

Memory Deficits in Parkinson's Disease

CRAIG J. WHITTINGTON,¹ JOHN PODD,² AND
STEVE STEWART-WILLIAMS³

¹University College London, Sub-Department of Clinical Health Psychology,
London

²Massey University, School of Psychology, New Zealand

³McMaster University, Department of Psychology, Neuroscience, and
Behaviour, Hamilton, Ontario, Canada

The purpose of this study was to investigate deficits in recognition, recall, and prospective memory among Parkinson's disease (PD) patients, and to ascertain whether task difficulty and disease severity moderate these deficits. Comparisons were made between 41 nondemented PD participants, divided into early-stage and advanced-stage groups, and 41 matched controls. PD participants exhibited deficits in recognition, recall, and prospective memory. The advanced-stage PD group produced greater deficits than the early-stage PD group in all tasks, suggesting that these deficits increase in step with overall disease severity. The results of the task difficulty manipulation provide a partial explanation for the inconsistencies in the literature concerning the existence of recognition memory deficits in PD.

Introduction

Until relatively recently, the sole focus of research in Parkinson's disease (PD) was the motor symptoms associated with this neurodegenerative disorder. In the last few decades, however, it has become increasingly apparent that PD involves a variety of cognitive impairments. Among these are impairments in declarative memory processes. The present article reports some of the findings of a large-scale study examining deficits in recognition, recall, and prospective memory among PD patients. The article begins with a discussion of the various memory deficits found in PD. Particular attention is paid to recognition memory, as there is a widespread belief that PD involves little or no impairment in this form of memory. Following this, we turn to the issue of the moderators of memory impairment in PD, including task difficulty and disease severity.

Recognition and Recall in PD

Although there is some consensus that PD involves cognitive impairments, the nature of these impairments is controversial. On the one hand, there is good evidence for deficits in

Received 9 June 2004; accepted 26 August 2004.

This research was supported by the Massey University Research Fund Grant 1-0575-67302A, the Massey University Research Equipment Fund Grant 1-0575-98025, and the School of Psychology at Massey University. Parts of this article were presented in 2000 at the annual conference of the British Psychological Society, Winchester, England.

Address correspondence to John Podd, Massey University, School of Psychology, Palmerston North, New Zealand. E-mail: j.v.podd@massey.ac.nz

recall memory among nondemented PD patients.¹ These deficits affect both verbal and nonverbal recall memory, and appear to be independent of antiparkinsonian medication (Brown & Marsden, 1990; Cooper, Sagar, & Sullivan, 1993; Sahakian et al., 1988). Beyond this, however, there is less agreement. It is widely held that, although recall is impaired in people with PD, recognition memory is relatively normal (Breen, 1993; Brown & Marsden, 1988; Emre, 2003; Ivory, Knight, Longmore, & Caradoc-Davies, 1999; Knight, 1992; Shibuya, Tachibana, Kawabata, & Sugita, 2001; Taylor, Saint-Cyr, & Lang, 1988). Three studies (Flowers, Pearce, & Pearce, 1984; Lees & Smith, 1983; Taylor, Saint-Cyr, & Lang, 1986) appear to have been instrumental in establishing this view. However, the research concerning recognition memory is inconsistent, with various studies reporting some form of impaired recognition in nondemented PD participants (Barnes, Boubert, & Harris, 2003; Minamoto, Tachibana, & Sugita, 2001; Owen et al., 1992; Sahakian et al., 1988; Stebbins, Gabrieli, Masciari, Monti, & Goetz, 1999; Woods & Troester, 2003).

In an effort to resolve this issue, Whittington, Podd, and Kan (2000) conducted an analysis of the statistical power of studies investigating PD-related recognition memory deficits. They discovered that the power of the relevant research has generally been too low to reliably detect small- to medium-size effects. For instance, in 48 studies investigating memory functioning in PD, the mean power to detect small effects was just 20%. This provides at least a partial explanation for the discrepancies in the literature concerning the existence of deficits in recognition memory in PD: Underpowered studies increase the likelihood that researchers will conclude that there is no population effect when in fact there is one (a Type II error; Schmidt, 1996). (This is the case, at any rate, if researchers rely solely on statistical significance testing, as we discuss later.) To determine whether there really is a population effect, Whittington et al. (2000) ran a meta-analysis of studies investigating recognition deficits in PD. They found that small deficits in recognition memory do occur in nondemented PD patients. Thus, the view that PD involves relatively intact recognition memory may largely be an artifact of underpowered studies. One goal of the present study was to add to the literature investigating recognition memory in PD.

Prospective Memory in PD

In addition to deficits in recognition and recall, there is some evidence for PD-related deficits in prospective memory. Prospective memory involves remembering to execute a planned action at some point in the future. Einstein and McDaniel (1990) distinguished two forms of prospective memory task: time-based tasks (e.g., remembering to turn on the oven at 5 o'clock) and event-based tasks (e.g., remembering to turn on the oven when an alarm rings). Little research has investigated prospective memory deficits in PD. This is surprising, first, because of the prevalence in everyday life of activities that require this capacity (Katai, 1999), and second, because prospective memory is likely to be subserved by the frontal lobes and their connections to other cortical and subcortical areas, areas thought to be impaired in PD (Bondi, Kaszniak, Bayles, & Vance, 1993; McDaniel, Glisty, Rubin, Guynn, & Routhieaux, 1999). Furthermore, prospective memory is likely to involve both the memory functions and the planning abilities usually associated with the central executive in working memory, which are also typically impaired in PD (Katai, Maruyama, Hashimoto, & Ikeda, 2003).

¹Participants were assumed to be nondemented if they scored in the normal range on the Orientation-Memory-Concentration Test (OMCT; Katzman et al., 1983).

Those few studies that have investigated prospective memory in PD have found that it is indeed impaired. In a study of deficits in everyday memory, Katai (1999) found that PD participants performed notably worse than healthy controls on subtests of the Rivermead Behavioral Memory Test related to prospective memory. A later study by Katai et al. (2003) focused specifically on event-based and time-based prospective memory. The PD participants were impaired in the event-based tasks, although not the time-based tasks. In light of the paucity of research findings on prospective memory deficits in PD, a further goal of the present study was to replicate some of the findings of Katai et al. (2003).

Variables Moderating Memory Deficits in PD

Several variables may moderate the memory deficits in PD. Among these are task difficulty and disease severity. Task difficulty has been used to explain the fact that PD patients tend to perform less well on recall tests than they do on recognition tests (Breen, 1993; Knight, 1992; Stebbins et al., 1999; Taylor et al., 1988; Weingartner, Burns, Diebel, & LeWitt, 1984). The idea is that recognition tests require less effort, less self-initiated activity, or less processing resources than recall tests (Brown & Marsden, 1990; Craik & McDowd, 1987; Hasher & Zacks, 1979).² Consistent with this view, previous research hints at an association between task demands and memory impairment in PD, such that tasks that are more difficult produce greater deficits (Weingartner et al., 1984). The present study sought to confirm this association. To the best of our knowledge, no one has yet directly tested the impact of task difficulty on recognition memory performance in PD participants. In the present study, we varied *within-task* difficulty. In this way, we were able to avoid the potential confounding of *type of task* with *level of difficulty* when both are simultaneously changed.

Another potential moderator of memory deficits in PD is disease severity. Although the relationship between disease severity and cognitive decline is not straightforward (Berger et al., 1999; Starkstein & Robinson, 1991), there is some evidence that disease severity is related to memory dysfunction (Huber, Freidenberg, Shuttleworth, Paulson, & Christy, 1989; Mortimer, Pirozzolo, Hansch, & Webster, 1982). For instance, individuals in the advanced stages of PD show greater deficits in recognition memory than do those in the early stages. Sahakian et al. (1988) found that nondemented PD patients with advanced symptoms performed significantly worse in a pattern recognition task than a matched control group, but that controls and mild PD patients did not differ. The Whittington et al. (2000) meta-analysis further supported the possibility that deficits in recognition memory increase in step with disease severity. Specifically, they found that only those PD patients who were medicated showed a deficit in recognition memory. At first glance, this would appear to suggest that the medication caused the deficit. This is unlikely, however. There is research suggesting that dopaminergic medication has no effect on memory performance, and can even improve it (e.g., Cooper, Sagar, Doherty, Jordan, Tidswell, & Sullivan, 1992; Cooper et al., 1993; see Whittington et al., 2000, for further discussion). Thus, it is more likely that medicated PD patients exhibited greater deficits on the recognition task because these patients tend to have more advanced PD. One goal of the present study was to build on the previous research by seeking to confirm this speculation. A further goal was to determine whether symptom severity also moderates deficits in recall and prospective memory in PD. To date, there has been little research directly examining the impact of disease severity on these different types of memory.

²A similar issue has been discussed in the literature on age-related decline in memory (e.g., Craik & McDowd, 1987) and memory deficits in schizophrenia (e.g., Calev, 1984).

Hypotheses

Several hypotheses derive from the foregoing discussion:

Hypothesis 1. Recognition memory will be impaired in PD patients, especially at the harder level of task difficulty. This hypothesis derives from the Whittington et al. (2000) power analysis and meta-analysis, which challenged the view that recognition memory is relatively intact in PD. It also derived from the observation that the recognition-recall dissociation may be a product of differential task difficulty.

Hypothesis 2. Recognition memory will be impaired in PD, with greater impairment associated with more advanced stages of the disease. This hypothesis derives from the fact that cognitive deficits in PD tend to increase in step with overall disease severity (e.g., Owen et al., 1992; Sahakian et al., 1988; Whittington et al., 2000).

Hypothesis 3. Recall will be impaired in PD, with greater impairment associated with more advanced stages of the disease. Like Hypothesis 2, this hypothesis derives from the speculation that cognitive deficits in PD increase in step with disease severity.

Hypothesis 4. Prospective memory will be impaired in PD patients relative to healthy controls. This hypothesis is based on past PD research (Katai, 1999; Katai et al., 2003).

Hypothesis 5. Deficits in prospective memory will be more pronounced in PD patients with greater disease severity. Again, this hypothesis derives from the speculation that the cognitive deficits in PD increase in step with disease severity.

In order to address these hypotheses, we examined recognition, recall, and prospective memory in nondemented PD participants. To examine the variable of task difficulty, we designed a nonverbal recognition memory task with two levels of difficulty. To examine the effects of symptom severity, we categorized the PD participants as early-stage or advanced-stage. Comparisons were made (with a complete replication six months later) with controls matched on sex, age, education, and premorbid IQ.

Method

Participants

Fifty-eight PD patients volunteered for the present study. On first contact, volunteers were screened via a telephone interview. Exclusion criteria included: a poor response to antiparkinsonian medication, any significant history of psychiatric or neurological disorder not related to their diagnosis (e.g., stroke or head trauma with loss of consciousness greater than one hour), past or present alcohol abuse or other drug abuse, neurosurgery, and any other serious medical condition. Seven individuals were excluded in light of these criteria. The remaining participants were screened for dementia using the Orientation-Memory-Concentration Test (OMCT; Katzman, T. Brown, Fuld, Peck, Schechter, & Schimmel, 1983).³ Three participants scoring greater than 10 on this instrument were excluded from further analysis. Finally, another seven participants were excluded from the final sample because they could not take part in the second testing session. The final sample consisted

³The OMCT is particularly suitable for use with PD participants, because it does not include a copying task that may be affected by the physical symptoms of PD independently of dementia.

of 41 PD patients (16 females and 25 males). The initial diagnosis of idiopathic PD had been made by each participant's General Practitioner, and in 93% of cases had subsequently been confirmed by a neurologist.

Symptom severity was assessed using the Hoehn and Yahr (1967) disability rating scale. There are a number of potential objections to the use of this scale that must be addressed. First, the scale is relatively coarse. However, as there were only 41 participants in the study, a scale with just four categories was sufficient. Second, the scale is not an ideal measure of severity, as it is a classification of disease symptoms, only some of which are associated with disease severity. Nonetheless, we chose the Hoehn and Yahr scale over other measures, such as the Unified Parkinson's Disease Rating Scale (Fahn, Elton, & Members of the UPDRS Development Committee, 1987), because it can be administered rapidly and efficiently. Another advantage of the scale is that it is relatively insensitive to motor fluctuations caused by drug therapy (Diamond & Markham, 1983). Nonetheless, an attempt was made to avoid problems caused by drug-induced side effects by testing participants at their time of optimal therapeutic response.

The pool of PD participants was divided into two groups based on the severity of their clinical symptoms. Participants were classed either as early-stage (Stage I or II) or as advanced-stage (Stage III or IV). A subset of the classifications was double checked by a neurologist; in every case, the neurologist's classification agreed with that of the researchers. The early-stage PD group consisted of 21 of the 41 PD participants (10 females, 11 males). Five of these participants had a Hoehn and Yahr rating of Stage I, and 16 had a rating of Stage II. At the time of testing, a majority (80%) of the early-stage PD participants was receiving L-Dopa preparations, and 29% were receiving anticholinergic medication. The mean age at symptom onset for the early-stage group was 59.64 years ($SD = 8.24$), and the mean duration of symptoms was 8.60 years ($SD = 6.34$). The advanced-stage PD group consisted of the remaining 20 participants (6 females, 14 males). Seventeen of these participants had a Hoehn and Yahr rating of Stage III, and three had a rating of Stage IV. At the time of testing, 95% of the advanced-stage PD participants were receiving L-Dopa preparations, and 20% were receiving anticholinergic medication. The mean age at symptom onset for the advanced-stage group was 61.35 years ($SD = 9.16$), and the mean duration of symptoms was 9.45 years ($SD = 5.08$).

Forty-one healthy control participants (18 females, 23 males) were chosen to match the PD participants as closely as possible with respect to sex, age, education, and premorbid IQ.⁴ The same exclusion criteria were applied to the control group as were applied to the experimental group. Table 1 presents a summary of characteristics of each group. Statistical analyses confirmed that both PD groups were well matched with the control group.

Materials and Procedure

The assessment procedures were conducted in the participants' homes. Each testing session began with an invitation for participants to read an information sheet and a brief overview of the tasks, and to sign a consent form. Several of the tasks were computer-administered. Computerized testing provides consistency and uniformity of stimulus presentation, and automates scoring which reduces the effects of experimenter expectancy (Youngjohn, Larrabee, & Crook, 1992). The positioning of the computer was adjusted to

⁴Premorbid IQ was estimated using the National Adult Reading Test (NART; Nelson & O'Connell, 1978). There is good evidence that this test of an individual's ability to pronounce a series of uncommon words serves as a useful indicator of premorbid intelligence (Christensen, Hadzi-Pavlovic, & Jacomb, 1991; Nelson & O'Connell, 1978; Ryan & Paolo, 1992).

Table 1

Characteristics of the early-stage Parkinson's disease (early-stage PD) group, the advanced-stage Parkinson's disease (advanced-stage PD) group, and the control group

Variable	Early-stage PD (<i>N</i> = 21)		Advanced-stage PD (<i>N</i> = 20)		Control (<i>N</i> = 41)	
	M	SD	M	SD	M	SD
Age (years)	68.24	6.19	70.80	5.82	69.80	5.31
Premorbid IQ	108.69	8.13	110.20	12.58	110.88	8.94
OMCT	3.52	3.68	4.35	2.96	2.39	2.38
L-Dopa ^a	436.11	202.91	407.89	239.98	–	–

Note. Premorbid IQ = Premorbid full-scale IQ (WAIS-R) estimated from National Adult Reading Test; OMCT = Orientation-Memory-Concentration Test; L-Dopa = mean daily levodopa dose in milligrams.

^a*N* = 18 (early-stage PD), 19 (advanced-stage PD).

suit the participant. Generally, the screen was centered approximately 0.5 m away from the seated participant at eye-level. Each participant was taken through a series of automated tasks using both standardized verbal instructions and instructions presented on the computer screen. Practice trials were given where necessary to allow participants to become familiar with the test procedures. If a participant required clarification of the task instructions, or appeared initially to misunderstand them, standardized verbal prompts were provided. The order of tasks was arranged to avoid interference from tasks of a similar nature and to minimize fatigue.

Participants were tested on two separate occasions. The intersession interval was approximately six months, with little difference between groups in the mean intersession interval. Every effort was made to keep the time of day constant between Time 1 and Time 2 for each participant.

Nonverbal Recognition Memory Task

Two recognition tasks were designed specifically for this study: a nonverbal task with two levels of difficulty, and a verbal task. Both tasks were computer-administered, and designed using Experimental RunTime System (ERTS) software (Beringer, 1995). ERTS software operates with millisecond accuracy to control response registration and event timing. Stimuli were presented in yellow on a blue background, using a 24 cm color screen. The participants' responses were registered using two large metallic keys mounted 8 cm apart in the center of a response console.

The first task, the Nonverbal Recognition Memory Task (NRMT), was based on a pool of 80 abstract stimuli, modeled on those used in the Embedded Figures Test (Witkin, Oltman, Raskin, & Karp, 1971).⁵ Half of the stimuli served as targets, and the remaining half were used as distracters. An effort was made to select stimuli that were difficult to encode verbally. Although no explicit attempt was made to examine the modality of encoding, post-test feedback indicated that most participants found it difficult to assign

⁵Modification and reproduction of stimuli from the Embedded Figures Test by Herman A. Witkin was by special permission of the publisher, Consulting Psychologists Press, Palo Alto, CA.

verbal labels to the stimuli. Two levels of difficulty were created within the NRMT. This was done by pairing half the targets with similar distracters and half with dissimilar distracters. The dissimilar distracters differed in terms of both form and color, whereas the similar distracters differed only in terms of form.⁶ A pilot study confirmed that the two levels of the task produced scores as predicted, and with scores above chance for both tasks.

The NRMT consisted of two phases: an exposure phase and a test phase. In the exposure phase, participants viewed a single list of 40 abstract target stimuli. The stimuli were presented one at a time on the computer screen at a rate of one every 3 s. The second (test) phase consisted of a two-alternative, forced-choice recognition test. Two figures appeared side by side in the center of the screen, with the target appearing equally often on the left or right, for a maximum of 6 s or until a response was made. Participants pressed the left key if they thought the stimulus on the left was the target, or the right key if they thought the stimulus on the right was the target. They were told that, if neither stimulus seemed familiar to them, they were to guess, with the proviso that they try to use both response keys equally often for guesses. The order of presentation of stimuli in both the exposure phase and the test phase was randomized for each participant.

Verbal Recognition Memory Task

The computer-administered Verbal Recognition Memory Task (VRMT) was also designed specifically for the present study. A pool of 80 nouns (three to six letters each; $M = 4.35$, $SD = .74$) was created, with half the words serving as targets and the remainder serving as distracters. Administration of the VRMT followed exactly the same procedure as that described for the NRMT. In the test phase, target words were paired with distracters of equal word length. There was little difference in the frequency of usage of the distracters ($M = 35.38$, $SD = 39.93$) versus the target words ($M = 28.63$, $SD = 44.49$), $t(38) < 1$.

Kendrick Object Learning Task

The Kendrick Object Learning Task (KOLT) was used to assess recall memory. Although originally designed for use as part of a battery to screen for dementia in the elderly, Kendrick (1985) suggested that it could also be used independently as a measure of recall. It seemed particularly suitable for the present study because it has two equivalent forms and is relatively quick to administer. It also has the advantage that raw scores can be converted to age-scaled quotients (based on a mean of 100 and a standard deviation of 15), thus avoiding the potential confounding effect of age. Administration and scoring of the KOLT was conducted in the manner outlined by Kendrick (1985). Briefly, the experimenter showed four large cards with pictures of everyday objects to participants. Each card had 10, 15, 20, or 25 objects and the time allowed for inspection of each card was based on a 3 s viewing time per object. After viewing a card, participants were asked to recall as many objects as possible, in any order.

Prospective Memory Tasks

The two event-based prospective memory tasks used in the present study were adapted from Huppert and Beardsall (1993), who demonstrated that these tasks are sensitive to mild forms of cognitive impairment in people with dementia. The first task was the Prospective

⁶A similar manipulation was used by Craik & Jennings, 1992.

Memory for a Question Task (PMQT). Participants were told that an alarm would sound in 5 min, at which point they should stop what they were doing and ask the experimenter two prescribed questions (e.g., "when will the next session be?"). If participants failed to ask both prescribed questions spontaneously within 15 s of the alarm, they were prompted with the question: "What were you supposed to do when the alarm sounded?" Participants scored one point for each question they recalled and an additional point for each response made without a prompt. The maximum score was four. Different questions were used at each of the two testing sessions.

The second prospective memory task was the Prospective Memory for an Object Task (PMOT). This involved remembering to ask for an object when the testing session concluded. Around a third of the way through the session, participants were asked to hand over a small personal belonging. They were told that, when the session finished, they were to ask that the item be returned, and to remember where it was put during the session (it was placed in one of several available equipment boxes). At the end of the session, it was made clear that the session was over and the equipment was packed away. If the participant did not ask for the item during this time, they were prompted with the question: "Was there something you were going to ask for now that we have finished?" Participants scored one point for remembering to ask for the object, and another if they remembered the location of the object. They also received an additional point for each response made without a prompt. Again, the maximum score was four. Note that the PMOT involved a longer time delay than the PMQT.

Results

Our original hope was to document deterioration in cognitive performance across the two testing sessions separated by a six-month period. However, preliminary analyses indicated that there were no significant interaction effects involving the variable of testing session. Consequently, we combined the results from the two sessions.

The main analyses were planned comparisons using one-way ANOVAs. In addition to traditional tests of significance, the results were interpreted by examining point estimates of effect size (ES) and their 95% confidence intervals. The Whittington et al. (2000) meta-analysis (described in the Introduction) highlighted the danger of relying solely on statistical significance testing when using individual studies to assess the existence of specific cognitive deficits in PD. Small-sample studies often have insufficient power to detect small or medium effects, which opens the door to Type II errors. For this reason, in the interpretation of individual studies, Schmidt (1996) recommends de-emphasizing statistical significance and focusing instead on ESs, which are likely to provide a more reliable estimate of the true population effect (Wilkinson et al., 1999). Although greater confidence can be placed in a result that is statistically significant, results that are only marginally significant should not be discarded if the size of the effect is reasonable. We use Cohen's (1988) *d* statistic as a measure of ES, and adopt his guidelines for interpreting this statistic: small: $d = .20$, medium: $d = .50$, large: $d = .80$. When an ES for a between-group comparison was equal to or greater than $.20$, it was judged potentially important, even if the result was only marginally significant.

To provide additional tests of the relationship between memory and disease severity, correlations were calculated between scores on the various memory tasks and the Hoehn and Yahr stage. Correlations were determined with the Pearson product-moment coefficient, r .⁷

⁷It might be objected that, because the Hoehn and Yahr scale is not a true interval scale, Pearson product-moment correlations could provide misleading results. However, when the equivalent non-parametric correlations were calculated, the same pattern of results emerged.

Again, Cohen's (1988) conventions were used to interpret the size of the effects: small: $r = .10$; medium: $r = .30$; large: $r = .50$.

Hypothesis 1

The first hypothesis was that recognition memory would be impaired in PD patients relative to controls, particularly at the more difficult level of the task. The dependent variable used to assess this hypothesis was the percentage of correct responses on the NRMT. Looking first at the easy level of the NRMT, an ANOVA revealed that the combined early-stage and advanced-stage PD participants had poorer nonverbal recognition abilities ($M = 69.21$, $SD = 9.95$) than did the controls ($M = 72.73$, $SD = 8.32$), $F(1, 80) = 3.03$, $p = .09$, $d = .38$ (95% CI = $-.06 - .83$). This result was only marginally significant. However, the ES indicated a small-to-medium effect and it is quite possible that the result would have been significant given a larger sample size. Thus, the result should be judged potentially important. Clearer evidence was found on the harder level of the NRMT. Again, the combined early-stage and advanced-stage PD participants had poorer performance ($M = 62.07$, $SD = 9.77$) than did the controls ($M = 66.79$, $SD = 8.84$). However, in this case the result was significant, $F(1, 80) = 5.26$, $p = .02$, $d = .51$ (95% CI = $.06 - .95$). Overall, these results confirm Hypothesis 1. For both levels of difficulty on the NRMT, PD patients performed worse than controls, with small-to-medium deficits on the easy level and medium deficits on the harder level.

Hypothesis 2

The next two hypotheses dealt with the possible moderating effect of disease severity in PD-related memory deficits. Hypothesis 2 stated that deficits in recognition memory would be more pronounced in PD participants with greater disease severity. The dependent variables used to assess this hypothesis were the percentage of correct responses on the NRMT and the percentage of correct responses on the VRMT. The results are presented in Table 2. We begin with the nonverbal recognition task. (Note that performance was above chance for all groups at both levels of difficulty on the NRMT, suggesting that the difficult level of the task was not unduly difficult.) On the easy level of the NRMT, the early-stage PD group produced normal performance relative to the control group, $F < 1$. In contrast, the advanced-stage PD group were notably impaired on the easy level, with an ES approaching a large effect, $F(1, 59) = 7.58$, $p = .008$, $d = .75$ (95% CI = $.22 - 1.28$). A different pattern emerged for the harder level of the task: Both the early-stage PD group and the advanced-stage PD group performed worse than the controls, $F(1, 60) = 1.59$, $p = .21$, $d = .34$ (95% CI = $-.17 - .85$), and $F(1, 59) = 6.30$, $p = .02$, $d = .69$ (95% CI = $.16 - 1.21$), respectively. The result for the early-stage PD group was not significant, but the ES was small-to-medium and it can therefore be judged potentially important. In comparison, the advanced-stage group had a significant, medium-to-large deficit. Thus, for both levels of the task, the advanced-stage PD group exhibited greater deficits than the early-stage PD group. Note that the advanced-stage group had medium-to-large deficits regardless of the level of task difficulty.

A similar pattern of results was found for verbal recognition memory, as measured by the percentage of correct answers on the VRMT (see Table 2). Both the early-stage PD group and the advanced-stage PD group were impaired relative to the control group, $F(1, 60) = 2.90$, $p = .09$, $d = .45$ ($-.07 - .96$), and $F(1, 59) = 7.93$, $p = .007$, $d = .77$ ($.19 - 1.24$), respectively. The early-stage PD participants exhibited

Table 2

Mean scores on the recognition, recall, and prospective memory tasks for the early-stage Parkinson's disease (early-stage PD) group, the advanced-stage Parkinson's disease (advanced-stage PD) group, and the control group

Variable	Early-stage PD (<i>N</i> = 21)		Advanced-stage PD (<i>N</i> = 20)		Control (<i>N</i> = 41)	
	M	SD	M	SD	M	SD
NRMT Easy (PC ^a)	72.62	6.85	65.62	11.51	72.73	8.32
NRMT Hard (PC)	63.90	7.87	60.15	11.32	66.79	8.84
VRMT (PC)	81.57	7.73	79.21	7.23	85.02	7.73
KOLTQ	95.79	15.99	91.80	8.00	104.50	9.73
PMQT	2.84	0.80	2.00	1.23	3.09	0.88
PMOT	3.43	0.66	3.18	0.69	3.42	0.66

Note. NRMT = Nonverbal Recognition Memory Task; VRMT = Verbal Recognition Memory Task; KOLTQ = Kendrick Object Learning Task – age-scaled quotients; PMQT = Prospective Memory Question Task; PMOT = Prospective Memory Object Task.

^aPC = percentage correct.

small-to-medium deficits in verbal recognition, whereas the advanced-stage PD participants exhibited medium-to-large deficits. Again, the ANOVA for the early-stage PD group is only marginally significant, but the ES indicates a potentially important result. Overall, the planned comparisons for both verbal and nonverbal recognition memory support Hypothesis 2.

To further evaluate the relationship between recognition memory and disease severity, correlations were calculated between each of the recognition memory scores and the Hoehn and Yahr stage. As Table 3 shows, disease stage was negatively correlated with scores on the NRMT and on the VRMT. Although only the NRMT result was significant, the correlations for both recognition memory tasks were small-to-medium

Table 3

Zero-order correlations between the Hoehn and Yahr (H & Y) stage and scores on the recognition, recall, and prospective memory tasks

Variable	H & Y stage	
	<i>r</i>	95% CI ^a
NRMT ^b	-.32*	-.62 – -.02
VRMT	-.22	-.50 – .06
KOLTQ	-.05	-.33 – .24
PMQT	-.34*	-.60 – -.08
PMOT	-.35*	-.61 – -.09

Note. NRMT = Nonverbal Recognition Memory Task; VRMT = Verbal Recognition Memory Task; KOLTQ = Kendrick Object Learning Task – age-scaled quotients; PMQT = Prospective Memory Question Task; PMOT = Prospective Memory Object Task.

^a95% confidence interval of *r*(*N* = 41). ^bAveraged across difficulty level.

**p* < .05.

in magnitude, providing further support for Hypothesis 2. Taken together with the results of the ANOVAs, this provides strong evidence that recognition memory (both verbal and nonverbal) deteriorates in step with increases in overall disease severity.

Hypothesis 3

Hypothesis 3 stated that recall memory would be impaired in PD, and that greater impairment would be associated with greater disease severity. The dependent variable used to assess this hypothesis was the age-adjusted KOLT score. As Table 2 shows, both the early-stage PD group and the advanced-stage PD group had significantly impaired recall relative to the control group, $F(1, 60) = 7.11, p = .01, d = .72$ (.19 – 1.24), and $F(1, 59) = 25.58, p < .001, d = 1.38$ (.81 – 1.95), respectively. Furthermore, as predicted, the impairment was greater in the advanced-stage PD group: Advanced-stage PD participants had large deficits in recall memory, whereas early-stage PD participants had medium-to-large deficits. To further evaluate the relationship between recall memory and disease severity, a correlation was calculated between the recall memory scores and the Hoehn and Yahr stage. Contrary to our expectations, disease stage was not significantly correlated with scores on the KOLTQ and the ES was negligible (see Table 3).

Hypothesis 4

The next two hypotheses dealt with prospective memory. Hypothesis 4 stated that prospective memory would be impaired in PD participants relative to healthy controls. For the PMQT, the combined early-stage and advanced-stage PD participants had significantly poorer performance ($M = 2.43, SD = 1.10$) than did the controls ($M = 3.09, SD = 0.88$), with the ES indicating a medium effect, $F(1, 80) = 9.16, p = .003, d = .67$ (95% CI = .22 – 1.12).⁸ In contrast, there was no evidence of a deficit on the PMOT, $F < 1$. Thus, there was support for a PD-related deficit in prospective memory, but only on the PMQT.

Hypothesis 5

It is important to note, though, that this result is qualified by planned comparisons examining the relationship of disease severity and prospective memory. Hypothesis 5 stated that deficits in prospective memory would be more pronounced in PD participants with greater disease severity. Looking first at the PMQT, the advanced-stage PD group were clearly impaired, as indicated by a large ES, $F(1, 59) = 15.91, p < .001, d = 1.09$ (95% CI = .54 – 1.64). The evidence was less clear in the case of the early-stage PD group. Although relative to the control group they were mildly impaired, the difference was not significant, $F(1, 60) = 1.27, p = .26, d = .30$ (95% CI = -.21 – .81). Nonetheless, the difference represented a small ES, and one cannot rule out the possibility that there is a small but genuine population difference. In any case, it is clear that the advanced-stage PD group exhibited greater impairment. Turning to the PMOT, the early-stage PD group performed normally on this task relative to the control group, $F < 1$. In contrast, the advanced-stage PD group

⁸Because of the possibility that the score distribution was skewed given the limited response range on the prospective memory tasks, we also carried out the equivalent non-parametric tests for hypotheses related to these tasks. The tests yielded precisely the same pattern of results, justifying the use of ANOVA for these data.

showed some signs of impairment, $F(1, 59) = 1.76, p = .19, d = .36$ (95% CI = $-.15 - .88$), a non-significant result but a small-to-medium ES. Thus, for both prospective memory tasks, the advanced-stage PD participants showed greater evidence of impairment than the early-stage PD group. To further evaluate the relationship between prospective memory and disease severity, correlations were calculated between the two prospective memory task scores and the Hoehn and Yahr stage. As Table 3 shows, disease stage was negatively correlated with scores on both the PMQT and the PMOT, with statistically significant, small-to-medium correlations in both cases. Taken together with the between-group comparisons, these results provide support for Hypothesis 5.

Discussion

Several important conclusions can be drawn from the results of the present study. The main one is that PD involves deficits in various aspects of recall, recognition, and prospective memory. Although the effect sizes for the deficits in recognition and prospective memory may not be clinically significant, they may be of theoretical interest, helping to establish how the central nervous system deteriorates as the disease progresses. The deficits in recognition memory are particularly noteworthy as it is widely held that recognition memory remains largely intact in PD (Brown & Marsden, 1988; Emre, 2003; Knight, 1992; Shibuya et al., 2001; Taylor et al., 1988). The finding that early-stage PD participants had no deficit in recognition at the easy level of the task is also important, as it may help to explain some of the discrepancies in the literature concerning the existence of deficits in recognition memory among PD patients. Another major conclusion of the present study is that the progression of memory deficits in PD increases in step with the overall level of disease severity. This was demonstrated by the fact that the advanced-stage PD group had larger deficits than the early-stage PD group in all memory tasks, and the fact that scores on most of the memory tasks were negatively correlated with the Hoehn and Yahr stage.

Deficits in Recall, Recognition, and Prospective Memory

The results of the present study reveal a clear pattern of deficits in recognition, recall, and prospective memory in PD. These deficits emerged relatively consistently across several memory tasks. The least surprising finding was the deficit in recall, which is in line with previous research on this topic (Brown & Marsden, 1990; Cooper et al., 1993; Sahakian et al., 1988). The present study also confirmed previous research suggesting that there is a deficit in prospective memory in PD (Katai, 1999; Katai et al., 2003). Arguably, though, the most important result of the present study was the deficit in recognition memory. This finding is consistent with the conclusion of Whittington et al. (2000) that nondemented PD participants suffer from deficits in recognition memory. However, it contrasts with the findings of Lees and Smith (1983), Flowers et al. (1984), and Taylor et al. (1986), who reported that their PD participants did not have impaired recognition memory. How can we explain these apparently contradictory results?

In the case of the Lees and Smith (1983) study, the contradiction may be explicable in terms of sample differences. Lees and Smith's participants were, on average, 10 years younger than the participants in the present study, had shorter disease duration, and were more likely to be in earlier stages of PD. This may explain why they found no recognition memory deficits; after all, the present study found no recognition deficits in the early-stage PD participants at the easier level of the task. Thus, the findings of Lees and Smith

may be consistent with those of the present study. On the other hand, sample differences cannot account for the conclusions of Flowers et al. (1984) and Taylor et al. (1986). However, a reanalysis of these researchers' data in terms of ES indicates that their participants may actually have had impaired recognition memory, but the studies had inadequate power to detect the impairment (Whittington et al., 2000). Thus, re-couching the Flowers et al. (1984) and Taylor et al. (1986) data in terms of ESs brings them into line with the results of the present study. In short, the present study provides further support for the view that PD does commonly involve deficits in recognition memory, challenging the commonly held view that recognition is largely intact whereas recall is impaired.

Nonetheless, the general trend appears to be that the deficits in recognition are less severe than those found in recall. According to some theorists, the fact that researchers consistently find deficits in recall but not in recognition is a product of task difficulty (Stebbins et al., 1999; Weingartner et al., 1984). Breen (1993) suggested that if recognition tasks were made more difficult, PD participants would show consistent deficits relative to controls. The results of the present study are consistent with this view. The early-stage PD participants did exhibit deficits of an important magnitude (with a medium-to-large ES), but only at the more difficult level of the nonverbal recognition task. In contrast, the advanced-stage PD participants had medium-to-large deficits regardless of difficulty level. It may be that a task must reach a certain threshold of difficulty before it affects a participant's performance, and that as the severity of the participant's condition increases, this threshold becomes lower. The threshold of the early-stage PD participants was presumably lower than that of the control group; however, the easy level of the NRMT task was beneath the threshold and thus the performances of the two groups were indistinguishable. However, on the more difficult level of the task, the threshold of the early-stage PD group was reached, and their impairment became apparent. The threshold of the advanced-stage PD group was presumably lower than that of the early-stage PD group, and even the easy level of the task was sufficiently taxing to reveal their memory difficulties.

This finding may shed further light on the discrepancies in the literature concerning the existence of deficits in recognition memory among PD patients. We have already argued that many such discrepancies are the result of researchers relying solely on statistical significance testing to draw conclusions from underpowered studies. Flowers et al. (1984) and Taylor et al. (1986) fall into this category. However, the finding that early-stage PD participants exhibited no deficits at the easy level of the NRMT suggests that, at least in some cases, the discrepancies may have another explanation. In such cases, the null finding may be valid, not because recognition memory is intact but because the tasks used are not sufficiently difficult to reveal the impairment when early-stage PD patients are used. The Lees and Smith (1983) study falls into this category.

To date, there has been little research on how PD might affect prospective memory. Katai et al. (2003) carried out one of the few studies showing that PD participants have prospective memory impairments for an event-based task. We found some evidence for a deficit in both of the event-based tasks we used, at least for cases where PD was more advanced. The evidence for a deficit in prospective memory was stronger in the case of the PMQT than the PMOT, perhaps because the former was a more cognitively taxing task. Unfortunately, though, there is another interpretation of this result. It is possible that no deficits were found on the PMOT because this task involved participants remembering an object of their own. In contrast, the PMQT did not involve anything of comparable personal significance. Future research should seek to replicate our result while eliminating this potential confound.

Overall, the findings of the present study corroborate earlier research suggesting that patients in the advanced stages of PD have more severe memory deficits than those in the early stages (Lees & Smith, 1983; Owen et al., 1992; Sahakian et al., 1988; Whittington et al., 2000). For instance, the advanced-stage PD participants exhibited greater deficits than did the early-stage PD participants in verbal recognition (medium-to-large vs. small-to-medium deficits) and in recall (large vs. medium-to-large deficits). Furthermore, the present study extends previous research by suggesting that prospective memory may also deteriorate in step with increases in disease severity. The advanced-stage PD participants exhibited greater deficits than the early-stage PD participants both on the PMQT (large vs. small deficits) and on the PMOT (small-to-medium vs. no deficits). In addition to the between-group comparisons, correlational analyses revealed that, with the exception of the recall task, scores on the memory tests were negatively associated with disease severity. Overall, then, the progression of deficits in verbal and nonverbal recognition, recall, and prospective memory appears to relate closely to disease severity.

Future Directions

There are various directions that future research in this area could profitably take. First, the present study makes it clear that disease severity is an important factor in assessing cognitive decline in PD. Most studies evaluate the degree of severity in each PD participant but few go on to analyze the results using severity as an independent variable. This is understandable; after all, even dividing the PD group into just two subgroups can reduce the power of a study to detect effects. Nonetheless, our study demonstrates that disease severity can be an important variable in understanding cognitive decline in PD.

Second, almost all PD studies use a control group matched with the PD group for age, IQ, years of education, and so on. The problem with this simple design is that, although it certainly allows for conclusions to be made about the effects of PD relative to controls, it does not allow one to quantify that part of any cognitive decline that is due to aging (see, e.g., Kausler, 1991). An additional matched control group of young people might help in ascertaining the true degree of cognitive decline independent of the effects of aging. Furthermore, we believe there is justification for studies comparing PD participants with older healthy participants. In our own current research, we have been struck by the similarity in performance on recognition, recall, and prospective memory tasks between an older healthy group, with an average age of 87.7 years, and a PD group. If healthy old age causes a functionally equivalent decline in memory types as in PD, then further investigation will be required to find out whether the same or different parts of the neural systems underlying these types of memories are affected.

Finally, although little is known about the rate of memory decline in PD, we had hoped that an interval of six months would be long enough to show a further deterioration in some aspects of memory in the PD participants. It is interesting that this was not the case. Longitudinal analyses of memory decline in PD, ideally beginning in the earliest stages of the disorder and ending in the latest stages, are conspicuously absent in the PD literature, but would be an important contribution to this area.

Conclusion

The major conclusions of the present study are that PD involves deficits in various aspects of memory, and that these deficits increase in step with increases in overall disease severity. The present study provides further confirmation that recognition memory is impaired

in PD, and replicates the recent finding of a PD-related impairment in prospective memory. The results of the task difficulty manipulation may help to explain why some studies fail to detect recognition deficits: Some recognition tasks may not be sufficiently challenging to reveal deficits in early-stage PD patients. Finally, the present study supports and extends previous research suggesting that patients in the advanced stages of PD have more severe memory deficits than do those in the early stages (Lees & Smith, 1983; Owen et al., 1992; Sahakian et al., 1988; Whittington et al., 2000). A general theory of cognitive dysfunction in PD must take into account not only the fact that deficits in recognition, recall, and prospective memory can occur in this neurodegenerative disorder, but also that these deficits may grow with disease severity.

References

- Barnes, J., Boubert, L., & Harris, J. (2003). Reality monitoring and visual hallucinations in Parkinson's disease. *Neuropsychologia*, *41*, 565–574.
- Berger, H.J.C., van Es, N., van Spaendonck, K.P.M., Teunisse, J.-P., Horstink, M.W.I.M., van 't Hof, M.A., & Cools, A.R. (1999). Relationship between memory strategies and motor symptoms in Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology*, *21*, 677–684.
- Beringer, J. (1995). *Experimental run time system*, Version 3.11. Frankfurt: BeriSoft.
- Bondi, M.W., Kaszniak, A.W., Bayles, K.A., & Vance, K.T. (1993). Contributions of frontal system dysfunction to memory and perceptual abilities in Parkinson's disease. *Neuropsychology*, *7*, 89–102.
- Breen, E.K. (1993). Recall and recognition memory in Parkinson's disease. *Cortex*, *29*, 91–102.
- Brown, R.G., & Marsden, C.D. (1988). Internal versus external cues and the control of attention in Parkinson's disease. *Brain*, *111*, 323–345.
- Brown, R.G., & Marsden, C.D. (1990). Cognitive function in Parkinson's disease: From description to theory. *Trends in Neuroscience*, *13*, 21–29.
- Calev, A. (1984). Recall and recognition in chronic nondemented schizophrenics. Use of matched tasks. *Journal of Abnormal Psychology*, *93*, 172–177.
- Christensen, H., Hadzi-Pavlovic, D., & Jacomb, P. (1991). The psychometric differentiation of dementia from normal aging: A meta-analysis. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, *3*, 147–155.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.
- Cooper, J.A., Sagar, H.J., Doherty, S.M., Jordan, N., Tidswell, P., & Sullivan, E.V. (1992). Different effects of dopaminergic and anticholinergic therapies on cognitive and motor function in Parkinson's disease: A follow-up study of untreated patients. *Brain*, *115*, 1701–1725.
- Cooper, J.A., Sagar, H.J., & Sullivan, E.V. (1993). Short-term memory and temporal ordering in early Parkinson's disease: Effects of disease chronicity and medication. *Neuropsychologia*, *31*, 933–949.
- Craik, F.I.M., & Jennings, J.M. (1992). Human memory. In F.I.M. Craik & T.A. Salthouse (Eds.), *The handbook of aging and cognition* (pp. 51–110). Hillsdale, NJ: Erlbaum.
- Craik, F.I.M., & McDowd, J.M. (1987). Age differences in recall and recognition. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *13*, 474–479.
- Diamond, S.G., & Markham, C.H. (1983). Evaluating the evaluations, or how to weigh the scales of parkinsonian disability. *Neurology*, *33*, 1098–1099.
- Einstein, G.O., & McDaniel, M.A. (1990). Normal aging and prospective memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *16*, 717–726.
- Emre, M. (2003). What causes mental dysfunction in Parkinson's disease? *Movement Disorder*, *18*, 63–71.
- Fahn, S., Elton, R.L., & Members of the UPDRS Development Committee. (1987). Unified Parkinson's Disease Rating Scale. In S. Fahn, C.D. Marsden, D.B. Calne, & M. Goldstein (Eds.),

- Recent developments in Parkinson's disease* (pp. 153–163, 293–304). Florham Park, NJ: MacMillan.
- Flowers, K.A., Pearce, I., & Pearce, J.M.S. (1984). Recognition memory in Parkinson's disease. *Journal of Neurology, Neuroscience, and Psychiatry*, *47*, 1174–1181.
- Hasher, L., & Zacks, R.T. (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology: General*, *108*, 356–388.
- Hoehn, M.M., & Yahr, M.D. (1967). Parkinsonism: Onset, progression, and mortality. *Neurology*, *17*, 427–442.
- Huber, S.J., Freidenberg, D.L., Shuttlesworth, E.C., Paulson, G.W., & Christy, J.A. (1989). Neuropsychological impairments associated with severity of Parkinson's disease. *Journal of Neuropsychiatry*, *1*, 154–158.
- Huppert, F.A., & Beardsall, L. (1993). Prospective memory impairment as an early indicator of dementia. *Journal of Clinical and Experimental Neuropsychology*, *15*, 805–821.
- Ivory, S.J., Knight, R.G., Longmore, B.E., & Caradoc-Davies, T. (1999). Verbal memory in nondemented patients with idiopathic Parkinson's disease. *Neuropsychologia*, *37*, 817–828.
- Katai, S. (1999). Everyday memory impairment in Parkinson's disease. *Rinsho Shinkeigaku*, *39*, 913–919.
- Katai, S., Maruyama, T., Hashimoto, T., & Ikeda, S. (2003). Event based and time based prospective memory in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, *74*, 704–709.
- Katzman, R., Brown, T., Fuld, P., Peck, A., Schechter, R., & Schimmel, H. (1983). Validation of a short orientation-memory-concentration test of cognitive impairment. *American Journal of Psychiatry*, *140*, 734–739.
- Kausler, D.H. (1991). *Experimental psychology, cognition, and human aging* (2nd ed.). New York: Springer-Verlag.
- Kendrick, D.C. (1985). *Kendrick cognitive tests for the elderly*. Windsor, England: NFER Nelson.
- Knight, R.G. (1992). *The neuropsychology of degenerative brain diseases*. Hillsdale, NJ: Erlbaum.
- Lees, A.J., & Smith, E. (1983). Cognitive deficits in the early stages of Parkinson's disease. *Brain*, *106*, 257–270.
- McDaniel, M.A., Glisky, E.L., Rubin, S.R., Guynn, M.J., & Routhieaux, B.C. (1999). Prospective memory: A neuropsychological study. *Neuropsychology*, *13*, 103–110.
- Minamoto, H., Tachibana, H., & Sugita, M. (2001). Recognition memory in normal aging and Parkinson's disease: Behavioral and electrophysiologic measures. *Cognitive Brain Research*, *11*, 23–32.
- Mortimer, J.A., Pirozzolo, F.J., Hansch, E.C., & Webster, D.D. (1982). Relationship of motor symptoms to intellectual deficits in Parkinson's disease. *Neurology*, *32*, 133–137.
- Nelson, H.E., & O'Connell, A. (1978). Dementia: The estimation of premorbid intelligence levels using the new adult reading test. *Cortex*, *14*, 234–244.
- Owen, A.M., James, M., Leigh, P.N., Summers, B.A., Marsden, C.D., Quinn, N.P., et al. (1992). Frontostriatal cognitive deficits at different stages of Parkinson's disease. *Brain*, *115*, 1727–1751.
- Ryan, J.J., & Paolo, A.M. (1992). A screening procedure for estimating premorbid intelligence in the elderly. *The Clinical Neuropsychologist*, *1*, 53–62.
- Sahakian, B.J., Morris, R.G., Evenden, J.L., Heald, A., Levy, R., Philpot, M., et al. (1988). A comparative study of visuospatial memory and learning in Alzheimer-type dementia and Parkinson's disease. *Brain*, *111*, 695–718.
- Schmidt, F.L. (1996). Statistical significance testing and cumulative knowledge in psychology: Implications for training of researchers. *Psychological Methods*, *1*, 115–129.
- Shibuya, N., Tachibana, H., Kawabata, K., & Sugita, M. (2001). Memory function in patients with Parkinson's disease in relation to neuropsychological tests and cerebral blood flow. *Nippon Ronen Igakkai Zasshi*, *38*, 193–200.
- Starkstein, S.E., & Robinson, R.G. (1991). Dementia or depression in Parkinson's disease and stroke. *Journal of Nervous and Mental Disease*, *179*, 593–601.

- Stebbins, G.T., Gabrieli, J.D., Masciari, F., Monti, L., & Goetz, C.G. (1999). Delayed recognition memory in Parkinson's disease: A role for working memory? *Neuropsychologia*, *37*, 503–510.
- Taylor, A.E., Saint-Cyr, J.A., & Lang, A.E. (1986). Frontal lobe dysfunction in Parkinson's disease. *Brain*, *109*, 845–883.
- Taylor, A.E., Saint-Cyr, J.A., & Lang, A.E. (1988). Idiopathic Parkinson's disease: Revised concepts of cognitive and affective status. *Canadian Journal of Neurological Science*, *15*, 106–113.
- Weingartner, H., Burns, S., Diebel, R., & LeWitt, P.A. (1984). Cognitive impairment in Parkinson's disease: Distinguishing between effort-demanding and automatic cognitive processes. *Psychiatry Research*, *11*, 223–235.
- Whittington, C.J., Podd, J., & Kan, M.M. (2000). Recognition memory impairment in Parkinson's disease: Power and meta-analyses. *Neuropsychology*, *14*, 233–246.
- Wilkinson, L., & the Task Force on Statistical Inference, APA Board of Scientific Affairs (1999). Statistical methods in psychology journals: Guidelines and explanations. *American Psychologist*, *54*, 594–604.
- Witkin, H.A., Oltman, P.K., Raskin, E., & Karp, S.A. (1971). *A manual for the Embedded Figures Test*. Palo Alto, CA: Consulting Psychologists Press.
- Woods, S.P., & Troester, A.I. (2003). Prodromal frontal/executive dysfunction predicts incident dementia in Parkinson's disease. *Journal of the International Neuropsychological Society*, *9*, 17–24.
- Youngjohn, J.R., Larrabee, G.J., & Crook, T.H. (1992). Discriminating age-associated memory impairment from Alzheimer's disease. *Psychological Assessment*, *4*, 54–59.