Published in final edited form as:

Int J Psychophysiol. 2022 October ; 180: 1–9. doi:10.1016/j.ijpsycho.2022.06.016.

Age-related no-go P300 amplitudes are moderated by exposure to early-life stress

Elizabeth R. Paitel[a,b], **Sierra D. Peters**[a,c], **Michelle Lobermeier**[a,d], **Raquel A. Lopez**[a,*]

[a]Department of Psychology, St. Norbert College, United States of America

[b]Department of Psychology, Marquette University, United States of America

[c]Department of Psychology, Florida State University, United States of America

[d]Department of Psychology, Eastern Michigan University, United States of America

Abstract

Deficits in inhibitory control are common with advancing age and may underlie declines in other complex cognitive functions. The inhibitory P300 event-related potential (ERP) generally decreases in amplitude with age, reflecting deficits in inhibitory performance evaluation and adaptation, with possible generators including precentral and inferior frontal gyri and midcingulate and parietal cortex. Exposure to early-life stress (ELS) is also associated with deficits in inhibitory control, smaller P300 amplitudes, and dysfunction in regions associated with P300 generation. Although biopsychosocial effects of ELS are evident in older adulthood, the influence of ELS on neural processes in later life is unknown. In the current study, 13 young adults and 21 healthy older adults completed a high-accuracy go/no-go task and the Juvenile Victimization Questionnaire (JVQ), an indicator of ELS. Regression analyses revealed significant central-parietal models, with smaller P300 amplitudes predicted by both older age and greater exposure to ELS. Age group*ELS interactions moderated P300 prediction at central and centro-parietal electrodes, such that older age predicted smaller P300 amplitudes only in those with lower to moderate ELS. Amplitudes did not significantly differ by age in those with higher ELS. *Post-hoc* within-age group correlations showed that greater ELS was associated with smaller P300 amplitudes in young adults. However, greater ELS was modestly associated with *larger* central amplitudes in older adults, potentially suggestive of anterior age-related compensatory recruitment to maintain high task performance. These findings suggest long-lasting neural implications of ELS that interact with normative neuro-cognitive aging processes, such that ELS may be an important risk factor for age-related cognitive decline.

*Corresponding author at: St. Norbert College, 100 Grant Street, De Pere, WI, United States of America., raquel.lopez@snc.edu (R.A. Lopez).

Declaration of competing interest

The authors have no conflicts of interest to report.

Ethics approval

All procedures were performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Institutional Review Board at St. Norbert College (June 1, 2015, 14–02-006).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijpsycho.2022.06.016.

Keywords

Event-related potentials; Aging; Early-life stress; Inhibitory control; P300; Go/no-go

Complex cognitive processes like those involved in everyday decision-making require inhibitory control to suppress irrelevant or non-goal directed information (Miyake et al., 2000; Munakata et al., 2011). Deficits in inhibitory control may in fact account for more global cognitive declines with increasing age (Salthouse et al., 2003; Sweeney et al., 2001). Thus, investigation of factors that increase risk of inhibitory deficits, and characterization of the neural patterns underlying inhibitory performance, will contribute to better prediction of and intervention for age-related cognitive decline (Chen et al., 2021; Lustig et al., 2009).

A relatively large body of research highlights poorer inhibitory control in children and adolescents with exposure to early-life stress (ELS; e.g., Bruce and Kim, 2020; Carvalho et al., 2020; Cowell et al., 2015; Cross et al., 2017; Lund et al., 2020). ELS is operationalized in several ways in these studies, with key experiences including familial instability, direct victimization, neglect, or a combination of these factors. Less research has investigated the lasting relationship between ELS and inhibitory performance in adulthood, but the existing work generally suggests that inhibitory deficits endure at least into early/mid-adulthood (Daly et al., 2017; Gould et al., 2012; Marshall et al., 2016; Pechtel and Pizzagalli, 2011). In one case, adults exposed to childhood maltreatment performed more accurately than the control group specifically on the inhibitory trials with a negative background distractor image, suggesting a greater, disproportionate amount of attention to task-irrelevant, negative stimuli. Such over-allocation of attentional processes to negative but irrelevant stimuli may translate to poor directive attention in real-life, emotive applications (Demers et al., 2021).

Critically, negative biopsychosocial outcomes associated with ELS are evident even in older adulthood. Overall, this body of work reveals that those with greater ELS exposure have increased risk for accelerated biological aging and cognitive decline, depression and anxiety, altered stress responses, and premature mortality (e.g., Brown et al., 2009; Fogelman and Canli, 2018; Goldman-Mellor et al., 2012; Kiecolt-Glaser et al., 2011; Korten et al., 2014; Lähdepuro et al., 2019; Pechtel and Pizzagalli, 2011; Ritchie et al., 2009; Ritchie et al., 2011; Savolainen et al., 2014). For example, a large sample of older adult males with a history of parental separation in childhood evidenced lower verbal, visuospatial, arithmetic, and general cognitive functioning compared to those without such ELS (Fogelman and Canli, 2018). Moreover, a large-scale study highlighted impaired stress responses in older adults with exposure to ELS compared to their peers, with different trajectories for those exposed to ELS based on whether they also had recurrent psychological distress in adulthood. Older adults with both ELS and recurrent psychological distress in adulthood displayed blunted cortisol reactivity, while those with ELS who did not experience recurrent distress in adulthood had elevated baseline cortisol, prolonged cortisol responses, and greater overall cortisol production (Goldman-Mellor et al., 2012). This work points to altered stress responses in later life that are rooted in ELS, which interact with life course experiences, and imply negative outcomes for cognitive function, including the risk of dementia (Comijs et al., 2010; Ouanes and Popp, 2019).

Research aiming to characterize the neural correlates of ELS-related inhibitory deficits has largely used functional MRI (fMRI). Overall, those with greater ELS show less efficient activation of the inhibitory network, including hyperactivation of dorsomedial and inferior prefrontal cortex, pre- and post-central gyri, the anterior cingulate gyrus, and posterior insula (Lim et al., 2015; Mueller et al., 2010). One study also showed a lack of differential activation for correct versus incorrect no-go trials (typically activation for incorrect > correct no-go trials) in the dorsolateral prefrontal cortex. Notably, this finding was evident only when accounting for early-life stress, but not stress in adulthood (Harms et al., 2017). Hyperactivation accompanied by poorer task performance suggests that the greater resources being allocated to completion of the task are not sufficient to attain high-level cognitive performance (Reuter-Lorenz and Cappell, 2008). Despite evidence of the enduring effects of ELS in later life, research investigating the neural underpinnings of these effects is severely limited. If the inhibitory deficits that are evident in early life are indeed long-term effects, then older adults with a history of ELS are at an increased risk of compromised inhibitory control, increasing the likelihood for poorer overall aging outcomes, including the inability to live independently (O'Connor and Boyle, 2007).

While fMRI allows for spatially localizing brain activation during a given task, it collapses activation across a period of several seconds (e.g., Slotnick, 2017). This poor temporal resolution critically limits interpretation of the specific neural processes underlying successful inhibitory control (e.g., conflict monitoring, motor response inhibition, performance evaluation), which take place within only ~500 ms after an inhibitory stimulus (Pires et al., 2014). Electroencephalography (EEG) and event-related potentials (ERPs), on the other hand, have millisecond-level resolution (Luck, 2014), which enables separation of distinct subprocesses (Paitel and Nielson, 2021). One such subprocess is evaluation and adaptation of inhibitory performance, which is reflected by the P300 component (Huster et al., 2013; Huster et al., 2020). The inhibitory P300 is of particular interest in questions of age by ELS interactions. First, there is notable overlap in regions associated with generation of the inhibitory P300 and those implicated in fMRI studies of children and adolescents with ELS exposure (Enriquez-Geppert et al., 2010; Huster et al., 2010; Huster et al., 2013; Huster et al., 2020). Furthermore, the inhibitory evaluation and adaptation processes reflected by the P300 component may be affected early in the aging process (vs. conflict monitoring), making these processes particularly vulnerable to further insult from ELS exposure (e.g., Paitel and Nielson, 2021).

ERP studies examining the relationship between ELS, inhibitory control, and P300 amplitudes have reported mixed results. Some studies found no significant group differences in children (Bruce and Kim, 2020; Lamm et al., 2018) or adults (Wu et al., 2021; Xue et al., 2017). Yet, others reported smaller P300 amplitudes or P300 source activation with greater ELS in children (McDermott et al., 2012) and adults (Kim et al., 2018). In contrast, another study with adult participants reported larger P300 amplitudes specifically in those exposed to childhood physical and sexual abuse (Howells et al., 2012). These inconsistent findings across studies are likely attributable to the great variability in age of participants, different domains of ELS (e.g., institutionalization, cumulative ELS exposure, physical abuse, neglect, etc.), other sample characteristics (e.g., Xue et al., 2017 specifically compared groups with high aggression), and inhibitory task parameters.

The most common approach to assessing ELS in adults is through self-report questionnaires. The Juvenile Victimization Questionnaire (JVQ) is a multi-domain assessment of ELS that includes appraisal of assault, peer victimization, exposure to family violence, parent-child dysfunction, and witnessed violence (Hamby et al., 2011; Hamby et al., 2004). The JVQ accounts for the cumulative overlap of negative experiences in different domains, which may be particularly important for accurate assessment of the global impact of ELS compared to assessments of domain specific ELS. Indeed, those exposed to one domain of ELS often also report exposure to other domains, and that cumulative exposure has been shown to be a stronger predictor of psychopathology than individual types of ELS (Haahr-Pedersen et al., 2020; Wolfe, 2018). The JVQ provides a useful index of ELS, and when paired with examination of the P300, may provide new insights into the relationship between ELS, inhibitory control performance, and neural processing at different time points in the lifespan.

Specifically, the temporal resolution of ERPs enables targeting of inhibitory P300-related processes and networks, which may be particularly sensitive to aging and ELS-related deficits (Elverman et al., 2021; Kim et al., 2018; McDermott et al., 2012; Paitel and Nielson, 2021). Better characterization of the neural patterns associated with both age and ELS may provide greater opportunity to identify individuals most likely to experience age-related cognitive declines and increase the odds of an appropriate and timely intervention (Chen et al., 2021; Lustig et al., 2009). The current study uses a simple, high-accuracy go/no-go task to ensure comparable task accuracy between age groups and minimize task difficulty- and error-related activation. Based on the existing research focused on the effects of aging or ELS, we hypothesized smaller P300 amplitudes during accurate inhibitory control (i.e., no-go) trials in older vs. young adults. We expected this relationship to be moderated by ELS, such that the predicted effect would be largest (i.e., P300 amplitudes would be smallest) in those with greater exposure to ELS. Additionally, we expected that greater exposure to ELS across age groups would predict smaller P300 amplitudes.

1. Method

1.1. Participants

Older adult participants ($n = 21$) were recruited from the community and compensated monetarily. Young adult participants ($n = 14$) were recruited from psychology classes offering course credit. A priori power analysis for linear multiple regression recommended a total sample size of 32 participants to detect effects with an overall model effect size (Cohen's f^2) = 0.40 with three predictors (age group, ELS, and age group*ELS interaction) at $\alpha = 0.05$, power (1- β) = 0.80 (G*Power; Faul et al., 2009). These values were selected based on effect sizes from relevant literature (e.g., Ashford et al., 2011; Elverman et al., 2021; Paitel and Nielson, 2021; van Dinteren et al., 2014).

1.2. Materials

1.2.1. Go/no-go task—The go/no-go task consisted of a serial stream of letters visually presented at a rate of 2000 ms per letter with an interstimulus interval of 1000 ms. Participants were instructed to press the space bar for every letter presented, except for "x" (202 stimuli, 50 no-go trials). Outcome measures included accuracy, target response time

(RT), and no-go false alarm (i.e., incorrect) response time. Note that no metric of response time can be calculated for the primary trials of interest – accurate no-go trials.

1.2.2. Juvenile Victimization Questionnaire: Key Domains Short Form (JVQ)

—The JVQ short form is a 21-item self-report measure of early-life interpersonal victimization in the following domains: assault, peer victimization, exposure to family violence, parent-child dysfunction, and witnessed violence (adapted from Finkelhor et al., 2005). Scores on this metric provide an assessment of exposure to a wide range of stressful early-life experiences with good test-retest reliability and construct validity (Hamby et al., 2011; Hamby et al., 2004). Internal consistency in the current sample was Cronbach α = 0.73.

1.2.3. EEG data acquisition and ERPs—

Continuous EEG data were collected using a 32-electrode Brain Products actiCAP arranged according to the International 10–20 System with ground at AFz and reference at FCz. Data were recorded with a 500 Hz sampling rate with impedances kept under 30kΩ using Brain Products actiCHamp and BrainVision Pycorder software. Continuous EEG data were processed off-line using Brain Products Brain Vision Analyzer 2.0 and EEGLAB (Version 14.1.1; Delorme and Makeig, 2004) software via MATLAB (Version 9.1, The MathWorks).

Continuous data were visually inspected and channels with extreme artifacts throughout the session were interpolated as necessary (using spherical splines) to eliminate channel-level artifacts. The number of channels interpolated was low and comparable between age groups, with M_{young} = 0.23 (SD_{young} = 0.58; median = 0; range = 0–2) and M_{older} = 0.30 (SD_{older} = 0.46; median = 0; Range = 0–1). Second-order Butterworth filters from 0.2 to 50 Hz and a 60 Hz notch filter were applied. Next, an Adaptive Mixture Independent Component Analysis (AMICA; Palmer et al., 2008) was used to decompose data into individual components. Components reflecting eye blink, other ocular movements, and muscle contraction were removed based on visual inspection. Semi-automatic artifact detection was then used to detect voltage steps >40 μV, amplitudes greater than ±150 μV, and activity below 0.5 μV. The data were re-referenced to a common average of all electrodes.

Data from correct no-go trials were segmented from 200 ms prior to stimulus onset to 1500 ms post-stimulus. Segmented data were baseline corrected using the −200 ms pre-stimulus period. Epochs were then examined and rejected as needed based on visual inspection, which was guided by the semi-automatic artifact detection step. Artifact rejection was conducted on an individual channel level, allowing for an artifact that was limited to a particular channel to be removed without discarding the entire epoch. The average number of epochs contributing to the average was comparable by age group. In the young adult group, all channels used in the current analyses had an average of M = 45.54 (SD = 6.22) epochs. The older adult group average by channel ranged nominally from M = 47.35 (SD = 2.89) to M = 47.50 (SD = 2.97). Thus, both groups had a high and comparable number of data segments contributing to their averaged metrics.

Epochs were averaged and an additional second-order Butterworth low-pass filter at 20 Hz was applied to attenuate higher frequency noise. Peak amplitude of the P300 component was computed at central-parietal sites (C3, Cz, C4, CP1, CP2, P3, Pz, P4) based on automatic detection, which was confirmed by visual inspection to ensure the main peak was selected. Amplitudes were then averaged by region (central: C3, Cz, C4; centro-parietal: CP1, CP2; parietal: P3, Pz, P4). These sites were chosen given the predominant central-parietal scalp distribution of the P300 waveform (e.g., Patel and Azzam, 2005). ERP grand average waveforms and scalp maps corresponding with the P300 component can be found in Fig. 1.

1.3. Procedure

EEG data during the go/no-go task were collected as part of a larger study. Participants completed one individualized testing session that consisted of surveys, one task without EEG, and EEG data collection during two tasks. Participants were seated in front of a computer following EEG cap placement and were instructed to limit gross motor movements and speech to reduce noise in the EEG signal. The go/no-go task was presented in *E*-Prime 2.0, with instructions read aloud as they appeared on the screen and an opportunity for questions from participants. No feedback was provided during test blocks. All procedures were approved by the college's Institutional Review Board.

1.4. Statistical analysis

Sample demographics and behavioral task performance were compared by age group (young, older) using between-subjects analysis of variance (ANOVA), with the exception of sex distribution, which was compared using a chi-squared test. Behavioral task performance was also compared by a median split of JVQ scores.

Multiple regression (via PROCESS v3.5; Hayes, 2017) was used to investigate whether the relationship between age group (young, older) and P300 amplitudes depended on exposure to ELS (via JVQ score). Models were structured with age group predicting P300 amplitude, with ELS as the moderator, testing also the interaction between age group*-ELS. Significant age group*ELS interactions were further examined using the Johnson-Neyman technique with conditional values based on −1*SD*, mean, and + 1*SD* (Hayes, 2017). To address the multiple comparisons, a Bonferroni correction was applied, with the adjusted threshold for model significance $p < .017$ (α 0.05/3 models = 0.017).

2. Results

2.1. Descriptive statistics and excluded data

One young adult participant was excluded from analyses due to low task performance and one older adult participant was excluded due to an outlying JVQ score. These exclusions resulted in a final sample of 20 older and 13 young adult participants. Sample demographics are presented in Table 1. The older adults had, on average, one more year of formal education than young adults, which was statistically significant.

2.2. Task performance analyses

Behavioral performance data are shown in Table 2. Task accuracy did not significantly differ by age group. As expected, older adults had slower response times than younger adults to go target trials, as well as slower responses on trials with no-go commission errors (i.e., false alarms). Based on a median split of JVQ scores, there were no behavioral effects of ELS.

2.3. ERP analyses

Using the Bonferroni-corrected threshold of $p < .017$, regression models were significant in central ($R^2 = 0.30$, $p = .015$), centro-parietal ($R^2 = 0.49$, $p < .001$), and parietal regions ($R^2 = 0.50$, $p < .001$), with smaller P300 amplitudes predicted by both older age and higher JVQ scores (see Table 3 for regression model statistics, Table 4 for mean P300 amplitude by electrode and age group). Additionally, the interaction between age group and JVQ score moderated P300 amplitudes in central ($R^2_{change} = 0.21$; $p = .007$) and centro-parietal regions ($R^2_{change} = 0.13$; $p = .01$; see Fig. 2 and Table 3). Specifically, older age predicted smaller P300 amplitudes, but only in those with lower to moderate ELS (Johnson-Neyman significance region = fewer than 3.35 JVQ endorsements for central models; fewer than 5.43 endorsements for central-parietal). Internal consistency of P300 amplitude was assessed for each age group via split-half reliability of the odd and even trials. Split-half reliability corrected using the Spearman-Brown prophecy formula (Nunnally and Bernstein, 1994) at all electrodes was ≥0.83, indicating good reliability of P300 amplitude metrics in both age groups.

Post-hoc bivariate Pearson correlations were used to further interrogate the relationship between ELS and P300 amplitudes *within* young and older adults, separately (see Table 5). These correlations were computed at the individual (unaveraged) electrode sites, toward a preliminary assessment of midline vs. lateralized contributions. Within the young adult group, greater ELS was associated with smaller P300 amplitudes at electrodes C4 ($r = -0.65$, $p < .05$), CP1 ($r = -0.72$, $p < .01$), CP2 ($r = -0.69$, $p < .01$), and Pz ($r = -0.60$, $p < .05$). Non-significant trends also indicated smaller amplitudes in those with higher ELS at Cz ($r = -0.51$, $p = .07$) and P4 ($r = -0.53$, $p < .07$). Within the older adult group, greater ELS was associated with *larger* P300 amplitudes at C3 ($r = 0.47$, $p < .05$).

3. Discussion

The current study assessed the impact of age group (young vs. older) and exposure to ELS on inhibitory P300 amplitudes during a simple and high-accuracy go/no-go task. Regression analyses revealed significant central through parietal models, with smaller P300 amplitudes predicted by both older age and greater exposure to ELS. Age group*ELS interactions moderated P300 prediction in central and centro-parietal regions, such that older age predicted smaller P300 amplitudes only in those with lower to moderate ELS. Specifically, age group was a significant predictor only in those with fewer than four JVQ endorsements at central sites and fewer than six endorsements at centro-parietal sites. *Post-hoc* follow-up correlations within young adults showed that greater ELS was associated with smaller central-parietal P300 amplitudes. Thus, even within healthy young adults during a high-accuracy task, ELS was associated with deficits in underlying inhibitory

control networks. Follow-up correlations within the older adult sample revealed that greater ELS was associated with modestly *larger* central P300 amplitudes, suggesting that anterior recruitment may be necessary to maintain high task performance. Indeed, all effects were evident despite high task performance across age and exposure to ELS (no-go trial accuracy >93 %).

The inhibitory P300 component has a relatively diffuse pattern of activation, with potential central-parietal generators including the posterior midcingulate gyrus, pre-supplementary motor area, and precentral gyrus (Huster et al., 2010; Overbye et al., 2021; Pires et al., 2014). P300 activation in these regions tends to be right hemisphere dominant in healthy young adults, with greater bilaterality and left hemisphere recruitment in healthy older adults (Huster et al., 2010; Paitel and Nielson, 2021). Smaller no-go P300 amplitudes at central-parietal sites are commonly reported in healthy older adults compared to young adults (e.g., Elverman et al., 2021; Kardos et al., 2020; Kropotov et al., 2016). During stop-signal inhibitory control tasks, P300 tends to peak *after* the participant's estimated stop-signal reaction time, suggesting that while the onset of P300 may coincide with the suppression and withholding of a motor response, the P300 *peak* likely better reflects post-motoric inhibitory processes. Specifically, P300 peak activity is thought to index evaluation of inhibitory performance and behavior adaptation for future trials (Huster et al., 2013; Huster et al., 2020). Thus, smaller inhibitory P300 amplitudes in healthy older adults supports the idea that these post-motoric, evaluative processes may be particularly vulnerable to the normative aging process (Paitel and Nielson, 2021).

During more complex but high-accuracy inhibitory control tasks, smaller central-parietal P300 amplitudes are often accompanied by larger frontal P300 amplitudes in older adults. Increased anterior activation reflects the greater demand and increased cognitive control necessary for successful inhibition (Elverman et al., 2021; Kropotov et al., 2016; Paitel and Nielson, 2021). Compensatory theories of cognitive aging (e.g., Cabeza, 2002; Reuter-Lorenz and Park, 2014) have shown that increased brain activation in older adults compensates for age-related neural deficits and serves a functional role in maintaining high cognitive performance despite these deficits. Compensatory recruitment is often characterized by recruitment of frontal and non-dominant hemisphere resources. Consistent with these models, two recent studies using complex go/no-go and stop-signal tasks with comparable group accuracy showed that P300 amplitudes during successful inhibition were larger in older adults at frontal and left (non-dominant) hemisphere sites, with larger amplitudes in young adults at posterior and right hemisphere sites (Elverman et al., 2021; Paitel and Nielson, 2021). Despite the lack of significant frontal P300-related activity in the current study, within the older adult group, greater ELS was associated with larger central P300 amplitudes. This pattern was primarily driven by activity at the left (non-dominant) hemisphere site, most likely corresponding with pre-central and/or post-central gyri (Kim et al., 2007; Rich and Gillick, 2019; Rojas et al., 2018). These patterns suggest that greater activation in anterior regions, particularly from the non-dominant hemisphere, may be necessary to compensate for underlying neural deficits associated with the combination of older age and greater exposure to ELS to enable high-level performance even on this simple go/no-go task (Reuter-Lorenz and Park, 2014). Thus, using a more demanding high-accuracy go/no-go or stop-signal task may reveal important early compensatory

activation by increasing frontal-dependent conflict and inhibition processes (Reuter-Lorenz and Cappell, 2008).

Some studies have reported larger central P300 amplitudes in older compared to younger adults during a go/no-go task with equi-probable stimuli (i.e., 50 % go, 50 % no-go; Hong et al., 2014; Vallesi, 2011) and on no-go trials with low conflict only (Vallesi and Stuss, 2010; Vallesi et al., 2009). Larger central P300 amplitudes are most evident in older adults during very low-demand tasks, such as those with equi-probable stimuli, which create a weaker prepotent response. As task demand increases, central P300 resources are depleted, resulting in smaller central P300 amplitudes, and more resources are recruited from frontal and bilateral regions (Elverman et al., 2021; Paitel and Nielson, 2021; Reuter-Lorenz and Cappell, 2008). Importantly, when task demand surpasses a given threshold, decreased activation is evident across sites, concurrent with behavioral performance deficits, indicating that these compensatory resources are finite (Reuter-Lorenz and Cappell, 2008).

Beyond the effects of age group, smaller inhibitory P300 amplitudes in central-parietal regions were predicted by greater exposure to ELS. The few existing ERP studies assessing ELS with inhibitory control tasks primarily investigated these processes in childhood and adolescence. While findings were inconsistent overall, some studies showed that youth exposed to early-life stress had smaller N200 (Bruce and Kim, 2020; Loman et al., 2013), error-related negativity (ERN; Loman et al., 2013), and P300 (McDermott et al., 2012) amplitudes during go/no-go and Flanker tasks, highlighting early effects of ELS on inhibitory control networks. ERP research on the neural effects of ELS in adulthood is notably sparse, despite evidence of lasting biopsychosocial effects of ELS into later life (e.g., Fogelman and Canli, 2018; Korten et al., 2014; Levine et al., 2015; Pechtel and Pizzagalli, 2011; Ritchie et al., 2009). Consistent with the current findings, one ERP study with young and middle-adult participants revealed smaller no-go P300 activation localized to right anterior cingulate cortex and precentral gyri as well as bilateral medial frontal cortex and superior frontal gyri in participants with childhood trauma (Kim et al., 2018). During an equi-probable go/no-go task, early-life physical and sexual abuse correlated with delayed P300 latency and larger P300 amplitudes. Larger amplitudes in those with greater ELS suggests compensatory activation akin to the patterns found in healthy older versus younger adults during equi-probable no-go tasks (Howells et al., 2012; Vallesi, 2011).

To our knowledge, the only other study to investigate ERPs in older adults with exposure to ELS used a combined sample of healthy older adults and those with mild cognitive impairment (Wang et al., 2016). This study showed that physical neglect was associated with later P300 latencies and smaller P300 amplitudes during a simple auditory oddball task (Wang et al., 2016). The current findings address an important gap by examining P300 patterns within a group of healthy adults, using a more robust regression analysis, and analyzing ERP activation during a simple and high-accuracy inhibitory control task. The domain of inhibitory control is particularly important for investigating age by ELS interactions given that poorer accuracy and slower reaction time during inhibitory control tasks are well-established in children with exposure to ELS (e.g., Bruce and Kim, 2020; Carvalho et al., 2020; Cowell et al., 2015; Giuliano et al., 2018), and have also been described in adolescents (Kirke-Smith et al., 2014) and adults (Daly et al., 2017; Marshall

et al., 2016). The impact of inhibitory deficits with age is likely critical, with evidence that inhibitory deficits may in fact mediate more global age-related cognitive decline (Sweeney et al., 2001), and that they are a primary predictor of deficits in activities of daily living (i.e., ADLs), a critical requirement for independent living (O'Connor and Boyle, 2007).

The current findings suggest that exposure to ELS has long-lasting and dynamic neural implications that interact with normative neurocognitive aging processes. Whereas participants with lower to moderate ELS demonstrated the expected pattern of smaller central-parietal P300 amplitudes in older vs. young adults, amplitudes in those with higher ELS did not significantly differ by age group. Within young adults, greater ELS was associated with smaller central-parietal P300 amplitudes, indicative of potential early deficits in underlying inhibitory networks (e.g., Harms et al., 2017; Lim et al., 2015; Mueller et al., 2010). Notably, despite this association between greater ELS and lower P300 amplitudes, young adults were able to maintain high task performance without needing to recruit additional brain regions. However, within older adults, greater ELS was associated with modestly larger central P300 amplitudes, suggesting a greater demand on anterior resources for high-accuracy task performance in older adults with greater ELS exposure (Reuter-Lorenz and Park, 2014). Importantly, compensatory resources are finite, and while greater reliance on compensatory neural resources may be successful with low to moderate cognitive demands, they will likely become insufficient when faced with a higher cognitive load and/or advanced neurological aging (Reuter-Lorenz and Cappell, 2008; Reuter-Lorenz and Park, 2014). Thus, greater ELS may confer a heightened risk for earlier signs of age-related cognitive decline and greater risk for pathological aging (Reuter-Lorenz and Cappell, 2008; Reuter-Lorenz and Park, 2014; Wang et al., 2016).

3.1. Limitations and future directions

The current study included relatively small samples, and thus should be interpreted with caution. We hope that these findings will be used to guide future research with larger samples and thus, more statistical power, to validate and further tease apart the interactions between ELS and aging. Studies with larger samples will also provide an opportunity to explore the impact of varying degrees of task demand on the effect of age*ELS interactions on inhibitory ERPs. The current study used a high-accuracy task, which indeed is particularly well-suited to capture potential compensatory recruitment patterns compared to higher-demand tasks where such resources are expected to be depleted, concurrent with deficits in task performance (for review see Reuter-Lorenz and Cappell, 2008). Yet, higher-demand inhibitory control tasks that control for working memory load (e.g., stop-signal paradigm) may be particularly useful for examining contributions of frontal lobe activity (Elverman et al., 2021; Paitel and Nielson, 2021) and may furthermore reveal similar compensatory patterns in young adults with high ELS at a higher cognitive load (Reuter-Lorenz and Cappell, 2008). The role of conflict monitoring, detection, and resolution, for which the N200 component may be particularly important, would also be better examined with a more complex task (Elverman et al., 2021; Huster et al., 2010; Paitel and Nielson, 2021; Paitel et al., 2021).

While the current findings fit within the framework of compensatory recruitment (Reuter-Lorenz and Park, 2014), it is also possible that these results reflect alternative interpretations, such as neural dedifferentiation. The argument for dedifferentiation suggests that increasing age is associated with decreased activation in functionally specialized neural networks and increased activation in more general networks (for review see Koen and Rugg, 2019). Future studies with sufficient sample sizes may be able to address these theories by assessing the impact of age and ELS on the relationship between neural activation in different networks and at varying levels of task performance.

An important methodological point for future research is the advantages of using interviews to assess life stress (vs. self-report metrics). This approach allows for gathering of more detailed and precise information surrounding ELS, including distinguishing between acute events vs. chronic stressors and major vs. minor stressors as well as creating the opportunity for clarification and elaboration (Harkness and Monroe, 2016).

Finally, because using ERPs to assess interactions between ELS and age is relatively novel, immediate work characterizing healthy, normative cognitive aging is important. Given the relevance of inhibitory control and the P300 component to pathological aging, future research examining how these relate to mild cognitive impairment, Alzheimer's disease, and healthy adults with genetic risk for Alzheimer's may be particularly important (see Paitel et al., 2021).

4. Conclusions

Given the well-established effects of ELS on inhibitory control in children (e.g., Bruce and Kim, 2020; Carvalho et al., 2020; Cowell et al., 2015; Lund et al., 2020), knowledge of neural effects of ELS in earlier life (e.g., Lim et al., 2015; Mueller et al., 2010), and the critical role of inhibitory control in healthy cognitive aging (e.g., O'Connor and Boyle, 2007; Salthouse et al., 2003; Sweeney et al., 2001), the current study sought to address the impact of ELS on neural activation during successful no-go inhibition in young adults and healthy older adults. Our findings showed the typical pattern of smaller central-parietal P300 amplitude in older vs. young adults (Elverman et al., 2021; Paitel and Nielson, 2021; Patel and Azzam, 2005), but this pattern was consistent only in those with low to moderate exposure to ELS. Greater ELS was associated with smaller central-parietal P300 amplitudes within young adults but larger central P300 amplitudes within older adults. Given very high task accuracy across participants, these patterns suggest that older adults with greater ELS exposure recruited more neural resources to support inhibitory performance evaluation and adaptation (Park and Reuter-Lorenz, 2009; Reuter-Lorenz and Park, 2014). Because the current task uses a simple, high-accuracy paradigm, it is likely these differences would be more pronounced during tasks with a greater demand on working memory, with concurrent deficits more likely to be seen in behavioral performance as these compensatory resources are depleted (Reuter-Lorenz and Cappell, 2008). Thus, the current study indicates that older adults with greater exposure to ELS may be at greater risk for age-related cognitive declines. With this in mind, we advocate for inclusion of a simple screening measure for ELS in neuroimaging research, particularly in work with older adults.

Data availability

Data will be made available on request.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

The authors wish to gratefully acknowledge contributions to study recruitment and data collection from Developmental Decision-Making Lab research assistants: Jonathan Carroll, Christine Chen, Corinna Jauregui, Sarah Jensen, Claire Rosenberger, Gretchen Stutz, Jessica Tooley, Shelby VanRossum, Stephanie Weigman, and Max Whealon.

Funding

This study was supported in part by the Ronald E. McNair Post-Baccalaureate Achievement Scholars Program (ERP & RAL; CFDA Number 84.217A), a Collaborative Research Grant from the Center for Undergraduate Research at St. Norbert College (ERP), and the National Center for Advancing Translational Sciences, National Institutes of Health (UL1TR001436, TL1TR001437; the study contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH).

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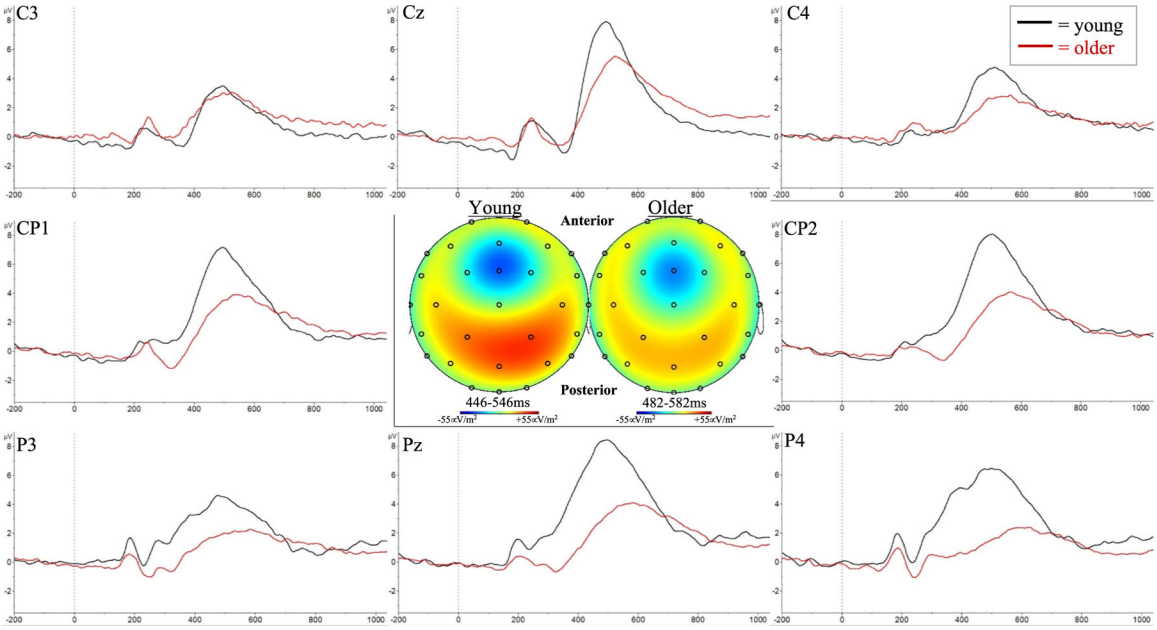

Fig. 1.

Correct no-go trial grand average waveforms by age group (**black** = young; **red** = older). x-axis = ms; y-axis = μv. Current source density maps show activity in the 100 ms window surrounding P300 peak by age group, both scaled to +/−55μv/m^2. Both groups had a posterior P300 maxima, with largest magnitude in young. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

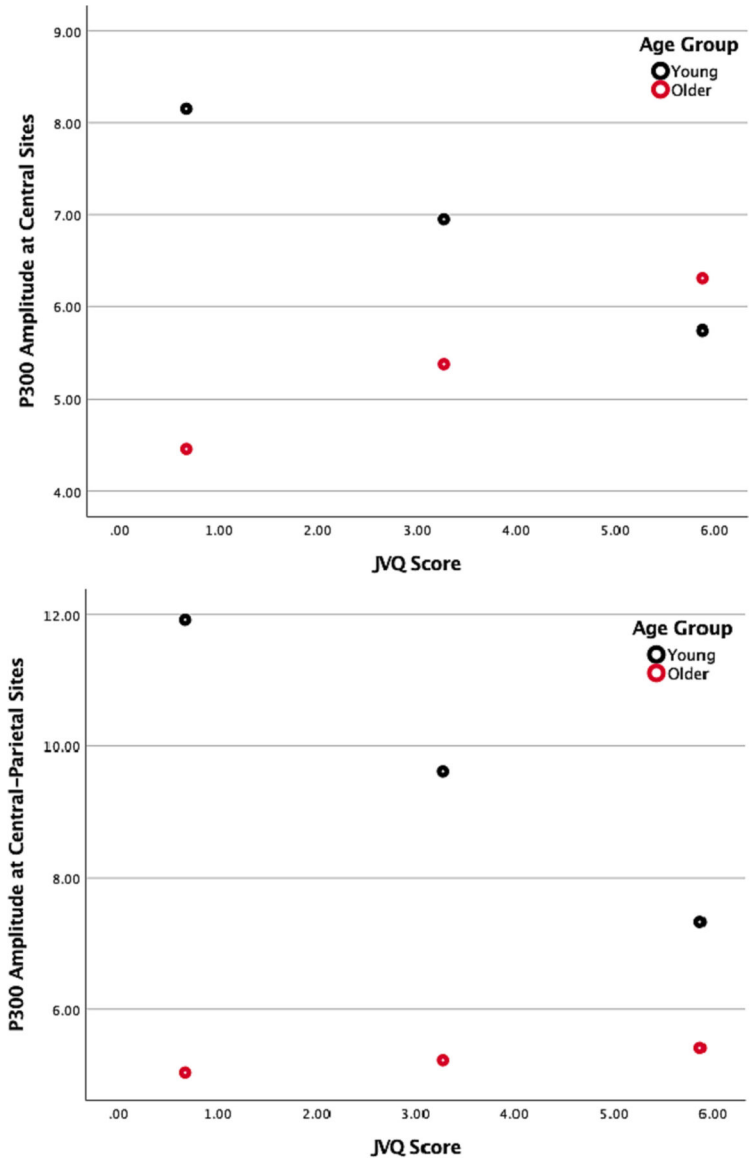

Fig. 2.

Plots showing the Johnson-Neyman technique for models with significant age group*ELS interaction terms. Red dots = older adults; black = young. Dots are positioned along the x-axis corresponding with JVQ scores −1 *SD* below the mean (left), at the mean (center), and +1 *SD* above the mean (right). Older age predicted smaller P300 amplitudes, *only* in those with lower (left; −1 *SD*) and moderate (middle; mean) JVQ scores. P300 amplitudes were not significantly predicted by age in those with higher ELS (right; +1 *SD*). See Table 3 for statistics, Table 4 for P300 values by age group. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 1

Demographics by age group (mean (±SD)).

	Older adults	Young adults
Age (years)	72.2 (8.6) ***	19.9 (1.0) ***
Education (years)	15.5 (2.2) *	14.1 (0.6) *
Sex (% female)	38 %	55 %

Note: Significant age group difference (older > young).

* $p < .05$.

*** $p < .001$.

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Table 2

Behavioral statistics for the go/no-go task.

	Older adults (*n* = 20)	**Young adults (*n* = 13)**	**Lower JVQ (*n* = 18)**	**Higher JVQ (*n* = 15)**
Overall accuracy (%correct)	98.35 %	98.69 %	98.61 %	98.33 %
No-go accuracy (%correct)	94.30 %	93.69 %	94.78 %	93.20 %
Go target RT (ms)	548.92 *	465.98 *	526.83	503.55
No-go commission RT (ms)	440.00 *	338.99 *	398.82	392.84

Note: JVQ groups are based on a median split, with those endorsing three or fewer items in the “lower” group and those endorsing four or more items in the “higher” group. Note that the paper’s primary analyses analyzed JVQ score as a continuous metric. This arbitrary grouping was used only to check for an effect of JVQ score on behavioral task performance. Go/no-go task accuracy did not differ by age group or JVQ score. Older adults had slower response time (RT). Significant age group difference (older > young).

* $p < .05$.

Table 3

Multiple regression models predicting P300 amplitude by region.

		Contribution of each variable		
	Coefficient	***SE***	***t***	***p***
Central				
Age	**−4.24**	**1.22**	**−3.46**	**0.002**
JVQ	**−1.28**	**0.44**	**−2.92**	**0.007**
Age*JVQ	**0.82**	**0.28**	**2.92**	**0.007**
		Model: R^2 = 0.30; MSE = 3.19; *F* = 4.10; *p* = .015		
Central-parietal				
Age	**−7.53**	**1.55**	**−4.85**	**0.000**
JVQ	**−1.84**	**0.56**	**−3.31**	**0.003**
Age*JVQ	**0.95**	**0.35**	**2.70**	**0.012**
		Model: R^2 = 0.49; MSE = 5.11; *F* = 9.18; *p* < . 001		
Parietal				
Age	**−6.55**	**1.54**	**−4.25**	**0.000**
JVQ	**−1.13**	**0.55**	**−2.04**	**0.051**
Age*JVQ	0.50	0.35	1.41	0.170
		Model: R^2 = 0.50; MSE = 5.05; *F* = 9.79; *p* < .001		

Note: Bonferroni correction was applied to adjust for multiple comparisons. Models with $p < .017$ (**bolded**) met the adjusted threshold.

Table 4

P300 amplitude (μV) during accurate no-go trials.

Region	Young adults		Older adults	
	Mean	***SD***	**Mean**	***SD***
Central	6.22	2.05	5.02	1.93
Centro-Parietal	8.24	3.25	5.15	2.14
Parietal	7.76	2.96	3.96	2.00

Note: *SD* = standard deviation; Central = C3, Cz, C4; Centro-parietal = CP1, CP2; Parietal = P3, Pz, P4.

Table 5

Pearson bivariate correlations between ELS and P300 amplitudes by age group.

	Young adults	Older adults
Site		
C3	−0.30	0.47*
Cz	−0.51^	0.15
C4	−0.65*	0.35
CP1	−0.72**	0.17
CP2	−0.69**	−0.08
P3	−0.36	−0.04
Pz	−0.60*	−0.23
P4	−0.53^	−0.11

C = central; CP = centro-parietal; P = parietal; odd numbers (i.e., 1, 3) = left hemisphere; z = midline; even numbers (i.e., 2, 4) = right hemisphere.

* $p < .05$.

** $p < .01$.

^ $p = .06–.08$.