Evidence for Intact Selective Attention in Alzheimer’s Disease Patients Using a Location Priming Task

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Researchers examining selective attentional mechanisms in patients with Alzheimer’s disease (AD) often report impairment in patients’ ability to inhibit irrelevant or distracting information. However, in many studies reporting such failures, researchers used tasks that require semantic processing, which can disrupt the full body of literature documents to be disrupted in AD. The authors of this study used a spatial location-priming task that minimized semantic processing to examine the phenomena of negative priming and facilitative priming in 13 AD patients and 13 healthy older adults. AD patients demonstrated facilitative and negative priming proportionately equivalent to that of older adults. These findings suggest that both the facilitative and inhibitory mechanisms involved in selective attention are preserved in patients with AD and can be revealed in tasks that minimize semantic processing.

*Keywords: Alzheimer’s disease, selective attention, negative priming*

Alzheimer’s disease (AD) is characterized by a breakdown of memory function, loss of spatial orientation, language impairments, and other symptoms (e.g., Heilman & Valenstein, 1993). Over the last decade, a great deal of research has revealed impairments in the domain of selective attention, particularly visual selective attention (Bell, Chenery, & Ingram, 2001; Buck, Black, Behrmann, Caldwell, & Bronskill, 1997; Foster, 2001; Foster, Behrmann, & Stuss, 1999; Parasuraman & Haxby, 1993; Perry, Watson, & Hodges, 2000; Tales, Muir, Bayer, & Snowden, 2002). The term visual selective attention refers to a constellation of mechanisms that enable individuals to maintain goal-directed behavior. Individuals accomplish this by using focal attention to identify regions or items within the visual array that are consistent with goals and by simultaneously ignoring or suppressing regions or items that are inconsistent with current goals. The rules that govern the activation and suppression of visual information are highly complex (see Dagenbach & Carr, 1994), and as suggested by Posner (1980), “it seems reasonable to suppose that orienting in semantic memory will take advantage of these same principles [component processes]” (p. 22) of attention. Thus, mechanisms involved in selective attention may not only serve to direct cognitive resources for sensory processing but may also function to orient the activation and suppression of specific internal representations, such as words, images, and memories (i.e., semantic representations; Posner, Werner-Inhoff, Friedrich, & Cohen, 1987).

Typical paradigms that are used to examine visual selective attention include search tasks (Esterman, McGlinchey-Berroth, & Milberg, 2000; Foster et al., 1999; Joseph, Chun, & Nakayama, 1997; Pavlovskaya, Ring, Grosswasser, & Hochstein, 2002), covert orientation tasks (Parasuraman, Greenwood, Haxby, & Grady, 1992; Posner, 1980), and priming tasks (Balota & Duchek, 1991; Langley, Overmier, Knopman, & Prod’Homme, 1998). The latter class of tasks allows for the examination of opposing facilitative and inhibitory mechanisms proposed to be operating in the course of normal selective attention. Facilitation refers to the enhancement of the relevant target, and inhibition refers to the suppression of distractors, both of which have been shown to be active in efficient selection of the target (Dagenbach & Carr, 1994; Houghton & Tipper, 1994). Researchers have shown that AD patients tend to show a differential impairment among the mechanisms of selective attention; specifically, they show intact facilitative mechanisms but impaired inhibitory mechanisms. For example, in one study examining facilitative and inhibitory processing during sentence comprehension, researchers required AD patients and healthy older adults (OAs) to read sentences with ambiguous meaning and then decide whether a target word was appropriate to the overall meaning of the sentence (Faust, Balota, Duchek, Gerbasi, & Smith, 1997). Compared with those of OAs, AD pa-

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This research was supported by Veterans Affairs Merit Review Awards to Regina McGlinchey and William Milberg. We thank Alan Mandell, Margaret O’Connor, and Ginette LaFleche for referring patients to us. We also thank the Harvard Cooperative Program on Aging and Fifty Plus Advocate for additional recruiting and David Wilkinson and Mieke Verfaellie for their comments.

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patients’ decision latencies were disproportionately slowed when ambiguous target words were inappropriate to the sentence context. The authors concluded that this “result is consistent with the notion that DAT [dementia of the Alzheimer type] individuals are less efficient at suppressing contextually inappropriate meanings of ambiguous words than are healthy older individuals” (Faust et al., 1997, p. 235). Of note, AD patients and OAs showed similar facilitated responses to target words that were appropriate to the sentence context.

The negative priming (NP) paradigm has also been used to investigate facilitative and inhibitory processes involved in selective attention. In NP, participants are explicitly instructed to attend to a stimulus defined by a specific trait (target) and to ignore any other stimuli defined by different traits (distractors; Tipper, 1985). When the stimuli are presented consecutively as prime–probe pairs, facilitation can be revealed as a reduction in response time due to the repetition of the prime target in the probe display, and inhibition can be revealed as an increase in response time due to the repetition of the prime distractor (ignored stimulus) in the probe display. Sullivan, Faust, and Balota (1995) used overlapping color pictures as prime–probe pairs in an NP experiment involving both healthy OAs and AD patients. Their results showed that AD patients, compared with healthy OAs, demonstrated no evidence of NP but did show equivalent facilitative (FAC) priming.

In a similar study, AD patients and healthy OAs were presented overlapping pictures and instructed to attend only to pictures in the target color (Grande, McGlinchey-Berroth, Milberg, & D’Esposito, 1996). Participants then read aloud a centrally presented word that was related to the attended picture, related to the unattended picture, or unrelated to either picture. Healthy OAs showed a facilitated response to the target word when the attended picture and the target word were related, and they showed an inhibited response when the unattended picture and the target word were related. However, AD patients showed a facilitated response when the target word was related to the attended picture as well as when it was related to the unattended picture. These findings suggest an inability of AD patients to suppress distracting semantic information.

In another study, researchers used a cue-priming experiment to investigate selective attention in patients with AD (Higgins, Duncan, Milberg, & McGlinchey, 2005). In this study, a valid or invalid cue (arrow) preceded a lateralized presentation of a real drawing in one visual field and a nonsense drawing in the other visual field. This was followed by a centrally presented target word to which participants decided to be related or unrelated to the real drawing. Whereas healthy OAs demonstrated FAC priming with the valid cue only, AD patients showed FAC priming with both valid and invalid cues. Thus, AD patients not only failed to suppress information not indicated by the cue but also processed the information enough to activate semantic representations.

These studies show a consistent report of intact facilitative mechanisms but impaired inhibitory mechanisms in AD patients. However, it may be critically important that many of the studies reporting this differential impairment involve access to semantic information; that is, semantic memory must be used to properly encode the stimuli. This semantic information is either passively activated, such as when participants are exposed to pictures but respond only to a perceptual component of the stimulus (Tipper, Brehaut, & Driver, 1990), or when semantic processing is actively used, such as in word naming tasks (Faust et al., 1997; Grande et al., 1996). In either case, semantic processing plays a definitive role in these tasks, and thus, caution must be imposed on present conclusions regarding the inhibitory failures observed in tasks of selective attention in AD, as these failures may be specific to tasks involving a semantic component. As noted earlier, disruption of the semantic memory system occurs relatively early in the course of AD (Faust et al., 1997; Grossman, Robinson, Bernhardt, & Koenig, 2001; Milberg, McGlinchey-Berroth, Duncan, & Higgins, 1999) and therefore may be contributing to the observed inhibitory failures in measures of selective attention.

Our aim in the current study was to examine whether selective attention impairments typically observed in AD patients would carry over to a visual selective attention task that minimized semantic processing. The NP paradigm was adapted to associate participants’ response with the locus of the target postselection rather than with the identity of the target (Tipper et al., 1990). This set of location-based NP experiments successfully replicated NP measures previously observed in identity-based NP paradigms. Because the response in this task is made to a location rather than to a property that is intrinsic to the target, there is no need for stimulus categorization or high-level semantic processing, making the paradigm ideal for examining selective attention in populations known to have semantic memory dysfunction. For example, a location-priming task was used to assess measures of attention in patients with frontal and posterior damage (Stuss et al., 1999). In other studies, researchers have found that detection of NP in certain populations is dependent on whether tasks are identity based or location based. For example, researchers found that children do not show NP effects on identity-based tasks (Tipper, Borque, Anderson, & Brehaut, 1989) but do show NP effects on location-based tasks (Tipper & McLaren, 1990). Differential results across identity-based and location-based tasks have also been found in the aging population, in which OAs failed to show NP on identity-based tasks but showed NP on location-based tasks (Connelly & Hasher, 1993).

We predicted that if previously observed inhibitory deficits in AD patients are due to the untoward influence of an impaired semantic memory system, AD patients would demonstrate intact NP as well as intact FAC relative to healthy OAs in this location-based NP task. If confirmed, these findings would reveal significantly slower responses on NP trials compared with the neutral baseline and significantly faster responses on FAC trials compared with a neutral baseline for both AD patients and healthy OAs.

Method

Participants

Participants included 13 older adults diagnosed with probable AD by neurologists or clinicians following the criteria of the National Institute of Neurological and Communications Disorders—Alzheimer’s Disease and Related Disorders Association (McKhann et al., 1984). Additional measures included the Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975), the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983), and the American New Adult Reading Test (Grober & Sliwinski, 1991). Mean scores on neuropsychological tests suggested mild or moderate impairment of AD (see Table 1). Patients were referred and
Table 1

Means, Standard Errors, and Ranges of Demographic and Clinical Measures for Participants

<table>
<thead>
<tr>
<th>Group and measure</th>
<th>Age (years) M</th>
<th>Age (years) SE</th>
<th>MMSE M</th>
<th>MMSE SE</th>
<th>BNT M</th>
<th>BNT SE</th>
<th>ANART M</th>
<th>ANART SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s disease</td>
<td>77.54</td>
<td>1.71</td>
<td>22.61</td>
<td>1.14</td>
<td>46.58</td>
<td>3.49</td>
<td>39.63</td>
<td>2.24</td>
</tr>
<tr>
<td>Range</td>
<td>68–87</td>
<td>8–25</td>
<td>16–29</td>
<td>22–60</td>
<td>17–50</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Older adults (3 men, 10 women)</td>
<td>76.69</td>
<td>0.91</td>
<td>29.54</td>
<td>0.14</td>
<td>56.69</td>
<td>1.09</td>
<td>47.38</td>
<td>1.58</td>
</tr>
<tr>
<td>Range</td>
<td>73–82</td>
<td>12–20</td>
<td>29–30</td>
<td>45–60</td>
<td>41–60</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. MMSE = Mini-Mental State Examination; BNT = Boston Naming Test; ANART = American New Adult Reading Test.

Results

The primary dependent variables of this experiment were errors on probes and decision latencies made on probes. Trials with response errors made during the prime were excluded from the error analysis. In the FAC condition, this exclusion resulted in an average loss of 0.85% of trials for OAs as well as for AD patients. In the NP condition, this resulted in an average loss of 0.85% of trials for OAs and 1.3% of trials for AD patients. Mean percentages of error rates can be found in Table 2. Trials with response errors on either the prime or the probe were eliminated from the decision latency analysis. In the FAC condition, this resulted in an average loss of 4.5% of trials for OAs and 6.7% of trials for AD patients. In the NP condition, this resulted in an average loss of 3.4% of trials for OAs and 5.0% of trials for AD patients. Raw latency measures can be found in Table 3. Because group differences between AD patients and OAs are often driven by an overall slowed performance by AD patients (Faust, Balota, Spieler, & Ferraro, 1999), we transformed decision latency data into proportion scores to correct for AD-related slowing. We calculated proportions by forming ratios of the mean decision latency in each cue type (baseline and FAC; baseline and NP) divided by the overall reaction time across FAC and NP conditions for each participant. This calculation can be expressed by the following equation, in which cond stands for condition and DL stands for decision latency: proportion cond = DLcond/DLoverall.

Facilitation

We submitted mean percentages of error rates of probes to a mixed two-factor analysis of variance (ANOVA) to examine the effects of group (AD patients vs. OAs) and cue (baseline vs. FAC). There was no main effect of group, F(1, 24) = 1.78, but there was a main effect of cue, F(1, 24) = 31.10, p < .01, showing that baseline trials (M = 0.072, SE = 0.012) produced a higher error rate than FAC trials (M = 0.014, SE = 0.005). There was a significant interaction between group and cue, F(1, 24) = 4.29, p < .05. Mean error rates and standard errors can be found in Table 2 for FAC and NP. A means comparison showed that although both groups made errors on baseline trials, AD patients made more errors than did OAs, F(1, 24) = 7.73, p < .05. Means comparisons showed improvements on FAC trials over baseline trials in both the AD patients, F(1, 24) = 29.25, p < .01, and OAs, F(1, 24) = 6.14, p < .05. Although AD patients produced more baseline errors than did OAs, a means comparison showed that there was no difference in error rates on FAC trials between AD patients and OAs, F(1, 24) = 0.02, p = .88, suggesting that the AD patients’ error rate was reduced to a greater degree than that of OAs.
We also submitted decision latency data to a mixed two-factor ANOVA to examine the main effects of group (AD patients vs. OAs), cue (baseline vs. FAC), and the interaction between group and cue. The proportion transformation was effective in eliminating a main effect of group, $F(1, 24) = 0.06, p = .80, \eta^2 = 0.005$; however, there was a main effect of cue, $F(1, 24) = 14.75, p < .01, \eta^2 = 0.388$, indicating significantly faster decision latencies on the FAC trials ($M = 0.944, SE = 0.011$) than on baseline trials ($M = 1.033, SE = 0.018$). There was no significant interaction between group and cue, $F(1, 24) = 0.79, p = .38, \eta^2 = 0.029$. We performed planned comparisons between baseline and FAC conditions and confirmed the presence of significant FAC priming in both the AD ($p < .01$) and OA ($p < .05$) groups.

NP

We submitted mean percentages of error rates of probes to a mixed two-factor ANOVA to examine the effects of group (AD patients vs. OAs) and cue (baseline vs. FAC). There was no main effect of group, $F(1, 24) = 0.07, p = .80$, but there was a main effect of cue, $F(1, 24) = 4.23, p = .05, \eta^2 = 0.15$. There was no significant interaction between group and cue, $F(1, 24) = 0.79, p = .38, \eta^2 = 0.029$.

Table 2
Mean Percentages of Error Rates (and Standard Errors) Made on Probes

<table>
<thead>
<tr>
<th>Group</th>
<th>Facilitative priming Baseline</th>
<th>Facilitative priming Cued</th>
<th>Negative priming Baseline</th>
<th>Negative priming Cued</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer's</td>
<td>0.092 (0.018)</td>
<td>0.013 (0.007)</td>
<td>0.06 (0.014)</td>
<td>0.004 (0.003)</td>
</tr>
<tr>
<td>Older adults</td>
<td>0.051 (0.014)</td>
<td>0.015 (0.007)</td>
<td>0.028 (0.011)</td>
<td>0.027 (0.007)</td>
</tr>
</tbody>
</table>

Table 3
Means (and Standard Errors) for Decision Latencies in Milliseconds

<table>
<thead>
<tr>
<th>Group</th>
<th>Facilitative priming Baseline</th>
<th>Facilitative priming Cued</th>
<th>Negative priming Baseline</th>
<th>Negative priming Cued</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s</td>
<td>683 (69.84)</td>
<td>603 (48.76)</td>
<td>633 (34.46)</td>
<td>661 (40.26)</td>
</tr>
<tr>
<td>Older adults</td>
<td>513 (17.99)</td>
<td>479 (15.87)</td>
<td>491 (14.88)</td>
<td>517 (13.24)</td>
</tr>
</tbody>
</table>

Figure 1. A schematic of displays used in each trial of this experiment. The top pathway follows the succession of the negative priming condition, and the bottom pathway follows the succession of the facilitative priming (facilitation) condition. The bottom arrow represents each stage of the trial, including the exposure duration of the stimulus and the interstimulus interval. Targets (O) and distractors (X) appear at critical locations during the prime and probe to establish the negative priming cue or facilitative priming cue.
effect of cue, \( F(1, 24) = 8.22, p < .01 \), showing that baseline trials \((M = 0.042, SE = 0.009)\) produced a higher error rate than NP trials \((M = 0.016, SE = 0.005)\). There was a significant interaction between group and cue, \( F(1, 24) = 7.50, p < .05 \). A means comparison showed that AD patients had a higher error rate than OAs on baseline trials, \( F(1, 24) = 4.63, p < .05 \). OAs did not show a difference of error rate between baseline and NP trials, \( F(1, 24) = 0.008 \), but AD patients showed a significantly lower error rate on NP trials than on their baseline trials, \( F(1, 24) = 15.70, p < .01 \). The difference in NP trials between AD patients and OAs was not significant, \( F(1, 24) = 2.96, p = .098 \).

We submitted decision latency data for NP to another mixed two-factor ANOVA to examine the effects of group (AD patients vs. OAs) and cue (baseline vs. NP). Again, the proportion transformation eliminated the main effect of group, \( F(1, 24) = 0.06, p = .80, \eta^2 = 0.002 \), and there was a significant effect of cue, \( F(1, 24) = 20.27, p < .01, \eta^2 = 0.44 \), indicating significantly slower decision latencies on the NP trials \((M = 1.035, SE = 0.012)\) compared with baseline trials \((M = 0.988, SE = 0.097)\). There was no significant interaction between group and cue, \( F(1, 24) = 0.33, p = .57, \eta^2 = 0.010 \). Planned comparisons between baseline and NP conditions showed significant inhibition on NP trials for both AD patients \((p = .01)\) and OAs \((p < .01)\). Plots of NP and FAC proportions can be found in Figure 2.

### Discussion

The proportional scores from the decision latency data clearly demonstrated intact facilitative and inhibitory processing among AD patients relative to healthy OAs in a location-based priming task. The error rate data showed a higher error rate in AD patients, but this was reflected mostly in baseline trials; in the FAC condition, they showed an identical error rate to the OA group when given a facilitative cue. AD patients also produced fewer errors on NP trials compared with baseline trials, a result that hints at an NP deficit. However, AD patients’ error rate on NP trials was not significantly different from that of OAs, and additionally, any trend in differences was a matter of only a few trials. Thus, even though the error rate data did not strictly conform with the notion of intact inhibitory processing for the patients, we argue that this trend was due to increased errors in the baseline condition and not to decreased errors in the NP condition. Taken together, these data provide strong evidence for intact selective attention in this task with AD patients, which contrasts with several studies in the literature that demonstrated impaired inhibition in AD patients.

The results of this experiment suggest that previously observed deficits may have occurred because of the involvement of semantic processing.

This semantic–nonsemantic dichotomy was suggested in an earlier study in which AD patients and healthy OAs were first presented with valid, invalid, or neutral arrow cues and then presented letters appearing left or right of center (Parasuraman et al., 1992). In the discrimination task, participants were required to report whether the letter presented was a vowel or consonant; in the letter-detection task, participants responded only to a letter X. AD patients showed impaired performance to the invalid cue and not valid cues, but these effects were evident only with the letter-discrimination task and not the letter-detection task. The authors raised the possibility that the discrimination task required more focal attention and thus elicited the attentional impairments in AD.

An alternative explanation may be that the letter discrimination task required the retrieval of semantic information to accurately categorize the stimuli for the response, whereas the detection task required a perceptually driven response. This provides an example in which inhibitory mechanisms in AD were impaired in the presence of a semantic component but were intact in its absence.

In a more recent study, researchers also reached the conclusion that differential impairment of inhibitory mechanisms may be dependent on the presence of a semantic component (Amieva et al., 2002). In this study, inhibitory measures were systematically characterized on an identity-based NP task, a Stroop task, a no-go task, and a stop-signal task. AD patients demonstrated a lack of NP that is consistent with results from a study by Sullivan, Faust, and Balota (1995). On the Stroop task, AD patients showed increased response latencies, which were concomitant with increased errors, suggesting a failure to inhibit irrelevant information despite increased processing time. Both the NP task and Stroop task involved semantic processing to encode the stimuli (i.e., picture identification and word reading). However, AD patients showed limited impairments on the no-go task and normal performance on the stop-signal task. The need for semantic processing was minimized in the no-go and stop-signal tasks, in which participants responded to a red circle or a blue triangle. Thus, in this characterization of inhibitory processes, the need for semantic information was reduced in the tasks, inhibition was less impaired in AD patients.

These results are similar to studies in the literature on aging. Decreased NP effects have been found in OAs compared with younger adults on identity-based NP tasks (Hasher, Stoltzfus, Zacks, & Rypma, 1991), although this result is controversial (Grant & Dagenbach, 2000; Schooler, Neumann, Caplan, & Roberts, 1997). However, on location-priming tasks, NP effects were equal in both groups (Connelly & Hasher, 1993). These findings are summarized in a model proposed by Connelly and Hasher (1993), which states that changes in response inhibition are due to alternative pathways through which input may flow. With this model, the researchers claimed that responses to properties of the target identity enable processes that use more of the parvocellular (“what”) pathway, whereas responses to the target location enable processes that use more of the magnocellular (“where”) pathway.

Although the results of the current study are analogous to the aging data that support this model, caution must be taken when claiming that changes in selective attention are solely dependent on visual pathways, for this assumption potentially eliminates factors outside of vision (see Kramer, Humphrey, Larish, Logan, & Strayer, 1994). Additionally, some researchers have noted damage to the magnocellular pathways in AD (Gilmore, Wenko, Naylor, & Koss, 1994; Jacob, Hache, & Pasquier, 2002).

In an alternative model, researchers propose that differences in NP reflect two attentional systems, a posterior system and an anterior system (Posner, 1992; Posner & Peterson, 1990). The posterior system, which includes the parietal cortex, the pulvinar nucleus of the thalamus, and the superior colliculus, mediates attention to perceptual and spatial characteristics of the target. The anterior system, which includes the anterior cingulate and prefrontal area, mediates executive, decision-making aspects of selective
attention, as well as semantic processing. Posner (1994) cites evidence for this model in an EEG study in which participants presented with word stimuli were instructed to locate a visual feature (search task) or to categorize whether the word referred to a natural or artificial object (semantic task). Performance on the semantic task activated several frontal regions bilaterally, whereas performance on the search task activated the right posterior area.

In a recent functional MRI investigation, researchers showed that anterior regions of the brain were sensitive to manipulations in target-relevant information and that posterior regions were sensitive to manipulations in target-irrelevant information in Stroop tasks (Banich et al., 2000). The authors noted that posterior regions are only influenced by target-irrelevant information when it is categorically related to target-relevant information, as suggested by past studies. This provides evidence for the modulation of target-irrelevant information in target selection, as in inhibitory components of selective attention models (Dagenbach & Carr, 1994; Houghton & Tipper, 1994). Banich et al. (2000) postulated that an executive role of the anterior system is to alert regions that are sensitive to certain information, including the posterior system.

In the context of semantic information, perhaps impairments of semantic processing and/or categorization in AD preclude sending

Figure 2. Interaction plots of facilitative priming (A) and negative priming (B). The y-axis represents the ratio of the mean decision latency in each condition over the overall mean decision latency for each participant. Error bars represent the standard error. RT = reaction time; BASE = baseline; FAC = facilitative priming; AD = Alzheimer’s disease; OA = older adults; NP = negative priming.
target-irrelevant information from anterior to posterior regions, resulting in a lack of inhibition. In our experiment, in which we have minimized the role of the anterior system, processing of target-irrelevant changes (i.e., location of the distractor) remains intact, thus resulting in intact NP.

The current results appear to fit within this posterior–anterior dichotomy, in which, by minimizing semantic processing, activity is concentrated more toward the posterior system rather than the anterior system. This model would suggest that previously observed inhibitory deficits in AD could be attributed to the anterior system that mediates executive functions (Vogt, Finch, & Olson, 1992). This area is known to undergo severe neuronal loss in AD (Vogt, Crino, & Volicer, 1991), and although it is not clear when this occurs in the course of the disease, it has been suggested that the degradation occurs in early to mid stages (Albert & Moss, 2002).

Interest in dissecting the causes of NP has led to the development of noninhibitory models that have emerged to challenge the inhibitory model (for a comprehensive review, see Fox, 1995), including the feature mismatch hypothesis (Park & Kanwisher, 1994) and the episodic retrieval model (Neill & Valdes, 1992; Neill, Valdes, Terry, & Gorfein, 1992). The feature mismatch hypothesis states that NP occurs because of a discrepancy between the features of the prime distractor and the probe target. In a spatial priming task in which participants were instructed to switch the identity of the target and distractor between prime and probe presentation, Park and Kanwisher (1994) demonstrated NP when the identity of the target was switched but remained in the same location between prime and probe (symbol mismatch). Additionally, they showed that when a prime distractor maintained its location as it became a probe target, facilitation was observed (symbol match).

The episodic retrieval model states that exposure to the probe activates the episodic retrieval of the prime, and a mismatch between the episodic states results in slowed responses. Thus, the temporal direction of the effect is retroactive from the probe to a trace of the prime response, whereas an inhibitory account purports to have more of a proactive effect: The prime response affects the probe response. A critical difference between these hypotheses (according to early findings, e.g., Tipper, 1985; Tipper & Cranston, 1985) regards persistence: The inhibitory model originally asserted that representations of the inhibited distractor remain activated, but inhibitory mechanisms block the response to the distractor. The episodic retrieval hypothesis states that the representation of the distractor is suppressed, that is, deactivated, and thus, NP represents retrieval after the mismatch (of episodes and response tags) has been reconciled. Therefore, inhibitory accounts predicted a persistence of NP effects, whereas retrieval accounts predicted a decay of NP over time. Evidence for the episodic retrieval hypothesis comes from a study by Neill et al. (1992), who demonstrated decay in NP over longer ISIs as long as ISI was maintained within participants. This decay was shown in a letter-matching task (Neill & Valdes, 1992) and a location-priming task (Neill et al., 1992). Unlike the feature mismatch hypothesis (and like the selective inhibition hypothesis), the episodic retrieval hypothesis requires the probe target (or a feature of the probe target) to formerly act as a prime distractor to generate NP. In a recent review, Tipper (2001) attempted to reconcile inhibitory and episodic retrieval accounts, stating that integrating the accounts results in a comprehensive model of the NP phenomenon. Tipper also cited countering studies and methodological problems with the feature mismatch hypothesis; suggestive evidence against feature mismatch can be found in results from a study by Stuss et al. (1999).

Although the current study was not constructed to compare these differing hypotheses, the finding that AD patients show intact NP may provide insight into this debate. The typical episodic memory impairments found in AD patients potentially contend with any model that supposes an episodic retrieval component, and the current results could suggest a differential role of episodic retrieval between identity- and location-based priming tasks. Sullivan et al. (1995) briefly referred to this memory impairment as a possible explanation for lack of NP in AD patients on a picture-priming task, and although our current findings challenge this possibility, the difference in elapsed time between primes and probes in the two experiments (300 ms in the current study vs. 600 ms in the Sullivan et al., 1995, study) prevents a means of comparing any episodic factors. This would be a critical comparison in future studies because of Tipper’s (2001) assertion that NP is best described as a tandem effort between inhibitory mechanisms and episodic retrieval, and thus, perhaps the difference between the identity- and location-based tasks could be best explained as a function of emphasis between inhibitory and retrieval mechanisms. That is, one task benefits differentially from one mechanism and vice versa. Perhaps the next step in determining the impact of AD on NP is to examine (a) the presence or absence of semantic components to further characterize selective attention in AD and (b) the manipulations of intertrial intervals to examine the presence of episodic factors and whether they affect patient performance. Such studies promise to provide an improved characterization of attention and memory in AD.

References


Received December 14, 2003
Revision received June 8, 2004
Accepted June 15, 2004