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Research Report

Modality-specificity of sensory aging in vision and audition: Evidence from event-related potentials

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ABSTRACT

Major accounts of aging implicate changes in processing external stimulus information. Little is known about differential effects of auditory and visual sensory aging, and the mechanisms of sensory aging are still poorly understood. Using event-related potentials (ERPs) elicited by unattended stimuli in younger (M=25.5 yrs) and older (M=71.3 yrs) subjects, this study examined mechanisms of sensory aging under minimized attention conditions. Auditory and visual modalities were examined to address modality-specificity vs. generality of sensory aging. Between-modality differences were robust. The earlier-latency responses (P1, N1) were unaffected in the auditory modality but were diminished in the visual modality. The auditory N2 and early visual N2 were diminished. Two similarities between the modalities were age-related enhancements in the late P2 range and positive behavior–early N2 correlation, the latter suggesting that N2 may reflect long-latency inhibition of irrelevant stimuli. Since there is no evidence for salient differences in neuro-biological aging between the two sensory regions, the observed between-modality differences are best explained by the differential reliance of auditory and visual systems on attention. Visual sensory processing relies on facilitation by visuo-spatial attention, withdrawal of which appears to be more disadvantageous in older populations. In contrast, auditory processing is equipped with powerful inhibitory capacities. However, when the whole auditory modality is unattended, thalamo-cortical gating deficits may not manifest in the elderly. In contrast, ERP indices of longer-latency, stimulus-level inhibitory modulation appear to diminish with age.

1. Introduction

Aging is associated with sensory, motor, and attentional declines. All major accounts of aging implicate impaired processing of external stimulus information and the proposed mechanisms include impaired local/lateral inhibition (Allman et al., 1985; Dustman et al., 1996; Grossberg, 2001), prefronto-thalamo-cortical gating (Knight et al., 1999; Zikopoulos and Barbas, 2006), prefrontal cortico-cortical facilitation (Chao and Knight, 1998; Knight et al., 1999), dedifferentiation (Dustman et al., 1981), general slowing (Salthouse, 1996), and higher-order compensation of inefficient lower-level processing (Cabeza et al., 2002). Indeed, links between perception and cognition have been observed in large-scale behavioral studies that found a substantial overlap of age-related variability in perceptual and cognitive skills, with the degree of overlap

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Table 1 – Auditory ERP findings in aging studies

Peak	Enhanced amplitude or shortened latency	Diminished amplitude or prolonged latency	No change
P1	Amplitude: Aine et al., 2005; Amenedo and Díaz, 1998a,b; Friedman et al., 1993; Kovacevic et al., 2005; Pekkonen et al., 1995; Pfefferbaum et al., 1980		
N1	Amplitude: Amenedo and Díaz, 1998a, 1999; Anderer et al., 1996[a]; Woods, 1992a		Amplitude: Amenedo and Díaz, 1998a,b; Anderer et al., 1996; Brown et al., 1983; Iragui et al., 1993; Pfefferbaum et al., 1980; Picton et al., 1984; Tremblay et al., 2004; Tremblay et al., 2002, 2003
P2	Amplitude: Aine et al., 2005; Amenedo and Díaz, 1998a,b; Friedman et al., 1993; Kovacevic et al., 2005; Pekkonen et al., 1995; Pfefferbaum et al., 1980; Tremblay et al., 2004; Tremblay et al., 2002, 2003	Amplitude: Anderer et al., 1996 (after age 60); Latency: Iragui et al., 1993; Pfefferbaum et al., 1980; Picton et al., 1984; Tremblay et al., 2004; Tremblay et al., 2002, 2003	Amplitude: Barrett et al., 1987; Brown et al., 1983; Iragui et al., 1993; Picton et al., 1984; Latency: Amenedo and Díaz, 1998a,b; Anderer et al., 1996; Barrett et al., 1987; Brown et al., 1983; Ford and Pfefferbaum, 1991
N2		Amplitude: Bertoli and Probst, 2005; Chao and Knight, 1997b; De Chicchis et al., 2002; Pfefferbaum et al., 1980 (no statistics, waveforms only)	

[a]the only study that found enhanced N1 in response to unattended stimuli.

increasing with age (Anstey et al., 2001; Baltes and Lindenberger, 1997; Ghisletta and Lindenberger, 2003; Li and Lindenberger, 2002; Li et al., 2001a; Lindenberger and Baltes, 1994, 1997). Sensory measures (acuity and discrimination of simple features) mediated nearly all of the variance in the 14 tests on cognitive skills and were more predictive of cognitive functioning than processing speed. Further, higher-order sensory functions, aiding in perceptual object formation and recognition (Čeponienė et al., 2005; Hillyard and Anllo-Vento, 1998; Näätänen et al., 2001) were implicated in age-related deficits in language and memory (Murphy et al., 2000). Finally, perception critically affects attention. For example, perceptually salient stimuli may initiate attentional orienting; increased perceptual thresholds or decreased perceptual accuracy may increase attentional demands; sensory-level disinhibition may cause distractibility. Therefore, impaired perceptual abilities may both directly and indirectly (through attention) affect the processing load of the limited-capacity cognitive mechanisms (McCoy et al., 2005). Despite the prominent role that sensory derangement might play in aging, the nature or universality of age-related changes in the sensory domain is still not well understood.

2. Inhibition, facilitation, and activation in auditory aging

The most prominent account of age-related changes in auditory sensory ERPs has been that of inhibitory decline, postulating ineffective top-down modulation of sensory regions by prefrontal cortex (PFCx, Hasher and Zacks, 1988; Knight et al., 1999; Kok, 1999). Mammal and human lesion data suggest that the top-down inhibition of *primary* auditory and somato-sensory responses (visual data is lacking) is implemented by prefronto-thalamo-cortical gating, where the PFCx induces thalamo-reticular inhibition of sensory input to the cortex. Electrophysiologically, gating deficiency is reflected by an enhancement of middle-latency potentials originating from primary sensory fields when PFCx is incapacitated, and diminution of these potentials when thalamo-reticular nuclei are stimulated (Knight et al., 1989; Kraus et al., 1982; Skinner and Yingling, 1977; Wood et al., 1988; Yingling and Skinner, 1976). Connection between these findings and aging is provided by neuro-anatomic evidence showing that the frontal lobe atrophies the earliest and the most with age (Raz et al., 1997), and that behaviorally measured inhibitory capacities also decline with age (Chao and Knight, 1997b; Dustman et al., 1996; Kok, 1999). Given the "preventive" nature of the gating mechanism, it is likely that its major role is to dampen information that has become irrelevant as a result of an attentional set having been established elsewhere (various channel-level selective attention conditions). In contrast, processing of salient stimuli that do break through the attentional threshold appears to be down-modulated by cortico-cortical inputs from orbito-frontal regions (Rule et al., 2002).

Although it is clear that inhibition is pivotal for sensory, attentional, and cognitive functions, it operates in complementation with *facilitation* to provide the balance necessary for optimizing adaptive behavior. Human lesion data show that, in addition to inhibitory effects, the PFCx exerts robust facilitatory influence on auditory and visual sensory processing. This was reflected by diminution of ipsilateral auditory and visual N1 peaks in patients with PFCx lesions (Chao and Knight, 1998; Knight, 1997). Human and animal data indicate that these facilitatory effects are conducted by cortico-cortical tracts projecting from tertiary PFCx to the *secondary* sensory fields (Knight et al., 1999; Webster et al., 1994). The interaction between the two major modulatory mechanisms is likely to act upon optimizing sensory selectivity: early on, the PFCx-thalamo-cortical loop aids in the selection/suppression of relevant information, while later on, processing of the selected

information is boosted by cortico-cortical facilitation or dampened by orbito-cortical inhibition. Such a scenario is likely to play out in within-channel selection tasks (e.g., detecting targets among non-targets in the same stimulus channel).

Age-related changes in sensory cortices also need to be considered. While it has been reported that no substantial volume or neuronal loss occurs in the auditory cortex with aging (Sowell et al., 2003), disintegration of synaptic connectivity is a possibility (Peters, 2002; Peters et al., 1994). This may affect the capacity of sensory processors to activate in response to bottom-up activation or top-down facilitation. Further, the diminished capacity of local (lateral) inhibition may result in the diminution of response accuracy (specificity) and enhance the recorded ERP amplitudes. Finally, synaptic de-synchronization would delay maximal post-synaptic summation which can translate into increased processing speed.

Numerous aging studies on auditory processing have demonstrated a general trend of earlier-latency auditory ERP peaks enhancing with age, positive peaks (P1, P2) more so than negative (N1; Table 1). Few existing studies on longer-latency N2 peak unequivocally show that it diminishes with age (Table 1). Contrary to earlier assumptions, latency increases with age are not a universal finding (Table 1). When found, these increases affect longer-latency peaks (e.g., P2; Iragui et al., 1993). N1 and P2 latency increases were found by Tremblay, et al. (2004, 2002, 2003) in response to consonant–vowel syllables but not to tones.

The age-related enhancement of the auditory N1 and P2 peaks, as well as the diminution of the N2, have been interpreted in terms of declining prefrontal gating (Amenedo and Díaz, 1998a; Anderer et al., 1996; Bertoli and Probst, 2005; Friedman et al., 1993). However, it is unclear why some electrophysiological indices of stimulus processing would be enhanced (N1, P2), while others diminished (N2), by a deficit in the same modulatory function of inhibition. Furthermore, in lesion studies, gating deficits manifest by increase in middle-latency auditory evoked potentials. In addition, age-related enhancements of the auditory P1 and P2 peaks appear to reverse after age 60 (Anderer et al., 1996), which would require an additional explanation to that of disinhibition. Finally, the link between the long-latency N2 peak and inhibitory functions is less than clear. Based on its morphological resemblance to the sleep N350 and go-nogo N2, Bertoli et al. (2005) argued that the non-target N2 may index inhibition of irrelevant information. If so, its diminution with age could provide a valuable index of diminishing inhibitory capacity. Interestingly however, this peak is maximal during mid-childhood (6–8 years), at the age when frontal networks involved in inhibition are still maturing. The amplitude of the N2 begins to diminish from age 10 years, onwards (Ponton et al., 2000), while inhibitory abilities continue to strengthen until the fourth or fifth decade (Dustman et al., 1996). This discrepancy may indicate that the N2 is subject to, rather than an index of, inhibitory modulation. Understanding this is critical for correct interpretation of age-related changes in sensory electrophysiology.

Three aspects of processing could be looked at in order to understand the above inconsistencies. First, all of the major phenomena, including inhibition, facilitation, and local resource depletion should be considered while interpreting age-related findings. Further, it is critical to take into account the known information about the functional roles of ERP peaks at question. For example, the auditory P1, the latest of middle-latency peaks, is most likely to be susceptible to changes in PFCx–thalamic gating, delimiting the amount of bottom-up activation. Further, the auditory N1 and P2 peaks, which are strongly linked to sensory-attentional interactions such as stimulus detection and orienting (Čeponienė et al., 2005; Crowley and Colrain, 2004; Jääskeläinen et al., 2004; Näätänen, 1990), are likely to be subjected to top-down facilitatory and/or inhibitory modulations, depending on stimulus relation with an individual's state and direction of attention. Finally, the auditory N2 peak, proposed to reflect longer-latency, higher-order (integrative) sensory processing (Čeponienė et al., 2005, 2001; Karhu et al., 1997), is likely to be affected by local connectivity as well as top-down facilitatory or inhibitory modulations.

The third aspect to be considered in interpreting sensory data is attentional influences. Since one of the suggested key players in age-related sensory decline is inhibitory deficit, examining sensory processes under conditions of attentional facilitation or inhibition represent an important confound for informing about changes in the sensory domain proper.

3. Local disinhibition and deactivation in visual aging

Visual ERPs to simple visual stimuli consist of the C1-P1-N1-P2-N2 peaks. The visual C1 (60–90 ms) is generated in striate visual cortex and is considered to reflect processing of elementary visual features (the "analysis" stage). The visual P1 peak (90–120 ms) is generated in extrastriate visual cortices and is a marker of visuo-spatial selection (Clark and Hillyard, 1996; Hillyard and Anllo-Vento, 1998). The visual N1 has multiple generators, extending through the secondary sensory and supra-modal cortices of occipito-temporal and lateral parietal cortices (Di Russo et al., 2002). The different subcomponents of the N1 reflect visuo-spatial attention and object-level processing (Hillyard and Anllo-Vento, 1998; Saron et al., 2001). The only finding bearing on the functional significance of the visual P2 peak (200–250 ms) is it's enhancement in novel stimulus responses (Knight, 1997). The visual N2 (350–400 ms) peak is sensitive to motion stimuli but is also robust in object ERPs (Simon-Cereijido et al., 2006), indexing higher-level visual processing.

ERP studies on visual aging are extremely scarce. Virtually nothing is known about the differential aging of distinct stages of visual cortical processing, as reflected by cortical visual ERPs. Earlier studies of visual aging used pattern-reversal techniques, predominantly targeting activity in striate visual areas (Dustman et al., 1981; Snyder et al., 1981), and measured mean amplitudes over time intervals spanning several VEP deflections. Nonetheless, the overall pattern that emerged appears to be quite different from that seen in the auditory modality. Instead of the augmentation–diminution pattern

seen in the auditory modality, majority of VEP studies found a decrease of VEP latencies and amplitudes, however preferentially to high spatial frequency stimuli (Crognale, 2002; Fiorentini et al., 1996; Porciatti et al., 1992). An exception is a study by Taroyan et al. (2004) who found a large increase in visual P2 peak from the 2nd to 7th decade of life. Second, not only do VEP amplitudes diminish with age, but their waveform morphology loses complexity (Dustman et al., 1981). Third, VEP amplitude augmentation as a function of stimulus intensity has been found to increase with age (Dustman et al., 1981). The waveform dedifferentiation and amplitude-intensity findings were interpreted in terms of diminished visual feature selectivity due to declining inhibitory capacity in *local* visual networks (Dustman et al., 1990; Dustman et al., 1996). Indeed, there is both single-cell (Leventhal et al., 2003) and human psychophysical data demonstrating declines in center-surround inhibition with age (Betts et al., 2005). However, local disinhibition cannot explain the predominant finding of VEP amplitude diminution with age, especially in the context of minimal age-related neuro-biological changes in the striate system (review in Spear (1993)). Consistent with this, a recent study reported evidence suggestive of *activation* deficits in visual aging (Zaletel et al., 2005). In that study, both younger and older subjects enhanced VEP (and cerebral blood flow) in response to greater stimulus contrasts. However, the overall VEP amplitudes were smaller in the older than younger subjects, and this difference increased with higher-contrast stimuli. Therefore, unlike in the auditory modality, in which inhibitory deficits have been suggested to dominate, the deficits in visual modality point to the predominance of activation deficits.

The reasons for such robust between-modality differences are not known nor have been considered. However, it would be important to examine these differences in order to understand the degree of universality vs. specificity of inhibitory, facilitatory, and local sensory deficits. This is a pre-requisite for studying audio-visual integration and compensatory attentional mechanisms. To allow for a valid between-modalities comparison, data from both modalities must be collected in the same paradigm. In the present context, this also means eliminating active attention.

Therefore, the aims of the present study were: (1) based on the known functionality of the sensory ERP peaks, to determine whether, and how, age-related deficits manifest during sensory processing in auditory and visual modalities when the effects of attention are minimized; and (2) compare age-related findings between the two modalities with the goal of identifying modality-general and modality-specific patterns of sensory aging.

4. Results

4.1. *Behavioral performance*

4.1.1. *Accuracy*

Both groups were more accurate in visual compared to auditory attention tasks (93.8±7% vs. 87.1±11%; $F(1,36)=16.86$, $p<.0002$). There were no group differences (main effects or interactions) in the accuracy of response.

Both groups made more false-alarm responses (i.e., button presses to any stimulus but the target) during auditory than visual attention tasks (1.7±3% vs. .8±1%; $F(1,36)=4.33$, $p<.05$). Overall, older adults made more false-alarm responses than younger adults (2.0±2% vs. .6±1%; $F(1,36)=8.40$, $p<.007$), but no Group×Modality interaction was found.

4.1.2. *Reaction time*

Both groups responded more quickly to visual than to auditory targets (391.5±40 ms vs. 444.4±60 ms; $F(1,36)=57.17$, $p<.0001$). There were no group differences (main effects or interactions) in speed of response.

4.2. *Electrophysiological results*

In both groups, the unattended auditory and visual non-targets elicited a typical sequence of P1, N1, P2, and N2 ERP peaks (Figs. 1 and 2). In the older adults, the earlier-latency P1 and N1 peaks were largely intact in the auditory modality but were severely diminished in the visual modality. The long-latency N2 peak showed an opposite effect: it was intact in the visual modality but diminished in amplitude in the auditory modality. The only similarity across the two modalities was an age-related enhancement in the P2 latency range (Fig. 3).

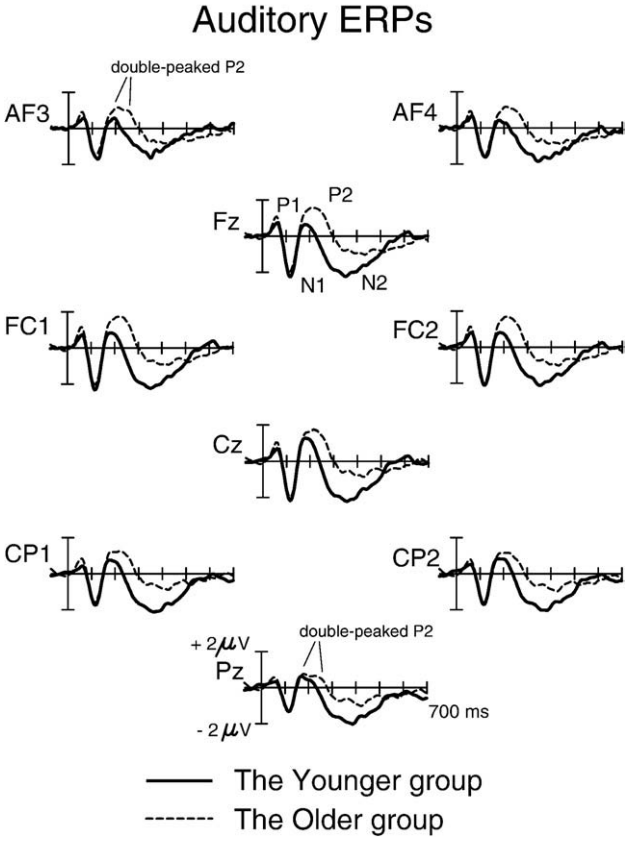

Fig. 1 – Auditory sensory ERPs elicited by the unattended auditory non-target stimuli in the Focus condition in the Younger (thin tracings) and Older (thick tracings) groups.

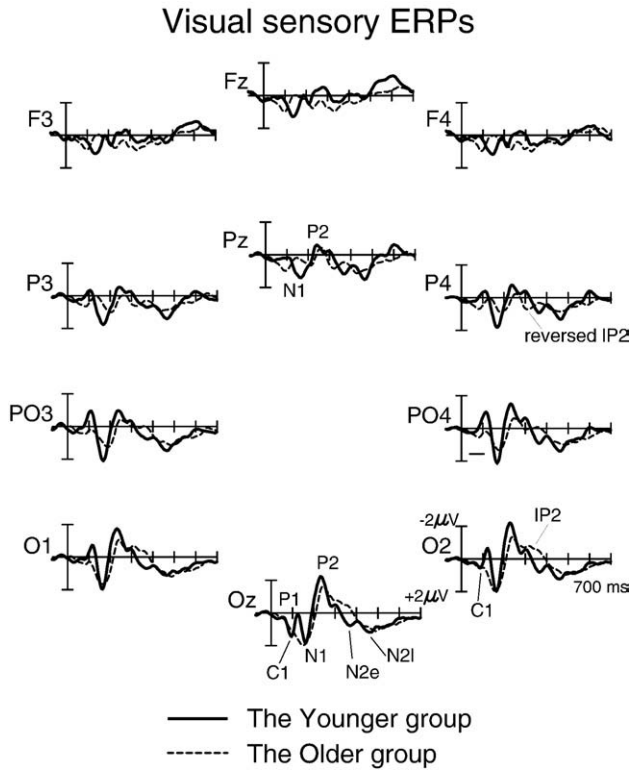

Fig. 2 – Visual ERPs elicited by the unattended visual non-target stimuli in the Focus condition in the Younger (thin tracings) and Older (thick tracings) groups.

4.3. *Auditory ERPs*

4.3.1. *P1*

In both groups this peak was predominant fronto-centrally (Electrode effect[1] $F(23,828)=8.17$, $p<.0001$, $\eta^2=.19$; Figs. 1 and 2) and showed no interactions involving Group. Although the P1 appeared larger in amplitude in the Older than Younger group (Table 2), this difference was not statistically significant. The peak latency of the P1 was equal in both groups (Table 3).

4.3.2. *N1*

The N1 was also predominant fronto-centrally in both groups (Electrode effect $F(23,828)=16.12$, $p<.0001$, $\eta^2=.31$) and showed no group differences in either the scalp distribution, amplitude (Table 2), or latency (Table 3).

4.3.3. *P2*

The auditory P2 peak showed a typical vertex-centered scalp distribution, with maximal values seen exclusively at the Cz electrode in both groups (Electrode effect: Younger, $F(23,414)=6.14$, $p<.0001$; $\eta^2=.25$; Older, $F(23,414)=12.81$, $p<.0001$, $\eta^2=.42$).

4.3.3.1. Group effects. Auditory P2 showed a strong trend to be larger in amplitude in the Older than Younger participants ($F(1,36)=3.40$, $p<.07$, $\eta^2=.09$ [5 parietal electrodes excluded]). Further, a significant Group×Electrode interaction ($F(18,648)=2.54$, $p<.04$, $\eta^2=.07$) indicated that the P2 had more activity over the frontal electrodes, but less activity over the parietal electrodes, in the Older than Younger subjects (Fig. 4). The P2 also peaked later in the Older than Younger group (($F(1,36)=6.37$, $p<.02$, $\eta^2=.15$; Table 3, Figs. 1 and 3).

In addition, the group difference, measured at the latency of the largest between-group difference in the later P2 range (250 ms; Fig. 3), was highly significant ($F(1,36)=31.25$, $p<.0001$, $\eta^2=.46$). The Older subjects showed positivity (.72 μV) and the younger subjects showed negativity (−.79 μV).

4.3.4. *N2*

In the Younger group, the auditory N2 peak was a robust, ubiquitous negativity that predominated over the frontal, central, and parietal regions at and around the midline (Fig. 1; Electrode effect $F(23,414)=8.66$, $p<.0001$, $\eta^2=.33$). In the Older group, the N2 was more poorly expressed and did not show an Electrode effect.

4.3.4.1. Group effects. The auditory N2 was smaller in amplitude ($F(1,18)=33.04$, $p<.0001$, $\eta^2=.65$) and longer in latency ($F(1,36)=8.20$, $p<.007$, $\eta^2=.19$) in the Older than Younger subjects (Tables 2 and 3). Further, it was posterior in scalp distribution in the Younger than Older group (Group×Electrode interaction $F(23,828)=2.88$, $p<.03$, $\eta^2=.07$; Fig. 5).

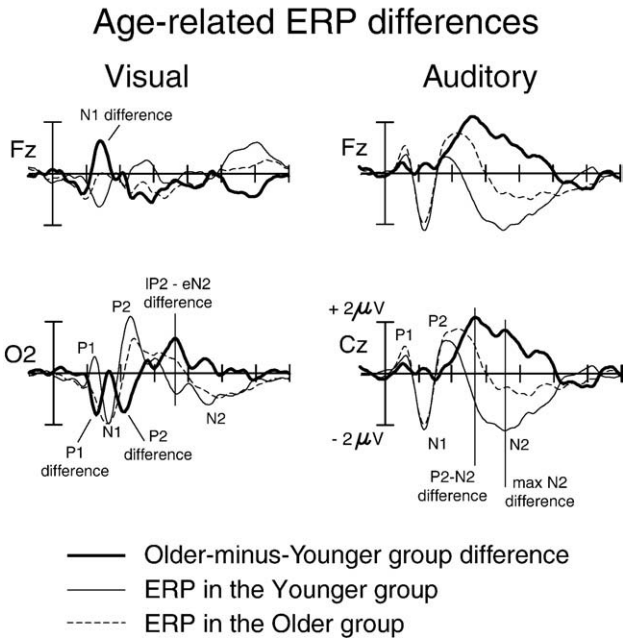

Fig. 3 – Age Group difference in the visual (left panel) and auditory (right panel) ERPs. The age effects are similar across the two modalities in the longer latency range (enhanced P2, diminished N2). In contrast, at the earlier latencies robust age-related changes are seen in the visual but not auditory ERPs.

[1] Electrode effects are provided to validate the described scalp distributions.

Table 2 – Peak amplitudes (μV (sem)) of the auditory ERPs in the Younger and Older groups

Peak	Younger					Older				
	Fz	FC1	FC2	Cz	Pz	Fz	FC1	FC2	Cz	Pz
P1	1.02[3] (.18)	1.02[3] (.19)	1.08[3] (.15)	1.04[3] (.20)	.69[2] (.21)	1.24[3] (.21)	1.35[3] (.23)	1.30[3] (.17)	1.28[3] (.24)	.90[3] (.19)
N1	−2.39[3] (.44)	−2.47[3] (.45)	−1.91[3] (.46)	−2.36[3] (.47)	−1.58[3] (.47)	−2.03[3] (.29)	−2.10[3] (.33)	−1.66[3] (.25)	−2.11[3] (.29)	−1.45[3] (.21)
P2	1.21[2] (.32)	1.38[3] (.35)	1.29[3] (.30)	1.78[3] (.38)	1.08[1] (.29)	2.01[3] (.30)	2.22[3] (.30)	1.64[3] (.24)	2.21[3] (.32)	1.27[3] (.27)
N2	−2.36[3] (.22)	−2.44[3] (.23)	−1.74[3] (.22)	−2.53[3] (.24)	−2.21[3] (.22)	−1.12[3] (.21)	−1.06[3] (.22)	−1.06[3] (.18)	−.98[3] (.23)	−1.04[3] (.23)

All reported values were significantly different from zero baseline: [1]$p<.01$, [2]$p<.005$, [3]$p<.001$.

4.4. *Visual ERPs*

4.4.1. *P1*

In the Younger group, the visual P1 peak was maximal at the parieto-occipital electrodes (Electrode effect $F(30,540)=5.06$, $p<.004$, $\eta^2=.22$). In the Older group, the P1 was severely impoverished and was clearly present only over the lateral parietal sites (Electrode effect: $F(30,540)=2.34$, $p<.08$, $\eta^2=.12$; Fig. 2, Table 4).

4.4.1.1. *Group effects.* Over the parieto-occipital scalp (electrodes: P7, P3, Pz, P4, P8, PO3, PO4, O1, Oz, O2), visual P1 was smaller in the Older than Younger subjects (.26 vs..97 μV; $F(1,36)=4.62$, $p<.04$, $\eta^2=.11$). Across 28 electrodes (all except O-s where the P1 was not expressed in the Older), visual P1 peaked later in the Older than Younger group (123 vs. 110 ms, respectively, $F(1,36)=4.73$, $p<.04$, $\eta^2=.12$).

4.4.2. *N1*

Consistent with its occipital and temporo-parietal components (Di Russo et al., 2002), in the Younger group, visual N1 predominated over the parieto-occipital regions and fronto-central midline (Electrode effect $F(30,540)=2.99$, $p<.04$, $\eta^2=.14$; Fig. 2; Table 4). Its latency was longer over the parieto-occipital than fronto-central areas (ca. 145 vs. 170 ms; $F(30,540)=9.38$, $p<.0001$, $\eta^2=.34$).

In the Older group, the visual N1 peak was better preserved than the P1 and was best seen over the parieto-occipital electrodes. However, the Electrode effect was not significant, indicating less expressed regional differences. As in the Younger group, in the Older group the N1 latency was longer over the parieto-occipital than fronto-central scalp (150 vs. 185 ms; Electrode effect $F(30,540)=7.48$, $p<.001$, $\eta^2=.29$).

4.4.2.1. *Group effects.* The visual N1 was larger in amplitude in the Younger than Older group ($F(1,36)=5.19$, $p<.03$, $\eta^2=.13$, Table 4), with no significant scalp distribution differences. Although the N1 peaked somewhat earlier in the Younger (158 ms) than in the Older (167 ms) group, this was not statistically significant.

Table 3 – Peak latency (SD) of the auditory ERPs in the Younger and Older groups at the Cz electrode

	P1	N1	P2	N2
Younger	61 (13)	119 (14)	184 (22)	341 (45)
Older	59 (11)	118 (11)	201 (26)	371 (42)

4.4.3. *P2*

In both groups, the visual P2 was maximal over the parietal and occipital regions (Electrode effect, Younger: $F(30,540)=8.47$, $p<.0001$, $\eta^2=.32$; Older: $F(30,540)=11.09$, $p<.0001$, $\eta^2=.38$; Fig. 2, Table 4).

4.4.3.1. *Group effects.* When measured at the peak, the visual P2 was smaller in amplitude in the Older than Younger subjects ($F(1,36)=7.04$, $p<.01$, $\eta^2=.16$), with no differences in scalp distribution or latency (means of 239 ms and 243 ms in the Younger and Older, respectively).

4.4.4. *Late P2 (lP2)*

Over the occipital electrodes, an additional positivity was seen in the Older group at the latency of 300–380 ms, here called the late P2 (lP2; Fig. 2). In this group, visual ERPs at 350 ms post-stimulus were positive over the occipital scalp but negative over the fronto-central scalp, with polarity reversal between the occipital and parietal sites (Electrode effect $F(30,540)=8.97$, $p<.0001$, $\eta^2=.33$; Fig. 2).

4.4.4.1. *Group effects.* Since the lP2 was not expressed in the Younger group, the group difference was assessed at 350 ms, the latency of the largest between-group difference (Fig. 3). While the main Group effect was not significant, the Electrode × Group interaction was ($F(30,1080)=4.39$, $p<.004$, $\eta^2=.11$). It originated from the opposite lP2 distributions in the two groups over the fronto-central vs. parieto-occipital areas (Fig. 6).

4.4.5. *N2, early window*

The early N2 peak was the first out of two peaks of a broad negativity following P2 in the latency window of 300–600 ms (Fig. 2). In the Younger group, the N2e was maximal over the back of the head and steadily diminished in amplitude

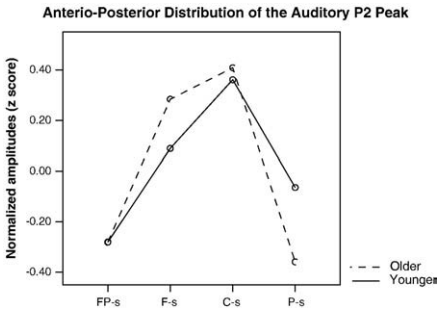

Fig. 4.

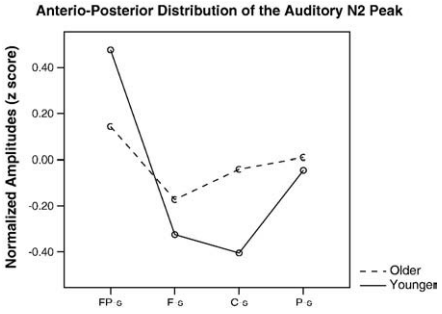

Fig. 5.

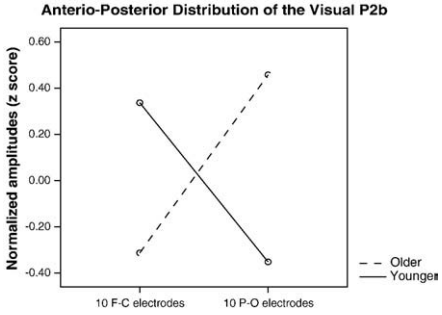

Fig. 6.

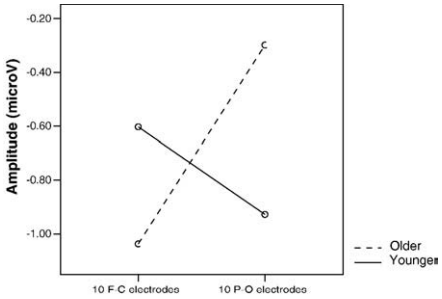

Fig. 7.

anteriorly (Electrode effect $F(30,540)=2.23$, $p<.08$, $\eta^2=.11$). In the Older group, a positivity (the P2b) replaced the eN2 of the Younger group over the parieto-occipital electrodes (Fig. 3; Electrode effect $F(30,540)=9.72$, $p<.0001$, $\eta^2=.35$).

Group comparison confirmed the eN2 pattern seen in the group analyses (Fig. 7): in the Younger subjects, the eN2 was prominent over the parieto-occipital, whereas in the Older group — over the fronto-central scalp (Group × Electrode effect ($F(30,1080)=6.03$, $p<.0001$, $\eta^2=.14$). The Older group showed shorter eN2 latencies (330 ms) than the Younger group (357 ms; $F(1,36)=10.97$, $p<.002$, $\eta^2=.23$).

4.4.6. *N2, later window*

In contrast to eN2, the lN2 was maximal over the parieto-occipital regions in both groups. In both groups, it steadily diminished in amplitude anteriorly (Fig. 2; Electrode effect: Younger, $F(30,540)=5.62$, $p<.0001$, $\eta^2=.24$; Older, $F(30,540)=5.22$, $p<.001$, $\eta^2=.23$).

4.4.6.1. Group comparison. There were no lN2 amplitude or scalp distribution differences between the groups. The lN2 latency showed a trend to be shorter in the Older than Younger subjects ($p<.07$; 451 ms vs. 466 ms, respectively).

4.5. *ERP–behavior correlations*

The most consistent association of behavior with the ERPs was with the auditory N2 and early visual N2 (Fig. 8). A larger unattended N2 was significantly associated with better target detection performance (more hits, fewer false alarms, correspondingly greater adjusted score) in the concurrently attended modality: visual ($r(37)=.46$, $p<.002$); auditory ($r(37)=.33$, $p<.025$). A larger unattended auditory N2 was also significantly associated with better performance in auditory modality, i.e., performance that occurred at a different time ($p<.025$). However, the auditory and visual N2 waveforms were also significantly correlated with each other (larger auditory N2 associated with larger visual N2, $r(37)=.46$, $p<.002$). Therefore, we computed partial correlations in which the N2 in the alternate modality was controlled for. When the common variance associated with the correlated ERP indices was controlled, we found that a larger (more negative) N2 was associated with a higher percent correct in an adjusted accuracy score in the concurrent, alternate modality (visual: $r(37)=-.363$, $p<.015$; auditory: $r(37)=-.287$, $p<.045$) but not with the performance in the same modality, which happened at a different time.

When partial (controlled) performance–brain response correlations were examined for Younger and Older groups separately, the effects were significant in the younger adults only: Auditory, Young: $r(16)=-.58$, $p<.006$; Auditory, Older: $p>.30$; Visual, Young: $r(16)=-.54$, $p<.1$, Visual, Older, $r(15)=-.30$, $p<.12$.

Table 4 – Peak amplitudes (μV (sem)) of the visual ERPs in the Younger and Older groups

Peak	Younger					Older				
	Fz	PO3	PO4	O1	O2	Fz	PO3	PO4	O1	O2
P1	−.09 (.26)	1.26[1] (.41)	1.56[3] (.34)	1.14 (.48)	1.28[1] (.44)	.08 (.23)	.27 (.20)	.38 (.16)	.23 (.22)	−.01 (.17)
N1	−2.06[3] (.22)	−2.52[3] (.40)	−2.74[3] (.39)	−2.36[3] (.44)	−2.24[3] (.40)	−1.50[3] (.21)	−1.68[3] (.29)	−1.85[3] (.34)	−2.03[3] (.46)	−2.09[3] (.53)
P2	.92[2] (.29)	1.58[3] (.29)	1.96[3] (.33)	2.21[3] (.31)	2.58[3] (.28)	−.06 (.25)	.83[1] (.27)	1.20[3] (.15)	1.49[3] (.30)	1.94[3] (.30)
eN2	−.73[2] (.20)	−.95[3] (.25)	−1.05[3] (.26)	−.90[2] (.28)	−.92[2] (.30)	−1.10[3] (.21)	−.29 (.19)	−.26 (.16)	.14 (.19)	.46 (.26)
lN2	−.41 (.28)	−1.52[3] (.35)	−1.55[3] (.31)	−1.38[3] (.27)	−1.41[3] (.29)	−.53 (.15)	−1.24[3] (.25)	−1.21[3] (.20)	−1.19[3] (.25)	−1.07[3] (.18)

Significance from zero baseline: [1]$p<.01$, [2]$p<.005$, [3]$p<.001$, or less unmarked values not significant.

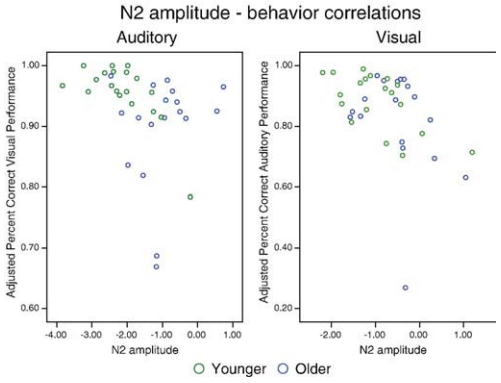

Fig. 8 – Partial correlations between behavior (Adjusted score=Hits minus False Alarms) and N2 amplitudes in auditory (left) and visual (right) modalities. The N2 amplitudes were controlled for the N2 magnitude in the alternate modality.

The N1-performance trends pointed to similar, positive correlations as the N2: Auditory N1 — Visual performance, $p<.09$, Visual N1 — Auditory performance $p<.07$.

5. Discussion

This study investigated age-related changes in auditory and visual sensory ERPs elicited by unattended non-target stimuli. In the auditory modality, the earlier responses (P1 and N1) were unaffected by age while later responses changed significantly (P2 increased, N2 decreased). In contrast, in the visual modality early ERP peaks (P1, N1, P2, early N2) diminished with age while the late N2 did not change. The only resemblance in age-related effects across the two modalities was enhanced positivities in the late P2 range.

5.1. *Auditory aging*

A prevailing account of age-related changes in perceptuo-cognitive functioning is failure of inhibitory control (Hasher et al., 1991; Knight et al., 1999; Kok, 1999). An early form of inhibitory function is prefronto-thalamo-cortical gating, reflected by enhanced MLAEPs in PFCx lesion patients (Knight et al., 1989). In aging literature, inhibitory deficit has been identified with the amplitude increases of the P1, N1, and P2 peaks (Amenedo and Díaz, 1998a, 1999; Chao and Knight, 1997a; Friedman et al., 1993; Kovacevic et al., 2005). However, only MLAEPs were enhanced due to the PFCx lesions, and only the P1 peak of long-latency ERPs can be assigned to MLAEPs. Therefore, only the P1 age-related effects can be somewhat reliably attributed to gating deficits. In the present study, the P1 showed a trend to be larger in amplitude in the Older than Younger group. Therefore, while the present sample of healthy aging adults might have had a tendency for the gating deficit, it was not robust. There was no correlation between the P1 amplitude and false-alarm rates. However, unlike the majority of earlier studies, the present study utilized a cross-modal inattention design. Cross-modal segregation is easier to achieve than within-modal segregation, which may render implementation of gating easier.

The auditory N1 and P2 peaks have been associated with sensory arousal and triggering of attentional orienting (Čeponienė et al., 2005; Näätänen, 1990), and therefore are likely candidates for top-down modulatory control. Consistent with this, Knight et al. (1980) found an enhanced auditory N1 in patients with prefrontal lobe lesions; the P2 was unaffected in those same patients, however. Newer brain lesion data indicate that during active attention tasks, auditory N1 can be diminished by lesions in ipsilateral tertiary PFCx (Chao and Knight, 1998; Knight et al., 1999). By comparison, when subjects with prefrontal lesions ignored auditory stimuli and performed a visual task, their N1 and P2 peaks were comparable to those of the controls (Alho et al., 1994). These results suggest that auditory N1 may be subjected to both inhibitory and facilitatory cortico-cortical influences. It may be the balance between these two modulations that determines the observed net ERP effect, which could explain variable age-related findings on auditory N1. Only one (Amenedo and Díaz, 1999) of the four studies (Amenedo and Díaz, 1998a,b; Anderer et al., 1996; Iragui et al., 1993) that found age-related increase in the auditory N1 examined responses to unattended stimuli, and in that study, a within-modality selective attention was utilized. In contrast, in the present study in which attention was engaged in another modality, no age-related N1 enhancement was observed. Therefore, it appears that when the top-down facilitation is minimal (no attention), PFCx–thalamic gating may prove sufficient in the elderly. It may be that only the interference-prone situations requiring, for example, within-modality selection can reveal age-related inhibitory deficits at the level of the auditory N1. In addition, in the present study, larger N1 amplitudes tended to correlate positively with the accuracy on the alternate-modality task. That is, larger N1 amplitudes did not index distraction; rather, they seem consistent with a level of sensory arousal favorable for task performance.

Another potential source of result variability in auditory aging literature is impact of hearing loss. A valuable observation was that high-frequency selectivity of the age-related hearing loss may yield larger N1 amplitudes in the elderly, since the N1 amplitude is smaller for high-frequency sounds and larger for low frequency sounds (Tremblay et al., 2003).

Overall, the intact auditory P1 and N1 peaks indicate that the earlier processing stages are fairly resistant to neurobiological aging and that in the Older group of the present study, acoustic information was delivered from auditory periphery to the cortex with reasonable fidelity and on time. This argues against age-related general slowing at the sensory level and is in line with the studies showing no effect of perceptual thresholds on cognitive performance (Lindenberger and Baltes, 1994; Lindenberger et al., 2001).

In contrast to N1, the auditory P2 peak showed a strong tendency to be enhanced in the Older group of the present study, corroborating previous findings (see Table 1). This peak is even more sensitive to acoustic salience than the N1 (Näätänen, 1990; Williams et al., 2006; Woods et al., 1984a); p. 217), and its amplitude and latency follow perception more closely than those of the N1 (Crowley and Colrain, 2004). Brain lesion data corroborate the association between the P2 and the perceived stimulus salience: the auditory P2 was intact in PFCx patients when tone pips were used as stimuli (Alho et al., 1994; Knight et al., 1989) but it was enhanced when environmental sounds

were employed (Chao and Knight, 1998). Further, in a patient with bilateral auditory cortex damage, both supra- and below-threshold stimuli elicited a comparable P1–N1 complex, but the below-threshold stimuli elicited small, or no, P2 (Woods et al., 1984b). Therefore, the P2 might reflect access of sensory information to conscious perception mechanisms depending on stimulus, subject's state, and/or task (Čeponienė et al., 2005) (see also Näätänen (1990) and Woods et al. (1984b)). If so, it is likely that the P2 enhancement, observed in the Older group of the present study, reflects a lowered perceptual threshold – an inhibitory deficit – which is consistent with an overall higher False-Alarm rates in this group. However, there was no correlation between the P2 measures and False-Alarm rates. Therefore, an alternative explanation is that the enhanced P2 in the Older group is caused by an overlap with a positivity that is not identical to the auditory P2 but is similar to that seen in the visual data (see Section 5.3).

In the present study, auditory N2 was diminished in amplitude and prolonged in latency in the Older, as compared with the Younger, group. Further, overall the N2 correlated positively with performance accuracy and correlated negatively with False-Alarm rates *in the alternate modality*. Data plotted in Fig. 8 show a clear relationship between the N2 amplitude and behavior in both the younger and older subjects. However, when the two groups were analyzed separately, the N2–behavior correlation in the Older group did not reach significance. In part, this appears to be caused by few outliers in behavior, a measure that could be contributed by numerous cognitive factors. However, the most direct interpretation of the existing result would be that, at least in part, the N2 reflects inhibition of unattended stimuli. If so, inhibition appears to be diminished with age, which is in line with the interpretation offered by Bertoli et al. (2005).

On the other hand, these stimuli belonged to the unattended channel, and therefore were inhibited at a channel level in a "gestalt" manner. Channel-level selection occurs early, by 50–100 ms post-stimulus and is indexed by Processing Negativity, a negative difference between attended and unattended stimuli (Näätänen, 1990), which was not examined in the present paper. If so, processes that are reflected during the late N2 range (300–450 ms) reflect stimulus-level, rather than channel-level, processing. Further, it is known that auditory N2 increases in amplitude when sensory processing is "disconnected" from perception, e.g., during sleep (Paavilainen et al., 1987) or when inhibitory capacities are under-developed, as in young children (Čeponienė et al., 2005). In a way, this resembles increased overall EEG amplitudes during relaxed vs. alert states (e.g., predominance of alpha vs. beta rhythm, respectively). This is in line with an earlier suggestion that during awake, attentive state the auditory cortex is under the influence of local tonic inhibition, which is removed by the impinging stimuli in a stimulus-specific manner (Näätänen, 1992). If so, efficient channel-level filtering occurring early in stimulus processing may provide a partial functional "disconnect" between sensory processing and perception, and remove some of the tonic inhibition, resulting in larger amplitudes of the N2. The net result of this and the first interpretation above is the same — larger N2 amplitudes signify better net inhibition of unattended sounds and, by extension, a deficit in inhibitory capacity in the Older group. However, the type of inhibition, and the implied functional role of the N2, differs between the two accounts. The latter is more consistent with the maturational trajectory of the N2, which parallels cortical cyto-architectonic development in frontal and auditory sensory regions (Eggermont and Ponton, 2002; Huttenlocher and Dabholkar, 1997; Moore, 2002), and its proposed role of integrative sound content processing (Čeponienė et al., 2005; Čeponienė et al., 2001; Čeponienė et al., in press).

Consistent with the latter, the N2 diminution with age could represent cyto-architectonic derangement in sensory regions, including diminished synaptic synchronization causing decreased processing speed (Peters, 2002; Peters et al., 1994). This is consistent with the fact that the diminished N2 amplitude is the only auditory ERP finding that is in agreement with age-related decrease in auditory acuity (Baltes and Lindenberger, 1997; Li et al., 2001b; Lindenberger et al., 2001; Plyler and Hedrick, 2002; Tremblay et al., 2003; Tun and Wingfield, 1999), and with the current finding of increased N2 latency in the Older group. The N2–behavior correlation, then, could simply reflect an overall better sensory processing.

5.2. *Visual aging*

In the present study, all visual ERP peaks but the late N2 were diminished in the older, as compared with the younger, subjects. This is in agreement with earlier VEP (Dustman and Snyder, 1981; Dustman et al., 1981; Snyder et al., 1981) and fMRI findings of diminished activity in primary visual cortices (Cabeza et al., 2004; Grady et al., 1994; Nielson et al., 2002) as well as with behavioral decline in visual perceptual abilities with age (Baltes and Lindenberger, 1997; Spear, 1993). Therefore, the first account to consider would be a decline in sensory neural recourses. In a thorough review of visual aging, Spear (1993) concluded that neuro-biological losses are minimal in the afferent visual system up to striate cortex. Consistent with this, brain imaging studies failed to reveal a significant volume loss in occipital cortex from 20 to 77 years of age (Murphy et al., 1996; Raz et al., 2005; Sowell et al., 2003). While an important piece of information, reliable synaptic counts, is still lacking, it appears that the basis for visual sensory decline may be functional rather than structural.

One hypothesis offering a functional explanation is that of local disinhibition (Dustman et al., 1990; Dustman et al., 1996). This notion gained recent support from findings of diminishing center-surround inhibition with age (Betts et al., 2005; Leventhal et al., 2003). However, while this is a fitting explanation for age-related decrease in visual acuity, it does not seem to be reflected in electrophysiological data, overwhelmingly demonstrating a decline of visual activity with age.

An alternative hypothesis, then, is one of a modulatory decline. Visual predominance in human attention is a reliably established phenomenon in both behavioral and electrophysiological experiments (Colavita and Weisberg, 1979; Golob et al., 2001; Posner et al., 1976). Indeed, before visual image can be processed in detail, it must be focused upon. Stimuli that are in visuo-spatial attention elicit greater electrophysiological activity and those that fall outside the attentional focus elicit diminished activity (Hillyard and Anllo-Vento, 1998). Therefore, optimal visual processing may require an integration of bottom-up input with top-down facilitation by visuo-

spatial attention mechanisms. This is corroborated by PFCx lesion data showing diminished visual N1 (Chao and Knight, 1998; Knight et al., 1999) as well as the present aging data demonstrating intactness of the attention-independent early visual C1 (Fig. 2). This latter finding resembles sparing of the early auditory peaks with age and suggests that the origin of visual ERP diminution is not peripheral. Therefore, severe age-related diminution of the visual P1, N1, and P2 peaks found in the present study could be explained by the diminished top-down facilitation of sensory input when attention is directed away from the eliciting stimuli. This is in line with the fact that frontal and superior parietal regions, involved in visuo-spatial attention, show volume depletion with age (DeCarli et al., 2005; Raz et al., 2005; Resnick et al., 2003). Interestingly, this is also in line with the finding that only those visual peaks that are most reflective of visuo-spatial attention, the P1 and N1, increased in latency with age.

Similar to auditory N2, the early portion of the visual N2 peak significantly and positively correlated with behavior. The age-related diminution of this peak, then, suggests a deficit in long-latency inhibition of visual stimuli in the older group. Due to the long latency of this effect, it is unlikely to represent lateral center-surround inhibition operative for stimulus features. Rather, this is a task-related inhibition which was more likely to happen at a stimulus level since visual stimuli occurred in the visual field.

Finally, cognitive resource sharing may have contributed to age-related diminution of early visual peaks. Since in the current experiment, the non-target visual stimuli occurred in front of the subject, the younger subjects may have been able to maintain a certain level of visuo-spatial facilitation of the visual stimuli while performing an auditory task. In contrast, the older subjects may not have been able to do that due to the insufficient attentional resources given the relative difficulty of the auditory task.

Therefore, overall it appears that facilitatory, rather than inhibitory, deficits prevail in visual sensory aging.

5.3. *Between-modality differences*

This study found profound differences in age-related changes in auditory and visual ERPs. While the former showed a pattern of intact–enhanced–diminished responses, the latter showed a nearly uniform diminution. Such differences are unlikely to be accounted for by gross morphological differences in auditory vs. visual cortices. Brain imaging studies show minimal, if any, cortical thinning in sensory cortices with no clear differences between the supra-temporal and occipital areas (Murphy et al., 1996; Raz et al., 2005; Sowell et al., 2003, 2002). GABA levels were found to decline with age in both auditory and visual sensory regions in mammals and non-human primates (Leventhal et al., 2003; Ling et al., 2005). The suggested consequence of this decline is diminished perceptual accuracy (Betts et al., 2005; Hua et al., 2006; Schmolesky et al., 2000; Yu et al., 2006). In the absence of direct comparison studies, it is not possible to compare the magnitude of GABA decline across the two cortical regions. However, the general impression is that the between-regional differences in GABA levels could not account for the salient differences found here between the auditory and visual ERPs. Therefore, alternative explanations need to be sought.

As described above, visual attentional predominance is a salient perceptual phenomenon. In contrast, auditory processing is far less dependent on attention. Quite to the opposite, acoustic information is free to enter the auditory system regardless of where the attention is directed. Accordingly, this modality is equipped with powerful inhibition and selection mechanisms serving to avoid sensory overload with irrelevant information (Näätänen, 1990). Therefore, processing of irrelevant stimuli in audition is relatively independent of concurrent top-down facilitation. Rather, irrelevant sounds are gated. Accordingly, age-related changes in the auditory modality are more likely to be caused by inhibitory deficits that appear as enhancements of the earlier-latency ERP peaks. In contrast, visual sensory processing strongly relies on facilitation by visuo-spatial attention. This suggests an explanation for the diminished visual sensory activity under the present inattention conditions that is also consistent with the behavioral findings of poorer visual than auditory perception in the elderly (Lindenberger and Baltes, 1994). The reason for the latter may be due to a confound between the visual and attentional aging.

The only similarity between the modalities was age-related enhancement of the positivities in the late P2–early N2 range. An additional positivity, following the auditory P2 in the Older group, could be seen in the frontal and parietal waveforms (Fig. 1), which resembled the two positivities found in the visual modality (the P2 and lP2; Fig. 2). Since the auditory late P2 was closely associated with the P2 proper, its scalp distribution could not be assessed. However, the visual lP2 of the Older group had a scalp distribution distinct from that of either P2 or the eN2: it inverted polarity between the occipital and parietal sites, whereas neither the P2 nor the eN2 inverted polarity over the scalp (Figs. 2 and 3). This suggests a source of the lP2 in the lateral parieto-occipital cortex. The functional significance of the hypothetical new late auditory P2 component, the visual lP2, or the visual eN2, seen in the younger subjects, is unknown.

Brain imaging studies of age-related changes in visual perception have demonstrated redistribution of activity from the lower-order to the higher-order non-primary sensory cortices, which was interpreted as compensatory activity for the loss in fluency, automaticity, or dedifferentiation of sensory functions. (Cabeza et al., 2004; Grady et al., 1994; Madden et al., 1996; Nielson et al., 2002). However, negative, not positive, voltage in the lP2–eN2 range in the visual modality correlated with better performance. Therefore, these ERP changes are not related to compensatory mechanisms. Rather, they may reflect more general neuro-anatomic changes, e.g., thinning of cerebral cortex (since positivities are generated in deeper layers, they may become more exposed). A similar, general change may underlie a ubiquitous age-related frontal rotation of the auditory P2 and N2 peaks (although the P2 increased anteriorly while the N2 decreased posteriorly) and the auditory and visual P300 (Amenedo and Díaz, 1998a, 1999; Friedman et al., 1997; Iragui et al., 1993; Pfefferbaum et al., 1980; Polich, 1996).

6. Conclusions

Age-related differences between the two modalities are robust and appear to be rooted in differential reliance of auditory and

visual systems on modulatory influences inhibition in the auditory modality and facilitation in the visual modality. An important modality-common finding was positive correlation between task performance and auditory N2 and early visual N2 amplitudes, likely signifying long-latency inhibition of unattended stimuli. These ERP indices diminished in with age. More general neuro-anatomic changes are implied by such modality-common ERP effects as amplitude enhancement in the late P2 range and frontal rotation of multiple ERP peaks.

7. Experimental procedures

7.1. Subjects

Thirty-eight healthy, cognitively intact individuals (18 males) between 20 and 89 years of age were recruited to the study through advertisements in the local newspapers and community centers. Participants aged 20 to 40 years comprised the Younger group ($n=19$) and those aged 67 to 89 years comprised the Older group ($n=19$, Table 5).

Participants had no history of major neurological, psychiatric, or medical disorders. All had normal or adjusted to normal vision and hearing (none wore hearing aids). Verbal and performance IQ were assessed using the WASI-III (Wechsler, 1997). There were no significant differences between the groups in IQ measures or years of education. Participants in the Older group received a battery of neuropsychological tests to assure normal cognitive functioning, including the Mini Mental State Exam (MMSE) (Folstein et al., 1975), Dementia Rating Scale (DRS) (Mattis, 1988), Wechsler Memory Scale (WMS-III) (Wechsler, 1997), and the California Verbal Learning Test (CVLT-II Delis et al., 2000). Additionally, all were re-tested 12–18 months following initial testing to assure that none were in the early stages of a dementing disorder that might not have been detected at the first testing. All older study participants scored in the normal or above normal range on these tests (Table 5).

The study was approved by the Institutional Review Board of the University of California, San Diego, and informed consents were obtained from all participants prior to the study. Participants received a nominal payment for participation.

Table 5 – Demographic (mean (SD)) and neuropsychological test scores in the Younger and Older subjects

	Younger	Older
n	19 (8 male)	19 (6 male)
Age	25.5 (5)	71.3 (6)
Years of education	16.1 (2)	15.9 (3)
Verbal IQ	111.61 (10.9)	115.53 (8.1)
Performance IQ	113.44 (13.3)	112.68 (13.5)
Full scale IQ	114.06 (12.0)	116.00 (10.2)
MMSE [a]	n/a	28.1 (2)
DRS attention ss [b]	n/a	11.5 (2)
DRS total ss	n/a	11.2 (2)
CVLT-II *t*-score	n/a	58.1 (9)
Verbal memory ss [c]	n/a	12.6 (3)
Non-verbal memory ss [d]	n/a	11.8 (3)

Scaled scores (ss) have a mean of 10 and a standard deviation of 3, *t*-scores have a mean of 50 and a standard deviation of 10.
[a] MMSE scores reflecting normal cognitive function are 27–30; a score of 23 or lower is considered to reflect cognitive impairment; no subject in our sample scored 23 or lower.
[b] Dementia rating scale scaled scores (ss) of 9 or above reflect cognitively intact performance.
[c] Verbal memory scaled scores (ss) are the mean of Immediate and Delayed Logical Memory scales from the WMS-III or the WMS-III Abbreviated.
[d] Non-verbal memory ss are the mean of Immediate and Delayed scales from the WMS-III Visual Reproduction subtest or the WMS-III Abbreviated Family Pictures subtest.

7.2. Stimuli

The auditory stimuli were 100-ms duration, 550-Hz (target) and 500-Hz (non-target) sine-wave tones, amplitude-modulated at 5 Hz to render a smooth, arch-shaped envelope. The tones were played via 2 loudspeakers located at the sides of a 21-inch computer monitor situated at the eye level 1.5 m in front of the subject. At the subject's head, tone loudness was 63 dB SPL (sound pressure level). The visual stimuli were displayed on the computer monitor. They were light-blue (target) and dark-blue (non-target) 8.4 cm^2 filled squares subtending 3.3 degrees of visual angle, presented for the duration of 100 ms on a light-grey background. Interspersed among the target and non-target stimuli were the cues, 200-ms bimodal words "Look" and "Hear".

In this paper, we report data on the *unattended non-target stimuli* obtained in a Focus attention task. Facilitation and inhibition are inherently intertwined with attention or its withdrawal and therefore comprise significant confounds to sensory processing. Therefore, in order to understand age-related changes pertaining specifically to the sensory domain, attentional confounds are better avoided. The stimuli from an irrelevant channel of a different modality, as opposed to non-targets in an attended channel or an unattended channel in the same modality, are well-suited to elicit predominantly sensory activity. In similar auditory studies, subjects usually read a book or watch silent cartoons. In visual studies, subjects usually perform an auditory discrimination task (e.g., Woods et al., 1992b).

7.3. Experimental design and procedure

The auditory and visual stimuli were presented intermixed in a pseudo-randomized sequence with the inter-stimulus interval (ISI, offset-to-onset) between any two consecutive stimuli varying between 100 and 700 ms (Fig. 9). The stimuli were presented in blocks of 264 for a duration of 2.64 min. In each block, there were 12 "Hear" and 12 "Look" cues. In the Shift condition, not reported in this paper, the subjects shifted their attention following the cue. In the Focus condition, reported in this paper, subjects ignored the cues and attended to the same modality during the whole block. The modality to attend was indicated prior to each block. Stimulus probabilities were the same between the Auditory and Visual conditions; direction of attention was the only difference between them. The subjects' task was to press a button upon a detection of a relevant-modality target.

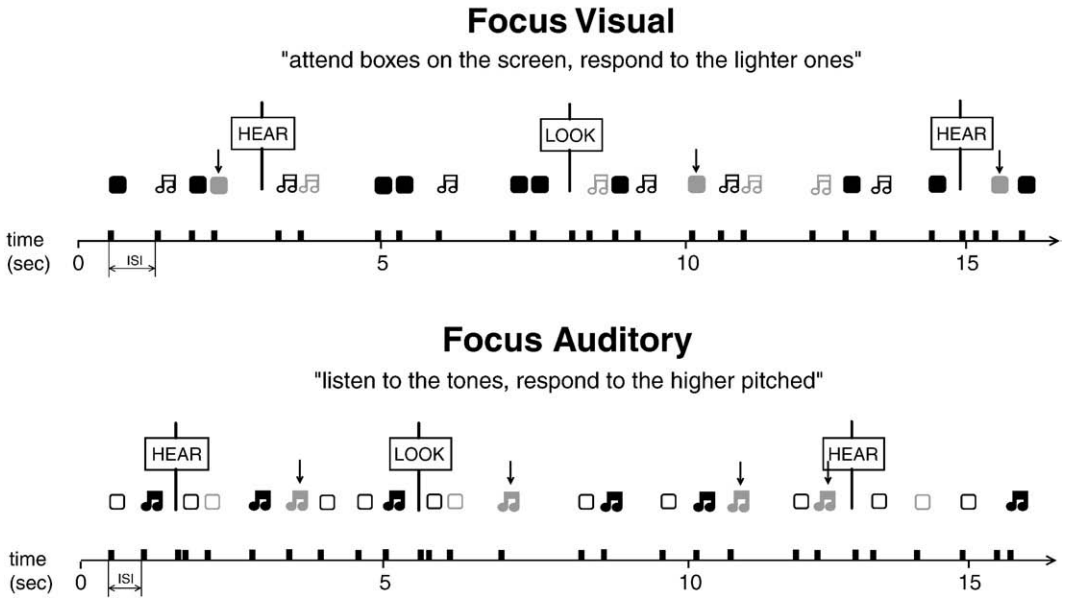

Fig. 9 – Layout of the experimental design. Subjects were asked to ignore the cues and attend to either the auditory or visual modality during the entire stimulus block. Filled shapes indicate attended stimuli, and unfilled shapes indicate unattended stimuli. Squares denote visual stimuli and note symbols denote auditory stimuli. Vertical arrows point to the targets. ISI — inter-stimulus interval.

Targets of each modality occurred at an overall probability of 10% (24 out of 240 stimuli, not counting the cues) and at a within-modality probability of 20% (24 out of 120 stimuli, respectively). The order of the Focus Auditory (FA) and Focus Visual (FV) sequences was randomized across subjects. Altogether, 384 unattended non-target stimuli per modality were presented during the Focus attention task. At all times, subjects had to fixate on a cross continuously presented in the middle of the computer monitor. They were visually monitored to ensure proper fixation.

7.4. *EEG recording and averaging*

Continuous EEG (amplification × 10.000, high- and low-pass filtered at .01 Hz and 60 Hz, respectively) was recorded from 33 scalp sites (FP1, FPz, FP2, AF3, AF4, F7, F8, F3, Fz, F4, FC1, FC2, FC5, FC6, T7, T8, C3, Cz, C4, CP1, CP2, CP5, CP6, P3, Pz, P4, P7, P8, PO3, PO4, O1, Oz, O2) of the International 10–20 system. The right mastoid served as a reference. Offline, the data were re-referenced to the average of the left- and right-mastoid recordings and corrected for blink and eye movement artifacts using independent component analysis (ICA, Jung et al., 2000).

The EEG was segmented into stimulus-locked epochs including 200 ms pre- and 900 ms post-stimulus onset time. Epochs containing body movement artifacts, as well as those with false-alarm button presses, were excluded from averaging. The remaining epochs were baseline-corrected in relation to the 200-ms pre-stimulus interval, digitally filtered using Gaussian finite impulse response function for frequencies 50 Hz and higher, and averaged by condition and stimulus type. Epoch overlap due to the short ISIs was mitigated by the even, pseudo-random distribution of ISI values between 100 and 700 ms. No evidence of significant overlap was seen in the waveforms.

7.5. *ERP measurements*

Auditory ERPs were measured at 24 electrode sites (FP1, FPz, FP2, AF3, AF4, F7, F8, F3, Fz, F4, FC1, FC2, FC5, FC6, C3, Cz, C4, CP1, CP2, CP5, CP6, P3, Pz, P4); visual ERPs were measured at 31 electrode (FP1, FPz, FP2, AF3, AF4, F7, F8, F3, Fz, F4, FC1, FC2, FC5, FC6, C3, Cz, C4, CP1, CP2, CP5, CP6, P3, Pz, P4, P7, P8, PO3, PO4, O1, Oz, O2). The different numbers of electrodes in the two modalities were defined by the respective ERP distribution. The ERP peak search windows (Table 6) were defined by visual inspection of the grand average waveforms (Figs. 1 and 2). Peak mean amplitudes were calculated over the time intervals roughly equaling 20% of that peak's duration in the grand average waveform. For each subject, these intervals were centered at their peak latency at each electrode.

Additional efforts were made to evaluate the ERP waveform in the P2–N2 latency range. In the visual ERPs, a positivity

Table 6 – ERP peak search windows and mean amplitude integration intervals

	Auditory		Visual	
Peak	Search window (ms)	Amplitude integration (ms)	Search window (ms)	Amplitude integration (ms)
P1	30–90	10	70–130	10
N1	70–170	16	120–220	16
P2	150–270	20	170–280	20
N2-w1	280–480	50	300–400	50
N2-w2	–	–	410–550	50

following but contiguous with the P2 peak could be seen at the parieto-occipital sites (Figs. 2 and 3). Similarly, in the auditory ERPs, the maximal between-group difference occurred at the latency following the P2 peak (Figs. 1 and 3). Therefore, in addition to measuring the P2 amplitudes, group difference waves were constructed to determine the timing of the largest between-group differences in the P2 latency range (Fig. 3). These latencies (auditory, 265 ms; visual, 350 ms) were used to center the 20-ms windows for mean amplitude measurements of the largest between-group difference in the late P2 range. The two peaks (early and late) of the visual N2 were measured separately, as indicated in Table 6.

7.6. *ERP analyses*

To determine whether activity of the ERP peaks was significant in comparison with the pre-stimulus baseline, peak amplitudes were subjected to two-tailed *t*-tests for independent samples at 5 most representative electrodes in each modality (auditory, Fz, FC1, FC2, Cz, and Pz; visual, Fz, PO3, PO4, O1, and O2). To account for multiple measurements in these analyses, alpha level was adjusted to .01.

The amplitude and latency differences between the age groups were tested using ANOVA with Age Group (Younger, Older) × Electrode as the factors. Unless noted otherwise, the 24 electrodes were used for auditory and 31 electrode for visual ERP data. When other electrode sets were used, these were determined based on the distribution of the ERP effect under analysis. *All* scalp distribution analyses (interactions involving Electrode factor) were done using z-score normalized data. Huynh–Feldt adjustment was applied when necessary. Only the significant results are reported.

7.7. *ERP–behavior correlations*

The primary measures of interest were correlations between ERP indices and behavior that occurred at the same time. Therefore, unattended visual ERPs were correlated with performance to auditory stimuli, and unattended auditory ERPs were correlated with performance to visual stimuli. In order to reduce the number of correlational analyses performed, we analyzed adjusted accuracy scores (correct responses minus false alarms). As a control for non-specific effects, we also examined correlations between ERP indices and behavior to stimuli in the same modality (i.e., behavior that occurred at a different time than when the unattended ERPs were recorded).

We used composite mean amplitude measures computed from groups of electrodes where the peak of interest was maximal. Electrode groups from which composite amplitudes were computed were as follows: fronto-central sites for auditory P1, N1, P2 and N2: Fz, F3, F4, Fc1, Fc2, Fc5, Fc6, Cz, C3, and C4; posterior sites for visual P1: PO3, PO4, O1, Oz, and O2; central and posterior sites for visual N1, P2 and N2: CP1, CP2, CP5, CP6, Pz, P3, P4, P7, P8, PO3, Po4, O1, Oz, and O2.

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