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The Nature of the Relationship Between Anxiety and the Error-Related Negativity Across Development

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Abstract
Purpose of Review I review the literature on the relationship between anxiety and the error-related negativity (ERN), a neurophysiological marker of performance monitoring, across development. I cover the development of the ERN, its functional significance, and its different relationship with anxiety in young children compared to adolescents and adults.
Recent Findings Contemporary research indicates that the ERN becomes larger with age and shows primary sources in cingulate, frontal, and motor cortices. Functional accounts of the ERN and its relationship with anxiety emphasize either cognitive control or affective mechanisms. Converging evidence across development suggests a reduced ERN characterizes anxious young children whereas an enlarged ERN characterizes older children, adolescents, and adults.
Summary The mechanisms involved in the developmental change in the relationship between the ERN and anxiety have important implications for better understanding interactions between cognitive control, anxiety, and motivation across the life span. Further research is needed to address extant methodological limitations and make stronger links to related neuroscience findings and theory on the development of anxiety and self-control.

Keywords Error-related negativity · ERN · Anxiety · Development · Children · Adolescence

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Introduction

The error-related negativity (ERN) is a negative deflection in the human event-related brain potential (ERP) that peaks within 100 ms of an erroneous response at frontocentral recording sites [1–3]. Localization studies have shown that the ERN is generated by a network of frontal brain regions including the anterior cingulate cortex (ACC; particularly the dorsal subdivision), prefrontal cortex (PFC), and supplementary motor areas [3–5]. Others have also found that the ERN has a strong source in the posterior cingulate cortex (PCC) [6, 7•]. The ERN is generally considered to be an index of cognitive control-related performance monitoring that is involved in coordinating optimal responding following mistakes [3, 8–11]. Indeed, the ERN is listed as a marker of Performance Monitoring, a subconstruct of Cognitive Control, in the Cognitive Systems domain of the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC) matrix.

Of relevance to the current review, research on the ERN has exploded over the past 15 years or so because of a robust relationship demonstrated between its amplitude and anxiety-related problems in children and adults [12•]. This relationship has led several research groups to challenge the traditional "cognitive" explanation of the functional significance of the ERN referenced above as well as to better understand the nature of the relationship between the ERN and anxiety across development [13•]. In this paper, I will build on previous reviews by focusing on the nature of the relationship between the ERN and anxiety in youth, how this relationship seems to change across development, and what such developmental changes suggest about the utility of the ERN as a neural marker of anxiety-related processes. I begin, however, with a bit of background on the functional significance of the ERN irrespective of its relationship with anxiety.

The Functional Significance of the ERN

A complete coverage of the ERN literature is beyond the scope of this review. The primary purpose of this section is to provide a brief overview of major theories of the functional significance of the ERN. As a matter of practice, the ERN is typically elicited by error responses in speeded two-choice reaction time tasks involving non-emotional stimuli. Although some have looked at the ERN in the context of more than two response options and in the context of emotional stimuli, non-emotional two-choice reaction time tasks are by far the most common approach for eliciting errors in research examining the relationship between the ERN and anxiety [12, 14]. Although this may seem like an unimportant detail, I argue that such task parameters are critical when evaluating the functional significance of the ERN and the nature of its association with anxiety.

Some of the first studies of the ERN suggested that it might reflect error detection itself [1, 2]. In short, the error detection theory of the ERN suggested that its amplitude indexes the comparison of the neural representation of the just-executed error response with the neural representation of the correct response—thus, the mismatch between representations was thought to produce the ERN [15]. In response to noted shortcomings of the error detection hypothesis—i.e., that the model does not account for why the brain would execute an error response in the first place—Yeung and colleagues [10] proposed the conflict monitoring theory of the ERN that leveraged computational models of task performance. The conflict monitoring hypothesis suggests that the ERN reflects the concurrent activation of the erroneous response and the subsequent error-correcting response that is triggered by continued stimulus processing [16]. A third explanation of the ERN is the reinforcement learning theory proposed by Holroyd and Coles [8], which is also based on a computational model. The reinforcement learning theory suggests the ERN reflects the evaluation of events (in this case, an error) as worse than expected. Specifically, the ERN reflects the impact of a phasic dip in mesencephalic dopamine—produced when events occur that are worse than expected—on the ACC. More recently, theorists have generated new models that combine conflict monitoring and reinforcement learning aspects [9, 17]. All of these models assume that the computation that gives rise to the ERN is involved in optimizing performance.

In large part because several studies suggested that the ERN has a principal source in the dACC or midcingulate cortex (MCC), "affective" interpretations of the ERN have emerged. The dACC/MCC is considered to be a hub for integrating emotional and cognitive information given its distributed connections with other cortical and subcortical regions throughout the brain [18]. Thus, if the ERN is a dACC/MCC signal, then it followed that it, too, might represent an index of "emotion-cognition" interaction. Initial individual-differences evidence seemed to support this account, as Luu and colleagues [19] found that college students scoring high on negative affect and negative emotionality demonstrated an enlarged ERN early in their experiment. Numerous correlational studies followed, further confirming that individuals high on symptoms of or diagnosed with negative affective psychopathology show an enlarged ERN [12•, 14]. Beyond correlational studies, several experimental manipulations that aim to increase the affective or motivational significance of errors seem to produce an enlarged ERN. For instance, Hajcak et al. [20] showed that the ERN is enlarged under evaluative stress and when errors are committed on trials worth more points toward a monetary bonus. Along these same lines, the ERN is larger under conditions of punishment such as when errors are followed by an aversive loud noise burst [21]. A recent, comprehensive instantiation of this affective hypothesis specifically suggests that the ERN reflects the degree to which an error is evaluated as threatening—so-called sensitivity to endogenous threat [13•]. This hypothesis claims to account for within- and between-subject variations in the ERN and has been leveraged to have the ERN included as a physiological marker of the Sustained Threat construct in the Negative Valence System domain of the NIMH RDoC matrix.

The abovementioned accounts need not be mutually exclusive, and, in fact, at the current time, the ERN is included as a marker of three different NIMH RDoC constructs: Performance Monitoring (a subconstruct of Cognitive Control), Sustained Threat, and Reward Learning. Thus, it might be that the ERN reflects a combination of cognitive and affective/motivational processes. The threat sensitivity hypothesis is particularly relevant to the current review, in that it is often used to characterize the nature of the relationship between the ERN and anxiety. I reference this hypothesis in subsequent sections and compare it to another conceptualization of the ERN-anxiety relationship that we recently proposed, the compensatory error monitoring hypothesis (CEMH) [12•, 22]. Before addressing the nature of the relationship between the ERN and anxiety, however, I next turn to research on development and the ERN.

Development and the ERN

A full understanding of the nature of the relationship between the ERN and anxiety must not only be grounded in functional accounts of the ERN itself, but also projected onto the developmental trajectory of the amplitude of the ERN and its neural sources. Studies generally show that the amplitude of the ERN increases from childhood to adolescence and adulthood. The increase in ERN across development has generally been interpreted as reflecting improvements in cognitive control mechanisms [23]. Indeed, performance on a number of cognitive control-related tasks increases with age, which is accompanied by changes in activity in frontoparietal and

frontostriatal circuits implicated in cognitive control [24•, 25, 26]. The increase in ERN is presumed to reflect such neural and behavioral changes across development insofar as it is an index of cognitive control-related performance monitoring.

The earliest developmental studies of the ERN suggested that an ERN might not be observable prior to adolescence [27]. Subsequent studies, however, showed that the ERN could be elicited in children between the ages of 4 and 11 [28–32], challenging the view that the neural mechanisms underlying the ERN were not online until adolescence. Grammer et al. [33] further demonstrated the presence of an ERN in children as young as 3 years of age using a developmentally sensitive Go/No-Go paradigm—the "Zoo Task." Interestingly, Grammer et al. did not observe a relationship between age and the ERN in the 3- to 7-year-old children in their study, which led them to conclude that the ERN may undergo its largest maturation during adolescence. However, our group [34] later showed that older age was associated with an increased ERN in children between 3 and 8 years of age using a developmentally sensitive version of the Flanker task—the "Fish Flanker"—while we, at the same time, replicated the null relationship between age and the ERN using the Zoo Task. We replicated the null relationship between age and the ERN using the Zoo Task again in a sample of children 5–8 years [35•]. Overall, results from extant studies indicate that an ERN can be observed as young as 3 years old and increases with age. Exactly during which developmental stage the ERN increases the most is less clear, as some findings point to the possibility that the ERN increases mostly during later childhood and adolescence whereas results from Lo et al. [34] suggest that age-related changes in ERN amplitude can be observed even earlier depending on the task used. Despite inconsistencies in the relationship between the ERN and age during early childhood, findings generally show that older age is associated with better performance across tasks.

Two limitations of developmental studies of the ERN reviewed above are that they rely on cross-sectional designs and do not fully account for known measurement problems in children—i.e., intra-individual variation in behavior and ERP latencies across trials. To address these issues, DuPuis et al. [36•] examined the average amplitude of the ERN as well as theta-band decompositions of its constituent parts—signal strength and temporal consistency—elicited during a Go/No-Go task in a three-wave longitudinal study of children 5–7.5 years old. Confirming previous cross-sectional work, DuPuis et al. found that the average amplitude of the ERN increased across the 2-year follow-up period. Importantly, they also found that the increase in ERN amplitude was primarily due to increases in temporal consistency of the underlying theta signal, and not theta signal strength—in fact, signal strength decreased over time. The authors concluded that the increase in temporal consistency likely reflects development of a more efficient performance monitoring system characterized by greater temporal precision in neuronal firing at post-synaptic sites involved in the generation of the ERN. DuPuis et al. further showed that both the average ERN amplitude and temporal consistency measure demonstrated low test-retest reliability over time, whereas the theta signal strength measure demonstrated fairly high test-retest reliability. These results challenge the view that ERN amplitude reflects a stable trait marker [13•]—in that both the average amplitude and temporal consistency measure changes over development demonstrated poor stability across 2 years. Instead, the signal strength measure might represent a stable trait, but contributes less to the developmental changes in the scalp-recorded ERN amplitude. It may also be the case that, irrespective of measurement, the processes that underlie the ERN do not comprise a stable trait until maturation of relevant neural systems reaches a certain threshold later in development, at which point they may become more stable within and across individuals. Such measurement challenges, and advanced signal processing methods aimed at addressing them, must continue to be evaluated to inform models of cognitive control development as well as those aimed at characterizing the nature of the relationship between the ERN and anxiety across development.

Finally, a recent source localization study of the development of the ERN also points to potentially important changes in neural generators over time. For their study, Buzzell et al. [7•] combined high-density EEG with structural MRI data to capture sources of the ERN in individuals 9–35 years of age. This approach allows for a much more precise localization of ERP sources by constraining the source space with the individual subject's structural scan information. Consistent with more recent source localization studies of the ERN [6], Buzzell et al. found primary sources in the PCC and dACC, with the strongest source in the PCC. Other sources of the ERN were also noted, including frontoparietal regions, insula, orbitofrontal cortex (OFC), and inferior frontal gyrus (IFG). PCC and dACC sources of the ERN were stable across age, whereas source strength of insula, OFC, and IFG increased later in development. These results suggest that the ERN should not only be considered a dACC signal. Indeed, studies by Agam et al. [6, 37] have shown that the ERN can be anatomically and genetically dissociated from error-related dACC activity, although localization of the ERN in these studies may still indicate a dACC source. These results also indicate that the neural sources of the ERN comprise stable and developmentally sensitive components. Despite these important implications, a significant limitation of the Buzzell et al. study is its small sample size (total $N = 43$ across ages).

Together, developmental work on the ERN suggests its amplitude increases with age, which may primarily be due to increases in temporal stability of the underlying theta signal, and that its neural sources comprise both stable and developmentally sensitive components. This review also revealed that

measurement issues related to intra-individual variability in latency of the ERN and task differences are important factors to consider in interpreting whether and how the ERN and its underlying neurocognitive mechanisms change across development. It seems fair to conclude, however, that the processes underlying the generation of the ERN present in early childhood begin undergoing developmental changes during this time and continue to mature throughout adolescence and adulthood. Moreover, performance in Flanker and Go/No-Go tasks improves with age beginning in early childhood and continues through adolescence and adulthood. A particularly interesting avenue for future research will be to test how developmental changes in the ERN might account for developmental changes in behavioral performance. A recent neuroimaging study found that executive network activity mediated age-related changes in working memory performance in 8–22-year olds [38]. Demonstrating the mediating role of the ERN in Flanker and Go/No-Go performance would likewise provide strong evidence for the role of the ERN in the development of cognitive control abilities.

A few pieces of data from the literature reviewed above warrant special attention inasmuch as they may inform the nature of the relationship between the ERN and anxiety that I cover next. First, that the ERN should not be considered synonymous with error-related dACC activity has implications for conceptions of the functional significance of the ERN and its relationship with anxiety. At the very least, researchers should be cautious in drawing parallels between the ERN and error-related dACC activity. Moreover, that the PCC should be considered a significant neural source of the ERN indicates that we must become more familiar with research on the role of the PCC in the brain. In short, a recent review of PCC function suggests its activation and connections with other regions help coordinate arousal state and the balance of internal versus external and broad versus narrow focus of attention [39•]. Finally, changes in the source contributions to the ERN across development open up interesting possibilities about the dynamic interplay of neural mechanism involved in conflict monitoring, salience detection, interoception, error correction, and hedonic processing, especially as they might emerge along different time courses during adolescence [24•].

The Relationship Between the ERN and Anxiety Across Development

As noted at the beginning of this paper, the amplitude of the ERN tends to be enlarged in anxious adults and children. The relationship between an enlarged ERN and anxiety seems to be particularly strong for anxiety problems characterized by anxious apprehension or worry (generalized anxiety disorder; GAD; OCD) as opposed to anxious arousal and panic (phobic and panic disorders) [12•, 40, 41]. We also recently reported meta-analytic evidence that adults with OCD demonstrate a larger ERN than those with other anxiety-related problems [42•]. Multivariate work by [43] further suggests that enlarged ERN may serve as a transdiagnostic marker of compulsive checking behavior across adult OCD, GAD, and major depressive disorder (MDD) patients. Because the adult literature has been widely covered in descriptive and meta-analytic reviews referenced herein, I do not expand on it here but rather now focus on the relationship between the ERN and anxiety in children and adolescents.

Cross-sectional Studies in Youth Although most accept that anxiety problems are associated with an enlarged ERN, a study by Meyer et al. [44] revealed that the relationship might be developmentally sensitive. In a modest sample ($N = 55$) of 8–13-year olds, Meyer et al. [44] found no relationship between ERN and parent-rated anxiety symptoms when looking across the full sample. However, age moderated this effect such that the expected relationship between higher anxiety and enlarged ERN was evident in older children (≥ 12.5 years) whereas there was a small (non-significant), unexpected relationship between higher anxiety and a smaller ERN in younger children (≤ 9.5 years) in the sample. This was an intriguing finding and stands in contrast to the zeitgeist that anxiety is related to an enlarged ERN in adults *and* children [12•].

Since the Meyer et al. [44] report, a few other studies have also demonstrated that anxiety measures are related to a smaller ERN in young children. Torpey et al. [45] showed a smaller ERN in 6-year-old children who displayed fearful behaviors in laboratory temperament tasks. We replicated this effect in a small sample of 5–7-year olds [46]. We also recently showed that parent-reported separation anxiety symptoms were associated with a smaller ERN in 5–8-year olds [35•]. The Lo et al. [35•] and Torpey et al. [45] studies were particularly strong tests of this effect because they both comprised large samples (133 and 326, respectively). In a related study, Lo et al. [34] reported that a smaller ERN was associated with larger startle eyeblink response elicited by unpleasant video clips and greater resting right-lateralized parietal activity in 3–7-year olds. Both startle eyeblink and right-lateralized parietal activities reflect reactivity of the brain's negative valence system and show associations with anxiety [47–50]. By zooming in on early childhood years (≤ 8 years of age), this collection of findings replicates and bolsters what Meyer et al. initially noted in their younger subsample.

In line with the notion that early childhood years might represent a period of development characterized by a reversed relationship between the ERN and anxiety, prior developmental studies suggesting enlarged ERN in anxiety included samples mainly comprised of older children and adolescents (≥ 11 years of age) [51–54]. More recent work continues to replicate the association between an enlarged ERN and anxiety in older children and adolescents [55, 56]. Interestingly, even in a sample of all adolescent females (13.5–15.5 years

old), Weinberg et al. [13•] found that older adolescent females demonstrated a relationship between higher reports of compulsive checking behavior and larger ERN whereas younger adolescent females did not. Findings from McDermott et al. [57] further suggest that the relationship between the ERN and anxiety in adolescence is sensitive to developmental processes in that they found an enlarged ERN was only related with anxiety diagnosis among adolescents previously rated as being behaviorally inhibited during toddler and early childhood years.

A fly in the ointment, however, is the study by Meyer et al. [58] that revealed an enlarged ERN in 6-year-old children diagnosed with anxiety disorders. To my knowledge, this is the only study to demonstrate a larger ERN in anxiety in young children and thus warrants further examination. A strength of the study is that the size of the sample was fairly large ($N = 96$), exactly half of which comprised children with anxiety disorders. Three issues with sample characteristics, however, muddy the waters. First, the anxious group included children with a number of different anxiety disorders, the majority of whom were diagnosed with specific phobia. Recall that in prior adult studies specific phobia and related anxious arousal phenotypes do not tend to show a reliable association with the ERN [12•, 40]. Second, the anxiety group included children with comorbid conditions that show blunted ERN (depression and externalizing problems) [13•, 59]. Third, the control group included several children who, too, were diagnosed with depression and externalizing problems and whose mothers had a history of anxiety problems, which has been linked to reduced ERN [45].

In sum, cross-sectional studies suggest that anxiety is generally associated with a smaller ERN in early childhood years and a larger ERN in later childhood and adolescent stages of development. The one exception to this pattern of findings is the Meyer et al. [58] study showing an enlarged ERN in 6-year-olds diagnosed with anxiety disorders, but potential confounds introduced by diagnostic heterogeneity in both the anxiety and control groups render these results difficult to interpret.

Prospective Studies in Youth Several additional investigations have examined the role of the ERN in predicting later anxiety problems. Meyer et al. [60] were the first to demonstrate the predictive validity of the ERN. Specifically, they showed that an enlarged ERN measured at 6 years of age predicted the onset of new anxiety disorders at age 9. To my knowledge, this is the only study to demonstrate the utility of the ERN in predicting later anxiety disorders. Despite the fact that an enlarged ERN predicted later emergence of anxiety disorders it *did not relate to anxiety disorder symptoms at age 9*—neither was the ERN related to concurrent anxiety disorder symptoms at age 6. One concern with this discrepancy is that the analysis involving onset of new anxiety disorders was much less robust because of a small sample size—there were only 26 children with a new anxiety disorder diagnosis out of 236 in the full sample—compared to the analysis involving anxiety symptoms, which took advantage of the full sample size. Moreover, the utility of the ERN in predicting later emergence of anxiety disorders only held for one of the three electrodes tested.

Other studies suggest an indirect, rather than a direct, relationship between early recorded ERN and later anxiety problems. For instance, one study showed that children with a larger ERN at age 6 only demonstrated later increases in anxiety and depression symptoms if they were previously rated as persistently irritable at age 3 [61]. Lahat et al. [62] also found that among children with a larger ERN at age 7, only those rated highly behaviorally inhibited as toddlers and preschoolers went on to develop more severe social anxiety symptoms at age 9. An even more complex set of interactions was revealed by Meyer et al. [63•], who found that children demonstrating a larger ERN at age 6 only later developed increases in anxiety and depression symptoms if they were rated as more temperamentally fearful at age 3 and lived in households that experienced greater stressors following Hurricane Sandy.

A final set of studies turns the ERN-anxiety relationship on its head and uses early indicators of anxiety or stressful life events as predictors of later ERN amplitude. For instance, both McDermott et al. [57] and Lahat et al. [62] found that higher rated behavioral inhibition in toddlers and young children related to a larger ERN later in development (15 and 7 years old, respectively). Harsh parenting (authoritarian and hostile) reported at age 3 also seems to relate to a larger ERN at 6 years old, although only in children carrying at least one methionine allele of the BDNF genotype that has been associated with risk for psychopathology [64]. Curiously, however, not all early risk factors predict later increases in the ERN. Early psychosocial deprivation associated with institutional rearing was not related to later recorded ERN at age 8 in one study [65] and was actually related to a *smaller* ERN measured at age 12 in another [66]. In fact, these studies suggested that a larger ERN served a protective role in that institutionalized children with larger ERNs tended to have fewer externalizing problems and better academic outcomes.

Together, prospective studies paint a complex picture of the relationship between the ERN and anxiety-related problems. Indeed, only one study suggests a direct relationship between earlier recorder ERN and later emerging anxiety [60]. Methodological concerns render this finding tenuous, however. The other prospective findings indicate a more indirect relationship between the ERN and anxiety. Aside from showing indirect relationships between early recorded ERN and later measured anxiety, these other studies also failed to relate the ERN itself to anxiety and instead decomposed interactions by looking at how other risk factors predicted later anxiety

problems in children either characterized by a large or small ERN. The prospective studies showing that early anxiety risk predicts later ERN amplitude further suggest a bidirectional or reciprocal relationship between the ERN and anxiety-related indices.

The Functional Significance of the ERN in Anxiety Across Development

In the context of developmental findings on the relationship between the ERN and anxiety, we are now left with the important question: What does it all mean? That is, what does the amplitude of the ERN index in anxiety? The dominant account of the relationship between the ERN and anxiety leverages the more general theory that the ERN reflects sensitivity to *endogenous* threat (i.e., errors; [13•]). Based primarily on the adult and adolescent literature, this account proposes that an enlarged ERN reflects the greater evaluation of mistakes as threatening in anxious individuals. Our group has recently suggested an alternative account, the compensatory error monitoring hypothesis (CEMH), which leverages existing theory and evidence regarding cognitive deficits in anxiety. Specifically, we proposed that enlarged ERN in anxiety reflects compensatory engagement of cognitive control (i.e., reactive control; [67]) following errors that is required because of the distracting effects of worry on active goal maintenance [12•, 22]. We also based our suppositions on adult and adolescent findings. I evaluate the evidence for each in the following sections and draw special attention to how they account for developmental findings.

Sensitivity to Endogenous Threat The sensitivity to endogenous threat hypothesis (hereafter referred to as SETH) is based on the notion that errors represent internal (i.e., endogenous) threats that trigger a cascade of neural, physiological, and behavioral responses aimed at correcting and preventing them [13•]. In this context, the ERN is the early evaluative signal carrying information about the degree to which the error is threatening and therefore what sort of control processes are required to adapt to the situation. This theory garners support from experimental studies showing enlarged ERN during conditions in which errors are punished, socially evaluated, or cost participants points toward a payout. Weinberg et al. further argue that studies showing specificity of the relationship between the enlarged ERN and anxiety to OCD, GAD, social anxiety disorder (SAD), and related symptom dimensions (worry and compulsions) support the claim that the ERN represents sensitivity to endogenous threats because these conditions all share a common focus on internal sources of threat.

The SETH also leverages these ideas to explain developmental changes in the relationship between the ERN and anxiety. Weinberg et al. [13•] and, more recently, Meyer [68] propose that developmental differences in the relationship between the ERN and anxiety reflect an interaction between symptom severity and the degree of concern about external versus internal threat. Specifically, as outlined by Meyer, children with low levels of normative anxiety show an average-sized ERN that remains stable from early to late childhood. Children with high levels of normative anxiety, on the other hand, show a smaller than average ERN during early childhood, when anxious children are more concerned with external sources of threat, whereas these same children show a larger than average ERN during late childhood when they begin to develop greater concern with internal sources of threat, such as their own mistakes [69]. Clinical levels of anxiety, however, are proposed to be associated with an especially large ERN beginning in early childhood because they already bias individuals toward concern about internal sources of threat.

Despite the intuitive appeal of the SETH, there are several issues with its conceptual and empirical foundations. First, the link between the ERN and sensitivity to internal threat is loose. Perfectionists, for instance, who report significant concerns about mistakes do not show an enlarged ERN [70–72]. As we have reviewed before, the ERN is also unrelated to other threat-related responses to errors such as skin conductance and startle eyeblink responses [22]. Moreover, the ERN is not enhanced in OCD patients when errors are made more threatening by punishing them with monetary loss [73]. If the ERN reflects the degree to which errors are experienced as threatening or aversive, as SETH suggests, then the ERN should be related to other threat responses elicited by errors and be enlarged in OCD patients when errors are made more aversive, neither of which pans out. SETH does not make a specific prediction that there is a ceiling on the aversiveness of errors to help explain the lack of punishment effect on the ERN in OCD. Weinberg et al. also argue that the transdiagnostic relationship between compulsive checking behaviors and enlarged ERN supports the SETH; however, checking only related to enlarged ERN after accounting for other symptom dimensions in multivariate analyses and it is unclear how checking behaviors reflect sensitivity to endogenous threat. Checking compulsions (and compulsions generally) function as compensatory responses aimed at alleviating anxiety-provoking obsessions. Does the ERN relate to checking because checking is one of the many possible defensive reactions the evaluative signal underlying the ERN can trigger? Why would the ERN not relate to obsessions, then, as obsessions could also be considered evaluative signals of endogenous threat (e.g., "I forgot to turn off the stove") that trigger compulsions (e.g., check stove)?

Extant data are also not supportive of how the SETH accounts for the complex relationship between the ERN and anxiety across development. First, only one study [58] has demonstrated enlarged ERN in young children with anxiety

disorders and it suffers from several methodological limitations mentioned above—most importantly, diagnostic confounds across patient and control groups. Thus, it seems premature to conclude that young children with clinical levels of anxiety demonstrate an enlarged ERN. Moreover, the majority of patients in this study's anxious group suffered from a specific phobia, which is characterized by concern about external and not internal threat. Second, the SETH conceptualization does not explain why separation anxiety would be related to a smaller ERN [35•], because separation anxiety is characterized by worry—i.e., internal threat—in young children [74]. Third, findings from existing studies in children do not provide support for a non-linear relationship between the ERN and anxiety that the SETH suggests. For example, although only a minority of participants crossed clinical threshold (≥ 70 T) for separation anxiety disorder in the Lo et al. [35•] study, none of those who did cross the threshold demonstrated an enlarged ERN. Given the relatively high prevalence of childhood anxiety disorders [75] and the dimensional nature of anxiety problems [76], it stands to reason that at least some of the children scoring above clinical threshold in the Lo et al. study would have met diagnostic criteria for an anxiety disorder. That none of these children demonstrated an enlarged ERN challenges the idea that clinical anxiety relates to an enlarged ERN in early childhood.

Fourth, there is little precedent for a relationship between external attention to threat and reduced ERN. On the contrary, one study in adults showed that increasing the salience of external threat by presenting spider phobics with a tarantula did not affect the ERN, but rather reduced the error positivity (Pe; [77])—an ERP component much more sensitive to attentional resource allocation to errors than the ERN. It is unclear why this process would work so differently in children, as the SETH suggests, such that young anxious children would show a decreased ERN but intact Pe. Fifth, the proposed timing of the developmental shift in the ERN-anxiety relationship does not seem to capture extant findings. Weinberg et al. [13•], for example, showed a significant relationship between checking symptoms and enlarged ERN only in older individuals in a large sample of all adolescent females (ages 13.5–15.5). The model proposed by Meyer suggests the developmental shift to a larger ERN with more anxiety should be in place by late childhood, specifically, by age 9.

Finally, the SETH does not make specific enough predictions to account for the complex interactions revealed between anxiety, the ERN, and related risk factors in developmental prospective studies. The SETH would seem to have particular difficulty accounting for why children who had experienced early psychosocial deprivation—a known risk factor for the development of later anxiety problems—would be characterized by an unchanged or smaller ERN and why, in these same children, an enlarged ERN would be related to better behavioral and academic outcomes [65, 66].

Compensatory Error Monitoring Hypothesis The CEMH is grounded in attentional control theory (ACT; [78]), which asserts that the distracting effects of worry on cognition generally impact processing efficiency (e.g., increased reaction time) but not processing effectiveness (e.g., decreased accuracy) because anxious individuals deploy compensatory effort. Our CEMH suggests that enlarged ERN in anxiety is a neural marker of such compensatory effort. Said in another way, enlarged ERN in anxiety is a sign of inefficient performance monitoring because greater resources are allocated to achieve an adequate, but not optimal, level of performance. We formalize the CEMH using the conflict monitoring theory of the ERN [10] and suggest that enlarged ERN in anxiety specifically reflects reactive control-induced increases in stimulus processing around and after the erroneous response that leads to enhanced conflict between the just-executed error and the correct response. *The clearest distinction between the CEMH and the SETH is that the former proposes the ERN is a control signal whereas the latter posits that the ERN is a threat signal*.

The CEMH finds support in studies demonstrating that worry or anxious apprehension relates more strongly to enlarged ERN than other forms of anxiety. This specific relationship occurs because worries, as opposed to physiologic sensations, are proposed to most interfere with online goal maintenance by consuming working memory resources and thus require compensatory effort to overcome [12•, 79]. That enlarged ERN is found in anxious individuals in the absence of compromised—or improved—performance is further support for CEMH inasmuch as the ERN serves a compensatory function.

Borrowing from ACT, CEMH also predicts that incentive and motivation manipulations should have a smaller effect on ERN in anxious compared to that in non-anxious individuals, which is what Endrass et al. [73] found in OCD patients. Because anxious individuals already deploy compensatory effort at baseline—i.e., demonstrate a larger ERN—they have few or no additional resources available to martial when incentives are provided for improved performance. The CEMH further attributes the relationship between enlarged ERN and compulsive checking to the fact that both are compensatory responses [13•, 43]. Although the ERN is seen as a compensatory signal aimed at maintaining adequate performance whereas compulsions are seen as compensatory behaviors aimed at decreasing anxiety provoked by obsessions, it may be that these two processes share underlying mechanisms in cognitive control brain networks or that the compensatory processes involved in the generation of the ERN maintain performance, at least in part, by suppressing obsessions.

A few newer pieces of supportive evidence also warrant mention, as they help to further specify the role of the enlarged ERN in anxiety. First, we recently showed that the enlarged ERN in worry served to compensate for decreased connectivity between medial and lateral prefrontal cortices indexed by

interchannel phase coherence [80•]. Specifically, worry was simultaneously associated with an enlarged ERN and decreased functional connectivity between medial and lateral prefrontal regions. These two opposing effects seemed to explain why, at the bivariate level, anxiety was unrelated to post-error behavioral performance. Indeed, worry was related to poor post-error performance through its association with decreased medial-lateral functional connectivity; however, worry's association with an enlarged ERN seemed to counteract this effect because an enlarged ERN was also related to increased medial-lateral functional connectivity. Although an enlarged ERN was not directly associated with post-error behavior, its indirect relationship through increased medial-lateral functional connectivity suggests it does have a control function so long as it elicits coordination between the monitoring and implementation units in the cognitive control network. In a subsequent study, we also found that decreased pre-error trial disengagement—indexed by a reduced error-preceding positivity (EPP)—mediated the relationship between worry and enlarged ERN [81].

Together, these findings provide a fuller picture of how worry is dynamically related to frontal control network function. Specifically, we propose that high worriers begin to engage compensatory effort (attentional focus) as attention naturally declines prior to an error, which results in increased target processing that is too late to avoid a mistake in the moment but increases conflict around the time of error commission and therefore produces an enlarged ERN. Enlarged ERN, then, helps to compensate for reduced integration of the frontal control network, which normalizes behavioral performance of worriers.

It is clear from this characterization that the control signal underlying the generation of the ERN in anxiety has motivational value. We have indicated such in a previous work [22]. To further expand on this point, I update our previous conceptualization of the anxiety-ERN relationship by drawing from the recently formulated expected value of control (EVC; [82•]) model of dACC function. In short, the EVC theory proposes that dACC integrates a range of valuation (e.g., from the amygdala) and salience (e.g., from the insula) information to determine the nature and intensity of the control signal that maximizes expected benefits relative to expected costs (i.e., expected value). This signal is then sent to and implemented by other regions in the cognitive control network (e.g., dlPFC). Thus, the dACC is involved in the decision to allocate a specific type and strength of cognitive control to a task given a set of costs and benefits associated with that control. The EVC theory further specifies that the dACC is principally involved in this decision process and not the valuation or implementation of control.

In the context of the EVC theory, we suggest that the ERN is one such dACC signal specifying the type and strength of cognitive control that will maximize the expected value on the task at hand. Thus, an enlarged ERN in anxiety can be understood as indexing the dACC specification of the type and strength of control signal that would maximize the expected value for anxious individuals. The most straightforward illustrative example that follows from our original CEMH is that enlarged ERN in anxiety may reflect an increase in the intensity of control specified by the dACC to overcome distracting worries. The dACC would make this decision presumably because the value of increased control intensity would be its ability to normalize performance in the face of distracting worries and reduced connectivity between dACC and dlPFC. This marriage of the CEMH with the EVC theory in accounting for the enlarged ERN in anxiety is further bolstered by the broader view that dACC is critically involved in the allocation of mental effort [83].

Because the CEMH conceptualizes the ERN as an index of control, and not evaluative (like the SETH), processes, it accounts for the developmental findings by again situating itself in the broader literature on the relationship between anxiety and cognitive control. For instance, the CEMH can accommodate a smaller ERN in anxious young children [35•] because it is consistent with related research showing that young children with anxiety-related problems show deficits in cognitive or effortful control (e.g., [84]). The Lo et al. [35•] study is particularly revealing because it showed that anxious young children not only had smaller ERNs but also performed less accurately and failed to demonstrate the expected relationship between an enlarged ERN and higher accuracy.

The CEMH seems to be able to accommodate many, but not all, of the developmental findings on the relationship between the ERN and anxiety. For instance, a developmental shift from a smaller to a larger ERN in anxiety may be attributable to developmental increases in cognitive control skills. Specifically, children who experience persistent anxiety over time may develop compensatory cognitive control, reflected in a larger ERN, that serves to maintain adequate performance at older ages. The complex, indirect relationships between the ERN and anxiety—identified in the aforementioned prospective studies—are more difficult for the CEMH to explain, however. Despite shortcomings of the CEMH in accounting for these complexities, there is convergence among many of the prospective studies that an enlarged ERN characterizes children who had previously been identified as at risk for anxiety and whose anxiety persists or grows [61, 62, 63•, 64]. The CEMH would attribute these effects to the development of compensatory cognitive control strategies to overcome the distracting effects of anxiety on performance that accumulate over time. Findings from McDermott et al. [65] and Troller-Renfree et al. [66] suggesting that a larger ERN relates to better behavioral and academic outcomes in institutionalized children support the compensatory conceptualization of the enlarged ERN in children at risk for anxiety and are consistent with our earlier findings that undergraduate

worriers with enlarged ERNs achieved higher grades in school than undergraduate worriers with smaller ERNs [12•].

Finally, it is important to consider the role that valuation and salience systems play in the generation of the ERN across development and how their contributions may influence the ERN's relationship with anxiety at different stages of development. Findings from Buzzell et al. [7•] suggest that whereas dACC and PCC sources of the ERN are stable across development, OFC, insula, and IFG sources increase with age. Together with the EVC model of dACC function, the Buzzell et al. findings suggest that OFC- and insula-based valuation and salience information contributes more to the error-related dACC decision process, indexed by the ERN, later in development. Thus, it is possible that this developmental increase in valuation and salience system contributions to the ERN accounts for the flip in the relationship between anxiety and the ERN across development. Early in development, error-related dACC function in anxiety might be reduced because worries interfere with the decision to allocate effortful control and there is less input from motivational regions to overcome them. Later in development, however, error-related dACC function in anxiety might be exaggerated because greater contributions of valuation and salience mechanisms increase the motivation to engage control because its expected value increases as the system learns it can compensate for interference from worries and impaired dACC-PFC functional connectivity by specifying increased control intensity. The increase in IFG contribution to the ERN with age found in Buzzell et al. also supports the notion that compensatory mechanisms play a larger role in the anxiety-ERN relationship later in development inasmuch as the IFG is related to inhibitory control and error correction.

Summary and Concluding Remarks

The relationship between the ERN and anxiety has received significant attention over the past 10+ years because of its potential to illuminate cognitive and motivational/affective mechanisms in anxiety that may ultimately serve as risk markers and intervention targets. The last 5 years, in particular, has seen an increased interest in understanding the nature of the relationship between the ERN and anxiety across development. There is a growing consensus in the literature that anxiety in young children may be associated with a reduced ERN whereas anxiety in older children, adolescents, and adults is characterized by an enlarged ERN. The trajectory of this relationship may reflect developmental shifts in cognitive control abilities and/or in concerns about internal sources of threat. Despite the boon of recent evidence to this area, however, there are a number of issues to address with future research that should help push the science forward.

First, a series of methodological concerns limits our ability to draw *clear* conclusions about developmental findings. For instance, measurement of the ERN is not consistent across development. Almost all the findings in children (e.g., [35•, 45]) demonstrate relationships between anxiety and the ERN—CRN difference score (ΔERN), and not the ERN itself, whereas adult studies demonstrate relationships between both the difference score and the ERN itself [12•]. This seems critical to address because these two ERN measures capture different variance across development, which may have significant implications for understanding their overlapping and dissimilar functional significances. Thus far, no research group has proposed an explanation for these measurement inconsistencies. Research has likewise not appreciated the contribution of different tasks to developmental relationships between the ERN and anxiety. Most research in children employs Go/No-Go tasks (e.g., [35•, 45]) whereas research in adolescents and adults typically employs Flanker tasks (e.g., [13•, 43]). These task choices might be important because they pull for different cognitive operations. Moreover, ERNs elicited by Go/No-Go and Flanker tasks only show moderate interrelations in adults [85] and adolescents [86] and no overlap in young children [34]. A better understanding of how different task parameters contribute to the relationship between the ERN and anxiety across development is therefore of great import. Research in this area must also better account for differences in measurement of anxiety-related problems across development. Studies of young children rely heavily on parent report and behavioral observation whereas adolescent and adult studies rely on self-report. Some studies focus on interviewer-rated diagnostic status [58] whereas others focus on interviewer-rated symptom dimensions [13•] or self-report [35•]. Even still, some studies employ specific measures of anxiety and its subcomponents of fear and worry [35•] whereas others utilize measures mixing anxiety and depression symptoms [63•]. Future research should also look to increase consistency of when anxiety and ERN measurements are taken that are informed by developmental theory.

Second, for the more complex interactions revealed by cross-sectional and longitudinal developmental studies, researchers should focus on understanding how the anxiety-ERN relationship changes over time or is affected by some other third variable (e.g., harsh parenting) rather than decomposing interactions to examine the relationship between anxiety and the third variable as a function of small versus large ERN amplitude (e.g., [63•]). The anxiety-ERN relationship is what we aim to understand. By focusing on the relationship between anxiety and another variable that might be moderated by ERN amplitude, the literature becomes more rather than less confusing because it is more difficult to draw conclusions about the relationship of interest—i.e., that between anxiety and error monitoring.

Third, considering recent evidence for a sex/gender difference in the relationship between enlarged ERN and anxiety in adults [42•], future studies should aim to be adequately powered to test sex/gender differences across development, particularly because clinically significant anxiety symptoms are more common in girls than boys from early childhood [87] and this female:male difference in anxiety disorder prevalence may increase in adolescence [88, 89]. Our group recently tested for a sex/gender difference in the relationship between the ERN and anxiety in young children and found no differences [35•]. Together with the noted sex/gender difference in adults and developmental changes in the relationship between the ERN and anxiety around adolescence, this result points to the interesting possibility that the pubertal transition might be an important stage of development to focus on, especially for females. This confluence of results also opens doors to considering other factors that might contribute to the ERN-anxiety relationship across development, namely sex hormones.

Finally, future work in this area would do well to create stronger links to related neuroscience research and theory on anxiety, cognitive control, and motivation/emotion across development. Our group, for example, has borrowed heavily from cognitive neuroscience research and theory on conflict monitoring [10] and cognitive control [67]. Our CEMH receives inspiration and support from these data and theories as well as others that demonstrate a compensatory function of the ACC in general [90] and with respect to anxiety in youth and adults in particular [91•, 92]. Exciting new themes emerging from research and theory on the development of cognitive and self-control include the proposition that frontal and limbic regions work in a reciprocal loop to build a regulatory circuit during adolescence to meet the developmental task of separation from caregivers [24•] and that increased modularization of the brain into functional networks supports the development of executive function from childhood to young adulthood [93•]. This work has widespread relevance to the ERN-anxiety literature. As mentioned previously, recent work suggesting that the PCC is another source of the ERN across development further underscores the importance of becoming familiar with PCC function. A recent integrative review by Leech and Sharp [39] suggests that the dorsal PCC, in particular, has strong connections with the frontal lobes and as such may serve to modulate attentional focus. Increased PCC function after errors may therefore reflect a shift to external focus of attention on corrective (target) information, consistent with conflict monitoring theory [10] and, as it relates to anxiety, the CEMH [12•, 37]. However, this interpretation of PCC function must also consider its role in the default mode network that has been implicated in cognitive control impairments in anxious populations (Fitzgerald et al. 2010; Menon 2011). Last, findings demonstrating error-related ACC activity does not seem to relate to online anxiety reactions, but rather to frustration [94], and ACC stimulation causes individuals to feel motivated to overcome an impending challenge [95] further call for us to consider the regulatory/motivational function of the ERN and its associated brain sources in anxiety across development.

Compliance with Ethical Standards

Conflict of Interest The author declares that he has no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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