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**Suppression of error-preceding brain activity explains
exaggerated error monitoring in females with worry**

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Abstract

Anxiety is consistently associated with hyperactive neural responses to errors. The majority of existing research has focused on a single marker of error-elicited brain activity – the error-related negativity (ERN), an event-related brain potential (ERP) elicited 50-100ms following an erroneous response. The ERN has accumulated growing interest for its use in clinical contexts as a potential biomarker and/or endophenotype. However, it is unknown whether anxiety's effects are specific to brain activity following erroneous responses; anxiety may affect processes prior to error commission, suggesting that the ERN might reflect the output of abnormal processing that begins before an error. Here, we examined the error-preceding positivity (EPP) – an ERP time-locked to the correct response immediately before errors – that reflects a gradual disengagement of task-focused attention preceding errors. Results revealed that female worriers demonstrated significantly attenuated EPP amplitude, indicating reduced pre-error disengagement. Moreover, reduced EPP mediated the relationship between worry and the enhanced ERN following errors. These results suggest that the temporal dynamics of anxiety's impact on error processing are more nuanced than previously thought such that effects emerge prior to the actual occurrence of an erroneous response.

Keywords: Anxiety, error monitoring, cognitive control, ERN, error-preceding positivity

Introduction

Anxiety and its disorders constitute some of the most common mental health problems in the world and cost billions of dollars each year in treatment and lost work productivity (e.g., Baxter, Scott, & Whiteford, 2013; Baxter, Vos, Scott, Ferrari, & Whiteford et al., 2014; Kazdin & Blase, 2011; Kessler, Chiu, Demler, & Walters, 2005). Recent years have seen an explosion of research focused on understanding the basic cognitive mechanisms that give rise to and maintain anxiety (e.g., MacLeod & Mathews, 2012; Teachman, Joormann, Steinman, & Gotlib, 2012). In particular, the methods of cognitive neuroscience have been seen as a promising tool for identifying novel markers of pathology and treatment targets (Cuthbert, 2014; First, 2012; Insel, 2007; Insel et al., 2010; Siegle, Ghinassi, & Thase, 2007; Simpson, 2012). The current study took such an approach to elaborate on a key cognitive bias associated with anxiety – exaggerated error monitoring.

Anxiety and the Error-Related Negativity (ERN)

One of the most consistently reported neurocognitive markers of anxiety and its disorders is increased amplitude of the error-related negativity (ERN), an event-related brain potential (ERP) elicited after errors in simple reaction time tasks (Falkenstein, Hoormann, & Hohnsbein, 1999; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Gehring, Liu, Orr, & Carp, 2012). The ERN occurs immediately (within 100ms) after response errors and has been reliably associated with regions of the medial frontal cortex such as the anterior cingulate cortex (ACC; Dehaene, Posner, & Tucker, 1994; Gehring et al., 2012; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004; van Veen & Carter, 2002). The ACC has been broadly implicated in the neural circuitry associated with monitoring for errors and conflicts in performance (Carter et al., 1998; Kerns et

al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000; van Veen & Carter, 2006) and signaling to other brain regions that behavioral adjustment is necessary (MacDonald et al., 2000; Shenhav, Botvinick, & Cohen, 2013). As such, the ACC has long been considered necessary for optimal performance and cognitive control (Shackman et al., 2011), and indeed, enhanced ACC activity often coincides with improved performance (e.g., Gehring et al., 1993; Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004). However, the vast majority of ERN studies of anxiety find enhanced ERN despite no improvements in performance (see Moser, Moran, Schroder, Donnellan, & Yeung, 2013; Weinberg, Riesel, & Hajcak, 2012 for reviews; however, see Moran, Bernat, Avieyente, Schroder, & Moser, 2015).

The ERN is enhanced among individuals diagnosed with generalized anxiety disorder (e.g., Weinberg, Olvet, & Hajcak, 2010; Xiao et al., 2011), obsessive-compulsive disorder (e.g., Gehring, Himle, & Nisenson, 2000; Klawohn, Riesel, Grützmann, Kathmann, & Endrass, 2014; Riesel, Endrass, Kaufmann, & Kathmann, 2011), as well as individuals with elevated levels of trait worry (e.g., Hajcak, McDonald, & Simons, 2003, 2004; Moser, Moran, & Jendrusina, 2012) and obsessive-compulsive symptoms (Grundler, Cavanagh, Figueroa, Frank, & Allen, 2009; Hajcak & Simons, 2002; Kaczurkin, 2013). Developmental studies indicate that enhanced ERN in anxiety is present among children and adolescents (e.g., Hanna et al., 2012; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; Torpey et al., 2013).

Given the robust relationship between anxiety and the ERN, understanding its clinical utility as a biomarker of risk for developing an anxiety disorder has become a focus of research (e.g., Manoach & Agam, 2013; Weinberg et al., 2012; Weinberg, Dieterich, & Riesel, 2015). This interest is readily apparent in the number of studies devoted to understanding the heritability (Anokhin, Golosheykin, & Heath, 2008), genetic associations (e.g., Beste et al.,

2013; Fallgatter et al., 2004), and basic psychometric properties of the ERN (e.g., Meyer, Riesel, & Proudfit, 2013; Riesel, Weinberg, Endrass, Meyer, & Hajcak, 2013). Indeed, many authors have argued the enlarged ERN may represent an endophenotype for anxiety and anxiety-related disorders (Olvet & Hajcak, 2008; Riesel et al., 2011; Riesel, Endrass, Auerbach, & Kathmann, 2015).

Despite the widespread interest in utilizing the ERN as a biomarker of anxiety, surprisingly few have considered the functional significance of enlarged ERN in anxiety. Early studies suggested the enlarged ERN was the result of a hyperactive response monitoring system (Gehring et al., 2000) whereas others suggested it reflected sensitivity to errors in anxious individuals (Hajcak et al., 2003). In a meta-analysis and theoretical conceptualization (Moser et al., 2013), we have recently incorporated cognitive models of anxiety (attentional control theory, ACT; Eysenck, Derakshan, Santos, & Calvo, 2007) and cognitive control (Braver, 2012; Yeung et al., 2004) to account for increased ERN in anxiety. Our meta-analysis found that the relationship between anxiety and the ERN is specific to the anxious apprehension / worry subtype of anxiety, as opposed to more physiological / unspecified types of anxiety. Our compensatory error-monitoring hypothesis (CEMH) attempted to account for this anxious neurocognitive profile of exaggerated error-monitoring brain activity but equivalent (and not improved) performance. We proposed that enlarged ERN is reflective of a compensatory effort process that worriers engage in to counteract the resource-depleting effects of worries. We hypothesized that the ERN could reflect the output of this process, suggesting that error-monitoring abnormalities associated with anxiety likely begin prior to the error. However, there has been no study examining pre-error brain activity in anxiety, a void the current study sought to fill.

Brain Activity Preceding Errors

Regardless of the specific cause, most errors are preceded by a gradual decline in task-related attention (Eichele et al., 2008; Eichele, Juvodden, Ullsperger, & Eichele, 2010; Steinhauser et al., 2012). This decline in attention has been captured by functional magnetic resonance imaging (fMRI) studies of the default-mode network (DMN), a collection of brain regions that are classically associated with resting states (Gusnard & Raichle, 2001) and off-task processing (Greicius, Krasnow, Reiss, & Menon, 2003; Raichle et al., 2001). As such, past work suggests that errors are preceded by increased DMN activity (Li, Yan, Bergquist, & Sinha, 2007).

ERPs have also been used to capture pre-error brain activity. Earlier research described an ERP that was time-locked to correct responses that precede errors, referred to as the error-preceding positivity (EPP; Allain, Carbonnell, Falkenstein, Burle, & Vidal, 2004; Hajcak, Nieuwenhuis, Ridderinkhof, & Simons, 2005; Ridderinkhof, Nieuwenhuis, & Bashore, 2003). Specifically, this response-locked ERP is larger (i.e., more positive) on pre-error trials versus pre-correct trials. The EPP has been suggested to reflect the gradual attentional decline immediately preceding errors (Hajcak et al., 2005; Li et al., 2007; Simons, 2010) and has been hypothesized to reflect DMN activity (Li et al., 2007). Unfortunately, few studies have examined the EPP, and despite the plethora of anxiety-ERN papers described above, no study has examined the relationship between anxiety and the EPP (or how anxiety relates to pre-error brain activity using fMRI). Thus, our knowledge about how anxiety influences error monitoring is constrained to brain activity that occurs after errors are committed.

This study sought to address this gap in knowledge by examining how anxiety impacts error processing before errors are committed. Investigating the relationship between anxiety

and pre-error brain activity (i.e., the EPP) would be informative for several reasons. First, it would provide for a more comprehensive understanding of how anxiety influences error monitoring by extending the temporal focus to trials leading up to the error. Indeed, the neural activity associated with the ERN does not occur in isolation; rather, it is part of a system involved in monitoring performance that likely waxes and wanes in activity during trials surrounding errors (Ridderinkhof et al., 2003; Steinhauser et al., 2012). Extending the window of focus would also allow us to test our hypothesis that error-monitoring modulations (compensatory effort) associated with anxiety are present before the error occurs (Moser et al., 2013). Second, from a clinical standpoint, the EPP may provide incremental understanding of the etiology and risk for anxiety, in addition to the ERN. Third, because it is captured on trials immediately before errors, the EPP can be extracted from any ERN dataset, regardless of paradigm (cf. Hajcak et al., 2005; Masaki, Murphy, Kamijo, Yamazaki, & Sommer, 2012), allowing for the re-analysis of existing datasets and therefore an amassing of a corpus of novel information related to anxiety and error processing.

Finally, although no study has examined error-preceding brain activity and anxiety specifically, extant neuroimaging research strongly suggests such a link might exist. Specifically, an increasing number of studies show DMN abnormalities in anxiety (Andreescu, Sheu, Tudorascu, Walker, & Aizenstein, 2013; Anteraper et al., 2014; Bijsterbosch, Smith, Forster, John, & Bishop, 2014; Fales et al., 2008; Gentili et al., 2007; Liu et al., 2014; Zhao et al., 2007; see Sylvester et al., 2012 for a review). Although most of these studies examined how anxiety relates to DMN activation during resting states, Fales and colleagues (2008) found that trait anxiety was associated with a sustained (task-wide) *deactivation* of DMN regions during a cognitive control task. Fales and colleagues suggested that sustained DMN deactivation reflected

greater compensatory effort among individuals with anxiety to either enhance performance or to suppress distracting anxiety-related thoughts when task attention began to decline. That is, the suppression of DMN activity was thought to be compensatory. Such an interpretation is entirely consistent with our model (Moser et al., 2013, 2014). Moreover, the regions identified in the Fales et al study – including posterior cingulate cortex, precuneus, perigenual ACC, and frontopolar cortex - overlap with those implicated in pre-error brain activity in other studies (e.g., Li et al., 2007). The overlap of regions between studies may suggest that individuals with trait anxiety show sustained DMN deactivation preceding errors. In this study, we therefore evaluated the relationship between worry, the EPP, and the ERN. We first predicted that worry would relate to a reduced EPP, consistent with Fales et al's (2008) findings of suppressed DMN activity in trait anxiety. Second, we assessed whether the EPP might explain (mediate) the relationship between worry and the ERN. If the EPP does explain the worry-ERN relationship, such a finding would provide additional support for the prediction of our compensatory error-monitoring hypothesis that the enlarged ERN associated with worry is the output of a compensatory effortful process that begins before error commission (Moser et al., 2013).

Method

Below, we report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study (see Simmons, Nelson, & Simonsohn, 2012).

Participants

Participants were 70 female undergraduates (M age = 19.60, SD = 3.22) who completed the study for partial course credit. Data from a subsample of these participants have been reported previously (Moran et al., 2015; Moran, Taylor, & Moser, 2012; Moser et al., 2012; Moser, Schroder, Heeter, Moran, & Lee, 2011; Schroder, Moran, Altmann, & Moser, 2012; Schroder, Moran, Infantolino, & Moser, 2013). None of these previous studies examined the EPP, and thus all of the analyses involving the EPP reported here are novel. We chose to analyze data from female participants because we have found both in an empirical study (Moran et al., 2012) and a meta-analysis (Moser, Moran, Kneip, Schroder, & Larson, 2016) that the anxiety-error processing relationship is only present among females. Of the 205 participants initially enrolled in the study¹, 28 were excluded due to failure to follow stimulus-response mapping instructions during the flanker task (see below for task details). A further 28 were excluded due to excessive artifacts in the raw EEG data or failure to commit at least six errors for error-related brain activity analyses (Olvet & Hajcak, 2009). Of the remaining 149 participants, 79 were female, and were chosen for the present analyses. Nine of the 79 female participants reported on in the Moran et al (2012) paper had unusable EPP data (fewer than six pre-error ERP trials), leaving a final sample of 70². Written informed consent was provided prior to experimental procedures and the University's Institutional Review Board approved the study. ERP data were available for all 70 participants; behavioral data were lost for two participants due to a computer error.

Materials and Procedure

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¹ The original aim of this project was to determine relations between psychopathology and error-related brain activity and potential sex differences. As such, we wanted to ensure adequate variability in personality traits and psychological distress for both males and females. We therefore recruited participants across four consecutive semesters, arriving at $N=205$.

² We reasoned that a sample size of 70 would be sufficient to explore relations between worry and the EPP, given the moderate effect size of the anxiety-ERN relation ($r = -.35$; Moser et al., 2013) and that the median sample size of similar anxiety-error monitoring studies was only $n = 40$ (Moser et al., 2013).

Participants completed a letter version of the Eriksen Flankers task (Eriksen & Eriksen, 1974). Participants were instructed to identify the center letter (target) of a string of five letters using the left or right mouse button. Each string included four distracter letters surrounding the target and were either congruent (e.g. M M M M M or N N N N N) or incongruent (e.g. M M N M M or N N M N N). Congruent and incongruent trials varied randomly with a frequency of 50% each. Flanking letters were displayed 35 ms prior to presentation of the target stimulus, which remained on the screen for 100 ms (total trial time of 135 ms). A fixation cross (+) was presented during a variable inter-trial interval (1200-1700 ms). Speed and accuracy were equally emphasized. No performance feedback was given during the task.

The experiment consisted of 480 trials grouped into 6 blocks of 80 trials each. To elicit a sufficient number of errors for ERP analysis (Olivet & Hajcak, 2009), letters making up the trial stimuli differed across blocks: Block 1: “M” and “N”, Block 2: “F” and “E”, Block 3: “O” and “Q”, Block 4: “T” and “I”, Block 5: “V” and “U”, and Block 6: “P” and “R”. Furthermore, mouse button-letter response mappings were reversed midway through each block (e.g. left-click for M through 40 trials of block 1, then right-click for M for the last 40 trials of block 1). To control the presentation and timing of stimuli along with determination of response accuracy and measurement of reaction times, a Pentium R Dual Core computer was used with Presentation software (Neurobehavioral Systems, Inc.). Characters were displayed in standard white font on a black background and subtended 1.3° of visual angle vertically and 9.2° horizontally.

Following completion of the Flankers task, participants completed a packet of questionnaires including the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) and

the Anxious Arousal subscale of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991)³.

Psychophysiological Data Recording and Reduction

Continuous electroencephalographic (EEG) activity was recorded using the ActiveTwo Biosemi system (BioSemi, The Netherlands) from 64 Ag-AgCl electrodes fitted in a stretch-lycra cap. Electrooculogram (EOG) activity generated by blinks and eye-movements was recorded at FP1 and additional electrodes placed on the left and right outer canthi as well as one placed below the left eye, all approximately 1 cm from the pupil. Two sensors were also placed on the left and right mastoids. During data acquisition, the Common Mode Sense active electrode and the Driven Right Leg passive electrode formed the ground. All signals were digitized at 512 Hz.

Offline Analyses were performed with 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 rolloff). Ocular artifacts were then corrected using a commonly used regression method (Gratton, Coles, & Donchin, 1988). Response-locked data were segmented beginning 200 ms prior to the response and continuing for 800 ms post-response. Physiological artifacts were rejected using a computer-based algorithm using the following criteria: a voltage step exceeding 50 μ V between contiguous sampling points, a voltage difference of more than 200 μ V within a trial, or a maximum voltage difference less than 0.5 μ V within a trial.

A baseline equal to the average activity in the 200ms window prior to response onset was subtracted from each data point subsequent to the response to quantify ERPs. Findings from a

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³ Other questionnaires included in the packet, but not analyzed here, included measures of perfectionism, depression, externalizing behaviors, and personality traits.

recent meta-analysis(Moser et al., 2013) indicate that worry is only significantly correlated with the ERN, and not the correct-response negativity (CRN). For this reason our subsequent analyses focused on the ERN. The ERN was quantified across five fronto-central recording sites (Fz, FC1, FCz, FC2, Cz) as the average voltage during the 0-100ms post-response time window on error trials. The EPP-1 was quantified as the average voltage of the same electrode sites in the 50-150ms post response time window averaged across correct trials immediately preceding error trials. An additional EPP (EPP-2) was quantified on the second correct trial before an error in the same time window.

ERP and behavioral data were statistically evaluated using SPSS General Linear Model software (Version 20.0). Inspection of distributions revealed one univariate outlier (which we defined as deviating from the mean by 3 or more standard deviations) for the EPP variable. We therefore analyzed the data with and without the outlier case included. Importantly, removing the outlier altered none of the primary findings; thus, analyses reported below included this case.

Results

Behavioral data

We first aimed to establish that worry was not associated with abnormal behavioral performance (accuracy or response times) in this simple flanker task (seeEysenck et al., 2007). Consistent with attentional control theory and the compensatory error-monitoring hypothesis, although accuracy was high across all participants (M percent correct=93.20, SD =5.69) it was not associated with PSWQ scores ($r(66) = -.04$, $p = .73$). Reaction times (RTs) were shorter on error trials compared to correct trials ($t(67) = 16.89$, $p < .001$, $d = 1.61$), but RTs were not associated with PSWQ ($r_s < -.04$, $p_s > .76$). We next examined RTs surrounding errors to

confirm the natural disengagement (increasingly fast RTs) that is often observed leading up to errors and the typical post-error slowing observed on trials following errors (Rabbitt, 1968). Correct responses that preceded errors had shorter RTs than correct responses that preceded corrects (1-back: $t(67) = 9.64, p < .001, d = 0.70$); 2-back: $t(67) = 5.98, p < .001, d = 0.49$). Moreover, correct RTs on the trials following errors were longer than correct RTs following correct responses ($t(67) = 12.75, p < .001, d = 1.37$). Accuracy did not differ significantly between post-error and post-correct trials ($t(67) = 0.36, p = .72, d = 0.04$). Thus, participants slowed down after their errors, but were no more accurate following errors than following correct responses. There were no associations between PSWQ scores and error-surrounding behavior ($ps > .29$) consistent with the predictions of attentional control theory and the compensatory error-monitoring hypothesis.

Event-related brain potentials (ERPs)

Error-monitoring ERPs

Waveforms are depicted in Figure 1. In the ERP analyses, we first established baseline error-monitoring effects. A robust ERN in the 0-100ms post-response time window was larger on error trials compared to correct trials ($t(69) = 7.23, p < .001, d = 0.78$). In the 50-150ms post-response window on correct responses immediately preceding errors, the positivity on pre-error trials (EPP-1) was larger than that on pre-correct trials ($t(69) = 3.18, p = .002, d = 0.25$). Consistent with previous research (Hajcak et al., 2005), amplitudes two trials before an error (EPP-2) were not significantly different from those two trials before a correct ($t(69) = 0.48, p = .63, d = .03$). Bivariate correlations indicated that all three ERPs were associated with one another such that larger EPP-2 related to larger EPP-1, and both larger EPP-2 and EPP-1 related to a

smaller (less negative) ERN:EPP-1 and EPP-2: $r = .60, p < .001$; EPP-1 and ERN: $r = .45, p < .001$; EPP-2 and ERN: $r = .52, p < .001$).

Relationship between worry and error monitoring

Duplicating results previously reported on this sample (Moran et al., 2012), PSWQ scores were associated with larger (more negative) ERN amplitudes ($r(68) = -.31, p < .01$)⁴. When PSWQ scores were entered into the Accuracy (Pre-Error vs. Pre-Correct) ANOVA for the EPP, the Accuracy x PSWQ interaction was significant ($F(1, 68) = 5.71, p = .02, \eta_p^2 = .08$). Consistent with our prediction, higher PSWQ scores were associated with reduced EPP-1 amplitude ($r(68) = -.38, p = .001$; see Figure 1). PSWQ was also related to the correct-preceding positivity (CPP; $r(68) = -.29, p = .01$), but, consistent with the interaction effect, PSWQ was related to the EPP difference ($r(68) = -.28, p = .02$). The correlation between PSWQ and EPP-2 was not statistically significant ($r(68) = -.19, p = .13$).

Our primary concern in this paper was to test the mediating role of the EPP in the worry-ERN relationship. Because of this, our analyses focus on the relation between worry and EPP among females based on our prior work showing that the worry-ERN relationship is significantly stronger among females – and not reliable in males (Moran et al., 2012; Moser et al., 2016). However, one reviewer inquired about the anxiety, ERN, and EPP relations in males in this study. Thus, we analyzed the males from the Moran et al (2012) sample with usable EPP data ($n=65$). As reported previously, the worry-ERN relationship was non-significant. Moreover, the relation between worry and EPP was non-significant and in the opposite direction ($r = .19, p = .14$). Importantly, the correlation between PSWQ and EPP was significantly different from that

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⁴ Because the ERN is a negative-going component, negative correlations indicate a larger ERN, whereas positive correlations indicate a smaller ERN.

in females ($r = -.38$), confirmed by Fisher's r - t - z transformation ($Z = 3.36$, $p = .0001$, two-tailed). Thus, like the relation between worry and the ERN, the relation between worry and the EPP appears to be much stronger in women.

Given the significant relationship between the ERN and EPP-1 in females described above, the conditions for mediation between PSWQ, EPP-1, and the ERN were established (scatter plots depicting the relationships are presented in Figure 2⁵). Formal mediation analyses (Preacher & Hayes, 2008) confirmed that EPP-1 mediated the PSWQ-ERN relationship (95% CIs: $-.77$, $-.12$; see Figure 3) such that after controlling for the EPP, PSWQ was no longer significantly associated with the ERN (partial $r(67) = -.17$, $p = .16$). Although controlling for the EPP did not *entirely* eliminate the relation between PSWQ and the ERN, the results are consistent with our prediction. In contrast, the PSWQ-EPP relationship remained significant even after controlling for the ERN (partial $r(67) = -.28$, $p = .02$). These findings indicate that the EPP accounts for considerable variance in PSWQ scores, even after taking the ERN into consideration.

Brain-behavior relationships

Brain-behavior relationships at different levels of worry were probed to further understand the functional interpretations of the EPP (cf. Schroder & Moser, 2014). This analysis specifically asked: does the EPP relate to behavior differentially at different levels of worry? Mean-centered PSWQ, EPP-1, and their interaction (PSWQ $\times$ EPP-1) were entered into a multiple linear regression predicting behavioral accuracy, which was arcsine transformed⁶. The interaction term was significant ($b = -.001$, $B = -.29$, $t = -2.37$, $p = .027$), and simple slopes

⁵ The outlier identified earlier (EPP z -score of above $|3|$) is evident in these plots. We reiterate that removing this outlier from the dataset had no effect on the results, and so the case was included in the analyses.

⁶ Results were unchanged when analyses were run without transforming the accuracy variable.

analyses were used to probe the significant interaction (Aiken & West, 1991). This analysis revealed that at one standard deviation below the mean on PSWQ (i.e., individuals with low worry), the EPP did not relate to accuracy ($b = .005$, $SE = .007$, $B = .18$, $t = 0.74$, $p = .46$). However, at one standard deviation above the mean on PSWQ, the EPP *did* relate to accuracy ($b = -.02$, $SE = .005$, $B = -.57$, $t = -3.42$, $p = .001$). These results indicate that at high, but not low levels of worry, a smaller EPP-1 related to greater behavioral accuracy.

Relationships to physiologic anxiety

As a final analysis, we examined how the EPP related to physiologic anxiety (anxious arousal, assessed with the MASQ-AA). The PSWQ and MASQ-AA showed a modest, albeit significant, correlation with each other ($r(68) = .26$, $p = .030$). MASQ-AA was also significantly related to smaller EPP ($r(68) = -.24$, $p = .045$). To evaluate whether MASQ-AA scores predicted unique variance in the EPP (over and above the PSWQ), we entered PSWQ and MASQ-AA into regression analyses to predict the EPP. Results indicated that PSWQ predicted significant variance in the EPP ($b = -.14$, $SE = 0.05$, $\beta = -.34$ (95% CI: $-.23$, $-.04$), $t = -2.94$, $p = .005$), but the MASQ-AA did not ($b = -.12$, $SE = .09$, $\beta = -.15$ (95% CI: $-.29$, $.06$), $t = -1.32$, $p = .192$). Thus, MASQ-AA scores did not statistically significantly predict unique variance in EPP amplitudes once controlling for PSWQ.

Discussion

Accumulating evidence indicates that anxiety is associated with increased cognitive-control brain activity after errors (Hajcak, 2012; Moser et al., 2013; Weinberg et al., 2015). The findings reported here significantly extend this research by demonstrating worry is also associated with abnormal brain activity leading up to errors, and that this pre-error activity

explains the relationship between worry and the enlarged ERN following error commission. In this way, our findings indicate that exaggerated ERN may reflect the output of a compensatory process beginning before the error among individuals with worry. Indeed, we found evidence that reduced EPP may be compensatory among worriers, as it was related to higher behavioral accuracy among high-, but not low-worriers.

The EPP's mediating role in the anxiety-ERN relationship is noteworthy for several reasons. First, it provides a potential explanation of exaggerated ERN among anxious individuals, which we expand on here from our model (Moser et al., 2013). Specifically, we suggest that reduced task-focused attention preceding errors prompts worriers to engage in compensatory target processing, which is first reflected in reduced EPP (less disengagement) and then enhanced ERN (increased post-error target processing; Yeung et al., 2004). In this way, effortful resources are mobilized just prior to, not after, errors. This hypothetical model is depicted in Figure 4. As can be seen in this figure, we propose that worriers have a lower threshold for engaging attentional effort and cross this threshold as task attention declines just before an error occurs (between one and two trials before an error). Recall that worry was related to reduced EPP-2 amplitude, albeit non-significantly ($r = -.19$). Enhanced attentional effort (i.e., increased target processing) in worriers immediately before the error is sufficient to increase the ERN amplitude but insufficient to prevent the error from occurring. The suppressed EPP amplitude in worriers may reflect an effortful response aimed at improving task-related attention, presumably to reduce the probability of an error. Although this hypothesis requires further testing and refinement, it is compatible with widely-accepted models of cognitive control and basic assumptions of error monitoring indicating 1) enhanced target processing potentiates the ERN (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Yeung et al., 2004; Yeung & Cohen,

2006; Yeung, Ralph, & Nieuwenhuis, 2007) and 2) anxious individuals are biased toward engaging in just-in-time (reactive) cognitive control (Braver, Gray, & Burgess, 2007; Gray & Braver, 2002). This hypothesis is also supported by the brain-behavior analyses revealing a tight coupling of brain activity and accuracy within the high-worry group. Among individuals with higher self-reported worry, reduced EPP was directly related to more accurate performance.

Second, it also suggests that the clinical utility of error-related ERPs is not confined to brain activity elicited following error commission. In fact, error-preceding brain activity may be more important in terms of understanding anxiety's impact on error processing and cognitive control more broadly. This is also especially encouraging as there are dozens of ERN datasets that can be reanalyzed to assess how the EPP relates to anxiety-related psychopathology. This is promising for neuroscience-based approaches to the study of psychopathology such as the National Institute of Mental Health's Research Domain Criteria (RDoC; Insel et al., 2010), which currently lists the ERN in three different constructs in its RDoC Matrix. The current findings suggest that different error-related ERPs may explain unique variance in manifestations of anxiety and related problems.

These findings are further in line with the growing movement to view cognitive control as a process of interacting brain networks, as opposed to the output of a single cluster of brain regions. For clinical psychology and psychiatry, this shift has resulted in a more nuanced view of how psychopathology impacts information processing and performance. This recognition has led more recent fMRI studies to consider both pre- and post-error neural activity in other clinical populations such as ADHD (Spinelli et al., 2011) and cocaine dependence (Bednarski, Zhang, Hong, Sinha, Rounsaville, & Li., 2011). The DMN is traditionally distinguished from "cognitive control" brain networks, as these are often anticorrelated (Greicius et al., 2003). Yet, a more

comprehensive model is evolving that considers DMN crucial to the execution of successful task performance (e.g., Braem et al., 2013; Cocchi, Zalesky, Fornito, & Mattingley, 2013; Leech, Braga, & Sharp, 2012; Leech, Kamourieh, Beckmann, & Sharp, 2011; Leech & Sharp, 2013; Mantini & Vanduffel, 2011). The current findings underscore this possibility to the extent that the EPP does, in fact, reflect pre-error DMN activity (Li et al., 2007).

The findings presented here may have implications for neurobehavioral treatment regimens aimed at reducing the distracting effects of worry on cognitive control and error monitoring. Some have previously suggested that exposure to errors may improve cognitive control functioning among individuals with anxiety (Sylvester et al., 2012). As our findings indicate that performance monitoring is modulated even before errors occur, alternative programs may work more effectively if they alter cognitive control processing more generally, and not just reactivity to errors. For example, recent efforts using proactive control training, which teaches participants to focus on the task at hand and keep task-relevant goals in working memory, may be particularly effective for individuals with anxiety/worry. Similar training programs are effective in reducing symptoms of depression and schizophrenia (Edwards, Barch, & Braver, 2010). Strategies to reduce the relationship between worry and error monitoring may also be effective in enhancing error-preceding brain activity among worried individuals. Expressive writing – or writing down emotional thoughts and feelings – just prior to engaging in a decision-making task has been shown to effectively eliminate the pernicious effects of anxiety on cognition (Ramirez & Beilock, 2011). More generally, our contention here is that strategies that reduce ERN and increase EPP amplitudes among worried individuals would likely be most effective in reducing the detrimental effects of anxiety on information processing. However, to the extent that these brain signals index compensatory processes aimed at maintaining

performance, it is possible that reducing ERN and increasing EPP among worriers may result in poorer task performance. Careful consideration of both the ERP and behavioral data will help evaluate these possibilities.

Our findings also call for a shift in focus from error-elicited activity to pre-error activity in the relationship between cognitive control and anxiety. The current work therefore suggests that more sophisticated cognitive control dynamics – perhaps involving multiple brain networks – relate to pathological worry. As the EPP has been hypothesized to reflect DMN activity (Li et al., 2007), reduced EPP among worriers implicates suppressed DMN activity leading up to errors. Moreover, the posterior cingulate cortex (PCC) may be one particularly promising region of interest in future studies examining cognitive control in anxiety, as recent work highlights its role in initial error detection (Agam et al., 2011; Agam et al., 2013), the detection of change to signal for shifts in task engagement (Pearson, Heilbronner, Barack, Hayden, & Platt, 2011), as well as its significance to various forms of psychopathology (Leech & Sharp, 2013).

This and past studies of cognitive control dynamics in anxiety have revealed one specific hidden cost of worry: inefficient performance. That is, anxious individuals expend added effort to offset the distracting effects of unwanted and intrusive worrisome thoughts. Compensatory effort and monitoring among anxious individuals, even in relatively simple tasks like the one described here, likely contributes to the fatigue and drained feeling that these individuals often report. Designing treatment programs aimed at reducing worry's burden on the brain prior to and after errors may be one way to help reduce the hidden costs of the world's most common mental health problem (Baxter et al., 2014).

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FIGURE CAPTIONS

Figure 1. Error-preceding positivity (EPP) and error-related negativity (ERN) among high and low scorers on the PSWQ. Trial Error-1 is shown on the left (only correct trials) and the error trial is shown on the right.

Figure 2. Scatter plots of correlations between ERN, EPP, and PSWQ.

Figure 3. Mediation model showing that EPP mediates relationship between PSWQ and ERN

Figure 4. Hypothetical attention dynamics in worriers.

Figure 1. Grand average waveforms of EPP and ERN

Figure 2. Scatter plots depicting relations between PSWQ, ERN, and EPP.

Note. $**p < .01$

Figure 3.

Note: PSWQ: Penn State Worry Questionnaire; EPP: error-preceding positivity; ERN: error-related negativity. $**p < .01$

Figure 4. Hypothetical attention dynamics in worriers. Attention gradually declines prior to an error trial across all participants, but the slope at which this occurs is reduced among those with high worry. This slope decline begins just prior to the trial before an error, after which task-focused attention (or target letter processing) is increased relative to those with low levels of worry, but not enough to prevent the error from occurring (attention still falls below the error response threshold). The higher degree of target processing just prior to the error response causes enhanced conflict between activation of the error and correct response, resulting in increased ERN immediately following the error.