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A Longitudinal, Within-Person Investigation of the Association between the P3 ERP Component and Externalizing Behavior Problems in Young Children

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Abstract

Background: Externalizing problems, including aggression and conduct problems, are thought to involve impaired attentional capacities. Previous research suggests that the P3 event-related potential (ERP) component is an index of attentional processing, and diminished P3 amplitudes to infrequent stimuli have been shown to be associated with externalizing problems and attention-deficit/hyperactivity disorder (ADHD). However, the vast majority of this prior work has been cross-sectional, and has not examined young children. The present study is the first investigation of whether *within*-individual changes in P3 amplitude predict changes in externalizing problems, providing a stronger test of developmental process.

Method: Participants included a community sample of children ($N=153$) followed longitudinally at 30, 36, and 42 months of age. Children completed an oddball task while ERP data were recorded. Parents rated their children's aggression and ADHD symptoms.

Results: Children's within-individual changes in the P3 amplitude predicted concomitant within-child changes in their aggression such that smaller P3 amplitudes (relative to a child's own mean) were associated with more aggression symptoms. However, changes in P3 amplitudes were not significantly associated with ADHD symptoms.

Conclusions: Findings suggest that the P3 may play a role in development of aggression, but do not support the notion that the P3 plays a role in development of early ADHD symptoms.

Keywords

P3 ERP; externalizing behavior problems; aggression; ADHD; early childhood

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Conflicts of Interest

No conflicts declared.

Externalizing behaviors in early childhood predict maladaptive outcomes in adulthood, including substance use and criminality (Petersen, Bates, Dodge, Lansford, & Pettit, 2015). One key feature of externalizing disorders is impaired attentional capacities. Event-related potentials (ERPs), stimulus-locked neuro-electrical activity measured using electroencephalography (EEG), can be used to study neural correlates of attentional processing that mark impaired attention in externalizing problems. The present study advances understanding of developmental process in externalizing problems by examining the longitudinal association between neural functioning and externalizing problems.

P3 ERP and Attention Processing

The oddball task, in which two stimuli are presented, one frequent and the other infrequent, is commonly used to assess neural correlates of attention. A widely studied index of response to infrequent stimuli in the oddball task, the P3 ERP component, the third positive deflection in the waveform post-stimulus, is considered an index of attention. The predominant theory of the P3 is that it indexes attention and memory processes, reflecting neural mechanisms associated with updating mental representations stored in working memory based on novel incoming stimuli (Polich, 2012). The cognitive interpretation of the P3 depends on the task in which it is elicited. In the current study's passive oddball task, in which no behavioral response was required, the P3 likely represents attentional orienting (Friedman, Cycowicz, & Gaeta, 2001). Attentional orienting involves rapid, passive attentional shifts to new/unexpected stimuli, and may reflect early evaluation of stimulus importance to determine whether further cognitive processing is necessary (Hermens et al., 2010).

A robust literature has examined the P3 cross-sectionally at different points in childhood. Meta-analytic evidence suggests that the auditory P3 amplitude increases across childhood, plateauing at age 20, whereas its latency decreases across the lifespan (van Dinteren, Arns, Jongsma, & Kessels, 2014). Additionally, there are slight variations in the electrode regions where the P3 is maximal across development (Hoyniak, Petersen, McQuillan, Staples, & Bates, 2015; Johnstone, Barry, Anderson, & Coyle, 1996). Despite differences in morphology and topography of the P3 elicited in children, an established literature suggests that the P3 is an index of attentional processing in childhood (Hoyniak et al., 2015; Johnstone, Barry, & Clarke, 2013; van Dinteren et al., 2014).

P3 and Externalizing Problems

Attentional orienting deficits play a key role in the social information processing style of individuals with high levels of aggression (e.g., failing to notice cues of non-aggressive intent; Dodge & Crick, 1990), explaining why diminished attention orienting capacities might be associated with externalizing problems. Therefore, the P3 may tap increased risk for externalizing problems. Given robust findings demonstrating an association between the P3 and externalizing problems, the P3 may be an endophenotype (i.e., an intermediate phenotype) of externalizing psychopathology, reflecting the biological processes underlying externalizing problems (Iacono & Malone, 2011). Meta-analyses have shown that smaller P3 amplitudes are associated with externalizing problems (Gao & Raine, 2009; Pasion,

Fernandes, Pereira, & Barbosa, 2018). The P3 shows rank-order stability across adulthood (Yoon, Malone, & Iacono, 2015), and has been shown to predict later criminality (Gao, Raine, Venables, & Mednick, 2013). Individual differences in the P3, which are highly heritable, may be a marker of genetic risk for externalizing psychopathology (van Beijsterveldt & van Baal, 2002), and the association between the P3 and externalizing problems is considered genetically mediated (Hicks et al., 2007). Additionally, the P3 is generated by dopaminergic neurotransmission (Pogarell et al., 2011) and by a distributed neural circuit including the lateral prefrontal cortex (Polich, 2007; Soltani & Knight, 2000), both of which have shown impairments in externalizing disorders (Gatzke-Kopp et al., 2009).

Despite considerable research linking the P3 to externalizing psychopathology, the vast majority of this prior work focused on adults and adolescents. We are aware of no studies that have examined the P3 in relation to externalizing problems in toddlers and preschoolers, so it is unclear whether diminished P3 amplitudes might play a role in development of externalizing problems in early childhood. Early childhood is an ideal window to study development of the P3 in relation to externalizing problems because (a) early childhood is characterized by rapid neurodevelopment supporting attention and self-regulatory processes (Diamond, 2002), and (b) externalizing behaviors are common in early childhood, especially physical aggression, which reaches its highest level during this era (Tremblay, 2002). Individual differences in aggression are highly stable and appear as early as toddlerhood (Olweus, 1979). Better understanding of early neural processes associated with development of externalizing problems may lead to earlier, more precisely targeted prevention efforts.

Prior research on the P3 across childhood has mostly been cross-sectional, and has not examined whether within-individual changes in the P3 predict within-individual changes in externalizing problems. Investigating the association within the individual removes between-subject confounds by using the individual as their own control, which is a stronger test of causality than between-subjects approaches (Duckworth, Tsukayama, & May, 2010). Based on prior work, we cannot ascertain whether the P3 plays a role in development of externalizing problems or whether it is simply a marker of risk for externalizing psychopathology in general. The P3 has associations with a number of disordered phenotypes, including aggression (Patrick, 2008), attention-deficit/hyperactivity disorder (ADHD; Tsai, Hung, & Lu, 2012), depression, and schizophrenia (Turetsky et al., 2015).

The Present Study

Longitudinal studies of the association between the P3 and externalizing problems in early childhood can elucidate neural processes associated with development of externalizing behaviors. The present study is the first, to our knowledge, to examine the *within*-individual association between the P3 and externalizing problems. Different associations can be observed at the group level (between-individual) and at the individual level (within-individual), and mistakenly attributing a between-individual association to a within-individual association is known as the ecological fallacy (Curran & Bauer, 2011). The present study examined the longitudinal association between a neural index of attentional processing, the P3, and externalizing behavior problems in early childhood, examining both

within- and between-individual associations. Children were followed longitudinally at 30, 36, and 42 months of age. Our main question was whether *within*-child changes in P3 amplitude predicted *within*-child changes in parent-reported externalizing behavior, examining aggression and ADHD symptoms separately.

Method

Participants

A community sample of children and their families ($N = 182$) were recruited from the Bloomington, Indiana area to participate when the children were 30, 36, and 42 months of age. Sample characteristics are reported in Table 1. Participants were assessed within one month of their target age. Children completed an oddball ERP task, and primary caregivers (97% mothers) reported on the child's behavior problems. To be included for analysis in the present study, children had to provide usable EEG data in the oddball task ($N = 165$, 91%). Exclusion criteria included non-febrile seizures ($n = 2$), head injury ($n = 9$), and psychotropic medication ($n = 2$ —none of which were psychostimulants), resulting in a final sample of 153.

Due to planned missingness and censoring (i.e., children not yet age-eligible), in the current sample, a total of 375 EEG assessments were possible. Of these 375 possible assessments, 73 assessments were not scheduled because the parent elected not to schedule a visit for their child (65 assessments) or equipment malfunctioning prevented us from collecting EEG data at that time (8 assessments). Hence, 302 EEG assessments were scheduled. Of 302 scheduled assessments, 257 provided usable EEG data. These 257 assessments were provided by 153 different children ($n_1 \text{ time} = 74$, $n_2 \text{ times} = 54$, $n_3 \text{ times} = 25$). Further details on missingness are in Supplementary Appendix S1 and Table S1.

Measures

Externalizing Behavior Problems.—Externalizing behavior problems were measured using mother report on the Child Behavior Checklist (CBCL 1½–5; Achenbach & Rescorla, 2000). We examined the two subscales comprising the Externalizing scale: Aggression (19 items) and Attention Problems (5 items). The Aggression subscale includes items about physical aggression, destruction, anger, noncompliance, and attention demands. The Attention Problems subscale has been interpreted as a measure of ADHD symptoms because it assesses the three dimensions of ADHD symptoms: inattention, hyperactivity, and impulsivity (Lifford, Harold, & Thapar, 2008). It is associated with other measures of ADHD, including the Conners Rating Scale and DSM-IV symptoms of ADHD (Derks et al., 2008). In addition, it has been shown to measure ADHD as accurately as the Conners Rating Scale (Derks et al., 2008), with strong sensitivity and specificity (Chen, Faraone, Biederman, & Tsuang, 1994).

The CBCL is among the best normed and most widely used measures for behavior problems in this age range, has good test-retest reliability and good validity (content, criterion, construct; Sattler & Hoge, 2006). Primary caregivers rated whether a behavior was *not true* (0), *somewhat or sometimes true* (1), or *very or often true* (2), and scores were summed

across items, with higher levels reflecting more behavior problems. This continuous approach to scoring behavior problems is consistent with evidence that externalizing problems and ADHD are dimensional not categorical (Coghill & Sonuga-Barke, 2012). For this sample, eight children's scores were above the borderline clinical threshold of a *T*-score 65 (i.e., above 1.5 *SD*) for externalizing problems. Cronbach's alpha in this sample was .90 for aggression and .69 for ADHD symptoms. Cross-time continuity was $r = .63$ for aggression and $r = .60$ for ADHD symptoms ($df = 189$, $ps < .001$). Children who had scores for externalizing problems were $n_{30\text{months}} = 146$, $n_{36\text{months}} = 120$, $n_{42\text{months}} = 101$.

P3 ERP.—Children participated in an oddball task and a go/no-go task while EEG data were collected during a lab visit. The present study focuses on the P3 ERP from the oddball task. A 6-minute auditory oddball (two-tone discrimination) task was used to elicit a P3 ERP component to infrequent sounds. The task was passive; children were not instructed to respond to any stimuli. Although the P3 from passive and active tasks have different latencies, spatial distributions, and cognitive interpretations (Polich, 2007), they both index attentional processing, and smaller amplitudes of both kinds of P3 have been associated with externalizing disorders (Rydckjær et al., 2017; Tsai et al., 2012). We chose to use a passive oddball task because more trials of usable data would be available than a task that required a behavioral response. ERP measures in a passive task may be especially useful in early childhood when behavioral response capacities are still developing and thus less stable, which would complicate interpretation of behavioral task performance.

Pure, low-frequency (1,000 Hz) and high-frequency (1,500 Hz) tones were randomly presented so that one tone occurred on 70% of trials (84 trials; frequent stimulus) and the other tone occurred on 30% of trials (36 trials; infrequent stimulus), with 120 trials in total. The two tones were counterbalanced as frequent versus infrequent across children. Each tone lasted for approximately 300 ms, and the task included an interstimulus interval that varied randomly from 2,300–2,500 ms to prevent habituation. Children were not asked to make a behavioral response. During presentation of the auditory tones, children watched a child-friendly cartoon video on a monitor with the video's audio turned off. On average, participants contributed 25.30 ($SD = 7.67$) usable infrequent and 58.22 ($SD = 18.25$) frequent trials.

Netstation Acquisition software version 4.4.2 (Electrical Geodesics, Inc., Eugene, OR) was used to collect and process EEG data from a 128-electrode Hydrocel Geodesic Sensor Net with a sampling rate of 250 Hz. Before recording began, electrode impedances were adjusted lower than 50 k Ω . Children's continuous EEG data were band-pass filtered from 0.3 to 30 Hz, and epochs 1,200 ms in duration were extracted, beginning 200 ms prior to the presentation of the target stimulus. Data were then visually inspected for artifacts. Following visual inspection, a channel was marked as bad if a voltage change of greater than 150 μV occurred during a given segment of length 80 ms, and a segment was marked as bad if it contained 20 or more bad channels. On average, participants had 10.07 ($SD = 3.83$) bad channels out of 128 channels.

Epoched data were then re-referenced to the average reference (i.e., subtracting the average potential of all channels from the potential at each channel), and baseline corrected by

subtracting the average activity over the 200 ms baseline period representing grand-averaged waveforms (Figure 1). After processing, primary components of the ERP waveform were statistically decomposed using a sequential temporo-spatial principal components analysis (PCA), which objectively and empirically determines regions of electrodes and time frames that parsimoniously account for the variance in the waveforms, and whose components correspond to ERP components (Dien & Frishkoff, 2005). Children's P3 amplitudes were calculated as their mean amplitudes for the temporo-spatial component reflecting the P3 based on timing, morphology, and spatial topography. We provide more information about the temporo-spatial PCA in Supplementary Appendix S2. PCA-derived P3 waveforms are depicted in Supplementary Figure SI. Cross-time continuity of the P3 amplitude was $r[87] = .21$ ($p = .043$, two-tailed), suggesting some rank-order stability but also considerable neurodevelopmental change from 30 to 42 months of age.

Statistical Analysis

Using hierarchical linear modeling (HLM), which handles missingness and unbalanced data (Singer & Willett, 2003), we fit growth curve models with random intercepts and slopes to each child's trajectory of externalizing problems. Growth curve models examined whether within-child changes in P3 amplitudes predicted concomitant within-child changes in externalizing problems, controlling for between-child associations of P3 amplitudes with externalizing problems. Models included the child's (a) mean P3 amplitude across time in association with the child's intercept of externalizing problems (i.e., their level of externalizing problems at 30 months of age): γ_{02} , (b) mean P3 amplitude across time in association with the child's linear slope of externalizing problems (i.e., their change in externalizing problems from 30 to 42 months of age): γ_{12} , (c) time-varying P3 amplitude (centered around the child's mean P3 amplitude across occasions) predicting concomitant within-child changes in externalizing problems: γ_{20} , and (d) covariates that could plausibly account for the association between P3 amplitudes and externalizing problems, including the child's sex as a time-invariant covariate to account for the well-established sex differences in externalizing problems, and time-varying covariates to account for potentially systematic ERP missingness (number of bad channels and number of infrequent trials kept). Centering the P3 amplitude around the child's own mean (so-called person-mean centering) follows best practice for disaggregating within- and between-individual effects (Curran & Bauer, 2011). An assumption of the disaggregation is that the person-level mean P3 amplitude is estimated without error. We were interested in both within-individual (γ_{20}) and between-individual (γ_{02} , γ_{12}) effects. The between-child effects examined whether children's mean P3 amplitudes across time were associated with their intercepts or slopes of externalizing problems. The within-child effect examined whether individuals' time-specific deviations in the P3 amplitude away from their own mean predicted their time-specific deviations in externalizing problems *over and above* their linear slopes (i.e., β_{1i}) of externalizing problems. Thus, the within-child effect examined whether within-child changes in the P3 amplitude predicted concomitant within-child changes in externalizing problems. We fit separate growth curve models for aggression and ADHD symptoms. Model equations and information about the models are in Supplementary Appendix S3. As a sensitivity analysis, we examined models with multiple imputation (Supplementary Appendix S3). Because the

substantive findings were unchanged (Supplementary Table S4), results from the raw data are presented.

Results

Descriptive statistics and correlations between study variables are in Supplementary Tables S2 and S3. Bivariate correlations showed that P3 amplitudes were negatively associated with aggression but not significantly correlated with ADHD symptoms (Supplementary Table S3).

Next, we examined the within- and between-child associations between P3 amplitudes and externalizing problems using HLM growth curve models. HLM growth curve model results are in Table 2. The child's mean P3 amplitude across time was negatively associated with their intercepts (γ_{02} : $B = -0.30$, $p = .039$), but not their slopes, of aggression. Within-individual changes in the P3 amplitude were negatively associated with within-child changes in aggression (γ_{20} : $B = -0.31$, $p = .004$). Findings suggest that smaller P3 amplitudes (relative to one's mean) were concurrently associated with more aggression (relative to one's level of aggression at other time points) above and beyond one's linear slope of aggression. Findings held even accounting for ADHD symptoms (Supplementary Table S5). The child's mean P3 amplitude across time was not significantly associated with their intercepts (γ_{02}) or slopes (γ_{12}) of ADHD symptoms. Within-child changes in P3 amplitudes were not significantly associated with within-child changes in ADHD symptoms (γ_{20} : $B = -0.06$, $p = .12$).

Discussion

The present study examined the longitudinal, within-person association between P3 ERP amplitudes and parent-reported externalizing problems in very young children. Our findings suggest that smaller mean P3 amplitudes across ages 30 to 42 months were associated with higher levels of aggression at 30 months of age. Additionally, findings suggest that within-child changes in the P3 amplitude were negatively associated with concomitant within-child changes in aggression. When children showed smaller P3 amplitudes (relative to their own mean level), they showed more concurrent aggression. However, within-child changes in P3 amplitudes were not significantly associated with ADHD symptoms.

Our findings of an association between smaller P3 amplitudes and aggression are consistent with prior meta-analyses examining externalizing problems (Gao & Raine, 2009; Pasion et al., 2018) and with conceptualizations of the P3 as an endophenotype of externalizing problems (Iacono & Malone, 2011). The auditory P3 amplitude increases across childhood (van Dinteren et al., 2014), and is considered an index of attentional orienting (Friedman et al., 2001).

Although the P3 amplitude is often found to be smaller in children with ADHD, as compared to controls (e.g., Tsai et al., 2012), contradictory findings have also emerged (e.g., Rydkær et al., 2017). Yoon and colleagues (2008) found that children with ADHD and a comorbid externalizing disorder (oppositional defiant disorder or conduct disorder) had smaller P3 amplitudes, whereas children with ADHD alone did not show such an effect.

These findings suggest that the smaller P3 amplitude typically noted in children with ADHD might actually reflect comorbid externalizing problems. Moreover, to our knowledge, no studies that identified an association between P3 amplitudes and ADHD examined whether within-individual changes in P3 were associated with ADHD. Our findings align with findings of prior studies that the smaller P3 amplitude in children with externalizing problems is not due to ADHD symptoms (Baving, Rellum, Laucht, & Schmidt, 2006). Thus, evidence does not support a causal interpretation of an association between the P3 and ADHD; prior findings of an association between the P3 and ADHD could reflect their common association with a third variable (e.g., broad factor of externalizing problems). This finding could reflect an important developmental fact about the meaning of the P3 in the oddball task, or less within-individual variation in ADHD symptoms than in aggression across 30 to 42 months (Supplementary Appendix S3). Or perhaps attention problems are a less coherent or stable construct in very early development. Or the finding could reflect measurement issues such as slightly weaker reliability of the shorter ADHD scale compared to the longer aggression scale.

By contrast, the within-individual association between changes in P3 amplitude and aggression provides stronger evidence consistent with a causal association, even though we cannot eliminate the possibility of time-varying confounds or the reverse direction of effect. How might a smaller P3 amplitude be involved in development of aggression? First, it is important to note that a smaller P3 amplitude may reflect not just under-processing of relevant information, but also over-processing of irrelevant information (Hermens et al., 2010), which could impair higher-order processes related to detecting and responding to subtle environmental and social cues. Within a social information processing framework, under-processing of relevant information (e.g., cues of safety) and over-processing of irrelevant information (e.g., ambiguous cues perceived as indicating threat) hypothetically could affect the first stage of social information processing, encoding of social cues. Altered encoding could, in turn, influence downstream attributions, making it more likely that individuals interpret ambiguous cues as hostile, and respond with aggression. Within a social information processing framework, impaired attention and encoding processes could explain, at a basic stimulus processing level, why individuals with smaller P3 amplitudes show more aggression, particularly reactive (as opposed to proactive) aggression. This is consistent with findings showing smaller P3 amplitudes in impulsive aggression, but not premeditated aggression (for a review, see Patrick, 2008). For instance, children with poorer novelty detection (e.g., smaller P3 amplitudes), may miss key changes in others' voice tone in daily interactions (Hoyniak et al., 2018), leading to agonistic conflicts with others, which in turn lead to future assumptions about hostile intent and negatively biased social information processing.

The present study had several key strengths. First, we examined the P3 ERP in very young children, an important group with high theoretical relevance for understanding how externalizing problems develop. Based on the morphology and topography of the P3, evidence suggests the P3 component elicited in the present study may correspond to the P3 elicited from older subjects. Our findings contribute to a relatively sparse literature focusing on ERPs elicited during toddlerhood. Second, the study was longitudinal with repeated measures of both the P3 and behavior problems. The repeated measures design allowed us to

examine whether within-child changes in P3 amplitude predicted concomitant within-child changes in behavior problems. We believe this is the first study to examine the within-individual association between the P3 and externalizing problems, providing a stronger test of causality.

The present study also had limitations. First, because of the correlational nature of the design, and the many likely determinants of psychological development, we cannot make definitive causal inferences. Next, our sample was predominantly middle class, which may limit generalizability of our findings. We hope to see future studies with more broadly representative as well as higher risk samples and the use of informants beyond parents. The extent of longitudinal ERP missingness warrants caution in interpreting our findings. Despite its clinical nonspecificity, the P3 may have transdiagnostic relevance. An interesting further question would be how the P3 relates in early childhood to additional dimensions of behavior problems, including internalizing and thought-disordered problems, given findings that the P3 is also associated with depression and schizophrenia (Turetsky et al., 2015).

Conclusion

The present longitudinal study is the first investigation of the *within*-individual association between the P3 and externalizing problems, which provides a stronger test of causality than previous studies of between-subjects effects. Findings indicate that children's within-individual changes in the P3 amplitude predicted concomitant within-child changes in their aggression but not ADHD symptoms. Importantly, this association was present in toddlerhood, an era when early targeted intervention efforts may efficiently prevent later, severe externalizing problems. Findings are consistent with the notion that the P3 may play a role in development of aggression. They are not consistent with the P3 playing a causal role in development of ADHD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Key points:

1. Previous research suggests that externalizing problems are characterized by attentional impairments and smaller P3 amplitudes.
2. Prior work has been mainly cross-sectional, has not examined young children, and has not examined whether *within*-individual changes in P3 amplitude predict changes in externalizing behavior.
3. Our findings indicated that children's within-individual changes in the P3 amplitude predicted concomitant within-child changes in their aggression but not ADHD symptoms.
4. Findings support the interpretation that the P3 may play a role in development of aggression. They do not support such a role in development of ADHD.

Figure 1. Children's grand-averaged waveforms for frequent and infrequent trials during the oddball task. The waveform depicted represents the mean waveform from those electrodes with a 0.6 or greater factor loading onto the PCA component reflecting the P3. For purposes of depicting waveforms, electrodes from the PCA-derived posterior/parietal electrode cluster (see Supplementary Figure S1) were averaged with equal, unit weighting. However, actual P3 amplitudes were calculated using PCA.

Table 1.

Sample characteristics.

Variable	<i>n</i>	%
Sex		
Males	83	54
Females	70	46
All	153	
Parent Ethnicity		
Non-Hispanic Caucasian	138	90
Hispanic	4	3
African-American	5	3
Asian-American	5	3
Mixed Race	1	< 1
Parent Education		
Some high school	1	< 1
GED	2	1
High school diploma	2	1
Some college	16	11
College degree	131	86
Marital status		
Single	10	7
Married	135	89
Divorced	6	4
Variable	<i>M</i>	<i>SD</i>
Child Age (months)	36.00	4.90
Parent Age (years)	33.26	4.85
Family SES	48.99	13.27

Note: one child was missing information on her parent's education, and two were missing parental marital status. Family socioeconomic status (SES) was calculated using the Hollingshead (1975) index.

Table 2.

Results of HLM growth curve models.

Outcome: Aggression	<i>B</i>	<i>β</i>	<i>SE</i>	<i>df</i>	<i>p</i>
intercept	15.52	0.01	2.27	142	<.001
Time	−0.11	0.00	0.13	90	.371
Sex	−1.07	−0.07	1.00	142	.288
Sex × Time	0.06	0.03	0.12	90	.607
Mean P3 amplitude	−0.30	−0.13	0.15	142	.039
Mean P3 amplitude × Time	0.02	0.05	0.02	90	.370
*Time-varying P3 amplitude	−0.31	−0.12	0.10	90	.004
*Number of bad channels	−0.09	−0.06	0.09	90	.306
*Number of infrequent trials kept	−0.13	−0.14	0.06	90	.022
Outcome: ADHD symptoms	<i>B</i>	<i>β</i>	<i>SE</i>	<i>df</i>	<i>p</i>
intercept	3.30	0.03	0.75	142	<.001
Time	−0.06	−0.07	0.04	90	.126
Sex	−0.66	−0.08	0.35	142	.064
Sex × Time	0.08	0.11	0.04	90	.053
Mean P3 amplitude	−0.03	−0.05	0.05	142	.538
Mean P3 amplitude × Time	0.00	0.01	0.01	90	.878
*Time-varying P3 amplitude	−0.06	−0.07	0.04	90	.122
*Number of bad channels	−0.01	−0.01	0.03	90	.849
*Number of infrequent trials kept	−0.01	−0.04	0.02	90	.546

Note: “Time” reflects the slope term, and is centered at the first time point so that the intercept reflects the child’s level at 30 months (i.e., 0, 6, 12 months from 30 months). “Sex” is coded with female = 1, male = 0. “Mean P3 amplitude” refers to a given child’s mean P3 amplitude across time (time invariant). “Time-varying P3 amplitude” refers to a given child’s P3 amplitude at a given time point that is centered around their mean P3 amplitude across time (time varying). Interaction terms with time essentially reflect the prediction of slopes of the outcome (e.g., “Sex × Time” reflects sex predicting slopes of the outcome). Asterisks reflect time-varying terms. Terms in bold reflect significant associations at $p < .05$ level.