



The persistent influence of pediatric concussion on attention and cognitive control during flanker performance

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ABSTRACT

This study investigated the influence of concussion history on children's neurocognitive processing. Thirty-two children ages 8–10 years (16 with a concussion history, 16 controls) completed compatible and incompatible conditions of a flanker task while behavioral and neuroelectric data were collected. Relative to controls, children with a concussion history exhibited alterations in the sequential congruency effect, committed more omission errors, and exhibited decreased post-error accuracy. Children with a concussion history exhibited longer N2 latency across task conditions, increased N2 amplitude during the incompatible condition of the task, and decreased P3b amplitude across task conditions. Children with a history of concussion also exhibited decreased ERN and Pe amplitudes, with group difference increasing for the incompatible condition of the task. The current results indicate that pediatric concussion may lead to subtle, but pervasive deficits in attention and cognitive control. These results serve to inform a poorly understood but significant public health concern.

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1. Introduction

Traumatic brain injury is a leading cause of death and disability in developing populations (Langlois, Kegler, & Butler, 2003), with more than a million cases being treated in the United States annually (Yeates et al., 1999). Although the majority of efforts are dedicated towards the diagnosis, tracking, and remediation of moderate-to-severe brain injuries (McKinlay, Grace, Horwood, Fergusson, & MacFarlane, 2010), mild or “concussive” injuries account for approximately 85% of all pediatric brain injuries (Langlois et al., 2003). Furthermore, a growing body of literature indicates that irrespective of severity, the immature brain is uniquely vulnerable to injury, not more resilient (Daneshvar et al., 2011; Giza & Prins, 2006; Prins & Giza, 2012). Specifically, the protracted development of frontal brain areas in terms of myelination, connectivity, and density appears to lead to extensive white and grey matter abnormalities following pediatric brain injury (Giza & Prins, 2006; Prins & Giza, 2012). Accordingly, these injuries warrant

increased attention from clinicians and researchers, as concussive injury may disrupt the developmental trajectory of frontally mediated cognitive functions (Baillargeon, Lassonde, Leclerc, & Ellemberg, 2012; Catale, Marique, Closset, & Meulemans, 2009; Moore et al., submitted for publication).

Indeed, although acute evaluations typically reveal global alterations in cognitive function (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004), long-term evaluations appear consummate in revealing selective alterations in frontally-mediated cognitive functions, such as attention and cognitive control (Baillargeon et al., 2012; Catale et al., 2009; Moore et al., submitted for publication). Cognitive control refers to higher-order cognitive functions, which serve to evaluate, regulate, and optimize goal-directed behaviors through the selection, scheduling, coordination, and maintenance of processes underlying aspects of perception, memory, and action (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Norman & Shallice, 1986). Core functions constituting cognitive control are working memory, inhibition and cognitive flexibility (Diamond, 2013). These functions are essential for learning, academic achievement and overall well-being (Diamond, 2013; Holmes, Gathercole, & Dunning, 2009), and deficits in these functions are believed to underlie many individual differences in cognition across the lifespan (Luna, 2009). Therefore, the long-term assessment of the

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functions following pediatric concussion is of particular importance.

One task, which examines multiple aspects of attention and cognitive control, is the Eriksen flanker task (Eriksen & Eriksen, 1974). Flanker tasks require individuals to ignore task-irrelevant information in order to correctly respond to a centrally presented target stimulus amid either congruent or incongruent flanking stimuli. While conceptually simplistic, this task has demonstrated high sensitivity for detecting deficits from years (Moore, Hillman, & Broglio, 2014; Pontifex, O'Connor, Broglio, & Hillman, 2009) to decades (de Beaumont, Brisson, Lassonde, & Jolicoeur, 2007) following injury in adults. This sensitivity is due to the rich variety of cognitive processes/effects, which can be evaluated during flanker performance.

For example, the congruency effect refers to the finding of decreased performance for incongruent relative to congruent trials (Eriksen & Eriksen, 1974; Spencer & Coles, 1999). This effect serves as a metric of interference control, as incongruent relative to the congruent trials require greater amounts of interference control in order to inhibit flanking stimuli, as both the correct (elicited by the target) and incorrect response (elicited by the flanking stimuli) are concurrently activated during stimulus evaluation (Spencer & Coles, 1999). An extension of this phenomenon, the sequential congruency or Gratton effect refers to the finding that lower interference occurs following an incongruent (i) trial relative to a congruent (c) trial (Botvinick et al., 2001; Gratton, Coles, & Donchin, 1992). This sequential modulation in performance is reflective of adjustments in cognitive control immediately following an incongruent trial, whereby participants strategically narrow attention to the central target, thus minimizing the interference of misleading flankers (Botvinick et al., 2001; Gratton et al., 1992). Lastly, the manipulation of stimulus–response compatibility, through which the response mappings to stimuli are reversed (e.g., a target stimulus which previously required a left thumb response, later requires a right thumb response in subsequent conditions), produces a compatibility effect (Friedman, Nessler, Cycowicz, & Horton, 2009). This manipulation allows for the examination of attention and inhibitory processes across multiple gradations, as individuals must inhibit the pre-potent response mapping in addition to regulating interference from flanking stimuli (Friedman et al., 2009; Pontifex et al., 2011; Pontifex, Scudder, Drollette, & Hillman, 2012). This manipulation also produces a sufficient number of errors to examine response evaluation and error correction (i.e., post-error accuracy, and RT).

Thus, flanker tasks afford a comprehensive assessment of attention and cognitive processes during conditions that elicit stimulus–response conflict. However, despite this dynamic utility and known sensitivity to concussive injuries, no one has utilized flanker paradigms in pediatric concussion research. Accordingly, the first aim of the current study was to examine the long-term influence of pediatric concussion on attention and cognitive control by employing a modified flanker task.

In addition to examining behavior, the current study also sought to examine the long-term outcomes of pediatric concussion on the neurophysiological level. Currently, little is known regarding the pathophysiological sequelae of pediatric concussion, or how this pathophysiology relates to functional behavioral outcomes. Thus, examining neurophysiological function is essential to gain a more comprehensive understanding of pediatric concussion outcomes (Baillargeon et al., 2012; Mayer et al., 2012). Indeed, examinations of neurophysiological function have been essential to understanding concussion outcomes in adults (Broglio, Moore, & Hillman, 2011; Slobounov, Johnson, & Zhang, 2012). Few pediatric studies, however, have examined neurophysiological function following concussion, and only two studies examined long-term outcomes (Baillargeon et al., 2012; Moore et al., submitted for publication).

Both studies employed electroencephalography (EEG) to evaluate event-related brain potentials (ERPs) during experimental task performance. Specifically, Baillargeon et al. (2012) recorded ERPs during a visual discrimination task (i.e., an oddball task) to evaluate children, adolescents, and adults 6 months after injury. The authors evaluated the P3b ERP component, which is believed to be a neural index of attentional resource allocation during stimulus engagement (Donchin & Coles, 1988; Polich, 2007). Irrespective of age, participants with a concussion history exhibited smaller P3b amplitude relative to control participants, indicating that a single concussion can lead to persistent alterations in the neurophysiology underlying attentional resource allocation in the service of working memory. Moore et al. (submitted for publication) recorded ERPs during switch and go–nogo tasks to evaluate the neuroelectric underpinnings of attention, and cognitive control in children an average of 2 years following injury. The authors observed subtle behavioral deficits in impulsivity (behavioral inhibition), working memory, and cognitive flexibility, with increasing cognitive load. Furthermore, these deficits were accompanied by alterations in the ERP indices of visual attention (N1; Hillyard & Anllo-Vento, 1998), stimulus–response conflict/inhibition (N2; Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003), and attention (P3b; Polich, 2007), suggesting that alterations in neurocognitive processing may become more pervasive with increasing cognitive load.

Together, these studies provide valuable information regarding the long-term outcomes of pediatric concussion and emphasize the utility of experimental and functional neurophysiological measures in pediatric concussion research. Although these results are valuable, much is still unknown regarding the nature and duration of neurophysiological outcomes of pediatric concussion. Thus, the second aim of the current study was to assess stimulus-locked and response-locked ERPs to examine the neurophysiological underpinnings of stimulus engagement and response evaluation in children with and without a concussion history.

In sum, the available evidence suggests that pediatric concussion may lead to persistent alterations in neurocognitive processing, but the answers to clinically relevant questions remain unknown. Specifically, how long do concussion-related deficits persist? What degree of specificity characterizes concussion-related deficits, and do deficits change with age at injury? Accordingly, the current study sought to answer these questions by comparing behavior and brain function in children with a concussion history relative to demographically matched controls. We predicted that children with a concussion history would exhibit both behavioral deficits and neuroelectric differences relative to matched control children during trials/trial sequences (incongruent/il), and task conditions (incompatible) requiring the modulation of attention and cognitive control. In accordance with previous research (Hessen, Nestvold, & Anderson, 2007; Moore et al., submitted for publication), we predicted that children injured earlier in life would exhibit the poorest outcomes.

2. Experimental procedures

2.1. Participants

Participants were 32 (16 concussion, 16 control) 8–10 years old residing in East-Central Illinois. Participants were recruited through university recruitment services and Central Illinois youth athletic associations (YMCA, hockey, football, soccer). Guardians confirmed that all participants with a concussion history had experienced a single medically diagnosed injury, while participating in sports and recreation 6+ months prior to testing. No participant incurred a complicated injury requiring surgical intervention or hospital admittance. All participants were physically active on a

Table 1
Participant demographic information.

Measure	Concussion	Control	Statistic
Age	9.2 (± 0.6)	9.0 (± 0.7)	
Age at injury	7.1 (± 2.1)		
Years since injury	2.1 (± 1.9)		
Loss of consciousness	8		
Gender	11M/5F	11M/5F	
Pubertal timing	1.28 (± 0.4)	1.38 (± 0.3)	$p = 0.47$
K-BIT (IQ)	115 (± 14.9)	117.4 (± 14.5)	$p = 0.64$
SES	2 (± 0.8)	2 (± 0.9)	$p = 0.99$
Fitness% (Vo2 rel.)	40.5 (± 6.5)	42.8 (± 9.6)	$p = 0.48$
ADHD-concentration	52.7 (± 33.2)	50.2 (± 37.5)	$p = 0.84$
ADHD-impulsivity	44.9 (± 31.9)	42.2 (± 31.6)	$p = 0.81$
ADHD-hyperactivity	55.4 (± 33.2)	55.4 (± 28.0)	$p = 0.79$
SSAP	2.2 (± 1.1)	2.2 (± 1.1)	$p = 0.99$
SSAS	1.5 (± 0.9)	1.5 (± 0.9)	$p = 0.98$

Note: K-BIT = Kaufman Brief Intelligence Test, SES = socioeconomic status, Fitness% = relative cardiorespiratory fitness percentile based on age, height and weight. SSAP and SSAS refer to social support for academics from parent and school-teachers, respectively.

regular basis and engaged in one or more sports, and all participants were required to be free of a history of severe brain injury, as well as special educational services related to cognitive or attentional disorders, neurological disease, or physical disabilities.

Following their initial visit, participants with a concussion history were matched with participants who had not sustained a concussion. Specifically, participants were matched on demographic factors of age, sex, pubertal timing, IQ, ADHD symptoms including impulsivity hyperactivity and attention, SES, social support for academics, and cardiorespiratory fitness; factors known to influence cognitive development (Hillman, Erickson, & Kramer, 2008; Luna, 2009) and moderate outcomes of pediatric brain injury (Anderson, Brown, Newitt, & Hoile, 2011). Demographic information is listed in Table 1.

2.2. Testing protocol

All participants completed a 2-day testing protocol, which was approved by the Institutional Review Board at the University of Illinois, and lasted 1.5–2 h per visit. During the first visit guardians provided written informed consent and participants provided written assent. Guardians then provided demographic information as described above. Pubertal stage was assessed via a modified Tanner Staging System questionnaire (Taylor et al., 2001), attention deficit and hyperactivity disorder (ADHD) symptoms were assessed via the ADHD rating scale IV (DuPaul, Power, Anastopoulos, & Reid, 1998), and socio-economic status (SES) was determined using a trichotomous index based on: (1) participation in free or reduced-price meal program at school, (2) the highest level of education obtained by the mother and father, and (3) number of parents who worked full-time (Birnbaum et al., 2002). Health issues exacerbated by physical exercise were screened via the Physical Activity Readiness Questionnaire (Thomas, Reading, & Shephard, 1992). While guardians completed demographic information, participants were administered the Kaufman Brief Intelligence Test (K-BIT; Kaufman & Kaufman, 1990) to assess intelligence quotient and completed a social support for academics questionnaire. Given the positive relation of cardiorespiratory fitness to brain and cognition (Hillman et al., 2008), children completed a maximal exercise test to assess aerobic fitness. For additional details, please see Moore, Drollette, Scudder, Bharij, and Hillman (2014).

On the second visit participants were outfitted with a high density EEG cap and sat in a sound and light attenuated room approximately 1 m from a monitor, and completed a modified flanker task. Participants were given a brief rest and encouragement

after each testing block. All participants were paid \$15 per hour for their participation.

2.3. Flanker task

To investigate attention and conflict/inhibitory control, participants completed a child friendly flanker task (Pontifex et al., 2011). First, participants completed a response-compatible flanker task, which required them to focus on a centrally located cartoon fish, while ignoring flanking fish, which were either congruent or incongruent with the target stimuli. Participants were instructed to manually press the button on a response controller (Neuroscan STIM system; Compumedics, Charlotte, North Carolina), which corresponded to the direction that the center (i.e., target) fish was swimming. Thus, during the compatible condition of the task, participants were instructed to make a left thumb press on a response controller when the target stimulus pointed left and a right thumb press when the target stimulus pointed right. Participants then completed an incompatible stimulus–response condition, wherein the response mappings to each of the stimuli were reversed such that a target stimulus, which previously required a left response, now required a right response (i.e., left thumb press to a right pointing target). Participants were given 24 practice trials before each condition, which included two blocks of 75 trials for each compatibility condition. Stimuli consisted of an array of five cartoon fish (i.e., one target and two flankers on either side) measuring 3 cm tall and separated by 1 cm for visual angle of 1.72° . All stimuli were presented focally on a blue background for 200 ms, with a 1700 ms inter-stimulus interval, and a 1650 ms response window. Mean response accuracy, reaction time, errors of omission (misses) and errors of commission (false alarms) were computed for each condition of the flanker task. In addition, sequential congruency as well as post-error reaction time and response accuracy was calculated.

2.4. Neuroelectric data acquisition

EEG activity was recorded from 64 sintered Ag-AgCl electrodes (10 mm sensors; FPz, Fz, FCz, Cz, CPz, Pz, POz, Oz, FP1/2, F7/5/3/1/2/4/6/8, FT7/8, FC3/1/2/4, T7/8, C5/3/1/2/4/6, M1/2, TP7/8, CB1/2, P7/5/3/1/2/4/6/8, PO7/5/3/4/6/8, O1/2), arranged according to the International 10–10 system (Chatrain, Lettich, & Nelson, 1985) using a Neuroscan Quik-cap (Compumedics, Inc, Charlotte, NC). EEG activity was referenced to averaged mastoids (M1, M2) with AFz serving as the ground electrode. Impedance was kept below $10\text{ k}\Omega$. Additional electrodes were placed above and below the left orbit and on the outer canthus of each eye to monitor electro-oculographic (EOG) activity with a bipolar recording. Continuous raw EEG data were collected using Neuroscan Scan software (v 4.5) and amplified through a Neuroscan Synamps 2 amplifier with a 24-bit A/D converter and ± 200 millivolt (mV) input range (763 $\mu\text{V/bit}$ resolution). Data was sampled at 500 Hz and amplified 500 times with a DC to 70 Hz filter, and a 60 Hz notch filter.

2.5. Neuroelectric data reduction

Prior to segmentation and averaging, an off-line electro-oculographic (EOG) reduction procedure was applied to individual trials via a spatial filter, which performed a principle component analysis (PCA) to determine the major components that characterize the EOG artifact between all channels. This procedure then reconstructed the original channels without the artifact components (Compumedics Inc., Neuroscan, 2003). Trials with a response error or artifact exceeding $\pm 75\text{ }\mu\text{V}$ were rejected and only artifact free data were retained for averaging.

Prior to segmentation and averaging, data were filtered with a zero phase shift 30-Hz low-pass cutoff (24 dB/octave rolloff) and a 0.1-Hz high-pass filter (24 dB/octave rolloff). Stimulus-locked ERP components included the creation of epochs from –100 to 1000 ms around stimuli and were baseline corrected using the 100-ms pre-stimulus period. The N2 component was identified as the mean amplitude within a 30 ms interval surrounding the largest negative-going peak within 150–350 ms latency. The P3 component was identified as the mean amplitude within a 50 ms interval surrounding the largest positive-going peak within a 300–700 ms latency window. Amplitude was measured as the difference between the mean pre-stimulus baseline and mean peak-interval amplitude; peak latency was defined as the time point corresponding to the maximum amplitude of the local peak.

Response-locked ERP components included the creation of epochs from –400 to 600 ms around the response and were baseline corrected using the –400 to –200 ms prestimulus period (Olvet & Hajcak, 2009; Pontifex et al., 2010). Data were filtered using a zero phase shift 1 Hz (24 dB/octave roll off) to 12-Hz (24 dB/octave roll off) band pass filter. The ERN component was identified as the mean amplitude within a 30 ms interval surrounding the largest negative going peak within a 0–150 ms window relative to the response. The Pe component was identified as the mean amplitude within a 50 ms interval surrounding the largest positive going peak within a 300–600 ms window relative to the response. Amplitude was measured as the difference between the mean pre-stimulus baseline and mean peak-interval amplitude; peak latency was defined as the time point corresponding to the maximum local peak amplitude.

2.6. Statistical analysis

Primary outcome measures were behavioral and neuroelectric indices of performance for the flanker task. Task performance measures for the flanker task were submitted to a 2 (Group: concussion, healthy match-control) \times 2 (Compatibility: compatible, incompatible) \times 2 (Congruency: congruent, incongruent) repeated measures ANOVA. Tertiary measures of post-trial task performance were also assessed using a 2 (Group: Concussion, healthy match-control) \times 2 (Compatibility: compatible, incompatible) \times 2 (Congruency: congruent, incongruent) \times 2 (Accuracy: post error, post match correct) repeated measures ANOVA.

Preliminary analysis of ERP epochs revealed that the number of trials retained for averaging did not significantly differ between groups for either stimulus- or response-locked components ($p's \geq 0.78$). Similar to previous research (Larson, Farrer, & Clayson, 2011; Moore, Drollette, & et al., 2014; Pontifex et al., 2011), preliminary ERP analyses were carried out to determine the sites of maximal activation. Analysis revealed that all participants exhibited topographical maxima at sites FCz and Cz for the N2 component, but exhibited differences in amplitude maxima (centrality and laterality) for the P3 component (CPz, CP1, CP2, Pz, P1, P2). Subsequent analyses were carried out on regions of interest (Frontal: Fz, FCz; Centro-Parietal: CPz, CP1, CP2, Pz, P1, P2), which were created by collapsing across the maximal sites of activation. ERP component values were then submitted to similar factorial ANOVAs as described above. All statistical analyses were conducted with $\alpha = 0.05$ using the Greenhouse–Geisser statistic with subsidiary univariate ANOVAs and Bonferroni corrected student's *t*-tests for *post hoc* comparisons. All values are expressed as means \pm SD, and given the number of comparisons, only significant findings are reported herein.

3. Results

Analysis failed to reveal group differences for any demographic factors, $ts(30) \leq 0.47$, $p's \geq 0.40$, (see Table 1), suggesting that sample matching was successful.

3.1. Behavioral data

Behavioral data are presented in Fig. 1. Omnibus analysis of response accuracy revealed a trend between groups, $F(1,30) = 3.13$, $p = 0.09$, $\eta^2 = 0.01$, suggesting that across conditions, and trial types, children with a concussion history tended to respond less accurately ($m = 78.6 \pm 11.1\%$) than children in the control group ($m = 84.6 \pm 6.9\%$). In addition, children with a concussion history exhibited significantly more omission errors ($m = 14.5 \pm 12.0\%$), relative to children in the control group ($m = 8.8 \pm 5.9\%$), $F(1,30) = 4.27$, $p = 0.05$, $\eta^2 = 0.52$. Furthermore, analysis revealed a significant group difference for the sequential-congruency effect, $t(30) = 3.01$, $p < 0.01$. That is, children with a history of concussion experienced greater stimulus–response conflict ($m = 61.5 \pm 18.1\%$), relative to children in the control group ($m = 74.7 \pm 26.6\%$), when completing an incongruent trial that was immediately preceded by an incongruent trial (ii).

Decomposition of post-error trials revealed a group \times compatibility interaction, $F(1,30) = 5.60$, $p = 0.03$, $\eta^2 \geq 0.16$. Post-hoc testing revealed that children with a concussion history demonstrated lower post-error accuracy ($m = 54.4 \pm 29.8\%$) relative to children in the control group ($m = 82.6 \pm 12.3\%$), but only during the incompatible condition of the task, $t(30) = 3.51$, $p < 0.01$. Lastly, age at injury correlated negatively with incompatible response accuracy, and positively with omission errors and post-error accuracy during the incompatible condition of the task, $r^2 \geq 0.52$, $p's \leq 0.04$. Thus, children who were injured earlier in life exhibited poorer performance when attention and cognitive control requirements increased.

3.2. Neuroelectric data

3.2.1. N2

Stimulus-locked ERP data are presented in Fig. 2. Analysis revealed a group \times compatibility interaction for amplitude, $F(1,30) = 4.34$, $p's = 0.05$, $\eta^2 = 0.13$. Post-hoc testing revealed that relative to children in the control group ($m = -4.0 \pm 3.4 \mu V$), children with a concussion history ($m = -7.6 \pm 4.6 \mu V$) exhibited greater amplitude during the incompatible condition of the task, $t(31) = 2.50$, $p = 0.02$, indicating that children with a concussion history experienced greater stimulus–response conflict during the incompatible condition. Analysis also revealed a main effect of group for latency, $F(1,30) = 4.30$, $p = 0.05$, $\eta^2 = 0.13$, indicating that across all conditions and trial types children with a concussion history ($m = 288.5 \pm 32.6$ ms) resolved stimulus–response conflict more slowly, relative to children in the control group ($m = 264.4 \pm 35.8$ ms).

3.2.2. P3

Analysis revealed a main effect of group for amplitude, $F(1,30) = 11.10$, $p < 0.01$, $\eta^2 = 0.27$. Thus, across all conditions and trial types, children with a concussion history ($m = 6.1 \pm 3.7 \mu V$) exhibited smaller P3b amplitude relative to children in the control group ($m = 10.7 \pm 3.7 \mu V$). Children with a concussion history therefore exhibited a general decrease in attentional resource allocation, irrespective of task demands.

3.2.3. ERN

Response-locked ERP data are presented in Fig. 3. Analysis revealed a group \times response-correctness interaction, for amplitude

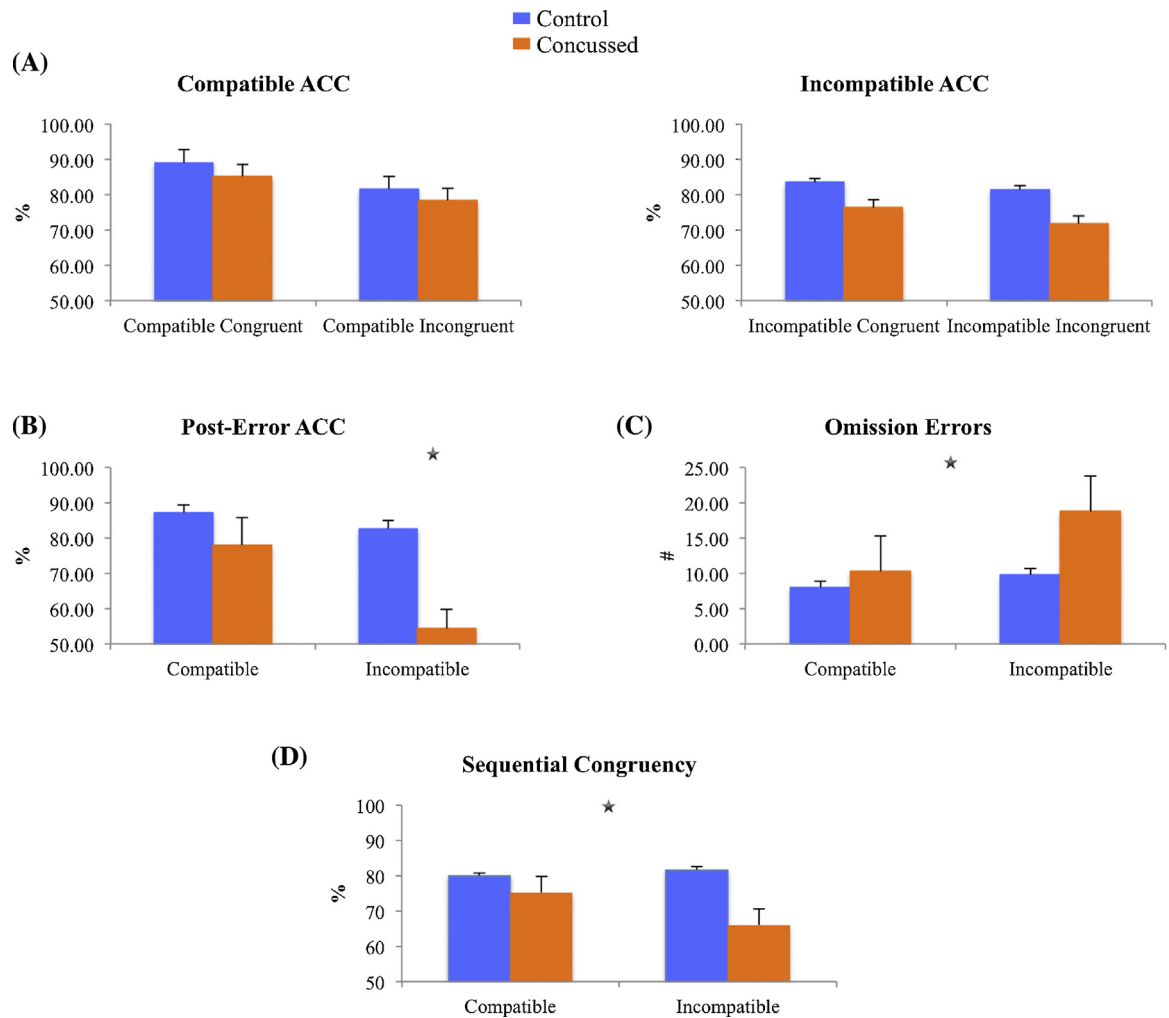


Fig. 1. Behavioral data for children with a concussion history and matched controls. Note: (A) accuracy data for congruent and incongruent trials for the compatible and incompatible conditions; (B) post-error accuracy data for the compatible and incompatible conditions; (C) number of omission errors for the compatible and incompatible conditions; (D) sequential congruency effect for (il) sequences; * denotes a significant difference at $p \leq 0.05$.

$F(1,30)=4.84$, $p=0.04$, $\eta^2=0.14$. Post-hoc testing revealed that relative to children in the control group ($m=-6.1 \pm 3.5 \mu v$), children with a concussion history exhibited smaller amplitude ($m=-3.1 \pm 2.1 \mu v$), following error trials, $t(29)=3.4$, $p<0.01$. Thus, children with a concussion history failed to adequately monitor actions/conflict following an erroneous response.

3.2.4. Pe

Analysis revealed a main effect of group for amplitude, indicating that irrespective of response outcome, children with a concussion history exhibited smaller amplitude ($m=2.6 \pm 1.7 \mu v$), relative to children in the control group ($m=5.0 \pm 2.8 \mu v$). This effect, however, was superseded by a group \times compatibility interaction, $F(2,62)=4.41$, $p=0.04$, $\eta^2=0.13$. Post-hoc testing revealed that irrespective of response outcome, children with a concussion history exhibited smaller amplitude during the incompatible condition of the task ($m=2.6 \pm 2.3 \mu v$), relative to children in the control group ($m=5.4 \pm 2.6 \mu v$; $t(30)=2.75$, $p=0.01$). However, post-hoc t tests revealed only a trend for the compatible condition of the task, $t(30)=2.23$, $p=0.03$. Lastly, bivariate correlations revealed a significant relation between Pe amplitude and post-error accuracy, $r^2=0.41$, $p=0.02$, indicating that those exhibiting

the greatest Pe amplitude demonstrated the greatest post-error accuracy.

4. Discussion

The current study sought to examine the long-term outcomes of pediatric concussion on aspects of attention and cognitive control during a modified flanker task. Relative to a matched control group, children with a concussion history exhibited behavioral deficits in modulating and maintaining attention, and flexibly adjusting behavior in order to correct erroneous response patterns. Furthermore, age at injury was significantly correlated with the magnitude of some deficits, suggesting that children injured earlier in life may exhibit poorer behavioral outcomes.

In addition to behavioral differences, children with a concussion history exhibited neuroelectric alterations during both stimulus engagement and response evaluation. These alterations were indicative of deficits in monitoring and resolving stimulus–response conflict (N2), attentional resource allocation (P3), action monitoring (ERN), and error awareness (Pe). Together, the current results suggest that a concussion incurred during childhood may lead to subtle, but pervasive deficits in attention and cognitive control for a sustained period of time. Lastly, the current

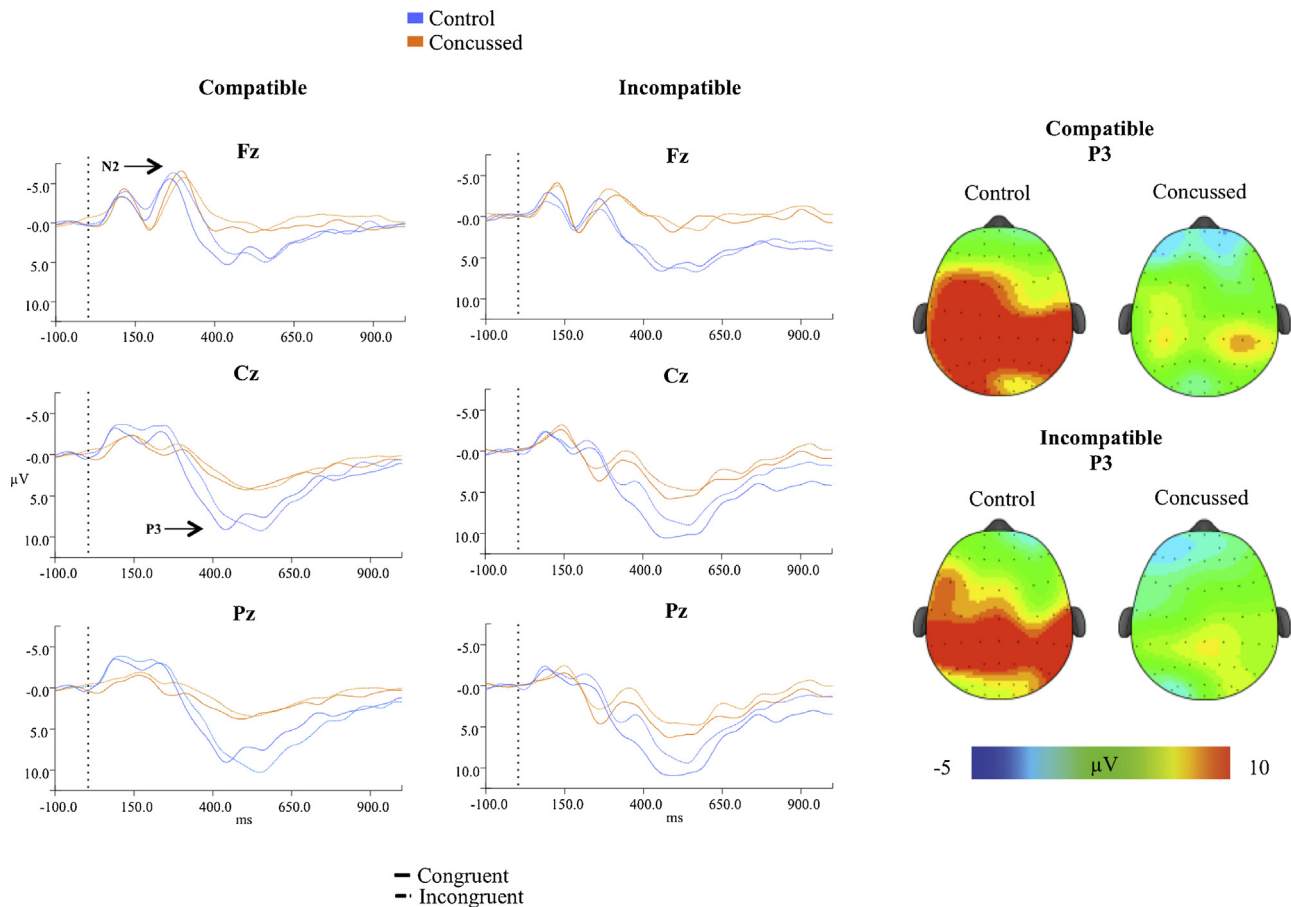


Fig. 2. Stimulus-locked ERP waveforms and topographic plots for children with a concussion history and matched controls.

results further validate the sensitivity of flanker paradigms and ERPs for detecting concussion-related deficits across the lifespan.

4.1. Task performance and brain function during stimulus engagement

Relative to children in the control group, children with a concussion history evidenced multiple deficits in attention and cognitive control during the flanker task. Although children with a concussion history only trended towards decreased accuracy during the flanker task, effect sizes for the group difference were larger during the incompatible ($d = 0.75$) relative to the compatible condition of the task ($d = 0.34$), suggesting that children with a concussion history trended towards behavioral deficits when inhibitory control demands were increased. Future studies employing larger sample sizes will be better positioned to evaluate the reliability of this observation.

Children with a concussion history did, however, exhibit a significantly greater number of omission errors. Evaluation of effect sizes revealed a large increase for between group differences for the incompatible ($d = 0.77$), relative to the compatible condition of the task ($d = 0.35$), suggesting that group differences magnified under increasing cognitive control requirements. Furthermore, omission errors were negatively correlated with age at injury, suggesting that those who were concussed earlier in life had the highest rates of omission errors.

Omission errors occurring during continuous performance/attentional vigilance paradigms such as the flanker task are believed to reflect lapses of sustained attention (Caggiano & Parasuraman, 2004; Fisk & Schneider, 1981). Previous adult concussion evaluations have observed deficits in sustained attention

during flanker performance (Pontifex et al., 2012), suggesting that flanker paradigms are well suited for detecting concussion-related lapses of attention. Sustained attention is a fundamental component of human cognition and is essential for success in academic and vocational environments (DuPaul et al., 1998; Posner, 2004). Furthermore, disruptions in sustained attention are believed to exacerbate impairments in other aspects of cognition (Sarter, Givens, & Bruno, 2001), due to the reciprocal interconnectivity of sustained attention and cognitive control networks (Dosenbach et al., 2006; Hilti et al., 2013). As such, concussion-related deficits in sustained attention may have contributed to group differences in other metrics of performance.

Indeed, children with a concussion history also exhibited a significantly smaller sequential congruency effect, exhibiting decreased accuracy, irrespective of compatibility, for (ii) sequences (although effect sizes were largest for the incompatible relative to the compatible condition, $d = 0.86$ vs. $d = 0.67$). This result indicates that pediatric concussion negatively influences the ability to strategically regulate attentional resources in order to manage interference and reduce stimulus–response conflict. Thus, pediatric concussion appears to not only influence the ability to sustain, but also to modulate, attention.

In addition to behavioral deficits, children with a concussion history exhibited neuroelectric alterations during stimulus engagement. Specifically, relative to control children, children with a concussion history exhibited increased N2 latency across both conditions of the flanker task and increased N2 amplitude during the incompatible condition of the task. Previous evaluations of adults have also observed alterations in N2 latency and amplitude following concussion (Broglia, Pontifex, O'Connor, & Hillman, 2009; Moore, Drollette, & et al., 2014). The latency of the fronto-central

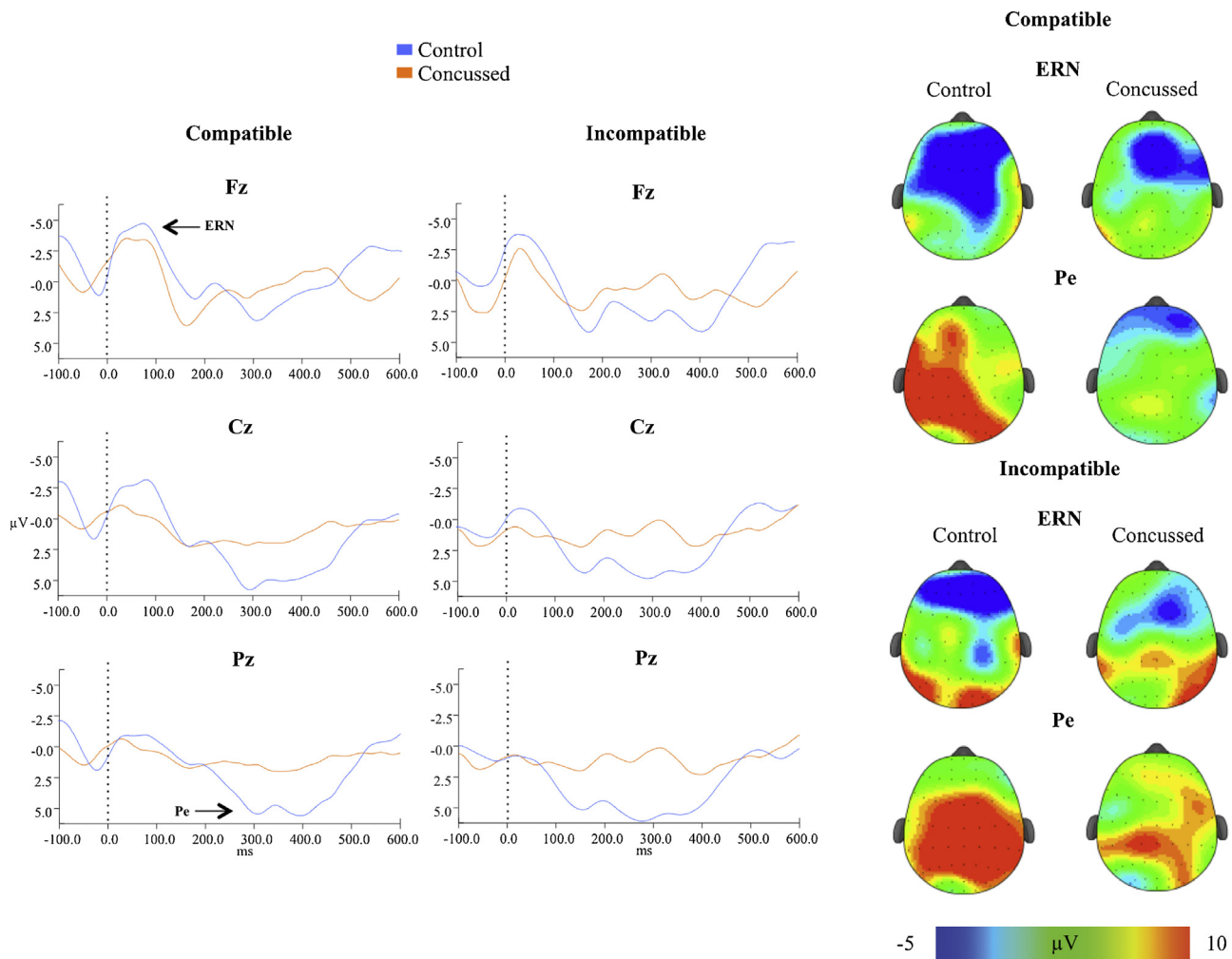


Fig. 3. Response-locked ERP waveforms and topographic plots for children with a concussion history and matched controls.

N2 is thought to reflect aspects of the response selection process associated with the resolution of stimulus–response conflict (Gajewski & Falkenstein, 2012; Gajewski, Stoerig, & Falkenstein, 2008). A concussion history therefore appears to negatively relate to the speed of conflict resolution. It should be noted, however, that this alteration was not modulated by congruency or compatibility, suggesting that concussion leads to a generalized delay in conflict resolution.

Consonant with our predictions, however, children with a concussion history exhibited a selective increase in N2 amplitude during the incompatible condition of the task, relative to control children. The amplitude of the fronto-central N2 is believed to reflect the magnitude of conflict arising from the competition between the execution and inhibition of a response (Folstein & Van Petten, 2008; Nieuwenhuis et al., 2003). Accordingly, N2 amplitudes are more negative during conditions of greater stimulus–response conflict (Clayson & Larson, 2011), indicating that children with a concussion history experienced increasing conflict with increasing inhibitory control demands.

Lastly, relative to children in the control group, children with a concussion history exhibited a decrease in P3b amplitude. P3b amplitude is believed to reflect the suppression of extraneous neuronal activity in order to facilitate attentional processing (Polich, 2007). Previous child (Baillargeon et al., 2012; Moore et al., submitted for publication) and adult (see Broglio et al., 2011, for review) studies demonstrate a robustly negative relation between concussion history and P3b amplitude. The current results further

demonstrate the sensitivity of the P3b for detecting subtle, yet persistent, concussion-related dysfunction in the neurophysiology underlying attention across the lifespan. Similar to N2 latency, P3b amplitude was not modulated by congruency or compatibility, suggesting a generalized deficit in the ability to allocate attentional resources irrespective of task demands.

Although this generalized deficit may seem perplexing at first, the result is logical when considered in relation to the generalized alterations in N2 latency. That is, the amplitude of centro-parietal P3b has been found to be in direct (negative) relation to the latency of the fronto-central N2 (Gajewski et al., 2008; Nieuwenhuis, Aston-Jones, & Cohen, 2005). Accordingly, the prolonged and generalized (across trial-types and conditions) stimulus–response conflict experienced by children with a concussion history may have yielded a generalized decrease in the ability to allocate/modulate attentional resources in accordance with task demands. Together, the current stimulus-locked ERP findings indicate that pediatric concussion leads to a complex mixture of general (N2 latency, P3b amplitude) and specific (N2 amplitude) deficits during stimulus engagement.

4.2. Task performance and brain function during response evaluation

Children with a concussion history exhibited deficits in post-error accuracy during the incompatible condition of the task. Furthermore, post-error accuracy was negatively correlated with

age at injury, suggesting that children injured earlier in life exhibited the lowest post-error accuracy. Previous adult studies have also reported persistent concussion-related deficits in post-error accuracy (Larson et al., 2011); however, this is the first study to demonstrate such deficits in previously concussed children. As post-error accuracy is believed to reflect the ability to implement top-down attentional control to correct errors (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Kerns et al., 2004), pediatric concussion appears to negatively influence the ability to dynamically modulate attention during (incompatible) conditions of increasing stimulus–response conflict.

This assertion is supported on the neuroelectric level, as children with a concussion history exhibited smaller ERN and Pe amplitudes, with the largest group differences observed for the incompatible condition of the task. The ERN is thought to reflect the activation of action monitoring processes to signal the up-regulation of top-down control (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring & Knight, 2000), and the Pe is believed to reflect the implementation of compensatory control following the conscious detection of an error (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). Accordingly, Pe amplitudes were positively correlated with post-error accuracy. Thus, the current results point to a cascade of deficits whereby children with a concussion history fail to adequately monitor their actions (ERN), leading to decreased error awareness (Pe) and the subsequent failure to correct erroneous response patterns (post-error task performance).

Although this is the first pediatric concussion study to examine the ERN, alterations in ERN amplitude appear to be a robust finding in concussion research, as this alteration has been observed across multiple age ranges and cognitive tasks (de Beaumont, Beauchemin, Beaulieu, & Jolicoeur, 2013; Pontifex et al., 2009). Therefore, persistent alterations in action monitoring may be a hallmark outcome of concussive injuries. This is, however, the first concussion study to reveal deficits in Pe amplitude, suggesting that the neural resources underlying the error awareness may be particularly fragile during development. Given the intimate roles of the ERN, Pe and post-error behavior in dynamic learning (Alexander & Brown, 2010; Holroyd & Coles, 2008), future research will be able to gain a more comprehensive understanding of functional outcomes by incorporating these measures.

4.3. Limitations and conclusions

In sum, children who were on average more than 2 years from a single concussive event exhibited multiple deficits in attention and cognitive control, relative to demographically matched controls. These deficits, though subtle, were pervasive, manifesting on the behavioral and neural levels. The current patterning of deficits points to an interconnected series of deficits, whereby children with a concussion history fail to adequately regulate attentional resources to reduce stimulus–response conflict, monitor actions, and correct erroneous response patterns. As many of these deficits were observed for measures of behavior (omission errors, sequential congruency, post error ACC) and brain function (ERN, Pe), which typically go unevaluated, the current results underscore the importance of going beyond standard analysis when evaluating pediatric concussion. Doing so will enable more accurate identification and tracking of concussion-related deficits.

Although meritorious, the current investigation is not without limitations. First, although participants were rigorously matched, the investigation was cross-sectional in design. Thus, it is impossible to know whether the subtle group differences detected herein represent the closure of concussion-related deficits or the emergence of injury-related developmental differences. Also, it is possible that some unmeasured variable contributed to group

differences in behavioral or neuroelectric function. That is, although exhaustive demographic information was collected and parents verified their child had a medically diagnosed concussion, we did not have access to children's medical records. Future research will benefit from longitudinal analysis and access to medical records that better characterizes the nature of the concussive injury.

Irrespective of these shortcomings, the current findings add valuable information regarding the nature and duration of deficits stemming from pediatric concussion. The results indicate that neurocognitive processing deficits extend well beyond the acute phase of recovery, and that children injured earlier in life may have poorer outcomes. Hopefully the current results will engender more comprehensive assessments and longitudinal tracking of children with concussive injuries, and aid the development and precise implementation of remedial measures.

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Appendix A.

Table A1.

Table A1

Table presents the correlations between stimulus- and response-locked ERP values and behavioral metrics of performance during the flanker task.

Measures	Compatible condition	Incompatible condition
N2 Amp-ACC	$r^2 = 0.29, p = 0.10$	$r^2 = 0.36, p = 0.04^*$
N2 Amp-RT	$r^2 = 0.32, p = 0.07$	$r^2 = 0.10, p = 0.57$
N2 Amp-omission errors	$r^2 = 0.22, p = 0.23$	$r^2 = 0.16, p = 0.36$
N2 Amp-commission errors	$r^2 = 0.23, p = 0.21$	$r^2 = 0.35, p = 0.05^*$
N2 Lat-ACC	$r^2 = 0.04, p = 0.81$	$r^2 = 0.15, p = 0.41$
N2 Lat-RT	$r^2 = 0.13, p = 0.49$	$r^2 = 0.13, p = 0.31$
N2 Lat-omission errors	$r^2 = 0.02, p = 0.76$	$r^2 = 0.35, p = 0.05^*$
N2 Lat-commission errors	$r^2 = 0.05, p = 0.71$	$r^2 = 0.16, p = 0.36$
P3 Amp-ACC	$r^2 = 0.45, p = 0.01^*$	$r^2 = 0.71, p < 0.01^*$
P3 Amp-RT	$r^2 = 0.17, p = 0.52$	$r^2 = 0.21, p = 0.43$
P3 Amp-omission errors	$r^2 = 0.37, p = 0.12$	$r^2 = 0.53, p < 0.01^*$
P3 Amp-commission errors	$r^2 = 0.34, p = 0.15$	$r^2 = 0.42, p = 0.02^*$
P3 Lat-ACC	$r^2 = 0.18, p = 0.51$	$r^2 = 0.27, p = 0.31$
P3 Lat-RT	$r^2 = 0.26, p = 0.34$	$r^2 = 0.30, p = 0.10$
P3 Lat-omission errors	$r^2 = 0.29, p = 0.1$	$r^2 = 0.16, p = 0.37$
P3 Lat-commission errors	$r^2 = 0.23, p = 0.2$	$r^2 = 0.22, p = 0.23$
Measures	Across conditions	
ERN Amp-post error ACC	$r^2 = 0.10, p = 0.58$	
ERN Amp-post error RT	$r^2 = 0.11, p = 0.59$	
ERN Amp-omission errors	$r^2 = 0.05, p = 0.77$	
ERN Amp-commission errors	$r^2 = 0.03, p = 0.86$	
ERN Amp-commission errors	$r^2 = 0.04, p = 0.81$	
ERN Amp-errors runs	$r^2 = 0.16, p = 0.36$	
Pe Amp-post error ACC	$r^2 = 0.41, p = 0.02^*$	
Pe Amp-post error RT	$r^2 = -0.33, p = 0.06$	
Pe Amp-omission errors	$r^2 = 0.05, p = 0.77$	
Pe Amp-commission errors	$r^2 = 0.04, p = 0.14$	
Pe Amp-errors runs	$r^2 = 0.35, p = 0.05^*$	

Note: ACC and RT refer to mean response accuracy and mean response time respectively.

* Denotes a significant correlation at $p \leq 0.05$.

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