

Sending mixed signals: worry is associated with enhanced initial error processing but reduced call for subsequent cognitive control

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Abstract

Worry is reliably associated with overactive action-monitoring processes as measured by the error-related negativity (ERN). However, worry is not associated with error-related behavioral adjustments which are typically used to infer increased cognitive control following errors. We hypothesized that this disconnect between overactive action monitoring and unimproved post-error adjustments in worriers is the result of reduced functional integration between medial and lateral prefrontal regions during generation of the ERN, understood to have an important role in mediating controlled processing. To test this, we examined ERN amplitude and interchannel phase synchrony extracted from scalp-recorded electroencephalographic data during error processing in 77 undergraduates who performed a Flankers task. Correlational and path analytic results demonstrated that worry was related to both an enlarged ERN and reduced phase synchrony. Although not directly related to post-error behavioral adjustments, results also revealed that worry was indirectly related to poor post-error adjustments through its association with reduced phase synchrony. Therefore, worry seems to affect multiple components of the action-monitoring system. It is related not just with the initial response to the error, but also with the transmission of information between networks involved in cognitive control processes.

Key words: anxiety; cognitive control; ERPs; ERN; theta synchrony

Introduction

Anxiety is a common experience characterized by perseverative worry, psychological arousal and pervasive cognitive impairments (Barlow, 2002; Eysenck *et al.*, 2007). Research into the neural underpinnings of anxiety have revealed a robust association between anxious symptomology and exaggerated action monitoring (for a review, see Moser *et al.*, 2013)—i.e. the monitoring of ongoing behaviors in service of optimizing performance (Shenhav *et al.*, 2013). In particular, anxiety has been associated with an enlarged error-related negativity (ERN; Moser *et al.*,

2013), an event-related potential (ERP) generated in the anterior cingulate and supplementary motor regions of the medial prefrontal cortex (ACC/mPFC) within 100 ms of an erroneous response (Gehring *et al.*, 2012). However, there appears to be no observable downstream effects of this exaggerated action monitoring. That is, while anxious individuals show an enlarged ERN, this does not appear to be translated into greater cognitive control (i.e. post-error behavioral adjustments). This article concerns this apparent disconnect between the initial response to an error and the failure to adaptively improve behavior in anxious individuals.

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The action-monitoring system

The study of action monitoring is rooted in Rabbitt's classic findings regarding the recruitment of cognitive control following an error (Rabbitt, 1966; Rabbitt and Vyas, 1981; also see Danielmeier and Ullsperger, 2011). Following the commission of an error, participants often, and automatically, take action to correct the error and engage in a speed/accuracy tradeoff on subsequent trials in order to minimize the likelihood of subsequent errors (Rabbitt, 1966; Danielmeier and Ullsperger, 2011). Theorizing on cognitive control posits that these control mechanisms are mediated by a dynamic loop of prefrontal cortex regions (Botvinick et al., 2001; Ridderinkhof et al., 2004). Specifically, the ACC/mPFC is hypothesized to monitor for circumstances that require cognitive control—such as conflicts or errors—and to specify the requisite adjustments in order to optimize behavior (Botvinick et al., 2001; Ridderinkhof et al., 2004; Gehring et al., 2012; Shenhav et al., 2013; Cavanagh and Frank, 2014). In this way, the ACC/mPFC (putatively the ERN) acts as an 'alarm' which indicates the need for increased cognitive control processes (Shenhav et al., 2013; Cavanagh and Frank, 2014). In response to the alarm signal, lateral prefrontal cortex regions (IPFC) are recruited to mediate control processes specified by the ACC/mPFC such as post-error slowing (Botvinick et al., 2001; Eichele et al., 2008; Shenhav et al., 2013). Along these lines, both error-related ACC/mPFC and IPFC activity have been found to predict post-error behavior (Garavan et al., 2002; Kerns et al., 2004; Kerns, 2006; Hester et al., 2007; Gehring et al., 2012; Shenhav et al., 2013 for reviews).

Studies have begun to more precisely map the mechanisms by which the initial alarm signal is transmitted to control regions. Based on findings suggesting that phase synchronization between brain regions is one mechanism by which different brain regions within a functional network interact (Engel and Singer, 2001; Varela et al., 2001; Fries, 2005), Cavanagh et al. (2009) examined scalp-recorded medial frontal theta activity (4–8 Hz; the frequency band thought to underlie the generation of the ERN; Luu et al., 2004; Trujillo and Allen, 2007) following the commission of errors. In this study, errors were associated with increased phase synchronization between electrodes over the ACC/mPFC and those over IPFC regions. We recently demonstrated how increases in these observed bivariate phase synchrony pairings during errors can also be understood in terms of increased small-world network organization involving these regions. Specifically, we utilized a node-based measure of small-world network organization to index contributions from each site to the overall network organization across the sites (Bolanos et al., 2013). Critically, sites consistent with the mPFC and IPFC were the central nodes in the small-world organization. Additionally, these small-world node-based measures fully accounted for the observed bivariate phase-synchrony increases during errors over the ACC/mPFC and IPFC regions, verifying that these bivariate and multivariate network measures indexed the same error processing activity and implicated the same regions. Together, these findings suggest that medial-lateral frontal functional integration is a strong candidate mechanism by which cognitive control regions become engaged during error processing.

The ERN and anxiety

A large corpus of data has accumulated demonstrating that anxiety is associated with an enlarged ERN (for a meta-analysis, see Moser et al., 2013). In particular, the ERN is associated with clinical obsessive-compulsive and generalized anxiety

symptoms (Gehring et al., 2000; Weinberg et al., 2010, 2012) as well as subclinical obsessive-compulsive symptoms (Hajcak and Simons, 2002; Kaczurkin, 2013) and worry (Moser et al., 2012). Furthermore, we have recently shown that the ERN is specifically associated with the worry/apprehension dimension of anxiety and much less strongly with arousal and general anxiety (Moser et al., 2012, 2013).

We have proposed that the association between anxiety and the ERN results from a compensatory process aimed at normalizing performance in anxious individuals (Moser et al., 2013, 2014; but see Proudfit et al., 2013). Specifically, our Compensatory Error Monitoring Hypothesis (CEMH) is predicated on the notion that anxious individuals are characterized by increased reliance on 'reactive control' (Gray et al., 2005; Fales et al., 2008; Krug and Carter, 2010, 2012). According to Braver's (2012) dual mechanisms of control model, reactive control is a form of cognitive control that involves recruiting attention on an 'as-needed' basis following conflicts or errors (Braver et al., 2007). This stands in contrast to 'proactive control', which Braver proposed is more effortful and involves maintaining task goals in a sustained manner. Anxious individuals' reliance on reactive control enhances target processing/cognitive control processes during and following the commission of an error which is known to result in an enlarged ERN (Yeung et al., 2004; Sarter et al., 2006; Yeung et al., 2007).

However, there appears to be discontinuity between studies of action monitoring in unselected populations and those conducted with anxious individuals. As noted earlier, ACC/mPFC-related activity (putatively the ERN) is assumed to function as the initial 'alarm' or monitor indicating that something has gone awry; IPFC regions are then signaled to implement cognitive control processes aimed at optimizing performance. Importantly, the successful recruitment of mPFC/IPFC-mediated control processes is inferred from post-error behavior such as slowed RTs and increased accuracy (Garavan et al., 2002; Kerns et al., 2004; Kerns, 2006; Hester et al., 2007; Danielmeier and Ullsperger, 2011; Schroder and Moser, 2014). The recruitment of cognitive control in anxiety, on the other hand, has generally been inferred from an enhanced ERN/ACC activity as anxiety (and worry in particular) is not reliably associated with post-error behavior (Hajcak and Simons, 2002; Hajcak et al., 2003; Hajcak et al., 2008; Hanna et al., 2012; Meyer et al., 2013; Moran et al., 2012; Weinberg et al., 2010, 2012; Aarts and Pourtois, 2010, 2012; Carrasco et al., 2013). This leads to the question 'if anxiety enhances error-related cognitive control processes (i.e. an enlarged ERN), why is improved post-error behavior not observed?'

One possibility is that the signal generated by the monitoring system (i.e. ACC/mPFC) is not being transmitted to subsequent cognitive control areas (i.e. IPFC). That is, while the ACC/mPFC may be overactive in anxious individuals, it is possible that control regions are not being effectively recruited following the commission of an error. In order to test this possibility, this study capitalized on Cavanagh et al. (2009) finding that the transmission of the alarm signal is likely instantiated by EEG-derived phase synchronization of the theta band between the ACC/mPFC and IPFC following an error. Using an existing dataset in which participants completed a Flanker task, we examined standard time-domain ERPs (i.e. the ERN) as well as error-related phase synchrony of the theta band between ACC/mPFC and IPFC. If it is the case that anxiety is associated with attenuated transmission of the error signal, then worry scores should predict decreased synchronization between electrodes over ACC/mPFC and IPFC. We also used path analyses to build a

model that could capture the nature of the interrelations between worry, ERN, synchronization between ACC/mPFC and LPFC regions and post-error behavior. In addition to predicting that worry would be simultaneously related to an enlarged ERN and decreased synchrony, we predicted that worry would be associated with poorer post-error behavior via its relationship with decreased synchrony.

Method

Participants

Participants included 79 undergraduate students who were recruited through Michigan State University's research participation pool as part of an ongoing study of the intersection between affect and cognitive control (see Moser et al., 2012, 2013). The data for two participants were lost due to technical failures resulting in a total sample size of 77. Participants ranged in age between 18 and 40 ($M = 19.82$, $SD = 2.86$); the sample included only women per Moran et al. (2012). No participants discontinued their involvement once the experiment began.

Materials/procedure

Participants completed a letter version of the Eriksen flanker task (Eriksen and Eriksen, 1974). On each trial, participants were presented with a five-letter string which was either congruent (i.e. all letters were associated with the same response; e.g. M M M M M) or incongruent (i.e. letters were associated with mutually exclusive responses; e.g. M M N M M) and were required to identify the center letter via a standard mouse. Characters were displayed in a standard white font on a black background and subtended $\sim 1.3^\circ$ of visual angle vertically and $\sim 9.2^\circ$ horizontally. All stimuli were presented on a Pentium R Dual Core computer, using Presentation software (Neurobehavioral Systems, Inc.).

Each trial began with the flanking stimuli (e.g. M M M M) which were present for 35 ms. The target stimuli was then embedded among the flankers (e.g. M M M M M) and remained present for 100 ms (135 ms total presentation time). The target/flanker stimuli were followed by a variable duration intertrial interval (varied randomly between 1200 and 1700 ms) during which a standard fixation cross was present (+). Participants were allowed up to 800 ms to respond beginning with the onset of the target.

The experimental session consisted of 480 trials which were grouped into 6 blocks of 80 trials. In order to increase the number of errors available for analysis, the letter strings were varied between blocks (e.g. M and N in block 1, E and F in block 2) and the stimulus-response mappings were reversed at the midpoint of each block (e.g. left-click for M through 40 trials of block 1, then right-click for M during the remaining 40 trials of block 1). Participants were not provided feedback during task performance.

After completion of the flanker task, participants filled out a packet of questionnaires including the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). This study focused on the worry dimension of anxiety as our previous work has shown that error-related brain activity is most closely coupled with worry (Moser et al., 2012, 2013).

Psychophysiological data recording and reduction

Continuous encephalographic (EEG) activity was recorded using the ActiveTwo Biosemi system (Biosemi, Amsterdam,

The Netherlands). Recordings were taken from 64 Ag-AgCl electrodes embedded in a stretch-lycra cap. Additionally, two electrodes were placed on the left and right mastoids. Electrooculogram (EOG) activity was recorded at FP1 and three additional electrodes placed inferior to the left pupil and on the left and right outer canthi (all ~ 1 cm from the pupil). The data were digitized at 24-bit resolution with a least significant bit value of 31.25 nV and a sampling rate of 512 Hz, using a low-pass fifth order sinc filter with a 3-dB cutoff point at 104 Hz to prevent aliasing. The Common Mode Sense (CMS) active electrode, located between POz and PO4 and Driven Right Leg passive electrode, located between POz and PO3 served as the online reference for all other electrodes.

Offline analyses were performed using BrainVision Analyzer 2 (BrainProducts, Gilching, Germany). Scalp electrode recordings were re-referenced to the mean of the mastoids and band-pass filtered with cutoffs of 0.1 and 30 Hz (12 dB/oct rolloff). Ocular artifacts were corrected using the method developed by Gratton et al. (1983). Response-locked data were segmented into individual epochs beginning 200 ms prior the response and continuing for 1000 ms. 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 μ V between contiguous sampling points, a voltage difference of 200 μ V within a trial, and a maximum voltage difference of less than 0.5 μ V within 100-ms intervals. Finally, response-locked averages were computed separately for error and correct trials.

Time-domain ERN measures

To quantify the ERN and CRN (i.e. the correct-response counterpart to the ERN), a baseline equal to the average activity in the -200 and -50 ms window was subtracted from each datapoint subsequent to the response. The ERN and CRN were then defined as the average activity occurring in the 0–100 ms post-response window at electrode site FCz (see Figure 1 for the topographical distribution of ERPs). Finally, the ERN is an inherently negative deflection which can make it difficult to interpret in correlational analysis (i.e. a negative correlation will indicate a positive association). In order to increase the interpretability of analyses, we multiplied the ERN and CRN by -1 . Thus, a numerically larger ERN will indicate a greater deflection. All figures and tables, however, will display the ERN/CRN in their original metric.

Interchannel phase synchrony (ICPS)

Using the same approach previously employed by our group (Bolanos et al., 2013), time-varying complex energy time-frequency distribution (TFD) of all the EEG signals at each channel were obtained using the reduced interference distribution (RID) Rihaczek distribution (Aviyente et al., 2011). The RID-Rihaczek distribution computes a complex TFD with uniform time-frequency resolution, avoiding the trade-off between time and frequency resolution inherent to wavelet analysis. To avoid spurious phase synchrony between scalp electrodes due to volume conduction (Srinivasan et al., 2007), all EEG epochs were first converted to current source density (CSD) using published methods (Tenke and Kayser, 2012). In these analyses, we computed the phase differences between pairs of electrodes across trials. Phase-synchrony was computed using a phase locking value (PLV; Lachaux et al., 1999) which represented the average difference in phase-synchrony between a pair of electrodes across epochs. The PLV is normalized so that values near 1

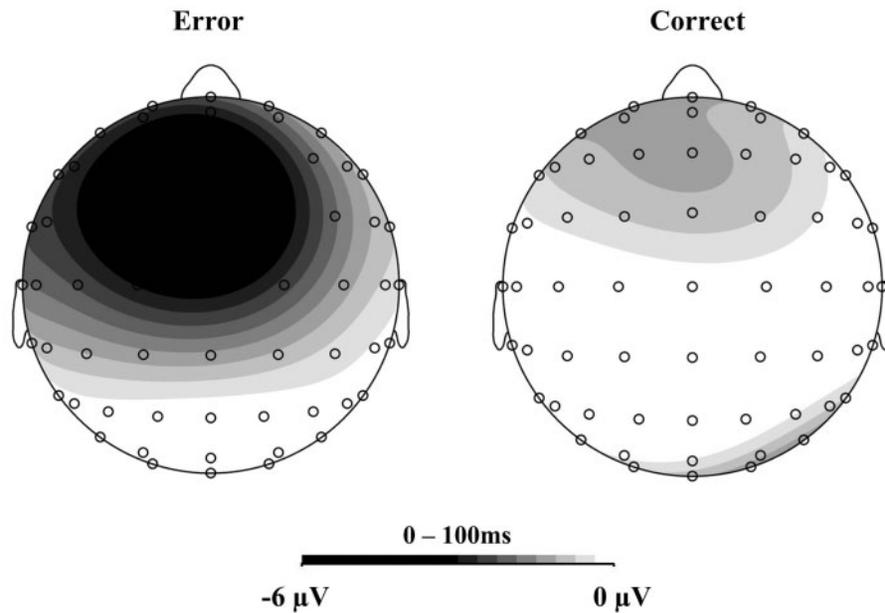


Fig. 1. Scalp maps depicting the topographical distribution of time-domain activity on error and correct trials.

indicate highly consistent phase difference between electrodes across trials and values near 0 indicate almost entirely unrelated phase between electrodes across trials. Thus, PLV was used to quantify the synchrony between EEG signals of electrode pairs independently for each time-frequency point on the TFD. In this article, we particularly focused on the average synchrony within the theta band in the ERN time window.

In order to be consistent with previous work (Cavanagh et al., 2009), this study will report ICPS for F5-FCz and F6-FCz pairs. The reader should note that, while increased ICPS recorded at these sites is consistent with increased coupling between the ACC/mPFC and lPFC, it is difficult to draw inferences regarding the underlying neuroanatomy from scalp-recordings alone.

Initial statistical analyses were conducted in SPSS v.22. Path modelling was conducted using LISREL v.9 software.

Results

Behavioral performance

Overall accuracy was high ($M = 92.23\%$, $SD = 5.38\%$). Participants were faster to respond on error trials ($M = 370.01$, $SD = 50.56$) than correct trials ($M = 446.69$, $SD = 41.61$; $F(1,76) = 368.81$, $P < 0.001$, $\eta_p^2 = 0.83$). Post-error response times (PERT; $M = 489.07$, $SD = 65.10$) were slower than RTs following correct trials (post-correct RT: $M = 442.67$, $SD = 42.87$; $F(1,76) = 80.03$, $P < 0.001$, $\eta_p^2 = 0.51$). Post-error accuracy (PEA) did not differ from post-correct accuracy ($F(1,76) = 0.11$, $P = 0.74$).

Neural differentiation between errors and correct trials

Time and time-frequency domain results are depicted in Figure 2. Replicating previous work, both time-domain ERPs (Gehring et al., 2012) and ICPS differed as a function of accuracy (Bolanos et al., 2013; Cavanagh et al., 2009). Specifically, a single factor (Accuracy) ANOVA demonstrated that time domain amplitudes (i.e. the ERN) were greater following errors relative to correct responses ($F(1,76) = 95.49$, $P < 0.001$, $\eta_p^2 = 0.56$). Secondly, a 2 (Accuracy: error vs correct) \times 2 (Electrode: F5-FCz vs F6-FCz)

repeated measures ANOVA confirmed that ICPS was also enhanced following errors ($F(1,76) = 11.02$, $P = 0.001$, $\eta_p^2 = 0.12$). There was no main ($F(1,76) = 0.26$, $P = 0.61$, $\eta_p^2 = 0.003$) or interactive ($F(1,76) = 0.11$, $P = 0.74$, $\eta_p^2 = 0.001$) effect of electrode on ICPS.

Correlational analyses

Given that we have recently shown that worry is most closely coupled with neural responses on error trials (Moser et al., 2013), correlational analyses focused on neural and behavioral measures recorded during and following error trials. Additionally, since ICPS recorded at F5-FCz and F6-FCz were highly correlated ($r = 0.64$, $P < 0.001$) and their correlations with worry did not differ ($z = 1.04$, $P = 0.29$), we averaged across error-related ICPS recorded at F5-FCz and F6-FCz. As shown in Table 1, correlations supported our predictions such that worry was associated with an enlarged ERN and reduced ICPS. Worry was not significantly related to either PERT or PEA.

To analyze the interrelationships between worry, error-related brain activity and post-error behavior, we conducted a path analysis—an extension of multiple regression that allows variables to act as both independent and dependent variables. This analysis also allowed us to test for the hypothesized indirect effects. Path analyses were conducted by applying the maximum likelihood estimator to the covariance matrix. In order to evaluate the path model, we report several indices of fit as is typically recommended (Kline, 1998). We report the chi-square statistic which reflects whether the difference between the observed and reproduced correlation matrices is statistically significant. As a significance test, the chi-square is highly influenced by sample size; thus, we also report the root mean square error of approximation (RMSEA), comparative fit index (CFI), Normed Fit Index (NFI), Tucker-Lewis index (TLI) and standardized root mean square residual (SRMR) which are less influenced by sample size. Hu and Bentler (1998, 1999) provided empirical cutoffs for these measures which are as follows: $RMSEA \leq 0.06$; $CFI \geq 0.95$; $NFI \geq 0.95$; $TLI \geq 0.95$; $SRMR \leq 0.08$.

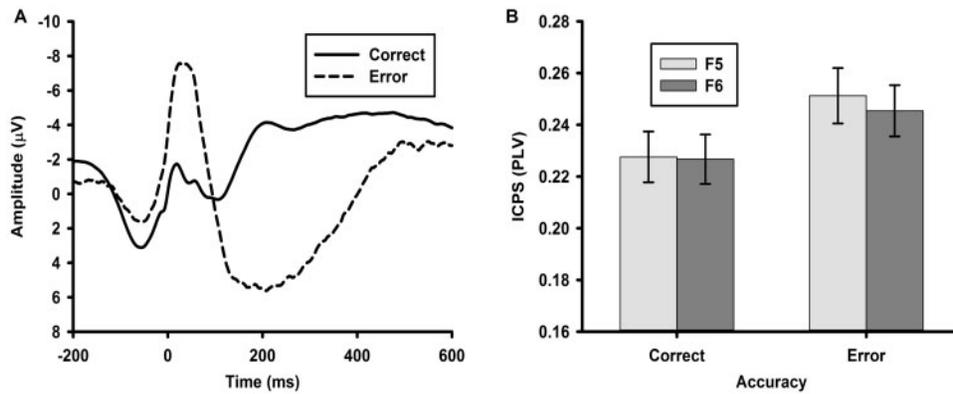


Fig. 2. (A) Waveforms depicting time-domain brain activity on error trials (ERN) and correct trials (CRN). (B) Interchannel phase synchrony as a function of accuracy and electrode. Error bars represent ± 1 SEM.

Table 1. Descriptive statistics and correlations for worry, error-related brain activity and post-error behavior

Variable	M	SD	Skew	Kurtosis	1	2	3	4	5	6
PSWQ	40.44	12.37	-0.08	-0.46	-					
ERN	-4.86	4.80	-0.93	1.12	0.26*	-				
CRN	-1.64	4.34	-0.34	-0.66	0.15	0.62**	-			
ICPS-E	0.25	0.08	0.42	-0.68	-0.23*	0.24*	0.31**	-		
ICPS-C	0.22	0.08	0.49	-0.17	-0.18	0.25*	0.29**	0.72**	-	
PEA	0.93	0.07	-1.03	0.68	-0.10	0.19	0.22	0.44**	0.37**	-
PERT	489.07	65.10	0.19	0.12	-0.05	0.15	0.25*	0.44**	0.55**	0.27*

Note: The means for the ERN and CRN are presented in their original metric. CRN, Correct-response negativity; ERN, error-related negativity; ICPS-E, interchannel phase synchrony on error trials; ICPS-C, interchannel phase synchrony on correct trials; PSWQ, Penn State Worry Questionnaire; PEA, post-error accuracy; PERT, post-error RT.

* $P < 0.05$, ** $P < 0.01$.

In our initial analysis, we assumed that worry influences error-related brain activity—both in the time and time-frequency domains—directly and that error-related brain activity influences post-error behavior. Additionally, we included a path from the ERN leading to ICPS. Note that this is not meant to suggest that the ERN itself causes theta synchronization following errors. Rather, it is meant to stand in for ACC/mPFC computations and their transmission to cognitive control regions. This model is depicted at the top of Figure 3. Overall, the model fit well ($\chi^2(3) = 0.94$, $P = 0.82$; RMSEA < 0.01 ; CFI > 0.99 ; NFI = 0.98; TLI = 1; SRMR = 0.02). However, an examination of the coefficients (Figure 3) suggested that the paths leading from the ERN to post-error behavior were small and not significant.

To further test our predictions, we aimed to test the fit of a model in which these insubstantial and nonsignificant contributions were removed (Kline, 1998). That is, we tested the model once paths leading from the ERN to post-error behavior were set to zero (see the bottom of Figure 3). The overall fit for this model was also very good across all metrics ($\chi^2(5) = 1.79$, $P = 0.82$; RMSEA < 0.01 ; CFI > 0.99 ; NFI = 0.96; TLI = 1; SRMR = 0.03). An examination of the path coefficients in Figure 3 indicated that results were consistent with expectations: worry was simultaneously related to an enlarged ERN and reduced ICPS; additionally, ICPS was related to greater post-error RT and accuracy. Results also supported our prediction that worry may be indirectly related to post-error behavior via ICPS. That is, a unit increase in worry affects ICPS, which, in turn, impacts

post-error behaviors. To formally test this prediction, we conducted Sobel's test for indirect effects (Sobel, 1982) which uses the indirect effect and pooled standard error to compute a t value. This indirect effect was significant (Sobel's $t = -2.37$, $P = 0.02$) for both PEA and PERT confirming our hypothesis that worry indirectly influences post-error behavior via ICPS. Specifically, worry is associated with faster PERT and lower PEA through its relationship with reduced ICPS. Interestingly, the indirect path between the ERN and post-error behavior through ICPS was significant (Sobel's $t = 2.43$, $P = 0.01$) indicating that the ERN predicts greater PERT and PEA through ICPS.

Discussion

This study examined the relationship between anxiety and neurobehavioral responses to errors. Our results (i) replicate previous findings demonstrating that inter-channel phase synchrony between electrodes over ACC/mPFC and electrodes over LPFC increase following the commission of an error (Bolanos et al., 2013; Cavanagh et al., 2009), (ii) demonstrate that worry predicts both an increased ERN and decreased theta band synchrony, and (iii) demonstrate that worry¹ is indirectly related to poorer post-error behavior via its association with decreased theta synchrony. These findings highlight novel mechanisms by which worry influences action monitoring. Anxiety appears to be associated with cognitive control abnormalities at several points between the commission and resolution of an error.

This study is the first to show that the transmission of error-related information between ACC/mPFC and LPFC is related to anxiety—specifically, that trait worry predicts attenuated

¹To demonstrate the specificity of this relationship, we also ran two additional path analyses in which the anxious arousal and loss of interest subscales of the MASQ were substituted for the PSWQ. We did not analyze the positive affect or anhedonic depression subscales as they were not related to any behavioral or physiological measures ($|r_s| < 0.15$). For the anxious arousal scale, fit was quite poor ($\chi^2(3) = 9.47$, $P = 0.01$; RMSEA = 0.17; NFI = 0.82; TLI = 0.48; CFI = 0.84; SRMR = 0.07); the paths leading from anxious arousal to the ERN and ICPS were non-significant ($t_s < 1.3$, $p_s > 0.1$). For the loss of interest scale, fit was, perhaps, marginal. The chi-square and SRMR showed acceptable fit ($\chi^2(3) = 6.45$, $P = 0.09$; SRMR = 0.06); however, the other metrics showed poor fit (RMSEA = 0.12; NFI = 0.88; TLI = 0.72; CFI = 0.91). Additionally, the path leading from loss of interest to ICPS was non-significant ($t = 0.12$).

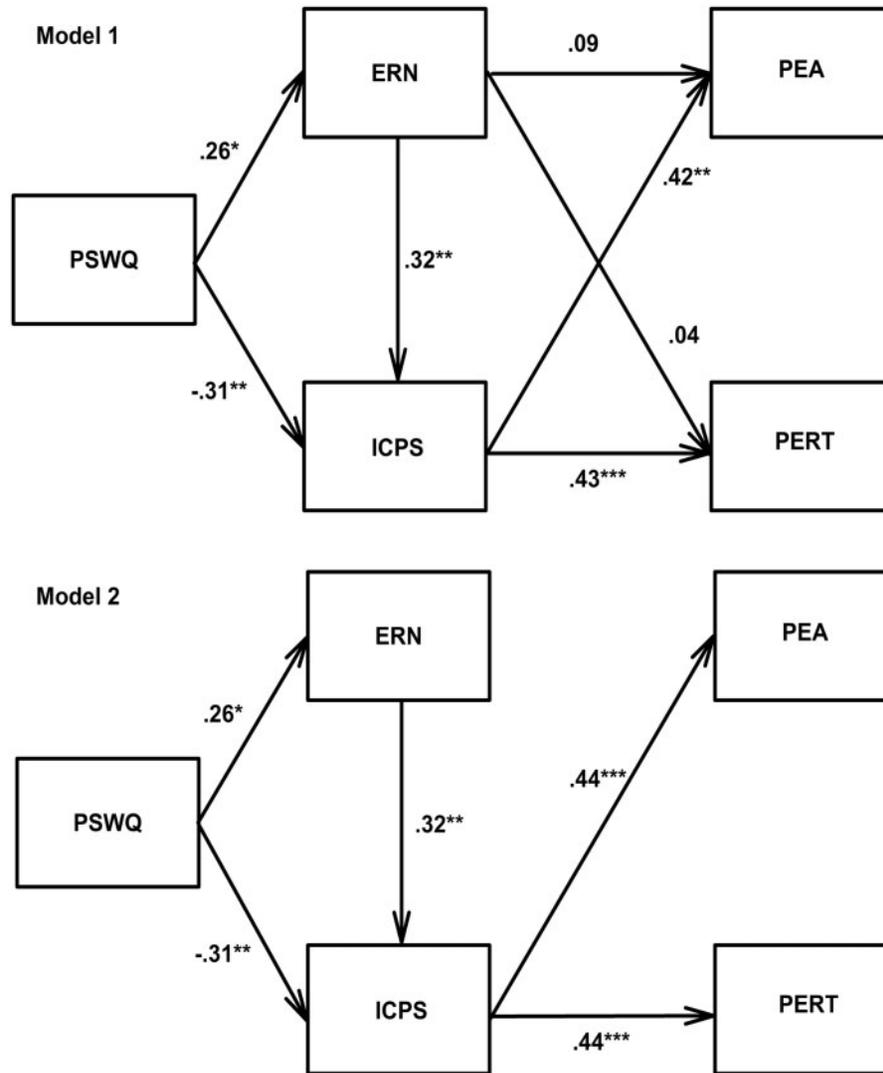


Fig. 3. The first (top) and second (bottom) path diagrams depicting the interrelationships tested in this study. ERN, error-related negativity; ICPS, interchannel phase synchrony for error trials; PEA, post-error accuracy; PERT, post-error RT; PSWQ, Penn State Worry Questionnaire. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

transmission between electrodes over these regions. Interestingly, a recent report suggests similar anxiety-related aberrations in the action-monitoring system. Fitzgerald et al. (2013) found that clinically anxious children show attenuated LPFC activity following the commission of an error suggesting hypoactive recruitment of the LPFC itself following errors. Thus, in conjunction with recent work, our data suggest that anxiety is associated with aberrations in multiple mechanisms of the action-monitoring system rather than specific aberrations in ACC/mPFC-mediated processes.

These findings provide an interesting extension of the attentional control theory of anxiety (Eysenck et al., 2007) and of our CEMH (Moser et al., 2013, 2014). The attentional control theory proposes that anxious individuals increase task-related effort in an attempt to maintain performance and that this effort is inferred from increased response times and neural activation in control areas while accuracy remains unchanged. We have recently extended this to suggest errors prompt anxious individuals to engage in extra effort in order to improve or 'normalize' performance (Moser et al., 2013, 2014). However, few, if any, studies have examined the specific mechanisms whereby

performance is maintained in anxiety. That is, anxious individuals appear to be recruiting ACC/mPFC regions to a greater degree than non-anxious individuals; however, this enhanced recruitment in control regions is not translating into improved performance—i.e. anxious individuals demonstrate cognitive inefficiency. Until now, the source of the inefficiency has largely remained opaque. This study suggests that, while the initial ACC/mPFC-mediated processes are enhanced in worry, this information is not being effectively transmitted to LPFC regions and is therefore not translated into improved behavioral performance. That is, worry appears to exert opposing forces on subsequent behavior. On the one hand, worry is associated with reduced connectivity between medial and lateral regions, as revealed by the significant path between the PSWQ and ICPS, which results in poorer PERT and PEA when considered in isolation. However, worry is also associated with an enhanced ERN which, in turn, is associated with greater ICPS, as revealed by the significant paths between the PSWQ and ERN and between the ERN and ICPS. The opposing effects thus result in a net effect near 0 as evidenced by the correlations in Table 1. These findings, then, (i) are in line with our CEMH in which we claim

that the ERN functions as a compensatory mechanism aimed at maintaining task performance and (ii) help further our understanding of compensatory processes in anxiety and its nuanced relationship with behavioral performance.

These findings are less easily integrated into previous theories of anxiety and the ERN. Previous accounts of anxiety and error-related brain activity have focused on the ERN as a motivational signal in response to potential environmental threats (i.e. errors; Weinberg et al., 2012). Within such frameworks, it is unclear how reduced transmission of error-related information to LPFC regions, which are thought to underlie attentional and motoric adjustments in response to errors (Botvinick et al., 2001; Eichele et al., 2008; Shenhav et al., 2013), aids in the survival of the organism.

The specific anxiety-behavior relationships our model revealed—that worry is indirectly related to decreased PERT and PEA—are noteworthy. Recent accounts suggest slow responses that follow errors actually reflect off-task orienting (Notebaert et al., 2009; Castellar-Nunez et al., 2010) or the consequence of error-related task disruption (van den Brink et al., 2014). In this way, reduced PERT may reflect another index of compensatory attention related to worry rather than poor post-error adaptation. Similarly, worry's negative relationship to PEA is consistent with reduced capacity for proactive control (Fales et al., 2008), as we have previously contended that PEA is more sensitive to effortful task-relevant behaviors (Schroder et al., 2013). Further study into worry's relations to post-error behaviors through theta synchronization will help build a more complete picture of the mechanisms underlying cognitive and behavioral disruptions in anxiety.

Finally, these findings may shed light on inconsistencies regarding the functional significance of the ERN. While ACC/mPFC activation is generally predictive of behavioral adjustments following conflicts and errors (Botvinick et al., 2001; Eichele et al., 2008; Shenhav et al., 2013), the ERN itself has been less reliably associated with behavior (see Gehring et al., 2012 for a review). Gehring et al. (2012) have proposed that this inconsistency might be due to intervening processes that occur between the elicitation of the ERN and subsequent behavior. Indeed, these findings suggest that the ACC/mPFC processes which are indexed by the ERN are not directly related to post-error behavior whereas the transmission of error-related information (i.e. ICPS) between ACC/mPFC and LPFC regions is predictive of subsequent behavior. Interestingly, the ERN indirectly predicted post-error behavior through ICPS. It seems, then, that there is little direct influence of the ERN on subsequent behavior. Rather, this association appears to be better accounted for by shared variance with ICPS.

Limitations

This study had a few limitations worth considering. First, this study included only female participants. We focused on female participants for two reasons: (i) anxiety disorders are nearly twice as prevalent in women as in men (Kessler et al., 2005) which is suggestive of possible sex differences in anxiety and (ii) the ERN relates to anxiety only in female participants (Moran et al., 2012). However, we note that this design choice makes it difficult to determine whether the findings regarding ICPS extend to male participants. Second, this study included undergraduates reporting on their anxiety. Although our previous work suggests that the ERN increases linearly across the entire spectrum of anxiety severity (e.g. Moser et al., 2013), it is possible that error-related ICPS functions differently in severely and

chronically anxious individuals. Further research will be needed to extend these findings to clinical anxiety. Third, we note that our sample size was 77. There are two general categories of recommendations for determining the sample size for path analytic work. The first category stresses the importance of the participant-to-variable ratio and suggests that a ratio of 5–10 is adequate (Nunnally, 1978; Kline, 1998; also see Hair et al., 1995); by such standards, the participant-to-variable ratio of this study (15.4) was more than adequate. The second category stresses the absolute number of participants. These guidelines typically recommend samples exceeding 100 (Hutcheson and Sofroniou, 1999; also see Kline, 1998). Additionally, Sobel's *t* is asymptotically normal and *P* values are therefore only accurate in large samples (Aroian, 1947; Preacher and Hayes, 2008). Caution is urged when interpreting such results in smaller samples. Although our sample was somewhat small, we note that the current sample size (i) was larger than many studies examining the anxiety/ERN relationship (see Moser et al., 2013) and (ii) was comparable to other studies examining individual differences in ERN amplitude using path analytic methods (Batholow et al., 2012; Teber and Inzlicht, 2014). Nonetheless, future work should replicate this in a larger independent sample. Finally, we have interpreted our ICPS results as indicating that worry is associated with decreased functional integration between ACC/mPFC and LPFC regions. While our results are consistent with such an interpretation (Cavanagh et al., 2009), it should be borne in mind that connectivity was inferred from scalp recordings. Future research will benefit from functional connectivity analyses conducted with fMRI data.

Conclusion

The results of this study suggest that anxiety is not only related to the ERN but is also related to subsequent cognitive control stages aimed at adjusting future behaviors. Findings such as these help us to better understand the cognitive inefficiencies thought to underlie anxious pathology (Eysenck et al., 2007) as well as the effects of anxiety on the neurobehavioral mechanisms involved in cognitive control.

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