The Relation between Executive Functions, Error-related Brain Activity, and ADHD Symptoms in Clinically Evaluated School-Aged Children

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Abstract

Two event-related potentials (ERPs) elicited following errors, the *error-related negativity* (ERN) and *error positivity* (Pe), have been proposed to reflect cognitive control, though the specific processes remain debated. Few studies have examined the ERN and Pe's relations with individual differences in cognitive control/executive functioning using well-validated tests administered separately from the inhibition tasks used to elicit the ERN/Pe. Additionally, neurocognitive tests of executive functions tend to strongly predict ADHD symptoms, but the extent to which task-based and EEG-based estimates of executive functioning/cognitive control account for the same variance in ADHD symptoms remains unclear. The current study addressed these limitations by examining relations between the ERN/Pe and three core executive functions (working memory, inhibitory control, set shifting) in a clinically-evaluated sample of 53 children ages 8–12 (M_{age}=10.36, SD=1.42; 77.4% White/Non-Hispanic; 16 girls) with and without ADHD. Results demonstrated that neither the ERN nor Pe were related to overall cognitive control/executive functioning, or to working memory or set shifting specifically (all 95%CIs include 0.0). In contrast, a larger Pe was associated with better-developed inhibitory control (β=−.35, 95%CI excludes 0.0), but does not capture aspects of inhibitory control that are important for predicting ADHD symptoms. Neither the ERN nor Pe predicted ADHD symptoms (95%CIs include 0.0). Results were generally robust to control for age, sex, SES, ADHD symptom cluster, and anxiety, and emphasize the need for caution when interpreting the ERN/Pe as indices of broad-based cognitive control/executive functioning, as well as using the ERN/Pe to examine cognitive processes contributing to ADHD symptomatology.

Keywords

Error-related negativity; error positivity; executive function; cognitive control; ADHD

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Conflict of Interest:

The principal investigator (Michael Kofler) holds a patent for a neurocognitive intervention that targets central executive working memory and is licensed to Sky Therapeutics, where Michael Kofler serves as Chief Science Officer and consultant. This intervention was not used in the current study, and there are no other current licensing, financial, or other conflicts to report.

Event-related potentials (ERPs) are a useful tool for assessing cognitive processes because they are a direct measure of brain activity and have demonstrated clinical utility in predicting both risk for and developmental trajectories of psychopathology (Hajcak et al., 2019). Two ERPs that are elicited following errors on speeded decision-making tasks, the *error-related negativity (ERN)* and *error positivity (Pe)*, have been proposed to reflect cognitive control mechanisms given that errors represent a breakdown in performance that is detected and subsequently acted upon using performance monitoring processes (Cavanagh & Shackman, 2015; Falkenstein, 2004; Gehring et al., 2012; Overbeek et al., 2005). However, despite the common reification of the ERN and Pe as indices of cognitive control, relatively few studies have examined their relations with individual differences in cognitive control/executive functioning (Meyer & Hajcak, 2019). As such, it is also unclear if and how the ERN and Pe may overlap with the executive function/cognitive control processes that are associated with both the etiology and behavioral symptom expression of attention-deficit/hyperactivity disorder (ADHD), a common neurodevelopmental disorder (e.g., Kofler et al., 2019). The present study examined the extent to which the ERN and Pe are related to overall and specific components of cognitive control/executive functioning (working memory, inhibitory control, set shifting) to inform characterization of the ERN and Pe as proxies of cognitive control, as well as their interrelations with ADHD symptoms in a relatively small but carefully phenotyped and clinically evaluated sample of children with and without ADHD.

Executive Functions and Cognitive Control

Executive functions refer to a set of interrelated, higher-order processes that regulate and enable cognition, goal-directed behaviors, and problem solving (Miyake et al., 2000). *Cognitive control*, a closely related construct, refers to mental processes that enable the continuous adjustment of behaviors in accordance with actively changing goals and task demands to optimize performance (Botvinick & Cohen, 2014; Nigg, 2017)[i]. Executive function deficits are theorized to play an etiological role in several forms of psychopathology, and are considered to play a prominent/primary role in the development and behavioral expression of ADHD (e.g., Rapport et al., 2013). Miyake et al. (2000) proposed three primary executive function domains: working memory, inhibitory control, and set shifting. Briefly, *working memory* involves the active, top-down manipulation of information held in short-term memory through interrelated functions of updating, dual-processing, and temporal/serial reordering (Fosco et al., 2020). *Inhibitory control* refers to the processes that support the ability to deliberately withhold or stop a dominant and/or on-going response (Alderson et al., 2007). Finally, *set shifting* involves cognitive flexibility in shifting back and forth between mental sets or tasks (Irwin et al., 2019; Miyake et al., 2000).

[i]Nigg (2017) concluded that cognitive control overlaps significantly with specific facets of executive function, particularly the core ‘lower-level’ executive functions that appear first developmentally (working memory and response inhibition, followed by set shifting; Diamond, 2013; Karr et al., 2018; Nigg, 2017). The subtle distinction between the two terms is rooted in their development within different disciplines (Nigg, 2017). Given that cognitive control and executive functioning are closely related constructs from separate disciplines, we use the terms interchangeably in the present study. Relatedly, our use of the term ‘executive functions’ is consistent with Nigg (2017)’s conceptualization of ‘lower-level executive functions’, which reflect the ‘core’ executive functions in the Miyake et al. (2000) model (Kofler et al., 2019).

ERN, Pe, and Executive Function

The ERN is characterized by a negative deflection about 50 to 100 ms after an individual makes an error, and is most readily observed at frontocentral scalp locations (Falkenstein et al., 1991; Gehring et al., 1993, 2012). Evidence suggests that the ERN is generated in the anterior cingulate cortex (ACC; Mathewson et al., 2005; van Veen & Carter, 2002), a brain region implicated in conflict and outcome monitoring (Botvinick et al., 2004). The Pe typically follows the ERN and is marked by a slow positive wave occurring centroparietally on the scalp between about 200 and 400 ms after an individual makes an error (Falkenstein et al., 1991; Overbeek et al., 2005).

Support for the ERN and Pe as proxies of executive functioning/cognitive control includes evidence that the ERN is associated with accuracy-speed tradeoffs (Gehring et al., 1993) and that a larger ERN is related to greater post-error slowing in adults during response inhibition tasks (Hirsh & Inzlicht, 2010; for review, see Cavanagh & Shackman, 2015). However, while behavioral data in some ERN studies found that fewer errors on inhibition tasks were associated with a larger ERN in children and adolescents (Lo et al., 2017; Mehra & Meyer, 2022; Overbye et al., 2019), others did not find this relation (Gorday & Meyer, 2018; Grammer et al., 2018; Kim et al., 2016). Similarly, an increased Pe in adults has been associated with fewer errors (Falkenstein et al., 2000; Mathewson et al., 2005) and increased post-error slowing (Chang et al., 2014; Hajcak et al., 2003) during response inhibition tasks. However, studies relating the Pe and inhibitory control in children and adolescents are mixed (Grammer et al., 2018; Kim et al., 2016; Overbye et al., 2019), and to our knowledge no studies have examined these relations specifically in school-aged children – a critical omission given that (a) the ERN and Pe follow different developmental trajectories, with the Pe maturing earlier and the ERN continuing to increase into early adulthood (Boen et al., 2022; Downes et al., 2017); and (b) school-aged children's executive functioning/cognitive control abilities also follow different developmental trajectories (Best et al., 2009; Best & Miller, 2010), with working memory and inhibitory control reflecting unique executive functions in early childhood (Lerner & Lonigan, 2014) and set shifting becoming a distinct ability in later school-aged children (Karr et al., 2018).

Although the extant literature is informative for understanding the relations between the ERN/Pe and inhibitory control, it is unclear if such findings extend to executive functioning/cognitive control overall, or to executive functions beyond inhibitory control (i.e., working memory, set shifting). In addition, the evidence is based primarily on performance parameters measured during the same inhibitory control tasks from which the ERPs are derived (Meyer & Hajcak, 2019), which questions the extent to which these findings may be artifacts of mono-task bias (i.e., attributable to illusory associations when multiple metrics from the same task/measure are compared). Given evidence supporting the reliability and validity of these ERPs as individual difference markers (Riesel et al., 2013; Weinberg et al., 2012), there is a need for studies using (a) construct valid executive function/cognitive control batteries completed separately from the task used to capture ERN/Pe parameters, and (b) measures of working memory and set shifting in addition to inhibitory control – yet few studies have used this approach (Meyer & Hajcak, 2019).

A partial exception to this critique comes from a recent adult study that suggests associations between the ERN/Pe and global estimates of executive functioning/cognitive control (Larson & Clayson, 2011). Similarly, a recent study with young children found that an increased ERN was associated with greater cognitive control (Meyer & Klein, 2018). However, the conclusions that can be drawn from these studies may be limited because they relied primarily on tests that were developed to assess gross neuropsychological functioning and questionnaire-based assessments of executive function/cognitive control processes that show poor correspondence with specific tests of these cognitive processes (for reviews, see Snyder et al., 2015 and Soto et al., 2020). However, two studies conducted with college students addressed these limitations with construct valid working memory tests, and found that a larger ERN and Pe were associated with better working memory (Coleman et al., 2018; Miller et al., 2012).

ADHD, Executive Functioning, and Error-related ERPs

Given that (a) the ERN and Pe have been reified as indices of executive functioning/cognitive control, and (b) many if not most children with ADHD have deficits in executive functioning (e.g., Kofler et al., 2019), it is unsurprising that multiple studies have examined the ERN and Pe in individuals with ADHD. However, as with the ERN/Pe-executive function findings in the developmental/cognitive literatures, results from these ADHD studies are mixed (for reviews, see Meyer & Hajcak, 2019; Shiels & Hawk, 2010). A meta-analysis of seven studies found that individuals with ADHD exhibit a blunted (i.e., significantly reduced) ERN compared to controls ($d = 0.50$), but that a blunted Pe may be detectable only during certain types of inhibition tasks such as the go/no-go task used in the current study ($d = 0.68$; Geburek et al., 2013). In contrast, a more recent meta-analysis found the Pe to be blunted in ADHD groups compared to non-ADHD groups ($d = -0.39$), while no significant group differences were found in the ERN ($d = 0.21$; Kaiser et al., 2020). Similarly, Meyer & Hajcak (2019) conducted a box score review and found that 14 studies showed a blunted ERN in ADHD, 11 showed no differences between ADHD and control groups, and still 1 showed an increased ERN in ADHD. Indeed, in a review of the use of EEG in ADHD research, McLoughlin and colleagues (2022) conclude that while research on group differences in the ERN remain inconclusive, a reduced Pe in ADHD groups is more consistently, albeit not always, found.

Few studies have taken a dimensional approach to examining the relation between ADHD and the ERN and Pe. While some have found greater ADHD symptoms to be associated with a smaller ERN amplitude in adolescents and adults (Marquardt et al., 2018; Rommel et al., 2019), others have not found this relation (Herrmann et al., 2009; Wiersema et al., 2009). Similarly, an association between greater ADHD symptoms and a reduced Pe has been found in some studies (Herrmann et al., 2009; Wiersema et al., 2009), but not others (Rommel et al., 2019). The mixed results at both the categorical and dimensional level have been hypothesized to be attributable to varying participant characteristics and methodological approaches across studies (Kaiser et al., 2020; McLoughlin et al., 2022).

Current Study

Taken together, the current literature provides some support for associations between the ERN/Pe and neurocognitive test-based estimates of executive functioning/cognitive control, as well as evidence linking test-based and potentially EEG-based executive function/cognitive control tests with ADHD symptoms. However, no study to date has examined whether the test- and EEG-based indices predict the same variance in ADHD symptoms. The current study addresses this limitation and is the first to be conducted specifically with school-aged children, use well-validated assessments of all three core executive functions (working memory, inhibitory control, and set shifting) that were administered separately from the tasks used to evoke the ERN and Pe, and consider the extent to which the ERN/Pe are capturing the aspects of executive functioning/cognitive control that are predictive of ADHD behavioral symptoms. We hypothesized that an increased ERN and Pe would be associated with better-developed executive functioning/cognitive control overall, and with better-developed working memory and inhibitory control specifically; no specific hypotheses regarding set shifting were offered due to the paucity of prior research. Additionally, we hypothesized that blunted ERN and Pe metrics would be associated with greater ADHD symptoms, and would capture the same variance in the executive function/cognitive control tests associated with ADHD symptoms.

Method

Participants

The sample included 53 children between the ages of 8 and 12 years (M = 10.36, SD = 1.42; 16 girls) from the Southeastern U.S. who were (a) recruited through community resources from 2018–2020 for a clinical research study of neurocognitive mechanisms underlying pediatric attention and behavior problems; and (b) completed EEG testing during evaluation. Recruitment to this study was closed in March 2020 due to the COVID-19 pandemic. The sample size reflects consecutive cases whose evaluations were completed prior to the COVID-19 shutdown. The Florida State University IRB approved the study prior to and throughout data collection, and parents and children gave informed consent/assent. Sample ethnicity consisted of 41 White Not Hispanic or Latino (77.4%), 6 Black or African American (11.3%), 2 Hispanic or Latino (3.8%), and 4 multiracial (7.5%) children. All children spoke English.

All children and caregivers completed a comprehensive evaluation that included semi-structured clinical interviewing and multiple norm-referenced parent and teacher questionnaires. A detailed account of the comprehensive psychoeducational evaluation can be found in the larger study's preregistration: [LINK]. The final sample comprised 53 children, including 14 children with ADHD only; 31 children with ADHD and common comorbidities (18 anxiety, 1 depression, 6 oppositional-defiant disorder, 6 autism spectrum disorder); and 8 with common clinical diagnoses but not ADHD (5 anxiety, 2 depression, 1 autism spectrum disorder). Further, eight children with ADHD and two children without ADHD met diagnostic criteria for a learning disorder. Psychostimulants ($N_{prescribed}$ = 14; 26.4%) were withheld ≥24 hours for neurocognitive testing. Psychoeducational evaluations were provided to caregivers. Children were excluded from the larger study if they presented

with gross neurological, sensory, or motor impairment; non-stimulant medications that could not be withheld for testing; or history of seizure disorder, psychosis, or intellectual disability.

Procedure

Children completed the executive function tasks and EEG recording as part of a larger battery of neurocognitive testing that involved two sessions of approximately three hours each. All tasks were counterbalanced to minimize order effects. EEG data collection was conducted at the end of the second testing session following an extended break. Children received breaks after each task and preset longer breaks every 2–3 tasks to minimize fatigue. Task performance was monitored at all times by the examiner, who was stationed just outside of the testing room (out of the child's view) to provide a structured setting while minimizing performance improvements associated with examiner demand characteristics (Gomez & Sanson, 1994).

Measures

Executive Function Test Battery and Dimension Reduction—Descriptions, psychometric properties, and scoring information for the working memory, inhibitory control, and set shifting tasks are depicted in Table 1. Task impurity was controlled by computing Bartlett maximum likelihood component scores based on intercorrelations among all 7 executive function tests (Distefano et al., 2009), which parsed the 3 working memory, 2 inhibitory control, and 2 set-shifting tasks into 3 component scores (35.31% of variance explained; Supplementary Table 1). A three-component solution based on the larger sample (Soto et al., 2022) was specified *a priori* to derive separate estimates of working memory, inhibitory control, and set shifting based on theory and previous empirical work (e.g., Miyake et al. 2000). These principal components analysis-derived component scores provide estimates of reliable, construct-level variance attributable to domain-general working memory, inhibitory control, and set-shifting. This formative method for estimating executive functioning was selected because (a) such methods have been shown to provide higher construct stability relative to confirmatory/reflective approaches (Willoughby et al., 2016); and (b) estimating executive functioning at the construct-level rather than measure-level was expected to maximize associations with the study's outcomes via the removal of task-specific and error variance. These component scores were used for all analyses. Higher scores reflect better working memory and set shifting, but worse inhibitory control.

EEG Task—Children completed an age-appropriate go/no-go task that was administered while EEG was being recorded. Alien (go stimuli) and astronaut (no-go stimuli) images appeared on the screen one at a time for 200 ms (inter-trial interval of 1000–2000 ms). Children were instructed to "shoot" the aliens as soon as alien images appeared by clicking the mouse and "save" the astronauts by withholding from clicking when astronaut images appeared. After five practice trials, children completed three blocks of 100 trials, with a ratio of 75:25 go:no-go stimuli. Behavioral data were recorded, including task accuracy and reaction times for go and no-go trials.

EEG Data Acquisition and Processing—EEG data was recorded at 9 electrode sites (Fz, FCz, Cz, Pz, Oz, F3, F4, CMS, and DRL) using an elastic cap, along with

two electrodes on the left and right mastoids, using a BioSemi ActiveTwo system (BioSemi, Amsterdam, Netherlands) during the EEG go/no-go task described above. The electrooculogram (EOG) data were collected using four electrodes: two each 1 cm outside the outer edge of the right and left eyes to record horizontal eye movements and two each 1 cm above and below the right eye to capture vertical eye movements and blinks. The EEG signal was preamplified at the electrode and amplified with a gain of one. Online, all active electrodes were referenced to a common mode sense (CMS) active electrode producing a monopolar (non-differential) channel. EEG were recorded with a low-pass fifth order sinc filter with a half-power cutoff of 204.8 Hz and digitized at a 2-bit resolution with a sampling rate of 1024 Hz.

Offline, EEG data were referenced to the mean of the left and right mastoids and band-pass filtered between 0.1 and 30 Hz. Ocular artifacts (i.e., eye blinks and eye movement) were corrected following the steps outlined in Gratton et al. (1983). Specific intervals containing artifacts were eliminated from individual channels in each trial. Artifacts were detected and rejected using a semi-automatic approach if the interval included a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, or a maximum voltage difference of less than 0.50 μV within 100-ms intervals. All EEG processing was conducted by trained researchers masked to child diagnostic status and without access to the executive function/cognitive control data.

The EEG was segmented to −500 to 1000 ms before and after response onset for each trial and the response-locked ERPs (ERN and Pe) were averaged separately for correct and error trial types. Response-locked ERPs were baseline corrected using the pre-response interval between −500 and −300 ms. The ERN and Pe were extracted from electrodes where error-related brain activity was maximal (i.e., FCz, Pz) based on the current study data. The ERN was defined as the average voltage between 0 and 100 ms following the response for each child and calculated separately for error and correct trials. This method for extracting the ERN was selected over peak detection due to increased split-half reliability (Spearman-Brown adjusted) in the current sample (error trials: $r = .61$ vs. .49; correct trials: $r = .76$ vs. .66)[ii]. The Pe was defined as the mean voltage between 200 and 500 ms following the response and calculated separately for error and correct trials. The Pe's split-half reliability (Spearman Brown adjusted) in the current sample was similar to the ERN (error: $r = .67$; correct: $r = .68$).

Validity Check.: A minimum of six commission errors during the EEG go/no-go task was required for ERP extraction, as recommended (Meyer et al., 2014), and all participants in the current sample met this threshold ($M = 29.77$, $SD = 9.02$; range = 10–50). Inspection of task performance data showed a mean reaction time of 439.12 ms (SD=74.57) on correct go-trials. Within-subjects ANOVAs with trial type (error, correct) were conducted to confirm that error-related ERPs were elicited/captured by the task. For the ERN, neural activity was more negative during error trials ($M = −9.56$, $SD = 8.12$) compared to correct trials ($M = 1.21$, $SD = 5.67$; $\eta^2_p = 0.63$; $p < .001$) between 0 and 100 ms after response as expected (Figure 1, left). For the Pe, neural activity was more positive during error trials ($M = 14.42$,

[ii]Sensitivity analyses were conducted using Peak scores for the ERN and produced no deviations from the primary study analyses.

SD = 9.73) compared to correct trials (*M* = 3.62, *SD* = 5.69; η^2_p = 0.57; *p* < .001) between 200 and 500 ms after response as expected (Figure 1, right). In other words, error trials elicited significant and large/very large magnitude ERN and Pe.

ERN and Pe Dependent Variables.: Following Meyer et al. (2017), the ERN and Pe were computed as unstandardized residualized difference scores (average voltage for correct trials regressed out of average voltage for error trials) to reflect the difference wave between correct and erroneous responses[iii], separately for the ERN (R^2 = .09, *p* = .03) and Pe (R^2 = .11, *p* = .02). More negative values reflect a larger ERN and more positive values reflect a larger Pe.

ADHD Symptoms—The ADHD Rating Scale (ADHD-RS-5; DuPaul et al., 2016) was used to assess the frequency and severity of DSM ADHD symptoms in children and adolescents (18 items; 4-point Likert scale). The ADHD-RS-5 comprises two symptom subscales: Inattention (9 items) and Hyperactivity-Impulsivity (9 items). Psychometric support for the ADHD-RS-5 includes high internal consistency (α=0.94) and test-retest reliability (*r*=0.79 to 0.85; DuPaul et al., 2016). Teacher-reported ADHD symptoms were selected *a priori* given evidence that teacher-reported ADHD symptoms are more strongly associated with executive function task performance (Cho et al., 2011; Wang et al., 2015) and more predictive of ADHD diagnosis (Tripp et al., 2006). Higher raw scores reflect greater quantity/severity of ADHD symptoms.

Anxiety Symptoms—The Multidimensional Anxiety Scale for Children 2nd Edition Self-Report (MASC-2; March, 2012) was added for sensitivity analyses given evidence linking anxiety with increased error-related neural activity (Meyer, 2016). The MASC-2 Total Score measures the overall extent to which the child is experiencing anxiety symptoms. Child self-reported anxiety was used because self-reports appear to be more sensitive to early symptom emergence than parent reports (Cole et al., 2002); the presence/absence of anxiety diagnoses based on differential diagnoses considering all informant data was also probed as described below. The MASC-2 Total Score has demonstrated high internal consistency (α=.92) and 1- to 4-week test-retest reliability (*r*=.89; March, 2012). Higher scores reflect greater quantity/severity of anxiety symptoms.

Global Intellectual Functioning (IQ) and Socioeconomic Status (SES)—All children were administered the Short Form of the WISC-V (Wechsler, 2014). Hollingshead (1975) SES was estimated based on caregiver(s)' education and occupation.

Data Analysis Overview

We used a series of bias-corrected, bootstrapped conditional effects modeling using the R package medmod as implemented in jamovi v.1.6.23 (the jamovi project, 2021) separately for the ERN and Pe to examine the relations between each executive function (working memory, inhibitory control, and set shifting), the ERN/Pe, and ADHD symptoms. As such, these analyses were used to determine whether each executive function predicted

[iii]Sensitivity analyses were conducted using differences scores for the ERN and Pe (average voltage for error trials minus average voltage for correct trials) and produced no deviations from the primary study analyses.

ADHD symptoms and the extent to which these hypothesized relations were conveyed via associations between executive function and the ERN/Pe. Based on prior research indicating directional, if not causal, effects of executive functioning on ADHD behavioral symptom frequency/severity (e.g., Kofler et al., 2010; Rapport et al., 2009), the executive functions were modeled as predictors of ADHD symptoms. Each executive function was also modeled as a predictor of the ERN and Pe to determine the extent to which variability in the ERN and Pe reflected executive function abilities.

Finally, the ERN and Pe were modeled as predictors of ADHD symptoms as we were interested in the extent to which the hypothesized aspects of executive function shared between the ERN/Pe and executive function tests predict ADHD symptom expression. Our *a priori* plan called for modeling ADHD symptoms as a whole to conserve power; sensitivity analyses were added to probe the extent to which the findings were driven by one or both ADHD symptom domains (inattention, hyperactivity/ impulsivity). Age was controlled in all models. As described below, additional sensitivity analyses indicated that the primary model findings were generally robust to control for sex, SES, anxiety, separating overall ADHD symptoms into separate inattentive and hyperactive/impulsive symptom clusters, and modeling overall executive functioning/cognitive control instead of the 3 primary executive functions separately.

Notably, the current study is cross-sectional, inhibiting our ability to test competing models regarding the direction of effects (i.e., reversing arrows does not distinguish plausible models; Thoemmes, 2015). Effects are statistically significant if their 95% CI does not contain 0.0. Effect ratios (ER) for significant indirect effects indicate the proportion of the total effect (c pathway) that is conveyed via the indirect pathway (ab; i.e., ER=ab/c), and reflect the extent to which the ERN/Pe and the executive function performance metrics are capturing the same components of executive function/cognitive control that predict ADHD behavioral symptoms.

Results

Power Analysis

Power analysis for the conditional effects models was conducted using Monte Carlo simulation (Schoemann et al., 2017). Standardized path coefficients were imputed iteratively to delineate the proposed path model. Results indicated that for α=.05, our *N* of 53 is expected to detect significant effects at power=.80, assuming medium/large associations between executive functions and the ERN/Pe, medium associations between the ERN/Pe and ADHD symptoms, and conservatively assuming partial mediation (i.e., small/medium associations between executive functions and ADHD symptoms remaining after accounting for the ERN and Pe). Effects of this magnitude were considered reasonable given previous evidence of (a) medium to large relations between executive functions and the ERN and Pe (η^2=.19-.25, r=.27-.72; Coleman et al., 2018; Miller et al., 2012; Grammer et al., 2018; Mathewson et al., 2005; Larson & Clayson, 2011); (b) medium relations between the ERN and Pe and ADHD symptoms (d=0.21–0.68; Geburek et al., 2013; Kaiser et al., 2020); and (c) medium to large magnitude relations between executive functions and ADHD symptoms (d=0.60–1.40; Kasper et al., 2012; Kofler et al., 2019; Wright et al., 2014; Willcutt et al.,

2005). Thus, we concluded that the study was likely to be sufficiently powered to detect clinically meaningful effects.

Preliminary Analyses

Each of the independent and dependent variables were screened for univariate outliers, defined as values three standard deviations above or below the mean. Outliers were corrected to the next most extreme value in the sample (1.18% of data points affected). Missing data were imputed using expectation maximization based on all available data and were determined to be missing completely at random (Little's MCAR test: $\chi^2 = 34.08$, $p > .99$). This affected 2.25% of data points. Sample demographics are shown in Table 2. The zero-order correlation matrix is shown in Table 3.

Conditional Effects Models

Bias-corrected conditional effects models with 5,000 bootstrap resamples were conducted, separately for the ERN (Figure 2) and Pe (Figure 3); results are organized by pathway. Results of both models indicated that better working memory (path c; $\beta = -.34$) and inhibitory control (path c; $\beta = .31$) each uniquely predicted lower levels of ADHD symptoms. In contrast, set shifting did not predict ADHD symptoms (path c; $\beta = .02$, *ns*).

In the ERN model, working memory (path a; $\beta = -.04$, *ns*), inhibitory control (path a; $\beta = -.14$, *ns*), and set shifting (path a; $\beta = -.05$, *ns*) all failed to predict the ERN. Additionally, the ERN did not predict ADHD symptoms (path b; $\beta = .01$, *ns*). Further, there were no significant indirect effects of the three executive functions on ADHD symptoms through the ERN pathway (path ab; all 95% CIs include 0.0). Overall, these findings indicate that the ERN is not capturing meaningful variance in children's executive functioning/cognitive control either overall or with regard to the aspects of working memory and inhibitory control that predict ADHD behavioral symptom expression.

In contrast, in the Pe model, better-developed inhibitory control was associated with a greater Pe (path a; $\beta = -.35$), whereas working memory (path a; $\beta = .22$, *ns*) and set shifting (path a; $\beta = -.10$, *ns*) failed to predict the Pe. Similar to the ERN model, the Pe was not significantly related to ADHD symptoms (path b; $\beta = .10$, *ns*) and there was no evidence to suggest conditional effects of the executive functions on ADHD symptoms through the Pe (path ab; all 95% CIs include 0.0). In other words, the Pe is capturing meaningful variance associated with inhibitory control, but similar to the ERN is not capturing the aspects of working memory and inhibitory control that are predictive of ADHD behavioral symptom expression.

Taken together, these results indicate that better working memory and inhibitory control, but not set shifting, predict fewer ADHD symptoms, whereas variability in the ERN and Pe is not capturing the aspects of working memory and inhibitory control that underlie these relations. Interestingly, better-developed inhibitory control predicted an increased Pe as hypothesized, whereas none of the three executive functions predicted the ERN. In contrast, neither the ERN nor Pe were predictive of ADHD symptoms.

Sensitivity Analyses

Sensitivity analyses were conducted to probe the extent to which the pattern of results reported above was impacted by our *a priori* decisions to (a) model ADHD symptoms as a whole; (b) exclude sex and SES as covariates to conserve power; (c) model the three EFs separately rather than as a single indicator reflecting overall executive functioning/ cognitive control; and (d) include children with and without anxiety disorders. First, the overall ADHD symptoms variable was replaced with separate indicators for inattention and hyperactivity/ impulsivity symptoms. Better-developed working memory ($\beta = -.34$) and inhibitory control ($\beta = .28$) continued to predicted inattention symptoms, but were not significantly associated with hyperactivity/impulsivity (all 95% CIs included 0.0). Consistent with the primary models, the ERN and Pe were not significant predictors of either ADHD symptom cluster (all 95% CIs included 0.0; Supplementary Tables 2–5).

Next, we repeated the primary models again controlling for sex and SES. Results were highly consistent with those reported above, with inhibitory control significantly predicting the Pe ($\beta = -.36$); working memory ($\beta = -.38$) and inhibitory control ($\beta = .32$) predicting ADHD symptoms; and no other significant relations detected (95%CIs include 0.0; Supplementary Tables 6 & 7). Third, we probed the extent to which the ERN and/or Pe might show stronger associations with executive functioning/cognitive control in general rather than the specific executive functions. This involved creating a single executive function component score based on the same 7 executive function tests described above (21.29% of variance explained; Supplementary Table 1). We then repeated the primary models above with the overall executive functioning/cognitive control estimate instead of the three separate executive function predictors. Results were generally consistent with the primary model in that better executive functioning predicted fewer ADHD symptoms ($\beta = -.40$), whereas the ERN and Pe did not (both 95%CIs include 0.0). In contrast, overall executive functioning/cognitive control did not significantly predict the ERN ($\beta = -.04$, *ns*) or the Pe ($\beta = .24$, *ns*). There were also no indirect effects of executive functioning/ cognitive control on ADHD symptoms via the ERN or Pe pathways (95% CIs included 0.0; Supplementary Tables 8 & 9).

Finally, given converging evidence linking anxiety disorders with increased error-related neural activity (Meyer, 2016), we explored the extent to which the results may have been impacted by our *a priori* decision to include children with anxiety disorders (n=23 of 53). This involved re-running the primary models twice, once with anxiety disorder status (No/ Yes)[iv] and once with child-reported anxiety symptoms (MASC-2) as additional covariates. Results were generally consistent with the primary models, with better inhibitory control predicting a larger Pe (β=−.35 for both models) and no other significant relations between the ERPs and executive functions or ADHD symptoms, with one minor exception: the relations between working memory (β=−.34 in the primary model vs. −.27) and inhibitory control (β=.31 vs. .21) no longer significantly predicted ADHD symptoms as the relations were slightly attenuated when controlling for anxiety disorder status (95%CIs include 0.0).

[iv]Due to prior research demonstrating that generalized anxiety, social anxiety, and obsessive-compulsive disorders are most reliably associated with error-related neural activity (Meyer et al. 2016), the models were also run only covarying the presence of these three disorders. The results were unchanged from those reported when anxiety disorder status was included as a covariate.

However, working memory (β=−.33) and inhibitory control (β=−.28) continued to predict ADHD symptoms when covarying self-reported anxiety symptoms.

Discussion

Although the ERN and Pe are frequently interpreted as indices of cognitive control (Gehring et al., 2012), few studies have tested this interpretation empirically outside of data from the same inhibitory control tasks used to derive the ERPs (Meyer & Hajcak, 2019) or examined whether test-based and EEG-based estimates predict the same variance in behavioral outcomes of executive function/cognitive control difficulties (i.e., ADHD symptoms). The current study addressed these limitations in a clinically evaluated and carefully phenotyped school-aged sample by examining the ERN/Pe's shared and unique relations with ADHD symptoms and a well-validated battery of all three core executive functions. Contrary to our hypotheses, the ERN was not associated with general or specific executive functioning/cognitive control abilities, whereas a larger Pe was only related to better inhibitory control. These findings add to a largely mixed literature on error-related neural activity and cognitive control/executive function. Our results are inconsistent with studies finding relations between a larger ERN and better working memory and inhibitory control (Coleman et al., 2018; Miller et al., 2012; Overbye et al., 2019), as well as better overall cognitive control/executive function (Larson & Clayson, 2011; Meyer & Klein, 2018). In contrast, the significant relation between a larger Pe and better inhibitory control is consistent with most prior work finding that the Pe is related to performance metrics from inhibition tasks in adults (Chang et al., 2014; Hajcak et al., 2003; Mathewson et al., 2005) and children (Kim et al., 2016; Overbye et al., 2019; c.f. Grammer et al., 2018). Interestingly, the Pe was not related to cognitive control/executive function overall, and did not capture meaningful variance in inhibitory control that was associated with ADHD behavioral symptoms, potentially suggesting the need for increased specificity when interpreting this metric as discussed below.

The characteristics of the current sample may be an important factor contributing to our findings that the ERN was not associated with executive function/cognitive control, while the Pe was related to inhibitory control only. Prior work suggesting that an increased ERN is related to better working memory ability was conducted with undergraduate students (Coleman et al., 2018; Miller et al., 2012), while the current study examined a clinically evaluated, school-aged sample. Studies examining the Pe and inhibitory control have also largely focused on healthy adult populations, with the few pediatric studies focusing on children younger than our age group (Grammer et al., 2018; Kim et al., 2016) or a wider age range spanning school-age and adolescence (Overbye et al., 2019). This distinction is important given evidence for differences in the developmental trajectories of the ERN, Pe, and specific executive functions. For example, the ERN continues to increase into early adulthood and the Pe matures earlier in development (Downes et al., 2017). Similarly, working memory and inhibitory control reflect distinct abilities around the age of school entry, while set shifting only emerges as a distinct executive function in older school-aged children (Karr et al., 2018; Lerner & Lonigan, 2014). Thus, it is possible that the ERN and Pe are reflective of different cognitive functions in different age groups.

Alternatively, the linkage between inhibitory control and the Pe but not ERN may be attributable to the increased use of top-down/effortful control processes for the former relative to the latter. Inhibitory control abilities are characterized by the active and goal-directed withholding or stopping of a response that relies on deliberate and conscious control processes (Alderson et al., 2007; Mirabella, 2021). However, prior work suggests that an ERN is detectable regardless of whether an individual recognizes that they made an error, whereas the Pe is most pronounced following trials where an individual has conscious awareness of the error (Endrass et al., 2007; Nieuwenhuis et al., 2001; O'Connell et al., 2007). Thus, it seems reasonable to hypothesize that the Pe may reflect effortful inhibitory control, whereas the ERN may be a more automatic monitoring process. In other words, the Pe may represent top-down regulation of ongoing behavioral processes, while the ERN may be reflective of more automatic bottom-up processes that would be less affected by effortful cognitive control/executive function processes (Wells et al., 2019). Indeed, Boen and colleagues (2022) suggested such a distinction as a potential mechanism underlying the differing developmental trajectories of the ERN and Pe.

Interestingly, we found that the aspects of inhibitory control captured by the Pe did not overlap with those aspects that are predictive of ADHD behavioral symptoms, despite better inhibitory control abilities predicting fewer ADHD symptoms. These findings contribute to a fairly large body of mixed evidence in which about half of the studies find that a greater ERN and/or Pe is related to fewer ADHD symptoms and the other half find no such relations (Herrmann et al., 2009; Marquardt et al., 2018; Rommel et al., 2019; Wiersema et al., 2009). Similar results are seen in ADHD/Control between-group studies in the ERN (for review see Meyer & Hajcak, 2019), while a reduced Pe in ADHD groups appears to be a more consistent finding (Kaiser et al., 2020; McLoughlin et al., 2022). Given this pattern, it seems likely that these mixed results, at least for the ERN, reflect study-level sampling error around a true effect that is small or null. Alternatively, we speculated that the mixed findings may be related to most studies examining ADHD symptoms overall as opposed to probing across the ADHD inattentive and hyperactivity/impulsivity domains. Our sensitivity analyses suggest that this explanation is unlikely given that the ERN and Pe were also unrelated to both ADHD symptom domains, but future studies with larger samples would be helpful to confirm/refute this interpretation.

Limitations and Future Directions

The current study's sample size was relatively small due to discontinued recruitment as a result of the COVID-19 pandemic. Although our sample size was similar to most prior work on this topic (e.g., Grammer et al., 2018; Marquardt et al., 2018, Miller et al, 2012; cf. Meyer & Klein, 2018; Rommel et al., 2019) and our power analyses indicated that the sample size was sufficient to detect meaningful effects, it is possible that the sample was not large enough to detect small magnitude relations with the ERN and Pe in the current study. However, we found significant associations between better inhibitory control and an increased Pe, which is consistent with prior work (Kaiser et al., 2020; McLoughlin et al., 2022), and robust relations between working memory/inhibitory control and ADHD symptoms. To the extent that the true relation between the ERN and ADHD symptoms is close to zero as suggested by prior literature (Kaiser et al., 2020; Meyer & Hajcak, 2019),

it seems likely that much larger samples would be needed to reliably detect this effect if it is present. Interestingly, while go/no-go tasks seem to elicit greater group differences in the Pe than flanker tasks (Geburek et al., 2013; Lutz et al., 2021), the opposite may be true of the ERN (McLoughlin et al., 2022). Thus, it is possible that our use of a go/no-go task during EEG recording may have optimized the potential to observe relations between the Pe, but not the ERN, and ADHD symptoms. In contrast, the lack of ERP relations with overall and specific executive functions in 7 of 8 cases (with the only significant relation being the Pe and inhibitory control) – as well as the non-significant associations between the ERPs and commission errors on the EEG go/no-go task – suggests challenges for attributing the same construct to these performance-based and electrophysiological measures. That is, if the current findings are replicated in larger and more diverse samples, a parsimonious conclusion would be that both the performance-based tests and error-related ERP metrics are reliable assessment methods, but are primarily measuring different constructs – or at best that terms like 'cognitive control/executive function' are too broad to accurately capture the cognitive processes reflected by the ERPs. This latter hypothesis is consistent with our finding that the ERN/Pe were not related to ADHD symptoms, and the Pe is not capturing the aspects of inhibitory control important for understanding children's inattentive or hyperactive/impulsive behavior.

Importantly, the current study recruited a clinically heterogeneous sample. Although more representative of pediatric populations seeking assessment for behavior and emotional concerns that have been linked with executive function/cognitive control (e.g., Kofler et al., 2019; Snyder, 2013), it is possible that the diverse clinical symptom presentation complicated our ability to detect relations between the ERN/Pe and executive function/cognitive control. In particular, an increased ERN is frequently reported in children with anxiety disorders, and has been proposed to be a biomarker of anxiety (Meyer, 2016). In contrast, the relation between the Pe and anxiety is more variable (Hajcak et al., 2004; Ladouceur et al., 2006; McDermott et al., 2009). Thus, we conducted sensitivity analyses to examine the extent that our results were confounded by the inclusion of children with anxiety disorders in the sample. We found that the ERP primary findings held with anxiety disorder status and child self-report of anxiety symptoms covaried, suggesting increased confidence in the findings. A more nuanced examination of this topic in future research is warranted given (a) the empirical and theorized relations between anxiety and both error-related neural activity and executive functions (Derakshan et al., 2009; Eysenck et al., 2007; Meyer, 2016; Wong et al., 2013); (b) the multicomponent nature of inhibitory control (e.g., Alderson et al., 2008); and (c) the current finding that the Pe did not capture the variance in inhibitory control that is important for predicting ADHD symptoms. A speculative hypothesis is that the Pe may instead be capturing aspects of inhibitory control that are linked with anxiety symptoms and/or other behavioral expressions rather than ADHD symptoms (e.g., Lutz et al., 2021), particularly given evidence that anxiety may serve as a protective factor for children with ADHD in at least some domains (Chan et al., 2022; Klymkiw et al., 2020; Maric et al., 2018).

Conclusion

Taken together, the current study found that the Pe appears to be capturing meaningful variance associated with inhibitory control abilities, whereas neither the Pe nor the ERN were associated with overall cognitive control/executive functioning in school-aged children. These findings call into question the use of ERN and/or Pe as broad indices of cognitive control and, if replicated, suggest the need for more specificity in the construct reification of these reliable indices. Based on results from the current study, we hypothesize that the Pe may be reflective of the top-down, effortful components of inhibitory control (Boen et al., 2022.; Nieuwenhuis et al., 2003; O'Connell et al., 2007) that are distinct from the aspects of inhibitory control implicated in the behavioral symptomatology of ADHD. In contrast, the ERN appears to reflect more passive monitoring processes that do not overlap meaningfully with the active, top-down processes that characterize cognitive control/executive function (Boen et al., 2022; Nieuwenhuis et al., 2001; O'Connell et al., 2007). Further research is needed to replicate these findings and identify additional cognitive and non-cognitive processes that are reflected in the ERN and Pe, toward a more refined understanding of the processes that these reliable ERP metrics are, and are not, capturing, and their utility in study of neurocognitive processes in ADHD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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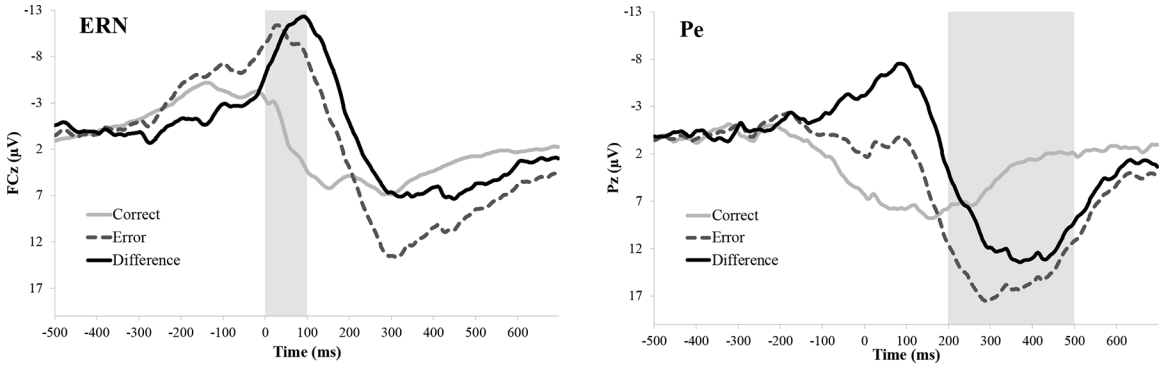

Figure 1.

ERN and Pe Grand Average Waveforms

Note. Grand average correct, error, and difference waveforms at FCz and Pz. The error-related negativity (ERN) was extracted from FCz (0 to 100 ms) and the error positivity (Pe) was extracted from Pz (200 to 500 ms). The difference wave represents activity during error trials minus activity during correct trials.

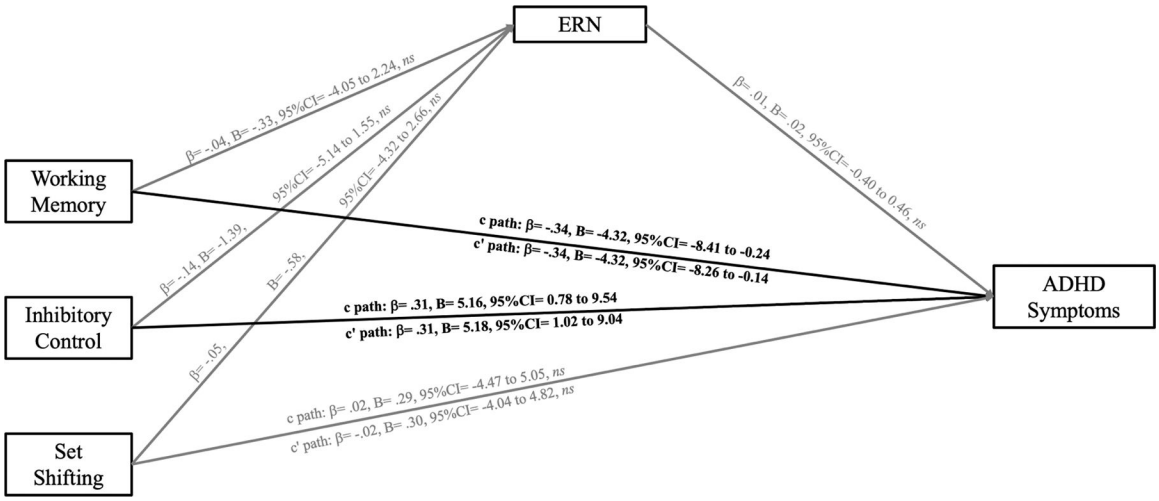

Figure 2.

Path diagram depicting primary ERN model results. Significant pathways are indicated by 95% CIs that exclude zero, and are shown in black/bold font. Nonsignificant pathways are shown in grey font.

Note. ERN: error-related negativity (residualized score); ADHD Symptoms: teacher-rated total ADHD symptoms. Age was included as a covariate but is not depicted for clarity.

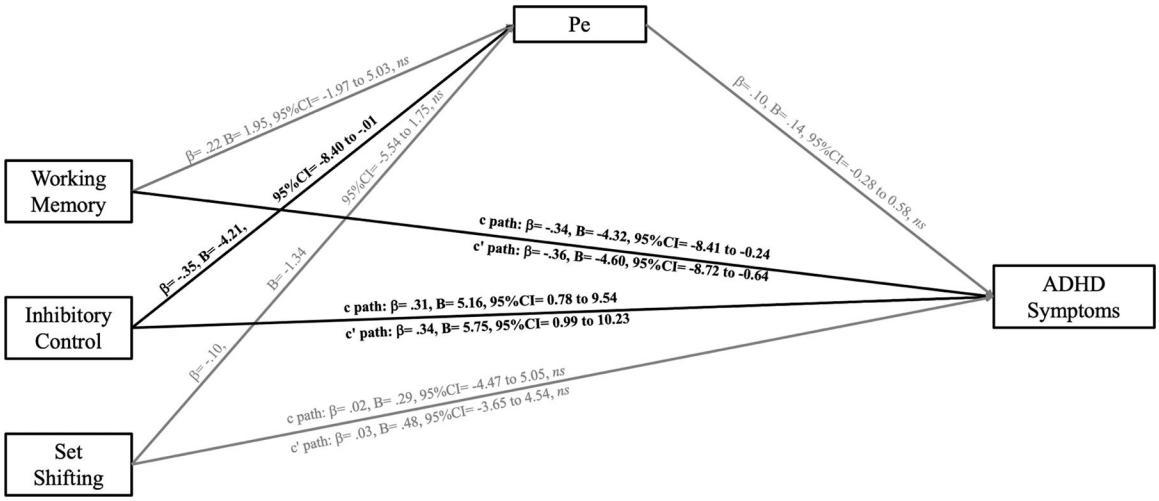

Figure 3.

Path diagram depicting primary Pe model results. Significant pathways are indicated by 95% CIs that exclude zero, and are shown in black/bold font. Nonsignificant pathways are shown in grey font.

Note. Pe: error positivity (residualized score); ADHD Symptoms: teacher-rated total ADHD symptoms. Age was included as a covariate but is not depicted for clarity.

Table 1.

Executive function test psychometric properties and scoring

Executive Function Test	Description/Key Task Parameters	Psychometric Support	Indicators and Scoring	Trials
Inhibitory Control				
Stop-Signal	-Go-stimuli were uppercase letters X and O. -Stop-stimulus was a 1000 Hz auditory tone presented randomly on 25% of trials. -Stop-signal delay (SSD; the latency between go- and stop-stimuli presentation) was initially set to 250 ms and dynamically adjusted ±50 ms contingent on the child's performance.	Internal consistency: α = .83–.89 3-week test-retest: r=.72 (Soreni et al., 2009) Internal consistency current study: α = .67	Speed of the inhibitory stop process (iSSRT) estimated separately for each of the task blocks (Verbuggen et al., 2013 integration method) Higher scores indicate worse inhibition	4 blocks 32 trials/block
Go/No-Go	-Children presented with a randomized series of vertical (go stimuli) and horizontal (no-go stimuli) rectangles. - A ratio of 80:20 go:no-go stimuli was selected to maximize prepotency (Kane & Engle, 2003; Unsworth & Engle, 2007).	Internal consistency: α=.95 (Kofler et al., 2019) Internal consistency current sample: α=.94	Commission errors (i.e., incorrectly responding to no-go trials) Lower scores indicate better inhibition	4 blocks 25 trials/block
Set Shifting				
Global-Local	-Navon (1977) figures were used, which consist of a larger, "global" shape made up of smaller, "local" shapes. Figures were presented one at a time in one of four quadrants. -To minimize memory demands, on-screen cues ("big shape," "small shapes") were positioned next to each quadrant. -Children were required to shift their response between global and local features depending on which quadrant the figures appeared (top quadrants: global; bottom quadrants: local).	Internal consistency: α=.86–.90) (Kofler et al., 2019) Increases in shift costs and accuracy costs relative to control conditions: ω^2 = .12, .14 (Irwin et al., 2019) Internal consistency current sample: α=.82–.87 for shift and no-shift trials, respectively	Speed shift costs (Speed shift cost = $RT_{shift} - RT_{non\text{-}shift}$ for correct trials) (Miyake et al., 2000; Irwin et al., 2019) Lower speed shift costs indicate better set-shifting	4 blocks 30 trials/block
Number-Color	-Children clicked either the larger or smaller single-digit number depending on the font color, which was presented semi-randomly to require shifting every other trial. -To minimize memory demands, on-screen instructions (blue bigger, yellow smaller) remained visible throughout the task.	Internal consistency: α = .87–.95 (Kofler et al., 2019) Internal consistency current study: α=.92–.95 for shift and no-shift trials, respectively	Speed shift costs (Speed shift cost = $RT_{shift} - RT_{non\text{-}shift}$ for correct trials) (Miyake et al., 2000; Irwin et al., 2019) Lower speed shift costs reflect better set-shifting	4 blocks 30 trials/block
Working Memory				
Rapport Phonological and Visuospatial Working Memory Tasks	-*Phonological:* A series of jumbled numbers and a capital letter presented. Children verbally recalled numbers in order from smallest to largest, and to say the letter last (e.g., 4H62 is correctly recalled as 246H). - *Visuospatial:* A series of 2.5 cm diameter dots (3, 4, 5, or 6) were presented sequentially in one of the nine squares arranged in three offset vertical columns. All but one dot presented within the squares was black —the exception being a red dot. Children reordered the dot locations (black dots in serial order, red dot last).	Internal consistency: α = .82–.97 (Kofler et al., 2019) 1- to 3-week test-retest reliability: r=.76–.90 (Sarver et al., 2015) Convergent validity: r = .61–.69 (Wells et al., 2018) Internal consistency current sample: α = .84–.85	Partial-credit unit scoring (i.e., stimuli correct per trial) (Conway et al., 2005) computed separately for the phonological and visuospatial working memory tests Higher scores reflect better working memory	24 total trials per task 6 trials/set size (3–6 stimuli/ trial)

Executive Function Test	Description/Key Task Parameters	Psychometric Support	Indicators and Scoring	Trials
Letter Updating	-Letters presented on the screen one at a time, and children kept track of the last three letters presented. -Children were instructed to rehearse out loud the last three letters by mentally adding the most recent letter and dropping the fourth letter back (Miyake et al. 2000).	Internal consistency: α=.75 (Kofler et al., 2019) Large magnitude ADHD/Non-ADHD between group differences (Fosco et al., 2020; Kofler et al., 2019). Internal consistency current sample: α=.82	Mean stimuli correct per trial (correct serial order) Higher scores reflect better working memory	4 blocks 3 trials/block (4–8 stimuli/ trial)

Table 2.

Sample and demographic characteristics

Variable	*M*	*SD*	*Min*	*Max*
N (Boys/Girls)	53 (37/16)			
Age	10.36	1.42	8.22	12.84
SES	49.28	9.46	31	66
FSIQ	101.70	14.37	73	128
Ethnicity (B, BR, H, W)	(6, 4, 2, 41)			
Teacher Total ADHD Symptoms (raw score)	28.42	12.96	2	51
Executive Functioning (z-scores)				
Working Memory Component Score	−.12	1.03	−3.27	1.65
Inhibitory Control Component Score	.05	.77	−1.37	3.14
Set Shifting Component Score	−.15	.70	−1.45	2.03
Executive Function Component Score	−.14	1.01	−3.27	1.74
EEG Error-Related ERPs				
ERN (residualized score)	0.00	7.73	−13.32	16.08
ERN (error trials) *	−9.56	8.12	−24.97	3.48
ERN (correct trials) *	1.21	5.67	−8.36	17.49
Pe (residualized score)	0.11	9.23	−17.56	27.07
Pe (error trials) *	14.42	9.73	−4.58	41.10
Pe (correct trials) *	3.62	5.69	−11.85	16.74

Note: B = Black and/or African American; BR = Biracial; H = Hispanic and/or Latino; SES = Hollingshead SES total score; FSIQ = Short-Form IQ; W = White/Non-Hispanic

* amplitudes are in microvolts (μV)

Table 3.

Zero-order and partial correlations

	ERN	Pe	WM	IC	SS	General EF	ADHD Symptoms	Sex	Age	SES	SFIQ	EEG GNG Commission Errors
ERN	--	*.36***	*−.08*	*−.14*	*−.06*	*−.03*	*−.01*	*.06*	--	*−.08*	*.07*	*−.22*
Pe	.36**	--	*.08*	*−.30**	*−.05*	*.18*	*−.02*	*−.17*	--	*−.01*	*.20*	*−.11*
WM	−.04	.06	--	*.20*	*.31**	*.93***	*−.22*	*.00*	--	*.16*	*.44***	*−.19*
IC	−.14	−.28	−.06	--	*.00*	*−.17*	*.25*	*−.01*	--	*−.07*	*−.11*	*.25*
SS	−.07	−.05	.13	.07	--	*.35**	*−.07*	*.19*	--	*.01*	*.39***	*−.03*
General EF	.00	.13	.96**	−.35*	.13	--	*−.31**	*.02*	--	*.18*	*.50***	*−.29**
ADHD Symptoms	−.02	−.02	−.36**	.33*	−.01	−.43**	--	*−.22*	--	*−.05*	*−.11*	*.26*
Sex	.06	−.17	−.01	.00	.19	.00	.20	--	--	*.20*	*−.07*	*.07*
Age	.04	−.01	.60**	−.35**	−.18	.66**	−.32*	−.02	--	--	--	--
SES	−.08	−.01	.09	−.04	.02	.09	−.03	.20	−.06	--	*.37***	*.21*
SFIQ	−.07	.20	.31*	−.08	.40**	.33*	−.08	−.07	−.07	.37**	--	*−.07*
EEG GNG Commission Errors	−.22	−.11	−.11	.21	−.04	−.17	.22	.07	.08	.21	−.07	--

*Note. ERN: error-related negativity (residualized score), General EF: general executive function component score, GNG: Go/no-go, IC: inhibitory control factor score, Pe: error positivity (residualized score), SES: Hollingshead socioeconomic status Total Score, SFIQ: WISC-V short-form IQ score, SS: set shifting factor score, ADHD Symptoms: teacher-rated total ADHD symptoms, WM: working memory factor score. = p <.05

** = p<.01.

Zero-order correlations are shown in the bottom triangle; partial correlations controlling for age are shown in italics in the upper triangle.