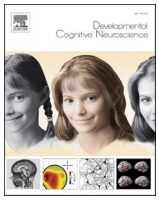
Developmental Cognitive Neuroscience 60 (2023) 101212

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn



Influences of age and pubertal development on P300 amplitude trajectory across two years in female adolescents

Nicholas J. Santopetro [a,*], C.J. Brush [a,b], Elizabeth M. Mulligan [a], Greg Hajcak [a]

[a] Department of Psychology, Florida State University, Tallahassee, FL, USA
[b] Department of Movement Sciences, University of Idaho, Moscow, ID, USA

ARTICLE INFO

Keywords:
ERP
Prospective
P300
Age
Puberty
Adolescence

ABSTRACT

The P300 event-related potential (ERP) has been extensively studied across the human lifespan. However, many studies examining age-related effects are cross-sectional, and few have considered the unique role that pubertal development may have on P300 developmental trajectories. The current study examined whether age, pubertal maturation or their interaction predicted changes in P300 amplitude over two years among 129 females between the ages of 8 and 15 years at baseline. Participants completed a flanker task while EEG was recorded at a baseline and two-year follow-up visit. Both baseline age and increased pubertal development were associated with smaller P300 amplitude at follow-up. However, the influence of age was qualified by an interaction between age and pubertal maturation: among younger girls only, increased pubertal development predicted decreases in P300, whereas decreased pubertal development predicted increases in P300. These data indicate that pubertal timing impacts neurodevelopmental changes in P300 amplitude – such that high versus low pubertal development among 8- to 10-year-old girls predicted differential trajectories of neural activity. In light of links between reduced P300 and mental health disorders, such as depression, future studies might examine whether neurodevelopmental changes influenced by early-onset pubertal development could account for increases in these mental health problems.

1. Introduction

The trajectory of cognitive functioning across the human lifespan has been commonly examined via neuropsychological tests (e.g., Wechsler Adult Intelligence Scale [WAIS], Wechsler, 1997a; Wechsler Memory Scale [WMS], Wechsler, 1997b), with research showing steady improvements in most cognitive abilities from childhood to early adulthood; in adulthood, these functions typically peak and then begin to decline (Hartshorne and Germine, 2015). More specifically, it is well established that children undergo critical cognitive, as well as social, emotional, and physical, changes during adolescence resulting in more effective cognitive abilities such as increased executive functioning and decision-making capabilities (Anderson, 2001). However, our current understanding of the underlying neurophysiological mechanisms associated with such changes in cognitive abilities during adolescence is quite limited.

Event-related potentials (ERPs), measured via electroencephalogram (EEG), are direct measures of brain activity recorded from the scalp during experimental paradigms. These brain potentials are non-invasive, and relatively inexpensive and rapid to acquire—especially in relation to other neural measures (e.g., fMRI; Hajcak, Klawohn and Meyer, 2019). The P300 ERP component is a positive deflection that peaks approximately 300 ms after stimulus presentation (i.e., auditory or visual) at midline parietal recording sites (e.g., Pz; Polich, 2012). Researchers have conceptualized the P300 in terms of information processing functions that include attention allocation, memory, and inhibitory control (Polich, 2012). The P300 has been investigated in past research to better understand the psychophysiology of these cognitive functions in relation to different areas of mental health such as aging, depression, substance use disorders, and schizophrenia (e.g., Bashore, 1990; Polich, and Luckritz, 1995; Carlson, McLarnon, and Iacono, 2007; Bauer, O'Connor, and Hesselbrock, 1994; Li et al., 2019; Bachiller et al., 2015; Bruder et al., 2012).

A recent review and meta-analysis examined how *auditory* P300 amplitude changes across the human lifespan. Consistent with findings that employed neuropsychological assessments of cognitive functioning (Hartshorne and Germine, 2015), researchers found that auditory P300 amplitude also appears to increase during childhood, plateau in late

* Correspondence to: Florida State University, 1107 West Call Street, Tallahassee, FL 32304, USA.
E-mail address: nicholas.santopetro@gmail.com (N.J. Santopetro).

https://doi.org/10.1016/j.dcn.2023.101212
Received 10 April 2022; Received in revised form 12 December 2022; Accepted 6 February 2023
Available online 8 February 2023

adolescence/early adulthood, and gradually decrease throughout life (van Dinteren et al., 2014). However, researchers find a slightly different trajectory regarding the *visual* P300 component as visual P300 amplitude appears to decrease as children age, which may be associated with more efficient processing of visual experimental stimuli requiring less cognitive resources (Courchesne, 1978; Katsanis et al., 1996; Hill et al., 1999; Batty and Taylor 2002; Carlson and Iacono, 2006; Riggins and Scott, 2020; Santopetro et al., 2021). In general, the vast majority of existing research on P300 amplitude and age is cross-sectional, and few studies investigate prospective changes in P300 amplitude (auditory or visual), which requires longitudinal study designs. Lastly, neurodevelopmental studies in younger populations typically utilize chronological age as a proxy for development status and do not consider other factors that are potentially influencing these changes in neural activity during this critical developmental period such as pubertal maturation.

Although pubertal development and age are highly interrelated variables, they are separate constructs and very few researchers have directly investigated these two variables concurrently in relation to P300 amplitude during childhood or adolescence. To date, only one study by Brumback and colleagues examined the cross-sectional relationships between age, pubertal status, and P300 amplitude elicited from a *visual* oddball task in a sample of 99 participants between the ages of 8 and 13 years (Brumback et al., 2012). In this study, the authors found that individuals in more advanced stages of puberty and older participants were both characterized by reductions in visual P300 amplitude. However, when placed in the same multiple linear regression model predicting P300 amplitude, neither age nor pubertal development uniquely related to P300, suggesting that they shared overlapping variance in the P300. These data may suggest that factors other than an individual's current level of pubertal development need to be investigated, such as pubertal timing and/or pubertal tempo, which consider pubertal status in different contexts. More specifically, pubertal timing is defined as a measure of whether pubertal development is occurring earlier, at the same time, or later than same-aged peers, while pubertal tempo refers to an individual's general rate of pubertal development (gradual or sudden developments). Moreover, it remains unknown if pubertal development, or specifically timing and/or tempo, impacts subsequent changes in P300 amplitude during childhood and adolescence.

In particular, pubertal timing may be a crucial factor to consider as past studies suggest that early pubertal timing (i.e., experiencing pubertal changes sooner than same-aged peers) has been inversely linked to cognitive abilities in adulthood (Beltz and Berenbaum, 2013), broad impairments in adjustment and functioning (Graber et al., 2004), and increased risk for developing mental health disorders such as substance use disorders and depressive disorders (Dick et al., 2000; Angold et al., 1998). Moreover, the relationship between pubertal timing and development of attentional processes is not fully understood as past studies have shown mixed results. Early pubertal timing has been linked to *worse* performance on task assessing attention and executive function (Stumper et al., 2020); however, there is also evidence suggesting that individuals who undergo earlier pubertal development *decrease* in attentional problems over time compared to similar-aged peers that are not as developed (Laitinen-Krispijn et al., 1999). To our knowledge, the relationship between pubertal timing and P300 amplitude during childhood or adolescence has never been examined which could potentially further our understanding of the exact neurophysiological changes linked to early pubertal timing and future cognitive and/or psychosocial functioning.

Therefore, the present study primarily sought to examine the effects of age and pubertal development on P300 amplitude change over two years in a large sample of adolescent females; as a secondary aim, we also examined the effects of age and pubertal development on changes in behavioral performance measures (i.e., error rate and reaction time) derived from the flanker task over two years. Participants in the current study completed self-report questionnaires including their current pubertal development and age, as well as a flanker task while EEG was recorded at a baseline assessment. Approximately two years later, the participants returned for a follow-up visit and completed the flanker task again while EEG was recorded. Based on past findings (Courchesne, 1978; Katsanis et al., 1996; Hill et al., 1999; Batty and Taylor, 2002; Carlson and Iacono, 2006; Riggins and Scott, 2020; Santopetro et al., 2021; Brumback et al., 2012), we predicted that both pubertal development scores and age would be significantly associated to baseline and follow-up P300 amplitude at the zero-order, such that older individuals and individuals higher in pubertal development would exhibit blunted P300 amplitude at both time points. Furthermore, we believe that baseline age and pubertal status would uniquely predict change in P300 amplitude over two years such that both older and more developed females at baseline would experience greater decreases in P300 amplitude. Considering the lack of existing research involving pubertal timing and P300 development in children and adolescents, we did not have any specific hypothesis regarding potential age and pubertal development interaction predicting concurrent P300 amplitude or change in P300 amplitude over two years.

2. Method

2.1. Participants

The current study was part of a larger longitudinal investigation of adolescent females recruited from the Long Island, New York area. Families were recruited via mailing lists, word-of-mouth, and other advertisements. A total of 317 adolescent females aged 8- to 15-years old, accompanied by a biological parent or legal guardian, participated in a baseline visit and were invited to participate in a follow-up visit, which occurred approximately two years later (M = 2.04 years between visits, SD = 0.19). Participants completed tasks while electroencephalogram (EEG) was recorded, as well as self-report questionnaires and clinical interviews at each visit. Participants were included in the study if they had the ability to read and comprehend questionnaires, were fluent in English, and if there was no parent-reported presence of an intellectual disability. Parents and adolescents provided consent and assent prior to completing study procedures, and families were compensated for their time (i.e., approximately $20 per hour). The study was approved by the Institutional Review Board at Stony Brook University.

The current study is a secondary data analysis of EEG data collected during a flanker task (Eriksen and Eriksen, 1974) from both the baseline visit and the two-year follow-up visits, as well as self-report data collected from the baseline visit (i.e., Pubertal Development Scale [PDS] score and age). Previous publications from this sample using the flanker task focused on response-monitoring ERPs (i.e., error-related negativity [ERN]; correct response negativity [CRN]; Meyer et al., 2018; Chong et al., 2020; Meyer et al., 2021) as well as examining the prospective relationship between baseline stimulus-locked neural activity (i.e., P300) and change in depressive symptoms over two years (Santopetro et al., 2020).

Of the original 317 participants recruited at baseline, 251 participants completed the flanker task at the baseline visit. Of these 251 participants, 155 returned two years later to complete the flanker task again at the follow-up visit. A total of 17 participants completed an "adaptive" version of the flanker task at the follow-up visit in which the task ended once the participant made approximately 20 errors; thus these 17 participants were excluded from the current study as they did not complete the same exact flanker task as they did at their baseline assessment. Additionally, four participants were excluded for poor baseline EEG data and three additional participants were excluded for poor follow-up EEG data based on visual inspection. Lastly, one participant did not complete the PDS at baseline and one participant had a follow-up P300 value that was more than 3 SD above the mean. Thus, the current study's final sample size was 129 (M = 11.66 years, SD =

1.84; baseline). The final sample was majority White ($n = 109$, 86.5%), with the remainder of adolescents identifying as either African American ($n = 7$, 5.6%), Hispanic ($n = 6$, 4.8%) or other ($n = 4$, 3.2%).[1]

2.2. Measures

2.2.1. Pubertal Development Scale (PDS)

The PDS is a five-item self-report scale that assesses development based on five common indices of pubertal growth in males and females (Petersen et al., 1988). All participants are asked about growth, body hair, and skin changes (e.g., acne). However, some items differ slightly depending on the participant's biological sex. Males are asked about changes to their voice and growth of facial hair, while females are asked about breast development and age of onset for menstruation. Besides menstruation status, every item is scored on a 4-point scale; not yet started (1), barely started (2), definitely started (3), and seems complete (4). Menstruation status is coded dichotomously: no (1) or yes (4). Therefore, the PDS total score ranges from 5 to 20. In the current sample, the PDS total score demonstrated good internal consistency ($\alpha = 0.83$).

Past studies have created a PDS category score to provide more context regarding the current stage of development participants were undergoing (Carskadon and Acebo, 1993; Herting et al., 2021). This category score is calculated using only three items in females (i.e., body hair, breast development, and menarche status) and is defined as: pre-pubertal = 2 and no menarche; early pubertal = 3 and no menarche; mid-pubertal = >3 and no menarche; late pubertal = <7 and menarche; and post-pubertal = 8 and menarche. At baseline in the present sample, 5.4% of the sample was pre-pubertal, 11.6% was early pubertal, 59.7% was mid-pubertal, 16.3% was late pubertal and 7% was post-pubertal.

2.3. Procedures

2.3.1. Flanker task

Participants completed a horizontal arrowhead version of the flanker task at the baseline and follow-up visits. The task was administered using Presentation software (Neurobehavioral Systems, Albany, California). Each trial consisted of five arrowheads presented for 200 ms, and the inter-trial interval (ITI) varied from 2300 ms to 2800 ms. Half of the trials were congruent (< < < < <, > > > > >), and the other half were incongruent (< < > < <, > > < > >), and the order of trials was random. The individual arrowheads are 1.5-cm long and the spacing between the arrowheads is 0.5 cm. Participants were instructed to try to respond as quickly and accurately as possible to the direction of the center arrow using the computer mouse. For example, if the central arrow was pointing to the right, participants were instructed to click the right button on the mouse. The full task consisted of 11 blocks, each consisting of 30 trials (330 trials total). Participants received feedback at the end of each block depending on how well they were performing. If accuracy was low (i.e., 75% or below), they would receive the message, "Please try to be more accurate." If accuracy was high (i.e., 90% or above), they would receive the message, "Please try to respond faster." Otherwise, they received the message, "You're doing a great job." Error rates at baseline and follow-up were calculated as the percentage of errors in relation to total responses for each participant.

2.3.2. EEG recording and processing

The continuous EEG was recorded during the flanker task using a 34 channel (sintered Ag/AgCl) ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). Electrodes were placed on the cap following the international 10/20 system. Two additional electrodes were also placed on both the left and right mastoid located behind the ears. Electrooculogram (EOG) was recorded from two electrodes placed approximately 1 cm above and below the right eye, and two approximately 1 cm to the side of each eye on the outer canthus. A common mode sense (CMS) active electrode served as the online recording reference. Data were digitized at a sampling rate of 1024 Hz, utilizing a low-pass fifth-order sinc filter with a half-power cutoff set at 204 Hz.

Offline analysis was conducted using Brain Vision Analyzer 2.2 (Brain Products, Gilching, Germany). First, all EEG data was re-referenced to the mastoid electrodes (TP9 and TP10). Next, data were filtered with low and high filter cutoffs set at 0.1 Hz and 30 Hz, respectively. Next, ocular corrections were made using the Gratton & Coles (1983) method. The continuous EEG data were then epoched to create stimulus-locked segments only on trials in which the participant responded correctly, starting 200 ms before stimulus onset (collapsed across congruent and incongruent stimuli) [2] and continuing for 1000 ms. Next, automatic artifact rejection was implemented to reject segments with a voltage step greater than 50 μV between sampling points, a voltage difference of 175 μV within 600 ms intervals, or a maximum voltage difference of less than 0.5 μV within 100 ms intervals. The epochs were baseline-corrected using the 200 ms pre-stimulus interval and averaged across all correct trials. The P300 was quantified as the mean amplitude at electrode site Pz from 400 ms to 600 ms following stimuli (collapsed across congruent and incongruent stimuli). Fig. 1 shows both the baseline and two-year follow-up stimulus-locked grand average waveforms and head maps of participants. The correlation between P300 amplitude on odd and even trials was examined to measure internal consistency (Levinson et al., 2017), which was then corrected using Spearman-Brown prophecy formula (Nunnally et al., 1967). The P300 exhibited excellent internal consistency at baseline (Spearman--Brown corrected $r = 0.96$) and follow-up (Spearman-Brown corrected $r = 0.98$). Lastly, flanker P300 amplitude demonstrated fair test-retest reliability over a two-year period (Pearson's $r = 0.51$, $p < .001$).

2.3.3. Statistical analyses

All statistical tests were conducted using $\alpha = 0.05$ (two-tailed) to denote significance, all data analyses were performed using SPSS Statistics software (Version 25.0; IBM Corp., Armonk, N.Y., USA), and the Johnson-Neyman analyses were conducted via the PROCESS macro for SPSS (Preacher and Hayes, 2008; Hayes, 2013). The Johnson-Neyman approach denotes all values of the moderator in which the simple slopes of the predictor variable are significant or not (i.e., regions of significance; Johnson and Fay, 1950). First, bivariate correlations (Pearson's *r*) were conducted between all baseline measures (P300, error rate, reaction time, PDS total score, age), and follow-up measures (P300, error rate, reaction time). Considering the number of statistical tests being conducted for each variable, all of the original correlation *p*-values were corrected using the false discovery rate (FDR) method. Next, a multiple linear regression was conducted to assess the *cross--sectional* influence of pubertal timing on P300 amplitude. This multiple linear regression used baseline P300 amplitude as the criterion, which was predicted from baseline pubertal development (centered), baseline age (centered), and their interaction (proxy for pubertal timing). Next, to determine whether baseline pubertal development, baseline age, and/or their interaction (proxy for pubertal timing) were *unique* predictors of P300 amplitude and/or flanker behavioral measure (error rate, reaction time) *changes over two years*, we conducted three separate hierarchical multiple linear regressions (predicting P300, error rate, or reaction time) each consisting of two steps. At step 1, baseline age

[1] Three participants did not endorse their race or ethnicity.

[2] At both baseline and follow-up visits, P300 amplitude elicited on incongruent trials (*T1*: 16.16 μV [*SD* = 6.60], *T2*: 16.76 μV [*SD* = 7.21]) was significantly larger than P300 amplitude elicited on congruent trials (*T1*: 13.24 μV [*SD* = 6.32], *T2*: 12.77 μV [*SD* = 6.87]), $t(128) > 8.81$, *p*-values < 0.001. However, results in the current project are consistent when utilizing either incongruent or congruent elicited P300 amplitudes (more detailed information in the Results section).

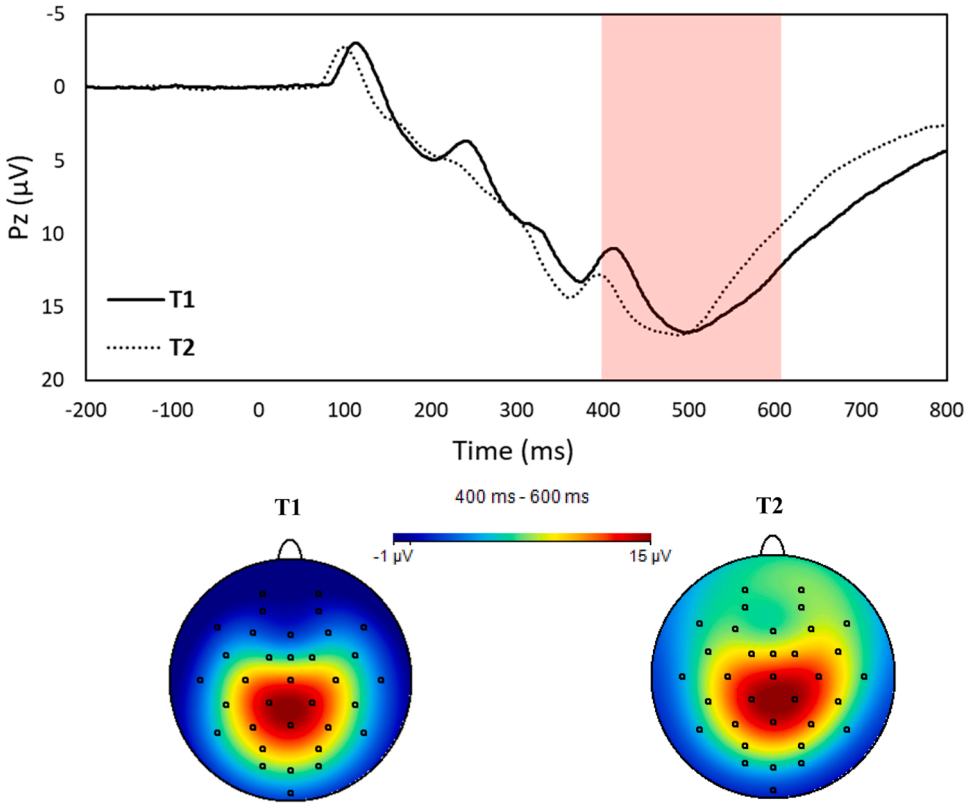


Fig. 1. Baseline (T1) and two-year follow-up (T2) stimulus-locked waveforms and head maps (n = 129).

(uncentered), baseline PDS total score (uncentered), and baseline P300 amplitude/behavioral measures (uncentered) were included as predictors of follow-up P300 amplitude/behavioral measures. At step 2, we included a baseline age x baseline PDS total score interaction term as an additional predictor along with mean-centered lower order terms. To decompose significant interactions, simple effects coefficients of the baseline age and follow-up P300 amplitude/behavioral measures relationship were computed at three different levels of baseline PDS total score (1-*SD* below the mean, at the mean, and 1-*SD* above the mean). Lastly, to further interpret results from the potential baseline age and baseline PDS total score interaction with change in P300 amplitude/-behavioral measures over two years, the Johnson-Neyman technique was utilized to determine at what specific age ranges the effect of baseline pubertal status on P300 amplitude/behavioral measures trajectory was significant (all variables uncentered to retain the variables' original scaling).

3. Results

3.1. Zero-order correlations

Means, standard deviations, and intercorrelations of all variables can be found in Table 1. At baseline, neither pubertal development ($p = .091$) nor age ($p = .717$) appeared to be significantly associated with P300 amplitude. Baseline task accuracy was not significantly associated with pubertal development ($p = .072$), but older participants did make fewer errors at baseline. Additionally, both older and more pubertally developed adolescents responded faster during the flanker task at baseline. Baseline P300 amplitude was not significantly associated with baseline error rate or reaction times during the task.

Older adolescents and adolescents reporting more advanced pubertal development at baseline exhibited reduced flanker P300 amplitude at follow-up. Again, follow-up flanker task accuracy was not significantly associated with baseline pubertal development ($p = .055$), but older participants made fewer errors at the follow-up assessment. Both older and more pubertally developed adolescents at baseline responded faster during the flanker task at follow-up. Furthermore, none of the follow-up behavioral measures (i.e., error rate or reaction time) were significantly associated with follow-up P300 amplitude. Fig. 2 displays the association between age and residual change in P300 amplitude over two years.[3]

3.2. Multiple linear regressions: P300 amplitude

3.2.1. Cross-sectional analysis

First, a multiple linear regression was conducted predicting baseline P300 amplitude employing baseline PDS total score, baseline age, and their interaction as independent variables to investigate if pubertal timing influenced current P300 amplitude. This overall model was not significant, $F(3, 125) = 2.37$, $p = .073$, and neither was the two-way interaction, $b = -0.07$, $t(128) = -0.89$, $p = .375$, 95% CI [− 0.23, 0.09].

[3] Flanker congruency did not appear to affect the relationship between baseline P300 amplitude and baseline pubertal development: increased pubertal development at baseline was significantly associated with reduced baseline P300 amplitude on congruent trials, $r(127) = -0.20$, $p = .021$, and trended significance on incongruent trials, $r(127) = -0.16$, $p = .080$ (Steiger's z-test: $z = -0.78$, $p = .784$). Again, flanker congruency did not appear to affect the relationship between follow-up P300 amplitude and baseline pubertal development or age: increased pubertal development/being older at baseline was significantly associated with reduced follow-up P300 amplitude on congruent trials, $r(127) = -0.38$, $p < .001$ and, $r(127) = -0.36$, $p < .001$, respectively, and on incongruent trials, $r(127) = -0.33$, $p < .001$, and, $r(127) = -0.29$, $p = .001$, respectively (Steiger's z-test: z's < -1.78, p's > 0.962).

Table 1
Flanker behavioral and P300 measures correlations with self-report measures.

Variable	*M*	*SD*	*Min-Max*	1	2	3	4	5	6	7	8
1. T1 P300 (μV)	14.57	6.19	2.07–32.29	-							
2. T2 P300 (μV)	14.12	6.78	2.07–39.01	0.51 * **	-						
3. T1 correct RT (ms)	494	115	316–880	-0.03	0.15	-					
4. T1 error rate (%)	16.18	9.28	2.12–48.16	-0.15	-0.09	-0.02	-				
5. T2 correct RT (ms)	408	70	297–697	-0.12	0.01	0.67 * **	0.11	-			
6. T2 error rate (%)	15.18	9.37	1.52–59.75	-0.18†	0.00	0.13	0.47 * **	-0.13	-		
7. T1 age (Years)	11.66	1.84	8–15	-0.03	-0.35 * **	-0.56 * **	-0.27 * *	-0.36 * **	-0.29 * *	-	
8. T1 PDS total	12.73	4.18	5–20	-0.19†	-0.36 * **	-0.41 * **	-0.19†	-0.27 * *	-0.19†	0.66 * **	-

Note. PDS represents the Pubertal Development Scale total score. T1 represents baseline assessment. T2 represents two-year follow-up assessment. RT represents reaction time. All *p*-values are FDR corrected. † indicates $p < .10$. * indicates $p < .05$. * * indicates $p < .01$. * ** indicates $p < .001$

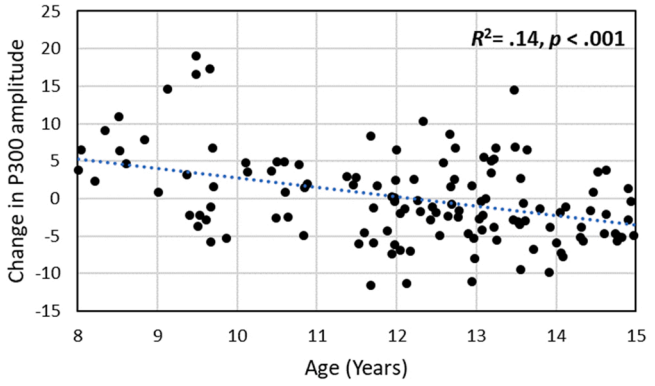


Fig. 2. *Association between age and change in P300 amplitude over two years (n = 129). Note.* Change in P300 amplitude was computed by saving the unstandardized residuals in a linear regression that predicted two-year follow-up flanker P300 amplitude from baseline flanker P300 amplitude.

3.3. Prospective analysis

Detailed information of the two-step hierarchical multiple linear regression analysis is presented in Table 2. At step one, baseline P300 amplitude, baseline age, and baseline PDS total score were entered as independent variables to predict follow-up P300 amplitude. A larger baseline P300 amplitude and younger age at baseline were significantly associated with a larger follow-up P300 amplitude; however, baseline PDS total score was not a significant predictor of follow-up P300 amplitude.

At step two, the baseline age x baseline PDS total score product term was entered as a predictor of follow-up P300 amplitude. At this step, a larger baseline P300 amplitude and younger age at baseline remained significantly associated with a larger follow-up P300 amplitude, while the main effect of baseline PDS total score was unrelated to follow-up P300 amplitude. However, there was a significant interaction between baseline age and baseline PDS total score, indicating that the association between baseline age and P300 amplitude over two years was evident only in adolescents lower in pubertal development at baseline.[4] That is, younger adolescents who were less pubertally developed (−1 *SD*), $b = -1.45$, $p < .001$, or experiencing average pubertal development, $b = -0.85$, $p = .020$, had *increases* in P300 amplitude over two years, while younger adolescents with larger baseline PDS total scores (+1 *SD*), $b = -0.26$, $p = .617$, experienced *decreases* in P300 amplitude (see Fig. 3). It is important to highlight that the y-axis of Fig. 3 is a residualized change score in P300 amplitude from baseline to two-year follow-up meaning that a more positive value represents P300 amplitude growth while a negative value represents reductions in P300 amplitude. The inclusion of the interaction term accounted for significantly more variance (ΔX^2 between step 1 and step 2 = 3.99, $p = .048$), with an additional 2% of variance explained in follow-up P300 amplitude with the addition of the interaction term.

Consistent with these follow-up analyses, the Johnson-Neyman interval indicated that the association between baseline PDS total scores

Table 2
Multiple linear regressions results using two-year flanker P300 amplitude as the criterion (n = 129).

Predictor	*b*	*b* 95% CI [LL, UL]	*β*	*β* 95% CI [LL, UL]	*p*-value	Fit
Step 1						
T1 P300	0.53	[0.37, 0.69]	0.48	[0.34, 0.63]	< 0.001	
T1 Age	-1.00	[− 1.70, − 0.31]	-0.27	[− 0.46, − 0.09]	0.005	
T1 PDS	-0.14	[− 0.45, 0.17]	-0.09	[− 0.28, 0.11]	0.378	
						$R^2 = .371$, $p < .001$ (adjusted $R^2 = .355$, $p < .001$) 95% CI [0.23,0.47]
Step 2						
T1 P300	0.54	[0.39, 0.70]	0.49	[0.35, 0.64]	< 0.001	
T1 Age	-0.85	[− 1.55, − 0.15]	-0.23	[− 0.42, − 0.04]	0.018	
T1 PDS	-0.19	[− 0.50, 0.12]	-0.11	[− 0.31, 0.08]	0.239	
T1 Age x PDS	0.14	[0.00, 0.28]	0.14	[0.00, 0.29]	0.048	
						$R^2 = .390$, $p < .001$ (adjusted $R^2 = .371$, $p < .001$) 95% CI [0.24,0.48]

Note. PDS represents the Pubertal Development Scale total score. T1 represents baseline assessment. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *β* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

[4] We conducted a multiple regression model using the P300 gain score (i.e., follow-up P300 minus baseline P300) over two years as the dependent variable to determine if the results were consistent with the primary analysis. The overall model was significant, $F(3125) = 6.98$, $p < .001$, $R^2 = .14$. There was also a significant interaction between baseline age and baseline PDS total score, $b = 0.18$, $t(128) = 2.20$, $p = .030$, 95% CI [0.02, 0.33]), such that younger adolescents who were less pubertally developed (−1 *SD*), $b = -1.78$, $p < .001$, and younger adolescents with mean pubertal development, $b = -1.05$, $p = .009$, had *increases* in P300 amplitude over two years. Younger participants with larger baseline PDS total scores (+1 *SD*), $b = -0.32$, $p = .578$, experienced *decreases* in P300 amplitude.

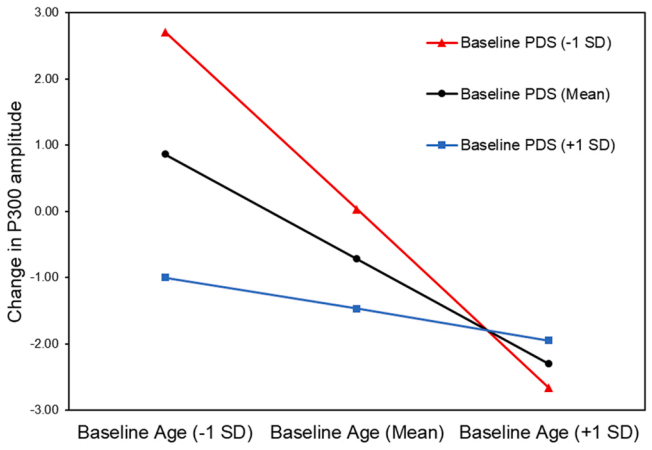


Fig. 3. *Two-way interaction between baseline age and baseline PDS total score in predicting change in flanker P300 amplitude over two years (n = 129). Note.* PDS represents baseline Pubertal Development Scale total score. Change in P300 amplitude was computed by saving the unstandardized residuals in a linear regression that predicted two-year follow-up flanker P300 amplitude from baseline flanker P300 amplitude. A more positive change value represents P300 amplitude growth while a negative value represents reductions in P300 amplitude. Compared to Fig. 2, the current figure does not present each individuals' change in P300 score which reduces the scale of the y-axis.

and follow-up P300 amplitude was significant only for participants 10.13 years or younger (i.e., uncentered raw values; reflecting 27.13% of the total sample); the relationship between baseline PDS total score and follow-up P300 amplitude was not significant for adolescents older than 10.13 years (Fig. 4).

3.4. Multiple linear regressions: error rate

3.4.1. Prospective analysis

Detailed information of the two-step hierarchical multiple linear regression analysis is presented in Table 3. At step one, baseline error rate, baseline age, and baseline PDS total score were entered as independent variables to predict follow-up error rate. Higher baseline error rate was significantly associated with a higher follow-up error rate; however, neither baseline age nor baseline PDS total score were significant predictors of follow-up error rate.

At step two, the baseline age x baseline PDS total score product term was entered as a predictor of follow-up error rate. At this step, higher baseline error rate remained significantly associated with higher follow-up error rate, while baseline age and baseline PDS total scores main effects were unrelated to follow-up error rate. However, there was a significant interaction between baseline age and baseline PDS total score, indicating that the association between baseline age and change

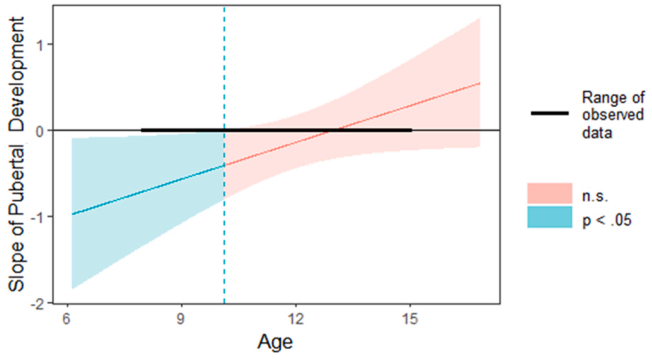


Fig. 4. Johnson-Neyman pubertal development and age interaction plot predicting change in P300.

Table 3
Multiple linear regressions results using two-year error rate as the criterion (n = 129).

Predictor	*b*	*b* 95% CI [LL, UL]	*β*	*β* 95% CI [LL, UL]	*p*-value	Fit
Step 1						
T1 Error rate	0.42	[0.26, 0.58]	0.42	[0.26, 0.58]	< 0.001	
T1 Age	-0.96	[− 2.02, 0.10]	-0.19	[− 0.40, 0.02]	0.076	
T1 PDS	0.03	[− 0.43, 0.49]	0.01	[− 0.19, 0.22]	0.890	
						R^2 = .246, p < .001 (adjusted R^2 =.228, p < .001) 95% CI [0.11,0.35]
Step 2						
T1 Error rate	0.42	[0.26, 0.57]	0.41	[0.26, 0.56]	< 0.001	
T1 Age	-0.56	[− 1.60, 0.48]	-0.11	[− 0.32, 0.09]	0.287	
T1 PDS	-0.10	[− 0.55, 0.35]	-0.04	[− 0.24, 0.15]	0.659	
T1 Age x PDS	0.36	[0.15, 0.57]	0.26	[0.11, 0.42]	0.001	
						R^2 = .312, p < .001 (adjusted R^2 =.290, p < .001) 95% CI [0.17,0.41]

Note. PDS represents the Pubertal Development Scale total score. T1 represents baseline assessment. A significant *b*-weight indicates the beta-weight and semi-partial correlation are also significant. *b* represents unstandardized regression weights. *β* indicates the standardized regression weights. *LL* and *UL* indicate the lower and upper limits of a confidence interval, respectively.

in error rate over two years was influenced by low levels of pubertal development at baseline. That is, younger adolescents who were less pubertally developed had *increases* in error rates (performed worse) over two years (−1 *SD*), $b = -2.06$, $p < .001$. Younger adolescents who were more (+1 *SD*), $b = 0.94$, $p = .213$, or at average, $b = -0.56$, $p = .289$, pubertal development demonstrated *no change or improvements* in task

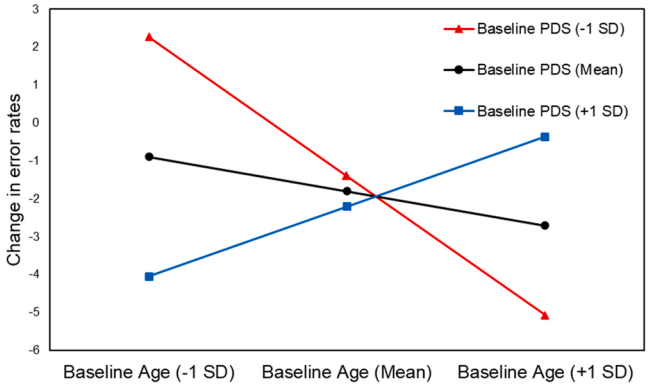


Fig. 5. *Two-way interaction between baseline age and baseline PDS total score in predicting change in error rates over two years (n = 129). Note.* PDS represents baseline Pubertal Development Scale total score. Change in error rates was computed by saving the unstandardized residuals in a linear regression that predicted two-year follow-up error rate from baseline error rate. A more positive change value represents growth in errors made while a negative value represents reductions in errors made.

accuracy over two years (see Fig. 5). The inclusion of the interaction term accounted for significantly more variance (ΔX^2 between step 1 and step 2 = 11.90, $p = .001$), with an additional 6.6% of variance explained in follow-up error rate with the addition of the interaction term.

The Johnson-Neyman interval indicated that the association between baseline PDS total scores and follow-up error rate was significant for participants 10.40 years or younger (i.e., uncentered raw values; reflecting 27.13% of the sample) and for participants 13.39 years or older (i.e., uncentered raw values; reflecting 17.83% of the sample); the relationship between baseline PDS total score and follow-up error rate was not significant for adolescents between the ages of 10.40 and 13.39 (Fig. 6).

3.5. Multiple linear regressions: reaction time

3.5.1. Prospective analysis

At step one, baseline reaction time, baseline age, and baseline PDS total score were entered as independent variables to predict follow-up reaction time. Higher baseline reaction time was significantly associated with a higher follow-up reaction time ($b = 0.41$, $t(128) = 8.46$, $p < .001$, 95% CI [0.32, 0.51]); however, neither baseline age ($b = 0.62$, $t(128) = 0.17$, $p = .867$, 95% CI [− 6.68, 7.92]) nor baseline PDS total score ($b = 0.10$, $t(128) = 0.07$, $p = .947$, 95% CI [− 2.84, 3.04]) were significant predictors of follow-up reaction time.

At step two, the baseline age x baseline PDS total score product term was entered as a predictor of follow-up reaction time. At this step, higher baseline reaction time remained significantly associated with higher follow-up reaction time ($b = 0.41$, $t(128) = 8.44$, $p < .001$, 95% CI [0.32, 0.51]). Baseline age ($b = 0.34$, $t(128) = 0.09$, $p = .929$, 95% CI [− 7.14, 7.81]), baseline PDS total ($b = 0.20$, $t(128) = 0.13$, $p = .896$, 95% CI [− 2.80, 3.19]), and their interaction ($b = -0.27$, $t(128) = -0.38$, $p = .703$, 95% CI [− 1.65, 1.11]) were each unrelated to follow-up reaction time.

4. Discussion

The current study examined the cross-sectional and prospective associations between age, pubertal development, flanker P300 amplitude, and behavioral performance measures over the course of two years in a large sample of adolescent females. At the baseline assessment, neither pubertal development nor age were significantly associated with baseline P300 amplitude. However, in terms of the impact of puberty and age on subsequent changes in the P300, older adolescents and adolescents with increased baseline pubertal score exhibited a reduced P300 amplitude at the two-year follow-up visit. Only age, not total pubertal score, emerged as a unique predictor of P300 amplitude change over two years when placed in a multiple linear regression together. However, there was a significant baseline age by pubertal score interaction predicting change in flanker P300 amplitude over two years, such that the influence of pubertal development on P300 amplitude change was most evident among younger participants. More specifically, among younger females (i.e., between the ages of 8 and 10 years), increased pubertal development predicted *decreases* in P300 amplitude over two years, while female participants experiencing less/average pubertal development increased in P300 amplitude over two years. A nearly identical interaction of age and pubertal development was evident regarding changes in error rates over two years such that among younger females (i.e., between the ages of 8 and 10 years), increased pubertal development predicted *decreases* in error rates (improved performance), while female participants experiencing less pubertal development *increased* in error rates.

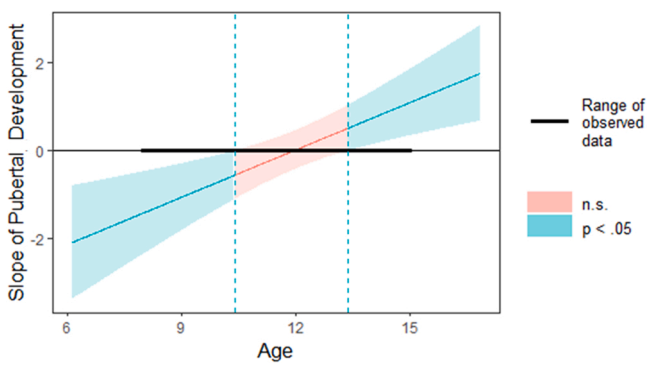


Fig. 6. Johnson-Neyman pubertal development and age interaction plot predicting change in error rates.

There was no significant relationship between baseline age and baseline flanker P300 amplitude which was unexpected and is at odds with the majority of past research that demonstrates that older children and adolescents typically are characterized by smaller *visual* P300 components (e.g., Courchesne, 1978; Katsanis et al., 1996; Hill et al., 1999; Batty and Taylor, 2002; Carlson and Iacono, 2006; Riggins and Scott, 2020; Santopetro et al., 2021). In a study by Overbye et al. (2018), the authors found that older participants (ages 9–19) exhibited *larger* P300 amplitudes during the rare target and distractor conditions of an oddball task; this effect was not present for P300 elicited from the more frequent standard condition. It is important to highlight the effect that task context has on our conceptualization of the functionality of different P300 components. Although there are various similarities between the oddball and flanker tasks employed in both studies (i.e., both are fast paced reaction time task involving visual stimuli), there are also significant differences in the frequency of stimuli presentation, which is a well-known factor that influences the P300 (Polich, 2007). The P300 component elicited from equiprobable flanker stimuli, as well as the frequent standard stimuli, might tap neural activity reflecting sustained attention and motivation to the experimental task, while P300 elicited from rare target and distractor stimuli could reflect novelty and inhibitory control processes which could potentially relate to age in different directions. In line with this conceptualization, there was a significant relationship between baseline age and P300 amplitude two years later, such that older participants are likely to exhibit reductions in later P300 amplitude, suggesting more efficient cognitive processing of experimental stimuli as children and adolescence age. In addition, age was also associated with better performance during the flanker task such that older females were both faster and more accurate than younger females at both visits, indicating better cognitive performance with increasing age during adolescence.

At the zero-order level, there was no significant association between baseline pubertal development and baseline P300 amplitude; however, adolescents reporting higher pubertal development scores at baseline were characterized by a reduced P300 amplitude at the two-year follow-up visit. This finding is consistent with results from a past study reporting smaller visual P300 amplitude related to increased pubertal development, which would also suggest that children at a higher level of pubertal development demonstrate more efficient information processing (Brumback et al., 2012). Furthermore, participants reporting more advanced pubertal development at baseline also performed better on the flanker task, in terms of response speed, at both the baseline and follow-up visits. Although chronological age and pubertal development were moderately related ($r = 0.66$) and thus shared approximately 44% variance in the present study, we simultaneously examined these two variables in relation to prospective changes in P300 amplitude. When employed as simultaneous predictors of two-year P300 amplitude, age explained unique variance in two-year P300 amplitude over and above pubertal development scores, suggesting that change in neural activity may be better accounted for by an individual's chronological age. In terms of behavioral performance, we found that neither age nor pubertal development appeared to independently predict changes in error rate or reaction times over two years suggesting that the shared variance between age and pubertal development explained the development of

these behavioral measures.

To further elucidate the relationship between age, pubertal development, and change in P300 amplitude during adolescence, an additional multiple linear regression model was conducted with the inclusion of an age and pubertal development score interaction term as a proxy for pubertal timing (i.e., assessing the influence of different levels of pubertal development at different ages on change in neural activity). This interaction was significant suggesting that, among younger females, increased pubertal development predicted decreases in P300 amplitude from baseline to follow-up, whereas both decreased and average pubertal development predicted increases in P300 amplitude. More specifically, this effect was driven by a subgroup of participants between 8 and 10 years of age. Results from a separate multiple linear regression model predicting change in error rate over two-years yielded highly similar results. More specifically, younger females who were less pubertally developed committed more errors in the flanker task over two years, while younger females at average or more advanced pubertal development demonstrated no change or improvements in task accuracy over this time period. Moreover, the effect between age and pubertal development predicting trajectory in error rate over two years appeared to also be driven, in part, by a sub-group of participants between the ages of 8- and 10-years old mirroring results from the model utilizing P300 amplitude. However, it is important to highlight that, although pubertal development and age interact in a very consistent way to predict changes in these measures of cognitive ability during development, these two measures are most likely explaining different aspects of neural/cognitive maturation that is occurring over this two-year time frame as residual change in P300 amplitude and residual change in error rate were not significantly associated with one another in the current study, $r(127) = 0.14$, $p = .125$. One possibility for this lack of overlap between these two measures could be due to method variance. That is, P300 reflects a distinct neuropsychological operation linked to cognitive processes such as attention allocation to significant stimuli, and error rate is an end-state measure of several neuropsychological operations, including early sensory, perceptual, cognitive, and motor processing. Lastly, age and pubertal development did not interact to predict trajectory of reaction times over two years during adolescence.

Taken together, these results implicate the role of pubertal timing in neural and behavioral development as female participants experiencing early pubertal timing in the present sample (i.e., increased pubertal development compared to same-aged peers) exhibited a significantly different developmental trajectory in both P300 amplitude and error rate over two years compared to female individuals experiencing less pubertal development in the same age range. It is important to note that this interaction was not evident at baseline, which would suggest that the influence of pubertal timing is preceding differences observed in P300 amplitude and task accuracy. Considering that past research has demonstrated that children and adolescents who undergo earlier pubertal timing are more at risk for psychopathology such as depressive and alcohol or substance use disorders (Dick et al., 2000; Angold et al., 1998), and that reductions in P300 amplitude have been consistently linked to these specific forms of mental health disorders during adolescence (Patrick et al., 2006; Polich et al., 1994; Iacono et al., 2002; Houston et al., 2003; Santopetro et al., 2020, 2021), future research should aim to examine the relationship between all of these factors during childhood and adolescence. For example, it is plausible that the relationship between early pubertal timing and increased risk for depression or externalizing disorders later in development may be partly mediated by this altered neurodevelopmental trajectory of motivational and cognitive functioning indexed by the P300 ERP component.

The current study has limitations worth discussing. First, the current sample is predominantly White (i.e., 87%) and comprises only female participants, which limits the generalizability of the current findings. Future studies need to investigate more ethnic and racially representative samples of both male and female children and adolescents to determine if the association between pubertal timing and P300 amplitude/error rate trajectory discussed in the present study is consistent. Additionally, it remains unclear what *specific* developmental changes are influencing this altered trajectory of P300 amplitude and error rates over two years observed in individuals experiencing early pubertal timing, and future studies will need to investigate more specific domains, such as biological, social, and emotional changes, to better understand these associations. Lastly, many, if not most, traditional studies of the P300 employ a version of the oddball task to elicit the component, whereas the current study utilized the flanker task. It is of the opinion of the authors of the present project that the positive neural component elicited from the flanker stimuli can be conceptualized as a P300 based on more recent and inclusive conceptualizations of the ERP (e.g., Bradley, 2009; Hajcak & Foti, 2020). However, it could also be argued that this positivity could be considered a Late Positive Complex (LPC) or Late Posterior Positivity (LPP). Overall, more research is needed to further refine our understanding of the mechanisms that underlie and potentially differentiate the P300 from the LPC or LPP components providing more insight into the exact functionality of these components.

Overall, the current study adds to past findings that observed cross-sectional reductions in visual P300 amplitude associated with older age/more advanced development during childhood and adolescence by expanding this work to include prospective analyses over a two-year time frame. Furthermore, the present study examined both chronological age and pubertal development scores, as well as their interaction, in an attempt to parse apart these two interrelated variables to further understand which factor may be uniquely influencing P300 amplitude and behavioral measures across development. Findings initially indicated that age independently predicted change in P300 amplitude over two years, while neither age nor pubertal development uniquely predicted change in behavioral performance measures over this time frame. More importantly, the interaction between these two variables significantly predicted P300 amplitude and error rate change such that younger females exhibiting heightened pubertal development demonstrated decreases in P300 amplitude and in error rates over two years, while similar-aged peers reporting less pubertal development actually increased in P300 amplitude and in errors committed during the task two years later. These results suggest that pubertal timing may directly influence neural development linked to motivational and cognitive processes during adolescence.

Funding

This work was supported by the National Institutes of Health under Award Number MH097767 to GH. EMM received support from the National Institute of Mental Health under Award Number F31MH125624. CJB received support from the National Institute of Mental Health under Award Number F32MH125504.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

Anderson, V., 2001. Assessing executive functions in children: biological, psychological, and developmental considerations. Pediatr. Rehabil. 4 (3), 119–136.

Angold, A., Costello, E.J., Worthman, C.M., 1998. Puberty and depression: the roles of age, pubertal status and pubertal timing. Psychol. Med. 28 (1), 51–61.

Bachiller, A., Lubeiro, A., Díez, Á., Suazo, V., Domínguez, C., Blanco, J.A., Molina, V., 2015. Decreased entropy modulation of EEG response to novelty and relevance in schizophrenia during a P300 task. Eur. Arch. Psychiatry Clin. Neurosci. 265 (6), 525–535.

Bashore Jr, T.R. , 1990. Age-related changes in mental processing revealed by analyses of event-related brain potentials.

Batty, M., Taylor, M.J., 2002. Visual categorization during childhood: an ERP study. Psychophysiology 39 (4), 482–490.
Bauer, L.O., O'Connor, S., Hesselbrock, V.M., 1994. Frontal P300 decrements in antisocial personality disorder. Alcohol.: Clin. Exp. Res. 18 (6), 1300–1305.
Beltz, A.M., Berenbaum, S.A., 2013. Cognitive effects of variations in pubertal timing: is puberty a period of brain organization for human sex-typed cognition? Horm. Behav. 63 (5), 823–828.
Bradley, M.M., 2009. Natural selective attention: orienting and emotion. Psychophysiology 46 (1), 1–11.
Bruder, G.E., Kayser, J., Tenke, C.E., 2012. Event-related brain potentials in depression: clinical, cognitive and neurophysiologic implications. Oxf. Handb. Event-Relat. Potential Compon. 2012, 563–592.
Brumback, T.Y., Arbel, Y., Donchin, E., Goldman, M.S., 2012. Efficiency of responding to unexpected information varies with sex, age, and pubertal development in early adolescence. Psychophysiology 49 (10), 1330–1339.
Carlson, S.R., Iacono, W.G., 2006. Heritability of P300 amplitude development from adolescence to adulthood. Psychophysiology 43 (5), 470–480.
Carlson, S.R., McLarnon, M.E., Iacono, W.G., 2007. P300 amplitude, externalizing psychopathology, and earlier-versus later-onset substance-use disorder. J. Abnorm. Psychol. 116 (3), 565.
Carskadon, M.A., Acebo, C., 1993. A self-administered rating scale for pubertal development, 190–5 J. Adolesc. Health 14. https://doi.org/10.1016/1054-139X(93)90004-9.
Chong, L.J., Mirzadegan, I.A., Meyer, A., 2020. The association between parenting and the error-related negativity across childhood and adolescence. Dev. Cogn. Neurosci. 45, 100852.
Courchesne, E., 1978. Neurophysiological correlates of cognitive development: changes in long-latency event-related potentials from childhood to adulthood. Electroencephalogr. Clin. Neurophysiol. 45 (4), 468–482.
Dick, D.M., Rose, R.J., Viken, R.J., Kaprio, J., 2000. Pubertal timing and substance use: associations between and within families across late adolescence. Dev. Psychol. 36 (2), 180.
van Dinteren, R., Arns, M., Jongsma, M.L., Kessels, R.P., 2014. P300 development across the lifespan: a systematic review and meta-analysis. PloS One 9 (2), e87347.
Eriksen, B.A., Eriksen, C.W., 1974. Effects of noise letters upon the identification of a target letter in a nonsearch task. Percept. Psychophys. 16 (1), 143–149.
Graber, J.A., Seeley, J.R., Brooks-Gunn, J., Lewinsohn, P.M., 2004. Is pubertal timing associated with psychopathology in young adulthood? J. Am. Acad. Child Adolesc. Psychiatry 43 (6), 718–726.
Hajcak, G., Klawohn, J., Meyer, A., 2019. The utility of event-related potentials in clinical psychology. Annu. Rev. Clin. Psychol. 15, 71–95.
Hartshorne, J.K., Germine, L.T., 2015. When does cognitive functioning peak? the asynchronous rise and fall of different cognitive abilities across the life span. Psychol. Sci. 26 (4), 433–443.
Hayes, A.F., 2013. Model templates for PROCESS for SPSS and SAS. Retrieved Dec. 12, 2013.
Herting, M.M., Uban, K.A., Gonzalez, M.R., Baker, F.C., Kan, E.C., Thompson, W.K., Sowell, E.R., 2021. Correspondence between perceived pubertal development and hormone levels in 9-10 year-olds from the adolescent brain cognitive development study. Front. Endocrinol. 11, 549928.
Hill, S.Y., Shen, S., Locke, J., Steinhauer, S.R., Konicky, C., Lowers, L., Connolly, J., 1999. Developmental delay in P300 production in children at high risk for developing alcohol-related disorders. Biol. Psychiatry 46 (7), 970–981.
Houston, R.J., Bauer, L.O., Hesselbrock, V.M., 2003. Depression and familial risk for substance dependence: a P300 study of young women. Psychiatry Res.: Neuroimaging 124 (1), 49–62.
Iacono, W.G., Carlson, S.R., Malone, S.M., McGue, M., 2002. P3 event-related potential amplitude and the risk for disinhibitory disorders in adolescent boys. Arch. Gen. Psychiatry 59 (8), 750–757.
Johnson, P.O., Fay, L.C., 1950. The Johnson-Neyman technique, its theory and application. Psychometrika 15, 349–367. https://doi.org/10.1007/BF02288864.
Katsanis, J., Iacono, W.G., McGue, M.K., 1996. The association between P300 and age from preadolescence to early adulthood. Int. J. Psychophysiol. 24 (3), 213–221.
Laitinen-Krispijn, S., Van der Ende, J., Hazebroek-Kampschreur, A.A.J.M., Verhulst, F.C., 1999. Pubertal maturation and the development of behavioural and emotional problems in early adolescence. Acta Psychiatr. Scand. 99 (1), 16–25.
Levinson, A.R., Speed, B.C., Infantolino, Z.P., Hajcak, G., 2017. Reliability of the electrocortical response to gains and losses in the doors task. Psychophysiology 54 (4), 601–607. https://doi.org/10.1111/psyp.12813.
Li, F., Wang, J., Liao, Y., Yi, C., Jiang, Y., Si, Y., Xu, P., 2019.). Differentiation of schizophrenia by combining the spatial EEG brain network patterns of rest and task P300. IEEE Trans. Neural Syst. Rehabil. Eng. 27 (4), 594–602.
Meyer, A., Carlton, C., Crisler, S., Kallen, A., 2018. The development of the error-related negativity in a large sample of adolescent females: Associations with anxiety symptoms. Biol. Psychol.
Meyer, A., Mehra, L., Hajcak, G., 2021. Error-related negativity predicts increases in anxiety in a sample of clinically anxious female children and adolescents over 2 years. J. Psychiatry Neurosci. 46 (4), E472–E479.
Nunnally, J.C., Bernstein, I.H., Berge., J.M.T. , 1967. Psychometric theory (Vol. 226). New York, NY: McGraw-Hill.
Overbye, K., Huster, R.J., Walhovd, K.B., Fjell, A.M., Tamnes, C.K., 2018. Development of the P300 from childhood to adulthood: a multimodal EEG and MRI study. Brain Struct. Funct. 223 (9), 4337–4349.
Patrick, C.J., Bernat, E.M., Malone, S.M., Iacono, W.G., Krueger, R.F., McGue, M., 2006. P300 amplitude as an indicator of externalizing in adolescent males. Psychophysiology 43 (1), 84–92.
Petersen, A.C., Crockett, L., Richards, M., Boxer, A., 1988. A self-report measure of pubertal status: reliability, validity, and initial norms. J. Youth Adolesc. 17 (2), 117–133.
Polich, J., 2007. Updating P300: an integrative theory of P3a and P3b. Clin. Neurophysiol. 118 (10), 2128–2148.
Polich, J. , 2012. Neuropsychology of P300. Oxford handbook of event-related potential components, 159, 88.
Polich, J., Luckritz, J.Y., 1995. EEG and ERPs in young and elderly subjects. Electroencephalogr. Clin. Neurophysiol. Suppl. 44, 358–368.
Polich, J., Pollock, V.E., Bloom, F.E., 1994. Meta-analysis of P300 amplitude from males at risk for alcoholism. Psychol. Bull. 115 (1), 55.
Preacher, K.J., Hayes, A.F., 2008. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav. Res. Methods 40 (3), 879–891.
Riggins, T., Scott, L.S., 2020. P300 development from infancy to adolescence. Psychophysiology 57 (7), e13346.
Santopetro, N.J., Kallen, A.M., Threadgill, A.H., Hajcak, G., 2020. Reduced flanker P300 prospectively predicts increases in depression in female adolescents. Biol. Psychol. 156, 107967.
Santopetro, N.J., Kallen, A.M., Threadgill, A.H., Amir, N., Hajcak, G., 2021. Blunted flanker P300 demonstrates specificity to depressive symptoms in females during adolescence. Res. Child Adolesc. Psychopathol. 1–12.
Stumper, A., Mac Giollabhui, N., Abramson, L.Y., Alloy, L.B., 2020. Early pubertal timing mediates the association between low socioeconomic status and poor attention and executive functioning in a diverse community sample of adolescents. J. Youth Adolesc. 49 (7), 1420–1432.
Wechsler, D., 1997a. Technical manual for the Wechsler Adult Intelligence Scale—Third Edition. Psychological Corp, San Antonio, TX.
Wechsler, D., 1997b. Technical manual for the Wechsler Memory Scale—Third Edition. The Psychological Corp, San Antonio, TX.