Implications of Ongoing Neural Development for the Measurement of the Error-Related Negativity in Childhood

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Abstract

Event-related potentials (ERPs) have been proposed as biomarkers capable of reflecting individual differences in neural processing not necessarily detectable at the behavioral level. However, the role of ERPs in developmental research could be hampered by current methodological approaches to quantification. ERPs are extracted as an average waveform over many trials, however, actual amplitudes would be misrepresented by an average if there was high trial-to-trial variability in signal latency. Low signal temporal consistency is thought to be a characteristic of immature neural systems, although consistency is not routinely measured in ERP research. The present study examined the differential contributions of signal strength and temporal consistency across trials in the error-related negativity (ERN) in 6-year-old children, as well as the developmental changes that occur in these measures. The 234 children were assessed annually in kindergarten, 1st, and 2nd grade. At all assessments signal strength and temporal consistency were highly correlated with the average ERN amplitude, and were not correlated with each other. Consistent with previous findings, ERN deflections in the averaged waveform increased with age. This was found to be a function of developmental increases in signal temporal consistency, whereas signal strength showed a significant decline across this time period. Additionally, average ERN amplitudes showed low-to-moderate stability across the three assessments whereas signal strength was highly stable. In contrast, signal temporal consistency did not evidence rank order stability across these ages. Signal strength appears to reflect a stable individual trait whereas developmental changes in temporal consistency may be experientially influenced.

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Keywords

Intra-individual variability; ERN; Developmental Change

Introduction

Neural processes have received increasing attention in the study of individual differences in affective and cognitive systems that may underlie risk for adverse behavioral outcomes. Several researchers have proposed that event-related potentials (ERPs) may have endophenotypic properties, reflecting core individual traits that are thought to be stable across time (Hajcak, 2012; Iacono & Malone, 2011; Olvet & Hajcak, 2008). If supported, ERPs would have enormous potential in research elucidating developmental mechanisms associated with adverse outcomes. However, the majority of research supporting the association between ERPs and specific psychological profiles has been conducted in adolescent and adult samples. As researchers increasingly seek to assess ERPs in younger children, careful consideration must be paid to whether these components are developmentally invariant, particularly during phases of rapid neural change (Davies, Segalowitz, & Gavin, 2004). In this study, we focus specifically on the developmental characteristics of the error-related negativity (ERN) in a sample of children assessed annually in kindergarten, 1st and 2nd grades.

The ERN is typically measured in speeded response tasks where erroneous responses are likely to occur even when participants are aware of the correct stimulus-response rules. Typically within 100ms following an error, an ERN signal is generated that reflects the evaluation of the effectiveness of current behavior, allowing for the rapid recruitment of additional cognitive resources in pursuit of improving future task performance (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Scheffers & Coles, 2000; Wessel, 2012). Consistent with its perceived role in monitoring performance, ERN deflections increase as a function of the motivation for accuracy (Gehring et al., 1993; Hajcak, Moser, Yeung, & Simons, 2005; Kim, Iwaki, Uno, & Fujita, 2005).

Larger ERN deflections have been reported in association with better executive functioning (Larson & Clayson, 2011) and academic outcomes (Hirsh & Inzlicht, 2010). Larger ERN deflections have also been observed among individuals with increased sensitivity to errors and negative feedback such as those with clinical syndromes related to anxiety and depressive disorders (Gehring, Himle, & Nisenson, 2000; Hajcak, McDonald, & Simons, 2003; Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003) as well as non-clinical personality traits such as high negative affect and trait anxiety (Boksem, Tops, Wester, Meijman, & Lorist, 2006; Hajcak, McDonald, & Simons, 2004). Larger ERN deflections have also been observed among individuals shown to learn faster in response to negative reinforcement (i.e. avoid a negative consequence) than positive reinforcement (Frank, Woroch, & Curran, 2005). Conversely, muted ERN amplitudes have been found among individuals characterized by low sensitivity to negative feedback such as those with clinical syndromes

on the externalizing spectrum (Franken, van Strien, Franzek, & van de Wetering, 2007; Hall, Bernat, & Patrick, 2007; Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005).

Development of the ERN

An initial cross-sectional study of individuals from age 7 to 18 indicated that ERNs were not readily apparent in grand averaged waveforms until early adolescence (Davies et al., 2004). This appeared to result from individual ERNs in children being less easily identified and considerably more variable across individuals, resulting in a muted waveform when individual participants were averaged together. Additional cross-sectional studies similarly show a clear linear increase in grand-averaged ERN amplitudes into adulthood, suggesting that variability among individuals reduces across development (Davies et al., 2004; Kim, Iwaki, Imashioya, Uno, & Fujita, 2007; Wiersema, van der Meere, & Roeyers, 2007). More recent research has successfully documented the presence of an ERN signal in children under the age of 8, although developmental differences are often observed. One study identified an ERN signal in children ages 5-7, but unlike adults, these children's ERN deflections did not increase when they were told to focus on accuracy (Torpey, Hajcak, & Klein, 2009). However, another study with children 7 - 8 years old reported that the ERN signal was greater among participants who performed the task while being watched by a friend, suggesting that children may show the same association between ERN amplitude and motivation, but differ developmentally in the contextual factors capable of motivating them (Kim et al., 2005). In a study of anxiety symptoms, the expected association between greater ERN amplitude and symptom severity was detected in young adolescents (ages 11-13) but not in pre-adolescent children (ages 8-10) (Meyer, Weinberg, Klein, & Hajcak, 2012). The studies reported to date leave open many questions about whether the ERN reflects the same psychological constructs in children it is believed to index in adults, as well as whether experimental conditions must be developmentally adapted in order to improve ERN detection. However, the pattern of inconsistent evidence for the ERN in children may be, at least in part, due to the approach of assessing amplitude in averaged waveforms that may not accurately reflect the neural activity on any individual trial. In other words, the association between ERN amplitude and individual differences in young children may be confounded by measurement constraints.

Potential Impact of Trial-to-Trial Variability on the ERN

Averaging waveforms across repeated trials is common practice in efforts to amplify signal, assumed to be consistent across trials, while eliminating noise, assumed to be random across trials. This process, however, relies upon the validity of the assumption that the signal occurs in a temporally consistent place in each trial. For a response-locked component such as the ERN, variability in the temporal relationship between error recognition and motor response could occur as a function of variable cognitive processing speed as well as more global neural inefficiency in efferent motoric responding. If this assumption is violated such that there is variability in signal latency relative to the response, the averaging process will result in a muted waveform that is not representative of the actual signal amplitude in any trial. Traditional measures of ERN amplitude will thus be a function of both the signal strength in each trial *and* the temporal consistency of the signal across trials.

Intra-individual variability, previously presumed to be noise, has received increasing attention as a valid indicator of the functional state of behavioral systems. Behavioral variability, typically measured as the standard deviation of response times, is thought to reflect inefficiencies in neural systems (Segalowitz & Segalowitz, 1993). Children routinely exhibit greater levels of intra-individual variability in behavioral measures, such as reaction time, when compared with adults, and increased behavioral variability is thought to reflect developmentally immature neural systems (Aggarwal & Lillystone, 2000; Hultsch & MacDonald, 2004; Li et al., 2004). It is possible that the greater variability in reaction time observed in children reflects greater variability in neural signaling that also hampers detection of the ERN signal. Both the ERN and behavioral variability have been associated with dopaminergic networks (Bäckman, Nyberg, Lindenberger, Li, & Farde, 2006; Holroyd & Coles, 2002; Li & Lindenberger 1999), and developmental changes observed in ERN amplitude as well as behavioral variability mirror the developmental trajectory of prefrontal dopamine (DA) receptors (Tarazi & Baldessarini, 2000). In addition, developmental changes in myelination are thought to increase the efficiency of the transmission of neural signals resulting in decreased behavioral variability and improved cognitive capabilities (Nagy, Westerberg, & Klingberg, 2004; Schmithorst, Wilke, Dardzinski, & Holland, 2005).

Although no research to date has directly examined ERN amplitude in children as a function of neural maturation, some research has been conducted in elderly populations. Neural declines in advanced aging may in some ways mirror the developmental inclines in neural systems seen in childhood. Advanced aging is characterized by a decrease in frontal DA receptors, an increase in behavioral variability, anda muting of ERN deflections (Bäckman et al., 2006; Band & Kok, 2000; Falkenstein, Hoormann, & Hohnsbein, 2001; Hultsch, Macdonald, & Dixon, 2002;). Similarly, decreased white matter integrity has been linked to age-related cognitive decline (Charlton et al., 2006; Madden et al., 2009; Sullivan & Pfefferbaum, 2006), as well as increased behavioral variability (Bunce et al., 2007; Fjell, Westlye, Amlien, & Walhovd, 2011; Ullén, Forsman, Blom, Karabanov, & Madison, 2008). At least one study has reported an association between reduced white matter integrity and decreased ERN deflections in a sample of healthy adults (Westlye, Walhovd, Bjornerud, Due-Tonnessen, & Fjell, 2009). Interestingly, one study reported that decreases in the average ERN amplitude seen in old age were due not only to a general decrease in signal strength, but also to an increase in the variability of the ERN signal (Kolev, Beste, Falkenstein, & Yordanova, 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 (Segalowitz & Dywan, 2009), this hypothesis remains largely unexplored. Evidence suggests that behavioral variability is associated with lower ERN amplitudes in children ages 7 - 9 years (Richardson, Anderson, Reid, & Fox, 2011). Thus it is possible that behavioral variability commonly seen in children is associated with trial-to-trial variability in signal latency that could underlie the muted ERN waveforms also observed in children.

Time-Frequency Decomposition

Evidence is increasing that signal temporal consistency specifically is associated with a range of cognitive processes such as successful memory encoding (Fell, Ludowig, Rosburg, Axmacher, & Elger, 2008; Klimesch et al., 2004) and memory retrieval (Schack & Klimesch, 2002). However, to our knowledge no study has assessed the developmental changes of signal temporal consistency or the association between changes in temporal consistency and changes in the amplitude of averaged waveforms. To examine this, we sought to decompose the ERN signal into separate components reflecting signal strength and signal temporal consistency across trials. Researchers have postulated that the ERN signal results from an increase in phase-locked activity in the theta (4-8 Hz) waveband (Luu, Tucker, & Makeig, 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 in 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 indicates the temporal location of the peaks and troughs of the sine waves in a given trial. This approach allows for an ERN signal to be decomposed into signal strength, computed as the average theta power across trials, and temporal consistency, computed as the phase coherence in the theta waveband across trials. Combined, these two components explain the majority of the variance of the ERN (Cavanagh, Zambrano-Vazquez, & Allen, 2012). By examining these components separately, their respective contributions to changes in the average ERN waveform over time can be assessed.

The Present Study

As reviewed above, researchers are increasingly interested in assessing the ERN in younger children with the presumption that this component will serve as a biomarker of individual differences that may be in effect prior to the development of full behavioral syndromes. The ability of the ERN to serve in this role is predicated on several qualities that have, to date, not been fully assessed. First, we examine whether there are developmental changes in the ERN by conducting 3 annual assessments with children from kindergarten through 2nd grade. To our knowledge, longitudinal assessments of the ERN have not been conducted in this age range. We hypothesize that the amplitude of the ERN deflection will increase within individuals over this range, and that behavioral variability will decrease. Furthermore, we hypothesize that significant increases in signal strength and temporal consistency will occur across this time period, accounting in part for the increase in amplitude in the average signal. Second, we examine whether signal temporal consistency is associated with behavioral variability. Given the wealth of evidence of sexually dimorphic effects in neural processes (see Cahill, 2014), we include sex in each of these models in order to determine whether males and females differ in any respect of developmental maturation.

Finally, in addition to the changes occurring as a function of ongoing brain development, we further examine whether the ERN possess trait-like properties by examining individuals' rank-order stability within the sample. The ERN has shown high test-retest reliability ($r = .74$) over a two-week period in a sample of undergraduate students (Olvet & Hajcak, 2009) and ($r = .62$) over the course of 3-6 weeks in middle adolescence (Segalowitz et al., 2010).

Moderate rank-order stability ($r = .65$) of the ERN has also been found amongst college students over the course of 1-2 years (Weinberg & Hajcak, 2011). However, the stability of the ERN in younger samples is unknown. High stability across the 3 assessments would provide strong support for the ERN as an early emerging trait-like factor. In contrast, low stability would indicate that children can undergo differential developmental trajectories during this developmental range and suggest that the ERN does not possess strong trait-like properties until later.

Methods

Participants

Data for this study were drawn from a larger study of children's socioemotional development in conjunction with a school-based preventive intervention, the full details of which can be found in [BLINDED]. Briefly, teachers were asked to respond to a brief behavioral screening questionnaire in the fall of the kindergarten year for all students in the class. Children identified as having behavioral risk (assessed through teacher report of aggressive or oppositional behavioral symptoms; $n = 207$) were recruited, and half were randomly assigned to participate in the intervention component of the study. An additional $n = 132$ children with no symptoms were recruited as comparisons. Because the focus of the present study is on normative developmental progression, only data from the $n = 234$ (65% male) children who were not exposed to the intervention condition, and who had at least one ERP assessment are included. Of these 234 participants 70% were African American, 20% Hispanic, 9% Caucasian, and 1% Asian. Once pooled, this sample of children (which included both children identified as having behavioral risk and comparison children), filled the full range of both externalizing and internalizing symptoms, with normal distributions at the sample level (see BLINDED for more details).

Children were assessed longitudinally in the fall of their kindergarten year (K), spring of their 1st grade year (G1), and spring of their 2nd grade year (G2). At first assessment, children ranged in age from 5.18 to 7.47 years ($M = 6.10$, $SD = .39$). As with any longitudinal study, participant attrition resulted in a smaller sample size at G2 ($n = 163$) compared to K. Participants who did not participate at all three assessments did not differ from those with complete data at the G2 assessment with regard to behavioral variability or ERN amplitude at the initial assessment ($ps > .29$).

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. Research assistants (RAs) escorted the child to the vehicle where they explained the task to the child, who was then asked to give verbal assent. RAs then affixed autonomic and EEG electrodes, and seated the children in front of a computer monitor where they completed a Go/No-go task (described below) followed by an emotion induction task. Only EEG data from the Go/No-go task at the K, G1, and G2 assessments are examined in the present study.

Go/No-go—The Go/No-go task was a modified version of a program developed by M. Lewis and J. Stieben (Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). At the start of each trial a stimulus consisting of either a cartoon character (K) or acomputer-generated human face (G1 and G2) was presented. Participants were instructed to press a button on 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 lasted approximately 10 minutes and consisted of 225 Go trials interspersed with 105 No-go trials. Prior to beginning the task research staff explained the game to the child using full-page color illustrations. The child then engaged in a short practice round to familiarize him/her with the rate of stimulus presentation and to establish task difficulty. In order to ensure an appropriate number of error trials for analyses task difficulty was standardized across all participants. Children with an error rate between 40% and 60% at the end of the practice session began the full game. Children with a higher error rate were given the practice session again until they were able to improve their performance. Children with a lower error rate were also re-administered the practice session with a faster stimulus presentation rate in order to make the task more challenging. At the kindergarten assessment all children began the task at the slowest presentation rate, which was 900ms. During the game an adaptive algorithm made dynamic adjustments to the inter-stimulus interval to help maintain the targeted error rate for the duration of the task.

Prior to beginning the task, children were informed that they would be winning points and that if they earned enough points they would be given a prize that consisted of a gift bag filled with stickers and small toys. Points were awarded for correct responses and subtracted for incorrect responses. Because of the age of the sample, points were presented visually rather than numerically. At slightly variable intervals of approximately 10 trials, cumulative point total was shown with a thermometer bar 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 that lasted approximately 2.5 minutes each. A 30s break was given between each block. During the 1st and 3rd block, participants earned more points for correct responses than they lost for incorrect responses. During the second block the scoring algorithm was changed such that they lost points, a manipulation designed to examine hypotheses related to the role of affect in cognitive processing (in the 3rd block all points are earned back and all children won the prize). However, because that manipulation was unrelated to the developmental questions examined here, the analyses presented below only make use of data from the error trials extracted from the 1st and 3rd blocks. Notably, the pattern of results is identical when examining the error trials from all three blocks.

EEG Recording—EEG data were recorded from a BioSemi ActiveTwo system (BioSemi, Amsterdam, Netherlands) with 32 standard extended 10-20 scalp channels using sintered-silver electrodes. 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. The analog signal was digitized according to the BioSemi zero-reference principle (the voltage at each site is quantified relative to the common mode sense and driven right leg loop) at 1024 Hz and online low-pass filtered at 512 Hz. The recording was

made from DC amplifiers with a gain of one using 24-bit A-D conversion, and filtered offline with a 1-30 Hz bandpass in Brain Vision Analyzer. As a substitute for impedance measures quantifying signal quality, electrode offsets were maintained below 50 μV.

EEG Post-Processing—Data were post-processed using Brain Vision Analyzer 2.0. The time-series for each electrode was 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, Coles, & Donchin, 1983) and data were baseline corrected relative to the average voltage in the window −600 to −400ms prior to the incorrect response so that the baseline would capture a period prior to stimulus onset. Segments containing a voltage step greater than 100μV between sampling points or data outside the range of −75μV to 75μV were marked as artifact and removed.

Additionally, assessments where fewer than 6 No-go error trials remained after artifact rejection were excluded from analyses (loss of 36 assessments from 32 participants). Finally, assessments where error rates exceeded 60% were deemed as indicative of participants' disengagement from the task and were excluded from analysis (loss of 16 assessments from 16 different participants). Clean, processed data from the Fz electrode (total of 494 assessments from 234 participants) were exported to MATLAB and analyzed using custom written software that extracted signal strength and temporal consistency as described below.

Time-Frequency Decomposition—Following procedures described by Mallat (2008), the spectral properties of Fz voltage fluctuations during each trial were described using a set of complex Morlet wavelets given by

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

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 (see also Cavanagh et al., 2012). 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 trial data, one trial at a time, to yield $W_k(t,f)$. The resultant wavelet transformation, represented in Cartesian coordinates as $W_k(t,f) = a + bi$ (where i is the square root of −1), can alternatively be described in Polar coordinates as a function of magnitude and phase offset as shown in Equations 2 and 3.

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

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

These representations of neural activity were subsequently used to calculate average signal strength and signal temporal consistency across trials (described below).

Measures

ERN Amplitude—ERN amplitude was measured for each child at each assessment (K, G1, G2) using a peak-to-peak method. First, a composite waveform was obtained for each participant by averaging the waveforms from artifact-free No-go error trials. ERN amplitude was then derived from the average waveform as the difference in µV between the most negative peak in the 0-100ms post-response window and the most positive peak in the −100-0ms pre-response window.

Average Signal Strength—Using the wavelet transformations described above, average signal strength at each assessment was defined as the average spectral power in the theta waveband (4-8 Hz) from 0 to 100ms following erroneous responses on No-go trials. Specifically,

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

where n is the number of trials, t is time in ms, f is frequency in Hz, and c is a scaling factor such that the result equals the average power at each time point from 0 to 100ms at 4-8Hz.

Signal Temporal Consistency—Similarly, signal temporal consistency at each assessment was defined as the extent of phase coherence in the theta waveband from 0 to 100ms post-response.

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

Behavioral Variability—Behavioral variability at each assessment was measured as the within-person standard deviation of response times on Go trials in which the child responded within a 100 ms and 1000 ms post-stimulus window. Given increasing accuracy with age, behavioral variability was indicated by responses to, on average $M = 150.31$ ($SD = 34.78$; range = 14 – 209) Go trials at the K assessment, $M = 196.59$ ($SD = 35.01$, range = 70 – 252) trials at the G1 assessment, and $M = 217.17$ ($SD = 24.01$, range = 113-254) trials at the G2 assessment.

Data Analysis

Our main interest was to describe and examine (a) age-related changes and sex differences in ERN amplitude, signal strength and signal temporal consistency, (b) the association between behavioral variability and signal temporal consistency, and (c) the extent of stability in between-person differences in these measures across this age range.

Age-related Changes in ERN Measures—To examine age-related changes and sex differences in development, multilevel models of change (Singer & Willett, 2003) were fit to the repeated measures of ERN amplitude, average signal strength, signal temporal consistency, and behavioral variability. Specifically, the model of change was specified as

$$Y_{tj} = \beta_{0j} + \beta_{1j} Age_{tj} + e_{tj}$$

where Y_{tj} is observed outcome (e.g., ERN amplitude, average signal strength) at assessment t for participant j, β_{0j} is a person-specific intercept indicating level of the outcome at age 6 years, β_{1j} is a person-specific linear rate of age-related change, and e_{tj} are assessment-specific residuals. Person-specific intercepts and rates of change are, in turn, modeled as

$$\beta_{1j} = \gamma_{10} + \gamma_{11} Sex_j + u_{ij} \qquad \beta_{0j} = \gamma_{00} + \gamma_{01} Sex_j + u_{0j} \tag{6}$$

where γ_s are sample-level parameters indicating the expected score for the prototypical 6 year old child (γ_{00}), the average rate of age-related change (γ_{10}) the extent of sex differences in those expected scores (γ_{01}), and rates of change (γ_{11}), and residual between-child differences unrelated to sex, u_{0j} and u_{1j}, that may be correlated but are uncorrelated with assessment specific residuals e_{tj}. Sex was coded such that males = 1 and females = −1. Following usual trimming practices, random effects were trimmed to obtain the best fitting error structure. In the present context this meant constraining the between-child variance in rates of age-related change to zero when the parameter did not contribute significantly to model fit. Follow-up analyses were used to examine if and how age-related changes in the typically examined ERN amplitude may be mediated by changes in signal temporal consistency using the Sobel (1982) method (see Krull & MacKinnon, 2001). All models were fit using SAS 9.3 (proc mixed, Littell, Milliken, Stoup, & Wolfinger, 1996) using minimum variance quadratic unbiased estimation (Rao, 1972) and treating incomplete data as missing at random (Little & Rubin, 1987). Maintaining conservative evaluation with respect to the sample size, observations more than 3 standard deviations away from the sample mean within a given assessments (i.e. 3 or 4 observations) were excluded from each analysis (results from analyses with or without the outliers removed do not differ). Significance of effects was evaluated at α = .05.

Associations among ERN Amplitude, Signal Components 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. Due to data missing for individual assessment points either due to attrition or exclusion of artifactual data the sample sizes for these correlations range from $n = 106$ to $n = 199$.

Trait-like properties of ERN

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.

Results

Descriptive statistics for all measures are given in Table 1. Average amplitude of ERN deflections in μV increased across the three assessments (from −6.00 to −7.35). The effect sizes for year-to-year change in the sample-level averages were small ($d = .18$ and $d = .20$ respectively). Highlighting the importance of development time-scale, these sample-level changes accumulate to what is a moderate effect size when looking at change from K to G2 ($d = .39$). Average behavioral variability decreased across the three assessments (from 138.33 to 109.13). Notably, the sample also became more homogenous with respect to the dispersion of children's behavioral variability (between-child variance going from 34.19 to 21.51). Effect sizes for year-to-year change were moderate ($d = .33$ and $d = .68$ respectively), accumulating into a large effect when looking at change from K to G2 ($d = 1.12$). Average signal temporal consistency increased across the three assessments (from .23 to .28) with effect sizes for year-to-year change in the sample-level averages being in the small to moderate range ($d = .38$, $d = .22$ respectively) and accumulating to $d = .56$ for K to G2. Signal strength decreased across the three assessments (from 33.34 to 32.88) with year-to-year effect sizes ($d = .20$, $d = .27$ respectively) comparable to the other ERN metrics, accumulating to $d = .45$ for K to G2.

Age-related Changes in ERN Measures and Behavioral Variability

Results from the multilevel models of change examining age-related change and sex differences are given in Table 2 and illustrated in Figure 1. The pattern seen in the sample-level averages noted above was reflected in the formal models of age-related change, with significant age-related changes apparent in all four measures of neural activity and behavior. For example, the prototypical trajectory for ERN amplitude was characterized by a deflection of $\gamma_{00} = -5.90$ ($p < .05$) at age 6 years, which increased in negativity at a rate of $\gamma_{10} = -0.61$ ($p < .001$) units per year. Similarly, the prototypical trajectory of signal strength declined at a rate of $\gamma_{10} = -0.22$ ($p < .001$) per year. In contrast, signal temporal consistency increased from $\gamma_{00} = 0.24$ ($p < .001$) at a rate of $\gamma_{10} = 0.02$ ($p < .001$) units per year. No main effects for sex or sex × age interactions were apparent for any of the three ERN measures ($ps > .10$). Behavioral variability exhibited age-related decline at an average rate of $\gamma_{10} = -12.64$ ($p < .001$) units per year. Sex differences in levels of behavioral variability did not significantly differ ($\gamma_{01} = 3.55$, $p = .11$; boys not significantly higher than girls) around an average level at age 6 ($\gamma_{00} = 139.76$, $p < .001$). However, boys showed significantly faster decline over time than girls ($\gamma_{11} = -2.00$, $p = .05$).

Signal Temporal Consistency as a Mediator of Developmental Changes in ERN Amplitude

Given that signal temporal consistency increased with age, the hypothesis that increases in ERN amplitudes were mediated by increases in signal temporal consistency was directly examined. A mediational model for signal strength was not examined because the hypothesis that signal strength would increase with age was not supported. In order to formally test the mediational effect, signal temporal consistency (person-centered) was added to the model of change (Eq. 6) as a time-varying predictor (γ_{20}) of ERN amplitude.

Results of the mediation model are presented in Table 3. Increases in signal temporal consistency over time significantly predicted more negative ERN amplitudes ($\gamma_{20} = -12.64$, $p < .001$). Sobel's z-test (Sobel, 1982) revealed that changes in signal temporal consistency significantly mediated the association between age and ERN amplitude ($z = -3.4$, $p < .001$), and accounted for 38% of the total effect of age on ERN amplitude.

Associations among ERN Amplitude, Signal Temporal Consistency and Behavioral Variability

Pearson's Correlations among behavioral variability and ERN amplitude, average signal strength, and signal temporal consistency are reported in Table 4 and illustrated in Figure 2. As expected, more negative ERN deflections were associated with greater signal strength and greater temporal consistency at each assessment. Signal strength and temporal consistency did not correlate with each other, consistent with the notion that each contributes independently to ERN amplitude.

The hypothesized relation between lower ERN amplitude and greater behavioral variability was $r = .13$, $p = .06$, at K, behavioral variability at K was significantly associated with lower signal temporal consistency ($r = -.15$, $p = .03$), which in turn was associated with less negative ERN amplitudes ($r = -.43$, $p < .001$). At G1, behavioral variability was not associated with any of the ERN measures. At G2, there was a significant association between greater behavioral variability and less negative ERN amplitudes as hypothesized ($r = .26$, $p = .002$). Greater behavioral variability at G2 was correlated $r = -.15$, $p = .07$, with lower signal temporal consistency and $r = -.25$, $p = .003$ with lower signal strength.

Rank Order Stability of ERN metrics and Behavioral Variability

Rank order stability coefficients among the K, G1, and G2 assessments are presented in Table 4. As expected, rank order stability in behavioral variability was moderate across all 3 assessments (rs = .44 to .52, ps < .001). ERN amplitude measured at K ($r = .18$, $p = .06$) and at G1 ($r = .23$, $p = .02$) were both positively correlated with ERN amplitude at G2, somewhat suggestive of stability. Contrary to expectations however, amplitude measured at K did not significantly correlate with amplitude measured at G1 ($r = .11$, $p = .21$). Average signal strength showed high stability across all time points (rs = .56 to .70, ps < .001). In contrast, rank order of signal temporal consistency at any one assessment was not associated with rank order at other assessments (rs = .03 - .09, ps > .30).

Discussion

The present study characterized the ERN signal in terms of both signal strength and temporal consistency and examined how these aspects of the ERN changed over 3 years during childhood. Although the ERN has been well studied in adults, far less is known about the presence of the ERN in children younger than age 8, and a lack of longitudinal data leaves much unknown about the stability of the ERN over developmental time. The longitudinal design of the present study allowed for the investigation of the relation of within-person changes in behavioral variability and neural activity underlying performance monitoring as well as the stability of between-person differences in this age range. Consistent with cross-sectional studies, ERN amplitude became increasingly negative with age (Davies et al., 2004; Wiersema et al., 2007). Although ERN amplitudes are related an individual's confidence in their performance and thus increase with learning (see Walsh & Anderson, 2012), it is unlikely that the effects observed in this study are a function of repeated experience with the task across assessments. The task was designed to minimize learning effects as task rules were made explicit through visual instructions and practice trials prior to the start of ERP recording. Further, task difficulty was adjusted over time to account for increases in skill. Most importantly, the decomposition of the ERN into elements of signal strength and temporal consistency suggests that the increasingly negative deflections are not a function of greater signal strength but rather an increase in temporal consistency of the neural signal over this time period, supporting the conjecture that these changes are a result of developmental maturation. Results also indicate that these two distinct components of the ERN signal show different levels of rank-order stability during this developmental period.

Within-Person Changes in the ERN

To our knowledge, this is the first study to examine how signal strength and signal temporal consistency contribute to ERN amplitude during childhood. In particular, this study examines children from approximately age 6- through age 8- years, when the ERN signal is thought to be emerging. Difficulty in identifying a clear ERN signal on grand averaged waveforms is thought to result from greater inter-individual variability in this age range. The current study further suggests that ERN amplitude in younger children may be affected by greater levels of *intra*-individual variability in the temporal consistency of the neural process under investigation. Low levels of signal temporal consistency could explain why some children fail to show an ERN signal in their average waveform despite having the ability to behaviorally exhibit performance monitoring capabilities. Furthermore, the pattern of findings presented here indicates that only age-related increases in signal temporal consistency accounted for the increased ERN amplitudes, as signal strength showed an unpredicted decline across the three assessments.

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 that affect the polarization of post-synaptic neurons. Post-synaptic neurons integrate signals from numerous sources and fire an action potential only if the threshold potential is reached. The

effect of a single action potential on a post-synaptic neuron, however, will diminish with time. Therefore, 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 maximize functionality 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. Thus signal strength may decline as temporal precision increases, allowing the system to achieve the same level of function with less energy expended. This suggests that, at least in this age range, amplitude extracted from the average waveform may not accurately reflect capacity as it is unduly affected by low temporal consistency.

Some previous studies support the supposition that signal strength serves a compensatory role early in development when temporal consistency is low. Results from a previous study investigating age-related changes in the dynamics of auditory perception across childhood found a decrease in average signal strength and an increase in signal temporal consistency similar to the changes reported in the current study (Müller, Gruber, Klimesch, & Lindenberger, 2009). This pattern of EEG findings could also be considered consistent with developmental data from neuroimaging. Neuroimaging studies lack the temporal precision to differentiate between signal strength and trial-to-trial temporal consistency, but studies often find that children exhibit more diffuse fMRI activation patterns than adults, even in the absence of performance differences. This could reflect less efficient neural systems that require a greater number of resources to process the same amount of information (Casey, Giedd, & Thomas, 2000; Durston et al., 2006).

Although some studies have reported differences in ERN amplitude between males and females (Davies et al., 2004; Larson, South, & Clayson, 2011), no significant sex differences were observed here. Our results are consistent with the lack of sex differences in other ERN research with pre-pubertal participants (Davies et al., 2004; Torpey, Hajack, Kim, Kujawa, & Klein, 2012). The current findings thus extend support for a lack of sex differences in ERN amplitude as well as the signal strength and temporal consistency components of amplitude, and the rate of developmental change at these ages. However, sex differences did emerge in the developmental progression of behavioral variability. Boys on average showed more rapid declines in variability across the 3 assessments.

Stability in Between-Person Differences in ERN

Despite evidence of significant within-person changes, between-person stability in relative rank could still be maintained if developmental changes occurred in a homogeneous manner (i.e. all children change in a similar fashion). Previous studies have found the ERN to be a stable trait-like measure in adulthood and evidence of stability in younger children would

suggest that the ERN is a strong biomarker of individual differences that could be inferred regardless of the assessment age (Olvet & Hajcak, 2009; Segalowitz et al., 2010). In this sample, however, evidence was inconsistent for the rank-order stability of the ERN across time. Both K and G1 ERN amplitudes were modestly correlated with G2 ERN amplitude suggestive of some degree of stability. Unexpectedly, amplitude measured a K did not correlate significantly with amplitude measured at G1. It is not clear what explains this lack of relation, but the finding reduces confidence in the trait-like stability of ERN amplitude at this age. However, despite low stability in the ERN amplitude, evidence of high stability was found across all three assessments for average signal strength. The highly trait-like nature of average signal strength suggests that it may be a better measure of trait-like individual differences in younger children than the more traditionally measured average amplitude. In contrast, signal temporal consistency showed no rank-order stability over this age range. Thus the relatively modest stability seen in the average amplitude may occur because amplitude contains one highly stable component (signal strength) and one unstable component (signal temporal consistency). Future research is needed to identify the experiential factors contributing to developmental changes in temporal consistency as well as when individual differences in temporal consistency stabilize and become trait-like.

The lack of correlation between the K and G1 amplitude measures, despite both being correlated with G2, is unexpected and warrants caution in interpretation. The G1 assessment is further anomalous in that this was the only time point during which behavioral variability showed no correlation with average amplitude. Although it is unclear what mechanisms underlie this pattern, it is possible to speculate on a developmental explanation. It is possible that the age range captured in this study spans a tumultuous transitional phase in neural organization as temporal consistency increasingly accounts for ERN amplitude. At young ages, individual differences in neural processing may be driven primarily by signal strength as individuals are near a developmental floor on signal temporal consistency. As children age, they will presumably approach a developmental ceiling at which further individual increases in consistency are unlikely. Near asymptote, temporal consistency would become more trait-like. The rate of this transitional process of decreasing signal strength and increasing signal temporal consistency may be highly variable between persons leading to instability in rank when measured at a time when the system is still in flux. It is possible that although individuals differ in their developmental rate, individuals may still arrive at a somewhat stable point within the distribution. As an example, pubertal development is characterized by a nonlinear progression between uniform beginning and endpoints. Although all individuals start at the same biologically pre-pubertal state and end at full sexual maturity, individual differences in timing and duration of the transition lead to temporary fluctuations in rank order in a variety of attributes (Marceau, Ram, Houts, Grimm, & Susman, 2011). Because of this, height measured in childhood correlates more strongly with adult height than height measured in adolescence, despite adolescence being a more proximal time point (see Wohwill, 1980 for discussion). Similarly, differences in timing and rate of development during this period of rampant neural change could temporarily obscure the appearance of trait-like stability in ERN amplitude. Far more research is needed to identify the factors contributing to the developmental parameters of this change process as well as its predictive validity for psychological outcomes.

Association between Neural and Behavioral Variability

Consistent with previous findings, the present study showed that behavioral variability decreased as children moved from K through G2, thought to reflect increased efficiencies in neural processing (Eckert & Eichorn, 1977; Segalowitz & Segalowitz, 1993). Despite the significant degree of within-person change, between-person stability for behavioral variability was comparable to that reported in adults (Flehmig, Steinborn, Langer, Scholz, & Westhoff, 2007; Saville et al., 2011). The rate of change was observed to be somewhat faster in boys than in girls in this sample.

Higher levels of behavioral variability were associated with less negative ERN amplitudes at K and G2, with no association observed at G1. The finding that behavioral variability also does not correlate with ERN amplitude at G1 further supports the notion that the developmental flux in the neural systems at this stage may be temporarily obscuring the presence of otherwise stable associations. Behavioral variability was, as expected, correlated with signal temporal consistency at K and G2. At the G2 assessment, a correlation also emerged between behavioral variability and signal strength. The emergent correlation between signal strength and behavioral variability may reflect the developmental changes in the distribution of behavioral variability. As the children aged, behavioral variability declined in both level and variance, presumably reflecting the effects of maturation over that 2.5 year window. High behavioral variability at K could be the result of many factors including inefficiencies in any neuronal system involved in completing the task (e.g. stimulus processing, response selection, and motor execution). As these various systems mature, behavioral variability that remains high at G2 may be a more specific indication of serious performance monitoring deficits as reflected by low error-related signal power in addition to low signal temporal consistency.

Limitations

Although this sample size of this study is relatively 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. Assessments for this study were also conducted at fairly long intervals and thus provide a coarse depiction of the timing of developmental changes and the short term stability of these metrics.

The participants in the current study were predominantly African American and resided in an urban, low socioeconomic status (SES) region, which could affect the generalizability of the study's findings. Socioeconomic factors have been shown to influence neural development (Bradley & Corwyn 2002). Although this may temper comparability of developmental timing of neural changes with other samples, the developmental change itself is presumed to be characteristically human and thus generally consistent across all potential samples. Future replications with samples from different demographic strata should examine whether the developmental trends reported here occur earlier or proceed more rapidly in

higher-resource populations, as well as what types of contextual and experiential factors affect this developmental timing.

The present study also oversampled for aggression and half of the participants were reported to have at least some problem behaviors at the start of schooling. Both externalizing and internalizing disorders have been linked to differences in ERN amplitude and the greater range of these symptoms in the present sample compared with typical community-recruited samples in studies of normative development may have contributed to greater variability across participants. Using a sample that covered the full symptom range, and thus reducing concern about restriction of range present in more homogeneous samples, provided for a relatively stronger test of stability. However, this must also be taken into account when comparing findings to different samples. For instance, externalizing disorders have been characterized by relative developmental immaturity and thus the timing of the transitions in signal strength and temporal consistency reported here may occur at even earlier ages in higher SES samples. Given the interest of using the ERN to obtain better understanding of the developmental risk for psychopathological outcomes, it is especially critical to track the developmental progression of this ERP in high-risk samples, keeping in mind that both the characteristics of the ERN *and* how those characteristics develop over time could differ between groups.

Summary

Overall, this study demonstrates the potential utility of decomposing the ERN into two independent components: average signal strength and temporal consistency across trials, when examining the ERN in children. The increase in ERN amplitude observed as children age appears to be due to an increase in trial-to-trial temporal consistency and occurs despite a decline in signal strength over this time period. Moreover, signal strength demonstrated high rank-order stability over time, suggesting that this measure may be particularly useful for studying trait-like differences associated with the ERN amplitude. Conversely, signal temporal consistency did not demonstrate stability over time and therefore may be more sensitive to experiential or situational influence. Although more work is needed to understand the developmental mechanisms underlying changes in neural processing over time, our results indicate the value of considering both signal strength and temporal consistency when examining the ERN in younger children.

Acknowledgments

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Research Highlights

- We extracted signal strength and signal variability on a trial-to-trial basis, both of which correlated with the ERN measured as an average across trials, but did not correlate with each other.

- Across 3 annual assessments from an average age of 6 to age 8, signal strength showed high inter-individual stability whereas temporal consistency did not.

- Across the 3 years, average ERN amplitude increased, consistent with previous developmental research.

- The increase in average ERN amplitude was mediated by an increase in signal temporal consistency. Contrary to predictions, signal strength declined over this period.

- Brain development during this period may be characterized by a shift from a more resource-intensive allocation of signal strength toward a more efficient pattern of temporal consistency.
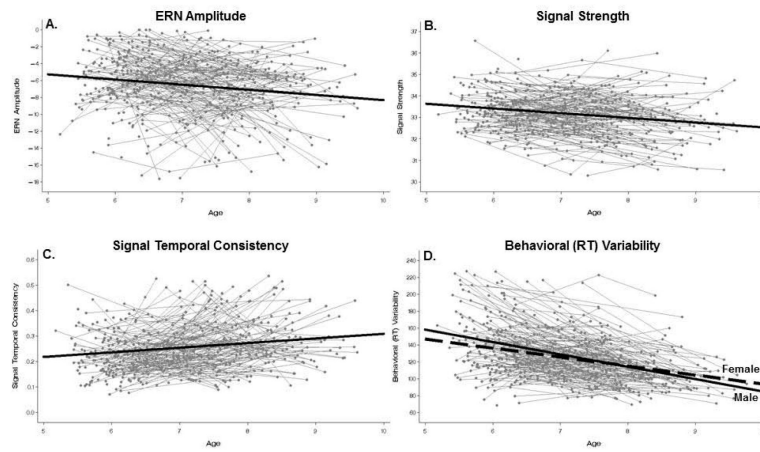
Figure 1.
Plots of ERN amplitude(A), signal components (B, C), and behavioral variability (D) across time. Bold black lines illustrate the prototypical trajectory in each measure over time. Data points and trajectories are combined across males and females for models with no significant effect of Sex. Prototypical male and prototypical female lines are included for behavioral variability to illustrate the significant Age x Sex interaction effect for that variable.
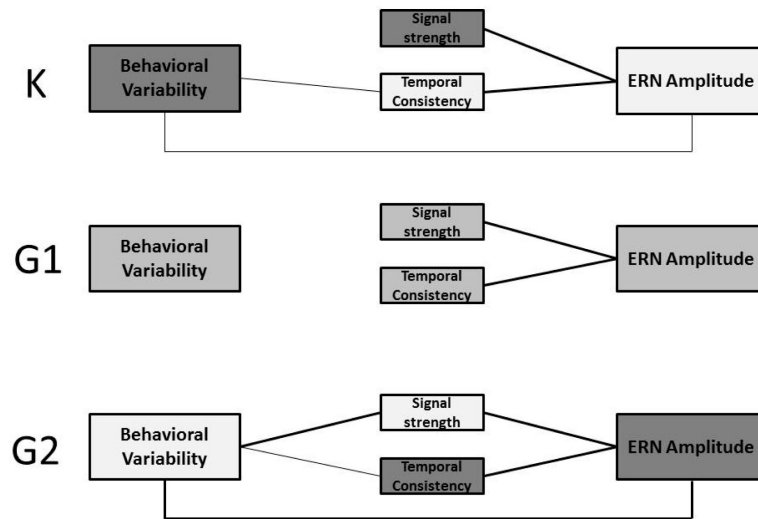
Figure 2.
Concurrent associations among ERN amplitude, signal components, and behavioral variability. Bold lines reflect significant correlations whereas thin lines reflect associations at the trend level. Developmental changes in average levels across the 3 years are depicted by changes in shading where darker boxes represent higher values.

Table 1

Sample-level descriptives across Kindergarten, Grade 1, and Grade 2.

Assessment	Age	ERN Amplitude	Signal Strength	Temporal Consistency	Behavioral Variability
K (n = 218)	6.10 (.39)	−6.00 (3.41)	33.34 (0.88)	.23 (.08)	138.33 (34.19)
G1 (n = 172)	7.20 (.41)	−6.62 (3.61)	33.16 (1.02)	.26 (.09)	126.96 (26.38)
G2 (n = 162)	8.27 (.56)	−7.35 (3.58)	32.88 (1.09)	.28 (.10)	109.13 (21.51)

Note. Empirical sample-level means (standard deviations in parentheses). K = Kindergarten, G1 = Grade 1, G2 = Grade 2. ns vary as all children did not participate or provide usable data at all waves. Participants with incomplete data did not significantly differ on the outcomes of interest from those with complete data.

Table 2

Results from multilevel models of change examining developmental changes and sex differences in ERN Amplitude, Average Signal Strength, Signal Temporal Consistency and Behavioral Variability.

Parameter	ERN Amplitude		Average Signal Strength		Signal Temporal Consistency		Behavioral Variability	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Fixed Effects								
Intercept, γ_{00}	−5.90*	(0.25)	33.40*	(0.07)	0.24*	(0.01)	139.76*	(2.19)
Sex, γ_{01}	−0.11	(0.25)	−0.03	(0.07)	−0.01	(0.01)	3.55	(2.19)
Age, γ_{10}	−0.61*	(0.16)	−0.22*	(0.03)	0.02*	(0.004)	−12.64*	(1.01)
Sex*Age, γ_{11}	−0.01	(0.16)	0.04	(0.03)	0.007	(0.004)	−2.00*	(1.01)
Random Effects								
Variance, intercept σ^2_{u0}	1.98*	(0.75)	0.65*	(0.08)	0.0005	(0.0005)	587.48*	(93.44)
Variance, Age σ^2_{u1}	--	--	--	--	--	--	27.24	(32.10)
Cov intercept, Age σ_{u0u1}	--	--	--	--	--	--	−122.56*	(42.92)
Residual variance	10.40*	(0.89)	0.32*	(0.03)	0.0082*	(0.0007)	402.53*	(50.03)
−2LL	2596.6		1245.5		−890.8		5116.5	

*
$= p < .05$. −2LL = −2 Log Likelihood. Analyses based on between 485 and 552 Go-/No-go trials nested within 234 children.

Table 3

Results from multilevel model of change examining extent to which association between Age and ERN Amplitude is due to developmental changes in Signal Temporal Consistency.

Parameter	ERN Amplitude	
	Estimate	SE
Fixed Effects		
Intercept, γ_{00}	−6.11[*]	(0.24)
Sex, γ_{01}	−0.20	(0.24)
Age, γ_{10}	−0.38[*]	(0.15)
Signal temporal consistency, γ_{20}	−12.64[*]	(2.21)
Sex[*]Age, γ_{11}	0.09	(0.15)
Random Effects		
Variance, intercept σ^2_{u0}	3.11[*]	(0.72)
Variance, signal temporal consistency σ^2_{u1}	136.80[*]	(79.32)
Cov intercept, temporal consistency σ_{u0u1}	9.33	(5.74)
Residual variance	7.21[*]	(0.80)
−2LL	2495.5	

Notes. Signal temporal consistency entered as a time-varying person-centered variable; age centered at 6 years; Sex coded males = 1 and females = −1.

[*] = p<.05. −2LL = −2 Log Likelihood. Analyses based on 482 Go-/No-go trials nested within 226 children.

Table 4

Pairwise Pearson Correlations Amongst Measures.

	1	2	3	4	5	6	7	8	9	10	11
1. K ERN Amp	--										
2. G1 ERN Amp	.11	--									
3. G2 ERN Amp	.18*	.23*	--								
4. K Signal Strength	-.29*	-.32*	-.17 t	--							
5. G1 Signal Strength	-.19*	-.37*	-.23*	.62*	--						
6. G2 Signal Strength	-.15 t	-.28*	-.36*	.56*	.70*	--					
7. K Temporal Consistency	-.43*	.10	-.06	.11	.02	-.01	--				
8. G1 Temporal Consistency	-.05	-.22*	.02	.01	-.04	.05	.08	--			
9. G2 Temporal Consistency	-.09	-.06	-.37*	.05	.02	.06	.03	.09	--		
10. K Behavioral Variability	.13t	-.04	-.03	.00	.00	.02	-.15*	-.17*	-.13	--	
11. G1 Behavioral Variability	.09	.08	.08	-.08	-.08	-.09	-.14 t	-.07	-.04	.52*	--
12. G2 Behavioral Variability	.22*	.13	.26*	-.11	-.16 t	-.25*	-.03	-.12	-.15t	.44*	.50*

*
Note. p < .05,

t
p< .10. *ns* for each cell range from 106 to 199.