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Development of the Error-Related Negativity and Behavioral Variability in Childhood: Clarification of Neural Processes Using Wavelet Analysis

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ABSTRACT

The error-related negativity (ERN) is an event-related potential that is thought to reflect neural activity related to performance monitoring. The ERN can be measured reliably in adults and relates to individual differences in cognitive abilities, psychopathology, and personality. However, questions remain about the reliability and validity of the ERN signal in children. As the ERN is typically measured over many trials, low levels of signal temporal consistency across trials in childhood may interfere with researchers' ability to detect an ERN signal in a child's averaged waveform. The present study sought to clarify the developmental trajectory of the ERN signal in childhood by assessing both average signal strength and signal temporal consistency of the ERN, using wavelets to examine dynamics in the theta waveband, in a sample of children assessed longitudinally in kindergarten, 1st, and 2nd grade. Consistent with previous findings, ERN deflections in the averaged waveform increased with age. It was found that signal temporal consistency also increased with age, lending support to the hypothesis that development of the ERN signal is characterized by an increase in signal temporal consistency. Additionally, the present study sought to determine the rank-order stability of ERN amplitude, signal temporal consistency, average signal strength, and behavioral variability in childhood. ERN amplitude and signal temporal consistency showed low levels of rank-order stability while average signal strength and behavioral variability showed high levels of rank-order stability over the age range assessed.

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Introduction

Electroencephalogram (EEG) measured neural activity during perceptual or behavioral events, termed event-related potentials (ERPs), have been used to assess neural information-processing. ERPs have been widely used to understand the neural basis of individual differences in cognition by examining differences in the amplitude and latency of ERP components between individuals of the same age. ERPs have widely been useful in understanding the neurological basis of cognitive systems in adults and researchers have leveraged the knowledge gained from adult populations to study the development of the cognitive systems underlying these ERPs in children. The use of ERPs in children, however, is complicated by ongoing developmental processes that have unknown effects on the ERPs. Careful consideration of the root cause of age-related differences in ERPs has the potential to increase understanding of the development of the cognitive systems underlying these ERPs.

The error-related negativity (ERN), which occurs within 100 ms following an erroneous response in various speeded response tasks, has been used to investigate cognitive processing related to performance monitoring (Falkenstein et al., 1991; Gehring et al., 1993; Scheffers & Coles, 2000). Performance monitoring is the process of evaluating the effect of one's actions in service of improving effectiveness of future behavior (Scheffers & Coles, 2000). Not surprisingly given the fundamental contributions that performance monitoring makes to behavioral learning, individual differences in ERN amplitude have been demonstrated in association with a broad range of domains including personality and psychopathology when studied in adults (Frank et al., 2005; Liotti et al., 2005; Olvet & Hajcak, 2008). Given this, investigations into ERN amplitude in children have the potential to provide developmental insights into the neurological basis of these domains. However, little is known about the

developmental progression of the ERN component and few studies have been conducted in children under the age of 8 years.

Early developmental research regarding the ERN indicated a lack of evidence for this component in children (Davies et al., 2004). However, individuals behaviorally exhibit the cognitive abilities associated with the ERN before the age at which an ERN signal is measureable, calling into question the reliability of measuring ERN amplitude in young children (Wiersema et al., 2007). It is possible, however, that the disconnect between the timing of the development of the ERN signal and the cognitive process the ERN is thought to reflect is an artifact of how the ERN is measured due to greater levels of intra-individual variability in ERN signaling in children. This possibility deserves added consideration given that children behaviorally exhibit greater levels of intra-individual variability than adults (Li et al., 2004). The present study investigated this possibility to increase understanding of the development of the ERN in children.

Performance Monitoring and the ERN

Measurement of the ERN signal is thought to index performance monitoring process necessary for the maintenance of goal oriented behavior and minimization of errors (Scheffers & Coles, 2000). Even when participants are aware of the correct stimulus-response mapping rules, erroneous responses increase when task instructions emphasize speeded responses. Following such an errors, an ERN signal is generated that reflects early error awareness (Wessel, 2012). A related ERP, the feedback error related negativity (fERN) is thought to reflect similar neural activity (Holroyd and Coles, 2002, Miltner et al., 1997). In tasks where external feedback is required for the participant to know an error has been committed, a fERN is generated following feedback of a mistake in similar neural regions as the ERN (Miltner et al., 1997). In both cases,

the neural activity reflects evaluation of the effectiveness of current behavior, allowing for the rapid recruitment of additional cognitive resources, when needed, in pursuit of improving future task performance. As expected from a signal that indexes performance monitoring, ERN deflections, a measure of the strength of the ERN signal, increase as a function of the motivation for accuracy. Individuals show a greater ERN deflections when instructed to prioritize response accuracy (Gehring et al., 1993), when monetary rewards are used to incentivize accuracy (Kim et al., 2005), and when performance is observed by a friend (Hajcak et al., 2005). Additionally, studies consistently show a link between larger ERN deflections and success in performing speeded response tasks (Gehring et al., 1993).

Two hypotheses have been put forth to further clarify the role of the ERN signal in performance monitoring. The Error Detection hypothesis posits that the ERN is the output of a generic error detection system (Gehring et al. 1993; Holroyd & Coles, 2002). During a speeded response task, participants may be forced to initiate a motor response before they can be certain of the correct response. Even after initiation of an incorrect motor response, participants will continue to process the stimulus and improve their internal representation of the correct choice. When the correct response becomes evident following the initiation of an incorrect motor response, an ERN signal is produced signifying that a mistake has been made (Falkenstein et al., 2000). This signal serves to increase cognitive resources to decrease the likelihood of future mistakes (Botvinick et al., 2001; Ridderinkhof et al., 2004).

Alternatively, the Conflict Monitoring hypothesis postulates that the ERN emerges from conflicts in information processing (Botvinick et al., 2001). Internal conflict is a sub-optimal state in which an individual experiences ambiguity. This ambiguity may cause a delay in choosing a behavior resulting in an ineffective exploitation of resources within given time

constraints. In the context of a speeded response task, the level of conflict will depend on the relative activation of competing correct and incorrect responses. Even after the initiation of an incorrect motor response, the correct response may be identified, resulting in conflict due to multiple response plans being simultaneously active. The ERN, according to the Conflict Monitoring hypothesis, signifies the co-activation of multiple incompatible response plans and serves to recruit additional cognitive resources to resolve the conflict and decrease the likelihood of future conflicts.

The cognitive functions attributed to the ERN by the Error Detection and Conflict Monitoring hypotheses are similar. Both view this activity as part of a performance monitoring system in pursuit of improving future behavior (Botvinick et al., 2001; Holroyd & Coles, 2002). This performance monitoring system recruits additional cognitive resources when sub-optimal performance is detected. Although not primarily specified in the Conflict Monitoring hypotheses, a conflict monitoring system can function as an error detector (Yeung et al., 2004). Since more conflict occurs on error trials, a conflict-error cutoff can be defined such that any trial with conflict levels above the cutoff will be presumed to be an error. Computer simulations have shown that such a system can detect errors at a level consistent with that seen in experimental studies (Rabbitt, 2002; Yeung et al., 2004). Because of this, it has been argued that the Conflict Monitoring hypothesis is more general, with the Error Detection hypothesis explaining a subset of the cases explained by the Conflict Monitoring hypothesis (Yeung et al., 2004).

Neural Basis of the ERN. Post-error performance monitoring activity, believed to occur in the anterior cingulate cortex (ACC), recruits additional cognitive resources, primarily believed to be the dorsolateral prefrontal cortex (DLPFC), to decrease the likelihood of future errors (Botvinick et al., 2001; Bush et al., 2005). EEG dipole source localization techniques implicate

the ACC as the probable generator of the ERN (Dehaene et al., 1994; Holroyd et al., 1998) and ACC activity has been detected following errors by single cell and local field potential recordings in non-human primates (Emeric et al., 2008; Ito et al., 2003; Niki & Watanabe, 1979), intra-cranial recordings from epileptic patients (Brázdil et al., 2005), and fMRI activation patterns (Debener et al., 2005; Mathalon et al., 2003) Additionally, a study of patients with lesions to the ACC supports the role of the ACC in the generation of the ERN signal (Stemmer et al., 2004). This post-error ACC activity predicts DLPFC activity on subsequent trials and successful behavioral adjustments (Cavanagh et al., 2009; Kerns et al., 2004; MacDonald et al., 2000).

Individual Differences in Performance Monitoring

The ERN signal provides insights into the neural basis of individual differences and has been broadly linked to cognitive abilities, psychopathology, and personality. One study found that individuals who show greater ERN deflections performed better at tasks indexing working memory and set shifting (Larson & Clayson, 2011). Another study found that greater ERN deflections predicted higher academic performance among college students (Hirsh & Inzlicht, 2010). These larger ERN deflections may reflect greater performance monitoring allowing for more effective engagement of cognitive capacities. Larger ERN deflections are also seen in individuals with anxiety disorders (Hajcak et al., 2003), depression (Tucker et al., 2003), and obsessive-compulsive disorder (Gehring et al., 2000). These differences extend to related personality differences in normative, non-clinical samples with individuals high in negative affect and trait anxiety also showing more negative ERN deflections (Boksem et al., 2006; Hajcak et al., 2004). These findings are interpreted as representing greater levels of performance

monitoring amongst populations that have increased sensitivity to errors and negative feedback (Olvet & Hajcak, 2008).

Contrarily, muted ERN amplitudes have been found in individuals with substance use disorders (Franken et al., 2007), and attention deficit-hyperactivity disorder (Liotti et al., 2005), as well as individuals high in a latent externalizing factor (Hall et al., 2007). The muted ERN deflections seen among these populations are believed to reflect decreased sensitivity to errors and negative feedback, which may result in continuation of maladaptive behaviors despite negative outcomes. A study on learning style provides additional support for these interpretations of individual differences in ERN amplitude representing differences in error sensitivity, as individuals who learn by avoiding negative consequences (i.e. negative reinforcement) have larger ERN deflections than those who learn by seeking positive outcomes (i.e. positive reinforcement) (Frank et al., 2005). An understanding of the ERN in children may provide insights into the development of these individual differences, but the development of the ERN must be well delineated first.

Development of the ERN

Although children at a young age are able to recognize errors, accurately complete speeded response tasks, and exhibit performance monitoring abilities, the reliability of measurement of an ERN signal in younger children is questioned. Children and adolescents consistently show smaller ERN deflections than adults and detailed cross-sectional studies show a clear linear increase in ERN signal strength into adulthood (Davies et al., 2004; Kim et al., 2007; Wiersema et al., 2007). The age at which the ERN signal becomes apparent, however, is less clear. While some children show a signal at 7 years old, an ERN signal does not become apparent when averaged across individuals until early adolescence (Davies et al., 2004). This

contradiction between an individual's behavioral capability to exhibit performance monitoring and our inability to capture brain processing believed to index performance monitoring remains unexplained.

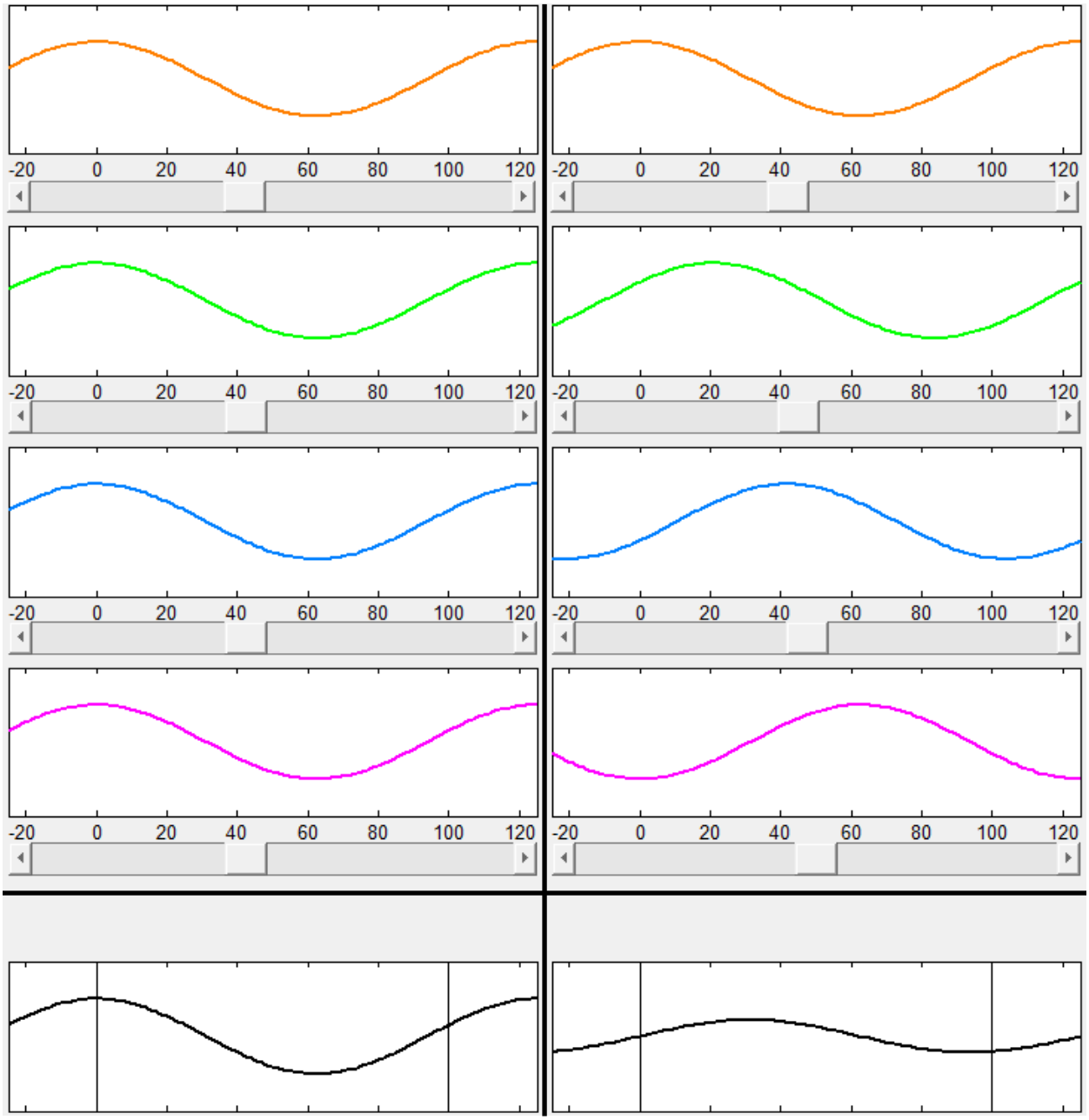
Given the ongoing development of the neural regions generating the ERN signal through adolescence and the variability between individuals in the age at which the signal can be detected, the earliest age at which measurement of the ERN can provide meaningful insights remains unclear. One study was able to identify an ERN signal in children aged 5-7 (Torpey et al., 2009). But unlike adults, these children's ERN deflections did not increase when they were told to focus on accuracy, indicating that the ERN did not reflect the level of performance monitoring the participants were exhibiting. In a group of children 7 to 8 years old, researchers identified an ERN signal and found that participants who performed the task under supervision of a friend had greater ERN deflections than those who performed the task alone (Kim et al., 2005). Another study found the expected relationship between greater ERN deflections and anxiety in 11-13 year olds, although they failed to find the relationship in 8-10 year old children (Meyer et al., 2012). The failure to consistently find the expected linkages with ERN deflections in younger children could be explained by lack of consideration of within-person temporal variability in signal responding, as discussed below.

Potential Impact of Variability on the ERN

To help distinguish neural activity related to a cognitive process of interest from background neural activity that is unrelated to the cognitive process of interest, EEG measured activity is measured over many trials. When trials are averaged, waveforms deriving from non-systematic activity cancel each other and waveforms locked to the event of interest (i.e. ERPs) emerge more clearly. This process, however, assumes that the signal occurs in a temporally

stable place in each trial. If there is variability in signal latency, the average waveform will show a muted signal that is not representative of the actual signal in any trial. Traditional measures of ERN amplitude will thus be a function of the signal strength in each trial and the temporal stability of the signal across trials, as shown in Figures 1 and 2. If traditional ways of quantifying the ERN are correct in assuming there is no temporal variability in signal location, average ERN amplitude across trials will accurately reflect the average signal strength in each trial. However, to the extent that there is temporal variability in the signal across trials, traditional measures of ERN amplitude will underestimate the average signal strength in each trial.

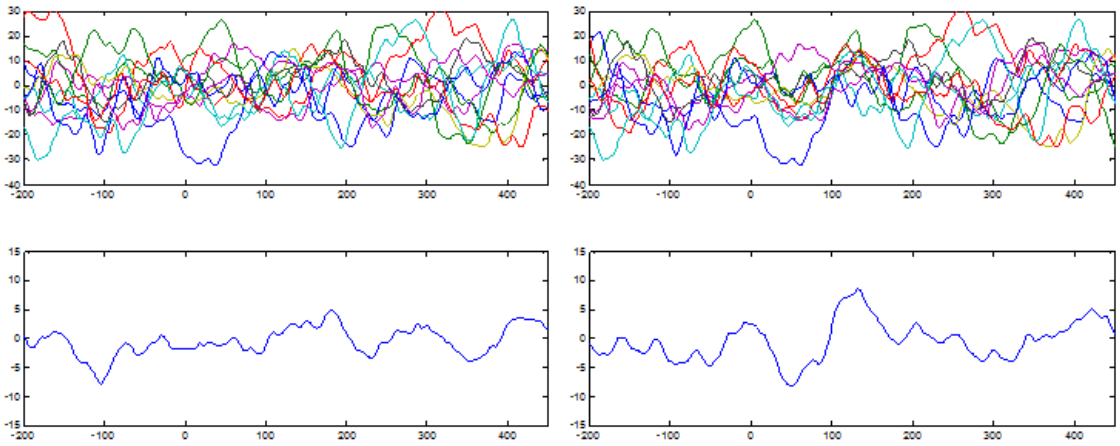
Figure 1. Effect of Low Signal Temporal Consistency on an Average Waveform.



Notes: Depiction of trials (upper 4 boxes on each side) and average waveform of those trials (bottom box on each side) for a simulated participant with high signal temporal consistency (left side) and low signal temporal consistency (right side). For the participant with low signal temporal consistency, the average waveform (bottom right box) is muted and not representative

of the amplitude in the trials that were used to make up the average waveform (upper 4 boxes on the right side).

Figure 2. Single and Averaged ERN Waveforms and the Effect of Signal Temporal Consistency.



Notes: Depiction of response-locked, post-error single trials (upper 2 boxes) and averaged ERN waveform (bottom 2 boxes) from a sample participant before correcting for inconsistency in signal latency (left side) and after correcting for inconsistency in signal latency (right side). All trials are from the same participant. A clear ERN deflection 50 ms post-response is evident in the corrected average waveform (bottom right box) but not the uncorrected average waveform (bottom left box).

Assessing Variability with Time-Frequency Decomposition

Researchers have postulated that the ERN signal apparent in average waveforms results from an increase in phase-locked activity in the theta (4-8 Hz) waveband (Luu et al., 2004; Trujillo & Allen, 2007). In this model, signal strength in each trial is estimated using sine waves with frequencies in the theta range. The sine waves that describe each trial can differ in their power and their phase offset. The power of the sine waves that describe a trial is defined by the amplitude of the sine waves and indicates the strength of the signal in a given trial. The phase offset of the sine waves that describe a trial indicate the temporal location of the peaks and

troughs of the sine waves in a given trial. Summary scores of average theta power and phase coherence in the theta waveband can then be created across trials. Average theta power, representative of average signal strength across trials, is a simple average of the power in the theta waveband in each trial. Phase coherence in the theta waveband across trials, representative of signal temporal consistency across trials, measures how consistently the peaks and troughs of the sine waves that describe each trial align in temporal space. Average theta power and phase coherence in the theta waveband across trials explains a majority of the variance of the ERN (Cavanagh et al., 2012). Thus, characteristics in the theta waveband are a validated metric to explore how differences in average ERN amplitude are due to both average signal strength and signal temporal consistency across trials.

Explanatory Value of Signal Temporal Consistency. Researchers have begun to show the utility of assessing signal temporal consistency in neural processes related to diverse cognitive functions. Consideration of signal temporal consistency has proved beneficial for predicting successful memory encoding (Fell et al., 2008; Klimesch et al., 2004) and memory retrieval (Schack et al., 2002). Additionally, decreased levels of signal temporal consistency in neural activity related to working memory, attention, and information processing have been used to differentiate participants with schizophrenia from controls (Ergen et al., 2008; Rentrop et al., 2001; Roth et al., 2007). These studies cast doubt on the underlying assumption of ERPs that the signal occurs in a temporally stable location and highlights the potential utility in including signal temporal consistency in the analysis of ERP components.

Importance of Consideration of Signal Temporal Consistency in ERN Development

Additionally, some research has shown support for age-related differences in neural activity to be characterized as a function of differences in both average signal strength and signal

temporal consistency. However, the only study specifically investigating age-related changes in ERN as a function of age-related changes in average signal strength and signal temporal consistency focused on aging in older adults. Mirroring the developmental increases in average ERN amplitude that are seen through late adolescence, researchers have shown a developmental decrease in ERN deflections late in life (Band & Kok, 2000; Falkenstein et al., 2001). Researchers found that the age related decrease in average ERN amplitude seen in old age was due to both a decrease in average signal strength and a decrease in signal temporal consistency (Kolev et al., 2009).

Although researchers have speculated that the developmental increase in average ERN amplitude through adolescence may be due to an increase in signal temporal consistency in addition to an increase in average signal strength (Gatzke-Kopp et al., under review; Segalowitz & Dywan, 2009), the hypothesis remains largely unexplored. A study investigating age-related differences in auditory perception found children to have greater levels average signal strength and lower levels of signal temporal consistency than adults (Müller et al., 2009). The researchers postulated that the increased neural activity allowed the children to perform the task proficiently despite lower levels of signal temporal consistency than adults.

Age-Related Differences in Behavioral Variability

An increased level of behavioral variability in children compared to adults (Li et al., 2004) is one reason to believe that ERN development may be partially characterized by an increase in signal temporal consistency. Behavioral variability indicates the level of inconsistency with which an individual performs a task and is typically measured as the standard deviation of response times. This inconsistency in responding is thought to reflect inefficiencies in neural systems underlying task performance and has been found to provide insights into

individual differences beyond mean response times and task performance (Epstein et al., 2003; Jensen, 1992; Segalowitz & Segalowitz, 1993). This variability has been found to be related to cognitive abilities, and the increased level of behavioral variability in children is thought to reflect developmentally immature neural systems (Aggarwal & Lillystone, 2000; Hultsch & MacDonald, 2004; Jensen, 1992; Li et al., 2004). Similarly, an increased level of behavioral variability has been found in the elderly compared to middle aged and young adults (Anstey, 1999). This increase in behavioral variability in the elderly has been attributed to decreased efficiencies of neuronal processing and has been linked to cognitive decline (Hultsch et al., 2002; MacDonald et al., 2003). It is possible that inefficiencies in developmentally immature neural systems, and corresponding inefficiencies related to cognitive decline, could result in increased variability in neural processing, resulting in greater signal temporal inconsistency.

Neural Systems Potentially Impacting ERN Signaling and Behavioral Variability

Dopamine System. There is strong correlative evidence to link dopaminergic networks to both behavioral variability and the ERN (Bäckman et al., 2006; Holroyd & Coles, 2002; Li & Lindenberger 1999). Developmental changes in the ERN and behavioral variability mirror the developmental trajectory of prefrontal dopamine (DA) receptors. Just as behavioral variability reaches minimal levels and the ERN reaches maximal levels in adulthood, DA receptor density in the prefrontal cortex of rats and prefrontal innervation by DA neurons in non-human primates increases throughout childhood until reaching stable levels in early adulthood (Lambe et al., 2000; Tarazi & Baldessarini, 2000). Aging is characterized by a decrease in frontal DA receptors, a muting of ERN deflections, and an increase in behavioral variability (Bäckman et al., 2006; Hultsch et al., 2002). One study found that age related increases in behavioral variability are related to a decrease in dopamine D_1 receptor binding potential in frontal regions, including

the ACC (MacDonald et al., 2012). Individuals with Parkinson's disease, a neurodegenerative disorder affecting midbrain DA neurons, show diminished ERN amplitudes and increased behavioral variability (Willemssen et al., 2008). DA agonists increase ERN deflections and decrease behavioral variability, while DA antagonists decrease ERN deflections (de Bruijn et al., 2006; Jonkman et al., 2007; van der Meere, 1995).

White Matter Integrity. Structural myelination patterns show mirrored trajectories early and late in life and could potentially provide a neural basis for a link between ERN signaling and behavioral variability. White matter integrity increases into early adulthood (Benes, 1989; Yakovlev & Lecours, 1967) and increased myelination has been linked to improved cognitive capabilities in children and adolescents (Nagy et al., 2004; Schmithorst et al., 2005). Similarly, white matter integrity decreases late in life and decreased white matter integrity has been linked to age-related cognitive decline (Charlton et al., 2006; Madden et al., 2009; Sullivan & Pfefferbaum, 2006). Reduced white matter integrity has also been found to be associated with behavioral variability in samples of healthy adults (Bunce et al., 2007; Fjell et al., 2011; Ullén et al., 2008). Increased levels of myelination have been interpreted, in these studies, as allowing for the more efficient transmission of neural signals resulting in decreased behavioral variability. Although less evidence exists to support the role of white matter integrity in ERN signaling, one study has linked decreased ERN deflections to decreased white matter integrity in a sample of healthy adults (Westlye et al., 2009).

Relationship Between the ERN and Behavioral Variability. Although there is considerable overlap in the research focus of those studying the ERN and behavioral variability, rarely have these two measures been investigated concurrently. One such study, however, found a strong relationship between these two measures (Richardson et al., 2011). Amongst a group of

7- to 9-year olds, individuals with greater behavioral variability showed muted ERN deflections. No published study has attempted to find associations between behavioral variability and temporal variability in ERN signaling.

Gender Differences in the ERN and Behavioral Variability

Considering that females are more likely than males to be diagnosed with anxiety disorders and depression, disorders in which individuals exhibit greater ERN deflections, and that males are more likely than females to be diagnosed with externalizing disorders, disorders in which individuals exhibit muted ERN deflections, it may be expected that females would exhibit greater ERN deflections than males (Kessler et al., 1994). The relation between gender and the ERN amongst normative populations, however, is mixed. Studies have found no gender difference in the ERN in young children, although gender differences appear to emerge in late adolescence with males having greater ERN deflections than females (Davies et al., 2004; Torpey et al., 2012). In adults, one study found no gender differences in fERN amplitude (Hiraki et al., 2006), but gender differences in ERN amplitude were observed in another study such that that males exhibited greater ERN deflections than females (Larson et al., 2011). Findings from studies assessing gender differences in error-related ACC fMRI activation patterns are inconsistent as well with reports in the literature of greater activation in males (Li et al., 2006), greater activation in females (Liu, 2012), and no gender differences in activation (Hester et al., 2004). The functional significance of potential gender differences in ERN and error-related ACC signaling is unclear and potential interpretations may be premature given the lack of robust findings, but it is possible that males and females may engage different brain regions and use different strategies to achieve similar levels of performance (Li et al., 2006). Additional research must be done to clarify potential sex difference in these neural processes.

The relation between gender and behavioral variability is less mixed, with males typically showing lower levels of behavioral variability than females in adults (Deary & Der 2005; Dykiert et al., 2012). These gender differences appear to emerge in adolescence (Dykiert et al., 2012) and studies have reported no gender differences in behavioral variability amongst younger children (Dykiert et al., 2012; Eckert & Eichorn, 1977). The functional significance of gender differences in behavioral variability in adulthood is unclear and it is possible that controlling for potential gender differences may be the most prudent method for investigating the relation between behavioral variability and neural functioning (Dykiert et al., 2012; Tamnes et al., 2012).

Rank-Order Stability of the ERN and Behavioral Variability

As reviewed above, the ERN and behavioral variability are associated with individual differences in a variety of domains in adults and older children, and individual differences found in young children could therefore be predictive of later developmental outcomes and could provide insight into the etiology of certain mental disorders (Olvet & Hajcak, 2008; Segalowitz et al., 2010). However, the predictive validity of these measures in young children is dependent on the rank-order stability, or the trait-like nature, of the measures over development. Any measure with high rank-order stability could be considered a trait-like measure as it would reliably index enduring patterns of individual differences. The rank-order stability of a measure is affected by both the reliability of the measure at any given point in development and the degree to which differential developmental trajectories amongst individuals shift the rank-ordering of individuals. The rank-order stability of a measure is not, however, influenced by normative (i.e. mean level) changes over development. If individuals develop at a similar pace, the rank-order of individuals will not change even if there are mean level changes over a given age range. Age can be used as a proxy into development, but different individuals and different

systems within an individual may develop at different paces. Individual differences found in a system under development at any given age may be capturing lasting individual differences or they could reflect temporary between-person differences due to uneven developmental paces of a system. An investigation into the rank-order stability of a measure over development can provide insights into the persistence of individual differences and thus is complimentary to the investigation of normative development.

Very little research has assessed the rank-order stability of the ERN and behavioral variability over development in childhood, although a handful of studies have provided evidence for high test-retest reliability of these measures over short time periods in adolescents and adults. Using Cohen's suggestions (1992) where Pearson's correlations of .10, .30, and .50 indicate a small, moderate, and high level of association between measures, respectively, the ERN has shown high test-retest reliability over a two-week period in a sample of undergraduate students and over the course of 3-6 weeks in middle adolescence (Olvet & Hajcak, 2009; Segalowitz et al., 2010). Moderate rank-order stability of the ERN has also been found amongst college students over the course of 1-2 years (Weinberg & Hajcak, 2011). Behavioral variability shows moderate to high test-retest correlations for adult and college-aged populations with 7 days between assessments (Flehmig et al., 2007; Saville et al., 2011). The rank order stability of the ERN and behavioral variability amongst younger populations, however, has yet to be assessed.

Aims and Hypotheses

The overarching goals of the current study are to assess the development of behavioral variability and the ERN and the relation between behavioral variability and the ERN in childhood. To achieve these aims, neurophysiological and behavioral assessments of children followed longitudinally in kindergarten, 1st grade, and 2nd grade were analyzed.

Aim 1: Assessing Normative Development of the ERN and Behavioral Variability.

Previous research indicates that ERN deflections become more negative throughout late childhood and adolescence and that there are no gender differences in ERN amplitude for participants in this age range (Davies et al., 2004; Torpey et al., 2012). No study has assessed the extent to which an increase in average signal strength and/or signal temporal consistency might explain the increase in ERN signal seen across childhood or if there are gender differences in average signal strength and signal temporal consistency. A study assessing the decreased ERN deflections in old age, however, found that decreased ERN signaling was caused by a decrease in both average signal strength and a decrease in signal temporal consistency (Kolev et al., 2009).

Consistent with previous research, it is hypothesized that individuals' ERN amplitudes will become more negative over the course of the study and that there will be no gender differences in the magnitudes of ERN deflections. Mirroring changes in late adulthood of average signal strength and signal temporal consistency, it is further hypothesized that an increase in average signal strength and signal temporal consistency will account for the age-related changes in ERN amplitude in the present study. Replicating previous findings of participants in similar age ranges, it is hypothesized that behavioral variability will decrease over this time period and there will be no gender differences in levels of behavioral variability (Eckert & Eiehorn, 1977).

Aim 2: Assessing Rank-Order Stability of the ERN and Behavioral Variability.

Previous studies have indicated that the ERN and behavioral variability show moderate test-retest reliability (Hagen et al., 2007; Olvet & Hajcak, 2009; Segalowitz et al., 2010; Weinberg & Hajcak, 2011), however the longer term rank-order stability of these measures has not been assessed in this age range. It is hypothesized that there will be moderate to low rank-order

stability of the ERN and behavioral variability since differential developmental paces may interfere with the rank ordering of these measures. Similar to ERN amplitude, low to moderate stability of average signal strength and signal temporal consistency is also expected since these two measures define ERN amplitude.

Aim 3: Assessing the Relationship between the ERN and Behavioral Variability. A plethora of indirect evidence suggests a potential relationship between the ERN and behavioral variability, with one study finding a direct link between the ERN and behavioral variability in a group of 7- to 9-year old children (Richardson et al., 2010). It is not clear, however, if the relationship between ERN and behavioral variability is stable across development. Additionally, no study has attempted to link behavioral variability to average signal strength or signal temporal consistency. Replicating the earlier finding, it is hypothesized that the ERN will be related to behavioral variability such that individuals with more negative ERN amplitudes will show less behavioral variability. Mirroring the predicted relationship between the ERN and behavioral variability, it is additionally hypothesized that behavioral variability will be negatively correlated with both average signal strength and signal temporal consistency.

Methods

Participants

Data for this study were drawn from a larger clinical trial of an intervention program for early-onset aggression that assessed children's development across three years (kindergarten through 2nd grade). The clinical trial was implemented in cooperation with 10 elementary schools in a district serving a low-income urban community. Prior to the study, kindergarten teachers were asked to complete a short screening form assessing aggressive behaviors for each child in their classroom (the Teacher Observation of Child Adaption-Revised; Werthamer-Larsson et al., 1991). From these reports, 339 children and their parents were recruited into the study. Of these, 207 children were among the upper 20% of the aggression ratings within each classroom, and 132 were among the bottom 20%. A random half of the children rated as the most aggressive were designated to receive the intervention. Physiological assessments were conducted with all participants during their kindergarten (K), 1st grade (G1), and 2nd grade (G2) school years. Further details about participant recruitment, procedures, and the clinical trial can be found elsewhere (Gatzke-Kopp et al., 2012). In the present study examining typical development, we focus only on longitudinal data obtained from the $N = 234$ non-intervention participants who had usable EEG data for at least one assessment. Sample demographics for those included in the study are presented in Table 1. This sample did not differ from the full sample on ethnicity ($\chi^2(3) = .10$, $p = .99$), gender representation ($\chi^2(1) = .04$, $p = .84$), or age upon entering school ($t = 1.36$, $p = .17$).

Table 1. Sample Demographics.

Characteristic	*n*	(%)
Gender		
Male	153	(65%)
Female	81	(35%)
Ethnicity		
Black	163	(70%)
Caucasian	20	(9%)
Hispanic	49	(21%)
Asian	2	(1%)

Notes: *N*=234.

Procedures

Physiological assessments took place in a recreational vehicle that was retrofitted as a mobile laboratory. The mobile laboratory was driven to the school allowing for testing during the school day. Children were escorted from class to the mobile laboratory and the testing procedures were explained to them. After obtaining the child's verbal assent, RAs affixed autonomic and EEG electrodes onto the children. Children were seated in front of a computer monitor and RAs initiated recording of physiological data. The testing procedure for each participant included a Go/No-go task followed by an emotion induction task. Only EEG data from the Go/No-go task was examined in the present study.

Go/No-go. The Go/No-go task used was a modified version of a program developed by M. Lewis and J. Stieben (Lewis et al., 2006). At the start of each trial, either a cartoon character (K) or an animated human face (G1 and G2) was presented. Participants were instructed to press a response box after the presentation of every stimulus (Go trial) unless the newly presented stimulus matched the immediately preceding stimulus (No-go trial). The task consisted of 330 trials, 105 of which were No-go trials. The task adaptively modified the difficulty for each participant by increasing or decreasing the speed of response required for a Go trial to be

considered correct to target a 50% error rate on No-go trials. Due to a speed-accuracy tradeoff, an increased response speed requirement on Go trials will result in more errors on No-go trials. This was done to ensure there were enough No-go error trials to analyze, as well as to avoid confounds between performance and ability.

Points were awarded for correct responses and subtracted for incorrect responses. A cumulative point total was graphically shown with a thermometer bar after each set of approximately ten trials, presented alongside a cartoon character who was smiling and giving a "thumbs up" or frowning and giving a "thumbs down" depending on the participant's performance since the previous feedback.

The task consisted of three blocks and during the 1st and 3rd block, participants earned more points for correct responses than they lost for incorrect responses. During the 2nd block, participants lost more points for an incorrect response than they gained for a correct response. These rules resulted in participants, on average, gaining points during the 1st and 3rd block and losing points during the 2nd block, regardless of actual performance. To maximize the number of trials available for the present analyses, data were aggregated across the blocks. The contextual effect of winning versus losing points was precluded by this aggregation procedure.

EEG Recording. Brain activity was measured throughout the behavioral task using a 32-electrode Biosemi EEG System (BioSemi, Amsterdam, Netherlands). To detect and later correct ocular artifacts, additional electrodes were placed on the left and right suborbital ridge under the pupil and 1 cm outside the left and right lateral canthi. Data were sampled at 512 Hz using Actiview Software.

EEG Post-Processing. Data were post-processed using Brain Vision Analyzer 2.0. Specifically, time-series for each electrode were re-referenced to the average of all sites and

bandpass filtered between 1 and 30 Hz. No-go trial errors of commission were segmented from –800ms to 800ms with respect to button presses indicating the erroneous responses. Corrections for eye blinks were made using the Gratton and Coles algorithm (Gratton et al., 1983) and data were baseline corrected relative to the average amplitude in the window –600 to –400ms prior to the incorrect response. Segments containing a voltage step greater than 100μV between sampling points or a data point outside the range of -75μV to 75μV were marked as artifact and removed. Assessment occasions at which participants had fewer than 5 No-go error of commission trials after artifact rejection were removed from the analysis (15 assessment occasions removed). Additionally, assessment occasions at which participants had error rates on No-go trials exceeding 60% were removed due to the participant potentially being disengaged from the task (7 assessment occasions removed). For the present study, processed data from the Fz electrode were exported to MATLAB and analyzed using custom written software.

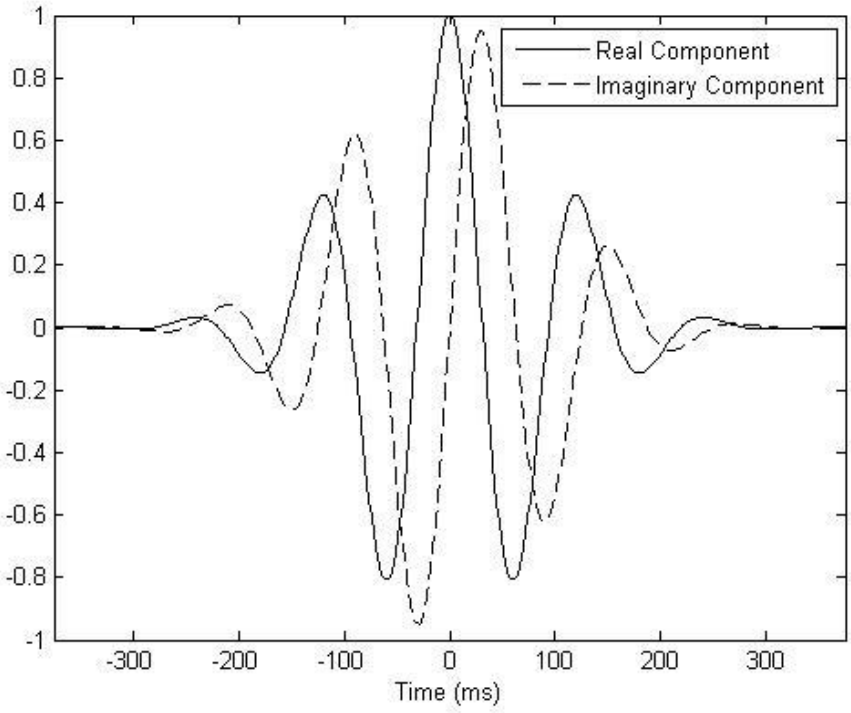
Time-Frequency Decomposition. Data from single trials underwent time-frequency decomposition using a set of complex Morlet wavelets to allow for the analyses of the spectral properties of participants' neural activity. The complex set of Morlet wavelets are depicted in Figure 3 and given by

$$\Psi(t,f) = \frac{1}{\sqrt{\pi\sigma_t}} e^{\frac{-t^2}{2\sigma_t{}^2}} (e^{2i\pi ft} - e^{\frac{-\alpha^2}{2}}) \quad (1)$$

where *t* is time, *f* is frequency, σ_t is the standard deviation in time domain around frequency f and α is a scaling factor typically set to 4.7 (Cavanagh et al., 2012; Grossmann & Morlet, 1984). Wavelets with a fixed width of 750 ms, corresponding to between 3 and 6 wave cycles for the frequencies of interest, were generated in 1 Hz frequency steps and were convolved with the No-go error of commission trials, one trial at a time, to yield $W_k(t,f)$. The resultant wavelet transformation represented in Cartesian coordinates is of the form $W_k(t,f) = a + bi$ (where *i* is

the square root of -1). $W_k(t, f)$ can alternatively be described in Polar coordinates as a function of magnitude and phase offset as shown in Figure 4 and Equations 2 and 3. These representations of neural activity will be used to obtain measures for average signal strength and signal temporal consistency across trials.

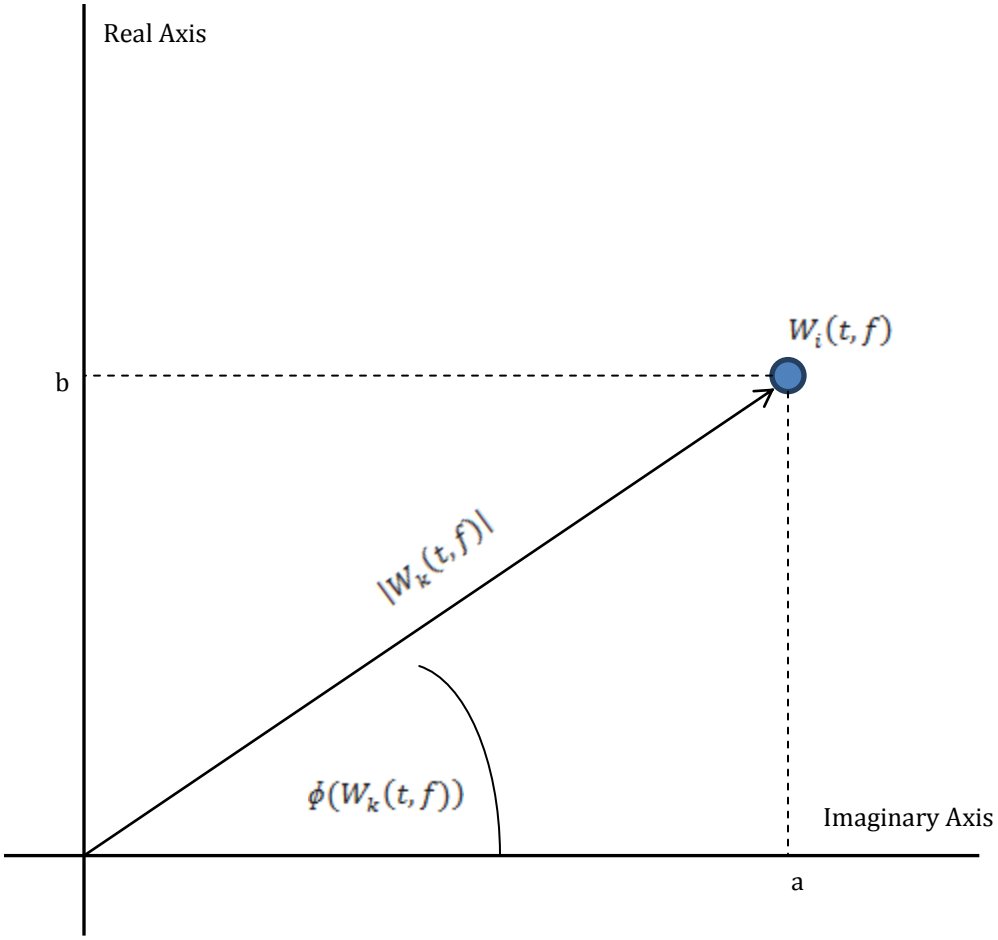
Figure 3. Sample Wavelet Corresponding to 8 Hz From the Set of Complex Morlet Wavelets Generated for the Analysis.



$$|W_k(t,f)| = \sqrt{a^2+b^2} \tag{2}$$

$$\phi(W_k(t,f)) = \begin{cases} \arctan\left(\frac{b}{a}\right) & if\ a < 0 \\ \arctan\left(\frac{b}{a}\right) + \pi & if\ a > 0\ and\ b \geq 0 \\ \arctan\left(\frac{b}{a}\right) - \pi & if\ a > 0\ and\ b < 0 \end{cases} \tag{3}$$

Figure 4. Transformation of Resultant of the Wavelet Transformation From Cartesian Coordinates to Polar Coordinates.



Measures

ERN Amplitude. ERN amplitude was calculated at each assessment (K, G1, G2) using a peak-to-peak method. First, a composite waveform was calculated for each participant by averaging the waveforms from artifact-free No-go error of commission trials. ERN amplitude was then calculated from the average waveform as the difference between the most negative peak in the window between 0 to 100ms after the response and the most positive peak in the window from −100 to 0ms prior to the erroneous response. At the sample level, ERN amplitude

showed a negative skew at each assessment. Data were reverse coded and log transformed to adjust for the negative skew. Data were then reverse coded again to preserve interpretation where more negative values for ERN amplitude indicate greater ERN deflections and more performance monitoring related neural activity.

Average Signal Strength. Average signal strength from each assessment was defined as the average spectral power across trials in the theta waveband, 4-8 Hz, from 0 to 100ms following erroneous responses on error trials as given by

$$Average\ Signal\ Strength = \frac{1}{n*c}\sum_{k=1}^{n}\sum_{t=0}^{100}\sum_{f=4}^{8}|W_k(t,f)| \quad (4)$$

where *n* is the number of trials, *t* is time in ms, and *f* is frequency in Hz.

Signal Temporal Consistency. Signal temporal consistency from each assessment was defined as the phase coherence in the theta waveband, 4-8 Hz, from 0 to 100ms following erroneous responses across error trials as given by Equation 5.

$$Signal\ Temporal\ Consistency = \frac{1}{n*c}\sum_{k=1}^{n}\sum_{t=0}^{100}\sum_{f=4}^{8} e^{i(\angle(W_k(t,f))-\overline{\angle(W_k(t,f))})} \quad (5)$$

The across-person distribution of signal temporal consistency was log transformed to adjust for a positive skew that was evident at each assessment.

Behavioral Variability. Behavioral variability from each assessment was defined as the standard deviation of response times on Go trials in which the participant responded between 100 ms and 1000 ms after presentation of the stimulus. Participants had, on average, 184 trials Go trials that met this criteria (range = 70 to 252).

Statistical Analysis

Assessing Rank Order Stability of Performance Monitoring and Behavioral Variability. Rank order stability across years was examined using pairwise Pearson's

correlations between the ERN amplitude, average signal strength, signal temporal consistency, and behavioral variability scores obtained at K, G1, and G2.

Assessing Normative Development of Performance Monitoring and Behavioral Variability. To examine age-related changes and gender differences in development, multilevel models of change were fit to the repeated measures of ERN amplitude, average signal strength, signal temporal consistency, and behavioral variability. Specifically, the model was specified as

$$\begin{aligned} Y_{tj} &= \beta_{0i} + \beta_{1j} Age_{tj} + e_{tj} \\ \beta_{0j} &= \gamma_{00} + \gamma_{01} Gender_j + u_{0j} \\ \beta_{1j} &= \gamma_{10} + \gamma_{11} Gender_j + u_{1j} \end{aligned} \quad (6)$$

where Y_{tj} is observation *t* for participant *j*, β_{0j} is the individual specific intercept for participant *j*, β_{1j} is the individual specific slope for age for participant *j*, γ_{00} is the mean intercept of the outcome, γ_{01} is the mean effect of gender on the intercept, γ_{10} is the mean linear slope for age, γ_{11} is the mean effect of gender on the linear slope for age, e_{tj} is time-specific residual error, u_{0j} is the individual specific deviation from the mean intercept for participant *j* after accounting for gender differences, and u_{1j} is the individual specific deviation from the mean slope for age for participant *j* after accounting for gender differences. For these models, age was centered at 6 years, which was roughly the average age of the sample at the first assessment (K) occasion. Gender was centered and coded such that males = 1 and females = -1. The model was estimated with PROC MIXED in SAS version 9.3 (Littell et al., 1996). Estimation of the models with restricted maximum likelihood was attempted, but the estimated G matrices were not positive definite. So, the models were then estimated using minimum variance quadratic unbiased estimation (Rao, 1972).

Assessing the Relations Between the Development of Performance Monitoring and Behavioral Variability. Pairwise correlations between concurrently measured behavioral

variability and ERN amplitude, average signal strength, and signal temporal consistency were conducted for each assessment to measure sample level associations of between person differences. Additionally, separate parallel process growth models were used to examine the relations between developmental trajectories of behavioral variability and developmental trajectories of ERN amplitude, average signal strength, and signal temporal consistency to measure sample level associations of between person differences in developmental trajectories. To accomplish this, Pearson's correlations were examined amongst the empirically derived Bayes estimates for intercepts (β_{0j}) and slopes for age (β_{1j}) extracted from the change models specified by Equation 6 (Littell et al., 1996).

Results

Univariate statistics for all measures are found in Table 2 and Pearson's correlation among K, G1, and G2 values for ERN amplitude, average signal strength, signal temporal consistency, and behavioral variability are presented in Table 3.

Rank Order Stability of Performance Monitoring and Behavioral Variability

ERN amplitude measured at K and G2 was significantly correlated (r = .20, p < .05), but pairwise correlations between ERN amplitude at K and G1 and G1 and G2 were not significant (ps > .10) indicating little rank-order stability of the ERN over the age range assessed. Average signal strength showed high stability across K to G2 with significant correlations among K and G1, K and G2, and G1 and G2 measures (rs = .61 to .70, ps < .01). Correlations among K and G1and K and G2 measured signal temporal stability were not significant (ps > 10), but the correlation between G1 and G2 measurements was trending toward significance (r = .16, p < .10). K and G1, K and G2, and G1 and G2 measured behavioral variability showed moderate to high stability and statistically significant correlations (rs = .39 to .69, ps < .01).

Table 2. Univariate Statistics for Measures.

Measurement	Mean	Standard Deviation	n
Age			
K	6.10	.39	218
G1	7.20	.41	172
G2	8.27	.56	162
ERN Amplitude			
K	-4.95	3.25	211
G1	-5.70	3.39	162
G2	-6.40	3.78	157
Average Signal Strength			
K	33.35	.89	211
G1	33.14	.98	162
G2	32.91	1.05	157
Signal Temporal Stability			
K	.19	.08	211
G1	.23	.10	162
G2	.25	.12	157
Behavioral Variability			
K	510.14	55.76	218
G1	483.74	67.82	172
G2	414.55	61.25	162

Table 3. Pairwise Pearson's Correlations Amongst Measures.

	1	2	3	4	5	6	7	8	9	10	11	12
1. K ERN Amplitude (log)	--											
2. G1 ERN Amplitude (log)	.03	--										
	(143)											
3. G2 ERN Amplitude (log)	.20*	.14	--									
	(139)	(126)										
4. K Average Signal Strength	-.36*	-.23*	.01	--								
	(211)	(143)	(139)									
5. G1 Average Signal Strength	-.23*	-.25*	-.05	.70*	--							
	(143)	(162)	(126)	(143)								
6. G2 Average Signal Strength	-.21*	-.17^{t}	-.23*	.61*	.69*	--						
	(139)	(126)	(157)	(139)	(126)							
7. K Signal Temporal Stability (log)	-.32*	-.02	-.11	.09	.00	-.03	--					
	(211)	(143)	(139)	(211)	(143)	(139)						
8. G1 Signal Temporal Stability (log)	.13	-.39*	-.08	-.05	.08	.05	.00	--				
	(143)	(162)	(126)	(143)	(162)	(126)	(143)					
9. G2 Signal Temporal Stability (log)	-.07	-.08	-.37*	.11	.15	.06	-.03	.16^{t}	--			
	(139)	(126)	(157)	(139)	(126)	(157)	(139)	(126)				
10. K Behavioral Variability	.12^{t}	.10	.13	.02	.02	.03	.01	-.17*	-.04	--		
	(211)	(148)	(145)	(211)	(148)	(145)	(211)	(148)	(145)			
11. G1 Behavioral Variability	.03	.17*	.20*	.02	-.06	.01	.00	-.11	-.11	.39*	--	
	(152)	(162)	(133)	(152)	(162)	(133)	(152)	(162)	(133)	(159)		
12. G2 Behavioral Variability	.12	.21*	.28*	-.02	-.09	-.14^{t}	-.01	-.05	-.14^{t}	.43*	.69*	--
	(144)	(129)	(157)	(144)	(129)	(157)	(144)	(129)	(157)	(151)	(138)	

Note. Pairwise sample sizes are reported in parentheses. Log signifies measure was log transformed. * = $p<.05$. ^{t} = $p<.10$.

Normative Development of Performance Monitoring and Behavioral Variability

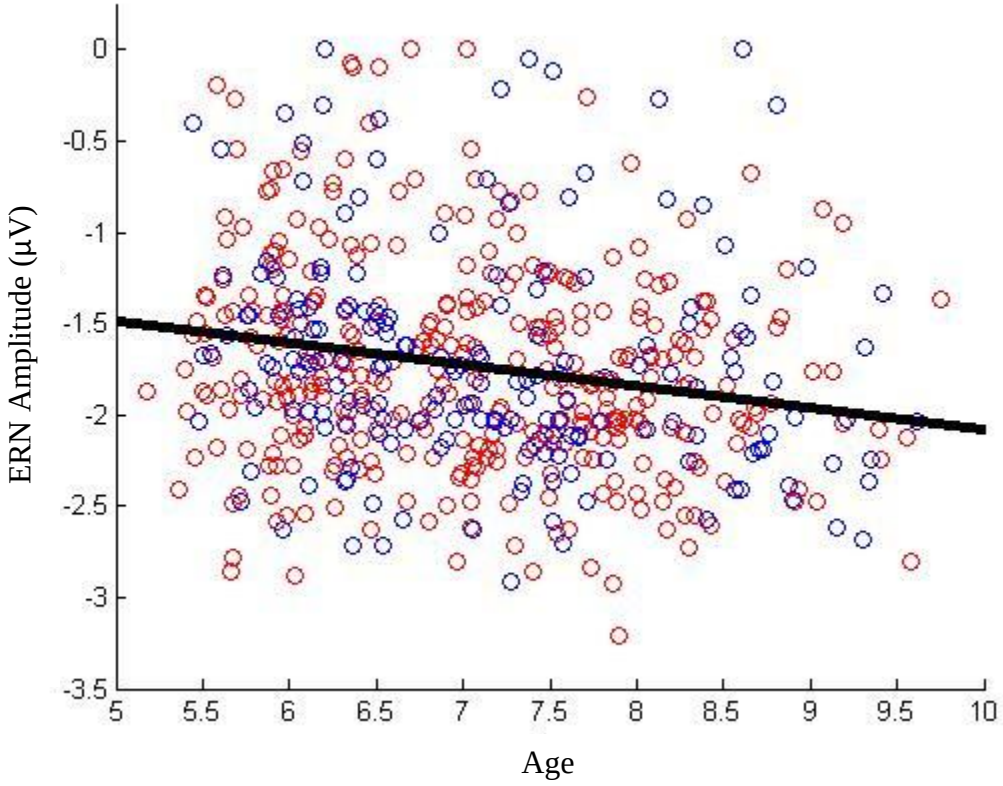
Age and gender related effects on the outcome measures were assessed with the model specified by Equation 6 and are presented in Table 4. A significant effect of Age was found for ERN amplitude (γ_{10} = -.118 p < .01) indicating that older participants had more negative amplitudes. The effect of Gender and the Gender by Age interaction were not significant (ps > .10). For average signal strength, an effect for Age was significant (γ_{10} = -.2190, p < .01) and indicated that average signal strength decreased over the time period assessed. The effects for Gender and the Gender by Age interaction were not significant (ps > .10). For signal temporal consistency, the effect of Age (γ_{10} = .094, p < .01) was significant and indicated that participants signal temporal consistency increased with age. The Gender by Age interaction was significant (γ_{11} = .042 p < .05) while the effect for Gender (γ_{01} = -.049, p < .10) trended on significance for signal temporal consistency. These gender effects indicated that signal temporal consistency was lower for boys than girls at the initial assessment, but the increase in signal temporal consistency was greater over this time period for boys than girls. A significant effect of Age was found for behavioral variability (γ_{10} = -34.267 p < .01) and indicated that behavioral variability was decreasing with age. The effect of Gender for behavioral variability was not significant (p > .10), but the Gender by Age interaction (γ_{11} = -10.126 p < .10) trended on significance and indicated that boys, on average, exhibited a steeper decrease in behavioral variability than girls. Participant values and modeled trajectories for ERN amplitude, average signal strength, signal temporal consistency, and behavioral variability are graphed in Figures 5-8.

Table 4. Modeled ERN Amplitude, Average Signal Strength, Signal Temporal Consistency and Behavioral Variability by Age, Gender, and Age by Gender Interaction with Occasion Nested Within Individual as Specified by Equation 6.

	ERN Amplitude		Average Signal Strength		Signal Temporal Consistency		Behavioral Variability	
Parameter	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Fixed Effects								
Intercept, γ_{00}	-1.609*	(.045)	33.393*	(.065)	-1.713*	(.028)	518.930*	(4.268)
Gender, γ_{01}	-.023	(.045)	.015	(.065)	-.049^{t}	(.028)	-.591	(4.268)
Age, γ_{10}	-.118*	(.034)	-.219*	(.027)	.094*	(.019)	-39.330*	(2.835)
Gender*Age, γ_{11}	-.003	(.034)	.016	(.027)	.042*	(.019)	-5.063^{t}	(2.835)
Random Effects								
Variance intercept σ^2_{u0}	.098	(.082)	.599	(.098)	.015	(.017)	1435.64	(451.05)
Variance slope σ^2_{u1}	.054	(.005)	.004	(.014)	.006	(.007)	317.93	(618.26)
Cov intercept, slope σ^2_{u0u1}	.018	(.002)	.034	(.026)	-.007	(.010)	442.58	(314.37)
Residual variance	.319	(.031)	.260	(.031)	.152	(.015)	2355.08	--
-2LL		1008.9		1295.0		569.1		6,028
AIC		1016.9		1303.0		577.1		6,036

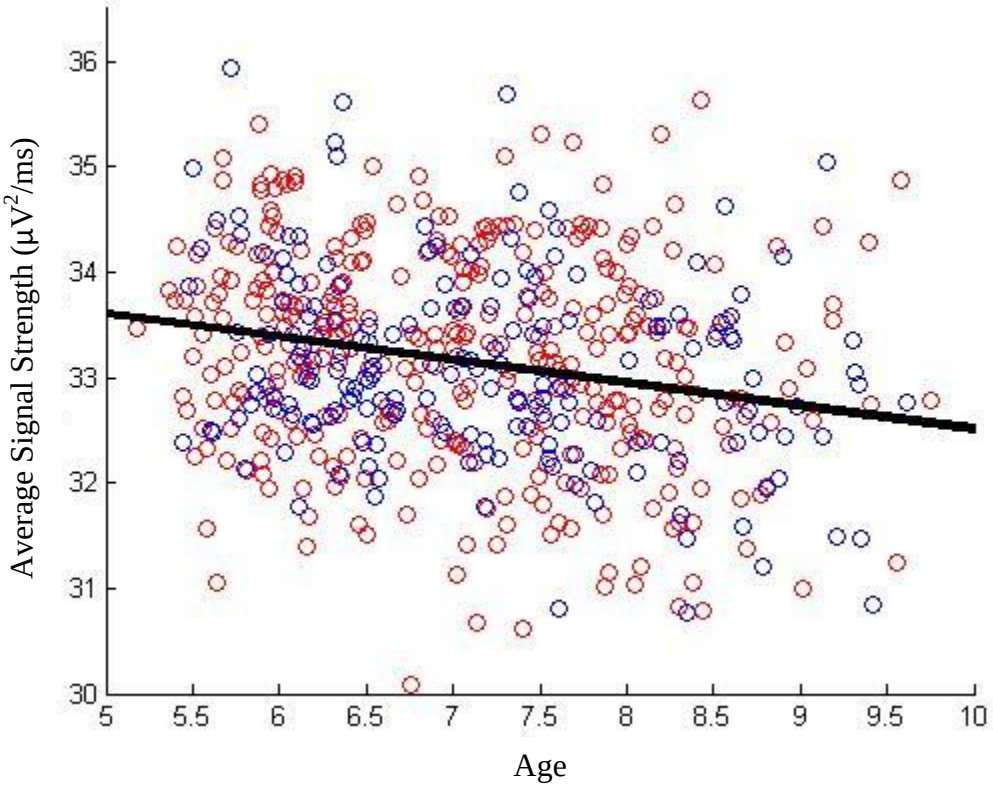
* = p<.05. ^{t} = p<.10. AIC = Akaike Information Criterion; −2LL = −2 Log Likelihood, relative model fit statistics.

Figure 5. ERN Amplitude and Modeled ERN Amplitude Over Time.



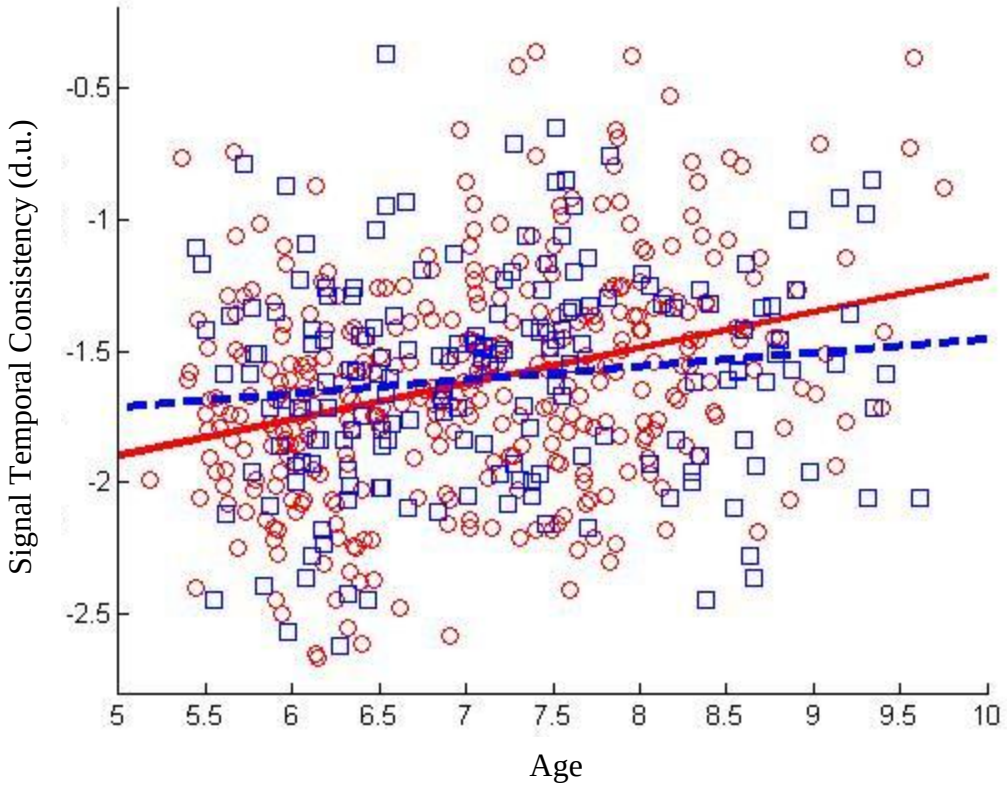
Notes. Red indicates male participant values and blue indicates female participant values. Average trajectory of ERN amplitude was collapsed across gender. More negative values indicate greater ERN deflections.

Figure 6. Average Signal Strength and Modeled Average Signal Strength Over Time.



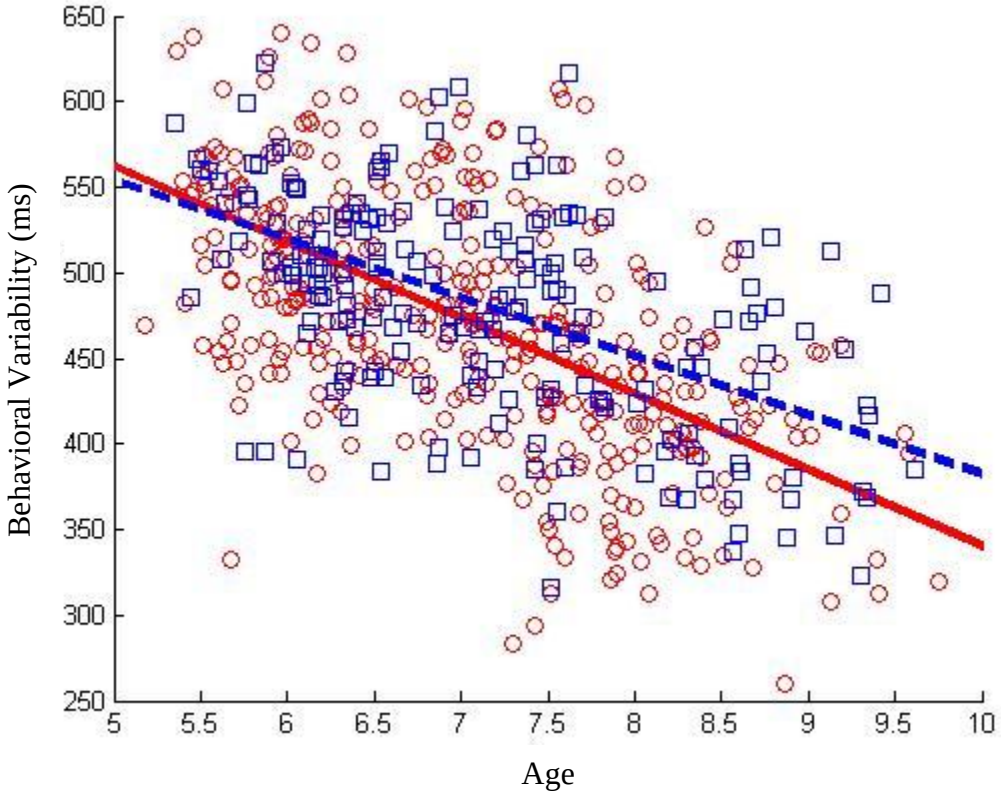
Notes. Red indicates male participant values and blue indicates female participant values. Average trajectory of average signal strength was collapsed across gender. Greater values indicate greater average signal strength.

Figure 7. Signal Temporal Consistency and Modeled Signal Temporal Sability Over Time by Gender.



Notes. Red circles indicate male participant values and the red solid line represents the modeled trajectory for male participants while blue squares indicate female participant values and the blue dashed represents the modeled trajectory for female participants. Less negative values indicate greater signal temporal stability.

Figure 8. Behavioral Variability and Modeled Behavioral Variability Over Time by Gender.



Notes. Red circles indicate male participant values and the red solid line represents the modeled trajectory for male participants while blue squares indicate female participant values and the blue dashed represents the modeled trajectory for female participants. Lesser values indicate less behavioral variability.

Assessing Normative Development of Performance Monitoring and Behavioral Variability

Pearson's correlations among concurrently measured behavioral variability and ERN amplitude, average signal strength, and signal temporal consistency were examined and are found in Table 3. The correlation between ERN amplitude and behavioral variability showed a trend level significance at K (r = .12, p < .10). Correlations between these two measures reached statistical significance in G1 (r = .17, p < .05) and G2 (r = .28, p < .01). Average signal strength and signal temporal consistency were not significantly correlated with behavioral variability in K

or G1 (*p*s > .10). The correlations with behavioral variability reached trend level significance for both average signal strength and signal temporal consistency in G2 (*r*s = -.14, *p*s < .10).

Pearson's correlations among individual specific intercepts and slopes for age from the model estimated by Equation 6 were conducted between behavioral variability and ERN amplitude, average signal strength, and signal temporal consistency and are presented in Table 5. Model derived intercepts and slopes for age for ERN amplitude and behavioral variability were moderately and significantly correlated (*r*s = .26 to .28, *p*s < .01) and graphical depictions of associations between modeled slopes and intercepts for these measures are shown in Figures 9 and 10, respectively. Modeled intercepts and slopes for age for average signal strength and signal temporal consistency were not significantly correlated to modeled intercepts and slopes for behavioral variability (*p*s > .10).

Table 5. Correlations Between Model Derived Trajectories for Behavioral Variability and Model Derived Trajectories of ERN Amplitude, Average Signal Strength and Signal Temporal Consistency

	Behavioral Variability	
Parameter	Intercept, β_{0j}	Slope, β_{1j}
ERN Amplitude		
Intercept, β_{0j}	.26*	.26*
Slope, β_{1j}	.27*	.28*
Average Signal Strength		
Intercept, β_{0j}	-.04	-.07
Age, β_{1j}	-.06	-.08
Signal Temporal Consistency		
Intercept, β_{0j}	-.07	-.05
Age, β_{1j}	-.02	-.02

Note: * = p<.05.

Figure 9. Parallel Process Model Scatterplot of Modeled Individual Specific Estimated Intercepts for ERN Amplitude and Behavioral Variability.

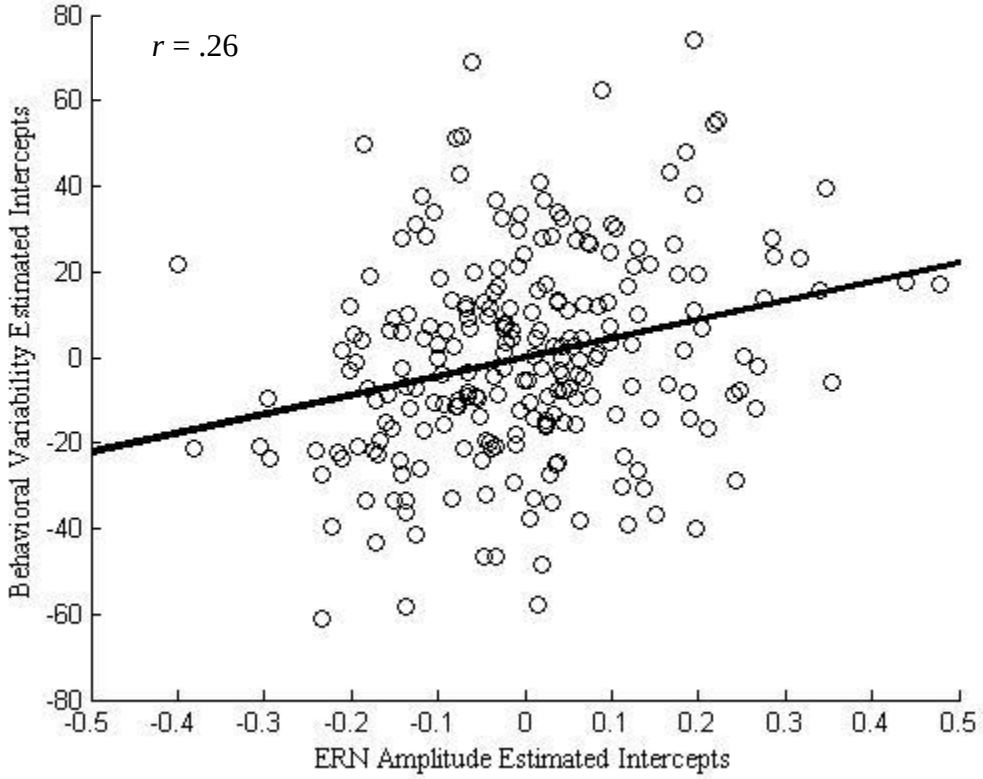
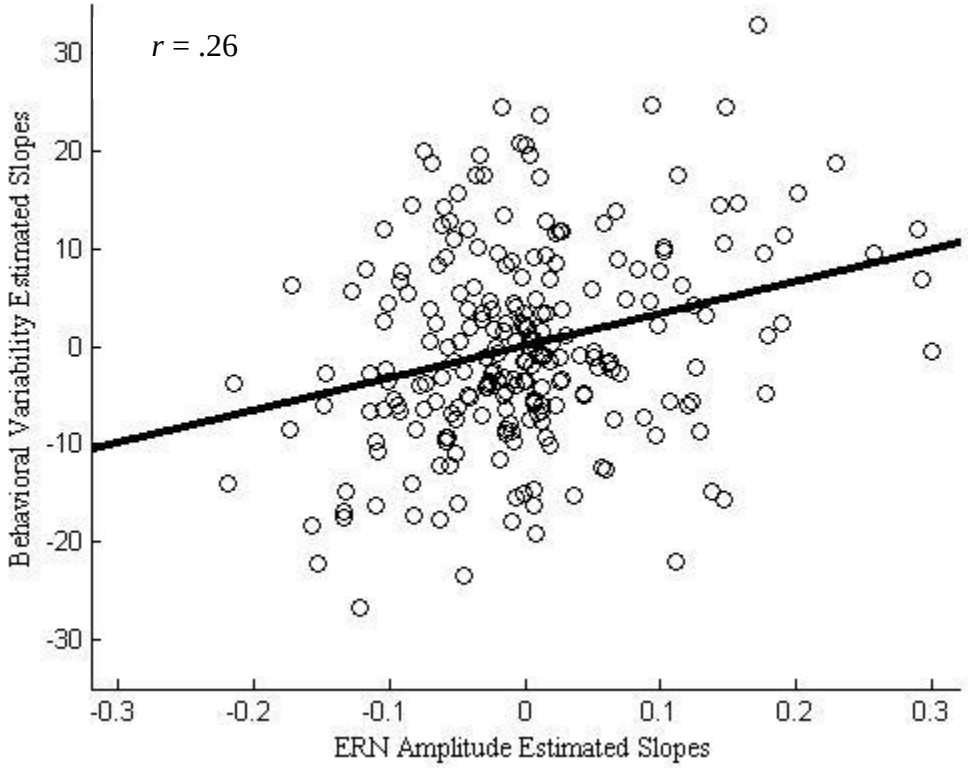


Figure 10. Parallel Process Model Scatterplot of Modeled Individual Specific Estimated Slopes for ERN Amplitude and Behavioral Variability.



Discussion

The present study sought to characterize the development of the neural activity underlying performance monitoring and behavioral variability in middle childhood. Although well studied in adults, these measures have rarely been assessed in childhood. Additionally, researchers investigating these measures primarily employ cross-sectional designs. The longitudinal design of the present study minimized the chance that age-related differences in the measures were due to cohort effects, allowed for the assessment of rank-order stability of the measures, and allowed for the investigation of the relationship of within-person changes in behavioral variability and neural activity underlying performance monitoring.

Normative Age-Related Changes in Performance Monitoring

Consistent with previous research, this study revealed developmental increases in ERN deflections in children assessed repeatedly in K, 1st, and 2nd grade (Davies et al., 2004; Wiersema et al., 2007). This study builds upon prior research by investigating age-related changes in average signal strength and signal temporal consistency of performance monitoring across trials. It was hypothesized that increases in both average signal strength and signal temporal consistency would underlie the increase in ERN deflections with age. Consistent with this hypothesis, signal temporal consistency increased with age. This is the first study to show that the developmental increase in ERN deflections in childhood could be partially due to an increase in consistency of signal latency across trials. Since the ERN is typically measured across many trials, low levels of consistency in the latency of the ERN component would result in a muted signal in the average waveform. Low levels of signal temporal consistency could also explain why some children fail to show an ERN signal in their average waveform despite having the ability to behaviorally exhibit performance monitoring capabilities. Surprisingly, it was found

that average signal strength decreased with age. This is incompatible with prevailing theory that the increase in ERN deflections in average waveforms in children and adolescents is due to an increase in the signal strength.

Potential Indicator of an Increasingly Efficient Neural System. The age-related changes in signal temporal consistency and average signal strength may be evidence of increasingly efficient functioning of neural systems underlying performance monitoring. Neurons transmit information with signals encoded in action potentials. These action potentials affect the polarization of a post-synaptic neuron. The post-synaptic neuron integrates signals from numerous sources and will fire an action potential if the threshold potential is reached. The effect of a single action potential on a post-synaptic neuron, however, will diminish with time and for a neuron to reach threshold potential it must receive signals from many action potentials in a short temporal window. This highlights the importance of timing of the receipt of a signal, as information encoded in a signal with imprecise timing could be lost. A neural system characterized by low temporal precision would process information less efficiently due to information loss during transmission (Barlow, 1961). However, an inefficient neural system could reach the functional effectiveness of an efficient neural system by compensating for information-loss by increasing the number of signals that are sent. Thus, an equally functional but less efficient neural system could be differentiated by greater activity and less temporal precision. Viewed through this lens, the increases in signal temporal consistency found in the present study could be evidence of maturing neural systems that are able to more efficiently monitor performance. Additionally, the increased levels of average signal strength seen early in the developmental period examined could be necessary for effective performance monitoring in spite of less efficient neural systems.

Results from a previous study investigating age-related changes in the dynamics of auditory perception across childhood that found a decrease in average signal strength and an increase in signal temporal consistency could be similarly interpreted as evidence of increasingly efficient neural systems (Müller et al., 2009). Additionally, fMRI studies often find that children exhibit more diffuse fMRI activation patterns than adults (Casey et al., 2000; Durston et al., 2006). Although the temporal precision of fMRI does not allow for the investigation of variability in signal latency, the more diffuse activation patterns could be a result of less efficient neural systems that require a greater number of resources to process the same amount of information.

Age-related structural changes in myelination patterns may underlie the age-related functional changes observed in the present and prior studies. White matter integrity increases into early adulthood and has been related to age-related increases in cognitive abilities (Benes, 1989; Yakovlev & Lecours, 1967; Nagy et al., 2004; Schmithorst et al., 2005). Myelin increases the conduction speed of action potentials allowing for neural signals to be transmitted at a distance more efficiently. If multiple neural regions require similar pre-processing of information, it is possible that this increase in white matter integrity allows for an increase in the centralization and specialization of information processing. A centralized information processing unit would be able to pre-process the information and more rapidly transmit this information to distant regions in a system with greater levels of white matter integrity. A system with decreased integrity of white matter tracks may require this pre-processing to happen closer to each of the regions requiring the output, requiring similar processing to happen in multiple regions. The centralized system, in which a specialized unit processes the information, would likely be more efficient at processing specific types of information while the decentralized system would have

added inefficiencies due to redundant information processing. In this way, a system with greater white matter integrity would require fewer resources to process the same amount of information as a system with less white matter integrity.

Gender Differences in Performance Monitoring. Similar to a previous study with participants in a similar age range (Davies et al., 2004; Torpey et al., 2011), no gender differences were found in ERN amplitude in the present study. A previous study of adolescents found gender differences in ERN such that males had larger ERN deflections than females (Davies et al., 2004). Similar findings have been found in adult populations (Larson et al., 2011), although other studies have found no association between ERN amplitude and gender in adults (Fukushima & Hiraki, 2006). Despite the lack of effect of gender on ERN amplitude, gender effects on signal temporal consistency were found. At the initial assessment, girls had greater levels of signal temporal consistency than boys. This relationship reversed over time with boys having greater signal temporal consistency than girls during the final assessment. Gender effects on average signal strength were not found. Interpretations of gender differences in signal temporal consistency are discussed in a later section through the relationship between behavioral variability and signal temporal consistency.

Rank-Order Stability of ERN Metrics

Rank-order stability of the ERN in this study was low. The correlation between K and G2 measured ERN was significant while the correlations between K and G1 and G1 and G2 were not. The strength of the relationship between the ERN in K and G1 was negligible, while small to moderate relationships between K and G2 and G1 and G2 measured ERN were found. Although previous studies have found the ERN to be a stable trait-like measure in adulthood (Olvet & Hajcak, 2009; Segalowitz et al., 2010), the low year-to-year reliability of the ERN in

the present sample is not entirely surprising. The neural regions that generate the ERN signal are under development during the age range assessed in the present study and differential rates of development across persons may dampen the rank-order stability of the ERN. Although the ERN is viewed as a trait-like measure in adulthood, the trait-like nature of this measure has not yet developed for this population. As such, the ERN at this age may not provide much predictive power to explain future between person differences in outcomes.

Unlike ERN amplitude, large associations were found between each of K, G1, and G2 measured average signal strength. The highly trait-like nature of average signal strength suggests that it has potential usefulness as a predictor of future outcomes. In contrast, signal temporal consistency showed poor rank-order stability over this age range. The associations between K and G1 and K and G2 measured signal temporal consistency were negligible, but a small association that trended on significance was found between signal temporal consistency measured at G1 and G2. This may be evidence of the beginning of the development of trait-like rank-order stability of signal temporal consistency.

Since measurement of the ERN in an individual's average waveform depends on both average signal strength in each trial and signal temporal consistency across trials (Cavanagh et al., 2012), the rank-order stability of the ERN will depend on the rank-order stability of each of these measures. Although there is high rank-order stability of average signal strength, signal temporal consistency has much less rank-order stability over time, suggesting that the low rank-order stability of the ERN across this developmental period is due to a lack of rank-order stability of signal temporal consistency.

It is possible that the signal temporal consistency may not act as a trait-like measure during any developmental period. If this is the case, traditional measurement of the ERN would

be a function of a factor that can reliably provide insights into trait-like individual difference in average signal strength, and a factor that cannot reliably provide insights into trait-like individual difference in signal temporal consistency. The reliability of traditionally measured ERN amplitude would, in this case, be dampened by the unreliability of signal temporal consistency and an analysis using average signal strength may be more informative for researchers.

The reliability of signal temporal consistency, however, cannot be extrapolated to age ranges beyond those assessed in the present study. There is some evidence to suggest signal temporal consistency is increasing as Pearson's correlation between G1 and G2 measured signal temporal consistency was trending toward significance. Future studies would need to be conducted in order to determine if signal temporal consistency develops into a trait-like measure in older populations. If it does, analyses investigating individual differences in average signal strength and signal temporal consistency could provide greater insights into neurological functioning than that of ERN amplitude alone. The ERN is broadly related to cognitive abilities, personality, and psychopathology. It is possible that average signal strength and signal temporal consistency could be differentially related to these outcomes and knowledge of this could further our understanding of the neural basis of behavior.

Abnormal performance monitoring has been broadly related to psychopathology (Hall et al., 2007; Olvet & Hajcak, 2008), and it is possible that altering performance monitoring capabilities in at-risk individuals could alter their risk trajectories. The low rank-order stability of signal temporal consistency in this age range, then, may provide useful information for developmental scientists. It is believed that unstable processes under development are indicative of sensitive periods in which environmental effects can induce lasting changes in a system (Knudsen, 2004). In this sense, the lower rank-order stability of signal temporal consistency in

children might signify that it is a better target for intervention than average signal strength. If we can further understand signal temporal consistency and find strategies to intervene and improve performance monitoring through increased signal temporal consistency, interventions may have a lasting effect on behavior.

Normative Age-Related Changes in Behavioral Variability

Consistent with previous findings, the present study showed that behavioral variability decreased as children moved from K though G2, thought to reflect increased efficiencies in neural processing (Eckert & Eiehorn, 1977; Segalowitz & Segalowitz, 1993). A gender by age interaction was found such that behavioral variability decreased faster for boys than girls over the age range in this study. The study indicates that this gender difference is not apparent in kindergarten-aged children; rather, the difference emerges as the children mature. The current study is the first to identify the emergence of gender differences in behavioral variability in middle childhood. Previous studies have found no gender differences in behavioral variability in childhood, although males are often reported to have less behavioral variability than females in adulthood (Dykiert et al., 2012; Eckert & Eichorn, 1977). Methodological differences could explain the discrepancy between the effect of gender on behavioral variability in this study and previous studies in childhood. The reported studies finding no gender differences in behavioral variability in childhood defined behavioral variability for an individual as the coefficient of variation, which attempts to take into account differences in mean reaction time by dividing the standard deviation of response times by the mean response time (Dykiert et al., 2012; Eckert & Eichorn, 1977). The present study, however, defined behavioral variability for an individual just as the standard deviation of response times. Females typically have greater mean response times, which would deflate the measurement of behavioral variability of females in the previous studies

compared to the present study. There is debate as to the most appropriate way to characterize behavioral variability and researchers have found both metrics to capture explanatory variance (Golay et al., 2013; Wagenmakers & Brown). Interpretations of gender differences in behavioral variability are discussed in the following section through the relationship between behavioral variability and signal temporal consistency.

Rank-Order Stability of Behavioral Variability

Despite the significant normative change observed in this study, behavioral variability showed high levels of rank-order stability. There were large associations amongst the K, G1, and G2 measurements of behavioral variability. The level of rank-order stability is similar to that seen in adults (Hagen et al., 2007; Saville et al., 2011), indicating that behavioral variability is a reliable, trait-like measure that is likely to have predictive validity even when measured in childhood.

ERN Amplitude and Behavioral Variability

Consistent with the hypothesis, moderate associations between concurrently measured behavioral variability and ERN amplitude were observed such that greater ERN deflections are associated with less behavioral variability. Additionally, individual differences in growth trajectories for ERN amplitude over the course of the study were related to individual differences in growth trajectories for behavioral variability such that children who showed steeper increases in ERN deflections over the course of the study also showed greater decreases in behavioral variability. This is one of the few studies to explore and find a relationship between the ERN and behavioral variability (Kolev et al., 2009). The growth trajectories for behavioral variability were not related to the growth trajectories for either average signal strength or signal temporal consistency. Additionally, no association was found between concurrently measured behavioral

variability and average signal strength or signal temporal consistency in K or G1. Associations of trend-level significance were found between behavioral variability and both average signal strength and signal temporal consistency in G2, although the size of these associations were small. This pattern of correlations suggests that the relationship between behavioral variability and performance monitoring may be strengthening as children age. Interestingly, the relationship between behavioral variability and ERN amplitude is stronger than the relationship between behavioral variability and either average signal strength or signal temporal consistency. This suggests that a small amount of the variance from both average signal strength and signal temporal consistency are independently contributing to the association between ERN amplitude and behavioral variability. Although this may appear to suggest that traditional measures of the ERN amplitude are able to provide greater insights into neurological basis of individual differences than average signal strength or signal temporal consistency, it is important to note that this is only necessarily true for the outcome of behavioral variability and in the age range assessed. Future studies will need to determine if either average signal strength or signal temporal consistency provide additional explanatory value for other outcomes and/or in other age ranges.

Behavioral Variability and Signal Temporal Consistency. If low levels of signal temporal consistency and high levels of behavioral variability are thought to reflect inefficiencies in neural processing, it may be somewhat surprising that the two measures do not show strong associations with each other. The lack of association, however, may be due to these measures being indicative of different types of neuronal inefficiencies. Behavioral variability could be due to inefficiencies in any neuronal system involved in completing the task including stimulus processing, response selection, and motor execution. Decreased signal temporal consistency

could only be due to inefficiencies in performance monitoring and potentially motor execution since the performance monitoring components in the EEG waveform are locked to the timing of the motor responses. In this way, both could be separate and valid measures of neural efficiency with behavioral variability reflecting general neural efficiency and signal temporal consistency more specifically reflecting the efficiency of neural performance monitoring processes.

The decreased behavioral variability and increased signal temporal consistency in females at the later time points of this study could be due to potential contributions of inefficiencies in motor execution on both measures. Boys show greater motor control performance than girls in many tasks (Thomas & French, 1985). Differences in the efficiency of neural processes underlying motor execution may exist, which could cause the lower behavioral variability and greater signal temporal consistency found in this study for boys at later assessments. If this is the case, it may be possible to parse out the shared variance due to motor execution inefficiencies in these measures for researchers whose primary interests do not include inefficiencies in neural systems underlying motor execution.

Limitations

Although this study is large compared to previous studies assessing development of ERN signaling in youth, the present study covers a comparably limited age range (Davies et al., 2004; Wiersema et al., 2007). Future studies will need to be conducted to determine if the age-related changes in neural signaling underlying performance monitoring found in children in this study are general trends throughout childhood or if these changes are limited to a narrow developmental time frame.

The participants in the current study were predominantly ethnic minority, urban, and of low socioeconomic status (SES), which could affect the generalizability of the study's findings.

But, the overall processes assessed in this underrepresented sample are presumed to be universal. As such, the developmental trajectories found in the present study would be expected to be seen among other populations. However, SES has been related to timing of the development of cognitive abilities (Bradley & Corwyn 2002), so future studies may expect developmental changes similar to those found in the current study to be found in younger children, if assessing children of higher SES.

The present study oversampled for aggression and half of the participants were reported to have problem behaviors. Externalizing disorders have been linked to both muted ERN deflections and greater levels of behavioral variability, which would result in differences in absolute levels at corresponding ages for these measures in the present study compared to those with lower risk samples. Since younger participants also show decreased ERN deflections and increased levels of behavioral variability, it is also possible that differences amongst participants in this sample could be due to later development of these processes compared to participants in lower risk samples. To the extent that individuals with externalizing disorders are characterized by developmental delay in these processes, as opposed to mean level differences at a given age, participants' of this study could again show changes in ERN amplitude and behavioral variability similar to what would be expected to be found amongst younger participants in lower risk samples.

Although the developmental changes found in the present study, timing withstanding, are presumed to be generalizable to samples more representative of typical development, the gender differences found should be viewed with caution. As noted above, the high risk composition of the sample likely had effects on levels of ERN amplitude and behavioral variability. It is possible that the gender effects found were due to the preponderance of high risk participants, and lower

risk samples, which may have gender differences in the proportion of high risk participants, compared to the current study, will find a differential impact of gender on the neurophysiological indicators of performance monitoring and behavioral variability.

Differences in attention have the potential to impact findings related to neural processing in studies assessing ERPs. If a particular individual is not attending to the stimulus, that individual will not exhibit task-related neural activity. To screen out individuals who were not attending throughout the task, any participant who incorrectly responded on at least 60% of No-go trials were excluded. Still, it is possible for individuals to have periods of inattention throughout the task and still perform above the cutoff threshold for inclusion in the study. Individuals who had periods of inattention would show smaller ERN deflections, less average signal strength, and lower signal temporal consistency. Thus, the possibility remains that the age-related changes found in the current study are due to age-related increases in attention. Future studies could investigate the role attention plays in the generation of the ERN by investigating if individual differences in ERN signaling are related to individual differences in the visual N1, an ERP component sensitive to levels of attention (Haider et al., 1964; Luck et al., 2000). The degree to which variance in ERN deflections are due to individual differences in attention could then be assessed by the strength of the relationship between ERN amplitude and visual N1 amplitude.

Conclusion

The present study examines the developmental trajectories of both average signal strength and signal temporal consistency in error-related neural signaling across childhood. While the results confirmed previous findings of increasing ERN deflections over childhood, it was observed that average signal strength actually decreased while signal temporal consistency increased across the age range assessed. The decrease in average signal strength across trials contradicts conventional thinking that the increase in ERN deflections seen throughout childhood and adolescence is due to an increase in signal strength. Instead, this study supports the hypothesis that a decrease in intra-individual variability in signal latency causes the increase in ERN signal strength seen throughout childhood. The potential muting effect of low signal temporal consistency in younger children could also explain why previous studies have found no ERN signal in children who are behaviorally able to exhibit the cognitive abilities associated with the ERN. These findings highlight the importance of considering signal temporal consistency of ERP components, especially when assessing participants of different ages, which is typically neglected.

The present study also adds to the literature by assessing the rank-order stability of neurophysiological measures underlying performance monitoring and behavioral variability in an age range in which they had yet to be assessed. Although ERN amplitude shows trait-like stability in adulthood, low levels of rank-order stability of the ERN amplitude were observed among children in the current study. Similarly, there was low rank-order stability of signal temporal consistency, particularly between the kindergarten and G1 or G2 measures. In contrast, both average signal strength and behavioral variability showed stable, trait-like properties over

the age range assessed in the current study indicating that these measures, assessed in childhood, may be reliable predictors of future individual differences.

Finally, the present study investigated the relations between neurophysiological measures underlying performance monitoring and behavioral variability. In line with previous research, between-person differences in ERN amplitude were related to behavioral variability such that individuals with greater ERN deflections also had less behavioral variability. Additionally, an association was found between individual-specific growth trajectories for ERN amplitude and behavioral variability such that individuals whose ERN deflections were increasing at a faster rate than average also exhibited a greater than average rate of decrease in behavioral variability. This suggests that the same developmental process could be accounting for between-person change in both ERN signaling and behavioral variability.

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