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What We've Learned From Mistakes: Insights From Error-Related Brain Activity

Greg Hajcak
Stony Brook University

Abstract

The error-related negativity (ERN) is a neural measure of error processing that peaks just 50 milliseconds after subjects make mistakes. Although previously conceptualized in purely cognitive terms, data increasingly suggest that the ERN is related to motivation and affect. The ERN has consistently been linked to individual differences in trait anxiety. Indeed, the ERN is itself trait-like, demonstrating high heritability and stability over time. The evidence suggests that an increased ERN is a viable biomarker of risk for anxiety disorders. The ERN is also sensitive to variation in the perceived consequences of making an error (i.e., *error value*): Punishing errors has a lasting effect on the ERN—an effect that is larger among more anxious individuals. Collectively, these data indicate that individual differences in anxiety and learning history could interact to influence the ERN—and that the ERN could be used to better understand trajectories of risk for anxiety disorders across development.

Keywords

errors, ERN, psychopathology, anxiety, endophenotype, biomarker, punishment

The Cognitive Neuroscience of Errors

Nearly two decades ago, a unique neural response to errors was reported in two different labs: In the United States, it was called the error-related negativity (ERN; Gehring, Goss, Coles, Meyer, & Donchin, 1993), and in Germany, it was referred to as the negativity associated with errors (Ne; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; I refer to it as the ERN from here on). The ERN is an event-related brain potential (ERP) that can be recorded at the scalp via electroencephalogram (EEG). ERPs provide a direct measure of neural activity time-locked to specific events, and they are thought to be generated by large populations of pyramidal cortical neurons that are aligned parallel to one another and perpendicular to the cortical surface (Luck, 2005).

Traditionally, the ERN is elicited by having participants engage in a speeded response task for several minutes. In our lab, we briefly present arrays of arrows that can either be incompatible (e.g., “< < < < <”, “> > < > >”) or compatible (“< < < < <”, “> > > > >”) and ask participants to respond to the direction of the central arrow. Participants do quite well on this task, but they make errors every now and then. We record the ongoing EEG during this task and subsequently examine brain activity time-locked to mistakes and correct responses.

Figure 1 (left) presents ERP data from 45 participants. The ERN is observed as a sharp negative-going deflection in the ERP that is time-locked to incorrect responses. The high

temporal resolution of ERPs is highlighted in the timing of this neural process, which peaks just 50 milliseconds after participants make mistakes. On the surface of the scalp, the difference between errors and correct responses is evident as a focal negativity maximal over frontal-central midline recording sites (Fig. 1, right).

An ERN can be elicited using a range of tasks across various stimulus and response modalities; accordingly, the ERN appears to reflect the early activity of a general error-processing system. An ERN is even evident when participants are unaware of having made a mistake (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001).¹ A wealth of data suggests that the ERN is generated in the anterior cingulate cortex—a region of the medial prefrontal cortex that is richly interconnected with both limbic and frontal regions of the brain (Bush, Luu, & Posner, 2000).

A number of computational models of the ERN, rooted in cognitive neuroscience, have been developed (Holroyd & Coles, 2002; Yeung, Botvinick, & Cohen, 2004). Broadly speaking, these models suggest that error detection, reflected in the ERN, is utilized to increase cognitive control. For

Corresponding Author:

Greg Hajcak, Department of Psychology, Stony Brook University, Stony Brook, NY, 11794-2500
E-mail: greg.hajcak@stonybrook.edu

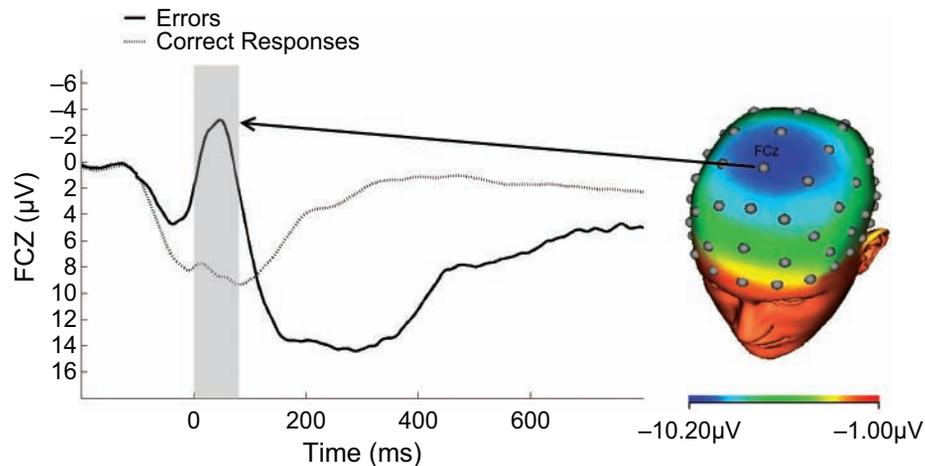


Fig. 1. Mean event-related potentials time-locked to correct and erroneous responses at electrode FCz (left). This graph plots voltage over time, where the response occurs at 0 milliseconds (ms). The error-related negativity (ERN) is observed as a sharp negative deflection that peaks around 50 ms (illustrated by the gray bar) after the commission of a mistake. The voltage difference between errors and correct responses in the time window of the ERN can be plotted over the scalp (right); the ERN is maximal at the frontal-central midline of the scalp.

instance, reinforcement-learning theory suggests that the ERN is elicited when actions are first evaluated as worse than expected based on recent performance—and that this learning signal is subsequently used to adjust behavior (Holroyd & Coles, 2002). On the other hand, conflict-monitoring theory proposes that the ERN reflects the coactivation of error and error-correcting responses—and that this conflict signal is utilized to increase cognitive control (Yeung et al., 2004). Both the reinforcement-learning and conflict-monitoring theories of the ERN propose that the magnitude of the ERN is directly related to performance measures and is utilized to improve subsequent behavior. Nonetheless, many dissociations between ERN and behavioral measures have been reported—both within and between subjects (see Weinberg, Riesel, & Hajcak, *in press*, for a review)—and evidence increasingly suggests that the ERN may reflect more than the “cold” cognitive process of error detection.

From Cognition to Motivation: Errors and Defensive Motivation

In earlier work, I and my colleagues found that errors not only elicit an ERN but also prompt a host of other physiological changes: Heart rate slows for several seconds following mistakes, and errors are associated with skin-conductance responses, which indicate engagement of the sympathetic nervous system (Hajcak, McDonald, & Simons, 2003b, 2004). Following from these data, we began to think about errors from the perspective of affective science, which focuses on response systems that are rooted in motivational tendencies to approach opportunities and avoid threats. From this perspective, errors can jeopardize an organism’s safety and thus are motivationally salient events. Although error detection could

be co-opted to improve performance in less survival-relevant situations, perhaps the evolutionary importance of error detection has more to do with protecting an organism. In other words, errors are threatening—and may, like external threats, activate defensive motivational systems designed to protect the organism.

To evaluate this possibility, we presented a sudden and brief loud sound (i.e., a 105-decibel sound for 50 milliseconds) following a subset of both erroneous and correct responses. This type of loud, jarring noise elicits a startle reflex across species, and it is most often measured by the eye-blink response in humans. The blink response and the startle reflex more generally are rapid and protective behaviors and are potentiated when organisms are in a more fearful state. The startle reflex has been used extensively to index fear across species (Lang, Davis, & Ohman, 2000). Consistent with the notion that errors are aversive events that prompt defensive motivation, we found that the startle reflex was larger after errors than after correct responses (Hajcak & Foti, 2008; Riesel, Weinberg, Moran, & Hajcak, *in press*). Moreover, a larger ERN predicted a larger startle response to errors—an effect that may especially characterize individuals with a large ERN (Riesel et al., *in press*). In this way, the ERN may be the earliest signal in a cascade of defensive responses that follow from making errors.

Consistent with the idea that the ERN relates to the motivational salience of errors, we have found that it is possible to affect the amplitude of the ERN by manipulating the perceived consequences of committing errors (i.e., error value). For instance, the ERN is slightly larger both when performance is evaluated explicitly and when monetary incentives are increased for correct responses (Hajcak, Moser, Yeung, & Simons, 2005). Other researchers have similarly found that

reward incentives and emphasizing accuracy can increase the magnitude of the ERN (reviewed in Weinberg et al., in press). Collectively, these data suggest that the ERN is larger in conditions when it is relatively worse to commit errors.

Individual Differences in Anxiety and the ERN

Errors are likely more catastrophic for some individuals than for others. The ERN is increasingly being used in translational research that uses variation in neural activity to better understand individual differences and psychopathology. Gehring and colleagues first demonstrated that patients with obsessive-compulsive disorder (OCD) were characterized by hyperactive action monitoring, reflected in a larger ERN (Gehring, Himle, & Nisenson, 2000). Since then, this finding has been replicated more than 10 times (see Weinberg et al., in press, for a review). However, an increased ERN does not appear to be specific to OCD: The ERN is also larger among patients with generalized anxiety disorder (Weinberg & Hajcak, 2011a). In considering a more dimensional approach to psychopathology, the ERN is increased among nonclinical participants who score high on measures of worry (Hajcak, McDonald, & Simons, 2003a), trait anxiety (Olvet & Hajcak, 2009a), and related personality traits (see Weinberg et al., in press, for a review).

A larger ERN may actually relate to increased *risk* for certain anxiety disorders (Olvet & Hajcak, 2008; Vaidyanathan, Nelson, & Patrick, in press). We measured the ERN among a sample of pediatric patients with OCD both before and after successful cognitive-behavioral therapy. Following treatment, the sample on average did not “have” OCD based on OCD-severity scores; however, the OCD group continued to have a larger ERN than a group of nonanxious control participants did (Hajcak, Franklin, Foa, & Simons, 2008). These data suggest that the increased ERN in OCD may reflect a biomarker of risk that is relatively independent of whether a person has the disorder. Along the same lines, a recent report found that individuals who had a first-degree relative with OCD had larger ERNs than individuals who did not have a first-degree relative with OCD (Riesel, Endrass, Kaufmann, & Kathmann, 2011). Considering that the ERN demonstrates strong heritability, with estimates ranging from 45% to 60% (Anokhin, Golosheykin, & Heath, 2008), these data collectively suggest that an increased ERN is a viable endophenotype (or heritable biomarker of risk) related to anxiety disorders—one that is a mediator between genetic predisposition and disease state (see Olvet & Hajcak, 2008).

ERN as a Neurobehavioral Trait

The ERN appears to be relatively trait-like: An increased ERN characterizes more anxious individuals—even following successful treatment. If the ERN relates to variability in relatively stable individual differences and personality dimensions, then the ERN itself ought to be relatively stable over time. In a

series of studies, we have found that the ERN is a robust neural signal with excellent psychometric properties. The ERN becomes stable after approximately 10 trials (Olvet & Hajcak, 2009c) and has excellent test-retest reliability that is on par with many trait measures (i.e., self-report measures) over the course of 2 weeks (Olvet & Hajcak, 2009b) and even 2 years (Weinberg & Hajcak, 2011b). Interestingly, the reliability of the ERN exceeds that of behavioral measures, including both accuracy and reaction time—which could account for variability in findings regarding the relationship between ERN and behavioral measures across studies.

In light of these data, we view the ERN as a *neurobehavioral trait* (Patrick & Bernat, 2010): a neurobiologically based individual-difference measure. In terms of the ERN, we have suggested that variability in the ERN reflects individual differences in defensive reactivity (Weinberg et al., in press). In particular, we believe that the ERN relates more to the specific dimension of anxious apprehension (i.e., worry) than to either anxious arousal (i.e., somatic anxiety; Moser, Moran, & Jendrusina, 2011; Weinberg & Hajcak, 2011a) or fear. For instance, we have found that individuals with specific phobias are not characterized by an increased ERN (Hajcak et al., 2003a). Even when performing a speeded-response task under conditions of symptom provocation (i.e., next to a live tarantula), spider-phobic subjects did not increase their ERN (Moser, Hajcak, & Simons, 2005). Recent work by Bartholow and colleagues indicates that alcohol reduces the ERN, and that the mechanism of this effect is reduced negative affect, a finding consistent with numerous studies indicating that alcohol reduces anxiety but not fear (Bartholow, Henry, Lust, Sauls, & Wood, in press; Greeley & Oei, 1999).

Neurobehavioral traits can be leveraged in several important ways. First, they could be used as targets for genetic analyses. Shedding light on the genetic determinants of the ERN could be an intermediate step toward understanding the genetics of more complex phenotypes (e.g., anxious apprehension). Second, neurobehavioral traits can be utilized to validate and delineate boundaries between more complex psychiatric phenotypes (e.g., fear vs. anxiety disorders; see Vaidyanathan et al., in press). Rather than treating the ERN as a dependent variable (i.e., what distinguishes anxious from nonanxious groups), neurobehavioral traits such as the ERN *can* be utilized as independent variables that could help refine, or even define, distinct phenotypes that may otherwise not be obvious from factor-analytic approaches to self-report measures (an approach that is consistent with the recent Research Domain Criteria proposal from the National Institute of Mental Health; Sanislow et al., 2010).

Learning Experiences and the ERN

If approximately 50% of the variation in ERN is heritable, then there is substantial room for other influences on the ERN. On the basis of a recent study, we have suggested that environmental factors and learning experiences that alter error

significance might have an impact on the ERN (Riesel, Weinberg, Endrass, Kathmann, & Hajcak, 2012). To examine this possibility, we employed an experimental design derived from the fear-learning literature. In the first half of the experiment, participants were punished in half of the blocks of trials. For instance, one participant may have been punished after 50% of their errors in blocks when the arrow stimuli were yellow but never punished after errors in blocks when the arrow stimuli were blue. The punishment was a very aversive sound, played for a full second at 100 decibels. Consistent with the research on ERN and error value described above, we found that the ERN was larger in blocks when errors were punished (Riesel et al., 2012).

Importantly, punishment stopped halfway through the experiment; as with the correspondence between punishment and stimulus color, subjects had to learn this. At the end of the experiment, all participants correctly reported the contingencies between block color and punishment—and all were aware that punishment stopped during the second half of the experiment. However, the ERN continued to be larger in blocks that were formerly punished. In fact, this effect was largest in the final quarter of the experiment. Punishing errors appears to have a relatively long-lasting impact on the ERN, and this effect does not depend on the conscious evaluation of punishment likelihood.

Finally, we found that the punishment-related increase in ERN was larger as a function of trait anxiety: More anxious participants were characterized by a larger ERN difference between the punishment and control conditions, suggesting that trait-anxious individuals are more sensitive to potential punishment of their errors. Indeed, the modulation by punishment in both the learning and extinction phases of the experiment was driven by individuals who were more anxious. More anxious individuals, then, might be especially susceptible to contextual modification of error-related brain activity based on punishment and learning experiences. These data demonstrate two important things: First, environmental factors (i.e., punishment) that modify error value can increase the ERN; second, both relatively stable individual differences (i.e., anxiety) and situational factors (i.e., punishment) interact with one another to influence the ERN. Although speculative, these data provide a potential pathway linking early environmental adversity and certain punitive parenting styles to later risk for anxiety disorders via an increased ERN.

Development of the ERN and Trajectories of Risk

An exciting avenue of research on the ERN concerns development and trajectories of risk. We know that the trajectory toward anxious psychopathology begins early in life—and one strategy for understanding developmental pathways of risk is to study the development of neural markers of risk such as the ERN. Importantly, the ERN can be elicited in children as young as 6 (Torpey, Hajcak, Kim, Kujawa, & Klein, 2012), if not younger. In a recent paper, we examined the relationship between ERN and anxiety as

a function of age among 8- to 13-year-olds (Meyer, Weinberg, Klein, & Hajcak, 2012). We found that the relationship between ERN and anxiety was strongly moderated by age: Increased ERN was related to anxious symptoms, but only among older (i.e., 11- to 13-year-old) children. We are currently examining the ERN in relation to known risk factors for anxiety disorders among a large and longitudinal sample, at both ages 6 and 9. Eventually, we hope to use the ERN to retrospectively identify early trajectories of risk and thereby inform and improve subsequent efforts at early identification of children at increased risk for anxiety disorders.

Conclusions and Future Directions

The ERN is a stable, trait-like neural measure that is elicited by errors. Variation in the magnitude of the ERN is heritable, and it relates to both individual differences in anxiety and risk for anxiety disorders. However, the ERN can be modified by contextual factors that affect the value of errors, and this effect appears to vary with individual differences in anxiety.

It will be important for future studies to examine the ability of the ERN to prospectively predict meaningful individual differences in anxiety—research that will require longitudinal designs. Along these lines, it will be important to carefully examine the impact of early experience on the development of the ERN and its relationship with anxious symptoms. Finally, although we have focused on the link between ERN and anxiety, abnormal ERN has been documented in other forms of psychopathology (see Weinberg et al., in press, for a review). For instance, individuals high in externalizing and impulsivity appear to be characterized by a reduced ERN. It is essential for future work to clarify how anxious apprehension and externalizing symptoms together affect the ERN. From basic to applied science, we will continue to learn from errors for another two decades, if not longer.

Recommended Reading

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- Olvet, D. M., & Hajcak, G. (2008). (See References). An explication of the endophenotype construct and how it might relate to the ERN.
- Simons, R. F. (2011). The way of our errors: Theme and variations. *Psychophysiology*, *47*, 1–14. An overview of various ERPs that are used to study internal and external negative feedback.
- Weinberg, A., Riesel, A., & Hajcak, G. (in press). (See References). A more comprehensive and detailed version of the material presented here.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Note

1. Following the ERN, errors are characterized by a P300-like response around 200 to 400 milliseconds that is maximal at parietal recording site; this response is known as the error positivity (Pe). Although the current paper focuses on the ERN, the recommended readings include relevant discussions of the Pe.

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