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The relationship between depressive symptoms and error monitoring during response switching

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Abstract Heightened sensitivity to failure and negative information is thought to be an important maintenance mechanism for symptoms of depression. However, the specific neural and behavioral correlates of the abnormal reactions to errors associated with depression are not yet well understood. The present study was designed to shed new light on this issue by examining how depressive symptoms relate to error monitoring in the context of different task demands. We used a modified flanker task in which the stimulus–response (S–R) mappings were reversed between blocks, differentiating relatively easy nonreversal blocks from the more-demanding S–R reversal blocks. Undergraduates performed this task and then completed a self-report measure of anhedonic depression. The results revealed that depressive symptoms were related to poorer posterror accuracy in the more-difficult S–R reversal blocks, but not in the easier nonreversal blocks. Event-related brain potentials (ERPs) within a subsample of these participants further indicated that depressive symptoms were associated with reduced error positivity (Pe) amplitudes in both block types, suggesting that depressive symptoms were related to reduced attention allocation to errors across the easy and hard blocks. Finally, brain–behavior correlations indicated that highly depressed individuals failed to display a relationship between Pe amplitude and posterror accuracy in the S–R reversal blocks, a relationship that was intact in the low-depression group. Together, these results suggest that task demands play a critical role in the emergence of error-monitoring abnormalities in depression.

Keywords Depression · Posterror adjustments · Error monitoring · Response switching

The cognitive capacity to adapt after failures and setbacks is important to maintaining a healthy lifestyle. People who have a difficult time recovering from their failures are often at an increased risk for developing depressive symptoms. Decades of research have indicated that difficulties in coping with failures (e.g., attributing failures to a lack of ability, as opposed to a lack of effort or motivation) promote helpless behaviors (e.g., Dweck, 1975; Dweck & Reppucci, 1973). Helplessness—the feeling that the situation at hand is uncontrollable and will not improve—in turn, is associated with the onset and maintenance of depression (Klein, Fencil-Morse, & Seligman, 1976; Nolen-Hoeksema, Girgus, & Seligman, 1986). Moreover, depressed individuals are more likely to "overgeneralize" their failures, such that any particular failure indicates to the person that *he/she is a failure* (e.g., Beck, 2008). In sum, depressive feelings are associated with maladaptive reactions to failures.

Cognitive theorists have long conceptualized abnormal responses to failures as resulting from a deficit of basic information processing (Beck, Rush, Shaw, & Emery, 1979). More recent depression research has pointed to impairments in "cognitive control"—that is, the ability to monitor for errors and to implement appropriate behavioral adjustments (Miller & Cohen, 2001). Although these control impairments have been inferred from a number of studies, the precise mechanisms responsible for such impairments remain unclear. The present study was designed to shed new light on the contexts in which depressive symptoms relate to cognitive control deficits. First, we briefly review findings on depression and error monitoring and discuss the possible moderating role of

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task difficulty. Next, we describe an experiment designed to test whether task demands influence the effects of depression on error monitoring.

Depression and posterror adjustments: The moderating role of task difficulty

In addition to subjective reports of magnified reactions to failures (e.g., Wenzlaff & Grozier, 1988), emerging findings are indicating that individuals with depressive symptoms evidence impaired performance on trials following errors in cognitive tasks (e.g., Jones, Siegle, Muelly, Haggerty, & Ghinassi, 2010). However—as we review below—the findings are quite mixed. One way to understand the mixed findings regarding the relationship between depression and posterror performance is to more closely examine the tasks that have been used in previous studies. Cognitive theories indicate that depression significantly interferes with "effortful" processes, but only minimally with relatively "automatic" processes (Hartlage, Alloy, Vázquez, & Dykman, 1993; Roy-Byrne, Weingartner, Bierer, Thompson, & Post, 1986; Weingartner, Cohen, Martello, & Gerdt, 1981; see Austin, Mitchell, & Goodwin, 2001, and Pizzagalli, 2011, for relevant reviews). Whereas automatic processes are relatively simple and can be completed without attention or consciousness, effortful processes require conscious attention and are subject to capacity limitations (Hartlage et al., 1993). Austin et al. described the role of motivation as mediating the relationship between depression and poor effortful task performance. According to these authors, motivation, the ability to initiate activity either spontaneously or in response to environmental cues (Lezak, 1995), is inextricably linked with affect, and in particular with anhedonic symptoms of depression.

The anhedonic subtype of depression was particularly relevant for our focus on the effects of task demands and effort, as well as posterror adjustments, in the present study. Although anhedonia has traditionally been viewed as a condition defined by a lack of pleasure, more recent theorizing has emphasized that it is characterized by difficulties with decision-making. Specifically, Treadway and colleagues have suggested that anhedonia is associated with dysfunctional neurobiological mechanisms associated with reward motivation, and that this causes impairments in effortful cost–benefit decision-making analysis (Treadway, Bossaller, Shelton, & Zald, 2012; Treadway & Zald, 2011). Put another way, individuals with anhedonia often do not know when to engage in effortful behaviors. Because anhedonia is a core component of major depressive disorder (MDD; American Psychiatric Association, 2000) and is associated with motivational deficits in effortful tasks (Austin et al., 2001), some of the heterogeneity of the depression and error-monitoring findings may be attributed to the relative difficulty of the task at hand.

A review of the error-monitoring literature provides support for this notion. In more difficult neuropsychological batteries, depressed patients show marked performance deficits following errors (Beats, Sahakian, & Levy, 1996; Elliot, Sahakian, Herrod, Robbins, & Paykel, 1997; Elliot et al., 1996). Similar findings have emerged for individuals with depressive symptoms when performing variants of the Stroop (1935) and Simon (Compton et al., 2008; Holmes & Pizzagalli, 2007, 2008) tasks. In contrast, many studies using simpler flanker tasks (Eriksen & Eriksen, 1974) or go/no-go tasks have not shown any posterror performance impairments among depressed participants (Georgiadi, Liotti, Nixon, & Liddle, 2011; Ladouceur et al., 2012; Olvet, Klein, & Hajcak, 2010; Ruchsow et al., 2006; Ruchsow et al., 2004; Schrijvers et al., 2008; Schrijvers et al., 2009), and one study even revealed *enhanced* posterror accuracy in depressed patients (Chiu & Deldin, 2007). The only flanker task study to find behavioral differences used a stimulus degradation function to enhance difficulty on a trial-by-trial basis (Pizzagalli, Peccoralo, Davidson, & Cohen, 2006).

Although all of these conflict tasks require inhibition and selective attention to some extent, the Stroop and Simon tasks may be more difficult than flanker and go/no-go tasks because the former tasks require suppressing response tendencies that are well-practiced before coming into the lab—for instance, the automatic tendency to read the stimulus word (as opposed to naming its color) in the Stroop task. A recent within-subjects study showed that reaction times and error rates were higher in the Stroop task than in a flanker task and a go/no-go task (Riesel, Weinberg, Endrass, Meyer, & Hajcak, 2013). Because we are only aware of this one study comparing performance across these tasks, we compared overall reported accuracy from the 12 studies[1] (total $N = 464$) of error monitoring and depression listed above; the studies using Stroop/Simon tasks had significantly lower accuracy rates ($M = 87.82\%$) than did studies using flanker and go/no-go tasks ($M = 90.92\%$) [$t(462) = 2.98$, $p < .01$, $d = 0.28$].

Demands on cognition therefore may vary significantly across different types of tasks, and may have particularly important implications for depression-related impairments in behavior (cf. Austin et al., 2001). However, no study to date has directly compared how depressive symptoms relate to posterror performance in relatively simple versus more demanding task conditions.

Depression and error-related brain activity

Hundreds of studies have examined the neural mechanisms by which individuals detect, become aware of, and bounce

[1] The list of studies included in this analysis is provided in the supplementary materials.

back from their mistakes (for a review, see Gehring, Liu, Orr, & Carp, 2012). These studies often use simple reaction-time (RT) tasks such as the flanker task (Eriksen & Eriksen, 1974) to elicit errors and capture corresponding neural responses using event-related brain potentials (ERPs) or functional magnetic resonance imaging (fMRI). fMRI studies have shown that regions of the anterior cingulate cortex (ACC) and prefrontal cortex (PFC) are intimately involved in error monitoring and implementing appropriate behavioral adjustments to improve performance on subsequent trials (Carter et al., 1998; Hester, Madeley, Murphy, & Mattingley, 2009; Kerns et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000).

The most well-studied neural marker of error processing is the error-related negativity (ERN), a sharp negative deflection in the frontocentral ERP elicited immediately (50–100 ms) following an error (Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN has been broadly associated with the cognitive control operations of monitoring task performance for conflicts (Yeung, Botvinick, & Cohen, 2004) and errors (Holroyd & Coles, 2002) and of signaling for additional resources to implement behavioral adjustment (e.g., Debener et al., 2005). Consistent with its role in such performance monitoring, the ACC is considered to be a primary generator of the ERN (Dehaene, Posner, & Tucker, 1994; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004; van Veen & Carter, 2002). The ERN is followed by the error positivity (Pe), a positive-going ERP that reaches maximal amplitude at centroparietal locations (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Ridderinkhof, Ramautar, & Wijnen, 2009). As compared with the ERN, the Pe typically has a broader scalp distribution and a wider array of potential neural generators, including ACC (van Veen & Carter, 2002) and anterior insula (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010). Accumulating evidence has indicated that the Pe reflects conscious awareness of having made a mistake (e.g., Murphy, Robertson, Allen, Hester, & O'Connell, 2012; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). Thus, recent work has dissociated these two components and has suggested that the Pe reflects processes related to motivated awareness or attention allocation to errors, whereas the ERN is more indicative of a general conflict signal (Hughes & Yeung, 2011; Ridderinkhof et al., 2009; Steinhauser & Yeung, 2010). This dissociation nicely dovetails with evidence indicating that the Pe, more so than the ERN, relates to behavioral adjustments following errors, such as slower or more accurate responding (Frank, D'Lauro, & Curran, 2007; Moser, Schroder, Heeter, Moran, & Lee, 2011; Nieuwenhuis et al., 2001).

Although the relationship between the ERN/Pe and many forms of psychopathology has been studied extensively (Olvet & Hajcak, 2008), studies examining the ERN/Pe in relation to depression have yielded mixed results. For instance, whereas some studies have reported that MDD or depressive symptoms relate to an increased ERN (Alexopoulos et al., 2007; Chiu & Deldin, 2007; Georgiadi et al., 2011; Holmes & Pizzagalli, 2008, 2010; Kalayam & Alexopoulos, 2003), others have shown a *reduced* ERN (Ladouceur et al., 2012; Ruchsow et al., 2006; Ruchsow et al., 2004), and still others have shown no relationship between the ERN and depressive symptoms (Compton et al., 2008; Olvet, Klein, & Hajcak, 2010; Schrijvers et al., 2008; Schrijvers et al., 2009; Weinberg, Klein, & Hajcak, 2012).[2] The findings regarding the Pe are only slightly more consistent: Five studies have reported a reduced Pe in relation to depressive symptoms (Alexopoulos et al., 2007; Holmes & Pizzagalli, 2010; Olvet et al., 2010; Schrijvers et al., 2008; Schrijvers et al., 2009), four have shown no relationship between the Pe and depression (Chiu & Deldin, 2007; Compton et al., 2008; Georgiadi et al., 2011; Holmes & Pizzagalli, 2008), and four studies did not analyze the Pe (Kalayam & Alexopoulos, 2003; Ladouceur et al., 2012; Ruchsow et al., 2006; Ruchsow et al., 2004). In sum, the findings are quite inconsistent with regard to the ERN; however, a reduced or comparable Pe is more consistently associated with depressive symptoms. This might suggest that individuals with symptoms of depression have fewer resources to allocate attention in a way that would prevent future errors (e.g., Moser et al., 2011; Nieuwenhuis et al., 2001; Ridderinkhof et al., 2009).

The present study

Here, we sought to explicitly test the hypothesis that error-monitoring deficits associated with symptoms of depression vary as a function of task demands in a large nonclinical sample of undergraduates. We used a modified letter flanker task in which the S–R mappings were reversed between blocks. This design utilized a type of task switching known as *response switching* (Rushworth, Hadland, Paus, & Sipila, 2002; Rushworth, Passingham, & Nobre, 2002). Response switching increases task difficulty because of the well-known phenomenon that once a stimulus has been associated with a particular response, any later presentations of that stimulus will evoke the same response (Pavlov, 1927). When the original S–R mapping is repeated, this "binding" of stimulus to response facilitates performance, but when the

[2] Olvet et al. (2010) reported that within the MDD group, the ERN difference (ERN minus correct-response negativity, or CRN) amplitude was reduced with increasing depressive symptoms; this relationship was driven by the relationship to the CRN, not the ERN.

Weinberg et al. (2012) compared ERN amplitudes between patients with generalized anxiety disorder (GAD), with and without comorbid MDD, and a group of healthy control participants. They found a reduced ERN in the comorbid group relative to the GAD-only group, but no difference between the comorbid group and a group of healthy controls.

response rules change (i.e., when the same stimulus is now associated with a different response), this binding is detrimental to performance (e.g., Waszak, Hommel, & Allport, 2003).

We had previously found that this S–R reversal flanker task elicits reliable response-switching effects of increased RTs and error rates (Schroder, Moran, Moser, & Altmann, 2012). Therefore, this design differentiates the relatively easy blocks that precede the S–R reversal (which we refer to as "nonswitch blocks") from the more demanding blocks that follow the mapping reversals ("switch blocks"). Switch blocks require additional cognitive processes, such as inhibiting the previous (nonswitch) block's mapping and increasing focus on the current (switch) block's mapping to maintain adequate performance (Schroder et al., 2012).

Because successful switch-block performance requires relatively more effort to maintain the current mapping in memory, we expected that depression-related behavioral impairments would emerge in the switch blocks, and not in the nonswitch blocks. We focused on two indices of posterror adjustment: posterror slowing and posterror accuracy (see Danielmeier & Ullsperger, 2011, for a review). A ubiquitous finding of error-monitoring studies is that participants slow down on trials following errors (see Rabbitt, 1966); this is known as *posterror slowing*. Posterror slowing has been conceptualized as being adaptive (Botvinick, Braver, Barch, Carter, & Cohen, 2001) or as an automatic orienting response unrelated to task performance (Notebaert et al., 2009; Núñez Castellar et al., 2010). Posterror accuracy—the ability to correctly respond to the trial following an error—is conceptually adaptive in all task contexts (cf. Moser & Schroder, 2012). The precise "normative" pattern of slowing versus accuracy following error trials has not been systematically studied in a single large sample, and it appears that task types influence the dynamics of posterror adjustments (Danielmeier & Ullsperger, 2011). Nonetheless, in the extant literature, depressive symptoms more often relate to impaired accuracy, not RTs, following errors. Thus, we predicted that depressive symptoms would relate to poorer posterror accuracy in switch but not nonswitch blocks.

In a subsample of participants, we further examined how depressive symptoms related to error-related ERPs in nonswitch versus switch blocks. Unlike the behavioral findings in the extant studies reviewed above, the ERP results from previous studies cannot easily be accounted for by task demands. That is, studies using variants of the same task produce mixed results regarding ERN and Pe amplitudes as they relate to depression. As such, we were agnostic as to how ERPs would relate to depressive symptoms as a function of task demands. On the basis of previous research, however, we expected that, if anything, depression might be associated with a reduced Pe.

Method

Participants

A total of 205 undergraduates provided written consent and participated in our ongoing study of behavior, brain activity, and affective psychopathology. Data from some of these participants have been reported previously (Moran, Taylor, & Moser, 2012; Moser, Moran, & Jendrusina, 2012; Moser et al., 2011; Schroder et al., 2012). However, none of these studies examined depression; thus, the analyses reported here are novel. The experimental procedures were approved by the Michigan State University Institutional Review Board. All participants were compensated with partial course credit.

Task

Participants completed a letter version of the Eriksen flanker task (Eriksen & Eriksen 1974). They were instructed to respond to the center letter (target) of a five-letter string, with each string being either congruent (e.g., MMMMM) or incongruent (e.g., NNMNN). Characters were displayed in a standard white font on a black background and subtended 1.3º of the visual angle vertically and 9.2º horizontally. All of the stimuli were presented on a Pentium R Dual Core computer, using Presentation software (Neurobehavioral systems, Inc.) to control the presentation and timing of all stimuli, the determination of response accuracy, and the measurement of RTs.

During each trial, the flanking letters were presented 35 ms prior to target letter onset, and all five letters remained on the screen for a subsequent 100 ms (the total trial time was 135 ms). A fixation cross (+) was presented during the intertrial interval, which varied randomly between 1,200 and 1,700 ms at 100-ms intervals. Performance feedback was not given. The experimental session included 480 trials, grouped into 12 blocks of 40 trials, during which accuracy and speed were equally emphasized. Across the entire task, the ratio of congruent to incongruent trials was kept at 1:1.

The primary response-switching manipulation involved reversing the S–R mappings within a block pair (e.g., left button for a target "M" in Block 1, right button for a target "M" in Block 2). The letters making up the trial stimuli differed across block pairs: Blocks 1 and 2, "M" and "N"; Blocks 3 and 4, "F" and "E"; Blocks 5 and 6, "O" and "Q"; Blocks 7 and 8, "T" and "I"; Blocks 9 and 10, "V" and "U"; and Blocks 11 and 12, "P" and "R." Prior to each block, instructions regarding the letter–mouse button assignments (the S–R mappings) were presented on the computer screen. Nonswitch and switch blocks each comprised 240 total trials.

Self-report measures

Following completion of the flanker task, the electrodes and cap were removed, and participants completed several questionnaires, including the Anhedonic Depression (AD) and Anxious Arousal (AA) subscales of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991) and the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The MASQ asks participants to rate how much they have experienced feelings, sensations, and problems during the previous week, including today, on a Likert scale ranging from 1 (*not at all*) to 5 (*extremely*). The MASQ-AD subscale is composed of 21 statements related to feelings of anhedonia (e.g., "Felt really happy"; reverse-scored), and the MASQ-AA subscale is composed of 17 statements related to physiologic arousal (e.g., "Hands were cold or sweaty"). Both subscales had adequate internal reliability in the present sample (MASQ-AD α = .92, MASQ-AA α = .79). The PSWQ is a 16-item questionnaire designed to assess trait worry; it includes items such as "I have been a worrier all my life," and participants rate the degree to which each statement describes them on a 1 (*not at all typical of me*) to 5 (*very typical of me*) Likert scale. The PSWQ in the present sample demonstrated adequate internal reliability (α = .93).

The decision to use the MASQ-AD to assess depressive symptoms was motivated for two primary reasons. First, we wanted to collect a relatively "pure" measure of depression, distinct from related measures of anxiety. That is, the MASQ-AD subscale has been shown to have sound psychometric properties, in that it can be reliably differentiated from anxiety (Nitschke, Heller, Imig, McDonald, & Miller, 2001). These three measures—the MASQ-AD, MASQ-AA, and PSWQ—have been used in a number of studies differentiating effects of depression from effects of anxiety (e.g., Engels et al. 2007; Engels et al., 2010; Fisher et al., 2010; Larson, Nitschke, & Davidson, 2007; Nitschke, Heller, Palmieri, & Miller, 1999; Nitschke et al., 2001; Sass et al., 2010; Spielberg, Heller, Silton, Stewart, & Miller, 2011). Second, as we outlined above with regard to the effort and motivational deficits associated with depression, the MASQ-AD was particularly well suited for our focus on error monitoring in terms of task demands.

Psychophysiological recording

Continuous encephalographic (EEG) activity was recorded using the ActiveTwo BioSemi system (BioSemi, Amsterdam, The Netherlands). Recordings were taken from 64 Ag–AgCl electrodes placed in accordance with the 10–20 system. In addition, two electrodes were placed on the left and right mastoids. Electrooculogram (EOG) activity generated by eye movements and blinks was recorded at FP1 and at three additional electrodes placed inferior to the left pupil and on the left and right outer canthi (all approximately 1 cm from the pupil). During data acquisition, the common-mode sense active electrode and the driven right-leg passive electrode formed the ground, as per BioSemi's design specifications. All signals were digitized at 512 Hz using ActiView software (BioSemi). Offline analyses were performed using BrainVision Analyzer 2 (BrainProducts, Gilching, Germany). Scalp electrode recordings were re-referenced to the numeric mean of the mastoids and band-pass filtered with cutoffs of 0.1 and 30 Hz (12 dB/oct roll off). Ocular artifacts were corrected using the method developed by Gratton, Coles, and Donchin (1983). The response-locked data were segmented into individual epochs beginning 100 ms before response onset and continuing for 800 ms following the response. Physiologic artifacts were detected using a computer-based algorithm, such that trials in which the following criteria were met were rejected: a voltage step exceeding 50 μV between contiguous sampling points, a voltage difference of more than 200 μV within a trial, or a maximum voltage difference less than 0.5 μV within a trial. The ERN and its correct-trial counterpart (the correct-response negativity) were quantified as the average voltage in the 0- to 100-ms postresponse time window. The Pe and its correct-response counterpart were quantified as the average voltage in the 150- to 350-ms postresponse time window. Both ERPs were calculated using a 100-ms preresponse baseline correction. These ERP quantifications were identical to those reported in our previous response-switching study (Schroder et al., 2012).

Data preparation and analysis

The data were analyzed in two steps. First, the behavioral data were analyzed to determine whether depressive symptoms were related to posterror adjustment impairments in these data across the entire sample of usable participants (N = 171; see below for the exclusion criteria). Next, usable ERP data from a subset of participants (N = 67) were analyzed to determine whether depressive symptoms were also related to error-related ERPs as a function of block type (nonswitch vs. switch).

Behavior Participants' data were excluded prior to analysis if (1) they failed to follow the S–R mapping task instructions, which resulted in performance below 50% in any one block (N = 28); or (2) posterror accuracy data were missing in any one block type (nonswitch or switch; N = 6). Thus, the data from a total of 34 participants were excluded prior to analysis, leaving a final sample of 171 participants (102 female, 69 male) for the behavioral data analysis.

Their overall RT and accuracy data were analyzed with a two-level within-subjects factor (Block Type: nonswitch vs. switch) repeated measures analysis of variance (ANOVA). Posterror adjustment data (RT and accuracy) were analyzed using separate 2 (Response Type: posterror vs. postcorrect) × 2 (Block Type: nonswitch vs. switch) ANOVAs. To test the primary hypotheses of the present study, the same 2 (Response Type) × 2 (Block Type) analysis was rerun with MASQ-AD entered as a continuous covariate in the SPSS GLM repeated measures module (see Moran et al., 2012; Moser et al., 2012; Moser et al., 2011, for a similar procedure).

ERP data The 67 participants with usable EEG data previously reported on in our response-switching study (Schroder et al., 2012) were also included in the present analyses of the ERN and Pe and their relationships to MASQ-AD. In brief, of the original 171 participants, 53 were excluded from the EEG analyses for excessive artifacts in the raw data. An additional 51 participants were removed because they did not have a sufficient number (i.e., < 6) of usable error-trial ERPs in both the nonswitch and switch block types to generate reliable ERNs (Olvet & Hajcak, 2009). Although we acknowledge that this data loss was particularly large, our final sample size is well within the average size of those reported in other ERP studies of depression (e.g., Compton et al., 2008). Moreover, the included and excluded participants did not differ in MASQ-AD scores [included, M = 50.58, SD = 13.60; excluded, M = 48.91, SD = 13.16; $t(169) < 1$], gender distribution [36 females, 31 males included; 66 females, 38 males excluded; $\chi^2(1) = 1.60$, p = .21], or age [included, M = 20.29, SD = 3.87; excluded, M = 20.17, SD = 3.65; $t(167) = 0.19$, p = .85]. The overall effects of the response-switching task have been reported in our previous investigation (Schroder et al., 2012). For the purposes of the present investigation, we focused on the relationships between the ERN and the Pe and depressive symptoms using separate 5 (Site: Fz, FCz, Cz, CPz, Pz) × 2 (Block Type: nonswitch vs. switch) × 2 (Trial Type: error vs. correct) ANOVAs with MASQ-AD scores entered as a continuous covariate.

The data were statistically evaluated using SPSS (Version 20.0) General Linear Model (GLM) software. Measures with skewness or kurtosis values greater than 2 were transformed in order to normalize the distributions. MASQ-AA scores were successfully log-transformed. To achieve homogeneity of variances in accuracy, the mean accuracies were arcsine-transformed (Keppel & Wickens, 2007); descriptive statistics for these measures are reported using the raw data. The Greenhouse–Geisser correction for p values is reported, and partial eta squared (η^2_p) is reported as an estimate of effect size, such that .05 is considered a small effect, .1 a medium effect, and .2 a large effect (Cohen, 1969).

Results

Self-report measures

Across the whole sample (N = 171), scores for the MASQ-AD (M = 49.57, SD = 13.32), MASQ-AA (M = 24.87, SD = 6.77), and PSWQ (M = 50.60, SD = 12.99) were similar to those from previous reports in college samples (e.g., Nitschke et al., 2001). MASQ-AD was related to scores for both the MASQ-AA and PSWQ (rs = .41 and .33, respectively, ps < .001), and MASQ-AA scores were also related to the PSWQ [$r(169)$ = .27, p < .001]. These moderate correlations replicate previous findings and indicate that they are nonredundant measures of negative affective pathology (Nitschke et al., 2001).

Behavioral performance results (N = 171)

All behavioral measures are presented in Table 1. In line with the notion that switch blocks required additional cognitive processing and were more demanding (Schroder et al., 2012), they were associated with longer RTs [$F(1, 169) = 98.99$, $p < .001$, $\eta^2_p = .37$] and lower accuracy [$F(1, 169) = 27.68$, $p < .001$, $\eta^2_p = .14$], as compared with nonswitch blocks.

Posterror accuracy For posterror accuracy, the main effect of response type was significant [$F(1, 170) = 26.01$, $p < .001$, $\eta^2_p = .13$], indicating that overall accuracy was slightly lower following error trials (M = 92.13, SD = 7.99) than following correct trials (M = 93.41, SD = 5.07). The main effect of block type was also significant [$F(1, 170) = 26.78$, $p < .001$, $\eta^2_p = .14$]; posttrial accuracy was lower in switch blocks, mirroring the overall effect of switch block on accuracy.

Table 1 Means (and standard deviations) of our behavioral performance measures

Measure	Nonswitch	Switch
Error RT	351.60 (54.23)	394.15 (71.35)
Correct RT	447.70 (47.18)	459.10 (46.40)
Correct minus error RT	96.10 (43.80)	64.94 (61.62)
Accuracy (% Correct)	93.84 (5.22)	92.83 (5.30)
Posterror RT	475.47 (67.08)	510.65 (78.34)
Postcorrect RT	443.82 (47.56)	454.16 (46.61)
Posterror slowing (RT)	31.65 (49.22)	56.49 (61.59)
Posterror accuracy	94.82 (7.62)	89.70 (12.09)
Postcorrect accuracy	93.69 (5.33)	93.12 (5.21)
Posterror accuracy difference	1.13 (6.19)	−3.42 (11.43)

Behavioral data are here presented as means (and standard deviations) from the full sample (N = 171). Posterror accuracy difference scores were calculated as posterror accuracy minus postcorrect accuracy. Note that all contrasts between nonswitch and switch blocks were significant (p < .01), and that raw data are reported

Importantly, an interaction between response type and block type emerged [$F(1, 170) = 17.88$, $p < .001$, $\eta^2_p = .10$], indicating that posterror accuracy was significantly greater than postcorrect accuracy in nonswitch blocks [$t(170) = 8.24$, $p < .001$, $d = 0.61$], but was nonsignificantly *lower* than postcorrect accuracy in switch blocks [$t(170) < 1$, $p = .55$, $d = 0.05$; see Table 1]. Follow-up t tests revealed that the difference between switch and nonswitch blocks was larger for posterror accuracy [$t(170) = 4.83$, $p < .001$, $d = 0.45$] than for postcorrect accuracy [$t(170) = 3.15$, $p < .01$, $d = 0.14$]. This indicates that participants were more likely to commit consecutive errors during switch blocks.

Importantly, and as is depicted in Fig. 1, the response type × block type × MASQ-AD interaction was significant [$F(1, 169) = 5.82$, $p = .02$, $\eta^2_p = .03$].[3] Follow-up correlations indicated that higher MASQ-AD scores were negatively related to posterror accuracy in switch blocks [$r(169) = -.24$, $p = .001$], but were unrelated to posterror accuracy in nonswitch blocks [$r(169) = -.09$, $p = .24$]. MASQ-AD scores were unrelated to postcorrect accuracy in both block types (rs < .13, ps > .11). Moreover, MASQ-AD scores were related to posterror accuracy difference scores (posterror accuracy minus postcorrect accuracy) in switch blocks [$r(169) = -.24$, $p < .01$] but not in nonswitch blocks [$r(169) = -.03$, $p = .70$; see Fig. 1]. Importantly, these correlations were significantly different from each other ($Z = 1.98$, $p < .05$, two-tailed).

Posterror slowing A main effect of response type emerged [$F(1, 170) = 160.45$, $p < .001$, $\eta^2_p = .49$], such that RTs were longer for posterror trials ($M = 493.40$, $SD = 64.86$) than for postcorrect trials ($M = 448.99$, $SD = 46.04$), indicating the typical posterror slowing effect. Moreover, the main effect of block type was significant [$F(1, 170) = 66.60$, $p < .001$, $\eta^2_p = .28$]: Posttrial RTs were longer in switch blocks. The response type × block type interaction was also significant for RTs [$F(1, 170) = 25.40$, $p < .001$, $\eta^2_p = .13$], such that posterror slowing (posterror RT minus postcorrect RT) was greater in switch blocks (see Table 1). We found no significant interactions with MASQ-AD scores for posterror RTs (all Fs < 1).

Subsample analyses ($N = 67$)

Behavior In the subsample of participants with usable EEG data, the three-way interaction between response type, block type, and MASQ-AD was significant for posterror accuracy [$F(1, 65) = 7.01$, $p = .01$, $\eta^2_p = .10$], consistent with the effect from the overall sample. Follow-up correlations confirmed that MASQ-AD scores were related to poorer posterror accuracy difference scores (posterror minus postcorrect accuracy) in switch than in nonswitch blocks [$r(65) = -.31$, $p = .01$].

ERN Response-locked ERP waveforms are depicted in Fig. 2. In the 0- to 100-ms postresponse time window, we found no interactions involving MASQ-AD (all Fs < 1.31, ps > .25). That is, the ERN was not related to anhedonic depression symptoms in the present sample.

Pe In the 150- to 350-ms postresponse time window, a significant Trial Type × MASQ-AD interaction emerged [$F(1, 65) = 6.38$, $p = .01$, $\eta^2_p = .09$]. Follow-up correlations indicated that as MASQ-AD scores became higher, error-trial Pe amplitudes (averaged across nonswitch and switch blocks across all five midline sites) became smaller [$r(65) = -.32$, $p < .01$]. Correct-trial Pe amplitudes were unrelated to MASQ-AD scores ($r = .01$, $p = .91$). MASQ-AD scores were therefore significantly related to reduced Pe difference amplitudes [Pe minus Pe on correct trials: $r(65) = -.30$, $p = .01$]. We observed no interactions involving MASQ-AD and block type, nor between MASQ-AD and site; the relationships between MASQ-AD and Pe difference amplitude were similar across the five midline sites (Fz, $r = -.25$; FCz, $r = -.28$; Cz, $r = -.28$; CPz, $r = -.28$; Pz, $r = -.29$).

Given that depression was associated with poorer posterror accuracy and a blunted Pe, we further explored the relationships between these three variables. In our previous report on the overall ERP findings in this response-switching task (Schroder et al., 2012), we demonstrated that during switch blocks, a larger Pe was associated with greater posterror accuracy. To evaluate whether depression altered this association, we compared brain–behavior relationships between individuals scoring in the upper and lower halves of the MASQ-AD distribution (on the basis of a median split). In the low-AD group ($N = 32$), the switch-block Pe difference (error – correct) amplitude at CPz was significantly related to higher switch-block posterror accuracy differences [$r(30) = .41$, $p < .05$]. Importantly, this relationship was absent in the high-AD group ($N = 35$): $r(33) = -.03$, $p = .85$; $Z = 1.83$, $p = .07$, two-tailed. Correlations at nearby sites revealed similar results (Cz: low-AD, $r = .32$, $p = .07$; high-AD, $r = -.09$, $p = .62$; Pz: low-AD, $r = .40$, $p < .05$; high-AD, $r = .05$, $p = .79$). Thus, low-depressed individuals were primarily responsible for our previously reported overall correlation, whereas highly depressed individuals failed to demonstrate this functional association.

Effects of anxiety and sex

Controlling for anxiety (MASQ-AA and PSWQ) yielded nearly identical results for the effects reported above. For the posterror accuracy analysis, the response type × block type × MASQ-AD interaction remained significant when these variables were

[3] When considering possible fatigue effects by adding task half (first vs. second) to the response type × block type × MASQ-AD ANCOVA, virtually identical results were produced. This suggests that the effects of depressive symptoms on posterror adjustments in switch blocks were present across the entire task.

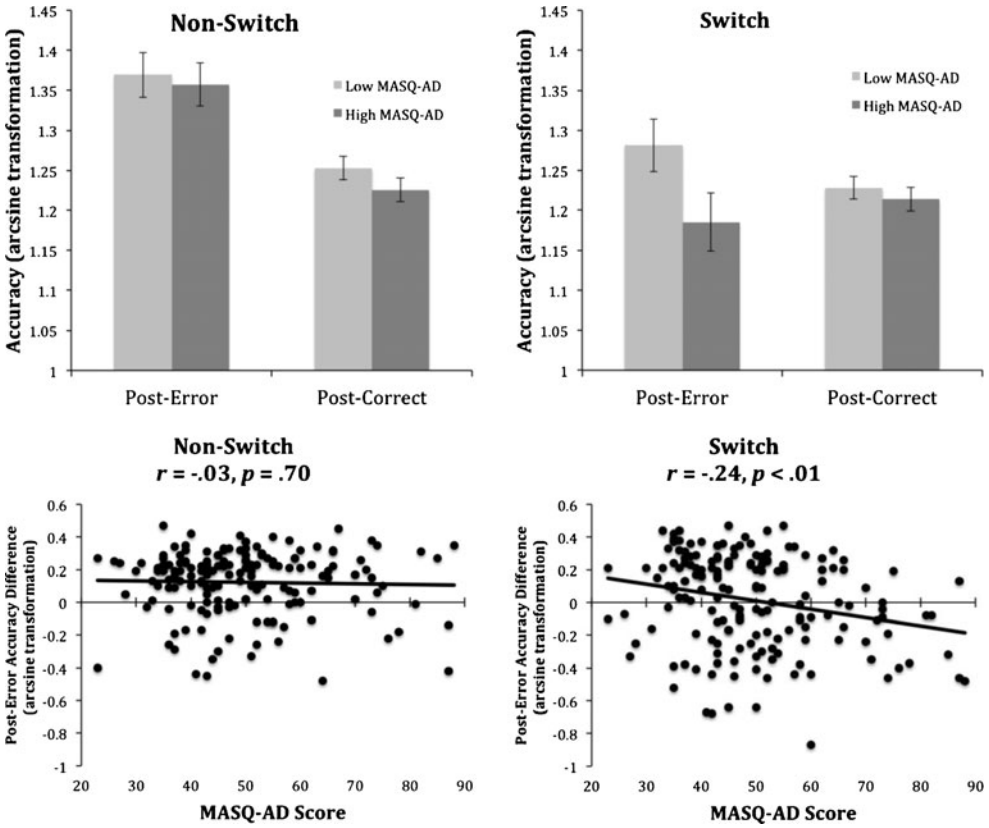

Fig. 1 Top: Bar graphs depicting the relationship between depression (MASQ-AD) and posttrial accuracy (arcsine-transformed) by block type from the full sample (N = 171). The MASQ-AD groups were created using a median split and are provided for illustrative purposes only (high MASQ-AD, n = 84; low MASQ-AD, n = 87). Error bars represent ±1 *SEM*. Bottom: Scatterplots depicting the relationship between MASQ-AD and posterror accuracy difference (arcsine-transformed) by block types

controlled for in the full [$F(1, 167) = 6.35$, $p = .01$, $\eta^2_p = .04$] and usable-ERP [$F(1, 63) = 6.91$, $p = .01$, $\eta^2_p = .10$] samples. For the Pe, the response type × MASQ-AD interaction was nearly significant [$F(1, 63) = 3.75$, $p = .06$, $\eta^2_p = .06$]. Controlling for participant sex yielded very similar results. For posterror accuracy, the response type × block type × MASQ-AD interaction remained significant in the full [$F(1, 168) = 5.68$, $p = .02$, $\eta^2_p = .03$] and the usable-ERP [$F(1, 64) = 6.60$, $p = .01$, $\eta^2_p = .09$] samples. For the Pe, when sex was added as a between-subjects variable in the ANCOVA, the interaction between response type and MASQ-AD remained significant [$F(1, 64) = 6.03$, $p < .05$, $\eta^2_p = .09$]. Thus, anxiety and participant sex were not the primary sources of depression's effects on error-related behavior and brain activity.

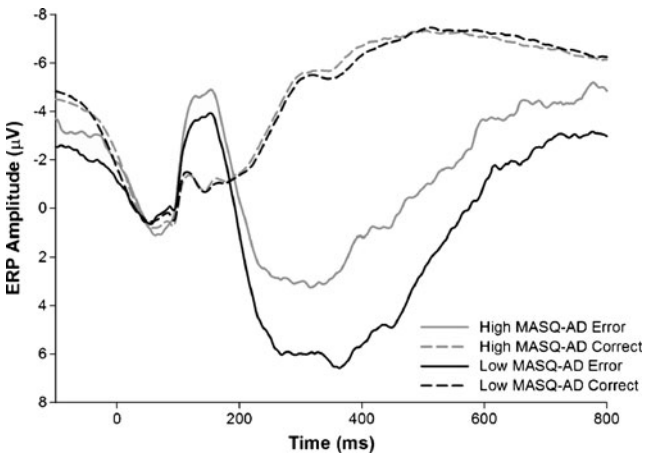

Fig. 2 Response-locked event-related potential (ERP) waveforms averaged across electrodes Fz, FCz, Cz, CPz, and Pz for high-MASQ-AD participants (n = 35) and low-MASQ-AD participants (n = 32). The groups were created using a median split of the usable-ERP subsample (N = 67) for illustrative purposes only

Discussion

The primary aim of the present study was to explore the moderating role of task demands on the association between error-monitoring deficits and depressive symptoms. Although abnormal reactions to failure have long been a hallmark feature of depression, the specific neural and behavioral underpinnings of this dysfunction are not yet well understood. Building from early theories that posited that effortful, but not automatic, tasks are most affected by depression (Hartlage et al., 1993), we used a modified flanker task that

differentiated relatively more demanding (switch) from relatively easier (nonswitch) blocks (Schroder et al., 2012). Consistent with these theories, depressive symptoms were associated with reduced posterror accuracy in the more-demanding switch blocks, and not in the nonswitch blocks. In a subsample of participants with available ERP data, we found that depressive symptoms were associated with reduced Pe amplitudes across both block types, but were not significantly associated with the ERN. We further found that individuals endorsing more symptoms of depression failed to show a positive association between Pe and posterror accuracy in the switch blocks, whereas this relationship was present in the lower-symptomatic group. Our findings suggest that task demands are important moderators of associations between error-monitoring deficits and depression. Below, we discuss these results in the context of current theories of depression and cognitive control.

Depression and posterror adjustments

In the present study, the relationship between depressive symptoms and posterror performance was specific in a number of ways. First, as hypothesized, depressive symptoms related to poorer posterror accuracy only in switch blocks. Because response switching is associated with increased control demands (Hsieh & Wu, 2011; Rushworth, et al. 2002a; 2002b), this finding suggests that individuals with greater symptoms of depression might not have adequate resources to bounce back from their errors, but only when control demands are high.

Second, depressive symptoms were correlated with posterror *accuracy*, and not with posterror slowing. This is an important distinction, as these measures have been dissociated in a number of studies (see Danielmeier & Ullsperger, 2011, for a review). Although the debate is ongoing, posterror slowing has been conceptualized as an orienting response that draws attention to an uncommon event (i.e., an error; Notebaert et al., 2009; Núñez Castellar et al., 2010). Thus, posterror slowing may not be inherently adaptive in every task context (Danielmeier & Ullsperger, 2011; Jentzsch & Dudschig, 2009). Rather, posterror *accuracy* may be a more reliable gauge of how "in control" an individual is after making an error, regardless of recovery time (slowing; Moser & Schroder, 2012). Our findings therefore further highlight the specificity of depression-related impairments to task-related behaviors aimed at improving performance following errors, rather than an automatic posterror orienting response in more difficult contexts.

Depression and error-related brain activity

In the present sample, depressive symptoms were negatively correlated with Pe amplitude across both block types. That the ERN was unrelated to depressive symptoms is inconsistent with several previous studies on depressed patients that reported enhanced ERNs (Alexopoulos et al., 2007; Chiu & Deldin, 2007; Georgiadi et al., 2011; Holmes & Pizzagalli, 2008, 2010), and with other studies that reported reduced ERNs (Ladouceur et al., 2012; Ruchsow et al., 2006; Ruchsow et al., 2004). However, our findings of unrelated ERNs but of reduced Pe amplitudes being associated with depressive symptoms are consistent with the only studies that have used the flanker task and evaluated the Pe component (Olvet et al., 2010; Schrijvers et al., 2008; Schrijvers et al., 2009). As we noted in the introduction, the ERN findings in depression are much less consistent than the Pe findings.

The specificity of depressive symptoms' relationship to the Pe is particularly significant for a number of reasons. Given the current conceptualizations of the Pe as an index of conscious error awareness and motivated attention allocation (Hughes & Yeung, 2011; Ridderinkhof et al., 2009; Steinhauser & Yeung, 2010; Ullsperger et al., 2010) aimed at facilitating behavioral adjustments that improve performance (Frank et al., 2007; Hajcak, McDonald, & Simons, 2003; Nieuwenhuis et al., 2001), these findings suggest the more-depressed participants allocated fewer resources to their errors across the task. Although this appeared to be sufficient during the relatively nondemanding nonswitch blocks (as no associated behavioral impairments occurred with these blocks), this reduced attention allocation was accompanied by behavioral deficits in the more-demanding switch blocks. These results might indicate that individuals with more depressive symptoms have fewer attentional control resources at their disposal in general, and that performance is unimpaired until more difficult task contexts are encountered.

Finally, brain–behavior relationships were moderated by depressive symptoms. Whereas larger Pe amplitudes in switch blocks correlated with improved behavioral performance after mistakes for the less-depressed participants, this relationship was absent among the more-depressed. These results are consistent with previous findings demonstrating a fundamental decoupling of brain activity and behavior in depression (Compton et al., 2008; Holmes & Pizzagalli, 2008; Pizzagalli et al., 2006). Clearly, these studies demonstrate that depression is associated with difficulties in optimally signaling for adaptive behavior following errors (see also Fales, Barch, Rundle, et al., 2008b).

Depression and effortful cognitive control

Although it has long been acknowledged that certain types of tasks may be more sensitive than others to depression-related impairments in cognitive performance (cf. Austin et al., 2001; Weinberg et al., 2012), this possibility has not been

directly tested in terms of error monitoring. Although we found evidence for taskwide, demand-independent reductions in motivated attention allocation to errors (i.e., the Pe; Ridderinkhof et al., 2009), depression-related difficulties in bouncing back from mistakes were only apparent in switch blocks. These findings provide novel support for the cognitive-effort hypothesis (Hartlage et al., 1993) and indicate that consideration of task demands is important in the evaluation of cognitive-control deficits associated with depression.

Finally, we speculate that cognitive-control deficits associated with depression might be conceptualized from Braver's "dual mechanisms of control" theory (Braver, 2012; Braver, Gray, & Burgess, 2007). This model suggests that cognitive control operates from two modes: a sustained *proactive* mode, in which task representations are continuously maintained in working memory and task conflicts can be resolved preemptively, and a transient *reactive* mode, in which goals are updated on an "as needed" basis *after* task conflicts arise. Here we speculate that depressive symptoms interfere with the engagement of proactive control, as this mode is more effortful and resource-demanding than is the reactive-control mode (Braver et al., 2007). Indeed, impairments in proactive-control engagement have been associated with related psychopathology such as anxiety (Fales, Barch, Burgess, et al., 2008; Krug & Carter, 2012; Osinksy, Alexander, Gebhardt, & Hennig, 2010). Moreover, depression has been associated with impairments in tasks that require task switching and working memory updating (e.g., De Lissnyder et al., 2012; Meiran, Diamond, Toder, & Nemets, 2010), as well as those requiring sustained cognitive control and emotional suppression (e.g., Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). In the present study, then, switch blocks likely required relatively more proactive control than did nonswitch blocks in order to keep the S–R mappings in mind (cf. Schroder et al., 2012). Although this account is speculative, in that studies examining reactive and proactive control do not typically consider the flanker task (Braver et al., 2007), the additional effort and resource demands associated with switch blocks were sufficient to elicit depression-related behavioral impairments following errors.

Limitations and conclusion

Despite the strengths of the present study, such as the relatively large sample size, one limitation is that full diagnostic interviews (e.g., structured clinical interviews from the DSM-IV) were not completed, potentially limiting the clinical implications of our findings. Moreover, we focused on a specific subtype of depression—anhedonic depression. However, the MASQ-AD—the measure utilized in the present study to identify depression symptoms—has been shown to be useful in screening for depressive disorders in undergraduate populations (Bredemeier et al., 2010). Furthermore, the dimensional nature of our findings indicates that the relationship between depression symptom severity and impaired attention allocation and posterror adjustment is dimensional in nature, thus complementing and extending patient studies. To our knowledge, this is only the second study to consider the relationship between depressive symptoms and error-monitoring functioning in healthy young adults (Compton et al., 2008). More studies considering depressive symptoms along a continuum of severity will therefore be necessary.

We must further acknowledge that our hypothesis that task demands account for the heterogeneity of findings in the error-monitoring literature is only tentative. Differences in task parameters other than "cognitive demand" are abundant in previous studies, and other explanations should also be considered in future work. Future studies examining how depressive symptoms relate to error monitoring using different types of tasks (e.g., Stroop, flankers) in the same sample would provide for additional tests of the hypotheses posited here. Finally, the task conditions in which the ERN and Pe are modulated by depressive symptoms were not made clear in the present study, as we found that taskwide reductions in Pe amplitude were associated with depression. Indeed, depression studies report inconsistent findings regarding the ERN and Pe, even when using the same types of tasks. Future research will need to evaluate more closely how these components relate to depressive symptoms in the context of different task conditions.

In conclusion, the present study offered some much needed specificity on the depression–error monitoring relationship. The primary findings were that symptoms of depression were related to reduced attention allocation to errors across the whole task, but to impaired posterror accuracy only during portions of the task that were more demanding.

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