

Article

Temporal Dynamics of Event-Related Potentials During Inhibitory Control Characterize Age-Related Neural Compensation

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Abstract: Aging is accompanied by frontal lobe and non-dominant hemisphere recruitment that supports executive functioning, such as inhibitory control, which is crucial to all cognitive functions. Yet, the spatio-temporal sequence of processing underlying successful inhibition and how it changes with age is understudied. Thus, we assessed N200 (conflict monitoring) and P300 (response inhibition, performance evaluation) event-related potentials (ERPs) in young and healthy older adults during comparably performed successful stop-signal inhibition. We additionally interrogated the continuous spatio-temporal dynamics of N200- and P300-related activation within each group. Young adults had left hemisphere dominant N200, while older adults had overall larger amplitudes and right hemisphere dominance. N200 activation was biphasic in both groups but differed in scalp topography. P300 also differed, with larger right amplitudes in young, but bilateral amplitudes in old, with old larger than young in the left hemisphere. P300 was characterized by an early parieto-occipital peak in both groups, followed by a parietal slow wave only in older adults. A temporally similar but topographically different final wave followed in both groups that showed anterior recruitment in older adults. These findings illuminate differential age-related spatio-temporal recruitment patterns for conflict monitoring and response inhibition that are critically important for understanding age-related compensatory activation.

Keywords: inhibitory control; executive function; event-related potentials; electroencephalography; N200; P300; cognitive aging; neural recruitment

1. Introduction

Various cognitive processes including episodic memory, spatial reasoning, and executive functioning tend to decline in the course of typical, healthy aging [1-3]. Deficits in executive functioning, particularly the ability to withhold attentional or behavioral responses to irrelevant or interfering stimuli [i.e., inhibitory control; 4], have received attention as potential mediators of more global cognitive decline [5,6]. However, the temporal sequence of neural activity underlying successful inhibition and, in particular, the effects of age on this sequence, are not understood [7].

Inhibitory control is commonly assessed using go/no-go and stop-signal tasks. In go/no-go paradigms participants respond to go stimuli (e.g., the letter A) while selectively inhibiting responses to no-go stimuli (e.g., the letter B), where the participant knows in advance which stimulus is to be inhibited (i.e., the letter B always signals inhibition). Thus, successful performance requires an “internal,” self-driven response selection process, aided by learning and memory [8,9]. In stop-signal tasks, participants respond to ‘go’ stimuli (e.g., the letter A) unless they are followed by an unpredictable stop-signal. The prepotent response to the ‘go’ stimulus is activated and must be effortfully retracted based on the externally generated stop-signal. This is therefore a better index of response inhibition than no-go [9]. Indeed, our recent study demonstrates the advantages of using the

stop-signal vs. no-go task in revealing age- and Alzheimer's disease risk-related differences in cognitive event-related potentials [ERPs; 10].

Neuroimaging studies of inhibitory control, typically using functional-MRI (fMRI), have implicated multiple sequential subprocesses involving right inferior frontal gyrus (IFG), insula, and cingulate cortex that are necessary for successful inhibition: interference/conflict resolution, action withholding, and action cancellation [11]. In addition, left pre-SMA and superior parietal gyrus contribute specifically to interference (i.e., conflict) resolution. However, although fMRI is well-equipped to determine which brain regions are active during a given task, the very slow (i.e., seconds-long) impulse response function greatly limits knowledge about the timecourse of activity in the relevant networks [12]. Temporal precision is particularly important for studying inhibitory control, for which relevant neural activity primarily occurs within the first ~400ms [7].

In contrast to fMRI, research with temporally precise event-related potentials (ERPs) has isolated two key components of inhibition: N200 and P300 [13-15]. First, the N200 is a fronto-central negativity that occurs approximately 150-350ms following an inhibitory cue. Source analyses have highlighted the IFG and dorsal anterior cingulate as likely generators [7,16-18]. Despite earlier conceptualization of N200 as reflecting response inhibition *per se* [19-21], more recent work suggests N200 is specifically tied to conflict monitoring and alerting of the need for inhibition *prior to* the motor 'response' underlying inhibition [18,22,23]. Indeed, N200 amplitudes during inhibitory control tasks are associated with concurrent increases in theta power, indicative of cognitive processes that precede motor processing [18], and are evident even on trials with conflict resolution that do not require motor inhibition [24].

The second component important in inhibitory control, P300, is a positive-going wave that occurs ~300-500ms post-inhibitory stimulus. In most tasks, P300 is maximal over parietal electrodes. However, in the context of inhibition it is often larger over fronto-central sites such as those corresponding with the precentral gyrus, pre-SMA, IFG, and cingulate cortex [i.e., no-go anteriorization; 25,26]. This activity is specifically linked to response inhibition, performance monitoring and evaluation, and error correction [17,18,23,24,27]. In line with this conceptualization, the inhibitory P300 is associated with increased delta power, thought to be associated with motivated attention and performance evaluation [18].

Understanding of the neural underpinnings of age-related differences during no-go and stop-signal inhibitory control tasks is relatively limited. This is particularly true for stop-signal tasks, despite their ability to better control for task demand-related activation [7,9]. We recently examined this with ERPs at midline electrodes. Using inhibitory tasks with high and equal level group accuracy, we found older adults had smaller posterior but larger frontal P300 amplitudes and overall larger N200 amplitudes specific to the stop-signal task [10]. Other inhibition studies have also reported larger or comparable frontal-central P300 activity across age groups [28-30], some also with smaller central-posterior P300 in older adults [15,31]. These patterns suggest age-related compensatory frontal recruitment in older adults [32,33], particularly in anterior relative to posterior sites during successful inhibition [34]. These results are consistent with findings from fMRI studies, which show greater frontal than posterior activation, as well as greater bilateral activation (particularly in frontal lobes) in older than young [32,34-39]. This evidence of recruitment has been associated with better maintenance of high-level cognitive performance [32,33,40]. Unfortunately, relevant ERP studies have thus far been limited to midline electrodes, with one exception. Hong, Sun, Bengson and Tong [30] reported elevated frontal N200 and P300 activation in older adults during response inhibition (i.e., no-go) that was particularly right lateralized.

Despite the conceptualization of inhibition as reliant upon a number of interacting subprocesses [7,11], ERP research has examined inhibitory control by collapsing data across epochs of several hundred milliseconds (i.e., ~100-350ms for N200, ~300-500+ms for P300), rather than taking advantage of its unique ability to capture data with millisecond-level precision. Thus, a finer-grained temporal analysis of recruitment during inhibition,

along with inclusion of a wider array of lateral electrodes to examine specific hemisphere differences could better characterize and disentangle the role of age-related recruitment in the each of the specific subprocesses of inhibitory control [12,35,37,40,41]. The current study sought to address these gaps. We first analyzed N200 and P300 amplitudes using traditional ERP time windows in young and older adults during a stop-signal task. The groups had comparable accuracy to preclude differences based on task difficulty. We then expanded the analysis to interrogate the continuous waveform temporal dynamics at all 64 electrodes within each age group, making a qualitative comparison. Based upon existing research and compensatory models of cognitive aging [32], we hypothesized that successful stop-signal inhibition would produce left hemisphere dominant N200 (conflict monitoring) and right hemisphere dominant P300 (response inhibition) amplitudes in young adults. We anticipated that older adults would exhibit bilateral activation, specifically attributable to recruitment of the non-dominant hemisphere (i.e., greater right hemisphere N200 and left hemisphere P300 recruitment). We expected these effects to be greatest at frontal and fronto-central electrodes.

2. Materials and Methods

2.1. Participants

Healthy older adult participants ($n = 49$) were recruited via newspaper advertisements and compensated monetarily. Young adults were recruited from psychology classes offering course credit ($n = 42$). The Mattis Dementia Rating Scale – Second Edition (DRS-2) was used to assure intact cognition in older adult participants, with a cut-off score of 130/144 for intact status [42-44]. One older adult participant was excluded due to a DRS-2 score below 130, reducing the older sample to 48. The depression subscale of the Brief Symptom Inventory (BSI; average six items scored 0 (none)- 4 (severe)) was used to assure normal mood and group comparability.

2.2. Materials

2.2.1. Stop-signal task

The stop-signal task consisted of a serial stream of letters visually presented at a rate of 750ms per letter with an interstimulus interval of 0ms. First, in the go condition, participants were instructed to press the space bar every time the letter “r” or “s” was presented (504 stimuli, 78 targets). This condition served to establish a prepotent response. Thereafter, in the stop condition, participants were instructed to press the space bar when the letter “r” or “s” appears (684 stimuli, 81 targets), except when the stimulus was followed by a red flash (i.e., the stop-signal, $n = 36$; flash duration = 100ms; stop-signal delays = 125ms and 200ms rather than a ‘staircase’ procedure to prevent predictability but also maintain high accuracy; see [10]). Outcome measures included target and inhibitory accuracy; target response time (RT); and stop-signal reaction time (SSRT), which is the latency for the process involved in stopping the motor response, as estimated from the distribution of observed target RTs combined with the inhibition function [8,45] .

2.2.2. EEG data acquisition and ERPs

Continuous EEG data were collected using a 64-electrode Brain Products actiCAP arranged according to the extended International 10-20 System with ground at AFz and reference at FCz. Data were recorded using Neuroscan SynAmps2 with impedances kept under 50 k Ω . EEG data were recorded in DC mode with a low-pass hardware filter at 100 Hz and a 500 Hz sampling rate using Neuroscan software (Scan 4.5). Continuous EEG data were processed off-line using EEGLAB (Version 14.1.0) software via MATLAB (Version 7.12, The MathWorks). The data were re-referenced to a common average of all

electrodes and filtered using a band-pass filter from 0.5 to 100 Hz and a notch-filter from 59 to 61 Hz.

Continuous data were visually inspected, and channels were rejected as necessary to eliminate channel-level artifacts. Data for rejected channels were interpolated based on an average of surrounding electrodes. Next, an Adaptive Mixture Independent Component Analysis [AMICA; 46] was used to decompose data into individual components. Components reflecting eye blink, other ocular movements, and muscle contraction were rejected and removed from the data based on visual inspection. These data were then segmented from 100 ms prior to stop-signal presentation (i.e., the red flash) to 1500 ms after stimulus onset. A baseline correction of 100 ms pre-stimulus was applied to all epochs. Epochs were then examined and rejected as appropriate based on visual inspection. Remaining epochs were averaged and an additional low-pass filter at 20 Hz (zero-phase, 4th-order, Butterworth) was applied. Peak amplitude was computed at frontal (F3, F4), frontal-central (FC3, FC4), central (C3, C4), and parietal sites (P3, P4) between the range of 100-350ms for the N200 component and 300-700ms for P300. To investigate the continuous temporal dynamics of these ERPs, grand average waveforms were computed in open-source Brainstorm software [47] using the full 64-channel array. Due to both the typical age-related delays in N200 and P300 components and notable variability of peak timing even within age groups (particularly for P300), the current temporal investigation describes the spatio-temporal patterns underlying group differences in the average waveforms without separate quantitative analysis of the continuous data.

2.2.3. *Procedure*

ERPs during the stop-signal task were collected as part of a larger study. Participants completed two testing sessions, separated by approximately one week, with individualized testing on both occasions. Participants were seated in front of a computer following EEG cap placement and were instructed to limit gross motor movements and speech as much as possible to reduce noise in the EEG signal. The stop-signal task was presented in MATLAB (version 7.12, The MathWorks). Instructions were read aloud as they appeared on the screen, and the participant had the opportunity to ask questions regarding task instructions. Corrective feedback was provided throughout the practice blocks of each task condition, but no feedback was provided during test blocks. All procedures were approved by the University’s Institutional Review Board.

3. Results

3.1. *Descriptive statistics and excluded data*

Two older adult participants and one young adult participant were excluded from analyses due to technical issues during collection of EEG data. These exclusions resulted in a final sample of 46 older and 41 younger adult participants. Sample demographics are presented in Table 1. Aside from age, the groups were comparable except that the older group had, on average, one more year of formal education than young adults, which was statistically significant.

Table 1. Demographics by age group (mean (± SD)).

	Older Adults (<i>n</i> = 46)	Young Adults (<i>n</i> = 41)
Age (years)	79.63 (4.68) ^a	19.95 (2.74) ^a
Education (years)	14.80 (2.65) ^a	13.77 (1.16) ^a
Sex (% female)	73.91%	73.17%
DRS-2	138.26 (2.88)	–
BSI-Depression	0.39 (0.53)	0.56 (0.61)

Note. DRS-2 = Dementia Rating Scale-Second Edition (maximum possible score = 144; minimum score allowed = 130); BSI-Depression = Brief Symptom Inventory, depression subscale. ^aSignificant age group difference (older > young), $p < .05$.

3.2. Task performance analyses

Task performance data are shown in Table 2. The groups did not significantly differ on task accuracy measures, either in the preliminary go task or during the stop-signal task, for target responses or the withholding of responses (i.e., inhibition). However, as expected, older adults had slower responses to targets and a slower SSRT than young adults.

Table 2. Descriptive statistics for the stop-signal task by group (mean (\pm SD)).

	Older Adults (<i>n</i> = 46)	Young Adults (<i>n</i> = 41)
<u>Go Task (prepotency):</u>		
PCTT	99.52 (0.83)	99.50 (1.51)
Target RT (ms)	678.71 (47.72) ^a	596.26 (39.51) ^a
<u>Stop-Signal Task:</u>		
PCTT	98.58 (2.63)	98.16 (2.52)
PCIT	75.00 (11.92)	77.64 (12.79)
Target RT (ms)	769.72 (63.36) ^a	684.01 (39.31) ^a
SSRT (ms)	541.47 (36.89) ^a	450.59 (44.94) ^a

Note. PCTT = Percent Correct Target Trials; PCIT = Percent Correct Inhibitory Trials; SSRT = Stop-Signal Reaction Time; ^a $p < .001$.

3.3. ERP analyses

Repeated measures 2x2x4 ANOVAs including Age (Young, Older), Hemisphere (Left, Right), Site (F, FC, C, P) were conducted to assess N200 and P300 amplitude. Greenhouse Geisser correction was applied where appropriate. Primary results of interest included main effects of Age and interactions of Age with Hemisphere and/or Site. Spatio-temporal dynamics of the continuous waveforms by age group was then examined qualitatively for the 64-channel waveforms for each group.

3.3.1. N200 component

N200 analyses revealed a significant main effect of Age ($F(1,85) = 6.85$, $p < .01$, $\eta_p^2 = .08$), with overall larger amplitudes in older adults ($M_{\text{older}} = -3.02$, $M_{\text{younger}} = -2.29$). Age by Hemisphere ($F(1,85) = 14.21$, $p < .001$, $\eta_p^2 = .14$) and Age by Site ($F(2,2,85) = 6.68$, $p < .001$, $\eta_p^2 = .07$) interactions were significant, with a non-significant trend for the three-way interaction ($F(2,38,85) = 2.56$, $p = .07$, $\eta_p^2 = .03$). The Age by Hemisphere interaction revealed greater amplitude in older compared to young adults specifically in the right hemisphere; older adults had right hemisphere dominant N200 amplitudes, while those of young adults were larger in the left hemisphere (see Table 3). The Age by Site interaction showed that older adults' amplitudes were significantly larger than young at central and parietal sites,

with a non-significant trend at frontal sites. Furthermore, young adults had an anterior N200 maximum, while older adults had maxima over both frontal and central sites, with less differentiation across the anterior to posterior sites (see Table 3; Figure 1).

Table 3. Summary of significant individual contrasts from *Age* by *Hemisphere* interactions for N200 and P300 amplitude, showing group contrast effects and hemisphere contrast effects.

		Group Contrasts				Hemisphere Contrasts		
		Direction	<i>F</i>	η_p^2		Direction	<i>F</i>	η_p^2
N200	Left	-			Young	L > R [^]	7.81	0.08
	Right	O > Y*	38.571	0.25	Older	R > L [▷]	6.4	0.07
	Frontal	-						
	Fronto-central	-						
	Central	O > Y*	34.54	0.29				
	Parietal	O > Y [▷]	5.38	0.06				
P300	Left	O > Y [▷]	5.63	0.06	Young	R > L*	50.35	0.37
	Right	Y > O*	22.47	0.21	Older	-		
	Frontal	-						
	Fronto-central	-						
	Central	-						
	Parietal	Y > O*	11.47	0.12				

Note. O = older adults; Y = young adults. L = left hemisphere (electrode 3); R = right hemisphere (electrode 4). * $p < .001$, [^] $p < .01$, [▷] $p < .05$.

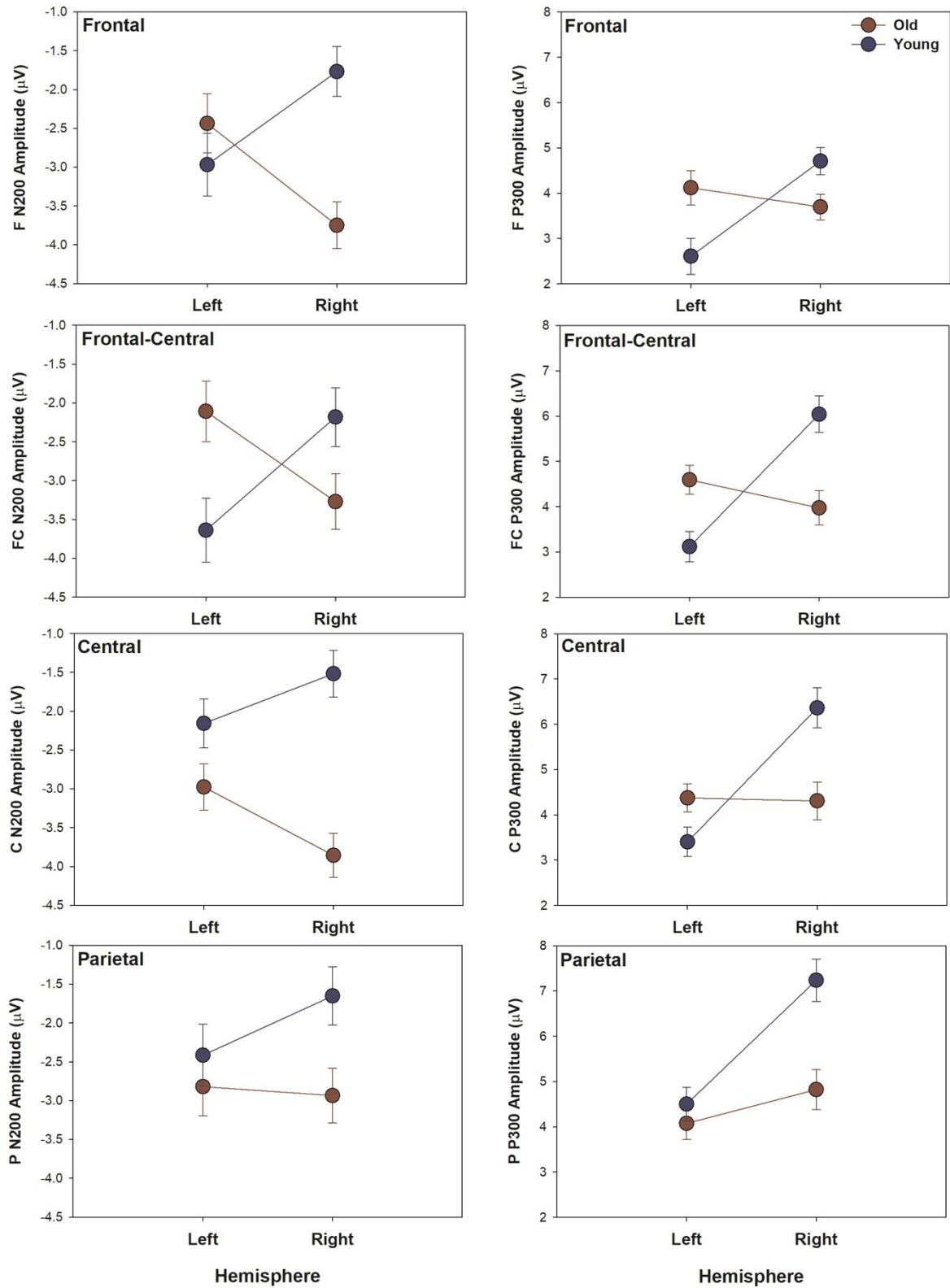


Figure 1. Average ERP amplitude ± SEM is shown by age group at left (electrode 3) and right (electrode 4) hemisphere sites for frontal through parietal regions (F = frontal; FC = fronto-central; C = central; P = parietal) for N200 (left column) and P300 (right column) components. Corresponding significant group differences are specified in Table 3.

Activation underlying the N200 component in the young group consisted of two short peaks (see Figure 2). The first phase of activation included left parieto-occipital sites (ordered by largest magnitude: PO9, PO7, P7), with the average peak at ~150ms. The second phase consisted of right parieto-occipital activation (PO8, P8, P6) and was largest at ~165ms. Older adults similarly had two phases of activation underlying the N200 component. As expected, these peaks were later than for young adults, but they also had a different scalp topography. The first phase included midline and left hemisphere centro-parietal sites (Cz, C1, CPz, CP1, C3, CP3), with the average peak ~245ms. Electrode Cz was among the first sites to activate, had the largest amplitude during the N200 window, and remained active for the longest time. The second phase of activation included more anterior and right hemisphere activation (C2, FCz, CP2, FC1, FC2, C4, CP4), peaking ~254ms.

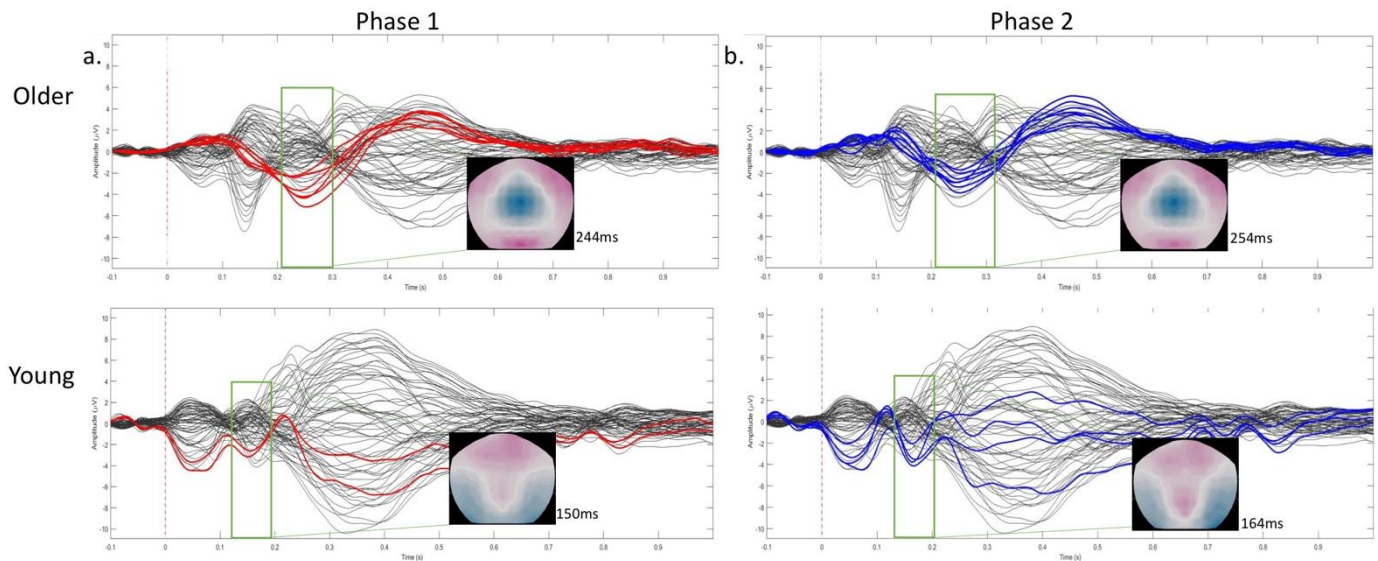


Figure 2. Continuous, 64-channel ERP waveforms (each tracing = individual electrode) in older (top) and young (bottom) adults. Scalp topography maps within the corresponding time windows display all activity at the scalp (i.e., not phase-isolated; time stamp relative to onset of stop-signal; scaled at $\pm 7.477 \mu V$). Plots include the pre- through post-stimulus recording epoch (y-axis = μV ; x-axis = ms). Two phases of activation were apparent within the N200 window in both groups, shown with green boxes. The phases were later in old than young and also differed in scalp topography. **Phase 1** (left panel, a) was largest in the left hemisphere in both groups, but in old, there was a left/midline centro-parietal negativity (largest at Cz, C1, CPz, CP1; red (top)), while in young, there was a left parieto-occipital negativity (PO9, PO7, P7; red (bottom)). **Phase 2** (right panel, b) was largest in the right hemisphere in both groups, but was anterior in old (C2, FCz, CP2, FC1; blue (top)) and parieto-occipital in young (PO8, P8, P6; blue (bottom)).

3.3.2. P300 component

Main effects of *Hemisphere* ($F(1,85) = 24.84$, $p < .001$, $\eta_p^2 = .23$) and *Site* ($F(2,2,85) = 11.90$, $p < .001$, $\eta_p^2 = .12$) were significant, indicating overall larger amplitudes in the right hemisphere, and in the parietal region, respectively. There was also a non-significant trend for a main effect of *Age* ($F(1,85) = 3.11$, $p = .08$, $\eta_p^2 = .04$), characterized by somewhat larger P300 amplitude in the young group. These effects were clarified by significant *Age* by *Hemisphere* ($F(1,85) = 28.46$, $p < .001$, $\eta_p^2 = .25$) and *Age* by *Site* ($F(2,2,85) = 4.48$, $p < .01$, $\eta_p^2 = .05$) interactions. The interactions revealed right hemisphere dominant P300 amplitudes in the young adults with no significant difference between hemispheres in the older adults (i.e., bilateral activation; see Table 3). That is, consistent with their right hemisphere dominance, young adults had larger amplitudes than old in the right hemisphere, but older adults had larger amplitudes than young in the left hemisphere. Furthermore, the young adults had maximal amplitudes at the parietal site, while older adults had a more diffuse pattern of activation that was largest at parietal sites, but not significantly different across sites. The age groups significantly differed

only in the parietal region, with greater P300 amplitude in young than old (see Table 3; Figure 1).

Investigation of the continuous waveform data in young adults revealed two phases of P300 peaks (see Figure 3). First was a clear early peak around 225ms primarily at left hemisphere parieto-occipital sites (ordered by largest magnitude: POz, Oz, PO3, O1, PO4, O2, PO7, P5). Many of these sites remained active during the second phase of activation, either peaking again at a smaller amplitude or sustaining activation in a “plateau” throughout the second phase. This second phase lasted ~250-550ms (maximal ~375ms), and was largest at midline and right hemisphere centro-parietal sites, with some fronto-central activation (CPz, Pz, Cz, P2, CP2, P1, CP1, C2, C1, CP4, P4, FCz, FC2, P3, FC1). Older adults exhibited three phases of activation, rather than two. They had an early P300-related peak (~320ms), similar to young adults, consisting of bilateral parieto-occipital sites (POz, PO4, PO3, Oz, O2, O1, P6, P5, PO8, PO7). These are the same electrodes that were active for an early visual negativity (see Figure 3). A second slow wave, present only in the older adult group, consisted of bilateral parietal activation (Pz, P2, P1, P4, P3) from ~300-550ms. Activity at these sites remained active throughout the duration of a third peak consisting primarily of bilateral anterior sites, with some centro-parietal (FCz, FC1, F1, Fz, FC2, Cz, F2, CPz, CP2, C2, C1, CP1, FC3, C4, F3, C3, FC4, CP3). This third phase was largest, lasting overall ~360-600ms (maximal ~460ms) and was similar but later, bilateral, and more anterior than the second phase produced by young adults.

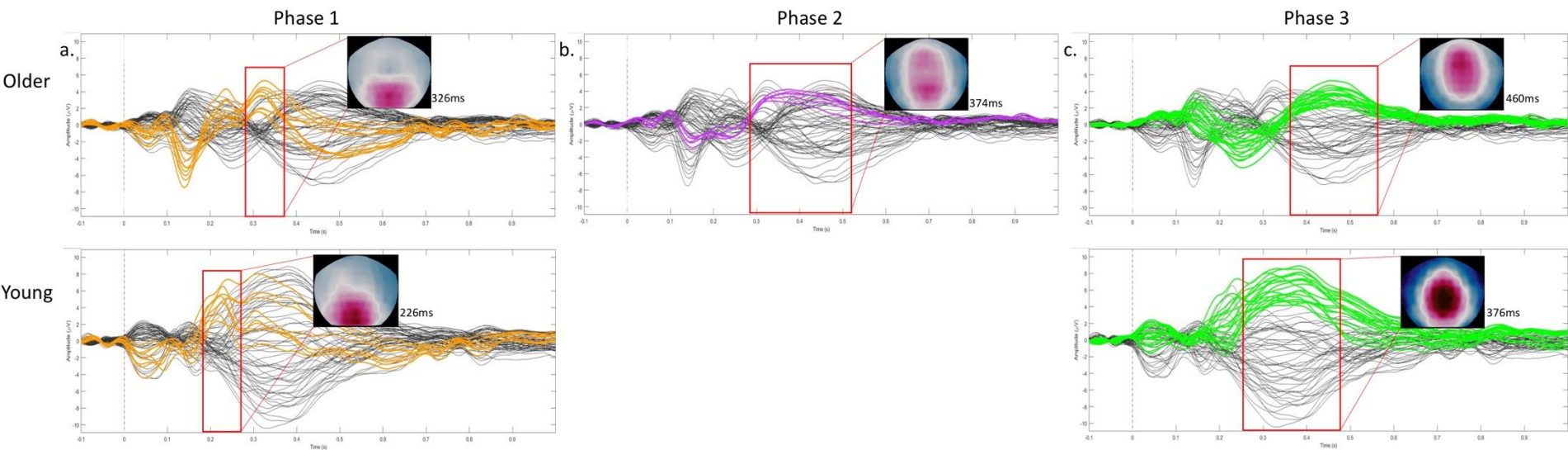


Figure 3. Continuous, 64-channel ERP waveforms (each tracing = individual electrode) in older (top) and young (bottom) adults. Scalp topography maps within the corresponding time windows display all activity at the scalp (i.e., not phase-isolated; time stamp relative to onset of stop-signal; scaled at $\pm 7.477 \mu V$). Plots include the pre- through post-stimulus recording epoch (y-axis = μV ; x-axis = ms). Phases of activation are highlighted within the P300 window via red boxes. Older adults had distinctly different spatio-temporal activation patterns than young. **Phase 1** (left panel, a) was largest at left hemisphere/midline parieto-occipital sites in young (largest at POz, Oz, PO3, O1; orange (bottom)) but bilaterally at parieto-occipital sites in old (POz, PO4, PO3, Oz; orange (top)). Note that in young, Phase 1 activity was sustained through the next phase, but in old it quickly returned to baseline. Instead, older adults uniquely generated **Phase 2** activation (middle, b), consisting of a bilateral parietal slow wave (Pz, P2, P1, P4; purple), possibly corresponding to the Phase 1 sustained parieto-occipital activity in young. Finally **Phase 3** activation (right, c) was largest at right hemisphere/midline centro-parietal sites in young (CPz, Pz, Cz, P2; green (bottom)), but at bilateral fronto-central sites (FCz, FC1, F1, Fz; green (top)) in old.

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4. Discussion

The current study used a high-accuracy stop-signal paradigm to examine age-related differences in lateralized N200 and P300 amplitudes, toward delineating the temporal sequence of compensatory recruitment during successful inhibition. Due to typical age-related delays in N200- and P300-related activity, as well as notable within-group variability in subcomponent timing, the continuous spatio-temporal dynamics are described qualitatively. Analyses revealed distinct hemispheric patterns, both within and between young and healthy, cognitively intact older adults. Young adults had left hemisphere dominant N200 activation and right hemisphere dominant P300. Older adults demonstrated right hemisphere dominant N200s, with overall larger N200 amplitudes than young, particularly in the right hemisphere. The older group also had distinctly bilateral P300 amplitudes, with between-group age effects dependent on electrode site; amplitudes in the left hemisphere were larger in the older group and the opposite was true in the right hemisphere. Further, a fine-grained temporal approach allowed for more in-depth characterization of the dynamics underlying the traditionally calculated N200 and P300 components. N200 activation was temporally similar between groups, with two phases of activation, but differing scalp topography. For P300, there was an initial peak that was common to both age groups, followed by a unique parietal slow wave in the older group and a temporally similar final wave in both groups that differed in topography, with evidence of anterior recruitment in the older group. These age-related patterns of neural recruitment were evident despite comparable task accuracy. Slower response times in the older group despite intact performance suggests that increased activation during successful task performance was compensating for age-related neural decline.

4.1. N200 findings

Inhibitory control studies with fMRI have generally highlighted a dominant role of the right hemisphere in paradigms such as the stop-signal task [11]. Because the N200 component is implicated in inhibitory processes [17], the left hemisphere dominant N200 in young adults might be unexpected. However, the significant temporal limitations of fMRI may be critical to this apparent inconsistency. Right frontal resources, including the IFG and precentral gyrus, are indeed critical for motor response inhibition [11,48]. These processes typically provoke a large magnitude of activation; thus the motor response in the fMRI impulse response function may obscure earlier, *non-motor* processes that contribute to successful inhibition. Indeed, a meta-analysis that separated the global concept of inhibitory control into contributing processes revealed the importance of right frontal regions for motor response inhibition, but in fact *left* frontal resources were dominant for earlier, pre-motoric conflict monitoring and detection processes [11]. EEG source localization studies similarly highlight *left* inferior frontal cortex and left dorsal anterior cingulate cortex as generators of the inhibitory N200, both of which are associated with conflict monitoring [16,18]. Thus, particularly during a paradigm with infrequent, prepotent stimuli such as the stop-signal task, we contend that the N200 during inhibitory control specifically reflects conflict monitoring processes critical to successful response inhibition [18].

Conflict processing that generates the N200 during stop-signal tasks includes monitoring for competing (i.e., high conflict) information, specifically co-activation of the prepotent ‘go’ and inhibitory ‘stop’ responses. Once conflict is detected, N200 may further reflect a sub-conscious alerting or activating of inhibitory mechanisms [18,49,50]. These types of conflict processing are thought to be generated by the dorsal anterior cingulate cortex [7,17,18]. Because cingulate cortex is a medial region, more lateralized activation likely reflects a combination of activity within the cingulate cortex and communication of the cingulate with dorsolateral and ventrolateral prefrontal (including inferior frontal gyri) regions toward modulating cognitive control for following trials [16,49].

Our results further highlight right hemisphere dominant N200 amplitudes in healthy, cognitively intact older adults, with activation greatest at central and frontal sites.

This hemispheric recruitment pattern contrasts with the left dominant pattern in young adults. Overall, the older group had larger amplitudes than young, particularly in the right, non-dominant hemisphere. Of note, amplitudes were still comparable between groups in the left, dominant hemisphere. Age differences were largest in central and parietal regions. Further, despite the young group’s frontal N200 maximum and the older group’s less differentiated anterior to posterior scalp distribution, follow-up contrasts revealed that older adults nevertheless had greater right, non-dominant frontal activation than young. Together, these patterns suggest that recruitment of *both* fronto-parietal and non-dominant hemisphere resources work in concert to enable successful conflict monitoring, detection, and resolution in older adults, which contributed to intact task accuracy despite typical age-related neural declines.

To our knowledge, there is only one study to date of age-related N200 effects using a stop-signal task. It revealed overall larger N200 amplitudes in older compared to young adults at midline electrodes, despite comparable task performance, suggestive of compensation in elders [10]. These findings contrast with previous research using go/no-go tasks, which has overall shown generally smaller N200 amplitudes in older compared to young adults. However, a 2019 meta-analysis revealed that go/no-go ERP studies also had overall poorer performance in the older adult groups. In addition, these studies often presented equal numbers of go and no-go trials, which over-represents motor inhibition trials relative to traditional paradigms. As such, those studies effectively de-emphasized the conflict processing that is tapped during prepotent responding to infrequent inhibitory targets [51]. Stop-signal tasks in general also provide a number of advantages over no-go tasks, including a better measure of response *inhibition* (i.e., vs. response selection) and limiting the role of working memory to allow for more comparable accuracy between groups [9]. To accurately capture compensatory recruitment, particularly in an already demanding domain such as inhibitory control, comparable performance between groups is crucial [52], which the current study achieved.

Examination of continuous waveform activation during the N200 window revealed a comparable underlying temporal pattern in both groups, with two phases of activation occurring in quick succession. For the young adult group, the phases consisted of left followed by right parieto-occipital activity, suggesting that only minimal visual conflict processing resources were necessary for successful inhibition. The older group showed first midline and left hemisphere centro-parietal activation followed by right hemisphere fronto-central activity. These patterns, combined with overall greater N200 amplitudes in older adults, suggest that greater conflict monitoring resources to inhibitory stimuli were necessary, including more anterior recruitment [18,34].

4.2. P300 findings

The young adult group exhibited right hemisphere dominant P300 amplitudes during successful stop-signal trials, with a clear parietal maximum. These findings are consistent with other studies showing right hemisphere dominant activation underlying the P300 component during inhibitory control [16,53]. P300 is associated with activation of a large network, including right hemisphere dominant precentral gyrus, pre-SMA, inferior frontal gyrus, and caudal anterior cingulate cortex, with purported roles in motoric response inhibition and performance evaluation [7,16,54]. These hemispheric patterns demonstrate the different lateralization of the N200 and P300 components in healthy young adults. Furthermore, P300 generally peaks *after* the participant’s SSRT, suggesting that this component more likely reflects *post-inhibitory* performance evaluation and adjustment, rather than response inhibition proper [18,24].

In contrast to young adults, older adults had distinctly bilateral P300 amplitudes with the greatest activation at parietal sites, but relatively little differentiation between anterior to posterior electrodes. Looking more closely at the bilateral activation in older adults, amplitudes in the left (non-dominant) hemisphere were larger in older compared to young adults, highlighting recruitment of non-dominant hemisphere resources [40]. Right (dominant) hemisphere amplitudes were, in contrast, significantly smaller in the

older group. This dominant hemisphere pattern differs from that of N200 wherein older adults still ‘matched’ the young group’s amplitudes in the dominant hemisphere, but ‘added’ recruitment of non-dominant hemisphere resources. Thus, while both components highlight important recruitment of the non-dominant hemisphere with age, the P300 pattern is unique given the smaller amplitudes, reflective of underlying neural deficits, in the task dominant hemisphere. Weaker dominant hemisphere activation during P300 but not N200 is consistent with previous research showing the sensitivity of the P300 component to early neural decline, including differentiation of healthy, cognitively intact elders with genetic risk for Alzheimer’s disease [see 55]. Thus, our findings suggest that the neural mechanisms underlying motor response inhibition and performance evaluation and monitoring via P300 likely decline earlier than N200-related conflict monitoring sources in healthy, typical aging.

In addition to bilateral recruitment, older adults also had less differentiated P300 amplitudes across anterior to posterior electrode sites. This pattern has been noted in previous studies that also controlled for task accuracy, including a recent study from our lab showing smaller midline centro-parietal P300 amplitude in older adults, but larger amplitudes at the frontal site [10]. This was interpreted as indicative of frontal recruitment. We suggest that recruitment of generally bilateral P300, along with frontal P300 in older adults contributes to maintenance of intact inhibitory performance with age [10,34,56]. Indeed, even during a simple auditory oddball task, frontal P300 recruitment was evident in older adults, with compensatory sources localized to precentral and parahippocampal gyri [57]. Oddball tasks are lower demand but overall similar to a stop-signal paradigm in that participants are instructed to respond to targets, in this case certain tones, but ignore others. Comparable task accuracy between groups is crucial in assessment of compensatory recruitment, and thus simple tasks such as the oddball paradigm have often been employed [see 55]. Specifically, compensatory activation is most consistently evident in elders at low to moderate levels of task demand, when performance is comparable with young groups [52]. Decreased activation becomes evident at high levels of task demand, coinciding with poorer task performance compared to young adults. Decreased activation during high task demand (e.g., high episodic/working memory load) is attributed to the depletion of compensatory resources or reserve [i.e., CRUNCH; 52]. Yet, to achieve comparable task demand does not necessitate assessment of only simpler cognitive processes, as with oddball paradigms. Indeed, using the stop-signal task, researchers can maintain control over demand while investigating a higher-order cognitive process that is not only critical in maintaining general cognitive functioning [58], but also maintenance of independence in older adulthood [59].

The only studies of which we are aware that have assessed age-related ERP differences during stop-signal tasks examined only midline electrodes. These studies revealed increased frontal activation in older adults, one with greater amplitudes in young at all except the frontal site [10], and the other showing no significant age-related differences, which the authors interpret as age-related compensation [15]. Relatedly, studies with go/no-go tasks have demonstrated larger P300 amplitudes in older compared to young adults specifically when task accuracy was controlled, pointing to compensatory recruitment [10,28-30,60]. One study localized the sources of greater no-go N200 amplitudes in older adults to the right pre- and post-central gyri and greater P300 activation to right dorsolateral prefrontal cortex and inferior frontal gyrus [30].

Exploration of the temporal patterns underlying P300 revealed an early peak that was common to both groups at parieto-occipital sites. This activation, bilateral in older adults, but right hemisphere dominant in young adults, was centered at the same electrodes that captured an early visual processing negativity in both groups. Activation at these parieto-occipital sites was sustained throughout the remainder of the P300 window in the young group, but quickly returned to baseline in older adults. This difference may point to age-related deficits in early visual aspects of motor response inhibition in older adulthood. Following the first peak, older adults exhibited a unique, bilateral parietal slow wave. Afterward, older adults had a third phase of activation that

was largest at bilateral anterior sites. This corresponded temporally with a second right hemisphere parietal/centro-parietal peak in the young. These later phases underlying P300 activation suggest that the parietal slow wave and recruitment of anterior resources may support successful inhibitory control via performance evaluation and monitoring in older adults [18,24,32]. Additionally, this finer-grained approach clarified that the bilateral activation evident in older adults' traditional P300 waveforms was in fact driven by consistently bilateral activity across *all three phases* underlying the P300 component. Thus, bilateral recruitment plays a uniquely critical role in successful motoric response inhibition and inhibition-related evaluation processes [24,40].

4.3. Limitations

Using continuous, millisecond-level temporal dynamics and a full 64-channel array to interrogate traditional ERP activation, which collapses across a temporal “window” of a couple hundred milliseconds, is a novel and potentially influential approach to ERP data analysis. A primary advantage of collapsing activation across a temporal “window,” however, is the ability to isolate and compare age-related peak activation despite the well-established delays in ERP latency with older age. Overcoming the latency delays to quantitatively compare age groups on different phases of activation underlying N200 and P300 is a challenging obstacle. Indeed, the timing of the phases even within age groups in the current data precluded the ability to define small windows to quantitatively compare age groups on the amplitude of N200 and P300 subprocesses. Future research exploring solutions to this unique issue will allow for more precise characterization of neuro-cognitive aging processes.

5. Conclusions

The current study uniquely reduces the inhibition construct into specific subprocesses to allow examination of how each is differentially impacted by aging. Compensatory theories of cognitive aging are largely based on fMRI research, which provides information on the scale of seconds. Yet, relevant neural activity primarily occurs within the first ~400ms of an inhibitory stimulus [7]. Thus, studies using event-related potentials are uniquely capable of filling the critical gap addressing the temporal sequence underlying compensatory activation such as is associated with aging. Beyond traditional ERP metrics, which collapse activation over several hundred milliseconds, we also provided novel exploration of continuous electrode-level activation to discern patterns underlying the N200 and P300 components. We further highlight the importance of hemisphere-specific patterns of activation that may be critical to understanding both the subprocesses that contribute to successful inhibition in healthy young adults, and the maintenance of cognitive function in older adulthood [32,40]. Thus, we encourage analysis of both anterior to posterior *and* hemispheric patterns of activation by including lateral electrodes in ERP analyses of complex cognitive functions, especially when assessing contributions of age.

Given the prominent effect of N200 amplitude during our stop-signal task and the unique underlying spatio-temporal patterns between age groups, attention to the specific neural mechanisms underlying conflict processing during inhibitory control may be a particularly important target for research on healthy, normative aging as well as risk for pathological aging [10]. Investigation of continuous waveform data also revealed particularly unique spatio-temporal patterns that differentiated age groups during the P300 window. Thus, given the particularly long time window typically used for P300 analyses, finer-grained temporal analysis of the P300 component will likely reveal important subprocesses that may be differentially impacted by aging.

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administration, KAN; funding acquisition, KAN. All authors have read and agreed to the published version of the manuscript.

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