Article

Time Course of Error-Potentiated Startle and its Relationship to Error-Related Brain Activity

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Abstract. Errors are aversive, motivationally-salient events which prime defensive action. This is reflected in a potentiated startle reflex after the commission of an error. The current study replicates and extends previous work examining the time course of error-potentiated startle as a function of startle lag (i.e., 300 ms or 800 ms following correct and error responses). In addition, the relationship between error-potentiated startle and error-related brain activity in both the temporal (error-related negativity, ERN/Ne) and spectral (error-related theta and delta power) domains was investigated. Event-related potentials (ERPs) were recorded from 32 healthy undergraduates while they performed an arrowhead version of a flanker task. Complex Morlet wavelets were applied to compute oscillatory power in the delta- and theta-band range. Consistent with our previous report, startle was larger following errors. Furthermore, this effect was evident at both early and late startle probe times. Increased delta and theta power after an error was associated with larger error-potentiated startle. An association between ERN amplitude and error-potentiated startle was only observed in a subgroup of individuals with relatively large ERN/Ne amplitude. Among these individuals, ERN/Ne magnitude was also related to multiple indices of task performance. This study further supports the notion that errors are aversive events that prime defensive motivation, and that error-potentiated startle is evident beyond the immediate commission of an error and can be predicted from error-related brain activity.

Keywords: startle reflex, error-related negativity, ERN/Ne, theta and delta power

The ability to monitor actions and rapidly detect errors is critical in a complex and often-changing environment. Cognitive theories of error monitoring (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Holroyd & Coles, 2002) suggest that detection of errors occurs in order to adjust performance in accordance with ongoing demands, and that errors signal the need to increase cognitive control and adjust behavior to improve performance (Botvinick et al., 2001; Holroyd & Coles, 2002; Rabbitt, 1966).

Studies of error monitoring have focused in particular on the error negativity (Ne; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) or error-related negativity (ERN; Gehring, Goss, Coles, Meyer, & Donchin, 1993), a negative deflection in the event-related potential (ERP) that is maximal approximately 50 ms after the commission of errors. The ERN/Ne is thought to reflect the action monitoring activity of the anterior cingulate cortex (Dehaene, Posner, & Tucker, 1994; Debener et al., 2005; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Van Veen & Carter, 2002). Several studies have suggested that the ERN/Ne emerges in part from the increased phase-locking of frontal midline theta activity on error trials (Luu & Tucker, 2001; Luu, Tucker, & Makeig, 2004; Trujillo & Allen, 2007; Tzur & Berger, 2009; however, for methodological concerns, see Yeung, Bogacz, Holroyd, Nieuwenhuis, & Cohen, 2007). Synchronization in the theta band may be an underlying mechanism of communication between networks by which action monitoring and cognitive control networks interact (Cavanagh, Cohen, & Allen, 2009). In addition to an increase in theta activity related to error processing, increased activity in the delta frequency band activity has also been noted (Beste, Domschke, et al., 2010; Beste, Kolev, et al., 2010; Kolev, Beste, Falkenstein, & Yordanova, 2009; Kolev, Falkenstein, & Yordanova, 2005; Yordanova, Falkenstein, Hohnsbein, & Kolev, 2004). Yordanova et al. (2004) suggest that two subprocesses are reflected in the ERN/Ne: one in the theta frequency band that is thought to be related to a general response monitoring (i.e., movement monitoring) and one error-specific subcomponent in the delta frequency band.

In addition to the cognitive functions of error monitoring, there is increasing evidence that error processing is influenced by motivational variables. Individuals and groups characterized by excessive concern over errors – for whom error commission may be both more salient and more aversive – show enhanced ERN/Ne amplitudes. Specifically, healthy individuals with obsessive-compulsive characteristics (Hajcak & Simons, 2002), high trait levels of

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anxiety (Hajcak, McDonald, & Simons, 2003a), and high negative affect (Hajcak, McDonald, & Simons, 2004; Luu, Collins, & Tucker, 2000; Wiswede, Munte, Goschke, & Russeler, 2009; Wiswede, Munte, & Russeler, 2009), as well as clinical groups with obsessive-compulsive disorder (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) and Generalized Anxiety Disorder (Weinberg, Olvet, & Hajcak, 2010), are all characterized by an enhanced ERN/Ne. Contrary to predictions derived from purely cognitive theories of the ERN, the enhanced ERN/Ne in these studies is rarely reflected in performance differences. Rather, there is increasing evidence that the magnitude of the ERN/Ne varies substantially as a function of motivational states (Falkenstein et al., 2000; Gehring et al., 1993; Hajcak, Moser, Yeung, & Simons, 2005; Wiswede, Munte, Goschke, et al., 2009) and traits (Hajcak et al., 2008; Olvet & Hajcak, 2008; Riesel et al., 2011), independent of behavioral measures (Weinberg, Riesel, & Hajcak, 2012).

Furthermore, it is likely that errors are not simply detected in the service of adjusting and improving future performance: errors prompt a cascade of physiological changes, including skin conductance response, heart rate deceleration, and pupil dilatation, that may indicate orienting or preparation for defensive action (Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Hajcak, McDonald, & Simons, 2003b; Hajcak, et al., 2004). Consistent with this notion, the defensive startle reflex has been shown to be larger 300 ms following errors than correct responses (Hajcak & Foti, 2008). Moreover, those subjects with larger error-potentiated startle may be characterized by an increased ERN/Ne (Hajcak & Foti, 2008). These data collectively suggest that errors not only signal the need to increase cognitive control, but also mobilize defensive motivational systems for action.

The first goal of the current study was to extend previous work (Hajcak & Foti, 2008) by examining startle potentiation using both early (i.e., 300 ms) and late (i.e., 800 ms) startle probes following errors and correct responses. That is, we sought to both replicate our previous report of increased defensive reactivity immediately (i.e., 300 ms) following errors, and to establish whether startle potentiation would similarly be evident 500 ms later (i.e., 800 ms after error commission). Second, we sought to determine whether error-potentiated startle was predicted by early neural correlates of error-processing, focusing specifically on the ERN/Ne and error-related increases in delta and theta power.

Methods

Participants

Thirty-two undergraduate students (13 men, 19 women, mean age = 20.53 years, *SD* = 5.41) were recruited from Stony Brook University's psychology subject pool. All participants received verbal and written information about the purposes and procedure of the study, and written informed consent was obtained. All subjects received course credit for their participation.

Stimuli and Procedure

Participants performed an arrowhead version of the Flanker task and were required to indicate the direction of the central arrowhead on each trial by using the right or left mouse button. The central arrowhead could be flanked by compatible (i.e., < < < < < or > > > > >) or incompatible (i.e., < < > < < or > > < > >) arrowheads. All trial types were presented with equal probability. All visual stimuli were presented for 200 ms in white against a black background using Presentation Software (Neurobehavioral Systems, Inc., Albany, CA). Subjects had up to 800 ms to respond, after response the intertrial interval varied randomly between 500 and 1,000 ms.

Participants performed 11 blocks of 30 trials. At the end of each block, they received performance feedback designed to encourage fast and accurate responding. If performance was above 90% correct, the message "Please try to respond faster" was displayed; performance worse than 75% correct was followed by "Please try to be more accurate;" intermediate performance prompted "You're doing a great job." All participants performed one practice block of 30 trials prior to beginning the experiment.

Standard procedures were used for eliciting the defensive startle response (Bradley, Moulder, & Lang, 2005; Bradley, Codispoti, & Lang, 2006; Grillon, Ameli, Merikangas, Woods, & Davis, 1993; Lang, Davis, & Öhman, 2000): A 105-dB burst of white noise with a 50-ms duration and instantaneous rise time was presented via earphones. Startle probes were delivered on 10% of all trials in the practice block. To control for the predictability of startle probes, on average 50% of errors and 50% of correct trials following errors were followed by startle probes throughout the experiment; this manipulation does not appear to differ from presenting startle probes randomly (see Hajcak & Foti, 2008). On startle trials, the startle probe was randomly presented either 300 ms or 800 ms following response (i.e., startle probe latencies were equally probable).

Psychophysiological Recording and Data Reduction

Continuous EEG recordings were collected using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). Sixty-four electrode sites were used, based on the 10/20 system, as well as two electrodes on the right and left mastoids. Electrooculogram (EOG) generated from eye movements and eyeblinks was recorded using four facial electrodes: horizontal eye movements were measured via two electrodes located approximately 1 cm outside the outer edge of the right and left eyes. Vertical eye movements and blinks were measured via two electrodes

placed approximately 1 cm above and below the right eye. The EEG signal was pre-amplified at the electrode to improve the signal-to-noise ratio and amplified with a gain of 1× by a BioSemi ActiveTwo system (BioSemi, Amsterdam, The Netherlands). The data were digitized at 24 bit resolution with a sampling rate of 1,024 Hz using a low-pass fifth-order sinc filter with a half-power cutoff of 204.8 Hz. Each active electrode was measured online with respect to a common mode sense (CMS) active electrode producing a monopolar (non-differential) channel. The startle response was recorded with two electrodes placed approximately 12 mm apart under the participants' left eye.

Offline, all EEG data were referenced to the average of the left and right mastoids, and band-pass filtered with low and high cutoffs of 0.1 and 30 Hz, respectively. Eye-blink and ocular corrections were conducted per Gratton, Coles, and Donchin (1983). Startle data were band-pass filtered (28–512 Hz; 24 dB/octave roll-off), rectified, then low-pass filtered at 30 Hz (24 dB/octave) and baseline-corrected. Startle response magnitudes were quantified in terms of the peak in the 20–120 ms window after the presentation of the startle probe. Trials were rejected if a naturally occurring blink began within 25 ms of the probe.

A semi-automatic procedure was employed to detect and reject artifacts in the EEG data. The criteria applied were a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a maximum voltage difference of less than .50 μV within 100-ms intervals. These intervals were rejected from individual channels in each trial. Visual inspection of the data was then conducted to detect and reject remaining artifacts.

Epochs for time-domain analysis of the response-related negativities were segmented for each trial, beginning 500 ms before the response and continuing for 1,000 ms. The interval from 400 ms to 200 ms prior to the response served as a baseline. The magnitude of response-related negativities was quantified as the average activity in a 0- to 100-ms window following response onset on both error and correct trials across three electrodes (Fz, FCz, and Cz). Additional analyses were conducted to assess the influence of stimulus congruency on response-related negativities. For the ERN/Ne, only participants with more than six artifact-free errors (Olvet & Hajcak, 2009) were analyzed. In order to conduct the time-frequency analysis to compute the power of oscillatory activity, a current source density transform was applied to the data. The time-frequency analysis was then conducted by applying a continuous Wavelet transform using complex Morlet wavelets (Lachaux, Rodriguez, Martinerie, & Varela, 1999; Samar, Bopardikar, Rao, & Swartz, 1999). The epochs for time-frequency analysis had a length of 3,000 ms (beginning 1,500 ms before response) to achieve an adequate evaluation of low frequencies. The complex Morlet wavelet is defined by the following formula:

$$\Psi(t,f) = A e^{-t^2/2\sigma_t^2} e^{i2\pi ct} \quad (1)$$

In this formula, t is time, e is the base of the natural logarithm, and f is the frequency, which increased from 1 to 30 Hz in 20 logarithmic steps. Factor A is the normalization parameter. Parameter c determines the number of oscillations of the wavelet. The complex Morlet transformation was applied with $c = 4$ to provide an adequate trade-off between temporal and frequency resolution. A 300-ms time window preceding the flanker stimuli (−1000- to −700-ms pre-response) was used for normalization (i.e., gabor normalization). Theta activity was scored by extracting the wavelet power between 4 and 8 Hz from the averages for correct and erroneous responses. Delta activity was scored between 1.5 and 4 Hz. The time-frequency data was analyzed across three electrodes (Fz, FCz, and Cz) between −100 and 200 ms around the response.

All psychophysiological measures were analyzed using BrainVision Analyzer and all measures were statistically evaluated using SPSS (version 17.0). Grand averages were filtered with a 15-Hz low-pass filter for visual presentation.

Results

Behavioral Data

The mean number of errors across subjects was 44.16 (SD = 26.31). On average, 50% of errors and 50% of correct trials following errors were followed by startle probes; the lag time of these startle probes varied randomly across trials. As a result, the number of startle trials at each time delay was similar for correct and incorrect responses (errors with startle: M = 20.87, SD = 11.85; errors followed by an early startle probe: M = 10.32, SD = 6.32; errors followed by a late startle probe: M = 10.55, SD = 6.41; correct trials with startle: M = 21.03, SD = 10.69; correct trials followed by an early startle probe: M = 10.61, SD = 5.83; correct trials followed by a late startle probe: M = 10.42, SD = 6.17). Analysis of reaction times revealed that incompatible trials (M = 432 ms, SD = 44) were associated with increased reaction times compared to compatible trials (M = 338 ms, SD = 31; $t(30) = 15.03$, $p < .001$). Furthermore, correct responses (M = 412 ms, SD = 37) were significantly slower than incorrect responses (M = 339 ms, SD = 43; $t(30) = 12.87$, $p < .001$). Since errors primarily occurred on incompatible trials (87.24%, SD = 10.92), stimulus congruency may have confounded the response type results. However, correct responses (M = 448 ms, SD = 44) remained slower than errors (M = 342 ms, SD = 43) when only incompatible trials were analyzed ($t(30) = 15.48$, $p < .001$). Post-error slowing was computed as difference between response times in trials following an error compared to response times in trials following a correct response. Increased slowing was found for trials following errors (M = 436 ms, SD = 55), compared to trials following correct responses (M = 402 ms, SD = 36; $t(30) = 6.42$, $p < .001$). As above, this pattern of results remained unchanged when only incompatible trials were analyzed ($t(30) = 5.27$, $p < .001$).

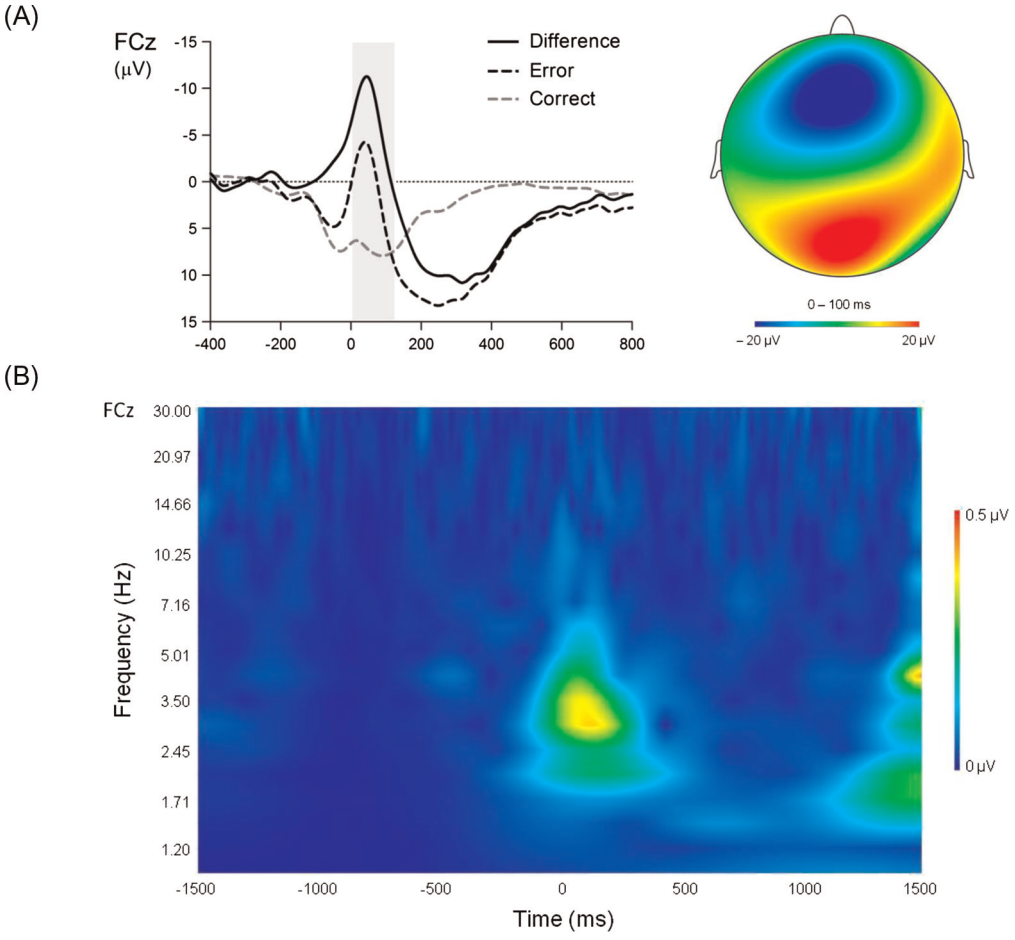

Figure 1. (A) Grand average waveforms for correct and incorrect responses and the difference wave at electrode FCz (top left) and the associated scalp topography (0–100 ms, current source density) for the ERN/Ne difference wave (top right). (B) Time-frequency plot (1–30 Hz, logarithmic scaling) depicts power over time for the difference of error compared to correct responses at FCz without startle presentation afterwards (bottom).

Error-Related Brain Activity

Figure 1A presents the response-locked ERP activity at FCz (top, left) and the scalp distribution of the error minus correct difference in the time range of the ERN/Ne (top, right). Consistent with previous work, the ERN/Ne was observed as a sharp negative deflection, peaking around 50 ms after an erroneous response. The ERN/Ne was larger following errors than correct responses ($t(31) = 12.45$, $p < .001$). Neither ERN/Ne ($t(12) = .38$, $p = .71$) nor the CRN ($t(31) = 1.87$, $p = .07$) differed in amplitude between compatible (ERN/Ne: $M = -0.50$, $SD = 6.29$; CRN: $M = 6.29$, $SD = 4.67$) and incompatible trials (ERN/Ne: $M = -0.92$, $SD = 5.50$; CRN: $M = 7.17$, $SD = 4.50$). However, the ERN/Ne remained larger compared to the CRN when only incompatible trials were analyzed ($t(31) = 9.73$, $p < .001$). A time-frequency plot portrays power over time for errors compared to correct responses in Figure 1B. Consistent with previous work, errors induced an increase in theta- ($t(31) = 7.83$, $p < .001$) and delta-band power ($t(31) = 5.53$, $p < .001$) relative to correct responses.[1]

Startle

A 2 (Probe: early vs. late) × 2 (Response: error vs. correct) repeated-measures ANOVA confirmed that startle was larger following errors ($M = 14.75$, $SD = 12.6$) than following correct responses ($M = 12.05$, $SD = 10.5$; $F(1, 30) = 12.57$, $p < .01$). Furthermore, the startle magnitude was significantly larger at the 300-ms probe delay ($M = 13.74$, $SD = 10.7$) than the 800 ms delay ($M = 12.67$, $SD = 12.2$; $F(1, 30) = 6.24$, $p < .05$). The interaction between response

[1] To score stimulus-related conflict, the stimulus-locked N2 was analyzed as the mean amplitude across three electrodes (Fz, FCz, Cz) between 250 and 350 ms for compatible and incompatible flanker trials (baseline -200 to 0 ms). The stimulus-locked N2 was significantly more negative for incompatible ($M = 1.15$, $SD = 4.94$) compared to compatible stimuli ($M = 2.78$, $SD = 5.05$, $t(31) = 6.32$, $p < .001$).

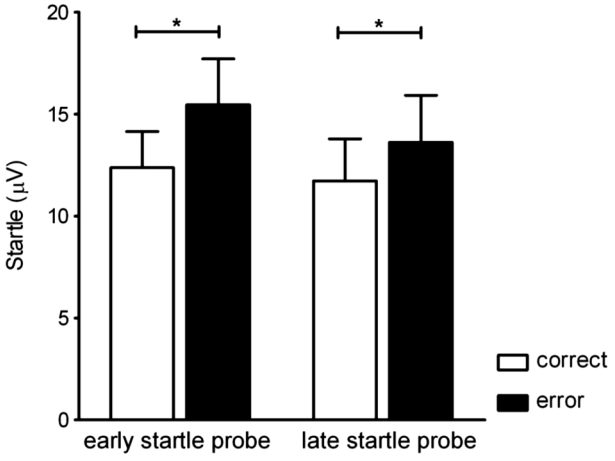

Figure 2. Mean startle magnitude and standard error as a function of response type and startle probe delay. *$p < 0.05$.

and probe delay was not significant ($F(1, 30) = 1.36$, $p = .25$), indicating that startle magnitude decreased for both correct and incorrect responses from the 300- to 800-ms lag. Like our previous study we found that the magnitude of the startle was larger after errors compared to correct responses for both the 300-ms ($t(31) = 2.75, p < .01$) and 800-ms startle probe delay ($t(31) = 2.51$, $p < .05$). Figure 2 displays startle magnitude for early and late startle probes following errors and correct responses.

Relationship Between Error-Related Brain Activity, Error-Potentiated Startle, and Behavior

To evaluate the extent to which error-related brain activity predicts startle potentiation, error-potentiated startle was defined as the average startle magnitude on error trials minus the average startle magnitude on correct trials, averaging over both time lags. Error-related brain activity was evaluated using the difference scores between error- and correct-related activity in both the time (i.e., ΔERN/Ne) and frequency (i.e., ΔTheta, ΔDelta) domains. We used zero-order correlations to examine the relationship between error-related brain activity, error-potentiated startle, and indices of task performance (see Table 1). ΔTheta and ΔDelta each related to the magnitude of error-potentiated startle. The relationship between error-potentiated startle and ΔTheta and ΔDelta was replicated when correcting for ΔDelta or ΔTheta respectively using partial correlations (ΔTheta: $r = .37$, $p < .05$; ΔDelta: $r = .33$, $p = .06$) suggesting that both show an independent association with error-potentiated startle. These results confirm that greater delta- and theta-band power after errors predicted a larger potentiation of the startle reflex after errors. However, ΔERN/Ne amplitude did not correlate with error-potentiated startle. ΔDelta power was negatively correlated with ΔERN/Ne magnitude, with more negative ERN/Ne amplitudes associated with higher delta power. All measures of error-related brain activity were significantly correlated with number of errors, such that an increased number of errors were associated with a *smaller* ERN/Ne amplitude (i.e., more positive), as well as with decreased theta- or delta band power. An increase in delta power following an error was also associated with enhanced post-error-slowing. In summary, the three measures of error-related brain activity show shared and distinct patterns of association with error-potentiated startle and performance indices, suggesting that these measures may overlap but are not redundant.[2]

Exploratory Post Hoc Data Analysis

Exploratory analyses were conducted to further examine the relationship between ERN/Ne amplitude and startle, and to determine whether the significance of errors, reflected in the magnitude of the ERN/Ne, might influence the relationship between the ERN/Ne and behavioral and defensive indices. Two groups (low ERN/Ne group: $N = 16$, high ERN/Ne group $N = 16$) were formed based on the median Δ ERN/Ne amplitude ($Mdn = -8.25$ μV). The groups did not differ in behavioral outcome (i.e., post-error slowing, reaction time, and number of errors), error-potentiated startle, or time-frequency power in the delta- and theta-band range (all p-values > .10). However, significant correlations between ΔERN/Ne magnitude and error-potentiated startle ($r = -.60$, $p < .05$), post-error slowing ($r = -.60$, $p < .05$), number of errors ($r = .62$, $p < .05$), and reaction time ($r = -.53$, $p < .05$) were observed in the high ERN/Ne group only. In the low ERN/Ne subjects, Δ ERN/Ne magnitude was not correlated with any of these measures (error-potentiated startle: $r = -.03$, $p = .92$; post-error slowing: $r = .21$, $p = .43$; number of errors: $r = .01$, $p = .97$; reaction time: $r = .35$, $p = .18$).[3] The scatter plots for the correlations between Δ ERN/Ne and error-potentiated startle and behavior in each

[2] Stimulus-related conflict evident in the enhancement in N2 (i.e., incompatible minus compatible) was not significantly associated with error-related brain activity (ΔERN/Ne: $r = .19$, $p = .29$, ΔTheta: $r = -.10$, $p = .57$, ΔDelta: $r = -.17$, $p = .35$), behavior (reaction time: $r = .33$, $p = .07$; number of errors: $r = .13$, $p = .49$; post-error slowing: $r = -.05$, $p = .79$), or error-potentiated startle ($r = .11$, $p = .55$).

[3] The observed pattern of correlations between ΔERN/Ne and behavior and error-potentiated startle was replicated when controlling for differences in stimulus-related conflict (i.e., difference in N2 between incompatible and compatible trials): high ERN/Ne group: error-potentiated startle: $r = -.59$, $p < .05$; post-error slowing: $r = -.58$, $p < .05$; number of errors: $r = .60$, $p < .05$; reaction time: $r = -.58$, $p < .05$; low ERN/Ne group: error-potentiated startle: $r = -.06$, $p = .83$; post-error slowing: $r = .20$, $p = .47$; number of errors: $r = .02$, $p = .95$; reaction time: $r = .32$, $p = .25$. Furthermore, the observed pattern of correlations also remained unchanged when only incompatible trials were analyzed.

Table 1. Bivariate Pearson correlations between error-related (ΔERN/Ne, ΔTheta, ΔDelta), error-potentiated startle, and indices of task performance

Variable	ΔERN/Ne	ΔDelta	ΔTheta	Error-potentiated startle	Number of errors	RT	post-error slowing
ΔERN/Ne	1						
ΔDelta	−.43*	1					
ΔTheta	−.25	.28	1				
Error-potentiated startle	−.13	.41*	.44*	1			
Number of errors	.39*	−.50**	−.35*	−.13	1		
RT	−.10	.20	−.09	.32	−.06	1	
Post-error slowing	.00	.43*	.13	.36*	−.24	.42*	1

Note. **$p < .01$; *$p < .05$. RT = reaction time.

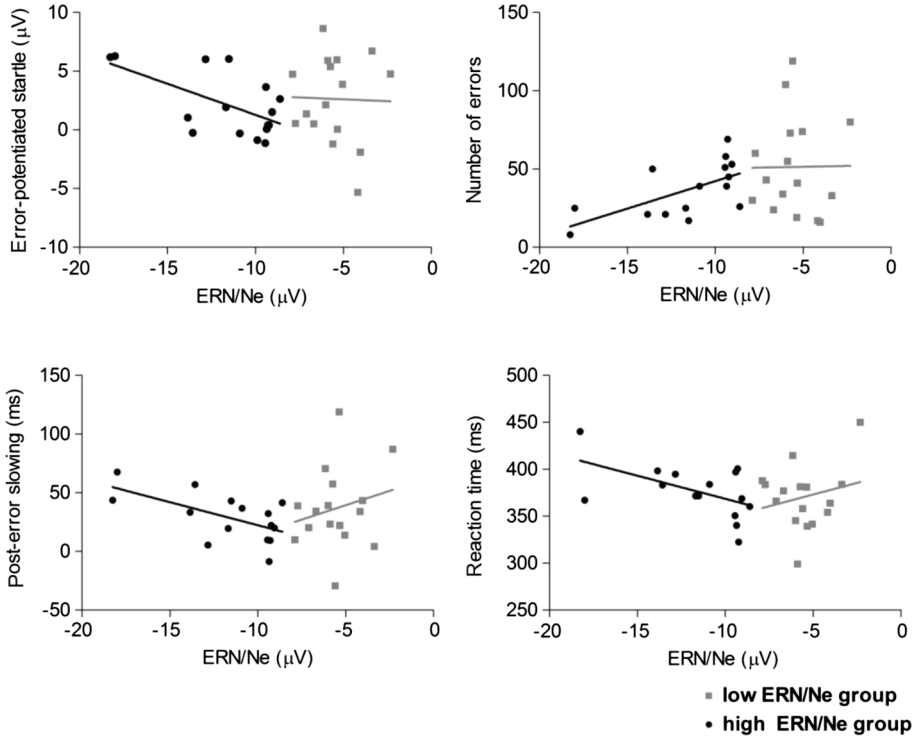

Figure 3. Scatter plots and linear regression lines for the relationship between ΔERN/Ne (averaged across Fz, FCz, and Cz) and error-potentiated startle or behavioral outcome for the low (gray) and the high ERN/Ne group (black).

of these groups are depicted in Figure 3. Note that ΔDelta and ΔTheta are related to error-potentiated startle in the high ERN/Ne group only (high ERN/Ne group: ΔTheta: $r = .55$, $p < .05$, ΔDelta: $r = .60$, $p < .05$, low ERN/Ne group: ΔTheta: $r = .48$, $p = .06$, ΔDelta: $r = .31$, $p = .24$), suggesting that the overall association between error-potentiated startle and delta- and theta-power was particularly driven by individuals with high ERN/Ne amplitudes.

Discussion

The present study investigated the time course of defensive motivation following commission of an error. The relationship between error-potentiated startle and neural correlates of error processing (i.e., ERN/Ne, error-related theta and delta power) was also evaluated. Consistent with previous research, we found startle potentiation following errors (Hajcak & Foti, 2008), further confirming that the commission of errors elicits a cascade of physiological changes reflecting defensive action (Critchley et al., 2005; Hajcak & Foti, 2008; Hajcak et al., 2003b, 2004). This study therefore adds to a growing body of literature that relates error monitoring to motivational variables (Hajcak & Foti, 2008; Luu et al., 2000; Olvet & Hajcak, 2008), and not just the recruitment of cognitive control to improve future performance (Botvinick et al., 2001; Falkenstein et al., 2000; Holroyd & Coles, 2002). Further, the present study extends previous work by demonstrating that increased defensive motivation following errors is sustained for at least

800 ms after responding. As in work that has employed aversive images (e.g., Bradley, Lang, & Cuthbert, 1993), our results indicate that the activation of defensive motivation following aversive events (i.e., errors) persists, suggesting longer-lasting motivational adaptations after errors.

To examine the neural correlates of error processing, the ERN/Ne as well as error-related delta and theta power were evaluated in the present study. In line with previous studies, an increase in power in the delta (Beste, Domschke, et al., 2010; Beste, Kolev, et al., 2010; Yordanova et al., 2004) and theta frequency ranges (Luu & Tucker, 2001; Luu et al., 2004; Trujillo & Allen, 2007) was observed following the commission of an error. In contrast to Yordanova and colleagues (2004), who reported a general response monitoring component in the theta frequency band and a error-specific subcomponent in the delta frequency band, the present results suggest an error-specific increase in both delta and theta power (Kolev et al., 2005). Task-related differences may account for these different results and require further examination.

Recently, we proposed that the magnitude of the ERN/Ne reflects individual differences in defensive reactivity following the commission of errors (Weinberg et al., 2012). And indeed, both the ERN/Ne (Endrass et al., 2008, 2010; Gehring et al., 2000; Hajcak, McDonald, & Simons, 2003a; Johannes et al., 2001; Riesel et al., 2011; Ruchsow et al., 2005; Weinberg et al., 2010) and startle reflex (Grillon, 2002; Kumari, Kaviani, Raven, Gray, & Checkley, 2001; Morgan, Grillon, Southwick, Davis, & Charney, 1995) have been linked to individual differences in defensive reactivity. In the present study, both error-related delta- and theta-band activity were associated with error-potentiated startle, such that an increase in power was associated with a stronger potentiation of startle reflex. This is in line with the results of Hajcak and Foti (2008) who found an association between ERN/Ne and startle potentiation after errors. However, in the present study, a relationship between the ERN/Ne and startle potentiation was only observed in the high ERN/Ne group. Synchronization in the theta band has been suggested as an underlying mechanism of communication and interaction between action monitoring and cognitive control networks (Cavanagh et al., 2009) as well as emotional networks (Pourtois et al., 2010). The present findings suggest that theta and delta activity may also reflect communication related to defensive mobilization following errors.

Despite straightforward predictions from cognitive theories of the ERN/Ne (Botvinick et al., 2001; Gehring et al., 1993; Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004) that an enhanced ERN/Ne might occur in the service of improved performance, there are numerous inconsistencies in the literature examining the relationship between ERN/Ne magnitude and performance measures (see, e.g., Weinberg et al., 2010 for a review). These inconsistencies raise questions about the nature of the relationship between error-related brain activity and adjustments in behavior. The results of this study suggest that variations in error significance and defensive reactivity reflected in ERN/Ne amplitude are a potential moderator that could explain discrepancies between studies. As in Hajcak and Foti (2008), increased ERN/Ne magnitude, as well as increases in theta- and delta-band power, were associated with increased startle magnitude in the group with larger ERN/Ne amplitudes. In the high ERN/Ne group, larger ERN/Ne amplitudes were also associated with a smaller number of errors, longer reaction times, and enhanced post-error slowing. Note that no behavioral differences were present between groups, and that none of the reported relationships were found in the low ERN/Ne group. Thus, although errors prompt adjustment in behavior and motivational disposition overall, a relationship between these measures and the ERN/Ne may be more robust among subjects with higher ERN/Ne amplitudes (Chiu & Deldin, 2007; Endrass et al., 2010; Hajcak et al., 2005; Pailing & Segalowitz, 2004). Nevertheless, these results should be interpreted with caution in light of the small sample size utilized for the exploratory post hoc analysis. Future studies could further examine how relatively large versus small ERN/Ne amplitudes impact other dependent measures using larger sample sizes – and directly address the question of whether differences in error significance modulate the relationship between ERN/Ne and defensive and behavioral adjustments.

It has been suggested that ERPs reflect phasic bursts of brain activity that are time-locked to a stimulus or response (Yeung et al., 2007) or that a reorganization and phase resetting of oscillatory EEG activity time-locked to an event can account for the ERP waveforms (Yeung et al., 2007). It is possible that both processes are reflected in the ERN/Ne, and indeed the moderate relationship between ERN/Ne and theta and delta power, combined with differences in their relationship to post-error adjustment, suggests that these measures of error-related brain activity overlap, but are not redundant measures. Our results suggest that the ERN/Ne had a heterogeneous structure partly composed of co-existing neuroelectric events in the delta and theta frequency ranges (Yordanova et al., 2004).

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