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Working Memory in Mild Alzheimer's Disease and Early Parkinson's Disease

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Alzheimer's disease (AD) and Parkinson's disease (PD) impair working memory (WM). It is unclear, however, whether the deficits seen early in the course of these diseases are similar. To address this issue, the authors compared the performance of 22 patients with mild AD, 20 patients with early PD and without dementia, and 112 control participants on tests of inhibition, short-term memory, and 2 commonly administered tests of WM. The results suggest that although mild AD and early PD both impair WM, the deficits may be related to the interruption of different processes that contribute to WM performance. Early PD disrupted inhibitory processes, whereas mild AD did not. The WM deficits seen in patients with AD may be secondary to deficits in other cognitive capacities, including semantic memory.

There has recently been an expanding interest in how neurological diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD) affect *working memory* (WM), which is defined as the online storage and manipulation of information for a short period of time (i.e., seconds). In a widely accepted model proposed by Baddeley and Hitch (1974), WM consists of three main components: the phonological loop, the visuospatial sketch pad, and the central executive. The phonological loop and visuospatial sketch pad are recruited for the storage and manipulation of verbal and nonverbal information, respectively. The central executive (Baddeley, 1992), modeled as the supervisory attentional system (Norman & Shallice, 1980), is a limited-capacity attentional system that selects goal-relevant behavior by focusing and switching attention. Putative functions of the central executive include inhibiting an automatic or prepotent response, shifting attentional set, coordinating information from the phonological loop and visuospatial sketch pad, and strategically (e.g., to meet specific goals or task requirements) retrieving information from long-term memory stores.

The functions of the central executive are closely tied to the idea of cognitive control: the ability to coordinate actions and thought processes directed toward a desired outcome (i.e., a goal). There are several influential models of cognitive control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Braver, Cohen, & Servan-Schreiber, 1995; Miller & Cohen, 2001). At the core of these models is the idea that top-down signals (posited to arise from prefrontal cortex) bias the processing of lower level, posterior brain regions (Desimone & Duncan, 1995; Miller & Cohen, 2001). Thus, processing of task-relevant information continues while computations conducted on distracting information are halted. Through such top-down control, prefrontal cortex can allow maintenance or change of goal states and can bias processing in accord with those goals.

Working Memory Performance in AD and PD

Although long-term memory deficits are the hallmark of AD, numerous studies have shown that AD can also result in deficits in short-term memory of information as well as higher level deficits related to the ability to coordinate multiple tasks or to inhibit irrelevant information. For example, patients with AD have been shown to have impairments in dual-task performance (Baddeley, Baddeley,

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Bucks, & Wilcock, 2001; Baddeley, Bressi, Della Sala, Logie, & Spinnler, 1991; Baddeley, Logie, Bressi, Della Sala, & Spinnler, 1986; Becker, 1988; Greene, Hodges, & Baddeley, 1995; Morris & Baddeley, 1988; Perry, Watson, & Hodges, 2000), inhibitory ability (Simone & Baylis, 1997), and set-shifting ability (Dorion et al., 2002; see Perry & Hodges, 1999, for a review).

Similarly, despite James Parkinson's (1817) initial description of PD as a disease with symptoms circumscribed to the motor domain, recent studies have shown that PD also results in a host of cognitive deficits (Brown & Marsden, 1988; Growdon, Corkin, & Rosen, 1990; Ogden, Growdon, & Corkin, 1990; Sagar, Sullivan, Gabrieli, Corkin, & Growdon, 1988), including difficulties updating verbal and visuospatial representations (Blonder, Gur, Gur, Saykin, & Hurtig, 1989; Cronin-Golomb, Corkin, & Growdon, 1994; Postle, Jonides, Smith, Corkin, & Growdon, 1997; Postle, Locascio, Corkin, & Growdon, 1997) and inhibiting processing of task-irrelevant information (Brown & Marsden, 1988, 1991; Cronin-Golomb et al., 1994; Ogden et al., 1990; Owen, Roberts, Polkey, Sahakian, & Robbins, 1991; Postle, Jonides, et al., 1997; Postle, Locascio, et al., 1997).

Despite the growing literature examining WM changes in AD and PD, no studies have directly compared the performance of patients with AD and those with PD on a series of tasks that assess various cognitive capacities related to WM. Critically, many studies have assessed WM function using complicated tasks that require simultaneous use of multiple cognitive capacities (e.g., short-term memory, updating of representations, inhibiting task-irrelevant information). Although these studies have been useful in providing a base from which to investigate the WM deficits in these patient groups, it is currently difficult to sift through the literature to find agreement in whether the core deficits in AD and PD are distinct.

Contributing to the opacity is the use of various disease stages within and across studies. Many researchers have combined patients at multiple stages of the disease or, in the case of PD, have compared performance of a group with early, nonmedicated PD with that of a group with late-stage, medicated PD (e.g., Cooper, Sagar, Jordan, Harvey, & Sullivan, 1991; Owen, Iddon, Hodges, Summers, & Robbins, 1997; Zimmermann, Sprengelmeyer, Fimm, & Wallesch, 1992).

Present Study

The primary goals of the current study were (a) to investigate whether patients with mild AD and patients with medicated, early PD without dementia show differential impairment on measures of inhibition (Hasher & Zachs, 1988); (b) to examine whether the inhibitory ability in these two groups is related to other processing deficits (e.g., short-term memory deficits); and (c) to examine whether the groups performed similarly on commonly administered tests of WM (2-back and reading span).

One of the reasons we chose to focus on inhibition is because there is evidence that, at least in moderate to late stages of the disease, AD and PD alter inhibitory ability. On many tasks, patients with PD show deficits that parallel those of patients with frontal lobe lesions (Brown & Marsden, 1988; Owen et al., 1993; Taylor, Saint-Cyr, & Lang, 1986). Frontal lobe dysfunction likely results from the deafferentation of the frontal lobe from the basal ganglia, and the disruption of the numerous reciprocal connections between the basal ganglia and the frontal lobe (Alexander & Crutcher, 1990; Alexander, DeLong, & Strick, 1986). Although inhibitory ability has been less thoroughly investigated in AD, there are a number of studies indicating that inhibition is reduced at least in moderate AD (Fimm, Bartl, Zimmerman, & Wallesch, 1994; Simone & Baylis, 1997; Sommers, 1998). It was, therefore, important to examine whether these deficits were present early in the disease.

We also chose to focus on inhibition because of its contribution to many cognitive tasks, as well as its clinical relevance. In daily life, as well as in the laboratory, task performance often requires suppression of interference from a combination of internal sources (e.g., response tendencies or associations) and external sources (e.g., salient stimuli). This ability to selectively attend to task-relevant information, and to suppress interference from distractors, is central to our ability to carry out a variety of tasks.

Because of its importance, deficits in inhibitory ability can contribute to deficits in other domains, including short-term memory: Given the limited capacity of short-term memory, if irrelevant information is kept online, task-relevant information is denied access to short-term memory (Collette, Van der Linden, Bechet, & Salmon, 1999; Engle, 1996). We wanted to assess performance on tasks of verbal short-term memory to allow us to elucidate whether the correlations between inhibitory ability and short-term memory were altered by the disease processes.

Our motivation for including the 2-back and reading span tasks was that these are commonly administered tasks used to assess WM function. Successful performance on each task requires a number of cognitive processes (e.g., short-term memory, updating of representations, inhibiting inappropriate motor or verbal output; see Whitney, Arnett, Driver, & Budd, 2001, for discussion). We were therefore interested in examining (a) whether the group with AD and the group with PD showed similar levels of performance on these tasks and (b) whether the performance of the group with AD and the group with PD on these tasks correlated with similar or distinct cognitive capacities. This question has not been addressed in prior studies comparing these patient populations, and it seemed useful to compare not only their overall levels of performance but also whether their impairments on these complex tasks resulted from different core deficits.

Method

Participants

The participants comprised 22 patients with AD, 20 patients with PD, and 112 control (CON) participants. Patients with AD and PD were referred to the study from the Memory Disorders Unit or Movement Disorders Unit at the Massachusetts General Hospital. All patients met research criteria for probable AD (Na-

tional Institute of Neurological Disorders and Stroke–Alzheimer's Disease and Related Disorders Association; McKhann et al., 1984) or PD (Ward & Gibb, 1990). CON participants were recruited through flyers posted throughout the Boston–Cambridge area and through the Harvard Cooperative on Aging; all had a normal neurological examination within 1 year of testing, and none showed signs of dementia. The CON group's mean Blessed Dementia Scale score (Information, Memory, and Concentration section; Blessed, Tomlinson, & Roth, 1968) was 0.3 ($SD = 0.2$). All participants were screened to eliminate those with a history of alcoholism, major heart disease, cancer, or neurological or psychiatric disorders other than the primary diagnosis (AD or PD). All participants were native English speakers. Patients with PD were all taking Carbidopa–Levodopa, and patients with AD were all taking Donepezil. Participants were not taking any other medication (e.g., anticholinergics, antidepressants, anxiolytics) that could affect cognition. No participants were depressed or had a prior diagnosis of depression. All scored below a 4 on the Geriatric Depression Scale (Sheikh & Yesavage, 1986; scores less than 7 are considered not depressed).

The group with AD had a mean Blessed Dementia Scale score of 6.1 ($SD = 3.2$). No patient with AD had a Blessed Dementia score greater than 10. The average time since diagnosis of AD was 1.6 years ($SD = 1.1$). For the group with PD, Hoehn and Yahr stage ranged from 0 to 2, Unified Parkinson's Disease Rating Scale (Fahn & Elton, 1987) scores ranged from 4 to 35, and the average time since diagnosis was 4.7 years ($SD = 4.3$). None of the patients with PD had dementia (mean Blessed Dementia Scale score = 0.5, $SD = 0.6$).

The patients with AD (14 men and 8 women) had a mean age of 73.4 ($SD = 6.6$), mean of 15.5 years of education ($SD = 2.9$), and a mean Wechsler Adult Intelligence Scale (3rd ed.; WAIS-III; Wechsler, 1997) Vocabulary age-adjusted scaled score of 12.3 ($SD = 1.2$). The PD patients (15 men and 5 women) had a mean age of 68.7 ($SD = 8.9$), a mean of 16.6 years of education ($SD = 2.9$), and a mean WAIS-III Vocabulary age-adjusted scaled score of 12.7 ($SD = 1.1$). CON participants (64 men and 48 women) had a mean age of 70.6 ($SD = 7.2$), a mean of 15.2 years of education ($SD = 2.6$), and a mean WAIS-III Vocabulary age-adjusted scaled score of 12.4 ($SD = 1.5$). The three groups did not differ significantly in age, $F(2, 151) = 2.6$, $p > .15$; education, $F(2, 151) = 2.2$, $p > .15$; or WAIS-III Vocabulary score, $F(2, 151) = 0.84$, $p > .40$. The three groups did not differ in movement time or response time (as assessed by the go/no-go task; Canavan, Sprengelmeyer, Diener, & Homberg, 1994; Kischka, Mandir, Ghika, & Growdon, 1993) or processing speed (as assessed by the digit/symbol and digit/digit task; Salthouse & Meinz, 1995).

Cognitive Tasks

Below we describe the tasks administered to achieve each of the study's goals. Completion of the test series required 3–4 hr. Participants were given breaks approximately every hour, including an hour break for lunch; they were remunerated at a rate of $10 per hour.

Tests of Inhibitory Ability

The first goal of this study was to assess the inhibitory ability of the patients with mild AD and those with early PD. To this end, we tested participants on the Stroop task and the go/no-go task, and we tabulated perseverative errors on a category fluency task. Although it is impossible to find tasks that tap only one cognitive function, these three tasks provide measures of inhibitory ability, and they should vary in the degree to which they rely on other capacities.

Stroop task (Stroop, 1935). Participants first read as many words as possible in 45 s (Card 1); the words were *red, green,* and *blue,* written in black ink. Next, participants were asked to name the color of ink in which a series of *X*s were written (Card 2). Then, they were asked to name the color of ink in which a color word was written (e.g., the word *green* written in red ink would require the response of "red"; Card 3). The Stroop interference score was computed using the formula $100 + I - (W \times C)/(W + C)$, where *W, C,* and *I* were the number of items read on Card 1, Card 2, and Card 3, respectively. This formula subtracted a "predicted interference" score $(W \times C)/(C + W)$ from the raw score I (Golden, 1978), with 100 added to avoid negative numbers. The predicted interference score takes into account how much easier it is for a person to read words versus to name colors (i.e., how "automated" reading is for that particular individual). For example, if a person is able to read 80 words (Card 1) in 45 s and can name 40 colored *X*s (Card 2) in 45 s, he or she would be expected to have greater interference on Card 3 than a person who reads 50 words (Card 1) and names 45 colors (Card 2). This interference estimation differs for the two people because the first person appears to have a much easier time reading the words than saying the colors (i.e., reading is more automatic), whereas for the second person, that is not the case. Thus, the predicted interference score for the first person would be higher than for the second person.

Go/no-go task (adapted from Canavan et al., 1994, and Kischka et al., 1993). Participants placed their index finger on the near button of a two-button box and watched a computer screen as the instruction "Stay" or "Move" appeared. If the instruction was "Move," participants had to lift their finger from the near button, depress the far button, and return their finger to the near button. If the instruction was "Stay," participants did not lift their finger from the near button. "Move" occurred on 80% of the trials, and "Stay" appeared on 20% of the trials. We measured participants' accuracy on the "Stay" trials. Because "Move" appeared on 80% of the trials, and "Stay" on only 20%, the "Stay" trials required inhibition of a prepotent response. We, therefore, considered an error on a "Stay" trial to be a "false start" because participants lifted their index finger from the initial button despite the instruction to "Stay." There were 100 trials.

Category fluency (Martin & Fedio, 1983; Newcombe, 1969). Participants were asked to generate as many words as possible in 60 s that were members of a named category (e.g., fruits). Participants generated words from six categories (fruits, toys, parts of the human body, insects, articles of clothing, and carpenter's tools) and were told not to give the same exemplar more than once. We used perseverative errors (repeated generation of an exemplar) as a measure of inhibitory ability; specifically, we scored for the percentage of responses that were perseverative errors (i.e., number of perseverations/number of correct category exemplars generated). This correction was necessary because many patients with AD generated fewer category exemplars than CON participants or patients with PD; thus, considering only the number of perseverations would underestimate the frequency with which patients with AD made perseverative errors.

Tests of Short-Term Memory

The second goal of this study was to examine whether inhibitory ability was related to performance on measures of short-term memory in the patient groups or in CON participants. To address this question, we administered tests of verbal short-term memory (Digit Span, word span). These tasks measured people's ability to store information for a short period of time (less than 30 s). We could, therefore, examine the correlations between performance on tests of inhibition and tests of verbal short-term memory.

Digit Span (WAIS-III). This task measured verbal short-term memory. Digit strings were presented orally at a rate of one digit per second. Participants were asked to repeat the digits in the same order as they had been presented. Two attempts were allowed at each digit string length. The digit span was the longest string of digits that could be recalled correctly on at least one of the two attempts. The test was discontinued when a participant failed two attempts at a given digit string length. The longest string presented was nine digits.

Word span (Talland, 1965). The word span task measured verbal short-term memory. Task administration and scoring were identical to that of Digit Span. The longest string administered was nine words. All words were one-syllable, concrete nouns (e.g., *tool–horse–cake–tooth–bell*). This task was harder than Digit Span; data from young adults (not reported here) indicate that most have a word span of two less than their digit span.

Standard Tests of WM

Many studies have examined WM function using the reading span task or the *n*-back task. The *n*-back task also has been used frequently in neuroimaging experiments to assess the neural substrates of WM. These tasks recruit multiple cognitive capacities; both tasks require short-term memory and manipulation and updating of information as well as inhibition of motor or verbal output. We administered these tests in an attempt to determine what underlying capacities best correlated with the performance of the group with AD and the group with PD on these tasks. By examining the pattern of errors on these tasks, and by correlating performance with measures of short-term memory and inhibition, we sought to clarify whether the two patient groups were impaired on these more general WM tasks, and if so, whether their impairments stemmed from similar cognitive deficits.

2-back (Cohen et al., 1997). The stimuli were four-letter abstract nouns (e.g., *love, site, idea*), presented one at a time at a rate of one word every 2 s. Participants were asked to press one button whenever the current stimulus matched the stimulus that had appeared two before it (i.e., with one intervening stimulus) and to press a second button whenever the stimulus was not a match to the stimulus that occurred two before. On 30% of the trials, the item was a match; on 70% of the trials, the item was not a match. We computed the percentage of hits, the percentage of false alarms, and the corrected hit rate (percentage of hits − percentage of false alarms). This task required maintenance (participants had to hold online the words as they appear), updating (participants had to constantly update the words they are holding online), and inhibition of motor responses (because a target occurred less frequently than a nontarget, participants had to inhibit the tendency to always press the nontarget button).

Reading span (adapted from Daneman & Carpenter, 1980). A sentence was presented on a computer screen (e.g., "The boy ate four hamburgers for lunch."). Participants read the sentence aloud and were then asked to answer a simple comprehension question about it (e.g., "What did the boy eat?"). Participants answered the question aloud (e.g., "hamburgers") and were then shown another sentence (e.g., "The eagle flew high above the city"), followed by another comprehension question (e.g., "What flew?"), which the participant answered (e.g., "The eagle."). Participants were then prompted to recall the final word of each of the sentences (e.g., *lunch* and *city*). Participants performed five sets of these two-sentence presentations. If they responded correctly on at least three of the five sets, they moved on to three-sentence presentations. The task was discontinued if the participant failed to respond correctly on at least three of the five sets. All participants had near perfect comprehension (<1% of participants made any errors on a comprehension question), so it was not necessary to control for comprehension. The largest sentence set administered comprised 6 sentences. This task assessed maintenance (participants had to hold words online), updating (participants had to update the words they were holding online), and inhibition (participants had to inhibit the tendency to recall the words generated in response to the comprehension question as opposed to the final word of the sentences). We scored the test as the "span" length, where every correct sentence set received a score of 0.2. Thus, if a person completed 4 sets at length 2 correctly, and 2 sets at length 3 correctly, their score would be $(0.2 \times 4) + (0.2 \times 2) = 1.2$. We also computed the percentage of errors that were omission errors (a participant said he or she could not remember a word, e.g., recalled only *lunch*, but not *city*), perseverative (intrusion) errors (a participant gave a word that had been generated in response to a comprehension question rather than a final word in a sentence, e.g., recalled *hamburger* as a final word of a sentence), semantic errors (a participant gave a semantically related word to the final word in a sentence, e.g., *dinner* rather than *lunch*), or unrelated errors (a participant gave a word that was unrelated to any words in the sentence set). An individual could have more than one error type (e.g., an individual who generated *dinner* would have one omission error and one semantic error).

Test of Semantic Memory

We hypothesized that the WM deficits of some of the patients with AD (e.g., on the reading span task) could be related to their semantic memory impairments. In addition to category fluency, which provides one measure of semantic memory, we also administered the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1978; Huff, Collins, Corkin, & Rosen, 1986).

Participants were given two forms of this test; each form had 42 black-and-white line drawings of objects. Participants were asked to name each object. The Boston Naming Test scores for the patients with AD and the patients with PD were from a neuropsychological test series administered to all patients in a separate testing session, within 1 month of the administration of the WM tasks.

Results

Inhibitory Ability

Analysis of variance (ANOVA) indicated significant group effects on all three tests of inhibitory ability. For the Stroop interference score, the group effect was significant, $F(2, 151) = 8.50$, $p < .001$, and post hoc t tests indicated that the participants with PD performed significantly worse than CON participants, $t(130) = 1.68$, $p < .05$. On the go/no-go task, ANOVA indicated a group effect, $F(2, 151) = 6.72$, $p < .001$, and t tests showed that the group with PD again performed less well than the CON participants, $t(130) = 2.41$, $p < .01$. On fluency perseverations, the group effect, $F(2, 151) = 8.73$, $p < .001$, was also found to be dominated by the poor performance of the group with PD as compared with the group with AD, $t(40) = 3.35$, $p < .001$, or with CON participants, $t(130) = 3.24$, $p < .001$; see Table 1). The patients with AD did not show impairments on any measures of inhibitory ability.

Table 1
Scores for Each of the Administered Cognitive Tasks as a Function of Group

Cognitive function	Tasks	CON (n = 112)		AD (n = 22)		PD (n = 20)	
		M	*SD*	*M*	*SD*	*M*	*SD*
Short-term memory	Digit span (highest span achieved)	7.0	1.2	6.4	1.2	6.7	1.0
	Word span (highest span achieved)	5.1	0.8	4.8	1.3	4.7	0.8
Inhibitory ability	Stroop task						
	Stroop, Card 1 (total no. of words read)	98	14.7	85	13.7	88	15.8
	Stroop, Card 2 (total no. of colors named)[a]	64	14.4	49	13.5	63	12.7
	Stroop, Card 3 (total no. of colors named)[b]	36	10.1	26	11.4	25	10.4
	Stroop interference[c]	97	7.5	94	6.3	89	13.0
	Category fluency						
	No. of correct category exemplars[a]	14.8	2.6	10.6	5.1	13.8	2.5
	% responses that were perseverations[d]	1.5	1.7	1.5	1.3	3.8	4.1
	Go/no-go accuracy[c]	89	12.2	85	12.0	78	15.1
Working memory	2-back						
	% hits[c]	88	15.2	84	14.0	76	15.3
	% false alarms	9	16.7	11	11.3	12	18.1
	Corrected hit rate (% hits − % false alarms)[c]	80	18.3	72	15.2	63	19.7
	Reading span[e]	2.3	0.82	1.3	0.59	1.7	0.57

Note. CON = control; AD = Alzheimer's disease; PD = Parkinson's disease.
[a] Patients with AD performed less well than CON participants and patients with PD ($p < .01$). [b] Patients with AD and patients with PD performed less well than CON participants ($p < .05$). [c] Patients with PD performed less well than CON participants ($p < .01$). [d] Patients with PD performed less well than CON participants and patients with AD ($p < .01$). [e] Patients with AD performed less well than CON participants and patients with PD ($p < .01$); patients with PD performed less well than CON participants ($p < .05$).

Relation Between Inhibitory Ability and Short-Term Memory

To assess the relation between inhibitory ability and short-term memory, we computed correlations to see whether the two abilities were associated. We found no significant correlations between short-term memory tasks (Digit Span and word span) and measures of inhibition (go/no-go accuracy, fluency perseverations, Stroop interference) for the group with PD, the group with AD, or the CON group ($r < .35$).[1] ANOVAs indicated no group differences on the tests of verbal short-term memory (Digit Span and word span; see Table 1).

Standard Tests of WM

The next question we sought to address was how patients with AD and patients with PD performed on tasks that are commonly used to assess WM.

The 2-Back Task

On the 2-back task, ANOVA conducted on the corrected hit rate (percentage of hits − percentage of false alarms) indicated a significant effect of group, $F(2, 151) = 4.81$, $p < .01$, and t tests indicated that the patients with PD performed significantly less well than CON participants, $t(130) = 2.39$, $p < .01$. When hit and false-alarm rates were analyzed separately, ANOVA indicated an effect of group for the hits, $F(2, 151) = 4.50$, $p < .01$, but no effect for the false alarms ($F < 2$). We performed t tests, which indicated that the patients with PD had significantly fewer hits than the CON participants, $t(130) = 2.56$, $p < .01$, but did not differ in their false-alarm rate ($t < 1$). The group with AD performed similarly to CON participants on all measures, $t(132) = 1.05$, $p > .15$.

We then examined whether performance on tests of inhibition, or tests of short-term memory, correlated with performance on the 2-back task for the group with AD and the group with PD. We found that for the group with PD, the hit rate correlated significantly with performance on all three tests of inhibition (correlation with go/no-go, $r = .44$, $p < .05$; with Stroop score, $r = .57$, $p < .01$; with fluency perseverations, $r = -.45$, $p < .05$). False-alarm rate, in contrast, correlated with a test of verbal short-term memory (correlation with Digit Span, $r = -.45$, $p < .05$).

The group with AD showed no deficit on the 2-back task. Their false-alarm rate correlated with measures of verbal short-term memory (correlation with Digit Span, $r = -.64$, $p < .001$; word span, $r = -.39$, $p < .10$). Thus, as with the group with PD, false alarms (essentially errors in maintenance or updating) on the 2-back task were correlated with short-term memory ability as assessed on other tasks. Unlike the group with PD, the hit rate of the group with AD did not show significant correlations with measures of inhibitory ability ($r < .25$, $p > .2$). Thus, the difficulty in inhibiting the tendency to hit the "nonmatch" button appeared to be a problem specific to the group with PD.

[1] A correlation between a short-term memory composite (Digit Span and word span) and an inhibition composite (consisting of scores from go/no-go accuracy, Stroop interference, and category fluency perseverations) was also nonsignificant for the group with AD, the group with PD, and the CON group (all rs $< .20$).

Reading Span Task

On the reading span task, ANOVA indicated a significant effect of group, $F(2, 151) = 17.30$, $p < .0001$, and t tests indicated that the group with AD performed less well than CON participants and the group with PD, $t(130) = 2.61$, $p < .01$, and the group with PD also performed less well than CON participants, $t(132) = 1.73$, $p < .05$. We, therefore, wanted to assess whether the two groups' deficits on this task were related to similar cognitive abilities. Given the known deficits of patients with AD in semantic access, and the deficits of patients with PD in inhibitory ability (and the task's reliance on both of these capacities), it seemed possible that the patients with AD were impaired on this task because of difficulties related to semantic retrieval, whereas the patients with PD may have shown impairment due to deficits in inhibition.

One analysis compared the error patterns of the patients with AD and those with PD. We broke down the errors into four categories: omission, perseveration, unrelated, and semantic associate. For each person, we computed the percentage of errors of each type (see Table 2). Repeated-measures ANOVA with error type as a within-subject factor and group as a between-subjects factor confirmed that the two groups showed different error profiles: We found a significant effect of error type, $F(3, 120) = 3.7$, $p < .01$; no effect of group; and a significant Group × Error Type interaction, $F(3, 120) = 11.2$, $p < .0001$. Independent samples t tests confirmed that the group with AD had a greater proportion of errors of the semantic type, $t(40) = 4.45$, $p < .001$, than the group with PD, whereas the group with PD had a greater proportion of errors of the perseverative type, $t(40) = 5.56$, $p < .001$, than the patients with AD. The two groups did not differ in terms of their proportion of errors that were of the omission or unrelated type, $t(40) < 1$. These results suggest that semantic memory impairments may contribute more to the performance of patients with AD on the reading span task, whereas impairments in inhibitory ability may underlie the impaired performance of the group with PD.

Another way in which we analyzed the possible contributions of semantic memory and inhibition to reading span performance was by computing correlations between participants' performance on reading span and measures of short-term memory (Digit Span, word span), inhibition (go/no-go, Stroop interference, fluency perseverations), and semantic memory (the Boston Naming Test, category fluency). We found that for the patients with AD, scores on reading span correlated with measures of short-term memory (correlation with word span, $r = .55$, $p < .01$; with Digit Span, $r = .39$, $p < .10$) and with measures of semantic retrieval (correlation with the Boston Naming Test, $r = .89$, $p < .0001$; with category fluency, $r = .68$, $p < .0001$). This result suggests that for the group with AD, performance on the reading span task is related not only to the ability to store information but also to the ability to access semantic information. For the group with PD, reading span scores correlated with measures of short-term memory (correlation with Digit Span, $r = .55$, $p < .01$; word span, $r = .42$, $p < .10$) but not with measures of semantic retrieval or inhibitory ability ($r < .35$, $p > .10$). Thus, the correlation with semantic access may appear only in subpopulations that have deficits with semantic memory; in people with little semantic memory deficit, reading span appears to be most related to measures of verbal short-term memory. This view is consistent with the finding that the errors of the group with AD on the task predominantly resulted from the inability to retrieve any word or the retrieval of an incorrect associate.

Discussion

The first goal of this study was to examine how mild AD and early PD affect inhibitory ability. This investigation indicated that the ability to inhibit automatic or prepotent responses is disproportionately affected in early PD as compared with mild AD. Patients with PD were impaired on all tests of inhibitory ability, whereas patients with AD showed no deficits on the measures of inhibition when compared with CON participants. We return to this finding below.

The second goal was to examine whether inhibitory ability correlated with performance on tasks of short-term memory in either of the patient populations. Researchers have posited that inefficient inhibition can ostensibly impair short-term memory by cluttering the store with irrelevant information, thus leaving less space for task-relevant information (Engle, 1996). We did not find a significant correlation between these two processes. These results suggest that although inhibitory deficits can be linked to reductions in short-term memory capacity (Engle, 1996; Hasher & Zacks, 1988), inhibitory deficits in some cases may exist without exerting an effect on short-term memory capacity. This null finding must be interpreted with caution, however, because it could represent methodological features of our

Table 2
Reading Span: Total Number of Errors Made, and the Proportion of Errors by Type, as a Function of Patient Group

	No. of errors		Error type							
			Perseverative		Semantic		Omission		Unrelated	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
AD	11.5	6.6	6.4	10.2	39.1	24.4	39.1	31.9	15.4	18.9
PD	14.4	8.2	47.8	33.3	10.3	16.3	29.8	31.1	12.2	24.3

Note. AD = Alzheimer's disease; PD = Parkinson's disease.

study (e.g., low variability in short-term memory scores, relatively small sample sizes) rather than an independence between the constructs.

The third goal of the study was to investigate how patients with mild AD and those with early PD would compare on standard WM tasks that require a host of cognitive processes. Only the group with PD was impaired on the 2-back task. Patients with PD were also impaired on the reading span task, as compared with CON participants, although patients with AD were most impaired on this task, performing less well than patients with PD and CON participants. The different pattern of impairment on the two tests (i.e., patients with PD as the lowest performing group on the 2-back and patients with AD as the lowest performing group on the reading span task) suggests that the group effects are not merely due to task difficulty or task sensitivity. Rather, this dissociation of impairment suggests that the deficits result from different core deficits in each group. Indeed, the results of our analyses suggest that the WM deficits in the group with PD may be more associated with deficits in inhibition than is the case with the group with AD. In contrast, the semantic memory impairments of patients with AD may contribute to their WM deficits.

Inhibitory Ability Contributes to WM Deficits in Early PD

The patients with PD were impaired on all tests of inhibition, and on both administered WM tasks, their inhibition deficit appeared to affect their performance. On the 2-back task, their hit rate was correlated with inhibitory ability. We suggest that this correlation may have arisen because matches occurred less frequently than nonmatches. Thus, participants may have gotten into a "routine" of hitting the nonmatch button and may have needed to overcome this tendency when a match was presented. In the case of the patients with PD, then, those who were better able to inhibit the prepotent response (hitting the nonmatch button) had a greater percentage of hits. On the reading span task, the most common type of error for the patients with PD was a perseverative error. Again, this result suggests that a dominant contributor to the performance of the PD group was their inability to inhibit inappropriate responses: motor (in the case of the 2-back) and verbal (in the case of the reading span task).

This inhibitory deficit likely results from disruption of the striatal–prefrontal dopaminergic projections thought to be particularly important for selective attention. As proposed in an influential model of control (Casey, Durston, & Fossella, 2001; Casey, Tottenham, & Fossella, 2002), the basal ganglia and prefrontal cortex may both play critical roles in inhibition. The basal ganglia may inhibit inappropriate thoughts or actions (Mink, 1996). The prefrontal cortex may serve as the locus of top-down control that sorts task-relevant from task-irrelevant information and maintains the relevant information in the face of distractors (Desimone & Duncan, 1995; Miller & Cohen, 2001). Dopaminergic projections from prefrontal regions may provide the basis for this allocation of attention by potentiating synapses associated with a reward (e.g., correct recall) and thereby reducing the interference from other, less activated, networks (Schultz, Dayan, & Montague, 1997). This interpretation is consistent with a range of neuroimaging and neuropsychiatric data implicating the prefrontal cortex and basal ganglia in selective attention and inhibition (see Casey et al., 2002, for a review).

The inhibition impairment of the early PD group is consistent with the deficits seen in patients with more advanced PD. These patients show impairments on tasks requiring inhibitory ability, such as the Stroop task (Brown & Marsden, 1988), random generation task (Brown, Soliver, & Jahanshahi, 1998; Robertson, Hazlewood, & Rawson, 1996; Spatt & Goldenberg, 1993), Wisconsin Card Sorting Test (Beatty & Monson, 1990), and others (Dalrymple-Alford, Kalders, Jones, & Watson, 1994; Hsieh, Chuang, Hwang, & Pai, 1998; Hsieh, Lee, & Tai, 1995; Owen et al., 1993). The results of this study underscore the fact that an inhibitory deficit is present not only in advanced stages of PD but also in the early stages. Throughout the course of the disease, deficits in the ability to inhibit inappropriate responses appear to affect the cognitive performance of individuals with PD.

Semantic Memory Ability Contributes to WM Deficits in Mild AD

AD results in alterations in the semantic memory system. The structure of the semantic system and the ability to access semantic information may be affected (Grossman, Mickanin, Robinson, & D'Esposito, 1996; see Carlesimo & Oscar-Berman, 1992, and Tippett, McAuliffe, & Farah, 1995, for reviews). AD can lead to pronounced word-finding difficulties (Astell & Harley, 1996), naming deficits (Huff et al., 1986; Ripich, Petrill, Whitehouse, & Ziol, 1995), and impaired performance on some tasks of implicit semantic memory (Glosser, Friedman, Grugan, Lee, & Grossman, 1998). Deficits in this area are a critical component of AD, and performance on semantic memory tests has been found to be one of the most accurate ways to stage the progression of AD (Locascio et al., 1995).

This semantic memory impairment likely results from the damage to the temporal neocortex that is present even early in AD and that increases with disease progression (Jagust et al., 1993; Nagy et al., 1999). This hypothesis is consistent with studies of patients with circumscribed temporal-lobe lesions, showing that medial temporal-lobe structures are not necessary for normal retrieval from semantic memory (Kensinger, Ullman, & Corkin, 2001) but that lateral temporal neocortex probably is (Schmolck, Kensinger, Corkin, & Squire, 2002; Schmolck, Stefanacci, & Squire, 2000).

The results of our study suggest that the disruption in semantic memory also can affect other cognitive domains, including WM performance. The performance of patients with AD on the reading span task correlated significantly with their performance on semantic memory tasks. In addition, their errors on the reading span task consisted of a large proportion of semantic errors. Thus, what on the surface appears to be a WM deficit (impaired performance

on the reading span task) may actually be due not only to capacities typically thought of as related to WM (e.g., short-term memory) but also to the patients' ability to process and access semantic information.[2]

Conclusion

In conclusion, we found that early PD alters inhibitory processing, likely through alteration of striatal dopaminergic projections. This inhibitory deficit can contribute to poor performance on WM measures, such as the 2-back and reading span task. Mild AD, in contrast, relatively spares inhibitory ability. Deficits seen in the group with AD appeared, instead, to be related to alterations in semantic memory, likely resulting from changes in temporal neocortex and cholinergic function, rather than to attention deficits.

[2] The semantic deficits in the patients with AD may even have contributed to their verbal short-term memory performance. Although the group with AD was not impaired on the word span task as compared with CON participants, their performance on this short-term memory test did correlate with their performance on tests of semantic memory. Perhaps the patients with AD who are less able to access semantic information about words presented on the word span list have more difficulties with effective chunking, and thus lower word spans.

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