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Sex moderates the relationship between worry and performance monitoring brain activity in undergraduates

Tim P. Moran, Danielle Taylor, Jason S. Moser *

Department of Psychology, Michigan State University, United States

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ABSTRACT

Research suggests that abnormal performance-monitoring contributes to the etiology and maintenance of anxious pathology. Moreover, the anxiety–performance monitoring relationship appears to be specific to the worry dimension of anxiety. Given that anxiety (and worry in particular) is twice as prevalent in women as men, and most studies to date have employed small samples which are underpowered to detect sex-differences, it is possible that sex may be an important moderator of the worry–performance-monitoring relationship. No studies have directly compared the worry–performance-monitoring relationship between men and women, however. In the current study, we extended our recent work showing a unique relationship between worry and performance monitoring brain potentials in female undergraduates by comparing this relationship to that between worry and performance-monitoring brain potentials in male participants. Seventy-nine female and 70 male undergraduates from an ongoing study of anxiety and performance monitoring performed a letter-flanker task while their brain activity was recorded. Results revealed that worry was associated with exaggerated performance-monitoring, as indexed by increased error-related negativity/correct-response negativity, in female, but not male undergraduates. These findings suggest that the functional relationship between worry and performance-monitoring is sex-specific and have implications for understanding the role of performance-monitoring in the development and maintenance of anxiety. Specifically, linking the worry–performance-monitoring relationship to other female-specific biopsychosocial factors represents an important direction for future research.

1. Introduction

Anxiety is a common experience characterized by worried thoughts, physiologic tension and numerous cognitive deficits. In certain contexts, anxiety may represent an adaptive response to environmental stressors (Marks and Nesse, 1994). Persistent and maladaptive anxiety, however, is one of the most common mental health problems in the United States (Kroenke et al., 2007; Kessler et al., 2005). Thus, delineating factors that contribute to their etiology and maintenance is of great theoretical, as well as practical, importance. Particularly, recent studies have focused on identifying biomarkers that may be useful for identifying at-risk individuals and avenues for future treatment research (Gottesman and Gould, 2003). Research suggests that the anterior cingulate cortex (ACC) –a frontostriatal brain region-mediated performance monitoring processes, particularly those indexed by event-related potentials (ERPs), may represent one such set of important biomarkers (Olvet and Hajcak, 2008; Botvinick et al., 2004; Kerns et al., 2004). Importantly, recent research has demonstrated that exaggerated ACC activation is characteristic of many anxious populations.

Abnormal ACC activity has been observed in obsessive–compulsive disorder, undergraduates high in trait-anxiety, and individuals at risk for developing anxiety disorders (Ursu et al., 2003; Paulus et al., 2004; Simmons et al., 2008). In a related line of research, enhancement of the error-related negativity (ERN), an ERP consistently localized to the ACC (Dehaene et al., 1994; van Veen and Carter, 2002), has been related to anxiety (Olvet and Hajcak, 2008). The ERN is a sharp, negative deflection in the electroencephalogram (EEG) that occurs approximately 50 ms following the commission of an erroneous response in two-choice reaction time tasks (Gehring et al., 1990; Falkenstein et al., 1991) and is thought to reflect early error- or conflict-monitoring processes (Coles et al., 2001; Holroyd and Coles, 2002; Yeung et al., 2004). An enhanced ERN has been observed in obsessive–compulsive disorder and generalized anxiety disorder as well as obsessive–compulsive and worried and mixed anxious–depressed undergraduates (Gehring et al., 2000; Weinberg et al., 2010; Hajcak and Simons, 2002; Hajcak et al., 2003, 2004).

All types of anxiety are not created equal, however. Drawing on theoretical and psychometric distinction between anxious apprehension (i.e., worry) and anxious arousal (i.e. somatic tension; Barlow, 1991; Clark and Watson, 1991; Nitschke et al., 2001), our group recently showed that an enhanced ERN was uniquely associated with the worry dimension of anxiety in a sample of female undergraduates

* Corresponding author at: Department of Psychology, Michigan State University, East Lansing, MI 48824, United States. Tel.: +1 517 355 2159; fax: +1 517 353 1652.
E-mail address: jmoser@msu.edu (J.S. Moser).

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(Moser et al., 2012b). These findings dovetail nicely with previous research in that enhanced ERN is routinely reported in groups high in worry – i.e., patients and students with obsessive–compulsive disorder and generalized anxiety disorder – and not those characterized by somatic symptoms and specific fears (Hajcak et al., 2004; Moser et al., 2005). Additionally, our data fit with previous research showing that worry and anxious arousal are associated with unique patterns of regional brain activity. Specifically, worry has been associated with increased activity in frontal areas whereas anxious arousal has been associated with increased activity in parietal regions (Engels et al., 2007; Heller et al., 1997; Nitschke et al., 1999). Given that the ERN is consistently localized to the medial frontal cortex (i.e., ACC), demonstrating that worry is uniquely associated with an enhanced ERN is consistent with the broader literature suggesting worry's relationship to hyperactivity in frontal brain regions. We have therefore suggested that enhancements of ERN amplitude might result from the specific effects of the worry dimension of anxiety on performance-monitoring processes (Moser et al., 2012b). Specifically, we theorize that worry depletes working memory resources in the frontal cortex resulting in the need for enhanced compensatory performance monitoring to maintain a standard level of performance (c.f. Attentional Control Theory; Eysenck et al., 2007). The primary aim of the current study was to extend this work by comparing the association we found in our female sample with that of a new group of males.

The correct-response negativity (CRN) and error-positivity (Pe) are two additional indices of ACC-mediated performance-monitoring processes (Herrmann et al., 2004). The CRN is a small negative deflection that appears to reflect similar performance-monitoring processes as the ERN, albeit on correct trials (Bartholow et al., 2005; Vidal et al., 2000, 2003). The relationship between anxiety and the CRN is less clear, however. Some studies have noted enhanced ERN and CRN (ERN/CRN) amplitudes in anxious individuals (Hajcak and Simons, 2002; Hajcak et al., 2004; Moser et al., 2012b; Endrass et al., 2008, 2010), whereas others have reported a specific enhancement of the ERN (Gehring et al., 2000; Weinberg et al., 2010). Following the ERN/CRN is the Pe, a slow, positive deflection peaking 200–400 ms following the commission of an error at centroparietal recording sites (Overbeek et al., 2005). The Pe is generally thought of as reflecting the conscious awareness that an error has occurred (Overbeek et al., 2005; Kaiser et al., 1997; Nieuwenhuis et al., 2001). Studies examining the influence of anxiety on the Pe have reported equivocal results: some studies have failed to find an association (Endrass et al., 2008; Ruchsow et al., 2005), some report attenuations to the Pe and its correct-trial counterpart (i.e., N300; Hajcak et al., 2004; Moser et al., 2012b), whereas others show an enhanced Pe and N300 (Weinberg et al., 2010).

The association between the worry dimension of anxiety and the ERN appears to be most robust. Given that the ERN is a stable, trait-like measure (Moser et al., 2005; Hajcak et al., 2008) with excellent internal consistency and moderate-to-good test–retest reliability (Olvet and Hajcak, 2009a; Weinberg and Hajcak, 2011), it has been argued that the ERN may serve as a trait biomarker of anxiety and its disorders (Olvet and Hajcak, 2008; Riesel et al., 2011). However, despite the promise of the ERN as an index of liability for anxiety, no studies have examined demographic factors that might affect their relationship. Anxiety in general and worry in particular are considerably more common in women than men (Kroenke et al., 2007; Kessler et al., 2005; Stavosky and Borkovec, 1988). Furthermore, there is evidence suggesting sexual dimorphism in ACC activation. Christakou et al. (2009) reported increased ACC activation in women during a conflict-monitoring task and a recent meta-analysis reported greater emotion-related ACC activation in women (Wager et al., 2003). Conversely, Davies et al. (2004) reported greater ACC activation in boys relative to girls (ages 7–25) in a performance-monitoring task. Similarly, Larson et al. (2011) found an enhanced ERN in male college students. Therefore, sex may represent a particularly important moderator of the worry–ERN/CRN relationship. Previous studies have relied on relatively small samples ranging between 18 (8 males; Gehring et al., 2000) and 60 (30 males; Hajcak and Simons, 2002) participants and most times include a preponderance of female participants (e.g. n = 41, 11 male; Weinberg et al., 2010). Therefore, previous studies may have been under-powered to detect sex-differences in the worry–ERN/CRN relationship.

Thus, the purpose of the current study was to extend our recent work (Moser et al., 2012b) by being the first to directly examine the potential moderating effect of sex on the relationship between two dimensions of anxiety – worry and anxious arousal – and performance monitoring ERPs. Specifically, we combined our previous female sample with additional female subjects and a new sample of male undergraduates – recruited as part of an ongoing study of anxiety and performance monitoring – to test whether the association between worry and the ERN/CRN we previously reported in females would extend to males or not. We further examined whether the null association we reported between anxious arousal and the ERN/CRN would extend to males. Finally, we explored associations between worry and anxious arousal and the Pe/N300 across sexes.

2. Method

2.1. Participants

Undergraduates participated in our ongoing study of anxiety and performance monitoring for course credit. Participants were excluded from analyses if greater than 50% of trials were rejected due to EEG activity containing excessive artifacts (criteria described below) or if fewer than six errors were committed, as per Olvet and Hajcak (2009b). The final sample consisted of 149 participants (79 female, 70 male), of which 67 females were previously reported on in Moser et al. (2012b). Participants ranged in age from 18 to 40 ($M = 20.20$, $SD = 3.03$). Men ($M = 20.00$, $SD = 4.10$) and women ($M = 20.47$, $SD = 1.72$) did not differ significantly with respect to age ($t\ (148) = .88$, $p = .42$). Deviations in reported degrees of freedom resulted from missing behavioral or questionnaire data. No participants discontinued their involvement once the experiment had begun.

2.2. Task

Participants completed a letters version of the Eriksen Flanker task (Eriksen and Eriksen, 1974). Stimuli were presented on a Pentium R Dual Core computer, using Presentation software (Neurobehavioral Systems, Inc.) to control the presentation and timing of stimuli, the determination of response accuracy, and the measurement of reaction times.

During the task, participants were presented with a string of five letters. Each five-letter string was either congruent (e.g. FFFFF) or incongruent (e.g. EEFEE) and participants were required to respond to the center letter (target) via the left or right mouse button. Trial types were varied randomly such that 50% of the trials were congruent. Characters were displayed in a standard white font on a black background and subtended 1.3° of visual angle vertically and 9.2° horizontally. A standard fixation mark (+) was presented during the inter-trial interval (ITI).

Each trial began with the presentation of the flanking stimuli (i.e. EE EE). Flanking stimuli remained on the screen for 35 ms and were followed by the target (i.e. EEFEE), which remained for 100 ms (135 ms total presentation time). Each trial was followed by a variable ITI (1200–1700 ms). The entire experimental session consisted of 480 trials grouped into six blocks (80 trials each). The letters constituting each string were varied between blocks (e.g., M and N in block 1 and E and F in block 2) and response-mappings were reversed at the midpoint of each block (e.g., left mouse-button click for M through 40 trials of block 1, then right-mouse button click

for M for the last 40 trials of block 1) in order to elicit a sufficient number of errors.

Following the Flankers task, participants completed questionnaires including the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) and the Anxious Arousal (AAr) subscale of the Mood and Anxiety Symptom Questionnaire (MASQ; Clark and Watson, 1991). The PSWQ served as the measure of worry and the MASQ-AAr was used as the measure of anxious arousal (AAr) per Nitschke et al. (2001).

2.3. Psychophysiological data recording, reduction and analysis

Continuous electroencephalographic (EEG) activity was recorded from 64 Ag–AgCl electrodes fitted in a BioSemi (BioSemi, Amsterdam, The Netherlands) stretch-lycra cap. In addition, two electrodes were placed on the left and right mastoids. The electro-oculogram (EOG) generated by eye-movements and blinks were recorded by FP1, as well as by electrodes placed below the right eye 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 Driven Right Leg passive electrode formed the ground. All bioelectric signals were digitized at 512 Hz.

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 rolloff). Ocular artifacts were then corrected using the regression method developed by Gratton et al. (1983). Response-locked data were segmented into individual epochs beginning 200 ms prior to the response and continued for 1000 ms. Individual trials were rejected on the basis of excessive physiological activity: 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. Finally, the response-locked EEG was averaged across trials to yield error- and correct-trial ERPs for each site.

To quantify ERPs, a baseline equal to the average activity in the 200 ms window prior to response onset was subtracted from each data point subsequent to the response. The ERN and CRN were then defined on error and correct trials, respectively, as the average voltage occurring in the 0–100 ms post-response window at FCz, where they were maximal. The Pe and N300 were defined on error and correct trials, respectively, as the average activity in the 200–400 post-response window at CPz, where they were maximal.

2.4. Analytic approach

Data were statistically evaluated using SPSS General Linear Model software (Version 20). Measures with skewness or kurtosis values greater than 2 were transformed in order to normalize distributions. Thus, MASQ-AAr scores were successfully log transformed. The arcsine transform was performed successfully for accuracy (Keppel and Wickens, 2007; Kirk, 1968); descriptive statistics for these measures are reported using raw data. With respect to accuracy, the effects of anxiety measures and participant sex were established with Pearson's r and t-tests, respectively. Early action-monitoring ERPs, late action-monitoring ERPs and RTs were submitted to separate repeated-measures multiple linear regressions (Miller and Chapman, 2001; for a similar method, see Li et al., 2007) using the SPSS (v. 20) GLM repeated measures module. In order to establish baseline experimental and sex effects, analyses were first performed with 1 two-level within subjects factor (Accuracy: error vs. correct) and 1 two-level between subjects factor (Sex: female vs. male), excluding individual differences scores on anxiety measures. To test the primary hypotheses of the current study, the same 2 (Accuracy)×2 (Sex) repeated-measures multiple regression was re-run with anxiety measures (both PSWQ and MASQ-Aar) entered simultaneously as continuous predictors (see, Moser et al., 2012a,b as well as Moser et al., 2011 for a similar procedure). The focus of the current study was on the interaction effects involving the Sex factor and anxiety scores. Follow-up analyses are reported when effects involving participant sex or self-reports reached statistical significance in the omnibus analyses.

3. Results

Means and standard deviations for all measures are presented in Table 1.

3.1. Self-report measures

PSWQ and MASQ-AAr scores were moderately correlated across the whole sample ($r=.28$, $p=.01$). This correlation was similar in men ($r=.31$, $p<.01$) and women ($r=.27$, $p=.01$; $Z=.25$, $p=.81$, two-tailed). Men and women also did not differ on either PSWQ ($t(147)=1.1$, $p=.27$, $d=.18$) or MASQ-AAr scores ($t(144)=.54$, $p=.59$, $d=.08$).

3.2. Performance measures

3.2.1. Accuracy

Overall response accuracy was high ($M=93.27\%$, $SD=4.86$). MASQ-AAr scores were significantly, negatively correlated with Accuracy ($r=-.17$, $p=.05$). Accuracy was unrelated to PSWQ scores ($r=-.10$, $p=.22$) and did not differ by sex (t $(143)=.10$, $p=.92$, $d=.02$).

3.2.2. RT

Participants responded significantly faster on error trials than on correct trials ($F(1,144)=424.87$, $p<.001$, $\eta^2_p=.75$). There was no main effect of sex ($F(1,143)<2.1$, $p>.15$, $\eta^2_p=.01$), however the Sex×Accuracy interaction was significant ($F(1,137)=5.48$, $p=.02$, $\eta^2_p=.04$). Between-groups t-tests revealed that RTs did not differ between men and women on either error or correct trials (ts $(143)<2$, $ps>.05$, $ds<.32$). Within-groups t-tests revealed that, although RTs on error trials differed significantly from RTs on correct trials in male participants ($t(67)=11.26$, $p<.01$, $d=1.17$), this difference was considerably larger for female participants ($t(76)=19.20$, $p<.01$, $d=1.66$). A significant MASQ-AAr×Sex×Accuracy interaction was also found on RTs (F $(1, 137)=5.47$, $p=.02$, $\eta^2_p=.04$). Separate correlations were conducted between MASQ-AAr and the difference between correct-RT and error-RT (correctRT−errortRT) for men and women. For men, the correlation was small and positive ($r=.17$, $p=.17$). However, for women the correlation was small and negative ($r=-.19$, $p=.10$). There were no other significant effects of MASQ or PSWQ scores ($Fs<2.4$, $ps>.12$, $\eta^2_{ps}<.02$).

Table 1
Summary data: overall and as a function of sex.

	Females		Males		All participants	
	M	*SD*	*M*	*SD*	*M*	*SD*
PSWQ	50.75	12.49	48.4	13.47	49.64	12.97
MASQ-AAr	23.7	5.97	24.36	6.8	24.00	6.35
Accuracy (%)	93.23	5.38	93.32	4.24	93.27	4.86
Error RT (ms)	370.01	50.56	387.63	62.59	378.27	57.01
Correct RT (ms)	446.69	41.61	450.99	43.99	448.71	42.65
ERN (μV)	−5.01	4.85	−4.77	4.14	−4.89	4.52
CRN (μV)	−0.85	4.01	0.06	3.95	−0.42	3.99
Pe (μV)	7.49	7.35	7.15	6.42	7.33	6.91
N300 (μV)	−6.48	4.12	−4.5	3.85	−5.55	4.1

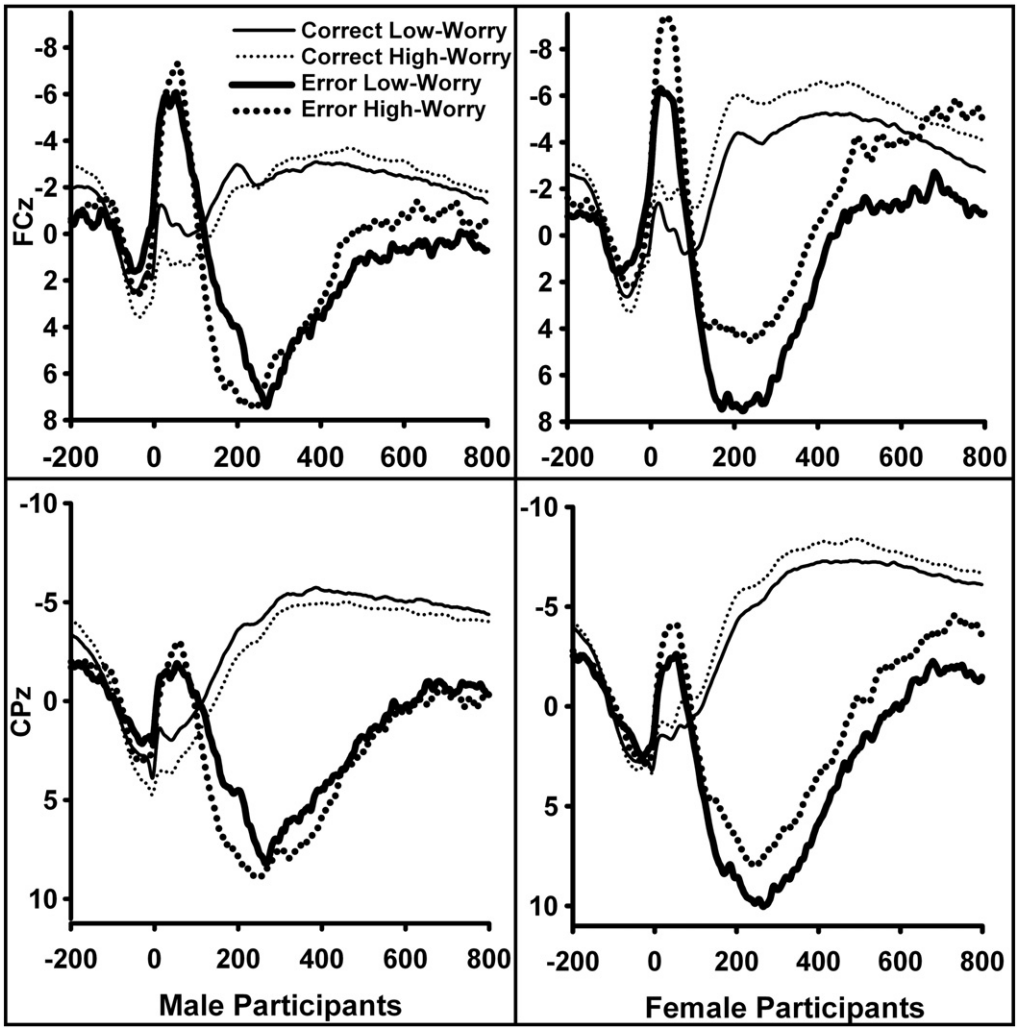

Fig. 1. Response-locked ERPs for the upper and lower quartiles of PSWQ scores plotted separately for men (left panels) and women (right panels) at electrode sites FCz (top panels) and CPz (bottom panels).

3.3. ERPs

3.3.1. ERN/CRN

Fig. 1 depicts the ERN and CRN as a function of sex and worry; participants were divided into upper and lower quartiles of PSWQ scores for illustrative purposes only. As shown in Fig. 2, brain activity immediately following responses (0–100 ms) was fronto-centrally distributed and was similar between men and women. Later response-locked ERPs (200–400 ms), however, were centro-parietally distributed and showed a broader distribution for females on correct trials.

ERP activity elicited by errors was significantly more negative than that by correct responses in the 0–100 ms time window, consistent with the ERN/CRN differentiation (F (1,148) = 144.57, p<.001, η^2_p = .49). There was no significant main effect of Sex[1] or Sex × Accuracy interaction on post-response negativity (Fs<1, ps>.30, η^2_{ps}<.01). A PSWQ × Sex interaction was found on overall post-response negativity amplitude (F(1,140) = 4.70, p = .03, η^2_p = .03). Follow-up correlational analyses were conducted separately for men and women. As shown in Fig. 3, a significant, negative correlation was found in female participants (r = −.27, p = .02) indicating that higher PSWQ scores were related to greater overall post-response negativity amplitudes. However, this correlation was near zero in male participants (r = .08, p = .49). When controlling for MASQ-Aar, the correlation in women remained significant (r = −.25, p = .03) and that in male participants remained non-significant (r = .09, p = .49). There were no other significant effects of PSWQ (Fs<1.7, ps>.19, η^2_{ps}<.02) and no effects involving the MASQ-AAr (Fs<1, ps>.7, η^2_{ps}<.01).

3.3.2. Pe/N300

The mean ERP amplitude between 200 and 400 ms post-response on error trials was significantly more positive than on correct trials, consistent with the Pe/N300 differentiation (Fig. 1; F(1,148) = 539.06, p<.001, η^2_p = .79). This was qualified by a significant Sex × Accuracy interaction (F(1,147) = 4.49, p = .04, η^2_p = .03). Between-groups t-tests revealed that the Pe did not differ by participant sex (t(147) = .31, p = .76, d = .04), however the N300 was significantly more negative in female relative to male participants (t(147) = 3.02, p<.01, d = .49). Within-groups t-tests revealed that the Pe and N300 differed significantly in both women (t(78) = 17.12, p<.001, d = 2.34) and men (t(69) = 16.26, p<.001, d = 2.20). There were no significant effects of worry or anxious arousal scores (Fs<3.56, ps>.06, η^2_{ps}<.03).

4. Discussion

The current study was the first to examine the potential moderating influence of sex on the relationship between anxiety symptoms and

[1] A visual inspection of our data suggested the possibility of peak amplitude differences between men and women. However, this did not reach significance (F(1, 147) = 2.43, p = .12, η^2_p = .02).

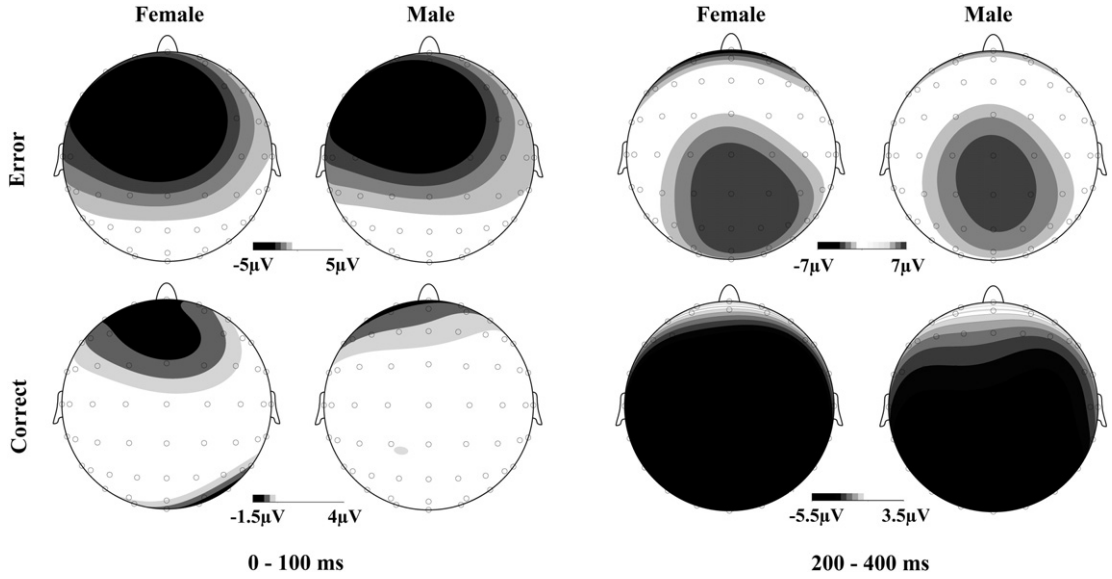

Fig. 2. Scalp topographies depicting voltages in 0–100 ms and 200–400 ms window following error and correct trials separately for men and women.

neural correlates of performance-monitoring. Results indicated that early performance-monitoring ERPs were most strongly related to worry in female participants. Specifically, in female participants, as worry increased, so did overall post-response negativity amplitude. In male participants, however, worry was not associated with early performance-monitoring ERP amplitude. Secondly, extending our previous findings, anxious arousal was unrelated to ERN/CRN in both sexes (Moser et al., 2012b). Later performance-monitoring brain potentials, as reflected by the Pe and N300 were not found to be associated with either dimension of anxiety across women and men.

The novel finding of the current study is that the worry–ERN/CRN relationship is specific to females. Interestingly, this difference emerged despite no significant differences in overall ERN/CRN amplitudes or PSWQ scores between the sexes. Several studies have pointed to the potential etiologic and maintenance role that abnormal ACC activation may play in anxiety. Specifically, enhanced ACC activity has been observed in obsessive–compulsive, trait-anxious undergraduates and individuals at risk for developing anxiety disorders (Ursu et al., 2003; Paulus et al., 2004; Simmons et al., 2008; Gehring et al., 2000; Weinberg et al., 2010; Hajcak and Simons, 2002; Hajcak et al., 2003; Moser et al., 2012b). Some researchers have suggested that abnormal ACC activation may index a vulnerability to developing anxiety disorders (Paulus et al., 2004). Importantly, the current findings suggest that enhanced ACC-mediated performance-monitoring processes (i.e. ERN and CRN) may only be useful biomarkers for denoting risk for anxiety disorders in women. It is possible that these findings are indicative of qualitative differences in the underlying mechanisms of worry between the sexes. Additionally, given that anxiety disorders affect women at approximately twice the rate at which they affect men (Kroenke et al., 2007; Kessler et al., 2005; Stavosky and Borkovec, 1988), the current results also suggest that abnormal ACC functioning may contribute to sex differences in the prevalence of anxiety. However, the causal mechanisms involved are not yet well understood. It is also possible that worry contributes to abnormal ACC functioning. For example, Shansky et al. (2004) noted that stress-induced impairments to prefrontal cortex functioning is specific to female rats; importantly, the specific manipulation in this study served to increase response-conflict during task execution (a process thought to characterize ACC functioning; see Yeung et al., 2004), suggesting that stress (a precursor to anxiety) may precipitate modulations in ACC-activation. Future research will benefit from further exploring the link between ACC functioning and risk for anxiety as well as female-specific biopsychosocial factors related to the etiology of anxiety.

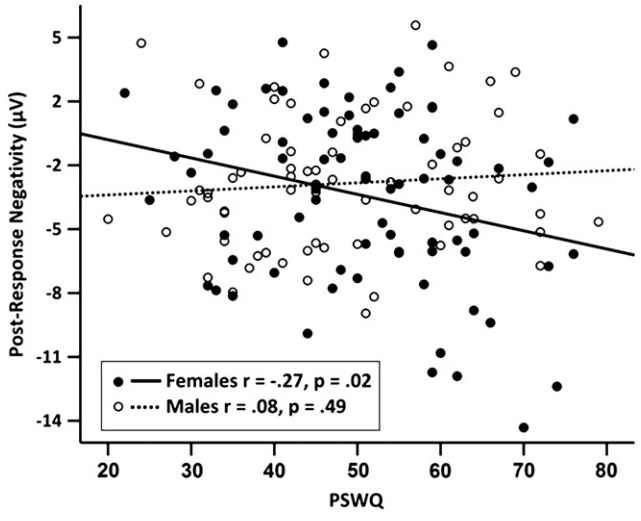

Fig. 3. A scatter-plot depicting the amplitude of overall post-response negativity as a function of Sex and PSWQ score. As post-response negativity scores are negative and PSWQ scores are positive, the negative correlation in female participants indicates that as PSWQ score increases, so does post-response negativity amplitude and vice vers.

The current study failed to replicate a recent investigation by Larson et al. (2011) which showed a larger ERN in men than women. Rather, in the current study ERN amplitudes appeared somewhat larger in female participants, although this was not a reliable finding. The current results are more consistent with sex differences in the prevalence of psychopathology. Several studies have noted that anxious pathology is approximately twice as prevalent in women as in men (Kroenke et al., 2007; Kessler et al., 2005; Stavosky and Borkovec, 1988); furthermore, externalizing pathology, known to be associated with an attenuated ERN (Hall et al., 2007), is considerably more common in men (Miller, 1991; Eagly and Steffen, 1986). Thus, in an unselected sample, one would expect a somewhat larger ERN in women due to anxiety-related enhancements and a smaller ERN in men due to externalizing-related attenuations. Alternately, the current task differed from that utilized by Larson et al. (2011); for example, in the current study, target stimuli differed by block and were present for considerably less time. Behavioral findings also differed from those reported by Larson et al. (2011). Finally, the discrepancy between this report and Larson's findings may reflect age-related differences in relative ERN amplitudes.

Both women and men in the current study were considerably younger than those included in the study by Larson and colleagues. Several studies have noted developmental changes in ERN amplitude (Davies et al., 2004; Kim et al., 2007; Wiersema et al., 2007; Ladouceur et al., 2004). Thus, this may represent sexual dimorphism in the development of performance-monitoring processes.

Neither anxious arousal nor worry was found to be associated with the Pe or N300. Previous work in this area has produced equivocal results. A study conducted in undergraduates noted an inverse association between negative affect and both the Pe and N300 (Hajcak et al., 2004); furthermore, we have recently noted reductions in both the Pe and N300 in female undergraduates high in worry[2] (Moser et al., 2012b). Work conducted in patient samples has also been mixed (Weinberg et al., 2010; Endrass et al., 2008; Ruchsow et al., 2005). The ambiguity in the existing literature suggests the possibility that the association between the Pe and anxiety may be moderated by other variables (e.g. symptom severity). Future research should focus on identifying the specific conditions under which the anxiety–Pe association emerges. Furthermore, the current results suggest that Pe amplitude does not differ between male and female participants; however, the amplitude of its correct-response counterpart was significantly more negative in female participants in the current sample. Bartholow et al. (2005) suggested this component (i.e., N300) may reflect bringing cognitive resources to bear on future conflict processing. Thus, one possibility is that the enhanced N300 in women reflects higher levels of cognitive control activity in order to maximize performance. However, given the current lack of research on this component, more work is needed to understand this finding.

Unlike worry, anxious arousal (i.e. MASQ-AAr scores) was more strongly associated with ineffective behavioral performance than performance-monitoring brain activity. That is, anxious arousal was associated with less accurate performance overall. Whereas worry was associated with enhanced neural activity and not accuracy, suggesting inefficient processing, anxious arousal was specifically associated with poorer accuracy. As anxious arousal has been linked to environmental scanning and threat detection (Nitschke et al., 2000), it is possible that the current findings resulted from reduced attentional resources allocated to the task. Also of note, this association held across the entire sample and was not moderated by participant sex. Future work will benefit from further parsing the specific effects of different dimensions of anxiety on cognitive performance across men and women.

It should be noted that, in the current study, we utilized a large set of female subjects previously reported on in Moser et al. (2012b) and, thus, this study should not be considered a replication of the female effect. However, it is also important to note that our finding in females is not controversial, as most other studies of anxiety and the ERN/CRN have utilized largely female participants and reported the same association we have here. That is, it is quite well accepted that anxiety, and worry, in particular, is associated with enhanced ERN/CRN in females (Olvet and Hajcak, 2008). The strength of our design, on the other hand, is the larger sample size. Indeed, this study represents the largest anxiety–ERN/CRN study to be published to date. The addition of a new sample of males gave us the opportunity to not only evaluate the worry–ERN/CRN relationship in males themselves, but also the chance to directly compare this association with that which we reported on in females – albeit with a slightly larger sample size than we had before (from 67 to 79). Thus, the current study shows a null association between worry and the ERN/CRN in males and demonstrates that this null association is significantly smaller that the moderate association in women.

Additionally, it should be noted that this study was conducted on an unselected sample of undergraduates displaying mostly subclinical levels of anxiety. This approach is in line with recent conceptualizations of psychopathology as dimensional in nature and suggestions that differences between clinical and subclinical severity levels represent quantitative, and not qualitative, differences (Brown and Barlow, 2009; Ruscio et al., 2001; Watson, 2005; Flett et al., 1997). Similarly, recent work has demonstrated that cognitive deficits previously examined in clinical populations extend across the full range of symptom severity (e.g. Moser et al., 2012a,b; Macleod and Mathews, 1988). Moreover, approximately 19% of those included in the current sample were likely to meet threshold for generalized anxiety disorder, as per Behar et al. (2003; PSWQ $\geq$ 62). Thus, the current findings are applicable to a wide range of individuals and levels of symptom severity.

The current study extends previous research by showing that the anxiety–ERN/CRN relationship is specific to the worry component of anxiety in female participants. The full implications of the current study require further investigation; however, these findings imply that the ERN and CRN may only be useful trait markers for the identification of anxious pathology in women. Future studies examining the association between worry and performance-monitoring brain activity should consider sexual dimorphism and focus on female participants as well as on identifying conditions under which the worry–ERN/CRN relationship emerges in males. Finally, these findings suggest that linking the ERN to other sex-specific biopsychosocial factors (e.g., hormones) that play a role in the etiology and maintenance of anxiety disorders represents an important avenue for further research.

[2] Our previous work has noted a negative relationship between worry and overall late performance-monitoring ERPs in a subsample of the females included here (Pe/N300; Moser et al., 2012b). Given the overlap between the samples used in our previous study and our current study, we examined this correlation for men and women separately to determine whether it replicated in female participants. The results indicated a moderate relationship in female participants ($r = -.23$, $p < .05$) and a near-zero relationship in men ($r = .02$, $p = .84$). The difference between these correlations was not sufficiently large to interact with sex in the omnibus analysis.

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