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Punishment has a lasting impact on error-related brain activity

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Abstract

The current study examined whether punishment has direct and lasting effects on error-related brain activity, and whether this effect is larger with increasing trait anxiety. Participants were told that errors on a flanker task would be punished in some blocks but not others. Punishment was applied following 50% of errors in punished blocks during the first half of the experiment (i.e., acquisition), but never in the second half (i.e., extinction). The ERN was enhanced in the punished blocks in both experimental phases—this enhancement remained stable throughout the extinction phase. More anxious individuals were characterized by larger punishment-related modulations in the ERN. The study reveals evidence for lasting, punishment-based modulations of the ERN that increase with anxiety. These data suggest avenues for research to examine more specific learning-related mechanisms that link anxiety to overactive error monitoring.

Descriptors: Error-related negativity, Punishment, Anxiety, Error processing, Conditioning

The ability to monitor our actions and to detect errors is a critical function for adaptive changes in behavior. Errors in speeded response tasks are associated with a characteristic pattern of brain activity that can be seen in the event-related potentials (ERP). Studies of error monitoring have focused in particular on the error-related negativity (ERN; Gehring, Goss, Coles, Meyer, & Donchin, 1993) or error negativity (Ne; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991), a sharp negative deflection that appears shortly after the commission of an error over medial frontocentral electrodes. Both functional neuroimaging (Debener et al., 2005; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004) and source localization studies (Dehaene, Posner, & Tucker, 1994; Hoffmann & Falkenstein, 2010) point to the anterior cingulate cortex (ACC) as a neuronal generator of the ERN.

The functional role of the ERN is still debated. However, most existing theories focus on cognitive functions (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Falkenstein et al., 1991; Gehring et al., 1993; Holroyd & Coles, 2002). These theories assume that the ERN signals the need to adjust behavior and to initiate increased cognitive control to improve future performance (Botvinick et al., 2001; Falkenstein et al., 1991; Gehring et al., 1993; Holroyd & Coles, 2002). In addition to this, a growing body of literature links alterations in error processing to psychopathology and individual difference measures of affective distress and defensive motivation (e.g., Olvet & Hajcak, 2008, Weinberg, Riesel, & Hajcak, 2011). An enhanced ERN has been demonstrated in individuals that seem to process the commission of errors as abnormally salient and aversive, such as obsessive-compulsive disorder (OCD) patients (Endrass, Klawohn, Schuster, & Kathmann, 2008; Endrass et al., 2010; Gehring, Himle, & Nisenson, 2000; Hajcak, Franklin, Foa, & Simons, 2008; Johannes et al., 2001; Riesel, Endrass, Kaufmann, & Kathmann, 2011; Ruchsow et al., 2005; however, see Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005) or highly anxious healthy individuals (Aarts & Pourtois, 2010; Hajcak, McDonald, & Simons, 2003). Further, this enhancement of the ERN among anxious individuals seems to be stable and largely state-independent (Hajcak et al., 2008; Moser, Hajcak, & Simons, 2005; Riesel et al., 2011). Combined with results demonstrating the excellent temporal stability and reliability of the ERN (Olvet & Hajcak, 2009a; Segalowitz et al., 2010; Weinberg & Hajcak, 2011), we have suggested that the ERN is a neurobehavioral trait (Patrick & Bernat, 2010; Weinberg et al., 2011). This raises important questions regarding how differences in ERN magnitude arise. Between 40% and 60% of the observed variability in ERN magnitude appears heritable (Anokhin, Golosheykin, & Heath, 2008). However, a substantial proportion of the variance in ERN magnitude cannot be explained by genetic factors, but may instead be attributed to environmental factors (Anokhin et al., 2008) such as learning experiences that alter error significance.

In addition to trait-like differences in ERN magnitude, there is evidence that the error-monitoring system is sensitive to situational context and variation in error significance in within-subject manipulations. Transient alterations in the magnitude of the ERN can be affected by experimental manipulations that modulate error significance, either via monetary incentives (Chiu & Deldin, 2007; Endrass et al., 2010; Ganushchak & Schiller, 2008; Hajcak, Moser, Yeung, & Simons, 2005; Pailing & Segalowitz, 2004), task instructions that emphasize accuracy (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring et al., 1993), or external performance evaluation (Hajcak et al., 2005; Kim, Iwaki, Uno, & Fujita, 2005). In this line, manipulations that reduce the perceived value of errors

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such as alcohol application (Easdon, Izenberg, Armilio, Yu, & Alain, 2005; Ridderinkhof et al., 2002) or sleep deprivation (Hsieh, Cheng, & Tsai, 2007; Hsieh, Li, & Tsai, 2010; Tsai, Young, Hsieh, & Lee, 2005; however, see Murphy, Richard, Masaki, & Segalowitz, 2006) have been linked to reduced ERN magnitude.

Combined, these findings suggest that the ERN is sensitive to the subjective value of errors and is influenced by both situational context and more trait-like characteristics. Moreover, situational variables and stable individual differences appear to interact in their influence on error monitoring (Amodio, Master, Yee, & Taylor, 2008; Dikman & Allen, 2000; Endrass et al., 2010; Luu, Collins, & Tucker, 2000; Olvet & Hajcak, 2011; Pailing & Segalowitz, 2004). However, no study to date has used punishment contingencies to manipulate error significance. Furthermore, it has not been investigated whether such within-subject manipulations of error significance can lead to lasting changes in the ERN (i.e., once punishment has stopped). The primary goal of the present study was to examine whether the ERN would be enhanced in conditions in which errors are punished, and if this effect would be evident once errors were no longer punished. Given the growing body of literature that links overactive error monitoring and anxiety (e.g., Olvet & Hajcak, 2008, Weinberg et al., 2011) we further wanted to explore whether the impact of punishment on the ERN would be increased among individuals higher in trait anxiety.

In addition to the ERN, other response-related ERP components may inform our knowledge about error processing. The ERN is typically followed by a centroparietal positivity called the error positivity (Pe) that occurs between 200 to 400 ms after an incorrect response (Falkenstein et al., 1991; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). Like the ERN, the Pe is also assumed to be generated in the ACC (van Veen & Carter, 2002). In contrast to the ERN, however, the functional significance of the Pe is less well understood (Overbeek et al., 2005), though one increasingly popular view relates the Pe to error awareness (Endrass, Reuter, & Kathmann, 2007; Hughes & Yeung, 2011; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). Furthermore, there is growing evidence for the notion that the Pe may reflect a P300, or more specifically a P3b, elicited by the commission of infrequent errors (Arbel & Donchin, 2009; Overbeek et al., 2005). In keeping with this, the Pe may be associated with the motivational significance of, or context updating after, an error (Overbeek et al., 2005). In contrast to the ERN, results regarding variations of the Pe as a function of psychopathology and personality are less clear (Overbeek et al., 2005).

The aim of the present study is first to extend previous work demonstrating the sensitivity of the magnitude of the ERN to manipulations of error significance by inducing threat (i.e., using primary punishment, specifically, an aversive noise following errors). We used a design that combines elements of instructed (i.e., instructing that errors in some conditions will be punished) and associative learning (i.e., pairing errors in some conditions with punishment). Moreover, drawing from the fear learning literature, we included both an "acquisition" and "extinction" phase—halfway through the experiment, errors stopped being punished—so that we could examine whether changing punishment contingencies would be reflected in the ERN. Based on previous results (Chiu & Deldin, 2007; Endrass et al., 2010; Ganushchak & Schiller, 2008; Hajcak et al., 2005; Pailing & Segalowitz, 2004), we assumed that the ERN would be increased in the punishment condition. We further hypothesized that punishment would lead to lasting changes in error monitoring, such that the ERN enhancement for the punishment condition would be evident throughout the experiment (i.e., increased in both acquisition and extinction phases). Furthermore, we examined the impact of trait anxiety on this effect. In particular, based on the literature suggesting enhanced fear conditioning and slower extinction for individuals with anxiety disorders (Hermann, Ziegler, Birbaumer, & Flor, 2002; Lissek et al., 2005; Orr et al., 2000) and high levels of trait anxiety (Barrett & Armony, 2009; Otto et al., 2007; Sehlmeyer et al., 2011; Zinbarg & Mohlman, 1998), we expected that individuals high in trait anxiety would demonstrate stronger and longer-lasting effects of punishment on the ERN.

Methods

Participants

Thirty-four undergraduate students (23 female) from Stony Brook University participated in this study. Three participants committed fewer than six errors (Olvet & Hajcak, 2009b) and were therefore excluded from further analysis. Data from three subjects were excluded from analysis due to excessive electroencephalogram (EEG) artifacts. The final sample consisted of 28 participants (20 female). The remaining participants had normal or corrected-to-normal vision and reported no history of head trauma or neurological disease. The mean age was 19.79 ($SD = 2.71$); 53.6% of the sample was Caucasian/European, 25% was Asian-American, 7.1% was Hispanic, 3.6% was African-American, and 10.7% identified as "other." All participants received verbal and written information about the aims and procedure of the study, and written consent was obtained. All participants received course credit for their participation.

Measures

The trait version of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) was administered to measure individual differences in state and trait anxiety. The inventory contains 20 items and measures relatively stable individual differences in anxiety proneness (scores range from 20 to 80). The STAI scores of the participants varied between 25 and 51 ($M = 38.03$, $SD = 8.25$). Compared to norms of healthy college students, none of these participants had a percentile rank over 90% and only four subjects had a percentile rank between 80% and 90%, suggesting that the majority of scores were within the normal range.

Task

An arrowhead version of the flanker task (Eriksen & Eriksen, 1974; Kopp, Rist, & Mattler, 1996) was administered using Presentation software (Neurobehavioral Systems, Inc., Albany, CA). On each trial, five horizontally aligned arrowheads were presented, and participants were instructed to respond with the left or right mouse button in accordance with the direction of the central arrowhead. Half the trials were compatible (e.g., flanker arrows and target point in the same direction) and half were incompatible (e.g., flanker arrows and target point in opposite directions). The trials were displayed in a pseudorandomized order. At a viewing distance of approximately 65 cm, the set of arrows filled 0.9° of visual angle vertically and 7.5° horizontally. All stimuli were presented for 200 ms, followed by an intertrial interval (ITI) that varied randomly from 900 to 1500 ms. Throughout the experiment, participants were encouraged to be both fast and accurate in their

performance. To encourage both fast and accurate behavior, performance-based feedback was presented at the end of each block. If performance accuracy was below 75%, a message appeared instructing participants to respond more accurately. When the performance was above 90%, participants were instructed to respond faster. Error rates between 10% and 25% were followed by the feedback, "You're doing a great job." The total duration of the task was approximately 45 min.

Procedure

Our experimental design combined elements of both instructed and associative learning—which have been shown to produce similar effects on psychophysiological measures in fear learning paradigms (Olsson & Phelps, 2004). There were blocks of trials in which errors were punished (punishment condition from here on), and blocks of trails in which errors were not punished (control condition from here on). The punishment and control conditions were distinguished by the color of the arrows presented, which were either blue or yellow. The assignment of color to control versus punishment condition was counterbalanced across participants. The conditions (control vs. punishment) varied blockwise in an alternating way whereby the order was counterbalanced across participants (e.g., ABABABAB or BABABABA). For instance, errors committed during blue arrow blocks might be punished, whereas errors committed during yellow arrow blocks would not be punished. In the beginning of the experiment, participants were explicitly instructed that errors in one color condition would sometimes be followed by a loud sound. However, they were not informed which color would be punished.

We refer to the first half of the experiment as the acquisition phase because of the presence of a contingency between errors and punishment in certain blocks. During the punishment conditions of the acquisition phase, the commission of an error was punished by presenting an aversive loud sound (100 db) with a 1-s duration (punishment condition) via two speakers to the right and left of the monitor; errors in the other color condition were never punished (control condition). The high-pitched sound (3500 Hz) was presented 1 s after the error was committed. We presented the aversive sound with a 1-s delay after the commission of an error to prevent a potential confound of response-locked ERPs. After a punishment sound was presented, the ITI was increased by 1500 ms, such that it varied between 3000 and 3900 ms. The reinforcement schedule during the acquisition block changed from continuous (i.e., the first five errors in the punishment condition were punished) to intermittent (50% of errors in the punishment blocks were punished subsequently). This change was introduced to ensure both fast and stable learning. The number of punished errors varied across participants, depending on their error rate (number of punished errors: $M = 12.08$, $SD = 4.97$).

We refer to the second half of the experiment as the extinction phase. During the extinction phase, errors were never followed by a loud sound, regardless of the condition (formerly punished or control). Each experimental phase consisted of 8 blocks of 64 trials per block (1024 trials total in both phases). Prior to the experiment, the participants performed a practice block containing 20 trials, half presented in blue and half in yellow. At the end of the experiment, participants were asked whether and when they realized that the punishment had stopped. Seventy-four percent of the participants stated that they were aware midway through the experiment that errors in the punishment condition were no longer punished. The remaining 26% were aware three quarters of the way through the experiment. Thus, all participants reported being aware that punishment was not presented in the last quarter of the experiment.

Psychophysiological Recording, Data Reduction, and Analysis

The continuous EEG was recorded using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, The Netherlands). Thirty-four electrode sites were used, based on the 10/20 system, as well as two electrodes on the right and left mastoids. All electrodes were sintered Ag-AgCl electrodes. The electrooculogram (EOG) was recorded using four additional facial electrodes: two electrodes placed approximately 1 cm outside of the right and left eyes and two electrodes mounted approximately 1 cm above and below the right eye. To improve the signal-to-noise ratio, the EEG signal was preamplified at the electrode with a gain of 1× by a BioSemi ActiveTwo system (BioSemi, Amsterdam). The EEG was digitized with a sampling rate of 1024 Hz using a low-pass fifth order sinc filter with a half-power cutoff of 204.8 Hz. A common mode sense (CMS) active electrode producing a monopolar (nondifferential) channel was used as recording reference. Offline, the data was referenced to the average of the left and right mastoids, and bandpass filtered with low and high cutoffs of 0.1 and 30 Hz, respectively. Eye movement artifacts were corrected per Gratton, Coles, and Donchin (1983).

Response-locked epochs with duration of 1500 ms including a 500-ms prestimulus interval were extracted. A semiautomatic procedure was used to detect and reject artifacts. Epochs containing a voltage step of more than 50 μV between sample points, a voltage difference of 300 μV within a trial, and a maximum voltage difference of less than .50 μV within 100-ms intervals were rejected. In addition, visual inspection of the data was conducted to detect and reject any remaining artifacts. Response-locked ERPs were averaged separately for each participant, each experimental condition and phase, and for incorrect and correct responses. Trials with response times <100 ms and >700 ms were excluded from averaging. The interval from 400 ms to 200 ms prior to the response served as a baseline (Weinberg, Olvet, & Hajcak, 2010). The response-locked negativities were evaluated as the mean activity between 0 and 50 ms after response at FCz, where error-related brain activity was maximal. For topographical display, the difference between the ERN and CRN (i.e., ΔERN) was also calculated. Finally, the Pe was evaluated on error trials as the average activity from 200 to 400 ms at Pz. Grand averages were filtered with a 15-Hz low-pass filter for visual presentation.

Statistical analyses were conducted using SPSS (Version 18.0). A 2 (*response*: error, correct) × 2 (*condition*: punishment, control) × 2 (*phase*: acquisition, extinction) repeated measures analysis of variance (ANOVA) was used to analyze the reaction time and electrophysiological data. For all ERP analyses, only subjects that had at least six artifact-free error trials were analyzed (Olvet & Hajcak, 2009b). Since sex has been shown to influence error processing (Larson, South, & Clayson, 2011) additional analyses with sex as a between-subjects factor were conducted. Error rate and post-error slowing were statistically analyzed by using a 2 (*condition*: punishment, control) × 2 (*phase*: acquisition, extinction) ANOVA. Paired *t* tests were performed for follow-up *post hoc* tests. Aside from *phase* and *condition* effects that were of central interest for the purpose of the present study, only significant main effects or interactions are reported in the Results section. The significance level was $\alpha = .05$, two-tailed. Correlational analyses (Pearson's *r*) were conducted to examine the association between error-related ERP components and self-reported anxiety.

Table 1. *Task Performance and Response-Related ERPs (Means and Standard Deviations) in the Control and Punishment Condition for the Learning and Extinction Phase*

	Learning phase		Extinction phase	
	Control	Punishment	Control	Punishment
Error rate	8.13 (3.88)	8.89 (3.35)	11.49 (4.35)	12.30 (5.17)
Correct RT in ms	418 (39)	421 (37)	411 (48)	413 (42)
Error RT in ms	344 (39)	356 (41)	347 (41)	356 (46)
Post-error slowing in ms	6 (29)	19 (24)	1 (16)	−1 (18)
ERN (FCz)	−0.13 (7.98)	−2.08 (7.72)	0.34 (6.64)	−1.59 (6)
CRN (FCz)	7.66 (5.69)	7.87 (4.86)	7.63 (5.14)	7.69 (5.43)
Pe (Pz)	18.98 (9.6)	19.89 (9.38)	18.52 (8.09)	17.91 (7.95)

Note. RT = reaction time.

Results

Behavioral Results

Behavioral results are presented in Table 1. The control and punishment condition did not differ with regard to error rates, $F(1,27) = 2.17$, $p = .15$, though the error rate was significantly included in the extinction phase compared to the acquisition phase, $F(1,27) = 32.29$, $p < .001$. Consistent with earlier studies, reaction times were faster for incorrect than correct responses, $F(1,27) = 419.50$, $p < .001$. Responses in the punishment condition, regardless of the phase of the experiment, were slower compared to responses in the control condition, $F(1,27) = 4.91$, $p < .05$.

Post-error slowing was analyzed by comparing the difference between reaction times following correct incongruent trials with reaction times following errors in incongruent trials. Only incongruent trials were included in this analysis in order to avoid a confound with the congruency effect, and because errors occurred mainly in incongruent trials. Furthermore, in the punishment condition of the acquisition phase, only reaction times after unpunished errors were evaluated to avoid a confound with immediate effects of the punishment. Only participants with more than six unpunished errors were statistically analyzed. There was a significant main effect for *phase*, which reflected a decrease in post-error slowing from the acquisition to the extinction phase, $F(1,24) = 9.98$, $p < .01$. However, *post hoc* tests indicated that only post-error slowing in the punishment condition showed a significant decrease from acquisition to extinction; punishment condition: $t(1,24) = 3.70$, $p < .01$, control condition: $t(1,24) = 0.90$, $p = .37$. This is also reflected in a trend for a *condition* × *phase* interaction, $F(1,24) = 3.21$, $p = .09$. While there was a pronounced post-error slowing for the punishment condition in the acquisition phase, $t(1,24) = 2.06$, $p = .05$, there was no difference in post-error slowing for both conditions in the extinction phase, $t(1,24) = 0.48$, $p = .64$.

Error-Related Brain Activity

Figure 1 presents the response-locked ERP waveforms for correct and error trials in the punishment and control condition for both experimental phases. Consistent with previous studies, the ERN was observed as a sharp negative deflection, peaking shortly after the commission of an error, with a frontocentral distribution. The ERN was significantly more negative than the CRN,

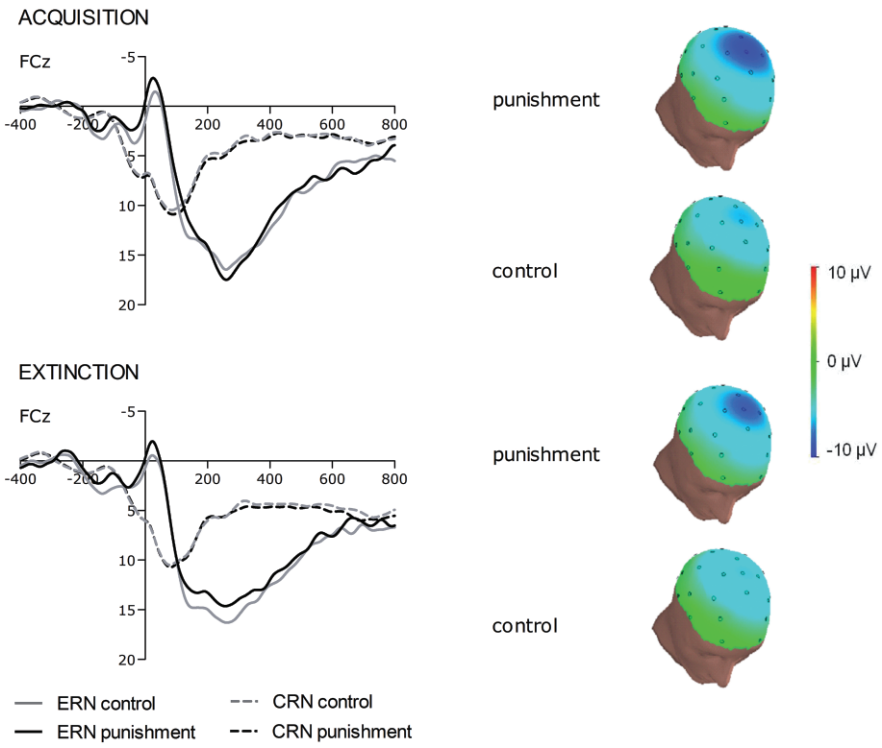

Figure 1. Response-locked ERP waveforms for correct and incorrect responses at FCz (left) and scalp topographies for the difference between the ERN and CRN in the time window from 0 to 50 ms (right) in the punishment and control conditions for the acquisition (top) and extinction (bottom) phases of the experiment.

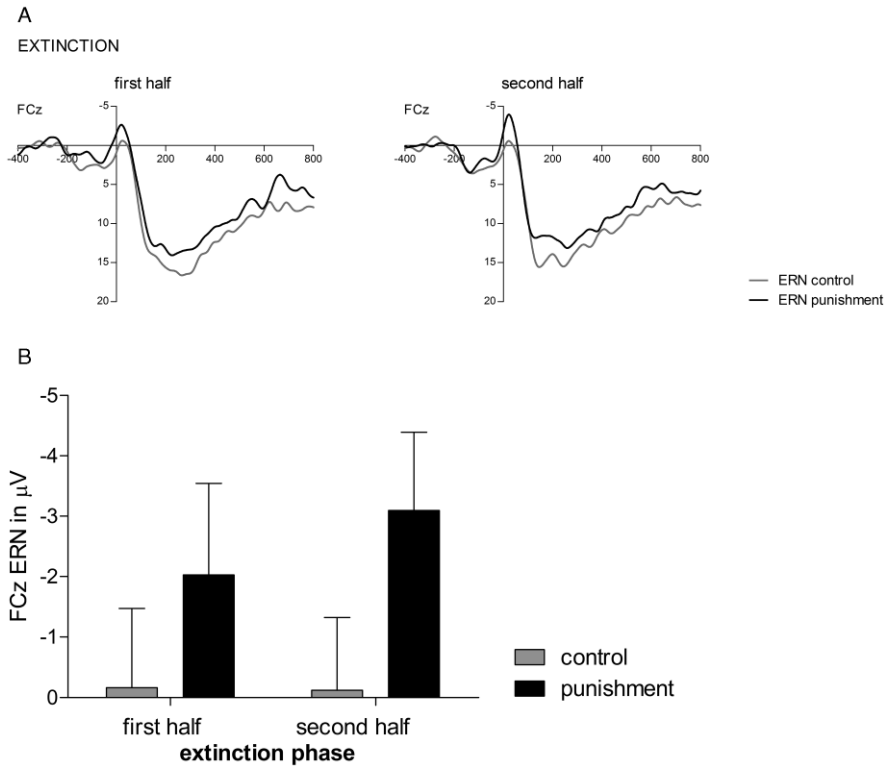

Figure 2. (A) Response-locked ERP waveforms at FCz, in the punishment (black) and control conditions (gray) for the first and second half of the extinction phase. (B) Bar chart showing the mean amplitude (0–50 ms) and standard errors in the punishment (black) and control conditions (gray) for the first and second half of the extinction phase.

$F(1,27) = 212.78$, $p < .001$. A significant main effect of *condition,* $F(1,27) = 7.56$, $p < .05$, and its interaction with *response*, $F(1,27) = 9.78$, $p < .01$, indicated that amplitudes in the punishment condition were selectively enhanced for the ERN. Importantly, there was no main effect of *phase*, $F(1,27) = 0.14$, $p = .71$, nor an interaction between *phase* and *condition*, $F(1,27) = 0.01$, $p = .93$, or *phase*, *condition,* and *response*, $F(1,27) = 0.01$, $p = .90$. Therefore, the ERN was larger in the punishment condition compared to the control condition in both experimental phases; acquisition phase: $t(1,27) = 2.18$, $p < .05$; extinction phase: $t(1,27) = 2.07$, $p < .05$. Thus, enhanced ERN amplitudes for the formerly punished condition compared to the control condition were also observed in the extinction phase, during which errors were no longer punished.

Sex did not influence this pattern of results. Neither a main effect of sex, $F(1,26) = 1.13$, $p = .30$, nor any interaction with sex reached significance, all p values > .10. Furthermore, the ERN in the punishment condition of the acquisition phase did not differ between error trials with ($M = -3.31$, $SD = 5.69$) and without the punishing sound ($M = -3.07$, $SD = 6.52$); $t(1,12) = .14$, $p = .89$), indicating that auditory processing did not confound the ERN results. One might argue that the observed ERN pattern could be explained by speed–accuracy tradeoffs, because previous research has shown increased ERN amplitudes when emphasizing accuracy over speed (Falkenstein et al., 2000; Gehring et al., 1993). To control for behavioral differences between the punishment and control condition, we introduced reaction time differences between the conditions as covariate in an analysis of covariance (ANCOVA). A significant effect of condition, $F(1,26) = 10.18$, $p < .01$, response, $F(1,26) = 169.68$, $p < .001$, and a significant interaction response by condition, $F(1,26) = 11.13$, $p < .01$, were still present when ERP analyses were corrected for the difference in reaction times. This pattern of results also remained unchanged when differences in error rates between the control and punishment condition were included as a covariate, main effect condition: $F(1,26) = 10.99$, $p < .01$, main effect response: $F(1,26) = 188.77$, $p < .001$, interaction response by condition $F(1,26) = 8.43$, $p < .01$. Together, these results suggest that behavioral differences between the conditions (i.e., differences in speed–accuracy tradeoff) do not account for the observed pattern of ERN results.

To further explore whether the continued effect of punishment experience on the ERN in the extinction phase could be driven by errors occurring before participants had the opportunity to realize that the punishment had stopped, ERN amplitudes in the extinction condition were reanalyzed comparing errors occurring in the first half and errors occurring in the second half of the extinction phase with a 2 (*condition*) × 2 (*half*) ANOVA. Six participants had to be excluded from this analysis due to insufficient error rates in each half (Olvet & Hajcak, 2009b). The magnitude of the ERN varied by *condition*, $F(1,21) = 7.84$, $p < .05$, as above, but not by *half,* $F(1,21) = 2.06$, $p = .17$. Nor did the interaction between *half* and *condition* reach significance, suggesting the effects of punishment persisted beyond the immediate application of the reinforcer even in the second half of the extinction phase, $F(1,21) = 0.45$, $p = .51$. Figure 2 displays the ERP waveforms for errors in the punishment and control condition separately for the first and second half of the extinction phase. If anything, the difference between the ERN in the punishment and control conditions became numerically larger from the first to second half of the extinction phase.

Grand average waveforms for correct and incorrect responses at Pz are displayed in Figure 3. The positivity after a response was significantly enhanced for errors compared to correct responses,

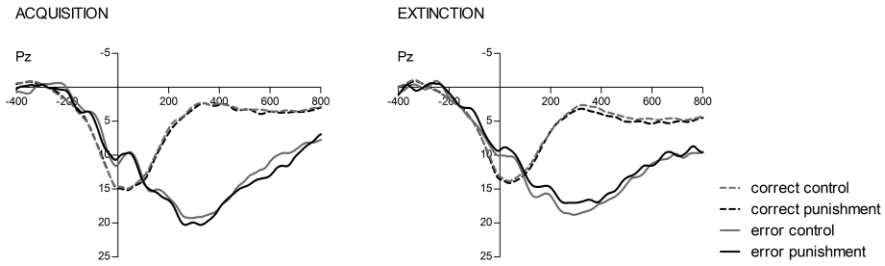

Figure 3. Response-locked ERP waveforms at Pz, comparing correct and incorrect responses in the punishment (black) and control conditions (gray) for the acquisition (left) and extinction (right) phases of the experiment.

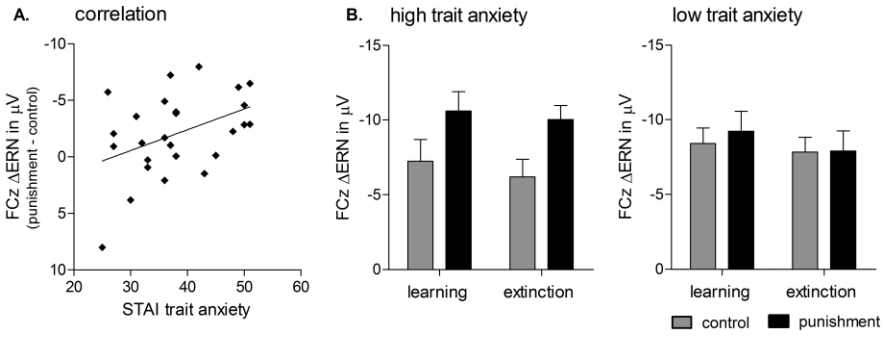

Figure 4. (A) Scatterplot depicting the relationship between trait anxiety assessed with the STAI and the difference between punishment and control condition in ΔERN (i.e., ERN minus CRN) across both experimental phases ($r = –0.42$, $p < .05$; left). (B) Bar charts of the ΔERN showing means and standard errors in the punishment (black) and control conditions (gray) for individuals with high (middle) and low trait anxiety (right) in both experimental phases.

$F(1,27) = 141.79$, $p < .001$. Additionally, the Pe decreased from the acquisition to the extinction phase of the experiment, which is reflected in a significant interaction between *phase* and *response*, $F(1,27) = 4.42$, $p < .05$. Even though the interaction between *phase, response, and condition*, $F(1,27) = 1.27$, $p = .27$, did not reach significance, *post hoc* tests indicated that only the Pe in the punishment condition showed a significant decrease from the acquisition to the extinction phase (acquisition compared to extinction in the control condition: $t(1,27) = 0.41$, $p = .69$; acquisition compared to extinction in the punishment condition: $t(1,27) = 2.25$, $p < .05$). There was no significant main effect of *condition* nor did any variable interact significantly with *condition* to determine the magnitude of the Pe response, all p values > .26. There was a trend for a main effect of *sex,* $F(1,26) = 3.33$, $p = .08$. Overall, females showed numerically larger positivities after errors and correct reactions. However, the observed pattern of results did not vary between sexes, and no interaction with sex reached significance, all p values > .25.

Effects of Trait Anxiety

To explore the effects of trait anxiety on the impact of punishment on the ERN, we correlated trait anxiety with the difference of ΔERN (i.e., ERN minus CRN amplitude) between punishment and control condition (i.e., ΔERN punishment minus ΔERN control). Trait anxiety was significantly negatively correlated with punishment-based modulations in ΔERN ($r = –0.42$, $p < .05$), such that higher scores for the trait measure of anxiety were associated with a larger difference in ΔERN between the control and punishment condition (see Figure 4).

To further examine the effect of trait anxiety, two groups were formed based on median level of trait anxiety (i.e., subjects above the median level of anxiety were formed into a "high" group ($N = 13$), and those below the median were formed into a "low group" ($N = 13$)). This was then introduced as a between-subjects factor in another 2 (condition) × 2 (phase) repeated measures ANOVA analyzing ΔERN. As above, we observed a significant main effect of *condition* suggesting that punishment led to enhanced ΔERN amplitudes, $F(1,24) = 9.69$, $p < .01$. This effect was specified by an interaction between *condition* and *trait anxiety* indicating that the effect of punishment in ΔERN amplitudes is specific to the high anxiety group, $F(1,24) = 5.87$, $p < .05$ (see Figure 4).[1] Accordingly, *post hoc* tests indicated that only individuals high in trait anxiety showed a modulation based on punishment in both phases (acquisition: $t(1,12) = 2.44$, $p < .05$, extinction: $t(1,12) = 3.62$, $p < .01$), whereas the ERN did not differentiate between the control and punishment conditions for low anxious individuals (acquisition: $t(1,12) = 0.59$, $p = .56$, extinction: $t(1,12) = 0.04$, $p = .96$).

Discussion

Consistent with previous work indicating that manipulations of error value and significance are associated with enhanced ERN amplitudes (Chiu & Deldin, 2007; Endrass et al., 2010; Falkenstein

1. There was no main effect of trait anxiety, $F(1,24) = 0.02$, $p = .89$, or phase, $F(1,24) = 2.53$, $p = .13$, and no interaction with phase, all p values > .68.

et al., 2000; Ganushchak & Schiller, 2008; Gehring et al., 1993; Hajcak et al., 2005; Pailing & Segalowitz, 2004), experimental manipulations of punishment modulated error-related brain activity in the current study. Whereas previous studies have utilized secondary reinforcers to modulate error significance, the present study used primary reinforcers. That similar effects were obtained is consistent with results indicating that both secondary and primary reinforcers have similar effects on learning (Delgado, Labouliere, & Phelps, 2006). These data suggest that early neural activity associated with error monitoring is sensitive to the motivational impact of potential punishment. The observed effect was specific to early neural response to errors: neither the CRN nor the subsequent Pe differentiated between the punishment and control blocks. In addition to enhanced ERN amplitudes, punishment also led to a more cautious response strategy, as reflected in longer reaction times for the punishment condition throughout the experiment, as well as increased post-error slowing in the acquisition phase of the punishment condition only. The Pe decreased from the acquisition to the extinction phase for the punishment condition, which may reflect a decrease in context updating to errors, or a reduction in more conscious evaluation of errors (Overbeek et al., 2005). Combined with the self-report data and the post-error slowing results, one could speculate this may reflect participants' awareness that errors were no longer punished in this condition.

Importantly, despite explicit knowledge that the punishment had stopped, the observed effect of punishment on both the ERN and reaction times remained stable throughout the extinction phase, in which errors were no longer punished. Moreover, errors in the formerly punished condition continued to elicit an enhanced ERN even in the second half of the extinction phase. This pattern of results remained stable when controlling for the difference in reaction times and error rates between the punishment and control conditions, suggesting that the ERN modulation was not driven by differences in speed–accuracy tradeoffs between conditions. These data are in line with a growing body of literature suggesting at least a partial dissociation between ERN magnitude and behavioral measures (Weinberg et al., 2011). Since differences in response strategy cannot account for the ERN results, the persistent effect of punishment throughout the experiment suggests that perceived threat surrounding errors can lead to lasting changes in the ERN. Since the time period examined in this study was relatively short, future studies should further explore the stability of experimentally induced alterations in ERN magnitude over longer periods of time.

Moreover, the results of this study suggest that certain personality features, such as trait anxiety, may lead to stronger effects of punishment-based modulations on error significance reflected in the ERN. Previous work has demonstrated that the impact of situational variables on the ERN can be moderated by personality traits and psychopathology (Amodio et al., 2008; Dikman & Allen, 2000; Endrass et al., 2010; Luu et al., 2000; Olvet & Hajcak, 2011; Pailing & Segalowitz, 2004). In line with this work, the current study revealed that trait anxiety was associated with enhanced punishment-based modulations in ERN amplitudes independent of the experimental phase, suggesting that individuals high in trait anxiety may be especially prone to acquire punishment-based modulations in error monitoring. This is consistent with assertions that enhanced fear conditionability and slowed extinction might be potential etiological factors in the development of anxiety disorders (Hermann et al., 2002; Lissek et al., 2005; Orr et al., 2000) and high levels of trait anxiety, more generally (Barrett & Armony, 2009; Otto et al., 2007; Sehlmeyer et al., 2011; Zinbarg & Mohlman, 1998). The results of this study suggest that the effects of enhanced conditionabilty may extend to overactive error monitoring due to punishment experiences that alter error significance. Future studies with larger samples and using more extreme variability in individual differences in anxiety levels are needed to further examine the effects of trait anxiety on how aversive learning experiences impact the ERN and whether high trait anxiety is also associated with slower extinction.

Differences in personality and psychopathology associated with affective distress and enhanced defensive motivation (Olvet & Hajcak, 2008, Weinberg et al., 2011) have been repeatedly linked to enhanced ERN amplitudes. The ERN has therefore been identified as a promising endophenotype for OCD (Riesel et al., 2011) or internalizing disorders broadly (Olvet & Hajcak, 2008). In addition to genetic effects, endophenotypes might also be influenced by shared environmental risk factors (Kendler & Neale, 2010). The results of this study emphasize the potential impact of environmental factors on error monitoring, such as punishment (or aversive learning experiences, more broadly) that can alter error significance and increase the ERN. These data suggest an etiological mechanism in addition to heritability (Anokhin et al., 2008) that can lead to overactive error monitoring and links anxiety to overactive error monitoring. Further, it is possible that this mechanism might link environmental adversity and parental behavior (e.g., overprotective/punitive parenting styles), which are thought to be etiological factors for a range of disorders in the internalizing spectrum (Hicks, DiRago, Iacono, & McGue, 2009; Hirshfeld-Becker, Micco, Simoes, & Henin, 2008), to increases in the ERN, which has also been repeatedly associated with these disorders (Olvet & Hajcak, 2008; Weinberg et al., 2011).

The present study has several limitations. The disproportionate representation of males and females in the current sample may have influenced the present results. Studies have reported sex differences in anxiety levels (Feingold, 1994), conditioning outcomes (Dalla & Shors, 2009; Kelly & Forsyth, 2007), and one recent study also reported sex differences in ERN amplitude (Larson et al., 2011). In the current study, the observed pattern of results did not vary between males and females. However, the disproportionate number of males in the study limited the ability to examine differences between sexes. The design of the current study did not permit exploration of the dynamics between ERN and the learning or extinction processes since no baseline or follow-up measures were included. Future studies with repeated measures over a longer period of time could examine the time-course of learning and extinction on ERN amplitudes. Further, the study design combines elements of instructed and associative learning. Future studies that use alternative designs can help to disentangle the specific learning processes involved. More specifically, it would be interesting to compare the impact of associative versus instructed learning on the ERN, as well as the effect of randomly presented punishment. We would note, however, that previous work demonstrated that the presence of a general threat (i.e., a tarantula, for spider phobic individuals) does not increase the ERN (Moser, Hajcak, & Simons, 2005). In the Moser et al. study (2005), participants were extremely afraid in one condition, though the threat was unrelated to their performance—and there was no corresponding increase in the ERN. In addition, the presence or absence of a loud startle probe on both error and correct trials does not appear to impact the ERN in between-subjects comparisons (Hajcak & Foti, 2008). Therefore, we think that our data, combined with existing literature, is most consistent with the view that the presence of error-related punishment, in particular, can lead to a rather sustained increase in the ERN.

The results of the present study have important implications for our understanding of processes that can lead to overactive error monitoring, which has been related to anxiety (Olvet & Hajcak, 2008; Weinberg et al., 2011). With regard to the functional role of the ERN, the present results are in line with previous evidence that the ERN amplitude relates to the motivational significance of errors (Hajcak & Foti, 2008; Hajcak et al., 2005; Luu et al., 2000; Weinberg et al., 2011). The ERN may be an early evaluation of the motivational salience of an error, and one that mobilizes additional cognitive processes as suggested by cognitive theories of the ERN (Botvinick et al., 2001; Falkenstein et al., 1991; Gehring et al., 1993; Holroyd & Coles, 2002) as well as defensive motivational systems to ensure adaptive behavior (Weinberg et al., 2011). The results of the present study demonstrate that, in addition to stable individual differences, punishment-related experiences may also lead to lasting changes in the ERN and motivational impact of errors. Furthermore, trait anxiety can facilitate the impact of punishment on the ERN. The identification of factors that reduce the impact of situational context, and conditions that reduce error significance (e.g., fearlessness), may also help to increase our understanding of personality features and psychopathologies that are characterized by abnormally small ERN responses. Thus, future research should examine these effects in multiple patient groups with different psychopathologies, as well as investigate this mechanism with longitudinal designs in individuals who are at risk for anxiety disorders. The results of this study show that punishment experiences that alter error significance have direct and lasting effects on the ERN. In a broader context, this points to the notion that the ERN can be shaped by both heritability and experiences that alter the subjective value of errors. This may be a promising starting point for future research focusing on both genetic and environmental influences on performance monitoring, which may inform our understanding of the development of multiple forms of psychopathology.

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