ERP measures indicate both attention and working memory encoding decrements in aging

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Abstract

We investigated age-related attention and encoding deficits, and their possible interaction, by analyzing visual event-related potentials from young and older adults during a modified Sternberg word recognition task. Young adults performed more accurately, albeit not significantly so. P1 latency was shorter in young adults and correlated negatively with task accuracy (with age partialed out). These data support proposals that P1 indexes attentional suppression, which is less efficient in older adults. N1 was larger in older adults but did not correlate with accuracy. Young adults had higher P2 amplitudes and P2 latency correlated with accuracy (age partialed), supporting the view that semantic operations during encoding are affected by aging. These data indicate that attention (P1) and encoding (P2) decrements may contribute to memory or related cognitive decrements in aging, and P1 and P2 latency measures from appropriate paradigms may be salient ERP markers of these decrements.

Cognitive aging typically incorporates decrements in one or more cognitive domains, particularly working memory, episodic memory, and attention (e.g., Buckner, 2004; Craik & Salthouse, 2000). Numerous studies have investigated the electrophysiological or neuroimaging correlates of such age-related cognitive deficits (e.g., Friedman, 2003; Grady, 2008; Van Petten, 2004). Regarding memory decrements in older adults, Friedman, Nessel, and Johnson (2007) posited that various event-related potential (ERP) data indicate that memory encoding decrements are more salient than retrieval deficits, and neuroimaging data also indicate significant encoding deficits in older adults (e.g., Morcom, Good, Frackowiak, & Rugg, 2003). Other ERP data indicate significant attentional decrements in cognitive aging (e.g., Bennett, Golob, & Starr, 2004). Except in a few recent cases (e.g., Zanto, Toy, & Gazzaley, 2010), many such studies have not systematically addressed or accounted for interdependencies between age-related deficits in multiple cognitive domains, yet attention and memory are inextricably linked. For example, Baddeley’s seminal model emphasizes the interdependence of attention and working memory (e.g., 1992). Indeed, behavioral data suggest that modulating attention can disrupt encoding in older but not younger adults (Hogan, Kelly, & Craik, 2006). Hence, memory, particularly encoding, decrements that are characteristic of cognitive aging may be exacerbated by attentional decrements. Recent electrophysiological results highlight the comprehensive nature of the interaction between working memory and attention (e.g., Zanto et al., 2010). For example, Missonniet al. (2006) reported evidence that event-related amplitude modulations during working memory tasks at least partly reflect attentional function. In the current study, we use electrophysiological methods to investigate the degree to which attentional decrements in cognitive aging may contribute to decrements in working memory updating or encoding during a visually presented modified Sternberg task.

In visual ERPs, the P1 and N1 components are generated in extrastriate cortex (e.g., Herrmann & Knight, 2001; Natale, Marzi, Girelli, Pavone, & Pollmann, 2006) and are modulated by attention (e.g., Gazzaley et al., 2008; Hackley, Woldorff, & Hillyard, 1990; Luck, Heinz, Mangun, & Hillyard, 1990; O’Connell et al., 2009; Parasuraman, 1998; Zanto et al., 2010). Hillyard, Vogel, and Luck (1998) proposed P1 and N1 to reflect “gain control” of sensory processing, and others report similar positions (Klimesch et al., 2004; Klimesch, Sauseng, & Hanslmayr, 2007; Klimesch, Sauseng, Hanslmayr, Gruber, & Freunberger, 2007; Natale et al., 2006). However, these two components apparently reflect distinct processing operations. For example, P1 may reflect sensory selection (e.g., Heinz, Luck, Mangun, & Hillyard, 1990) via top-down suppression (Hillyard et al., 1998), whereas N1 has been attributed to index the orienting of attention (Luck et al., 1990; Natale et al., 2006) via amplification of neural activation (Hillyard et al., 1998). The visual P2 component is evidently generated in parieto-occipital regions (Freunberger, Klimesch, Doppelmayr, & Holler, 2007). At distinct from the attentional operations attributed to P1 and N1, various outcomes indicate that the P2 component indexes working memory function (Lefebvre, Marchand, Eskes, & Connolly, 2005; Taylor, Smith, & Iron, 1990; Wolach & Pratt, 2001), particularly encoding (Chapman, McCrary, & Chapman, 1978; Dunn, Dunn, Languis, & Andrews, 1998).

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Hence, the P1, N1, and P2 components are salient neurophysiological markers with which to investigate the degree to which attentional decrements may contribute to working memory encoding decrements in cognitive aging. If attentional decrements do so, we expect P1 and/or N1 measures to differ between younger and older adults. Further, to the degree that working memory encoding deficits per se exist in the current older adult sample, we expect P2 outcomes to differ between these age samples. Some previous studies have observed differences in one or another of these components between healthy young versus older adults, but with a few exceptions (e.g., Gazzaley et al., 2008; Zanto et al., 2010), many have employed tasks that entail either no or relatively minimal cognitive demands (for a review, see De Sanctis et al., 2008). Such studies do not systematically address the central question of the current study. Moreover, various data indicate that the deleterious effect of aging on cognitive function is especially apparent when cognitive load or demands are high (e.g., see Buckner, 2004; Craik & Salthouse, 2000; Friedman, Nessler, Johnson, Ritter, & Bersick, 2008; Grady, 2008); hence, we employ a working memory load manipulation. We hypothesize that ERP differences between the age samples will be most pronounced when cognitive demands are highest.

Methods

Participants
The local ethics committee approved the study. All participants gave informed consent. Fifteen healthy young (age \( M = 21.05 \text{ years}, SD = 3.73 \)) and 15 cognitively healthy older adults (age \( M = 64.20 \text{ years}, SD = 7.87 \)) participated in this study. The young adult sample included 9 women and the older sample 8 women. All participants were right-handed and had normal or corrected-to-normal vision. Young adults were recruited from the local university School of Psychology undergraduate student participant panel and received course credit for participation. Older adults were recruited via newspaper advertisements and received reimbursement for travel expenses where appropriate. The older adult participants were part of a broader, longitudinal study of cognitive aging and, prior to recruitment, were screened for the potential presence of cognitive impairment or other exclusion criteria using clinical interview, neuropsychological testing, and magnetic resonance imaging as described previously (Cummins & Finnigan, 2007).

Task Design
Electroencephalogram (EEG) data were recorded continuously from all participants during the performance of a modified Sternberg (1966) recognition memory task. Procedural details of the task are illustrated in Figure 1.

The task was presented visually on a computer monitor at eye level approximately 50 cm in front of participants’ noses. Words were presented in 48-point black font against a gray background and subtended approximately \( \theta \) of visual angle. Study list length was either four words long (low load condition) or eight words long (high load condition), with a total of 40 study-test trials for each length condition across the entire task (presented at random). All words were five letters in length and of relatively high normative frequencies (> 50 occurrences/million; The Sydney Morning Herald Word Database, cited in Cummins & Finnigan, 2007). The task requirement was to determine whether each cue word was in the immediately preceding study list (old) or not (new). Across all 80 task trials, 50% of cue words were old and 50% were new (varied at random for each participant). The serial position of cue words in the study list was varied at random, with the only constraint being that a cue was never the final word in any study list. Once a word appeared in a given study-cue trial, it was not repeated in another trial. To minimize motor-related artifacts in the EEG/ERP data during the period of interest, participants were instructed to withhold responses until the appearance of the words “yes” and “no” on the screen. Participants’ index fingers each rested on one of two response panel buttons throughout the task, and responses were made by depressing the button corresponding to the position of the appropriate decision, which was varied at random from trial to trial; that is, across all 80 trials, “yes” appeared on the left a total of 40 times (hence, a left button press indicated a “yes” response in these cases) and 40 times on the right. The trials were presented in blocks of 20, with a break of up to several minutes (at the participant’s discretion) between these blocks.

As a consequence of the variation in word-list length, the participant could not predict whether the fourth item in each encoding list would be followed by a fifth (and then sixth, seventh, eighth) to-be-encoded item (high load condition) or a blank encoding list would be followed by a fifth (and then sixth, seventh) to-be-encoded item (low load condition) or a blank screen indicating a retention interval (low load condition). Hence, the fifth item in each eight-item list not only signaled that working memory load was increasing but was also a cue to continue encoding rather than switch from encoding mode to a retention/recognition mode.

EEG Data Acquisition
EEG data were acquired during the task from an elastic QuikCap with 30 embedded sintered Ag/AgCl electrodes (Neuro-medical supplies), SynAmps2 amplifier, and Acquire 4.2 software (Compumedics-Neuroscan). Scalp electrode locations corresponded to the following sites of the international 10–20 system (Jasper, 1958) and modification of same: FP1, FP2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T7, T8, P7, P8, CZ, FZ, PZ, FCZ, CPZ, CP3, CP4, FC3, FC4, TP7, TP8, FT7, FT8, OZ, FT7, with a midline frontal ground and referenced online to linked earlobe electrodes. Vertical eye movements and blinks were monitored via electrodes placed on the supraorbital ridge and below the left eye. Horizontal eye movements were monitored via electrodes placed on the outer canthus of each eye. Electrode impedances were kept at 10 k\( \Omega \) or less and data sampled at 500 Hz with an online bandpass filter (0.5–50 Hz; 12 dB/oct).

EEG and ERP Signal Processing
Off-line signal processing was performed primarily with Edit 4.3 software (Compumedics-Neuroscan) using methods similar to our past studies. An electro-oculogram (EOG) artifact reduction algorithm (Semlitsch, Anderer, Schuster, & Presslich, 1986) was applied where appropriate as in past studies. Continuous EEG data were segmented into one epoch per encoding list word presentation (~200 to 1846 ms after stimulus onset). The epoched data were bandpass filtered (zero phase shift; 0.50 [24 dB] to 40 Hz [24 dB]), then baseline corrected over the prestimulus interval (~200 to 0 ms). Epochs were excluded if amplitude at any EEG electrode exceeded the criteria of ± 80 \( \mu \text{V} \).

For each participant, separate grand ERP averages were computed across the four word presentations in the shorter encoding lists (S), across the first four words (L1) of the eight-
Analyses of ERP Measures

Peak amplitude and latency measures were separately submitted to repeated measures analysis of variance (ANOVA) employing the factors of age (young, old), hemisphere (see above), and encoding list position (L1, L2). Separate ANOVAs were carried out for each component (P1, N1, P2) and for posterior versus frontal electrode sites for reasons outlined above. (Moreover Figure 2 illustrates considerable differences between posterior versus frontal electrodes’ data in terms of ERP waveform morphology and polarity, and these outcomes further support separate ERP analyses for these respective scalp regions.) In addition, for each component (P1, N1, P2), component parameter (amplitude, latency), and scalp region (posterior, frontal), a separate ANOVA was conducted only for fifth-item (“continue encoding”) trials from the high load (eight-item list) condition. As noted above, attentional control is particularly salient to this condition, and fewer trials per participant contribute to this condition’s EEG/ERP averages (hence, generally lower signal-to-noise ratios are assumed, relative to L1 and L2 conditions); therefore separate analyses are warranted for this condition.

Comparisons of Recognition Accuracy with ERP Measures

To investigate the degree of putative relationships between cognitive performance and the ERP measures of interest, in the context of healthy cognitive aging, recognition accuracy measures from the modified Sternberg task were correlated with the P1, N1, and P2 amplitude and latency measures outlined above. These were performed separately for short-list and long-list measures, as discussed further below. Furthermore, any significant ERP–accuracy correlations were rerun as partial correlations with age partialed out, and in these cases partial ERP–age correlations, with accuracy partialed out, also were assessed.

Results

An alpha level of .05 was used for all statistical tests. For all ANOVAs reported hereafter, violations of the heterogeneity of covariance assumption were corrected using the Greenhouse–
Geisser procedure, and corresponding $F$ ratios are, where appropriate, reported with corrected degrees of freedom.

**Recognition Accuracy**

Mean accuracy (discriminability: hits minus false alarms) measures were $M = .930$ (short) and $M = .846$ (long) for the young adults and $M = .933$ (short) and $M = .755$ (long) for the older adult sample. These data were submitted to a $2 \times 2$ independent groups ANOVA with age and list length (load) as factors. A main effect of load was found with short-list accuracy ($M = .932$, $SD = .09$) being greater than long-list accuracy ($M = .801$, $SD = .156$), $F(1,22) = 70.2$, $p < .001$, $\eta^2 = .76$. Neither a main effect of age ($Ms = .888$ and .844 for young and older, respectively) nor a Load $\times$ Age interaction were observed.

**Event-Related Potentials**

Grand-average ERP waveforms for the young versus older adult samples are illustrated in Figure 2. Here the ERPs are averaged across encoding list position conditions, as ERPs for these respective conditions were similar within samples, and, indeed, no significant differences were obtained between these conditions (see below). Similarly for each sample, the group-average ERPs for the S and L1 conditions were virtually identical. This is not surprising, given that each condition represents the first four word presentations in each study-test trial, and for these reasons L1 and L2 condition ERPs only were submitted to the following analyses. The mean number of trials per participant per condition (and ranges across participants) for the L1 and L2 conditions were 127 (101–149) and 130 (105–148), respectively. The corresponding means computed separately for each sample differed by no more than three trials. As noted above the continue encoding condition ERPs, illustrated in Figure 3, contained fewer trials (mean 35, range 24–38) per participant than do the above conditions. Visual inspection of the grand-average ERP waveforms shown in these figures highlights the posterior scalp topography of the P1, N1, and P2 components, which are readily apparent at occipital and parietal electrode sites (with the exception of electrode Pz, at which P1 and N1 are generally less

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**Figure 2.** Grand-average ERP data for the young and older adult samples ($n = 15$ per sample), averaged across task conditions. A representative subsample of 12 electrode sites’ data are displayed. Y-axes display microvolts and X-axes milliseconds. The P1, N1, and P2 components are most prominent at posterior electrodes between approximately 100 ms and 300 ms. Note that although only a prestimulus baseline period of 100 ms is plotted here, these ERPs were baseline corrected over the $-200$ to $0$-ms interval.
distinct). A P2-like waveform (albeit less distinct) is evident at central electrodes and, in Figure 2, frontal electrodes. Both figures show that ERP amplitudes are relatively more positive in younger versus older adults' data over an interval that incorporates all three components. In addition, it is evident that posterior P1 peak latency is relatively earlier in younger than older adults. A similar trend is apparent for N1 at some electrodes (e.g., O1) but less so for P2.

**P1 amplitude analyses.** In the primary ANOVA computed on posterior P1 amplitude data (with load as a factor) P1 amplitude was greater in the young (M = 4.483, SD = 2.664) compared to the older adults (M = 3.491, SD = 1.943); however, a significant main effect of age was not obtained. There was a borderline-significant main effect of hemisphere, F(1,28) = 4.23, p = .056, which reflected the outcome that P1 amplitude was generally greater at right versus left posterior electrodes. No other significant main effects or interactions were observed in this ANOVA. In the ANOVA of posterior P1 amplitude data for the continue encoding condition, a significant main effect of age was obtained, F(1,28) = 8.77, p < .01, indicating that P1 amplitude was significantly greater in young (M = 6.562, SD = 3.763) than older adults (M = 5.121, SD = 2.960). Thus, a diminished P1 in the older adult sample was revealed when working memory task demands were highest in this paradigm. No other significant main effects or interactions were observed.

Above, we describe a significant age-related P1 amplitude difference specifically for the continue encoding condition. Visual inspection of the ERP waveforms for this condition (see Figure 3) indicates some apparent young greater than old amplitude differences over an interval of approximately 100 ms prior to the onset of the P1 (perhaps longer, incorporating some prestimulus interval, e.g., at O2 and Oz). We conducted a supplementary, exploratory analysis to inform consideration of

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**Figure 3.** Grand-average ERP data for the young and older adult samples (n = 15 per sample), for the continue to encode condition (the fifth item in eight-item lists; see text). Y-axes display microvolts and X-axes milliseconds. A representative subsample of 12 electrode sites' data are displayed. The P1, N1, and P2 components are most prominent at posterior electrodes between approximately 100 ms and 300 ms. Note that although only a prestimulus baseline period of 100 ms is plotted here, these ERPs were baseline corrected over the – 200 to 0-ms interval.
potential impact of this earlier potential difference on P1 amplitude measures; via independent groups t tests, we compared the age samples’ mean posterior amplitudes over both the −100 to 0 ms and the 0 to 80 ms intervals. These revealed no significant mean amplitude differences between the groups in either the former, t(28) = 0.547, p = .591, or the latter interval, t(28) = 1.674, p = .107.

P1 latency analyses. In the primary ANOVA of posterior P1 peak latency data, there was a significant main effect of age, F(1,28) = 9.47, p < .01, whereby P1 latency was significantly shorter in young (M = 113, SD = 10.931) compared to the older adults (M = 125, SD = 8). Similarly, in the corresponding ANOVA for the continue encoding condition, a significant main effect of age was obtained, F(1,28) = 9.46, p < .01, indicating that posterior P1 peak latency was significantly shorter in young (M = 109, SD = 17) than older adults (M = 125, SD = 14). No other significant main effects or interactions were observed in these posterior P1 peak latency analyses. No significant main effects or interactions were observed in the frontal P1 amplitude or latency analyses, including analyses incorporating both load conditions and those for the continue encoding condition alone.

N1 amplitude analyses. In the primary ANOVA of posterior N100 peak amplitude data (with load as a factor), there was a significant main effect of age, F(1,28) = 5.29, p < .05, whereby N1 amplitude was significantly less negative in the young (M = −2.743, SD = 1.619) compared to the older adults’ sample (M = −4.827, SD = 2.271). There was a trend for N1 to be largest (most negative) over the left hemisphere, but no significant main effect of hemisphere was obtained, F(1,28) = 3.94, p = .081. The ANOVA of posterior N1 amplitude data only for the continue encoding condition also revealed a significant main effect of age, F(1,28) = 5.18, p < .05; again N1 amplitude was significantly less negative in the young (M = 1.686, SD = 1.924) compared to the older adults’ sample (M = −1.774, SD = 2.026). A significant main effect of hemisphere was obtained, F(1,28) = 8.47, p < .01, which again indicated that N1 amplitudes were relatively more negative at left (M = −4.559, SD = 2.853) versus right hemisphere electrodes (M = −3.492, SD = 2.808). The ERP figures as well as source analysis outcomes (see below) demonstrate that this N1 hemispheric asymmetry is primarily driven by the younger adults’ ERPs. Hence, as with the P1, the impact of age on N1 is most pronounced when task demands were highest.

N1 latency analyses. No significant main effects or interactions were observed from the ANOVA of posterior N1 peak latency data or in the analyses of frontal N1 peak amplitude or latency data.

P2 amplitude analyses. A significant main effect of age, F(1,28) = 5.32, p < .05, resulted from the primary ANOVA of posterior P2 peak amplitude data, whereby the amplitude of this component was significantly greater in the young (M = 6.023, SD = 1.851) compared to the older adults’ ERP (M = 4.042, SD = 2.177). A highly significant main effect of hemisphere, F(1,28) = 9.23, p < .001, reflected the outcome that posterior P2 amplitude was greater at right (M = 6.234, SD = 1.667) versus left hemisphere electrodes (M = 5.133, SD = 1.621). The ANOVA of posterior P2 amplitude only for the continue encoding condition also revealed a significant main effect of age, F(1,28) = 5.29, p < .05, with P2 amplitude greater in young (M = 9.222, SD = 2.529) than older adults (M = 5.812, SD = 2.478). No other significant main effects or interactions were observed in this ANOVA. The ANOVA of frontal P2 peak amplitude data revealed no significant main effects or interactions.

P2 latency analyses. The ANOVA of P2 peak latency data revealed no significant main effects or interactions, nor did the analyses of frontal P2 peak amplitude or latency data.

Comparisons of Recognition Accuracy with ERP Measures
No significant correlations were observed between ERP and recognition accuracy measures in data from the short encoding list condition, albeit performance in this condition was at or near ceiling in the majority of participants. For analyses of long encoding list data, ERP measures were averaged over the L1 and L2 conditions for two principal reasons. First, recognition performance on the long-list condition was averaged across these two encoding list-position conditions. Second, as outlined in the above analyses, substantially different outcomes were not observed between these two conditions on the analyzed ERP measures. No significant correlations were observed between ERP amplitude measures and long-list recognition accuracy; this applies to P1, N1, and P2 amplitude measures. No significant correlations were observed between long-list N1 latency measures and accuracy. Left posterior P1 peak latency bore a significant negative correlation with long-list recognition accuracy (r = −.426, p < .05). In contrast, left posterior P2 latency bore a significant positive correlation with long-list recognition accuracy (r = .422, p < .05). These two significant ERP–accuracy correlations are illustrated as scatterplots in Figure 4. These demonstrate that each sample contains one participant who may possibly be considered an outlier in part because his or her accuracy was below .5; however, supplementary analyses excluding these participants’ data demonstrated that the significant correlations were not driven by same. Furthermore, when recomputed with age partialed out, both the accuracy–left posterior P1 latency (r = −.365, p < .05) and the accuracy-left posterior P2 latency (r = .398, p < .05) correlations maintained significance. Finally, partial correlations were computed between age and these ERP measures, with long-list recognition accuracy partialled out; here only left posterior P1 latency demonstrated a significant partial correlation with age (r = −.378, p < .05).

Current Source Analyses
ERP source analysis outcomes are illustrated in Figure 5. Frontal P1 and P2 sources are right-lateralized in older adults, but bilateral sources were indicated in the young adult sample, whereas N1 sources are bilateral in older adults and left-lateralized in the young adult sample. The same outcomes were obtained from both the sLORETA and the Minimum Norm algorithms’ current source analyses, albeit the former is a modification of the latter. Consistent with past reports (e.g., De Sanctis et al., 2008; Herrmann & Knight, 2001; Natale et al., 2006) these analyses reveal P1 and N1 sources in regions of extrastriate cortex. P2 sources incorporate adjacent, in some cases partially overlapping, regions that are right-lateralized in the older adults, whereas in the younger adult sample specifically, there is also a left hemisphere P2 source incorporating the junction of the occipital, temporal, and parietal lobes. It is noteworthy that no frontal sources were
revealed in either sample for any of these three components analyzed.

Discussion
A number of novel, age-related ERP differences were observed in this study. The peak latency of the posterior P1 component was significantly earlier in the young versus older adults in all conditions. Posterior P1 amplitude was greater in young versus older adults specifically in the continue encoding condition. N1 amplitude was significantly greater (i.e., relatively more negative) in the older versus younger adults in all conditions. In addition, P2 amplitude was significantly greater in the young than older adults’ grand-average ERPs. Furthermore, recognition accuracy demonstrated significant correlations with both posterior P1 peak latency and posterior P2 peak latency, and these held when age was partialed out. During passive viewing of alphanumeric stimuli De Sanctis et al. (2008) reported a lack of significant age effects on both P1 amplitude and latency. Using a task that modulates working memory load and encoding demands, we have obtained significant age effects on both P1 latency and in a specific condition, amplitude. Three past studies report significantly longer P1 latencies in older adults in data from visual, four-choice reaction tasks (see De Sanctis et al., 2008). The results summarized in the preceding four sentences collectively indicate that P1 latency may, under appropriate conditions, be linked or sensitive to some aspect of cognitive function.

P1 has been proposed to index attentional function, such as early sensory selection (e.g., Heinze et al., 1990). Moreover, Klimesch et al. (2004), Klimesch, Sauseng, and Hanslmayr (2007), Klimesch, Sauseng, Hanslmayr, et al. (2007), and Natale et al. (2006) have proposed P1 to be the earliest ERP index of
attentional control interacting with bottom-up sensory processing (via frontal-posterior attention networks). The attentional "gain control" model of Hillyard et al. (1998) proposes that suppression (of task-irrelevant processing) occurs during the P1 interval. Gazzaley et al. (2008) reported posterior P1 amplitude and other electrophysiological outcomes, indicating a selective deficit in older adults' top-down suppression of visual stimulus processing, which is specific to early processing stages. Given these proposals, the current P1 latency outcomes indicate that the older adults were slower to instantiate attentional suppression of irrelevant processing during working memory encoding. Curran, Hills, Patterson, and Strauss (2001) also observed slower P1 latencies in older adults but noted that this is not necessarily accompanied by information loss. Further to this, Gazzaley et al. proposed that their P1 and related results indicate that suppression is not abolished in aging but is delayed, thus resulting in greater interference from task-irrelevant information and poorer working memory performance in older adults. The current outcomes extend this literature by demonstrating not only slower P1 latencies in older adults but a significant inverse relationship between left posterior P1 latency and accuracy, even when age was partialled out. These observations indicate that the efficiency of suppression during working memory encoding is linked to subsequent recognition performance such that, in older relative to younger adults, slower P1 peaks are generally associated with lower recognition accuracy. The specificity of this correlation to left P1 latency well may be linked to our use of verbal stimuli. There was a trend whereby the older adults performed the recognition task less accurately than younger adults, albeit this was not significant; this is not an unexpected outcome in healthy older adults (e.g., Verhaegen, Marcoen, & Goosens, 1993). Hence, these outcomes are in general agreement with the positions of Curran et al. and Gazzaley et al., whereby attentional suppression is slowed with aging, often impacting detrimentally on working memory performance (perhaps via increased interference); yet at least a degree of (slowed) suppression generally remains intact in healthy older adults, and this would account for the current lack of significant working memory deficits in same. Finally, with regard to the aforementioned Natale et al. (2006) proposal, it is possible that the slower P1 latencies in older adults may reflect at least a degree of delayed sensory processing, perhaps concomitant with delayed attentional suppression.

Across all conditions, there was a trend whereby older adults exhibited relatively lower P1 amplitudes than younger adults, as illustrated in Figures 2 and 3. However, a significant age effect on P1 amplitude was observed only under conditions of increasing working memory load, when the task required ongoing encoding rather than transition to a recognition memory mode. These outcomes indicate an age-related decrement in modulating or (re)allocating attention to support ongoing working memory encoding and also are consistent with behavioral outcomes that indicate that attentional switching disrupts memory encoding to a greater degree in older versus younger adults (Hogan et al., 2006). Within the framework of Gazzaley et al. (2008; see above), the current P1 amplitude data may indicate that when varying task demands required ongoing encoding of new items, older adults were less effective at reallocating attention and suppressing irrelevant processing, such as that associated with retention/rehearsal of previously encoded items. However, it is salient to note that fewer trials contribute to the continue encoding condition ERPs; hence, these have lower signal-to-noise ratios. Similarly, Figure 3 illustrates that at some posterior electrodes (e.g., O2, Oz) a young greater than old ERP amplitude difference is apparent prior to the onset of the P1, despite baseline correction. We did not observe significant mean amplitude differences between the age samples over the −100 to 0-ms nor the 0 to 80-ms intervals; however, these were simple, supplementary analyses. Hence, the age effect on P1 amplitude that was significant in this condition alone may at least in part represent a carryover of a preceding, age-related amplitude difference (which may in turn possibly reflect an age-related difference in anticipatory-type potentials). As expected on the basis of past reports (e.g., Herrmann & Knight, 2001; Natale et al., 2006) P1 sources were identified in extrastriate cortex. However, an outcome that to our knowledge has not been reported is that these P1 sources were bilateral in younger adults but only right-lateralized in the older adults. These outcomes may reflect relatively more widespread attentional suppression of (irrelevant or interfering) sensory processing in younger adults. However, these proposals remain speculative and warrant further investigation, not only because our current source analyses are not based on data from a high-density electrode montage. Overall P1 peak latency (more so than P1 amplitude) appears to be a notable ERP marker of attentional decrements in cognitive aging, at least in the current study, as it was significantly slower in older adults across all encoding conditions, plus it bore a significant positive correlation with age (independent of accuracy) and a negative correlation with recognition performance (independent of age). These novel correlation outcomes extend the current and previously reported age effects on P1 to indicate that P1 latency measures acquired from appropriate cognitive paradigms may constitute a salient ERP marker of cognitive aging.

Posterior N1 amplitude was significantly greater (relatively more negative) in the older adults' ERPs across all conditions. Bennett et al. (2004) reported analogous auditory attention task N1 differences between young versus older adults, as did De Sanctis et al. (2008) using a passive viewing task. N1 evidently indexes distinct attentional operations to P1 (e.g., Hillyard et al., 2008).
Age effects on attention and memory encoding ERPs

1998; Klimesch, Sauseng, & Hanslmayr, 2007; see Natale et al., 2006; for example, N1 has been attributed to index the orienting of attention (Luck et al., 1990; Natale et al., 2006) or the “intersection between feature selection and working memory encoding” (Zanto et al., 2010, p. 22). In addition, Allison, Puce, Spencer, and McCarthy (1999) reported two intracranial ERP components incorporating the N1 interval (one of which is evidently analogous to the scalp-recorded N1) that appear to specifically index processing of words (nouns, and not faces, objects, or letter strings). Hence, the larger posterior N1 amplitudes in older adults may indicate that this sample generally oriented attention to the visual features of to-be-encoded words to a relatively greater extent in order to maintain task performance. Notably, previous N1 (and other electrophysiological) outcomes indicate that older adults do not exhibit a processing enhancement deficit during visual working memory encoding (Gazzaley et al., 2008). Evidently, cortical excitatory state and stimulus processing capacity are enhanced in an oscillatory manner, specifically during the interval of negative scalp potentials (e.g., Klimesch, Sauseng, & Hanslmayr, 2007). Hillyard et al. (1998) referred to such enhancement during the N1 interval as “gain control-amplification.” In the current study, older adults may have exhibited relatively enhanced visual processing, as indexed by amplification of N1 generators and larger N1 amplitudes. Current source analyses provide further insights: As in previous studies (e.g., Herrmann & Knight, 2001; Natale et al., 2006), N1 sources were identified in extrastriate cortex, bilaterally in older adults and left-lateralized in the young adult sample (see Figure 5). These outcomes, which are consistent with N1 source data reported by De Sanctis et al. (2008), indicate that, in the older adults, activity and processing (perhaps of orthographic features) in contralateral extrastriate regions was “amplified” to a relatively greater degree (perhaps as a form of neurocognitive compensation). However, it should be noted that neither N1 amplitude nor latency measures were found to be correlated with recognition performance, indicating that N1 is not as closely coupled to overall working memory function (at least to performance on the current task) as are P1 and P2. Finally, notwith-standing the differing N1 source outcomes from the age samples, regarding the N1 amplitude differences, we cannot definitively exclude the possibility that these may represent some degree of carryover of the preceding age-related P1 amplitude difference (albeit this was not significant in all conditions). Indeed the same caveat applies to the age-related P2 amplitude differences, which are discussed next.

Young adults’ posterior P2 amplitudes were significantly larger (across all task conditions) than those of the older adults. To our knowledge these are novel observations. Various outcomes indicate that the P2 component indexes working memory function (Lefebvre et al., 2005; Taylor et al., 1990; Woluch & Pratt, 2001), including encoding (Chapman et al., 1978; Dunn et al., 1998; Smith, 1993). The significant correlation between P2 latency and performance on the modified Sternberg task (discussed below) and the age-related P2 amplitude difference in parallel with a recognition accuracy difference between the samples are consistent with these proposals. Although significant age effects were obtained on neither recognition accuracy nor on P2 latency measures, and P2 latency did not correlate with age when accuracy was partialled out; perhaps task conditions incorporating higher cognitive load might yield such outcomes. Overall, the lower P2 amplitudes in older adults and the correlation between P2 latency and accuracy (independent of age) at least are broadly consistent with other evidence (e.g., Friedman et al., 2007; Hogan et al., 2006) that memory encoding is affected by cognitive aging. More generally, together with P1 and N1 outcomes discussed above, the current ERP data indicate that both attentional and encoding decrements can contribute to diminished working memory function in healthy older adults. Curiously, the directions of the significant ERP-recognition correlations were opposite, being negative for P1 but positive for P2 latency; the latter, positive correlation would perhaps seem less likely than the former. One potential explanation relates to the proposals outlined herein whereby P1 reflects attentional suppression (e.g., Gazzaley et al., 2008) but P2 indexes encoding. Within this framework, a later P2 component may reflect a relatively more comprehensive degree of encoding-related activity, such as semantic information processing, and thus generally better memory performance. Dunn et al. (1998) and Federman and Kutas (2002) have linked P2 with semantic processing. Indeed Friedman and colleagues (Friedman et al., 2007; Nessler, Johnson, Bersick, & Friedman, 2006) posited that their ERP data indicate that age-related encoding decrements reflect diminished semantic processing (e.g., retrieval or elaboration) during encoding of words, and such phenomena may also underlie the current P2 outcomes. Current source analyses revealed right posterior P2 sources in both samples but a left hemisphere source around the junction of the occipital, temporal, and parietal lobes only in the younger adults. The left hemisphere P2 source in younger adults incorporates the angular gyrus and Brodmann area 39 (“angular area 39”), which has frequently been linked to semantic processing on the basis of classical neuropsychological as well as more recent neuroimaging observations. These outcomes, together with proposed links between P2 and semantic processing summarized above, are consistent with the position that, relative to older adults, young adults can more successfully employ semantic processing during and in order to enhance the encoding of words. An important caveat is that our source analyses are not based on a high-density electrode array and only indicate candidate generators, although Freunberger et al. (2007) also reported P2 sources in parieto-occipital regions. Furthermore, it is notable that our observed correlation between P2 latency and recognition performance (independent of age) was specific to left hemisphere electrodes. These observations further support our proposal that longer-latency P2 is generally associated with better word recognition performance (at least in the current study) because the former reflects relatively more comprehensive semantic processing that increases the efficacy of encoding. On a different note, P1 is proposed to be linked to or modulated by alpha-frequency oscillations (e.g., Freunberger et al., 2008; Klimesch, Sauseng, Hanslmayr, et al., 2007) whereas P2 has been linked to oscillations in the relatively slower theta-frequency band (e.g., Freunberger et al., 2007). Hence, individual variations in EEG-ERP dynamics (e.g., individual alpha frequency) and links between same and cognitive function in aging may also relate to these outcomes and may prove fruitful subject matter for future investigations in this field, perhaps via additional methods such as event-related synchronization/desynchronisation analyses in concert with appropriate cognitive paradigms. Regardless, posterior P1 and perhaps P2 peak latency measures, acquired from appropriate cognitive paradigms, may be salient ERP markers of aspects of neurocognitive function in the context of aging.

The interesting dissociation in ERP source analysis outcomes between young and older adults, as well as positive components
versus N1, warrants some final consideration, particularly with regard to the observed ERP-recognition correlations. Young adults showed bilateral posterior source generators for P1 and P2 but only a left-posterior source for N1, whereas older adults exhibited an orthogonal pattern of only right-posterior sources for P1 and P2 but bilateral posterior sources for N1 (see Figure 5). Although these current results are based on fewer electrodes’ data, it is salient that they corroborate those reported by De Sanctis et al. (2008; who did not report P1 or P2 sources). Above, we note models (e.g., Hillyard et al., 1998) wherein, during the N1 interval, cortical activity is in an excitative state, whereas cortical activity may be in an inhibited or refractory state during the P1 and P2 intervals. Bilateral N1 sources in older adults may reflect a greater degree of attentional enhancement (perhaps compensatory), whereas one or both of the bilateral P1 and P2 source patterns in younger adults may partly reflect more effective attentional suppression (cf. Gazzaley et al., 2008). As noted above, peak latency measures for both the P1 and P2 components were the only ones of the analyzed encoding-trial ERP measures to demonstrate significant correlations with recognition accuracy measures. Furthermore, both of these correlations were specific to left posterior electrode sites; this is salient, given that left hemisphere sources of these components were specifically observed in younger adults. Finally, it is also noteworthy that the various ERP outcomes reported herein were obtained in the absence of significant recognition differences between the age samples. We have previously reported analogous outcomes for recognition-interval, frontal theta power measures (Cummins & Finnigan, 2007). Collectively, these data indicate that brain electrophysiological measures such as P1 or P2 latency, together with appropriate cognitive paradigms, should provide more sensitive markers of neurocognitive aging than behavioral measures alone.

There are several caveats or possible shortcomings to the current study. First, the age-related comparisons are cross-sectional rather than longitudinal, although the latter approach is not particularly feasible for comparisons between age ranges that differ by several decades. Nevertheless, we are currently preparing to repeat these measurements annually in a cohort of older adults. We cannot definitively reject the possibilities that some of the results may partly reflect possible differences between the age samples on more general factors such as intelligence or arousal. We do not assume that the ERP source results do not imply that no regions other than those illustrated in Figure 5 were active during those components’ intervals; similarly, we do not assume that the scalp ERPs acquired in this study index all cortical regions activated during such intervals. For example, on the basis of past reports, we may expect activity in left inferior prefrontal (e.g., Morcom et al., 2003; Nessler et al., 2006) and/or inferior temporal (e.g., Allison et al., 1999) cortical regions, but we do not necessarily assume that activity in such areas is substantially indexed by the scalp ERPs investigated herein. Also, as noted above, our source computations do not analyze data from a high-density montage, so the outcomes should be interpreted accordingly. This article focuses largely on posterior ERP component results, albeit no frontal sources were observed for any of the components analyzed; these data indicate that any associated frontal operations are not optimally indexed by ERP analyses between approximately 100 ms and 300 ms after stimulus onset. Rather, such operations may be instantiated in a relatively more tonic manner and may be further probed with methods noted above and measures such as frontal theta power (cf. Cummins & Finnigan, 2007; Gazzaley et al., 2008; Missonnier et al., 2006). Finally, we agree with Zanto et al. (2010) that ultimately it may not be possible to completely disentangle early encoding stages from some attentional operations. Nevertheless, in summary, the current data are consistent with behavioral and other neurobiological evidence that attentional decrements can manifest during working memory encoding and thereby contribute to memory decrements in aging, but further provide novel neurophysiological evidence that both attentional (indexed by P1 and perhaps N1) and working memory encoding (indexed by P2) decrements exist and can contribute to working memory decrements in healthy older adults. More broadly, the current data support the existence of interdependencies between working memory and attention and between age-related deficits in same. Finally, the peak latencies of the P1 and/or P2 components acquired from appropriate cognitive paradigms may be particularly useful ERP measures for assessing specific neurocognitive processes in older adults. Hence, these data generate new evidence and interpretations that can be further investigated by future studies that may use multidisciplinary methods to investigate the dynamics of such neurocognitive decrements not only in healthy older adults but also in clinical conditions such as mild cognitive impairment or dementia.

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