Spatial Attention and Response Control in Healthy Younger and Older Adults and Individuals With Alzheimer's Disease: Evidence for Disproportionate Selection Impairments in the Simon Task

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The authors examined the degree to which aging and Alzheimer's disease (AD) influence the ability to control attention when conflict is presented in terms of incongruent mapping between a stimulus and the appropriate response. In a variant of the Simon task, healthy older adults and older adults with mild or very mild AD showed disproportionately larger reaction time (RT) costs when the stimulus and response were in conflict relative to RT costs of healthy younger adults. Analyses of RT distributions provide support for a 2-process model of the Simon effect in which there is a short-lived transient effect of the irrelevant dimension in younger adults and a more sustained influence across the RT distribution in older adults. An analysis of error rates showed that the older adults with mild and very mild AD made more errors on incongruent trials, suggesting that AD leads to increased likelihood of selecting the prepotent pathway. The findings are discussed in terms of the special nature of the response requirements of the Simon task to better illuminate the attentional decrements in both healthy aging and early stage AD.

Keywords: aging, Alzheimer's disease, attention, cognitive control

The ability to control attention relies on fundamental cognitive operations that contribute to how information is processed and later remembered. Although memory impairments are often thought to be the hallmark of the possible onset of Alzheimer's disease (AD), there is accumulating evidence that attentional processes are impaired relatively early on in the progression of pathological cognitive aging associated with AD (see Balota & Faust, 2001, and Perry & Hodges, 1999, for reviews). In order to develop a better understanding of how AD influences cognitive functioning, it is especially important to examine tasks that involve both automatic and controlled attentional processes that eventually support higher order cognitive operations. In the present study we focused on how aging and AD influence the ability to appropriately control attention and subsequent response selection, in order to gain further insight into the cognitive changes that occur in old age and to examine possible delineations between normal aging and the onset of dementia.

There is abundant evidence that healthy older adults show various degrees of impairment on a wide range of cognitive tasks (Balota, Dolan, & Duchek, 2000). Performance on tasks that involve executive processes and frontal lobe function are thought to be especially impaired, leading to other attentional and memory deficits (West, 1996). Several prominent theories have been proposed to describe the changes in cognitive function in old age, centering on reductions in available processing resources (Craik, 1982, 2002), general slowing of processing (Myerson, Hale, Wagstaff, Poon, & Smith, 1990; Salthouse, 1996), and reductions in inhibitory control (Hasher & Zacks, 1988; Rabbitt, 1965). Although these frameworks typically apply to healthy aging, the notion that a breakdown in the ability to control partially activated but incorrect information has also been quite useful in terms of accounting for some of the cognitive deficits that are associated with AD (e.g. Balota & Ferraro, 1993, 1996; Dempster, 1992; Perry & Hodges, 1999; Spieler, Balota, & Faust, 1996).

A breakdown in attentional control systems could produce a variety of impairments, such as maintaining the task goals, activation of task-relevant information, and the ability to control highly active but inappropriate responses. The Stroop task (see MacLeod, 1991, for a comprehensive review) provides a useful

measure of attentional control, in which the Stroop effect refers to an increase in response latency to name the ink color of a presented color word when the word is an incompatible color name (e.g., the word *BLUE* printed in red), relative to an unrelated word. Spieler et al. (1996) found disproportionate interference effects in healthy older adults relative to younger adults in the Stroop task. In addition, older adults with AD showed a disproportionate increase in intrusion errors (naming the word, instead of the color, on incongruent color-naming trials). Thus, these results suggest that healthy older adults use additional time to keep under control the prepotent word response (thereby producing greater interference effects in response latencies), whereas AD individuals are more likely to produce an error and actually output the inappropriate response (see also Fisher, Freed, & Corkin, 1990; Koss, Ober, Delis, & Friedland, 1984).

Although the finding of larger Stroop interference in older adults (in absolute terms) has been documented as "almost universal" (West, 1996, p. 287) and is consistent with many theories of cognitive aging, the underlying mechanism of greater interference in the Stroop task for older adults has been widely debated. In a meta-analysis of aging and the Stroop effect, Verhaeghen and De Meersman (1998) found no significant difference between the effect size for younger and older adults, after taking into account overall response latencies, and concluded that age differences in Stroop interference can be largely attributed to general slowing as opposed to a reduction in inhibitory control in old age (also see Verhaeghen, 2000). Basak and Verhaeghen (2003) used a modified Stroop *subitizing* (enumerating digits) task, which required participants to report the number of digits in a display, in which the digits either were equal to the size of the set displayed or differed by 1 from the set size. By controlling the size of the focus of attention, the authors found that once individual differences in processing speed were accounted for, there were no age differences in susceptibility to this type of the subitizing Stroop effect. These results suggest that old age may lead to reductions in the size of the focus of attention but that speed of access to elements within the focus of attention may remain constant.

Given that the Stroop task involves multiple processes tied to lexical/semantic access of the word dimension, the strength of the perception of the color dimension, the maintenance of control signals across trials, and the resolution of conflict, this task may not be ideally suited for localizing age-related changes in control systems. For example, it is possible that the yellowing of the lens in older adulthood (Hood, Garner, & Truscott, 1999) could decrease the strength of the color dimension, thereby producing greater Stroop effects in some older adults. Thus, it is useful to examine attentional control processes in older adults by examining a task that involves the control of more general or primitive sets of stimuli–response (S-R) mappings.

The present experiments examine attentional control systems when conflict is presented in terms of incongruent mapping between a stimulus and the appropriate response. The compatibility of stimuli and responses produces clear effects on a variety of performance measures (Fitts & Seeger, 1953), and breakdowns in S-R compatibility can shed light on how attentional control functions. The Simon task has been used to measure visuospatial attention and inhibitory control, especially in terms of control over simple S-R mapping (see Lu & Proctor, 1995, and Simon, 1990, for reviews). In the present version of the Simon task, participants were required to make a button press response based on the identity of a stimulus (i.e., respond with a left or right keypress to target colors or symbols). Specifically, participants were instructed to press the left button when a left-pointing arrow appeared and to press the right button when a right-pointing arrow appeared. The critical observations from this task occur when a left arrow appears to the right of fixation or a right arrow appears to the left of fixation, creating interference between the stimulus and the appropriate response. When the location of the stimulus (relative to fixation) conflicts with the required response (e.g., a left arrow appears to the right of fixation), individuals are slower to respond compared with when the location and the stimulus match (e.g., a left arrow on the left side). In the conflict condition, the more primitive spatial location of the stimulus to response mapping needs to be overcome in order for an individual to respond to the directionality of the arrow. The Simon effect is typically expressed as the reaction time (RT) cost of responding to an incongruent trial relative to a congruent trial.

Although initial accounts of the Simon effect were similar to that of the Stroop effect (Simon, 1969; Simon & Berbaum, 1990), more recent evidence suggests that the locus of the effect is primarily at the response selection and execution stage (Ansorge & Wühr, 2004; Lu & Proctor, 1995). In support of this, there is evidence from the lateralized readiness potential indicating that there is indeed contralateral activation in primary motor cortex during the Simon task, suggesting that the onset of a stimulus at a specific location results in the partial activation of an associated (or prepotent) automatic spatial motor code that needs to be controlled in order for an individual to select the correct response (Umilta, 1995). Although some studies of the Simon effect use stimuli that require learned associative responses (e.g., the color of stimuli indicates what response is required) and others use arrows (e.g., participants must respond to the direction of the arrow while ignoring spatial location— somewhat akin to Stroop experiments), both types of studies lead to similar results. Thus, it appears that the Simon effect results mainly from conflict at a relatively late stage of processing, such as the mapping between the stimulus and the appropriate response codes (Ansorge & Wühr, 2004). Previous research that has examined healthy aging, interference, and response control in variants of the Simon task has found significantly larger costs in response latencies on incongruent trials (i.e., a magnified Simon effect) for healthy older adults relative to younger adults (Bialystok, Craik, Klein, & Viswanathan, 2002; Proctor, Pick, Vu, & Anderson, 2005; Van der Lubbe & Verleger, 2002), which is consistent with impaired inhibitory control in old age, although there are typically no differences in error rates between younger and older adults.

As noted, the principal mechanism hypothesized to be involved in the Simon effect is the activation of a response code corresponding to the general spatial location of the stimulus. This automatic response activation has been reported to decay over time (Hommel, 1994), such that when participants have to delay their responses, the Simon effect is reduced (Simon, Acosta, Mewaldt, & Speidel, 1976). The effect has also been found to decay with responses that have longer RTs (De Jong, Liang, & Lauber, 1994). Recent work by Wiegand and Wascher (2005) has shown that the Simon effect can be partitioned into two mechanisms: a fast, transient effect and a slower, sustained component. By using

Simon displays that consisted of both vertical and horizontal spatial orientations of response locations, coupled with the standard horizontal configuration of the response keys, Wiegand and Wascher found that the Simon effect was reduced at longer RTs in the horizontal condition but not in the vertical condition. These findings were interpreted as evidence that for more complex S-R mapping (such as in the vertical condition), the Simon effect is maintained even at longer RTs because participants must rely on controlled processing. Also, Wiegand and Wascher found evidence from lateralized readiness potential-difference waves that indicated a fast and transient influence of the horizontal but a slow and sustained influence of the vertical spatial stimulus feature. In essence, the model described by Wiegand and Wascher suggests that slower, more controlled processing will lead to reductions in the Simon effect due to the reliance on controlled processing in standard S-R mappings but that when these mappings are more complex, the magnitude of the Simon effect will remain stable across the range of RTs.

In the present study, we were interested in whether younger adults would show a reduced Simon effect at longer RTs, and we predicted that older adults would show a constant Simon effect across all RTs. We reasoned that older adults might show a constant Simon effect across the time course (after accounting for overall slowing) because irrelevant response code information remains active for a longer period of time; this prevents the use of controlled processing, owing possibly to the sustained activation of competing pathways.

Although previous research has shown larger Simon effects for healthy older adults relative to younger adults (Bialystok et al., 2002; Proctor et al., 2005; Van der Lubbe & Verleger, 2002), a pattern consistent with impaired attentional control in old age, no study that we are aware of has tested whether the larger Simon effect in older adults holds after a z-score transformation taking into account age-related general slowing (although researchers have used proportional measures; see Faust, Balota, Spieler, & Ferraro, 1999, for a discussion of the relative merits of a z-score transformation vs. a proportional analysis).

In the present study, as noted above, we used stimuli (arrows) that inherently contained response-related information, as opposed to experimentally induced learned associations (such as color) that indicate which response is required. This was done to examine how response conflict influences attentional control when the spatial codes of the stimuli are pre-experimentally salient and highly related to the response options. Moreover, no study we are aware of has examined how individuals with AD perform on the Simon task, and observations from this task might provide useful information regarding the delineation between healthy aging and AD. If the Simon task produces results similar to results of the Stroop task, we would expect an increase in intrusion errors—that is, responses based on the location instead of the direction of the arrow—in early stage AD. Furthermore, consistent with the Wiegand and Wascher (2005) study, we also investigated how the Simon effect changes across different regions of the RT distribution for younger, older, and AD individuals, as this pattern provides some evidence regarding the decay of irrelevant response code information and the use of controlled processing across time.

Experiment 1A

The goal of Experiment 1 was to examine the Simon effect in a large sample of healthy younger and older adults to determine whether (as suggested by previous work) older adults display greater interference on incongruent trials. Given the widespread general slowing in older adults and the concomitant increase in absolute effect sizes, we incorporated the use of z-score transformations for the RT data (e.g., Faust et al., 1999) to correct for possible scaling differences.

Method

Participants. One hundred thirty-seven younger adults ($M = 19.5$ years, $SD = 1.1$) were recruited from undergraduate courses at Washington University and received course credit for participating. One hundred twenty-two healthy older adults were recruited from the Washington University Aging and Development Research Volunteer Pool and received $10 for participating.

Apparatus and materials. The experiment was run on a Pentium II IBM-compatible computer with a standard 15-in. monitor; it was implemented using E-prime software (Schneider, Eschman, & Zuccolotto, 2001). Participants viewed the display from an approximate distance of 50 cm. The display consisted of a central fixation cross on the screen (1 cm $\times$ 1 cm) and arrow stimuli (measuring approximately 4 cm in length and 2 cm in height). The peripheral locations of the arrow (left and right) were situated 5° on the horizontal plane from the central fixation area. The central fixation cross and arrows were presented in white on a black background.

Procedure. The Simon task was embedded in a set of other tasks investigating memory performance. At the beginning of the Simon task, participants were given verbal instructions regarding the nature of the task. They were told that they would be presented with an arrow pointing to either the left or the right on the computer screen. The arrow could appear on the left half, right half, or center of the screen. Participants were told to ignore the location of the arrow and simply respond according to the direction of the arrow by pressing a key on either the left or the right side of the keyboard that corresponded to the direction of the arrow. Examples of the three different trial types are shown in Figure 1.

A practice session was first administered that consisted of 12 trials; participants were given accuracy feedback after each trial. Each trial began with a central fixation cross, which stayed on the screen for 500 ms, followed by the onset of the arrow, which stayed on the screen either until the participant made a response or until 5 s had elapsed. Once a response was made, the screen cleared and the participants received feedback for 400 ms as to whether they had made a correct or an incorrect response. After feedback, the next trial began 2 s later. In total, there were 12 practice trials (4 congruent, 4 incongruent, and 4 fixation) and 120 experimental trials (40 congruent, 40 incongruent, and 40 fixation). We included the 40 "neutral" *fixation* trials primarily to ensure that participants would keep fixated at the center of the screen. Of course, these trials should have produced the fastest response latencies because participants were already at fixation. The different trial types were randomly intermixed within each session for each participant, and the entire task took approximately 20 min.
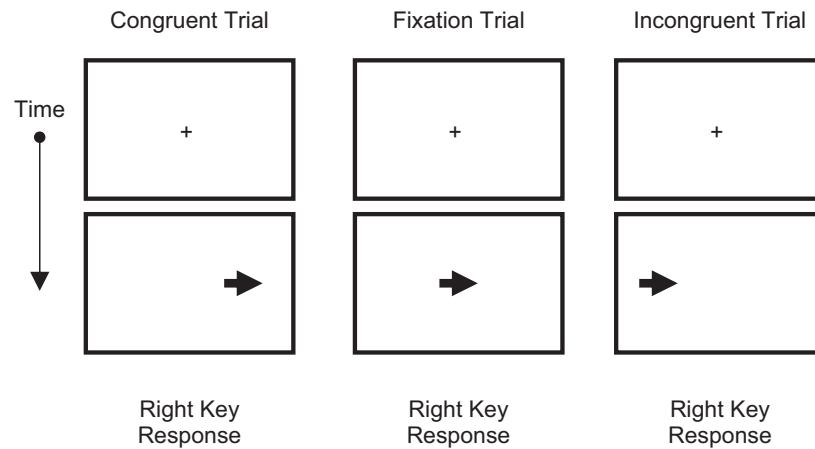
Figure 1. The sequence of events for a given congruent, fixation, or incongruent trial.

Results

The results from the Simon task for younger and older adults can be viewed in several ways. Consistent with prior research on the Simon task, the primary dependent measure is the RT for the arrow detection in the three different trial types (congruent, incongruent, and fixation), and the Simon effect is the difference between the incongruent and congruent trials. Also, in order to take into account general slowing of older adults relative to the younger adults, we transformed the RTs into standardized z scores; conclusions regarding the magnitude of the Simon effect can be made on the basis of these measures in light of overall slowing (see Faust et al., 1999). Finally, we explored whether there were age differences in error rates among healthy younger and older adults in the Simon task. Error rates do not typically differ between healthy younger and older adults in the Stroop task, but they are disproportionately higher in individuals with AD as compared with healthy older adults (Spieler et al., 1996).

RT analysis. The RTs for younger and older adults for correct responses for the three different trial types are presented in Table 1. RTs that were greater or less than 2.5 standard deviations from the mean for each participant were removed, and this resulted in the exclusion of less than 2.4% of RTs for each group. To examine differences in RTs for younger and older adults, we conducted a 2 (group) $\times$ 3(trial type) mixed-model analysis of variance (ANOVA). There was a significant main effect of group $F(1, 257) = 243.17$, $MSE = 55,594.37$, $\eta^2 = .47$, $p < .001$, indicating that the two age groups differed in overall RTs ($Ms = 458$ ms and 722 ms for the younger and older adults, respectively). There was also a main effect of trial type, $F(2, 514) = 444.83$, $MSE = 1,230.07$, $\eta^2 = .63$, $p < .001$. Congruent and fixation trials were significantly faster than incongruent trials ($p < .001$); fixation trials were not reliably faster than congruent trials ($p > .10$). As noted, the fixation trials were actually catch trials to make sure that participants were attending to the central fixation point, and so it is not surprising that these items produce relatively fast response latencies. Of note, there was a highly reliable Group $\times$ Trial Type interaction, $F(2, 514) = 52.43$, $MSE = 1,230.07$, $\eta^2 = .17$, $p < .001$.

To further examine the nature of the interaction, we calculated the Simon effect, typically defined as the difference between incongruent and congruent trials (Simon, 1990), for each group;

Table 1

Performance Measures From the Simon Task in Experiment 1A

Measure	Younger ($n = 137$)		Older ($n = 122$)	
	M	SD	M	SD
Response latencies (milliseconds)				
Fixation	434.07	74.68	689.69	174.27
Incongruent	493.42	79.02	792.71	203.80
Congruent	445.91	70.44	683.77	180.38
Simon effect	47.51	30.60	108.94	78.44
Response latencies (z scores)				
Fixation	−.33	.13	−.25	.11
Incongruent	.26	.18	.25	.22
Congruent	−.18	.15	−.28	.15
Simon effect	.44	.27	.53	.33
Error rates				
Fixation	.01	.02	.01	.03
Incongruent	.04	.05	.04	.08
Congruent	.01	.02	.01	.05

these values are shown in Table 1. These data were entered into a one-way ANOVA, which revealed a significant effect of group, $F(1, 257) = 71.79$, $MSE = 3,392.69$, $\eta^2 = .22$, $p < .001$, such that the Simon effect was significantly larger in older adults than in younger adults.

z-score transformation of RTs. To account for group differences in overall RT, we transformed mean RTs to z scores and repeated the analyses that were carried out on the original RT data. The transformed standardized z scores are presented in Table 1. There was no significant main effect of group, $F(1, 257) = 1.18$, $MSE = 0.002$, $\eta^2 = .01$, $p > .20$. As in the raw RTs, there was a significant main effect of trial type, $F(2, 514) = 641.98$, $MSE = 0.036$, $\eta^2 = .71$, $p < .001$, with fixation trials ($M = -.29$) being faster than both congruent ($M = -.23$) and incongruent trials ($M = .25$), $p < .001$. Mirroring the RT data, there was a significant Group × Trial Type interaction, $F(2, 514) = 11.84$, $MSE = 0.036$, $\eta^2 = .05$, $p < .001$.

To further investigate the nature of the Group × Trial Type interaction and to assess the interference effect, we calculated the Simon effect by subtracting incongruent trial z scores from congruent trial z scores for younger and older adults (see Table 1). A one-way ANOVA showed a main effect of group, $F(1, 257) = 5.54$, $MSE = 0.09$, $\eta^2 = .02$, $p < .02$, indicating that as in raw RTs, the Simon effect in z scores was again significantly larger in older adults than in younger adults.

Bin analyses of RTs. To examine the time course of activation in the Simon task, one can compute RT distribution analyses in order to view the Simon effect as a function of fastest to slowest RTs (see Proctor et al., 2005, for a similar procedure). As noted earlier, Wiegand and Wascher (2005) recently argued that two mechanisms likely contribute to the Simon effect: a fast, transient effect and a slower, sustained component. One way to examine these two components is to examine the RTs as a function of the temporal response pattern. Hence, we computed quartiles for each participant by obtaining RT measures from the 20th, 40th, 60th, and 80th percentile for each condition, and then the Simon effect was calculated at each of the four bins (or percentiles).

The mean Simon effect for each group at each bin percentile is plotted in Figure 2. The data in Figure 2 show that the Simon effect

Figure 2. Reaction time bin analysis for Experiments 1A and 1B.

for younger adults tended to decline as RTs became slower, whereas older adults showed an increasing Simon effect across bins. The data were entered into a 2 (group) × 4 (bin) repeated measures ANOVA, which revealed a significant main effect of group, $F(1, 257) = 77.35$, $MSE = 14,307.78$, $\eta^2 = .23$, $p < .0001$, but the effect of bin did not reach significance, $F(3, 771) = 2.05$, $MSE = 2,042.15$, $\eta^2 = .01$, $p = .11$. Of note, there was a highly reliable Bin × Group interaction, $F(3, 771) = 10.39$, $MSE = 2,042.15$, $\eta^2 = .04$, $p < .0001$. The presence of the interaction suggests that whereas younger adults show a decrease in the Simon effect as RTs increase, older adults display an increasingly larger Simon effect in later RTs. Thus, younger adults show a reduced Simon effect at longer RTs (as shown in previous research), suggesting that when controlled processing is present (and irrelevant response codes have decayed over time), the Simon effect is reduced. However, older adults do not show this trend, indicating that the irrelevant response code information still exerts an effect even at the longer RTs. This finding is important in terms of supporting a two-mechanism account of the Simon effect (Wiegand & Wascher, 2005) and suggests that older adults have difficulty selecting and controlling response pathways at longer RTs relative to younger adults. The two-stage model posits that the Simon effect is related to both an initial fast, transient effect and a slower, controlled component, and it appears that older adults do not (or cannot) utilize the slower, controlled process to reduce the Simon effect at longer RTs.

Error rates. The mean percentages of errors for each group for the three different trial types are shown in Table 1. Given that errors (especially on incongruent trials) were of critical interest in examining the ability to inhibit prepotent responses, the error rates on each trial type were entered into a mixed-model ANOVA for the younger and older adults. There was no significant main effect of group ($F < 1$). There was a significant main effect of trial type on errors, $F(2, 514) = 65.38$, $MSE = 0.01$, $\eta^2 = .21$, $p < .001$, with more errors on incongruent trials ($M = 4.3$) relative to both congruent ($M = 1.3$) and fixation trials ($M = 1.2$), $p < .001$. There was no significant Group × Trial Type interaction ($F < 1$).

Experiment 1B

One reason for age-related differences in the magnitude of the Simon effect may be that younger adults have better visual discrimination abilities for peripheral stimuli, whereas older adults need to engage in deliberate shifting of attention for these peripheral stimuli. This explanation centers on differences in the shifting and distribution of attention across the lifespan (e.g., Festa-Martino, Ott, & Heindel, 2004; Madden, Connelly, & Pierce, 1994; Tales, Snowden, Haworth, & Wilcock, 2002). To examine this possibility, we conducted an experiment in which younger adults participated in conditions in which the peripheral target was perceptually degraded, as has been done in other studies that have examined the allocation of attention to degraded stimuli (e.g., Castel, Pratt, Chasteen, & Scialfa, 2005). This allowed for the examination of how younger adults perform under more demanding perceptual conditions (possibly akin to older adults under nondegraded conditions) and, more specifically, the determination of whether younger adults, when producing longer RTs owing to the more challenging visual discrimination, would produce a greater Simon effect. Furthermore, in light of the RT distribution
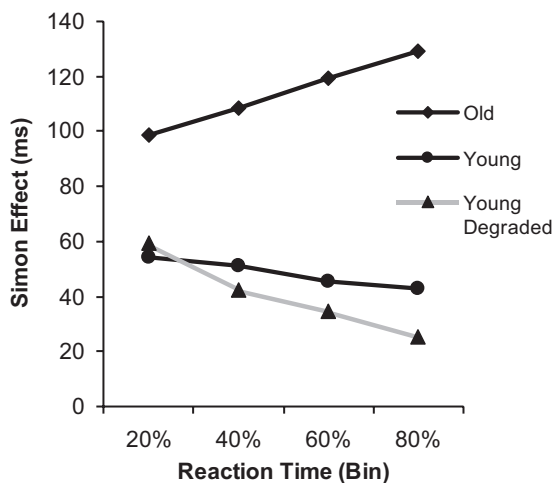
analyses, we were interested in whether younger adults under degraded conditions would show a greater Simon effect at longer RTs, suggesting that the Simon effect is directly related to RT—a point that is important in terms of the interpretation of the magnitude of the Simon effect in younger and older adults. If, however, there is no difference in the Simon effect across the RT bins or a greater Simon effect at fast RTs, then this would suggest that the younger adults' smaller Simon effect (relative to older adults) is likely attributable to differences in later stages of processing that are related to attention and response control.

Method

Participants. Ten younger adults were recruited from undergraduate courses at Washington University and received course credit for participating.

Apparatus, materials, and procedure. The experiment was very similar to Experiment 1A, the only change being that the target was perceptually degraded by reducing the luminance levels of the target. This was done by reducing the contrast between the target and background, in essence making the target harder to perceive and leading to longer RTs. The luminance of the target was reduced to approximately 5 candelas per square meter (cd/m^2), whereas luminance of the target in Experiment 1A was approximately 50 cd/m^2.

Results

The mean RTs for each trial type for the younger adults responding to the degraded target are as follows: congruent = 561 ms (SD = 84.5), incongruent = 600 ms (SD = 75.5), fixation = 491 ms (SD = 80.3); thus, there was a significant mean Simon effect of 39 ms (SD = 19.1), $t(9)$ = 6.45, p < .001, d = 0.49, which is actually slightly smaller than the Simon effect (48 ms) obtained with the younger adults in the nondegraded conditions of Experiment 1. Overall, these participants were about 93 ms slower than the younger adults from Experiment 1A (although it should be noted that this slowing is moderate relative to the 250 ms of slowing that was related to age difference in Experiment 1A). Similar effects were found when conducting these analyses on *z*-score transformation of the RTs: congruent = .034 (SD = .82), incongruent = .33 (SD = .74), fixation = −.56 (SD = .78).

As in Experiment 1A, error rates were very low for all three trial types (congruent = .01, incongruent = .05, fixation = .02), showing that degrading the target led to slower RTs but no differences in errors relative to nondegraded conditions in Experiment 1A. As in Experiment 1A, the RTs were sorted into bins to examine the temporal dynamics of the Simon effect across differing RTs; the mean Simon effects at each bin are presented in Figure 2. When examining these RTs in terms of bins (faster to slower RTs), we again found an interaction between bin and the magnitude of the Simon effect, $F(3, 27)$ = 4.31, MSE = 246.97, η^2 = .32, p = .013, suggesting that even at longer RTs (and with degraded targets), the magnitude of the Simon effect for young adults became smaller as RTs became longer. This pattern is consistent with both Experiment 1A and related findings in the literature.

In summary, the results from the degraded target experiment show that even when younger adults were required to respond

under more demanding visual conditions (which resulted in longer RTs but no change in errors), the Simon effect was still comparable to that found with younger adults in Experiment 1A. This suggests that delays in visual processing time of the peripheral target in the Simon task do not lead to increases in the magnitude of the Simon effect, and this is an important point in terms of interpreting the age-related differences in attentional and response control.

Experiment 2

The focus of the second experiment was to attempt to replicate the age-related changes observed in Experiment 1A and extend this investigation to older adults who have very mild, and mild, AD. We were particularly interested in performance on trials in which conflicting information is present, to determine whether AD leads to an increased likelihood of making response errors (via an impairment in inhibiting prepotent responses) in situations that demand high levels of attention and response control (cf. Stroop study by Spieler et al., 1996). We were also interested in how AD modulates the time course of the Simon effect.

Method

Participants. Participants were recruited from the Washington University Alzheimer's Disease Research Center (ADRC) and consisted of 66 healthy older adults and 50 individuals with early stage AD. The healthy older adults (n = 66) had a mean age of 78.96 years (SD = 8.1; range, 59–83), the individuals with very mild AD (n = 38) had a mean age of 79.9 years (SD = 5.9; range, 55–91), and the individuals with mild AD (n = 12) had a mean age of 84.4 years (SD = 8.7; range, 61–86). There were no significant differences among the groups of older adults in terms of mean age (all ps > .13). In addition, 31 younger adults (age 25 or younger; M = 20.8 years, SD = 1.5) were recruited from the Washington University student community and participated for course credit or were paid $10.

The healthy older adults and the individuals with AD were seen by a physician and completed a battery of psychometric tests approximately once a year, and were screened by a physician for neurological, psychiatric, or medical disorders with the potential to cause dementia. The inclusion and exclusion criteria for a diagnosis of AD have been described in detail elsewhere (e.g., Morris, 1993; Morris, McKeel, Fulling, Torack, & Berg, 1988) and conform to those outlined by the National Institute of Neurological and Communications Disorders and Stroke—Alzheimer's Disease and Related Disorders Association (McKhann et al., 1984). Dementia severity for each individual with AD recruited from the ADRC was staged in accordance with the Washington University Clinical Dementia Rating Scale (Hughes, Berg, Danziger, Coben, & Martin, 1982; Morris, 1993). According to this scale, a score of 0 indicates no cognitive impairment, a score of 0.5 indicates very mild dementia, a score of 1.0 indicates mild dementia, and a score of 2.0 indicates moderate dementia. At the ADRC, a Clinical Dementia Rating Scale score of 0.5 has been found to accurately indicate the earliest stages of AD (Morris et al., 1991). Both the reliability of this scale and the validation of the diagnosis (based on autopsy) by the research team have been excellent (93% diagnostic accuracy) and are well documented (e.g., Berg et al., 1998).

Psychometric test information. In addition to participating in the experimental task, the healthy older adults and those with AD who were recruited from the ADRC completed a 2-hr battery of psychometric tests as part of a larger longitudinal study of cognitive performance in healthy aging and AD. The results from the psychometric tests are displayed in Table 2. Memory performance was assessed with the Wechsler Memory Scale (Wechsler & Stone, 1973) and scored accordingly; Logical Memory (immediate, with no delayed recall; recall of Scoring Units 0–23) and Forward and Backward Digit Span (number of correct digits; 0–8 and 0–7, respectively). General intelligence was assessed with the Information (scoring range, 0–29), Block Design (scoring range, 0–48), and Digit Symbol (scoring range, 0–90) subtests of the Wechsler Adult Intelligence Scale (Wechsler, 1955). Visual perceptual–motor performance was assessed with the Benton Visual Retention Test (number correct) and the Benton Copy Test (number of errors; Benton, 1963) and Part A of the Trail Making Test (seconds to complete; Armitage, 1946). Finally, the Boston Naming Test (Goodglass & Kaplan, 1983) was administered as a test of semantic/lexical retrieval (number correct out of 60). These psychometric tests are scored such that greater scores indicate better performance with the exception of Trail Making A and Benton copy errors, for which higher scores indicate poorer performance. Psychometric testing always occurred within a 2-month window of the Simon task testing session. As shown in Table 3, as expected, the AD groups performed more poorly than the healthy older group on most tests. Because the younger adults were not recruited by the ADRC, they did not receive the psychometric battery.

Apparatus, materials, and procedure. The method, materials, and procedure of Experiment 2 were identical to those of Experiment 1.

Results

RT analysis. The mean RTs for the four groups for correct responses in the three different trial types are presented in Table 3. RTs that were greater or less than 2.5 standard deviations from the mean for each participant were removed, and this resulted in the exclusion of less than 3% of RTs for each group. The results of a 4 (group) $\times$ 3 (trial type) mixed-model ANOVA yielded a main effect of group, $F(3, 143) = 28.65$, $MSE = 76,622.88$, $\eta^2 = .38$,

Table 3

Means and Standard Deviations of Reaction Times (RTs) for Correct Responses to Fixation, Congruent, and Incongruent Trial Types by Group

| | Trial type | | |
Group and measure	Fixation	Congruent	Incongruent
Younger adults (*n* = 31)			
Mean RT (milliseconds)	484	498	533
SD	131	118	126
Older adults (*n* = 66)			
Mean RT (milliseconds)	701	683	779
SD	132	145	156
Very mild AD (*n* = 38)			
Mean RT (milliseconds)	781	746	892
SD	160	152	198
Mild AD (*n* = 12)			
Mean RT (milliseconds)	841	837	1,087
SD	226	244	474

Note. AD = Alzheimer's disease.

$p < .0001$, indicating that the groups differed in overall RTs ($M = 505, 722, 806,$ and 922 ms for the younger, older, very mild AD, and mild AD groups, respectively). A Tukey post hoc procedure showed that the younger adults were significantly faster than the other groups and that the mild AD group was slower than the three other groups ($p < .05$), whereas there was no significant overall difference between the healthy older and very mild AD groups. There was also a significant main effect of trial type, $F(2, 286) = 103.87$, $MSE = 5,238.04$, $\eta^2 = .42$, $p < .0001$, with congruent ($M = 691$ ms) and fixation ($M = 702$ ms) trial types being faster than incongruent trials ($M = 823$ ms), $p < .05$. Most notably, there was again a highly reliable Group $\times$ Trial Type interaction, $F(6, 286) = 9.37$, $MSE = 5,238.04$, $\eta^2 = .16$, $p < .0001$, likely driven by the slow RTs of the AD group for the incongruent trials. To further examine the nature of the interaction, we calculated the Simon effects (incongruent minus congruent trials) for each group; these values are shown in Figure 3A. These data were entered into a one-way ANOVA, which revealed a significant effect of group, $F(3, 143) = 11.29$, $MSE = 13,900.73$, $\eta^2 = .19$, $p < .0001$. Post

Table 2

Means (and Standard Deviations) of Scores on Psychometric Tests for Healthy Older Adults and Individuals With AD

Psychometric test	Healthy older adults	Very mild AD	Mild AD[a]	F(2, 110)	p
WMS Logical Memory	10.78 (3.41)	7.39 (4.07)	3.90 (2.10)	24.81	<.0001
WMS Digits Forward	6.55 (1.47)	6.41 (1.23)	6.57 (1.22)	0.586	=.559
WMS Digits Backward	5.02 (1.27)	4.36 (1.33)	4.00 (1.22)	4.31	<.05
Trail Making Form A (total seconds)	39.86 (21.64)	50.58 (26.07)	78.00 (40.50)	15.41	<.0001
Trail Making Form B (total seconds)	90.52 (37.78)	128.63 (41.91)	150.60 (40.33)	18.63	<.0001
Boston Naming Test	55.35 (5.61)	48.97 (8.65)	41.00 (10.65)	19.10	<.0001
Benton Copy Form D	9.77 (0.63)	9.69 (0.75)	8.66 (1.07)	10.40	<.0001
WAIS–R Block Design	31.93 (8.23)	23.36 (11.62)	20.02 (11.66)	16.79	<.0001
WAIS–R Information	21.52 (4.04)	18.36 (5.72)	13.20 (4.96)	17.62	<.0001
WAIS–R Digit Symbol	46.51 (11.65)	36.11 (12.69)	24.20 (12.77)	17.40	<.0001

Note. F and p values reflect one-way analyses of variance. AD = Alzheimer's disease; WMS = Wechsler Memory Scale; WAIS–R = Wechsler Adult Intelligence Scale—Revised.

[a] Complete psychometric data were available from only 9 of the 12 mild AD participants.
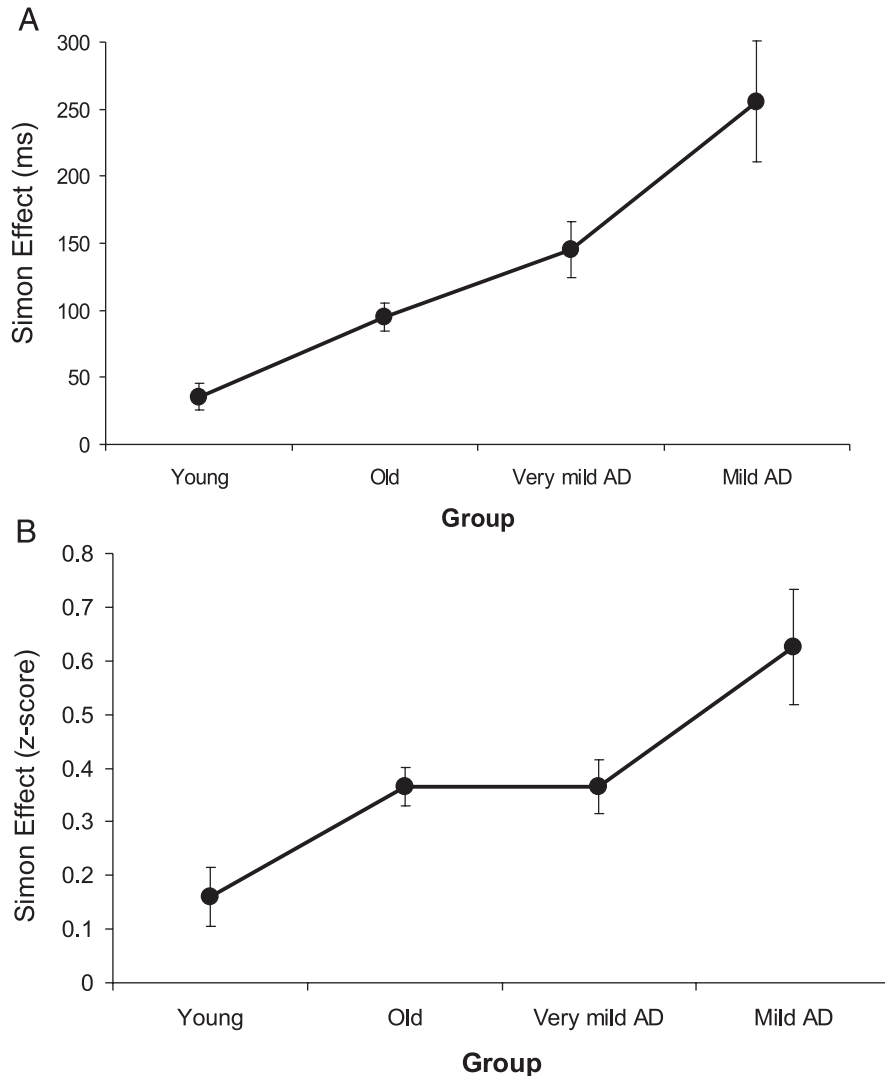
Figure 3. A: Mean Simon effect expressed as the difference in reaction times between congruent and incongruent trials for the four groups in Experiment 2. Error bars represent standard error of the mean. B: Mean Simon effect expressed as the difference in *z* scores between congruent and incongruent trials for the four groups in Experiment 2. Error bars represent standard error of the mean. AD = Alzheimer's disease.

hoc tests showed that the younger adults' Simon effect was significantly smaller relative to the three other groups ($p < .05$), whereas the older adults and very mild AD group did not differ from one another ($p > .15$) and the mild AD group was significantly greater than the four other groups ($p < .05$).

z-score transformation of RTs. The *z* scores are presented in Table 4. The ANOVA revealed that there was a significant main effect of group, $F(3, 143) = 5.46$, $MSE = 0.008$, $\eta^2 = .10$, $p < .01$, indicating differences in the overall pattern of *z* scores (*M*s = −.0329, −.0293, −.0175, and .0321 for the younger, older, very mild AD, and mild AD groups, respectively). A Tukey post hoc procedure showed that the mild AD group differed from the other three groups ($p < .05$), and no other differences reached conventional levels of significance ($p > .60$). As in the raw RTs, there was also a significant main effect of trial type, $F(2, 286) = 131.98$, $MSE = 0.041$, $\eta^2 = .48$, $p < .0001$, with

congruent ($M = −.152$) and fixation ($M = −.122$) trial types being faster than incongruent trials ($M = .202$), $p < .05$. Of note, as was found in the RT data, there was a significant Group × Trial Type interaction, $F(6, 286) = 8.93$, $MSE = 0.041$, $\eta^2 = .16$, $p < .0001$. To further investigate the nature of this interaction and to assess the interference effects, we calculated the Simon effect by subtracting incongruent trial *z* scores from congruent trial *z* scores. This measure of the Simon effect is shown in Figure 3B. A one-way ANOVA showed a main effect of group, $F(3, 146) = 9.26$, $MSE = 0.10$, $\eta^2 = .16$, $p < .0001$, and post hoc tests showed that the younger adults differed from the other three groups ($p < .05$), thereby replicating the results from Experiment 1. However, the older adults and very mild AD group did not differ from one another ($p > .80$), and the effect in the mild AD group was significantly greater than the three other groups ($p < .05$).

Table 4

Mean z-Score Transformations and Standard Deviations of Reaction Times (RTs) for Correct Responses to Fixation, Congruent, and Incongruent Trial Types by Group

Group and measure	Trial type		
	Fixation	Congruent	Incongruent
Younger adults (*n* = 31)			
Mean RT (milliseconds)	−.219	−.019	.140
SD	.196	.179	.153
Older adults (*n* = 66)			
Mean RT (milliseconds)	−.103	−.183	.199
SD	.153	.182	.148
Very mild AD (*n* = 38)			
Mean RT (milliseconds)	−.059	−.206	.187
SD	.134	.181	.148
Mild AD (*n* = 12)			
Mean RT (milliseconds)	−.166	−.231	.493
SD	.151	.188	.388

Note. AD = Alzheimer's disease.

Bin analyses of RTs. To examine the time course of activation in the Simon task (as done in Experiment 1), we conducted RT bin distribution analyses in order to view the Simon effect as a function of fastest to slowest RTs. Figure 4 shows that the Simon effect for younger adults again tends to decrease across bins (replicating the results of Experiment 1), whereas both older adults and the very mild AD group show a larger Simon effect for slower bins. The mild AD group showed an initial increase in the Simon effect, but the effect then decreased at the slowest bin. As described below, this decrease at the slower bins may be related to the slightly higher error rate in the mild AD group. Of interest, a similar pattern was found in the Spieler et al. (1996) Stroop study. This decrease in the Simon effect at longer RTs likely reflects some reliance on more controlled processing for the correct trials, which results in a smaller Simon effect, whereas other trials result in errors due to selecting the prepotent but inappropriate response.

To examine differences between the groups, we entered the data into a 4 (group) × 4 (bin) repeated measures ANOVA, which revealed a significant main effect of group, $F(3, 140) = 9.82$, $MSE = 63,144.42$, $\eta^2 = .17$, $p < .0001$, but the effect of bin did not reach significance, $F(3, 420) = 1.30$, $MSE = 5,363.81$, $\eta^2 = .01$, $p = .27$. More important, the Bin × Group interaction was significant, $F(9, 420) = 2.41$, $MSE = 5,363.81$, $\eta^2 = .05$, $p = .01$. The trends are similar to those found in Experiment 1, with younger adults showing a decrease in the Simon effect with increasing RT bins, whereas older adults and especially the very mild AD group showed greater Simon effects (for the most part) at longer RT bins. To compare these data with the findings from Experiment 1, we conducted an ANOVA with just the younger and healthy older adults, which yielded a main effect of group, $F(1, 95) = 16.29$, $MSE = 22,683.50$, $\eta^2 = .15$, $p < .0001$, but no significant effect of bin ($F < 1$). More important, replicating Experiment 1a, we again found a significant interaction, $F(3, 285) = 3.20$, $MSE = 1,887.79$, $\eta^2 = .033$, $p < .05$, suggesting different Simon effects for younger and older adults across the bins.

Error rates. The mean percentages of errors for each group for the three different trial types are shown in Figure 5. Given that

errors (especially on incongruent trials) were of interest in order to examine the ability to control prepotent responses, the error rates on each trial type were entered into a mixed-model ANOVA for the four groups. There was a significant main effect of group $F(3, 143) = 8.48$, $MSE = 0.014$, $\eta^2 = .15$, $p < .0001$, indicating differences in the overall error rates (*M*s = 1.1%, 1.7%, 6.9%, and 9.2% for the young, older, very mild AD, and mild AD groups, respectively). A Tukey post hoc procedure showed that the younger adults did not differ from the older group but that these two groups did differ significantly from the two AD groups ($p < .05$), whereas there was no significant difference between the two AD groups. There was a significant main effect of trial type on errors, $F(2, 286) = 34.62$, $MSE = 0.002$, $\eta^2 = .20$, $p < .0001$, with more errors on incongruent trials (*M* = 6.8) relative to both congruent (*M* = 2.4) and fixation (*M* = 3.1) trials ($p < .05$). Consistent with theoretical expectations, there was a significant Group × Trial Type interaction, $F(6, 286) = 4.54$, $MSE = 0.002$, $\eta^2 = .09$, $p < .001$, that reflected the relatively high error rates for both AD groups on incongruent trials. To further investigate this interaction, we subtracted error rates for incongruent trials from error rates for congruent trials and subjected them to a one-way ANOVA for the four groups. There was a main effect of group, $F(3, 146) = 5.48$, $MSE = 0.006$, $\eta^2 = .10$, $p = .001$, with both AD groups producing significantly higher error scores relative to the healthy younger and older adults ($p < .05$).

The RTs for trials in which an error occurred were compared with correct trial RTs in order to determine whether error RTs are relatively faster than the average overall RTs (possibly suggesting an inability to inhibit a prepotent response). However, owing to the fairly low error rates for most groups and the large amount of variability in error RTs for the AD groups, standard ANOVAs were not carried out (as many participants made no errors on certain trial types). Moreover, individual *t* tests indicated that there were no significant differences between correct RTs and error RTs for each group ($ps < .25$).

Intraindividual variability analysis. One way to examine differences in response control (for each participant) is to examine intraindividual variability. Although it is clear that variability
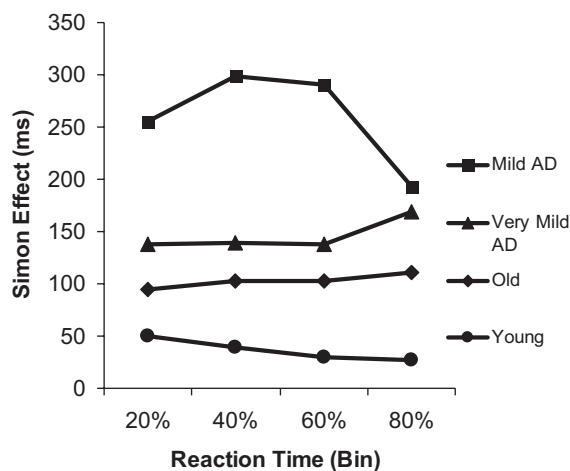

Figure 4. Reaction time bin analysis for Experiment 2. AD = Alzheimer's disease.
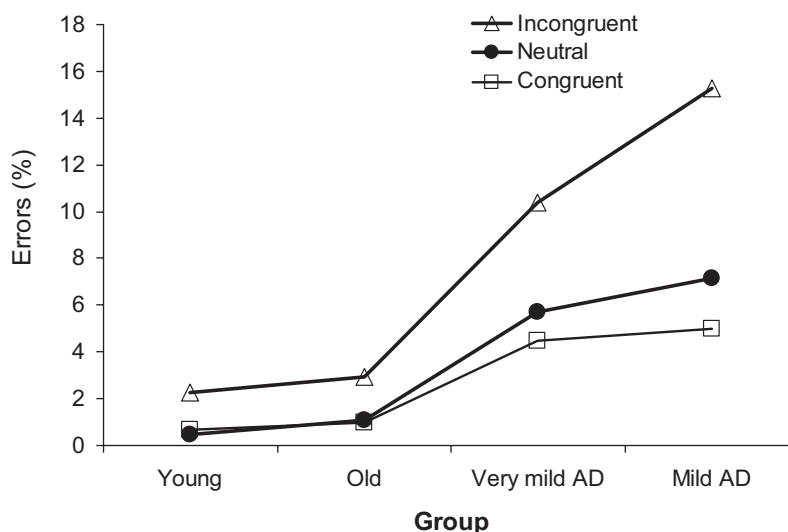
Figure 5. Mean proportion of errors for congruent, incongruent, and fixation trial types for the four groups in Experiment 2. AD = Alzheimer's disease.

across individuals increases in both healthy aging and AD, an intriguing question is whether there is increased variability *within* an individual over time (i.e., within a single testing session). This approach has been advocated by Hultsch, MacDonald, Hunter, Levy-Bencheton, and Strauss (2000), who have shown that individuals with AD demonstrate greater intraindividual variability relative to healthy younger and older adults.

To examine this in the present experiment, we calculated the intraindividual variability for each participant across all trial types and used the standard deviation as a measure of variability (see Hultsch et al., 2000, for a similar procedure). The two AD groups were combined into one general AD group in order to increase the sample size. The intraindividual standard deviation score (ISD) for each group is as follows: young = 170, old = 267, AD = 390. Entering these data into a one-way ANOVA yielded a significant between-groups effect, $F(2, 146) = 15.56$, $MSE = 31,495.71$, $\eta^2 = .18$, $p < .0001$, and follow-up t tests (Tukey) showed that each group was significantly different from each other ($p < .05$). To control for general slowing, we also computed the coefficient of variation, in which each individual's ISD is divided by his or her mean RT (as suggested by Hultsch et al., 2000), thus providing a measure of intraindividual variability relative to the individual's own level of performance (i.e., taking into account overall speed differences). Coefficients of variation for each group are as follows: young = .31, old = .34, AD = .42. An ANOVA revealed a significant between-groups effect, $F(2, 146) = 5.78$, $MSE = 0.026$, $\eta^2 = .07$, $p < .05$, mirroring the ISD analyses; however, follow-up t tests (Tukey) revealed that the younger and older adults did not significantly differ from one another ($p > .45$), although both groups were different from the AD group ($p < .05$). These findings suggest that AD leads to greater intraindividual variability relative to that demonstrated by healthy younger and older adults, above and beyond global slowing, consistent with Williams, Hultsch, Strauss, Hunter, and Tannock (2005).

General Discussion

The purpose of the present investigation was to examine how aging and AD influence the ability to control attention when conflict is presented in terms of incongruent mapping between the identity of a stimulus and the appropriate response. Building on previous research that has shown breakdowns in attentional control in old age and dementia in a variety of situations, the results from the Simon task are in line with prior research and provide further evidence regarding attentional control difficulties that are specific to the response level. In terms of healthy aging, older adults showed a larger Simon effect even after we corrected for general slowing, a finding consistent with prior investigations that involve the Simon task and aging. Previous research (e.g., Bialystok et al., 2002; Van der Lubbe & Verleger, 2002) has found that older adults display larger costs in the Simon task, and the present results converge on the point that aging leads to disproportionately larger impairments in RT on incongruent trials. We have replicated and extended these findings using *z*-score transformations.

The present results also demonstrate some intriguing differences in the temporal dynamics of the Simon effect in younger and older adults. Wiegand and Wascher (2005) have suggested that the Simon effect can be partitioned into two mechanisms: a fast, transient effect and a slower, sustained component based on controlled processing at longer RTs. Younger adults (but not older adults) show reduced Simon effects at longer RTs, suggesting that younger adults can use more controlled processing at longer RTs to reduce the magnitude of the Simon effect, and hence this observation supports Wiegand and Wascher's two-stage model. It may also be the case that this controlled processing occurs earlier for younger (but not older) adults and could contribute at early stages for relatively easy response code mappings, thus leading to reduced Simon effects for younger adults. Older adults do not appear to use this second stage of processing at longer RTs, possibly suggesting impairments of controlled processing in old

age. It is important to note that the results from Experiment 1B indicate that the observed age-related differences were not simply due to decreased visual sensitivity to peripheral information.

The present study is also the first we know of that has examined the Simon effect in AD, and it provides some insight into how this disease influences response mapping and conflict resolution, two processes that are thought to be related to frontal lobe function. Older adults diagnosed with very mild AD showed Simon effects comparable to healthy older adults but had disproportionate error rates, especially on trials that involved conflicting information. The finding of increased interference effects in response latencies in healthy older adults and increased interference in intrusion errors in individuals with AD is consistent with the pattern found in the Stroop task (see Spieler et al., 1996) and extends this to situations of response conflict, in which AD leads to a reduced ability to control prepotent responses.

Of interest, the present bin analyses also showed a decreased Simon effect at the slowest bin for the mild AD. This decrease in the Simon effect in the mild AD group is reminiscent of a similar pattern found by Spieler et al. (1996) in the Stroop task. In particular, Spieler et al. found that in contrast to the healthy older adults, early stage AD individuals did not show a disproportionate slowing in the tail of the distribution in the incongruent condition. However, they did find an increase in intrusion rate, that is, responding to the wrong code. Spieler et al. argued that instead of taking additional time to control the prepotent pathway on those difficult trials, the individuals with AD were more likely to respond to the prepotent stimulus, thereby reducing the overall interference effect in RTs but producing an effect in accuracy. As noted, there was a clear increase in error rates in the incongruent condition in the mild AD group in the present study, consistent with this notion.

Given that previous investigations of age differences in inhibitory control using the Stroop effect have yielded somewhat mixed results, with healthy older adults sometimes showing disproportionately greater interference effects (e.g., Spieler et al., 1996) and sometimes yielding age equivalence (e.g., Verhaeghen & De Meersman, 1998), it is important to consider why a greater Simon effect in old age has been reliably found in several studies, including the present investigation. One possibility is that age differences in the ability to detect and process a peripheral target can play a critical role in the magnitude of the Simon effect, and given that healthy older adults and those with AD have impairments in the allocation of peripheral attention (Festa-Martino et al., 2004; Tales et al., 2002), this factor might contribute to enhanced Simon effects in old age, independent of attentional and inhibitory control. Also, the use of arrow stimuli in the present study creates a strong overlap between the encoding of spatial information (arrow and location) and the response code, and the need to draw on attention to integrate feature information (e.g., Treisman & Gelade, 1980) likely influences the magnitude of the Simon effect. Although the Simon effect was not reduced in the present study with degraded targets, the cuing of attention to peripheral locations and incorporating longer delays between the cue and target can also diminish the Simon effect in younger adults (Stoffer & Yakin, 1994), suggesting that manipulations in the ability to allocate attention and incorporate features are important factors that can influence the magnitude of the Simon effect. Also, relative to the Stroop task, it is likely that the Simon task involves more primitive

processing (and conflict) of directionality and spatial mapping, whereas the Stroop task involves higher level lexical access at encoding and conflict between two acquired responses, that is, the name of the word and the name of the color. In this light, it is possible that the Simon task might be a purer measure of control of a prepotent response, and by measuring the ability to override strong S-R mapping, the task is able to produce reliable age differences due to its more primitive and simplistic nature. Once higher level systems are engaged (both at encoding and at retrieval), there is more opportunity for individual differences and strategic processes to play a role in performance, and this might lead to the inconsistent Stroop differences as a function of age group (once general slowing is taken into account). Hence, the Simon task presents a basic conflict situation between stimulus and response mapping and provides a useful tool to examine age differences in attentional and response control, as well as yielding useful information about how these mechanisms are impaired in AD.

One possible interpretation of the present findings is that participants with AD had difficulty maintaining the proper goal set during the testing session and would occasionally lapse and press a key that matched the side of presentation of the stimulus, rather than directing attention to the identity of the arrow. Indeed, Kane and Engle (2003) suggested that working memory is responsible for maintaining task set and response competition resolution. The healthy older and AD groups did differ in measures related to working memory (see Table 2), although the Simon effect was not reliably correlated with performance on the digit span tests. Of course, it should be noted that these measures may have limited value in capturing the executive component of working memory (see Kane & Engle, 2003). Clearly, task set has been shown to be a critical factor, in that previous research has shown that the Simon effect can diminish as the frequency of noncorresponding trials increases (e.g., Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002).

Although this interpretation is not entirely inconsistent with our findings, other current research converges on the point that AD may lead to a reduction in the ability to inhibit partially activated information. Recently, Duchek and Balota (2005) showed that AD leads to an overreliance on dominant pathways in a dichotic listening paradigm, and this pattern again did not appear to be due simply to differences in measures related to working memory. Specifically, very mild and mild AD groups showed a larger right ear advantage in free-recall performance compared with the healthy controls, indicating a tendency to respond to the prepotent left-hemisphere pathway for language processing. This tendency to respond with a prepotent pathway is somewhat similar to the errors made in the Simon task, in which individuals with AD would provide incorrect responses owing to the inability to override possibly automated S-R connections. Of note, Proctor et al. (2005) showed that the enhanced Simon effect in older adults can be reduced when a strong distinction is made between relevant and irrelevant properties of the target. Although difficulty in maintaining task set might contribute to the effects observed in the present study, it seems that failure to control prepotent response mappings is a more specific explanation that fits well with the current data and previous behavioral findings. Clearly, further work is needed to better understand the contributions of changes in inhibitory

control and changes in the maintenance of task set in accommodating both age-related and AD-related changes in attentional control systems.

It is likely that attentional control of response systems mediated by frontal lobe function contributes to performance on the Simon task, as indicated by recent work in the neuroimaging literature. Both Maclin, Gratton, and Fabiani (2001) and Peterson et al. (2002) found that the Simon task was associated with activation in the superior and inferior frontal gyrus, middle frontal gyrus, and right medial frontal gyrus. Although the present study does not provide direct evidence regarding the brain regions that contribute to performance (and errors) on the Simon task, Jonides, Badre, Curtis, Thompson-Schill, and Smith (2002) showed that inhibitory processing is associated with a lateral portion of the left prefrontal cortex, and MacDonald, Cohen, Stenger, and Carter (2000) further suggested that areas of the left prefrontal cortex are involved in representing and monitoring the control of attention. Thus, it is possible that healthy older adults and individuals with early stage AD may have the observed difficulty in inhibitory control in the Simon task because of changes in frontal lobe volume and function (Raz, 2000). The Simon task appears to consistently capture the behavioral consequences of these changes.

In summary, the present findings build on previous research that has shown attentional impairment in early stage AD (e.g., Balota & Faust, 2001) and extend this to areas of spatial S-R conflict resolution in the Simon task. Given the relatively high error rates for AD participants but not healthy older adults, one potential conclusion is that the "response selection system," which may be composed of various connections and mappings between stimulus identity and appropriate response output, is compromised by the onset of dementia. Furthermore, control over response selection may be crucial in many tasks that go beyond typical Simon-like response conflict situations. Behaviorally inappropriate output in language and motor domains also might reflect mechanisms involved in general response conflict resolution, and such output appears to be associated with early signs of AD.

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