



Dissociation of motor and sensory inhibition processes in normal aging

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HIGHLIGHTS

- Older individuals showed neural markers of sensory enhancement and suppression in line with previous work (Gazzaley et al., 2008).
- ERP markers of motor inhibition (N2 and P3 amplitude and latency) are presented for the first time in a cohort of older individuals performing a stop-signal task.
- Distinct inhibitory functions (motor, sensory) are differentially impacted in normal aging, as evidenced by behavioral and neural findings using EEG.

ABSTRACT

Objective: Age-related cognitive impairments have been attributed to deficits in inhibitory processes that mediate both motor restraint and sensory filtering. However, behavioral studies have failed to show an association between tasks that measure these distinct types of inhibition. In the present study, we hypothesized neural markers reflecting each type of inhibition may reveal a relationship across inhibitory domains in older adults.

Methods: Electroencephalography (EEG) and behavioral measures were used to explore whether there was an across-participant correlation between sensory suppression and motor inhibition. Sixteen healthy older adult participants (65–80 years) engaged in two separate experimental paradigms: a selective attention, delayed-recognition task and a stop-signal task.

Results: Findings revealed no significant relationship existed between neural markers of sensory suppression (P1 amplitude; N170 latency) and markers of motor inhibition (N2 and P3 amplitude and latency) in older adults.

Conclusions: These distinct inhibitory domains are differentially impacted in normal aging, as evidenced by previous behavioral work and the current neural findings. Thus a generalized inhibitory deficit may not be a common impairment in cognitive aging.

Significance: Given that some theories of cognitive aging suggest age-related failure of inhibitory mechanisms may span different modalities, the present findings contribute to an alternative view where age-related declines within each inhibitory modality are unrelated.

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1. Introduction

Normal aging has been associated with increased difficulty in managing conflicting situations in both the sensory and motor domains (Hasher et al., 1999; Lustig et al., 2007). For example, studies have revealed age-related deficits in restraining a response (Kok, 1999; May and Hasher, 1998) and ignoring/suppressing visually distracting information (Alain and Woods, 1999; Chao and Knight,

1997; Clapp and Gazzaley, 2010; Gazzaley et al., 2005, 2008; West and Alain, 2000; Zanto et al., 2010a,b). More specifically, older adults show diminished response inhibition when required to modify an executed plan of action (Andres et al., 2008; Bedard et al., 2002; Kramer et al., 1994; Potter and Grealy, 2008), as well as visual suppression deficits in the presence of distracting information (Clapp and Gazzaley, 2010; Gazzaley et al., 2008; Hasher et al., 1999).

Both motor inhibition and sensory suppression are believed to be mediated by top-down control processes originating from the prefrontal cortex (Andres et al., 2008; Aron et al., 2003; Gazzaley and D'Esposito, 2007). Given that the prefrontal cortex has been shown to undergo significant age-related structural and functional alterations (Dumitriu et al., 2010; Rajah and D'Esposito, 2005; Raz

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et al., 1997; Tisserand and Jolles, 2003; Tisserand et al., 2004; van Boxtel et al., 2001), it is possible that aging is associated with a general inhibitory deficiency that may be a universal feature of cognitive aging. For example, Hasher and Zacks' (Hasher et al., 1999) inhibitory deficit theory suggests that an age-related decline in inhibitory processing efficiency is reflected by deficits in both the sensory domain (Andres and Van der Linden, 2000; Clapp and Gazzaley, 2010; Gazzaley et al., 2005, 2008; Vallesi and Stuss, 2010; Zanto et al., 2010a,b), as well as in motor inhibition processes (Butler et al., 1999; May and Hasher, 1998; Nielson et al., 2002). The inhibitory deficit hypothesis proposed by Hasher and Zacks (Hasher et al., 1999) and supported by others (Alain and Woods, 1999; Butler and Zacks, 2006; Ossmann and Mulligan, 2003; West and Alain, 2000) suggests that an underlying failure of inhibitory mechanism(s) may account for an age-related decline in cognitive performance across different modalities.

Alternatively, age-related inhibitory deficits could reflect deterioration of independent neural networks supporting different functions (Greenwood, 2000), rather than a decline involving common inhibitory mechanisms. Indeed, evidence of inhibition devolving as a unitary construct or degrading equivalently across individuals has not been supported by behavioral studies. Several age-related studies have reported non-significant correlations among different inhibitory measures including response compatibility, negative priming, stop signal inhibition, spatial precuing, the Wisconsin Card Sorting Test (WCST), a Stroop task, and garden path sentences (Connelly and Hasher, 1993; Davidson and Glisky, 2002; Kramer et al., 1994; Rush et al., 2006; Shilling et al., 2002). These findings suggest that while the operational characteristic of interference suppression and motor inhibition may follow similar mechanisms, these inhibitory processes are independent, even in the presence of age-related cognitive declines. However, Collette et al. (2009) have argued that there are inherent difficulties in isolating inhibitory processes with *behavioral* measures due to the integrative nature of inhibitory functioning, i.e., the act of inhibition engages a series of mechanisms that act in concert with other cognitive processes. Thus, previous reports of non-significant correlates of inhibitory behavioral measures may reflect an insensitivity of these measures in probing this relationship. Here we hypothesize that the use of measures that probe the underlying neural processes associated with these distinct types of inhibition may provide a unique vantage point to better characterize these age-related inhibitory declines.

Gazzaley et al. have repeatedly examined sensory suppression utilizing both functional MRI and electroencephalography (EEG) during the engagement of a selective attention, delayed-recognition task in order to study both top-down enhancement and suppression of visual representations during a memory encoding period (Clapp and Gazzaley, 2010; Clapp et al., 2010; Gazzaley et al., 2005, 2007, 2008; Zanto et al., 2010a,b). These studies in younger adults have repeatedly shown an increase in neural activity for relevant stimuli versus a passive condition, as well as a decrease in activity for irrelevant stimuli versus the same passive condition. These changes in selective attention lead to a modulation of activity at the early stages of visual processing (Kloeber et al., 2005; Lopez et al., 2004; Schoenfeld et al., 2007), as reflected by greater P1 peak amplitudes (~100 ms) and earlier N170 peak latencies (~170 ms), with these event-related potentials (ERPs) localized to the lateral extrastriate cortices (Gomez Gonzalez et al., 1994). With respect to aging, while older adults show intact neural markers of increased cortical activity for task relevant stimuli relative to younger adults (i.e., enhancement), they also display a deficit in the suppression of cortical activity associated with task-irrelevant stimuli (Clapp and Gazzaley, 2010; Gazzaley et al., 2008; Zanto et al., 2010a). Specifically related to the study at hand, the characterization of the age-related effects on sensory suppression

on these ERPs has been reproduced on several occasions (Clapp and Gazzaley, 2010; Gazzaley et al., 2008; Zanto et al., 2010a), supporting the reliability of these neural measures.

Studies of response inhibition have regularly utilized the go/nogo paradigm (Butter, 1969; Passingham, 1972), where participants are instructed to respond (go) or not to respond (nogo) to predefined stimuli. ERPs associated with nogo stimuli have shown two consistent components: a fronto-central negative deflection developing at 200 ms post-stimulus (nogo N2), and a subsequent medial central positivity (nogo P3) (Eimer, 1993; Kok, 1986; Pfefferbaum et al., 1985). While the nogo N2 has been associated with a participant's recognition of their *need* for inhibition (Kok, 1986; Smith et al., 2008), the nogo P3 component has been considered a more precise indicator of the *effectiveness* of motor inhibition engaged (Falkenstein et al., 1995; Pfefferbaum et al., 1985; Smith et al., 2008; Vallesi et al., 2009). Age-related ERP studies of the go/nogo task have consistently demonstrated delayed peak latencies for both the N2 and P3 components in older adults (Fallgatter et al., 1999; Horvath et al., 2009; Pfefferbaum and Ford, 1988; Tachibana et al., 1996); however, it has been suggested that this slowing may reflect a general slowing with age versus a selective age-related slowing of inhibition processes (Falkenstein et al., 2002; Vallesi et al., 2009). Vallesi et al. (2009) have recently suggested these effects may reflect older adults strategically using more time to process conflicting stimuli; thus, a purer measure of motor inhibition may be revealed by controlling for strategies at an individual level.

Better control of strategies can be achieved using a stop signal task (Logan et al., 1984) with a dynamically changing "stop" stimuli based on a participant's inhibition accuracy (cf. Rubia et al., 2003). This task has a greater inherent inhibitory load than a go/nogo task in that participants attempt to inhibit an already triggered movement rather than withholding a prepotent response. In addition, the use of a staircase algorithm to ensure participants inhibit on 50% of trials (thus failing on the other 50%) ensures that each individual performs at the limit of their respective inhibitory capacity, without being able to resort to a 'slowing' strategy. Similar to the nogo P3, the stop signal P3 has also consistently been interpreted as the more precise indicator of the effectiveness of inhibition compared to the N2 (Beste et al., 2010; Kok et al., 2004; Ramautar et al., 2004). Each component in this N2/P3 complex has been associated with inhibition; however, the inhibitory role of the P3 has been strengthened by studies showing that its source is in or near the primary motor or premotor cortices (Kok et al., 2004; Ramautar et al., 2004). However, to date there has not been an EEG study of the stop signal task in older adults that characterizes how these components change with age.

In the present study, we assessed established neural markers of motor inhibition (N2 and P3 amplitude and latency) and sensory suppression (P1, N170) using a stop signal task and a selective delayed-recognition task, respectively, in the same cohort of older individuals. The goal was to evaluate these measures with an across-participant regression analysis to determine if there was a relationship between the impact of aging on motor inhibition and sensory suppression at the neural level. We hypothesized that those older individuals who showed greater markers of inhibition of motor responses would also show greater sensory suppression abilities. This would suggest a common system of inhibitory control equivalently impacted by aging. Alternatively, age-related declines in the underlying networks mediating each type of inhibition type may not degrade in an equivalent fashion, leading to a non-significant relationship between these factors. This distinction would inform previous behavioral reports that described the construct of inhibition as a distinct set of processes depending on modality (Andres et al., 2008; Collette et al., 2009; Kramer et al., 1994).

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