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Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity

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Abstract In the past 20 years, research concerning the error-related negativity (ERN), a negative-going deflection in the event-related brain potential (ERP) following an erroneous response, has flourished. Despite a substantial body of research, debate regarding its functional significance persists. In what follows, we selectively review literature on the ERN, and outline several prominent cognitive theories related to the generation and significance of the ERN. Cognitive theories predict that the size of the ERN should relate to variation in behavior, although there is substantial evidence that the ERN and behavioral measures are at least partially dissociable. Moreover, individual difference measures, psychopathology, and motivational factors all appear to impact basic mechanisms that generate the ERN to moderate the magnitude of the ERN, suggesting a need to integrate alternative perspectives into models of ERN amplitude. Insofar as errors prompt the mobilization of defensive responses, we view variation in the ERN in terms of error detection in the service of protecting the organism. Based on data indicating that the ERN is highly stable over time, heritable, and related to broad dimensions of personality, we propose that the ERN is a neural index of a neurobehavioral trait and variation in its amplitude is related in part to individual differences in defensive reactivity. Implications and future directions are considered.

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Introduction

Errors are motivationally-salient events. In extreme cases, errors may threaten an individual's safety; in most cases, errors require attention and corrective action (Hajcak and Foti, 2008; Hajcak et al. 2004; Hajcak et al. 2005a, b; Olvet and Hajcak, 2008). Before an adaptive response to errors can be mounted, however, an organism must identify the discrepancy between intended and actual behavior (Falkenstein et al. 2000; Frank et al. 2005; Hajcak and Simons, 2008; Holroyd and Coles, 2002). A unique neural response to the commission of errors was reported almost 20 years ago in two different labs, referred to as the error-related negativity (ERN; Gehring et al. 1993) and negativity associated with errors (Ne; Falkenstein et al. 1991). Because the ERN appears to index the activity of an executive system that monitors actions and triggers behavioral adjustment, interest in it has flourished: advances have been made in understanding its function, neuroanatomical origins, and relationship to personality dimensions and multiple psychiatric disorders.

Yet, two decades later there is ongoing debate regarding the precise functional significance of the ERN. Several cognitive theories have been formulated, premised on the notion that the ERN indexes an action monitoring system that functions to modify behaviors—both immediately and over the longer-term—and a wealth of evidence suggests that behavioral changes may relate to this error signal. However, there are also considerable inconsistencies in research surrounding the association between the ERN and

behavioral measures. Moreover, there is ample evidence that variation in the ERN relates to affective, motivational, and individual difference variables, as well as performance measures and behavioral adjustment, suggesting that theories of the ERN should account for motivational/affective processes. In what follows, we selectively discuss the current state of research surrounding the ERN, as well as our view regarding its functional significance. We will begin with a discussion of general mechanisms that give rise to the ERN, followed by theoretical models of these mechanisms; however, our interest here is primarily in characteristics that moderate the ERN across contexts and individuals.

Error-related negativity

The ERN presents as a response-locked negative deflection in the ERP resulting from the commission of an error. Typical response-locked ERPs for error and correct trials, along with a scalp distribution of the error response in the time-range of the ERN, are presented in Fig. 1. The ERN has a frontocentrally-maximal distribution, and peaks about 50 ms following erroneous responses. It has been observed across various levels of task difficulty (Falkenstein et al. 2000; Hoffmann and Falkenstein, 2010; Johannes et al. 2002; Mathewson et al. 2005; Pailing and Segalowitz, 2004a; Themanson et al. 2006), as well as across stimulus (e.g., Falkenstein et al. 1991) and response modalities (Bernstein et al. 1995; Holroyd et al. 1998; Nieuwenhuis et al. 2001; Van't Ent and Apkarian, 1999), and as such has been thought to represent the activity of a response-monitoring system that is generic and modality nonspecific.

Consistent with maturational trajectories for executive control, developmental variation in the ERN has been noted (Davies et al. 2004; Meyer et al. 2011; Nieuwenhuis et al. 2002; Pontifex et al. 2010; Segalowitz et al. 2010), although the ERN has been observed across the lifespan, in children as young as 5 years old (Davies et al. 2004; Torpey et al. 2009), and adults as old as 80 (Nieuwenhuis et al. 2002). Additionally, the ERN appears to be a remarkably robust signal—within a task, as few as six errors may yield a reliable estimate of the ERN (Olvet and Hajcak, 2009c; Pontifex et al. 2010); across time, excellent test-re-test reliability estimates have been observed after several weeks (Olvet and Hajcak, 2009b; Segalowitz et al. 2010), and even up to 2 years (Weinberg and Hajcak 2011). This is in contrast to performance measures (i.e., accuracy, reaction time), which show lower reliability estimates over time (Olvet and Hajcak, 2009b; Weinberg and Hajcak 2011). Furthermore, the ERN demonstrates substantial heritability, ranging between 45 and 60% (Anokhin et al. 2008).

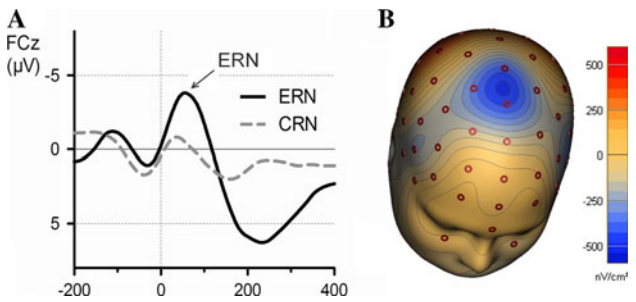

Fig. 1 **a** Response-locked ERP waveforms recorded from an arrow flankers task at FCz in 30 healthy individuals, used in a previous study (Riesel et al. 2011a) comparing correct and error trial waveforms. Response onset occurred at 0 ms and negative is plotted up. **b** Scalp topographies representing the error-related negativity (ERN). The map is derived from the average waveform for error trials (current source density, latency 66 ms) in the same healthy individuals

Correct-response negativity

Although it is sometimes assumed that errors and correct responses reflect a binary differentiation, this may not be the case. Partial errors, in which subjects initiate an incorrect response but self-correct before errors are committed, have also been shown to generate ERNs that are on par with uncorrected errors in terms of their magnitude (Burle et al. 2008; Carbonnell and Falkenstein, 2006; Gehring et al. 1993; Masaki and Segalowitz, 2004; Scheffers et al. 1996). Additionally, correct responses frequently elicit a small ERN-like component (Falkenstein et al. 2000; Ford, 1999; Scheffers and Coles, 2000; Vidal et al. 2000) called the Correct Response Negativity (CRN; Ford, 1999; See Fig. 1).

Stimuli that might activate incompatible response tendencies (e.g., stimuli from a flankers task when the central arrow points in a different direction from the surrounding arrows: "< < > < <"), appear to elicit a larger CRN than congruent stimuli (Bartholow et al. 2005). However, in the same study, the CRN was largest when the observed trial type violated subjects' expectations (i.e., when an incompatible trial occurred when a compatible trial was expected). Additionally, the magnitude of the CRN appears to be inversely related to the probability of committing an error (Allain et al. 2004), such that a larger CRN may act as a kind of "prophylactic" against errors (Simons, 2010). Collectively, this work suggests that the magnitude of the CRN may index the degree of engagement of response monitoring on correct trials. Similarities between the CRN and ERN in terms of their topography, time-course, and assumed source (Hoffmann and Falkenstein, 2010; Luu and Tucker, 2001; Roger et al. 2010; Vidal et al. 2000) have led many to argue that the two components reflect overlapping

or even identical neural and cognitive processes during response monitoring that are enhanced on error trials.

Proposed neuroanatomical substrates

Source localization (Holroyd et al. 1998; Pizzagalli et al. 2006) magnetoencephalography (Miltner et al. 2003), time–frequency analyses (Luu et al. 2004; Trujillo and Allen, 2007), and intracerebral recording (Brázdil et al. 2005) suggest that the ERN is generated in the anterior cingulate cortex (ACC), a part of the frontostriatal system, which also encompasses the orbitofrontal cortex (OFC) and areas of the basal ganglia (BG). Likewise, single-unit recording studies show increased error-related potentials in the ACC in monkeys (Gemba et al. 1986; Ito et al. 2003), and evidence from human lesion studies indicates that patients with ACC lesions have diminished ERNs (Stemmer et al. 2004).

The ACC has interconnections to both limbic and prefrontal areas, and responds to *both* cognitive conflict and aversive affective information (Bush et al. 2000). For example, ACC activation is reliably observed in situations involving response conflict, negative feedback, pain, and errors; based on these data, it has been suggested that the ERN may reflect the integration of cognitive and affective processes during error detection (Hajcak et al. 2008; Hajcak et al. 2005a, b; Luu et al. 2000a, b; Luu et al. 2003; Tucker et al. 2003). The ACC comprises two functional subdivisions: the rostral and dorsal subdivisions of the ACC have been linked to affective and cognitive processing, respectively (Bush et al. 2000). Although some fMRI and source localization studies have implicated the dorsal ACC as the principal generator of the ERN, consistent with the notion that the ERN may be linked to more cognitive processes, other studies have instead found the ERN is generated in the rostral, affective portion of the ACC (Ridderinkhof et al. 2004; Taylor et al. 2007). Emerging evidence further suggests that there may be important individual differences associated with anxiety in recruitment of these functional subdivisions of the ACC (Aarts and Pourtois, 2010).

Although the bulk of existing data implicates the ACC as the primary neural generator of the ERN (Brázdil et al. 2005; Carter et al. 1998; Debener et al. 2005; Holroyd et al. 1998; Miltner et al. 2003; Ridderinkhof et al. 2004; van Veen and Carter, 2002), there is now accumulating evidence that the lateral prefrontal cortex also plays a critical role in the error-monitoring network (Carter et al. 1998; Gehring and Knight, 2000; Kiehl et al. 2000; Turken and Swick, 2008; Ullsperger and von Cramen, 2006a). And indeed, the ability to process errors is contingent upon active maintenance of internal representations of task instructions and goals—operations that depend critically on structures such as the PFC. Combined with the proposed role of dopamine, discussed below, this suggests that it may be important in the future to consider the role of multiple brain regions working in concert towards the generation of the ERN, particularly in research concerned with individual differences. Variability in the ERN might reflect differential patterns of response across multiple, diverse brain regions.

The ACC is also richly innervated by dopaminergic neurons (Allman et al. 2001). Dopamine (DA) has been related broadly to behavioral facilitation and stimulus salience (Allman et al. 2001; Depue and Iacono, 1989), but most empirical investigations have focused on the relationship of DA and reward. Consistent with reward-based views of DA, a prominent model of the ERN suggests that the ERN reflects dopaminergic disinhibition of neurons in the ACC when events are evaluated as worse than anticipated (Holroyd and Coles 2002).

Ongoing discussions regarding the functional significance of the ERN will likely be informed through improved understanding of the role of multiple neurotransmitter systems and neural regions, as well as the role of specific subdivisions of the ACC, in generating error-related brain activity. Based on current knowledge, several computational models of the ERN have been formulated that have generated testable hypotheses—each has provided significant insight regarding the function of the ERN. Below, we will discuss three cognitively-oriented theories, followed by our own view of the ERN and potential meaning of variation in its amplitude.

Mismatch theory

Among the earliest attempts to explain the ERN was the Mismatch Theory, which posits a comparator system that evaluates the mental representations of the correct and actual response; mismatch between these two representations was hypothesized to elicit an ERN (e.g., Bernstein et al. 1995; Coles et al. 2001; Falkenstein et al. 1991). According to the Mismatch Theory, errors arise as a result of impulsive responses based on incomplete stimulus processing. Consistent with this view, errors occur much more quickly than correct responses. Furthermore, the *degree* of mismatch between the actual and correct response (e.g., response with an incorrect hand compared to response with an incorrect finger) appears related to the magnitude of the ERN (Bernstein et al. 1995; Falkenstein et al. 1991; however, see Gehring and Fencsik 2001).

Additionally, the impact of mismatch ought to be more potent when the mental representation of the executed response is stronger; consistent with this view, the magnitude of the ERN is enhanced when participants are more

confident in having made an error, regardless of whether or not they actually made a mistake (Scheffers and Coles, 2000). Functionally, this mismatch signal is thought to form a part of an internal feedback loop, by which errors may be remediated quickly in the short term (as in partial errors), but also by which behavior may be shaped over the long term in order to reduce the likelihood of future errors (e.g., Coles et al. 2001; Gehring et al. 1993). In fact, *correct* trials following error trials are characterized by dramatic response time slowing, suggesting that the response monitoring system utilizes error detection to reassert control over behavior (Allain et al. 2009; Rabbitt 1966).

However, observations of the CRN—even on compatible trials in which no inconsistency between actual and intended response should exist—have posed a challenge to the Mismatch Theory, and ultimately led to adaptation. For instance, Falkenstein and colleagues suggest that the ERN and CRN reflect the comparison process *itself* (i.e., response checking), rather than its outcome, and that the ERN signals this comparison process plus an additional error signal that may be unique to error trials (Falkenstein et al. 2000).

Conflict monitoring theory

Another prominent model of the ERN is rooted in theories of response conflict, which focus on the competition between possible responses; for instance, when a desired response must compete with and overcome a strong but contrary response tendency. Behaviorally, response conflict on error trials is evident in that participants often correct their errors, even when uninstructed to do so (Fiehler et al. 2005; Ullsperger and von Cramen 2006b). Conflict Monitoring models further suggest that the co-activation of the error- and error-correcting response can be observed at the level of the scalp-recorded ERP, and produces the ERN. For the Conflict Monitoring theory then, the ERN is taken to index increased response conflict on error trials in the response-locked ERP (Botvinick et al. 1999; Carter et al. 1998; Yeung et al. 2004). On correct trials with increased response conflict (e.g., "< < > < <"), conflict is resolved prior to the execution of a response, and is thought to give rise to the stimulus-locked N2 (Carter and van Veen 2007; Yeung et al. 2004).

The conflict theory further hypothesizes that it is the ACC that functions to monitor conflict between these simultaneously activated response channels (Carter and van Veen 2007; Yeung et al. 2004), and that when the ACC detects high levels of conflict, projections to the prefrontal cortex signal the need for increased cognitive control (Botvinick et al. 2001; Carter et al. 1998; Kerns et al. 2004; Yeung et al. 2004). In support of this view, ACC activity following errors has been associated with both dorsolateral prefrontal cortex (dlPFC) activity on subsequent trials and post-error slowing (Kerns et al. 2004; van Veen and Carter 2006). Additionally, neuroimaging studies demonstrate that the ACC is active on both errors trials and on correct trials with incongruent stimuli that elicit high levels of response conflict (Botvinick et al. 1999; Carter et al. 1998; MacDonald et al. 2000).

Related to this, there are notable similarities between the ERN and the stimulus-locked N2. The magnitude of the N2 is enhanced on incongruent trials (Kopp et al. 1996), and the ERN and N2 are morphologically similar and may share a common neural generator in the ACC (Kopp et al. 1996; van Veen and Carter, 2002; Yeung et al. 2004; however, see Mathalon et al. 2003). An attractive feature of the Conflict Monitoring model, then, is that it accounts for the presence of both the stimulus-locked N2 on incompatible correct trials and the response-locked ERN on error trials.

In addition, though early iterations of Conflict Monitoring theory did not account for the CRN, subsequent evidence that larger CRNs are associated with incongruent trial types (Bartholow et al. 2005) suggests that conflict can occur at multiple points in the information processing stream, including stimulus processing, response monitoring, and even conceptual processing (Carter and van Veen, 2007). Based on these findings, Conflict Monitoring theorists have proposed that there is no dedicated error monitoring system, and instead that errors represent one instance of increased conflict.

There are important differences between the Conflict and Mismatch theories of the ERN. As discussed, Conflict Monitoring theories suggest that the ERN represents just one example of ongoing conflict monitoring; the Mismatch Theory instead suggests a dedicated response checking process. These theories also have different explanations for the occurrence of the CRN. In addition, neither Conflict Monitoring nor Mismatch Theory account for the observation of a medial-frontal negativity following negative performance feedback. This feedback-related negativity (fERN, FN, FRN: hereafter referred to as the FN) is an apparent N2-like stimulus-locked component elicited when feedback indicates that outcomes are worse than anticipated (e.g., stimuli indicating monetary loss in simple gambling tasks; Foti and Hajcak 2009; Gehring and Willoughby 2002; Miltner et al. 1997).

The FN peaks approximately 250–300 ms after feedback presentation and is maximal at frontocentral recording sites (Holroyd and Coles 2002; Miltner et al. 1997). The FN appears similar to the response-locked ERN in terms of topography and neural generator (Miltner et al. 1997). Yet, because the FN occurs substantially later than response selection processes, it is not easily explained by either

Conflict Monitoring or Mismatch theories. However, it is possible that the response monitoring system produces error signals based on both internal and external information. Reinforcement Learning theories, discussed immediately below, may provide a cohesive explanation for both the ERN and the FN.

Reinforcement learning

Mismatch theories of the ERN were expanded upon and formalized by theorists interested in the specific process and mechanisms by which error detection might influence behavior, both in the short- and long-term. According to the Reinforcement Learning theory of the ERN (RL-ERN; Holroyd and Coles 2002), behavior is shaped through ongoing performance evaluation and feedback, consistent with reinforcement learning principles, such that the motor system is trained through reward and punishment. In the RL-ERN model, the ACC receives ongoing feedback via dopaminergic projections from the basal ganglia, and uses this signal to adapt the response selection process. In this view, the ACC and basal ganglia function much like actor and critic components of learning models (e.g., O'Doherty et al. 2004). The RL-ERN theory is rooted in non-human animal work indicating that the basal ganglia monitor both external feedback and internal evaluation of responses (for a review, see: Houk et al. 1995; Schultz 2002). When outcomes are better or worse than expected, the basal ganglia induce an increase or decrease in phasic midbrain DA activity, respectively. The ERN and FN, therefore, are both thought to reflect a reward-prediction error signal. In support of the RL-ERN hypothesis, there is evidence that DA agonists enhance the ERN (de Bruijn et al. 2004), while DA antagonists attenuate it (de Bruijn et al. 2004; Zirnheld et al. 2004).

Though the RL-ERN theory does not account for the observed stimulus-locked N2, a major strength of this theory is that it integrates literature concerning the performance-related ERN and the feedback-locked FN (Holroyd and Coles 2002; Holroyd et al. 2005). The topographical and morphological similarities between the FN and ERN components (Miltner et al. 1997) have therefore been presented as further evidence in support of a reward- and learning-based model of the ERN. This model contends that error monitoring occurs within a system which utilizes stimulus–response mappings to assign a hedonic value (i.e., good or bad) to ongoing events. The FN and ERN are both thought to reflect the activity of this system in trial-and-error learning tasks—the former reflecting internalization of external feedback and the latter reflecting an entirely internal feedback loop. The RL-ERN theory therefore predicts that, as this system begins to learn appropriate stimulus–response mappings, the magnitude of the FN and ERN will have an inverse relationship. And in fact there is evidence from reinforcement learning tasks that, as the magnitude of the ERN increases, the FN decreases (Holroyd and Coles 2002; Nieuwenhuis et al. 2002).

Although both the RL-ERN and Conflict Monitoring theories of the ERN elegantly explain the occurrence of the ERN by emphasizing specific cognitive functions that are thought to give rise to the ERN, *neither* adequately addresses the substantial individual differences that have been observed in the magnitude of the ERN. More specifically, both the RL-ERN and conflict models imply that variation in the magnitude of the ERN is predicted by behavioral measures: more infrequent errors will give rise to an increased ERN according to both models (Holroyd and Coles 2002; Holroyd et al. 2005; Yeung et al. 2004), and the degree of post-error slowing should be related to the magnitude of the ERN (Holroyd et al. 2005; Yeung et al. 2004). In short, both RL-ERN and conflict theories are predicated upon the notion that variation in the *magnitude* of the ERN is related to current behavior, and is utilized to shape subsequent behaviors. Yet, as we discuss below, there are multiple instances in which variation in the ERN occurs in the absence of behavioral differences, suggesting additional sources of variation in the magnitude of the ERN may exist and should be considered.

ERN and behavior

As we have noted, there is accumulating data to suggest at least a partial dissociation between ERN magnitude and behavioral measures. For example, though some studies have demonstrated larger ERNs associated with lower error rates (Holroyd and Coles 2002; Pailing and Segalowitz 2004a; Pieters et al. 2007; Santesso et al. 2005), others have reported no relationship between error rate and ERN amplitude (Falkenstein et al. 2000; Masaki et al. 2007; Weinberg et al. 2010). In addition, differences in accuracy rates have been observed between the same task either with or without performance feedback, although an equivalent ERN was observed in both conditions in the same subjects (Olvet and Hajcak 2009a).

Moreover, consistent with the notion that error detection might shape future behavior, reaction times are slower following errors; this post-error slowing is thought to reflect a compensatory action to enhance post-error performance (Rabbitt 1966). Conflict Monitoring theories specifically predict that the magnitude of the ERN should relate to this post-error slowing (Botvinick et al. 2001); the logic of Mismatch and RL-ERN theories should also predict behavioral adjustments after an error. Although some

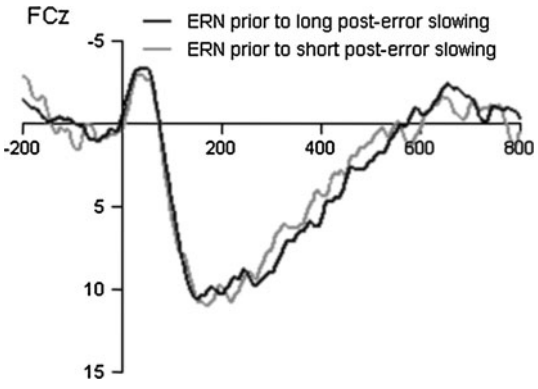

Fig. 2 Response-locked ERP waveforms recorded at FCz demonstrating the relationship between the ERN and post-error slowing in 16 subjects. For each subject, the median post-error correct reaction time was calculated. Based on this, two new error averages were created for each subject; one for errors preceding correct reaction times falling *below* that individual's median (fast responses), and one for errors preceding correct reaction times falling *above* that individual's median (slow responses). As suggested by the figure, the magnitude of the ERN did not predict the degree of post-error slowing ($t(15) = .84$, $p > .05$)

studies have indicated that the magnitude of the ERN is associated with post-error slowing (Compton et al. 2008; Debener et al. 2005; Gehring et al. 1993; Kerns et al. 2005; Scheffers et al. 1996; South et al. 2010), many others have failed to find such an association (Endrass et al. 2007; Gehring and Fencsik 2001; Gehring and Knight 2000; Hajcak et al. 2003b; Hajcak and Simons, 2002; Luu et al. 2000a, b; Riesel et al. 2011a; Scheffers et al. 1999). A dissociation between the ERN and post-error slowing is further supported by studies that demonstrate group differences in the magnitude of the ERN unaccompanied by group differences in post-error slowing (Endrass et al. 2010; Hajcak et al. 2008; Hajcak et al. 2003a; Mathalon et al. 2002; Riesel et al. 2011a; Weinberg et al. 2010); likewise, some studies find differences in post-error slowing in the absence of ERN differences (Endrass et al. 2007; Hajcak and Simons, 2008; Nieuwenhuis et al. 2001).

A number of pharmacological challenges also impact the magnitude of the ERN without any impact on the degree of post-error slowing (e.g. de Bruijn et al. 2004; Riba et al. 2005; Tieges et al. 2004; Zirnheld et al. 2004). Additionally, errors following errors elicit a normal ERN, but are associated with reduced post-error slowing (Hajcak and Simons 2008). Finally, although errors committed outside of awareness elicit intact ERNs, post-error compensatory slowing following such errors is reduced (Endrass et al. 2007; Nieuwenhuis et al. 2001), suggesting further instances in which a robust ERN signal may occur in the absence of accompanying behavioral changes.

In short, the relationship between the ERN and behavioral measures is far from clear, despite straightforward predictions from cognitive theories such as the RL-ERN and Conflict Monitoring models. However, it is possible that a relationship between ERN and behavior only exists within subjects, not between subjects. Relatively few studies have investigated an intra-individual coupling of ERN magnitude and behavioral measures (e.g., Debener et al. 2005; Gehring et al. 1993). In order to examine this, we reanalyzed data from 16 healthy control participants used in a previous study (Weinberg et al. 2010). Specifically, we analyzed the ERN for trials that preceded relatively more or less post-error slowing based on individual subjects' median post-error RT. In contrast to previous studies (Debener et al. 2005; Gehring et al. 1993), ERN amplitudes did not differentiate trials that were followed by greater post-error slowing (Fig. 2). Altogether, whereas cognitive theories explain basic processes leading to the generation of the ERN, the inconsistent relationship between the ERN and behavioral measures raises important questions about the functional significance of *variation* in the amplitude of the ERN. We believe that a significant portion of this variability is related to dispositional characteristics that moderate the ERN.

Motivational orientation, psychopathology, and the ERN

We have suggested that errors are motivationally-salient events that may convey more than just the need to increase cognitive control. Indeed, errors prompt a cascade of physiological changes that suggest defensive motivational response in preparation for action, including skin conductance response, heart rate deceleration (Hajcak et al. 2003b, 2004), potentiated defensive startle reflexes (Hajcak and Foti, 2008; Riesel et al. 2011b) and pupil dilation (Critchley et al. 2005). A recent study utilizing intracranial recordings also reported amygdala activity following errors (Pourtois et al. 2010).

The ERN also appears sensitive to the motivational salience of errors (e.g., Amodio et al. 2004, 2008a, b): the magnitude of the ERN is enhanced by manipulations that make errors more salient, either through incentives (Chiu and Deldin 2007; Endrass et al. 2010; Ganushchak and Schiller 2008; Hajcak et al. 2005a, b; Pailing and Segalowitz 2004b), task instructions that emphasize accuracy over speed (Falkenstein et al. 2000; Gehring et al. 1993), or external performance evaluation (Hajcak et al. 2005a, b; Kim et al. 2005).

In addition, there is a growing body of research indicating the ERN is enhanced among individuals who experience errors as abnormally salient and aversive. For example, an increased ERN has been observed for patients with obsessive–compulsive disorder (OCD; Endrass et al. 2008; Endrass et al. 2010; Gehring et al. 2000; Hajcak et al. 2008; Johannes et al. 2001; Riesel et al. 2011a; Ruchsow et al. 2005a, b; however, see also Nieuwenhuis et al. 2005),

depression (Chiu and Deldin 2007; Holmes and Pizzagalli 2008; Holmes and Pizzagalli, 2010; however, see also Olvet et al. 2010; Ruchsow et al. 2006a, b; Ruchsow et al. 2004; Schrijvers et al. 2009; Schrijvers et al. 2008) and generalized anxiety disorder (GAD; Weinberg et al. 2010; Xiao et al. 2010). Figure 3 presents an overview of ERN variation across a number of psychiatric disorders.

Moreover, healthy individuals with obsessive–compulsive characteristics (Grundler et al. 2009; Hajcak and Simons 2002; Santesso et al. 2006), high trait levels of anxiety (Hajcak et al. 2003a; Meyer et al. 2011; Pourtois et al. 2010), high negative affect (Hajcak et al. 2004; Luu et al. 2000a, b) and increased scores on the Behavioral Inhibition System scale (Amodio et al. 2008a, b; Boksem et al. 2006) are also characterized by increased ERN amplitudes. Finally, there is evidence that motivational manipulations that impact the magnitude of the ERN may be moderated by personality traits or clinical status (Amodio et al. 2008a, b; Dikman and Allen 2000; Endrass et al. 2010; Luu et al. 2000a, b; Olvet and Hajcak 2011; Pailing and Segalowitz 2004b).

For example, there is recent evidence that the relationship between the ERN and the impact of a sad film clip on mood was moderated by trait neuroticism: individuals with high trait levels of neuroticism demonstrated a stronger coupling between sadness following the film clip and the magnitude of the ERN (Olvet and Hajcak 2011). Another recent study demonstrated that the ERN was enhanced when errors were punished by monetary loss—but only among healthy controls participants; monetary penalty had no impact on the already-enhanced ERN among OCD patients (Endrass, et al. 2010). Similarly, punishment of errors appears to elicit a larger ERN, both in a learning and an extinction period (Riesel et al. 2011c); moreover, highly trait anxious individuals appear to be characterized by larger punishment-related modulations of the ERN (Riesel et al. 2011c). Thus, both stable individual differences and situation-specific variation in motivational factors seem to modulate the ERN; moreover, these factors may interact to influence the ERN.

Because cognitive theories of the ERN assert that this error signal relates to behavior, one might expect that this exaggerated processing of errors would be related to increased behavioral regulation; that is, groups of individuals with larger ERNs should be characterized by fewer errors, decreased reaction times suggesting better performance, or enhanced post-error slowing or accuracy. Surprisingly, only two of the above-cited studies that report increased error-related brain activity as a function of internalizing psychopathology also report behavioral differences between groups (Riesel et al. 2011a; Schrijvers et al. 2009). However, of these two, one study continued to find ERN differences between groups after controlling for behavioral differences (Riesel et al. 2011a).

In contrast to studies documenting an increased ERN in relation to anxious traits and psychopathology, *reduced* ERN amplitudes have been associated with schizophrenia (Alain et al. 2002; Bates et al. 2002; Bates et al. 2004; Foti et al., in revision; Mathalon et al. 2002; Mathalon et al. 2009; Morris et al. 2008; Morris et al. 2006), substance abuse (Franken et al. 2007; however, see Schellekens et al. 2010), ADHD (Albrecht et al. 2008; Groen et al. 2008; Herrmann et al. 2010; Liotti et al. 2005; van Meel et al. 2007; however, see Burgio-Murphy et al. 2007; Jonkman et al. 2007; Van De Voorde et al. 2010; Wiersema et al. 2005; Zhang et al. 2009), autism (Henderson et al. 2006; Sokhadze et al. 2010; South et al. 2010; Vlamings et al. 2008; however, see Groen et al. 2008), psychopathy (Munro et al. 2007; von Borries et al. 2010; however, see Brazil et al. 2009) and borderline personality disorder (de Bruijn et al. 2006; Ruchsow et al.

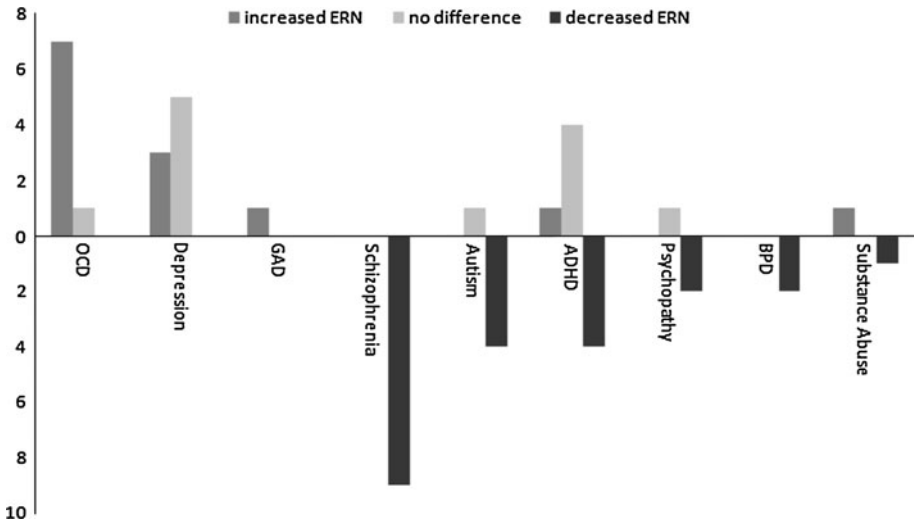

Fig. 3 A *bar graph* representing the findings of multiple studies examining the ERN in relation to psychopathology. The *Y-axis* represents the number of studies demonstrating either an enhanced ERN in that group (compared to healthy controls), no difference in the magnitude of the ERN, or a reduced ERN. Note that the ERN is most well-characterized and consistent in OCD and schizophrenia

2006a, b). Figure 3 presents an overview of ERN variation across these disorders.

Decreased ERN magnitude has also been reported among individuals high in trait levels of impulsivity (Potts et al. 2006; Ruchsow et al. 2005a, b), disinhibitory personality traits (Dikman and Allen 2000) and broad externalizing traits (Hall et al. 2007). Likewise, consumption of alcohol appears to result in a reduced ERN (Easdon et al. 2005; Ridderinkhof et al. 2002). Each of these groups and manipulations that relate to a reduced ERN have also been characterized by behavioral deficits. Indeed, the majority of the reported studies associating a *decreased* ERN amplitude with psychopathology also reveal poorer behavioral performance in these groups (Alain et al. 2002; Bates et al. 2002; Bates et al. 2004; de Bruijn et al. 2006; Franken et al. 2007; Henderson et al. 2006; Herrmann et al. 2010; Jonkman et al. 2007; Liotti et al. 2005; Mathalon et al. 2002; Mathalon et al. 2009; Morris et al. 2006; Schellekens et al. 2010; South et al. 2010; van Meel et al. 2007; Vlamings et al. 2008; von Borries et al. 2010; however, see also Dikman and Allen 2000; Hall et al. 2007).

Nevertheless, the source of this variation in both behavior and the magnitude of the ERN is far from clear; one possibility is that the reduced ERN is directly linked to poorer performance in these groups. However, another possibility is that the two are only indirectly associated, and are both driven by other factors. For instance, the reduced ERN among individuals with schizophrenia potentially reflects neurobehavioral variation in inhibitory control (Patrick and Bernat 2010) or differences in defensive mobilization following errors (Barch 2005; Braff et al. 1992; Volz et al. 2003). An alternative explanation for the attenuated ERN might be on-line processing impairments associated with fronto-cortical dysfunction, a deficit frequently observed in individuals exhibiting disinhibitory disorders and traits (e.g., Davidson et al. 2000; Dinn and Harris 2000; Morgan and Lilienfeld 2000). This possibility is consistent with evidence that the magnitude of the ERN is reduced in patients with lesions of the frontal cortex (e.g., Turken and Swick 2008; Ullsperger and von Cramen 2006a).

It is also possible that the reduced ERN observed in relation to externalizing traits and psychopathology reflects the motivational disengagement, disinhibition, and decreased conscientiousness often observed in these groups (Gard et al. 2007; Gurrera et al. 2000; Krueger and Markon 2006; Nigg et al. 2002). Decreases in the motivational salience of errors, along with task disengagement and fronto-cortical dysfunction, could thereby account for both the smaller ERN and poorer performance in these patient groups. Conversely, the increased ERN observed in anxiety disorders may reflect features common to these disorders, such as perfectionism (Frost and Steketee 1997; Kendall et al. 2004), excessive concern over errors (Coles et al. 2003; Pitman 1987), negative affect (Hajcak et al. 2003a; Mineka et al. 1998) and increased intolerance of uncertainty (Holaway et al. 2006; Tolin et al. 2003) which could relate to heightened motivational salience or hedonic value of an error for these groups and individuals. In our view, these diverse explanations for variability in the ERN (e.g., task engagement, fronto-cortical dysfunction, conscientiousness, motivational salience) are neither incompatible nor mutually exclusive. Instead, they suggest that distinctive dispositional factors may contribute in contrasting ways to the amplitude of the ERN—perhaps via differential activation of multiple components of the neural circuitry that underlies the ERN.

Taken together, these results suggest that, although general mechanisms responsible for the generation of the ERN may be similar across subjects, the *magnitude* of the ERN is moderated by both situational affective and motivational processes as well as by more stable emotional and motivational characteristics and individual differences—and that these may interact to determine the amplitude of the ERN. Overall then, motivational deficits and poor task engagement seem to be linked to a reduction in the ERN, whereas the increased motivational salience of errors seems to be associated with a larger ERN.

The ERN as a neural indicator of the neurobehavioral trait of defensive reactivity

Thus far we have argued that the ERN may reflect the activation of a response monitoring system that is highly responsive to the value of errors based on local context, personality, and learning history. Based on both the role of motivational factors in determining the ERN, and significant variation in the ERN as a function of individual differences, some have speculated that the ERN reflects an affective or emotional response to errors (Pailing and Segalowitz 2004b; Vidal et al. 2000). While we believe that cognitive theories too-frequently overlook the moderating impact of affective/motivational variables on the magnitude of the ERN, still we hesitate to say that *any* single physiological response *is* an emotional response, per se. Our perspective comes from the tradition that emotion constitutes a set of responses rooted in motivational tendencies, which broadly support approach- and withdrawal-related behaviors (Bradley 2000; Bradley et al. 2001; Cuthbert et al. 2000; Lang et al. 1997). Engagement of motivational systems depends on appraisal, occurring at multiple levels, that assesses the relevance of stimuli to survival needs and current goals (e.g., Lang and Davis 2006); mobilization of motivational systems is evident across multiple response systems (e.g., changes in heart rate, skin conductance, amygdala response, self-report). The holistic emotional response is then constructed across

response systems. For instance, changes in heart rate can be involved in both sexual pleasure and anger; this single measure may indicate a response to an emotional stimulus but does not, on its own, *constitute* 'an emotional response'.

We suppose that errors, like aversive visual stimuli, can signal a potential threat to the organism and its goals. A host of evidence suggests that threatening stimuli activate defensive motivational systems in the brain, which leads to physiological and neural changes that support increased attention, perception, and readiness for action (Bradley 2000; Bradley et al. 2001; Cuthbert et al. 2000; Lang et al. 1997). Whereas previous theories and models of error monitoring have discussed adaptive responses to errors in terms of behavioral adjustments in post-error slowing and subsequently improved performance (Holroyd and Coles 2002; Holroyd et al. 2005), we suggest that an adaptive response to errors also involves mobilizing defensive motivational systems. And indeed, as reviewed above, errors prompt a number of changes that indicate defensive mobilization (Critchley et al. 2005; Hajcak and Foti 2008; Hajcak et al. 2003b, 2004).

In our view, then, the ERN is not in and of itself a valenced neural response to errors. Rather, the ERN reflects the earliest component—i.e., the initial evaluation of the motivational salience of an error—in a dynamic process which rapidly mobilizes defensive systems, as well as additional cognitive processing, and signals the need to respond adaptively. In this view, increased cognitive control evident in behavioral measures would be just one type of adaptive response following error detection. The host of physiological changes following errors signaling the initiation of a defensive response may be another. Moreover, we suppose that error detection precedes the development of cognitive control processes—both ontogenetically and phylogenetically. In this view, the DLPFC develops such that it may increase cognitive control following error monitoring activity of the ACC—but we do not suppose that this is the only reason that error detection occurs in the first place.

Moreover, we would argue that the amplitude of the ERN is not *only* determined by predictions derived from ongoing performance measures, as suggested by cognitive theories of the ERN. Variation in neural and psychophysiological responses to errors may reflect meaningful and stable individual differences in defensive reactivity, much like responses to other threatening stimuli (Foti et al. 2010; Holmes et al. 2008; Lang et al. 2007; MacNamara and Hajcak 2009, 2010; Weinberg and Hajcak 2010). And indeed, though it is clear that changes in the ERN are observed through state-related manipulations (Chiu and Deldin 2007; Endrass et al. 2010; Ganushchak and Schiller 2008; Hajcak et al. 2005a, b; Pailing and Segalowitz 2004b), we believe that a great deal of the variation in the magnitude of the ERN is trait-like (Anokhin et al. 2008; Hajcak et al. 2008; Moser et al. 2005; Olvet and Hajcak 2009b; Riesel et al. 2011a). We would also note that situational and trait variables interact to determine the amplitude of the ERN in a given context (e.g., Endrass et al. 2010; Olvet and Hajcak 2011; Riesel et al. 2011c), suggesting a pathway by which inherited vulnerabilities may be shaped via interactions with stressors in the environment to become dispositional characteristics. That is, though it is far from the *only* trait variable to influence the ERN, we believe that a substantial amount of the variation in the ERN relates to stable individual differences in defensive reactivity—being larger for those individuals characterized by greater response to threat.

For example, hyperactive error signals have been assumed to be related to obsessive–compulsive pathophysiology (Pitman 1987). Enhanced ERN amplitudes have also been observed in unaffected first-degree relatives of patients with OCD (Riesel et al. 2011a). Combined with findings that indicate that the magnitude of the ERN is heritable (Anokhin et al. 2008) and insensitive to symptom reduction in OCD (Hajcak et al. 2008), it is possible that the enhanced ERN in OCD is a potential endophenotype for OCD—reflecting information-processing abnormalities that mediate the pathway between genetic predisposition and disease states (Gottesman and Gould 2003)—and may represent a biomarker of risk for the development of the disorder.

As noted above, however, an enhanced ERN is probably not specific to OCD; rather, an increased ERN is also observed in GAD (Weinberg et al. 2010; Xiao et al. 2010). GAD shares several clinical features with OCD, including enhanced reactivity to errors, negative affect (Hajcak et al. 2003a; Mineka et al. 1998) and increased intolerance of uncertainty (Holaway et al. 2006; Tolin et al. 2003). Furthermore, these disorders are frequently comorbid (e.g., Kessler et al. 2008; Krueger and Markon 2006; Ruscio et al. 2010). These shared symptoms may be reflected in similar underlying neurobiological processes and aberrations (e.g. ERN enhancement); along this line, the enhanced ERN may reflect a shared endophenotype for multiple forms of internalizing psychopathology (Olvet and Hajcak 2008). The specific phenotype to which the ERN most closely corresponds is not yet clear—and this requires larger studies that simultaneously assess multiple disorders and personality traits.

For example, although there is substantial evidence for a relationship between the ERN and some anxiety disorders, the evidence accumulated thus far suggests an absence of a relationship between the fear-based anxiety disorders (e.g., phobias) and the magnitude of the ERN (Hajcak et al. 2003a; Moser et al. 2005). Likewise, there is evidence that variation in the ERN is more closely related to symptoms

of anxious-misery/distress—related to sustained dispositional anxiety—than to symptoms of anxious arousal, which may capture acute fear responses (Moser et al. 2011; Weinberg et al. 2010; Vaidyanathan et al. 2011). Though this body of evidence is still developing, the distinction is consistent with a substantial animal and human literature pointing to a difference between neural circuitry underlying phasic, cue-specific defensive reactivity (i.e., fear) and more persistent, generalized defensive reactivity (i.e., anxiety; e.g., Davis et al. 1997; Grillon and Davis 1997; Rosen and Schulkin 1998; Vaidyanathan et al. 2009). Variability in the ERN appears to be linked more closely to disorders of generalized anxiousness, rather than heightened cue-driven fearfulness. This suggests that the ERN might be useful not only in making distinctions across broad diagnostic categories (i.e., internalizing from externalizing), but also *within* diagnostic categories (i.e., within the anxiety disorder spectrum).

Similarly, depression falls within the internalizing spectrum and is characterized by high lifetime comorbidity with a number of anxiety disorders (Clark 1989). In contrast to the literature in anxiety disorders, however, evidence related to ERN enhancement in depression has been inconsistent (see Fig. 3). Some studies have found increased amplitude of the ERN in clinically depressed populations (Chiu and Deldin 2007; Holmes and Pizzagalli 2008; Tucker et al. 2003), while others have not (Ruchsow et al. 2006a, b; Ruchsow et al. 2004). However, there is emerging evidence that mild-to-moderate levels of depressive symptoms are related to an enhanced ERN (Chiu and Deldin 2007; Compton, et al. 2008), whereas severe depression is associated with an attenuated ERN (Olvet et al. 2010; Schrijvers et al. 2008; Schrijvers et al. 2009). It is possible that depression is characterized by an enhanced ERN only insofar as depression is also characterized by high levels of NA, worry, and anxiety—and that severity of depressive symptoms may moderate this relationship, such that severe depression and the associated motivational disengagement may actually attenuate the ERN. Consistent with this, we have recently observed that, while individuals with GAD alone—which is associated with both persistent, generalized defensive reactivity and mild depression—are characterized by an enhanced ERN compared to controls, individuals with comorbid GAD and MDD—which is associated with more severe depression and decreased motivational engagement—instead display an attenuated ERN (Weinberg et al., under review). Increased attention to the complex and interacting influences of anxiety and depression on motivation and behavior will be imperative as neurobiological research on error-monitoring advances.

The literature reviewed above further suggests that the ERN may be a neurobehavioral marker that differentiates internalizing from externalizing disorders (Olvet and Hajcak 2008). Yet it is worth noting that the externalizing and internalizing spectra correlate positively with one another (Krueger 1999; Krueger and Markon 2006; Watson 2005), and that this relationship is unlikely to derive from criterion overlap (as is frequently the case for comorbidity within the internalizing spectrum; Angold et al. 1999). There is also evidence for shared genetic liability between internalizing and externalizing psychopathology (O'Connor et al. 1998), making precise examinations of differences in the pathophysiology of the two spectra difficult. However, the clinical presentation of the two domains is distinct, and we suggest that one area in which individuals with these disorders may differ is in their defensive reactivity; for instance, in their response to errors (see Patrick and Bernat 2010).

Given the evidence we have discussed, we view the ERN as a neural indicator of a *neurobehavioral trait* (Patrick and Bernat 2010): a stable individual difference measure that has a direct referent in both neurobiology (i.e., the ACC), and behavior. Based on the conceptual articulation of neurobehavioral traits by Patrick and Bernat (2010), we would argue that the ERN can form the basis for better understanding broad individual differences in cognition, personality, and psychopathology. We have suggested that the ERN is a trait-like neural response—formed by both environmental and heritable influences—that indexes error monitoring. Errors activate defensive motivational responses, and variation in the ERN relates to individual differences in sustained and generalized defensive reactivity. This view can explain why the ERN would be increased among those who are more anxious/fearful and decreased among those who are relatively low in anxiety/fear (i.e., externalizing). However, internalizing and externalizing disorders may also vary as a function of inhibitory control—which could relate to the ERN (Patrick and Bernat 2010; Vaidyanathan et al. 2011). It will be important to determine how the ERN relates to other measures of defensive reactivity (e.g., startle reflex potentiation, amygdala response, etc.)—and whether together these measures might form a profile of trait-like defensive reactivity (e.g., Patrick and Bernat 2010).

Refining how specific neurobehavioral traits and profiles relate to dimensions of personality and psychopathology—both cross-sectionally and prospectively—will be a critical direction for future research. For instance, some of the dispositional characteristics we have discussed (e.g., alertness, vigilance, perfectionism) may directly influence the general mechanisms that produce the ERN. On the other hand, variations in certain dimensional trait attributes (e.g., defensive reactivity) might account for systematic variance in the magnitude of the ERN across individuals while not contributing generally to ERN. Future research might better tease these interactions apart. Furthermore, it will be

important to more thoroughly examine whether the ERN is more related to the broad dimension of negative emotionality, or to more specific dimensions such as fear (Watson 2005), or anxious apprehension (Moser et al. 2011; Simons 2010; Watson 2005; Weinberg et al. 2010). We have suggested that examining the interactive effects of negative and positive emotionality on the ERN might be another fruitful avenue of study (Olvet et al. 2010). Moreover, one could treat the ERN as an independent variable, and examine other correlates as dependent variables—this approach might help refine, and possibly define, distinct phenotypes in personality and psychopathology. Further, continuing research into the ways in which trait-like variability in defensive reactivity or inhibitory control might interact with situation-specific variability (i.e., motivational manipulations) to influence the magnitude of the ERN will be critical. Such research has the potential to illuminate how individuals move from vulnerability markers to illness, and might begin to shed light on the development and course of multiple forms of psychopathology.

Finally, it is likely that neural indices like the ERN will also be highly sensitive to *cognitive* development and deficits. As described above, there are meaningful developmental changes in the ERN (Davies et al. 2004; Meyer et al. 2011; Nieuwenhuis et al. 2002; Pontifex et al. 2010; Segalowitz and Dywan 2009), consistent with maturational changes in the ACC, PFC, and dopaminergic systems underlying learning, executive control, and self-regulation (Ernst et al. 2006). These data reinforce the notion that the interplay between cognitive and motivational factors will likely be critical in determining the ERN. Studies on the normative developmental trajectory of the ERN will be crucial in this regard.

Summary

The ERN reflects the activity of an error monitoring system that is central to human behavior, and is situated at the crossroads of complex motivational and cognitive processes, as well as individual differences in these processes. Cognitive theories and models of the ERN are powerful, and flexibly explain *how* and *when* the occurrence of an error is processed in the brain—what, in other words, the ERN *is*. However, these theories and models are less adept at explaining variability in the ERN. We believe that the consideration of motivation—and the ways in which individual differences in tonic defensive reactivity might influence the evaluation of errors—will be critical to understanding the functional value of variation in the amplitude of the ERN. In addition, continued empirical and theoretical work is necessary to refine the conceptual model proposed here. For instance, because there is less extant research on the CRN, and its relationship to individual differences is less well-documented, we have largely not discussed the role that general response-monitoring processes might play in our model. In short, we have limited our musings to variation in the ERN. As the body of research contributing to the cognitive-affective neuroscience of error processing grows, a more exact portrait of this dynamic and flexible signal—and its relationship to cognitive and motivational variables—will hopefully inform our understanding of multiple areas of human functioning.

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