in CRN, examining the individual subscales revealed they had different relationships with CRN. Specifically, anxiety predicted CRN enhancement ($B = -0.13$, $SE = .06$, $t = -2.06$, $p = .046$, $\Delta R^2 = .09$, CI [−.26, −.003]), while brooding rumination ($B = 1.06$, $SE = .31$, $t = 3.43$, $p = .001$, $\Delta R^2 = .20$, CI [.44, .168])—but not reflective pondering—predicted CRN attenuation. Again, the effects of anxiety and brooding remained significant when worry, depression, and the anxiety by rumination interaction term were in the model, is shown in Table 8.[3]

To examine the relation of the clinical measures with Pe, the same regressions were repeated for Pe following error trials and Pe following correct trials. Results did not reveal any significant results; none of the clinical measures significantly predicted Pe following correct or error trials.

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[2] The significant effects of anxiety and brooding remained when reflective pondering was included in the model.
[3] The significant effects of anxiety and brooding also remained when reflective pondering was included in the model.
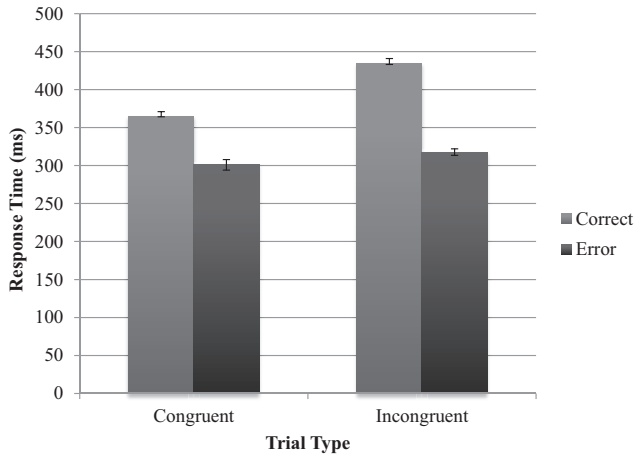
Figure 1. Mean response time by trial type.

Discussion

After controlling for anxiety, our results demonstrate that brooding rumination relates to diminished ERN magnitude. The significant relationship between anxiety and ERN replicates prior work demonstrating enhancement of ERN (i.e., more negative deflection) among clinically and subclinically anxious individuals. Additionally, it appears that this relationship is not specific to the context of errors: performance monitoring during correct responses as indexed by CRN is also enhanced in anxiety, consistent with previous work with similar populations (e.g., Hajcak, McDonald, & Simons, 2003; Moran, Taylor, & Moser, 2012; Moser, Moran, & Jendrusina, 2012). More anxious participants appeared to exhibit neural correlates of increased performance monitoring compared to those who were less anxious. The additional variance in ERN and CRN predicted by brooding rumination suggests that a tendency to brood is associated with decreases in performance monitoring.

As an index of error monitoring generated by the ACC, ERN can be thought of as a signal that an error has occurred, and as a call for more cognitive control to enhance performance on a given task (Shackman et al., 2011). Rumination may increase cognitive load thereby decreasing cognitive control resources available for the task. This explanation is consistent with the resource allocation hypothesis of depression, which holds that depression takes from the finite amount of resources that an individual can exert on other cognitive operations and that rumination may be a key mechanism for depletion of those resources (Hertel, 1998; Joormann & Ar-
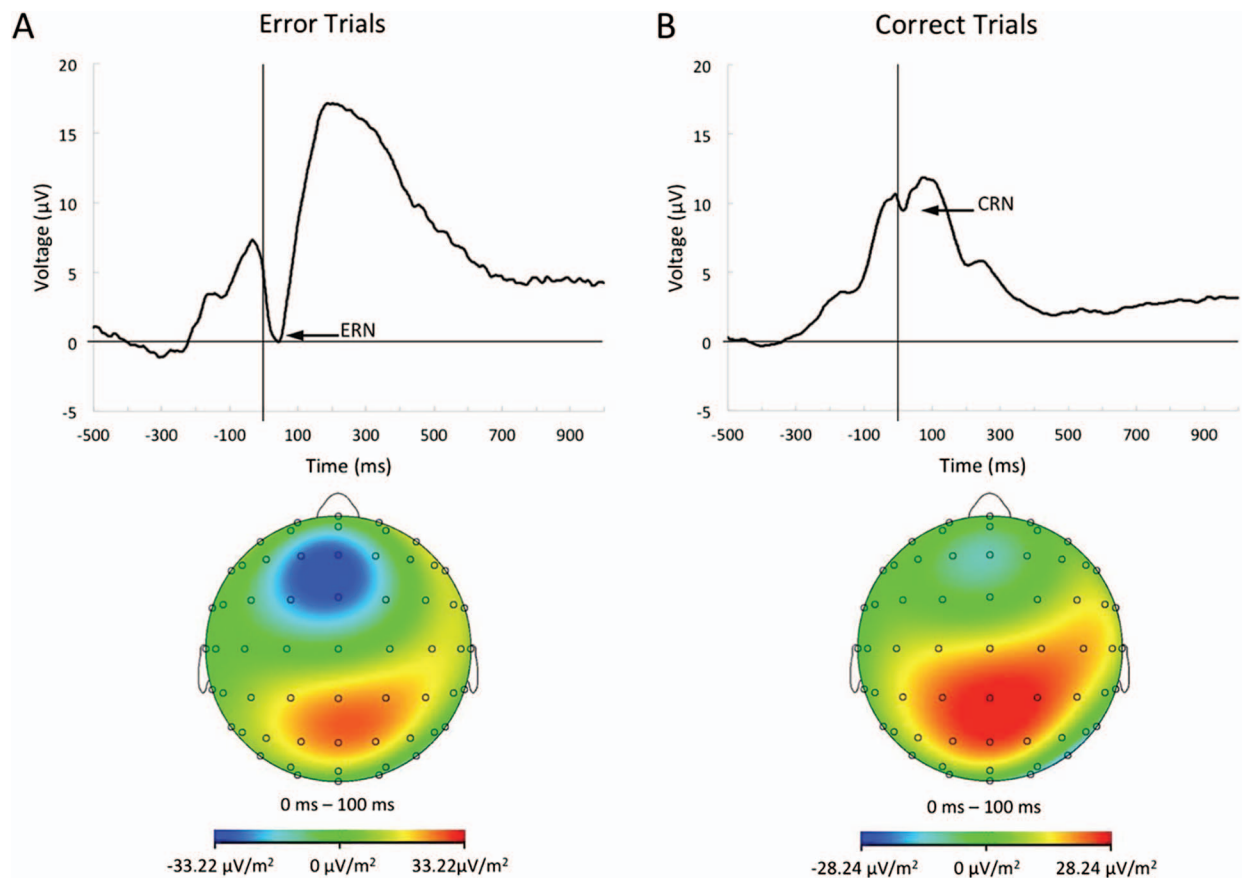

Figure 2. Grand average of response-locked *EEG* activity at FCz and Cz on error trials (top panels) and scalp topography of the grand average of response-locked activity between 0 ms and 100 ms (bottom panels). Trials where participants committed an error (A) are on the left, and trials where participants correctly responded (B) are on the right.

Table 3
Correlations Among ERP Components (n = 45)

	ERN	CRN	Pe (E)	Pe (C)
ERN	—			
CRN	.58**	—		
Pe (E)	.28	.20	—	
Pe (C)	.33*	.54**	.53**	—

Note. ERP = Event-related potentials; ERN = error-related negativity; CRN = correct response negativity; Pe (E) = error positivity following error trials; Pe (C) = positivity following correct trials.
* $p < .05$. ** $p < .01$.

ditte, 2014; Levens, Muhtadie, & Gotlib, 2009). The enhancement of ERN associated with anxiety may reflect the emotionally aversive nature of making mistakes, while the attenuation associated with rumination may reflect the decrease in available cognitive control resources for task-related goals.

The relation of rumination and error detection was specific to the brooding subscale of the RRS and not the reflection subscale. This is consistent with evidence reviewed earlier suggesting that brooding may be more pathognomonic than reflective pondering (Joormann et al., 2006; Nolen-Hoeksema et al., 2008). In addition to an emotional toll, brooding may also be cognitively taxing. Our findings provide electrophysiological evidence for this idea, by showing that brooding rumination is associated with changes in error processing while reflective pondering is not, and help to explain why brooding is a particularly harmful type of rumination (e.g., Joormann et al., 2006; Nolen-Hoeksema et al., 2008; Treynor et al., 2003).

When ERN is enhanced in relation to anxiety, our findings suggest that rumination attenuates this enhancement, thus "normalizing" ERN. If an enhanced ERN reflects neural activity associated with enhanced threat sensitivity in anxiety as some researchers have proposed and our results support (e.g., Proudfit, Inzlicht, & Mennin, 2013), then reduction of ERN may signal anxious individuals that a given event is not as threatening as it would otherwise be responded to by the performance monitoring system. Clinically, this helps explain the pernicious cycle of rumination.

Table 4
Correlations Among Clinical Measures and ERP Components (n = 45)

	ERN	CRN	Pe (error)	Pe (correct)
RRS	.10	.04	−.04	.01
RRS-B	.14	.21	−.13	.05
RRS-R	.07	.06	−.02	−.06
PSWQ	−.16	−.13	−.18	−.02
PAI-ANX	−.30*	−.30*	−.11	−.11
PAI-DEP	−.16	−.22	.06	−.01

Note. ERP = Event-related potentials; ERN = error-related negativity; CRN = correct response negativity; RRS = Ruminative Responses Scale; RRS-B = Ruminative Responses Scale-brooding subscale; RRS-R = Ruminative Responses Scale-reflective pondering subscale; PSWQ = Penn State Worry Questionnaire; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; PAI-DEP = Personality Assessment Inventory—Depression Scale; Stressful life events = number of events endorsed on Life Experiences Survey.
* $p < .05$.

Table 5
Multiple Linear Regression Model of Clinical Measures, Including Rumination, as Predictors of ERN Amplitude

	B	SE	β	95% CI	p	ΔR²	R²
Step 1					.048	.09	.09
PAI-ANX	−.13	.06	−.30	[−.25, −.001]	.048		
Step 2					.017	.09	.18
PAI-ANX	−.21	.07	−.48	[−.35, −.06]	.006		
RRS	.19	.09	.35	[.01, .37]	.039		
Step 3					.037	.01	.19
PAI-ANX	−.21	.07	−.50	[−.36, −.07]	.005		
RRS	.19	.09	.35	[.01, .37]	.038		
PAI-ANX × RRS	.004	.007	.09	[−.01, .02]	.522		
Step 4					.122	.01	.19
PAI-ANX	−.27	.13	−.64	[−.53, −.02]	.035		
RRS	.83	.38	.43	[−.02, .37]	.073		
PAI-ANX × RRS	.005	.007	.12	[−.01, .02]	.451		
PSWQ	.06	.10	.15	[−.14, .26]	.561		
PAI-DEP	.03	.12	.05	[−.21, .26]	.832		

Note. ERN = error-related negativity; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; RRS = Ruminative Responses Scale; PSWQ = Penn State Worry Questionnaire; PAI-DEP = Personality Assessment Inventory—Depression Scale.

Although rumination has been shown to worsen and prolong negative affect, individuals continue to engage in it. Perhaps one reason for this is that rumination may play a pacifying role on systems abnormally enhanced by anxiety, as reflected by electrophysiological indices.

The compensatory error monitoring framework proposed by Moser et al., (2013) suggests that the enhancement of ERN seen in anxiety is specifically related to the construct of anxious apprehension, which they define as being characterized by worry and verbal rumination (Moser et al., 2013). They posit that, because worry is distracting, additional cognitive control must be exerted to meet task demands and that this compensatory process is reflected in an enhanced ERN. Our results suggest an alternative interpretation. In contrast to worry, rumination is associated not with enhanced but with attenuated ERN. Individuals who ruminate may respond to aversive events, like making a mistake, by disengaging from present task-related goals to instead engaging in the negative thoughts that such an aversive event prompts, thereby diminishing ERN amplitude. In other words, our results support the notion that rumination is distracting but suggest that a compensatory process as reflected by an enhanced ERN does not accompany this distracting effect, contrary to what the compensatory error monitoring framework postulates about both worry and rumination. Instead, we argue that ERN enhancement is associated with trait anxiety and reflects the emotionally aversive nature of errors (Proudfit, Inzlicht, & Mennin, 2013). The compensatory error monitoring framework may benefit from considering rumination and worry independently, rather than subsuming them under the term of anxious apprehension. Further research is needed to disentangle these constructs, and our results suggest caution against assuming that these constructs are isomorphic.

The association of attenuated ERN and rumination is consistent with previous findings from Weinberg and colleagues (Weinberg, Klein, & Hajcak, 2012; Weinberg, Kotov, & Proudfit, 2015; Weinberg, Meyer et al., 2016), who have shown that ERN is enhanced

Table 6
Multiple Linear Regression Model of Clinical Measures, Including Brooding, as Predictors of ERN Amplitude

	B	SE	β	95% CI	p	ΔR²	R²
Step 1					.048	.09	.09
PAI-ANX	−.13	.06	−.30	[−.25, −.001]	.048		
Step 2					.005	.13	.22
PAI-ANX	−.23	.07	−.53	[−.37, −.09]	.002		
RRS-Brooding	.84	.31	.43	[.21, 1.47]	.011		
Step 3					.014	.004	.23
PAI-ANX	−.24	.07	−.56	[−.39, −.09]	.002		
RRS-Brooding	.89	.34	.46	[.21, 1.57]	.012		
PAI-ANX × RRS-Brooding	.01	.03	.07	[−.04, .07]	.662		
Step 4					.064	.00	.23
PAI-ANX	−.27	.12	−.63	[−.51, −.03]	.030		
RRS-Brooding	.85	.36	.44	[.13, 1.58]	.022		
PAI-ANX × RRS-Brooding	.01	.03	.05	[−.05, .07]	.738		
PSWQ	.01	.10	.04	[−.18, .21]	.882		
PAI-DEP	.04	.12	.07	[−.20, .27]	.753		

Note. ERN = error-related negativity; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; RRS-Brooding = Ruminative Responses Scale-Brooding subscale; PSWQ = Penn State Worry Questionnaire; PAI-DEP = Personality Assessment Inventory—Depression Scale.

in GAD but not in comorbid GAD and MDD or MDD alone. Their results and ours suggest that correlated phenotypes can influence the amplitude of ERN in opposite directions; when there are such suppressor effects, they are only evident using multiple regression. Along similar lines, the current study suggests that anxiety may enhance ERN, while rumination may diminish it.

In our study, the effect size of the correlation between ERN and PAI-ANX was medium ($r = -.30$), which is consistent with the average effect size reported in the literature ($r = -.25$ to $-.35$; Moser et al., 2013). It should be noted that while anxiety and brooding rumination together explained 22.2% of ERN variance, they each accounted for approximately equal amounts of the variance, 9% and 13%, respectively. In light of the substantial

Table 7
Multiple Linear Regression Model of Clinical Measures, Including Rumination, as Predictors of CRN Amplitude

	B	SE	β	95% CI	P	ΔR²	R²
Step 1					.046	.09	.09
PAI-ANX	−.13	.06	−.30	[−.26, −.003]	.046		
Step 2					.038	.06	.14
PAI-ANX	−.20	.07	−.44	[−.34, −.05]	.011		
RRS	.15	.09	.27	[−.04, .34]	.109		
Step 3					.088	.002	.15
PAI-ANX	−.19	.08	−.43	[−.34, −.04]	.015		
RRS	.15	.09	.27	[−.04, .34]	.116		
PAI-ANX × RRS	−.002	.007	−.04	[−.02, .01]	.778		
Step 4					.195	.02	.17
PAI-ANX	−.24	.13	−.53	[−.50, .03]	.080		
RRS	.15	.10	.26	[−.06, .35]	.154		
PAI-ANX × RRS	−.001	.007	−.02	[−.02, .01]	.919		
PSWQ	.09	.10	.22	[−.12, .30]	.394		
PAI-DEP	−.05	.12	−.10	[−.30, .20]	.668		

Note. CRN = correct response negativity; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; RRS = Ruminative Responses Scale; PSWQ = Penn State Worry Questionnaire; PAI-DEP = Personality Assessment Inventory—Depression Scale.

empirical support for the relation between ERN and anxiety, our work underscores the importance of examining rumination in future studies seeking to understand the cognitive processes associated with ERN.

No significant findings emerged regarding the relation between the Pe and clinical measures. This suggests that the relation between rumination and error processing is specific to the early stages of error detection, as indexed by ERN. The lack of Pe attenuation or enhancement in relation to anxiety and depression measures is consistent with some previous findings (e.g., Compton et al., 2008; Endrass et al., 2010) and suggests that this later component of error awareness is less affected by variation in internalizing psychopathology.

Theories about the ERN suggest that it represents increased resources to adjust behavior after committing an error. The ERP measures in our study were not significantly related to accuracy or response time, which is consistent with many, though not all, findings from previous studies that examined error commission and ERN and Pe (e.g., Masaki et al., 2007; Weinberg, Olvet, & Hajcak, 2010; for a review, see Weinberg, Riesel, & Hajcak, 2012). In meta-analyses, ERN does relate to behavioral adjustment—especially for within subjects comparisons (Cavanagh & Shackman, 2015). Yet, subjects who have a larger ERN rarely differ in behavioral measures (Weinberg et al., 2011). The current study was consistent with the latter, insofar as variables that related to the ERN did not relate to performance. Moreover, we did not find any overall relationship between ERN and behavioral measures (cf. Cavanagh & Shackman, 2015). The lack of significant relations among the clinical and behavioral measures suggests that rumination and anxiety may relate specifically to alterations in neural responses to errors that do not directly translate to behavior and may instead reflect earlier cognitive control processes that have complex, multipliciotous effects on behavior.

Our study had certain strengths and limitations. The number of male participants precluded examination of gender differences, and it is relevant that a recent meta-analysis reported that anxiety

Table 8

Multiple Linear Regression Model of Clinical Measures, Including Brooding, as Predictors of CRN Amplitude

	B	SE	β	95% CI	p	ΔR²	R²
Step 1					.046	.09	.09
PAI-ANX	−.13	.06	−.30	[−.26, −.003]	.046		
Step 2					.001	.20	.29
PAI-ANX	−.26	.07	.53	[−.40, −.12]	.001		
RRS-Brooding	1.06	.31	−.57	[.44, 1.68]	.001		
Step 3					.003	.00	.29
PAI-ANX	−.25	.07	−.57	[−.40, −.11]	.001		
RRS-Brooding	1.02	.33	.51	[.36, 1.69]	.004		
PAI-ANX × RRS-Brooding	−.01	.03	−.05	[−.07, .05]	.747		
Step 4					.015	.00	.30
PAI-ANX	−.26	.12	−.60	[−.50, −.03]	.030		
RRS-Brooding	1.01	.35	.51	[.30, 1.72]	.007		
PAI-ANX × RRS-Brooding	−.001	.03	−.03	[−.07, .05]	.852		
PSWQ	.04	.10	.09	[−.16, .23]	.701		
PAI-DEP	−.03	.12	−.06	[−.27, .20]	.776		

Note. CRN = correct response negativity; PAI-ANX = Personality Assessment Inventory—Anxiety Scale; RRS-Brooding = Ruminative Responses Scale-Brooding subscale; PSWQ = Penn State Worry Questionnaire; PAI-DEP = Personality Assessment Inventory—Depression Scale.

was associated with ERN enhancement in women but not men (Moser, Moran, Kneip, Schroder, & Larson, 2016). Findings from the current sample comprised of university students may not generalize to other populations. Participant scores on clinical measures spanned a range, with 13.3% in the clinical range for anxiety, and 4.4% for depression. Some may argue that a clinical sample is most apt to study cognitive-neural features of rumination, anxiety, and depression. Nonetheless, the range of symptomatology in our sample is consistent with a dimensional approach to capture meaningful subclinical variance (e.g., Sanislow et al., 2010).

In the present study, both ERN and CRN were enhanced by anxiety. After controlling for anxiety, brooding rumination predicted ERN attenuation. The variance accounted for by rumination was about equal to that accounted for by anxiety, and this finding extended to the CRN component of the waveform. The uniqueness of the relationship between ERN and rumination further suggests that worry and rumination may affect distinct cognitive-neural systems. Our results also show that the relation of rumination to early performance monitoring is not specific to the incidence of errors but instead reflects a more global reduction in cognitive resources for task-related goals.

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