EUROPEAN JOURNAL OF COGNITIVE PSYCHOLOGY
2009, 21 (2/3), 330–346

Psychology Press
Taylor & Francis Group

# The effect of attentional set-shifting, working memory, and processing speed on pragmatic language functioning in Parkinson's disease

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Parkinson's disease (PD) is traditionally associated with motor symptoms. However, impairments in language functioning may also accompany this disorder. The present study investigated pragmatic language deficits in PD and their relationship to cognitive functioning. Forty patients with PD were compared to age- and IQ-matched controls on measures of pragmatic language functioning using the Test of Language Competence–Expanded (TLC-E), and measures of attentional set-shifting, working memory, and processing speed. Overall, PD patients were impaired on aspects of language, working memory, and processing speed. Measures of cognition were significantly correlated with pragmatic language functioning. Path analyses revealed that deficits in pragmatic language functioning were mediated by verbal working memory and processing speed, but not attentional set-shifting. Regression analyses found that processing speed was a stronger determiner of pragmatic language performance than verbal working memory. Results suggest that pragmatic language deficits may be explained in terms of deficits in processing speed associated with the disease.

***Keywords:*** Parkinson's disease; Language; Working memory; Processing speed; Attention.

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We are indebted to neurologists Tim Anderson and John Fink for the referral of patients, advice on the interpretation of the UPDRS, and helpful comments on a draft of this manuscript. The research was supported by a grant from the Canterbury Medical Research Foundation.


http://www.psypress.com/ecp DOI: 10.1080/09541440802281266

Parkinson's disease (PD) is one of the most common neurodegenerative disorders in people over 50 years of age. Depletion of dopamine containing cells in the basal ganglia is considered to be a characteristic feature of the disorder (see Frank, 2005; Middleton & Strick, 2000, for review). As the basal ganglia are key to the execution of movement and cognitive tasks (Frank, 2005; Middleton & Strick, 2000), it is not surprising therefore that cognitive impairments are frequently reported in patients with PD, even in the absence of dementia (Pillon, Boller, Levy, & Dubois, 2001). Communication problems may also accompany PD, and include impairments in speech production and a range of language abilities, including comprehension and effective verbal expression (Grossman, Carvell, Stern, Gollomp, & Hurtig, 1992; Owen, 2004). However, there is considerable controversy regarding the degree to which language deficits in PD may be mediated by other cognitive skills (Murdoch, 2001).

Subtle cognitive deficits in PD resulting from the dysfunction of the basal ganglia are apparent early in the disease process, similar to that seen in patients who have experienced prefrontal lesions (Cooper, Sagar, Jordan, Harvey, & Sullivan, 1991; Frank, 2005; Royall et al., 2002). Difficulties with attentional control, working memory, processing speed, planning, problem solving, and memory have been reported (Pillon et al., 2001). Language-related deficits that have been reported in PD include reduced verbal fluency, difficulties with processing of past tense verbs, and impairments in detecting and correcting syntax errors (McNamara & Durso, 2003; Monetta & Pell, 2007; Ullman, 2001). The comprehension of sentences with complex or irregular grammatical structures is one of the most frequently reported deficits (Bodis-Wollner & Jo, 2006; Grossman, 1999; Grossman et al., 1991, 1992, 2003; Lieberman, Friedman, & Feldman, 1990; McNamara, Krueger, O'Quin, Clark, & Durso, 1996).

However, the degree to which language deficits in PD are dependent on the integrity of other cognitive skills is still being investigated (Murdoch, 2001). To date, researchers have found evidence of an association between impaired sentence comprehension in PD and a number of other cognitive tasks including attentional set-switching, inhibition, working memory and attention, and processing speed (Angwin, Chenery, Copland, Murdoch, & Silburn, 2005; Grossman et al., 2002, 2003; Hochstadt, Nakano, Lieberman, & Friedman, 2006). Indeed, Grossman and colleagues (1992) reported that working memory and attention accounted for over 97% of the variance in complex sentence comprehension for PD patients.

It seems likely that more complex aspects of language, such as the understanding of pragmatics, would be more dependent on other aspects of cognition. Pragmatics involves the ability to use and interpret verbal and nonverbal language appropriately within the social situation in which the communicative exchange occurs, requiring a degree of inference and interpretation (Perkins, 2005). Different aspects of pragmatic language are considered to be highly resource demanding (Monetta, Ouellet-Plamondon,

& Joanette, 2006). For example, the ability to interpret nonliteral elements of language such as metaphor, considered to be a part of pragmatic language, requires the comprehension of a false statement using cues from the current context (Glucksberg, 2003; Monetta & Pell, 2007).

Impairments in pragmatic language commonly result when the prefrontal cortex is compromised (see Martin and McDonald, 2003, for a review). Given that basal ganglia dysfunction has been associated with "frontal type" deficits, it would seem likely that pragmatic language problems would also be evident for individuals with PD. Moreover, given the complexity of skills associated with the comprehension of pragmatic language, it would seem likely that other cognitive skills would be implicated in its effective use. However, there is relatively little information regarding the association between pragmatic deficits and other cognitive functions for patients with PD.

The studies that have examined pragmatic language in PD have reported deficits when compared with healthy controls. For example, Lewis, Lapointe, Murdoch, and Chenery (1998) compared the language abilities of 20 nondemented PD patients (all Hoehn & Yahr Stage 3; Hoehn & Yahr, 1967) to healthy controls using the Boston Naming Test (BNT), the WORD Test (TWT; Jorgensen, Barrett, Huistingh, and Zachman, 1981), and the Test of Language Competence–Expanded Edition (TCL-E; Wiig & Secord, 1989). Parkinson's disease patients were significantly poorer at interpreting ambiguity, figurative language, and sentence construction. Furthermore, patients with lower levels of general cognitive functioning were more impaired than other PD patients.

McNamara and Durso (2003) examined the association between pragmatic language functions and frontal dysfunction, comparing 22 nondemented PD patients with healthy controls. Pragmatic skills were assessed using a formal pragmatic skills protocol devised by Prutting and Kirchner (1987). Two tests of frontal ability were also administered; the interference condition of the Stroop colour–word test to assess susceptibility to interference, and the Tower of London task as a test of planning ability. Significant deficits were evident for the PD group when compared to healthy controls on the test of pragmatic language ability and the test of susceptibility to cognitive interference. Deficits in pragmatic language ability were correlated with poorer performance on tests of planning and susceptibility to cognitive interference. These authors concluded that impairments in pragmatic language ability may be related to frontal lobe dysfunction.

Monetta and Pell (2007) examined the effects of verbal working memory deficits on metaphor; comparing 17 individuals with PD with mild to moderate motor symptoms to healthy controls. This study used a verbal working memory span task to assess working memory capacity, and a metaphor comprehension task following the methods of Gernsbacher, Keysar, Robertson, and Werner (2001). Parkinson's disease patients were less efficient

at processing metaphorical information (as measured by accuracy and speed). Further, there was a strong relationship between pragmatic language ability and working memory. However, when patients were divided into two groups according to their working memory performance (impaired and unimpaired), only those with impaired working memory performance were impaired in the processing of metaphorical language. These authors concluded that efficient metaphor interpretation was dependent on intact working memory systems that enabled the efficient storage and processing of metaphorical information. Further, that working memory relied on the integrity of the frontostriatal systems, which are compromised in patients with PD.

The current literature suggests that pragmatic language skills are impaired as part of the neurodegenerative process associated with PD. This present study was designed to examine the degree to which any deficits in pragmatic language are mediated by different cognitive skills including working memory, processing speed, and attentional set shifting. These aspects of cognition were selected as they have frequently been found to be impaired in patients with PD, even in the early stages of the disease process (Cooper et al., 1991; Gabrieli, Singh, Stebbins, & Goetz, 1996; Lewis, Dove, Robbins, Barker, & Owen, 2003; Muslimovic, Post, Speelman, & Schmand, 2005). We hypothesised that compared to health controls, PD patients would show deficit in pragmatic language tasks. We further hypothesised that these deficits would be mediated by deficits in other areas of cognitive functioning.

## METHODS

This study received approval from the Upper South B Regional Ethics Committee. Parkinson's patients in the Canterbury region of New Zealand who could be identified at the time of this study and had not been diagnosed with dementia, were invited by letter to participate.

### Participants

*Parkinson's patients.* Participants were required to meet the following inclusion criteria: (1) a diagnosis of idiopathic Parkinson's disease, confirmed by a specialist neurologist; (2) assessed at the Hoehn and Yahr Stages 1–4 (Hoehn & Yahr, 1967; numbers of patients at each stage were as follows: Stage 1, $n = 8$; Stage 1.5, $n = 6$; Stage 2, $n = 7$; Stage 2.5, $n = 10$; Stage 3, $n = 7$; Stage 4, $n = 2$.); (3) aged between 50 and 80 years; (4) adequate or corrected hearing and vision (self-report checked by examiner); (5) stable on PD medication; (6) English as the primary spoken language; (7) no suspicion of dementia (Mini-Mental Status Exam, MMSE $= 25$; Demential Rating Scale–II, DRS-II, Jurica, Leitten, & Mattis, 2001; DSM-IV criteria, American Psychiatric Association, 2000).

The following exclusion criteria were applied: (1) currently involved in a therapeutic trial; (2) a history of: (a) moderate or severe head injury, (b) stroke or other neurological impairment, (c) other major medical illness, (d) significant psychiatric illness requiring hospitalisation, (e) major depressive episode in the previous 6 months; (3) diagnosis of, or special education for, a learning disability; (4) premorbid IQ estimated at $< 85$ using National Adult Reading Test (NART); (5) currently taking medications known to have a significant effect on the central nervous system (other than medications prescribed for the control of PD symptoms); (6) Beck Depression Inventory–II (BDI-II; Beck, Steer, & Brown, 1996) score of $> 16$.

Of the 115 letters that were posted, 6 (5.2%) of individuals with PD could not participate due to illness, 6 (5.2%) were deceased, 8 (6.9%) declined, 34 (29.6%) did not respond, and 21 (18.3%) did not meet the inclusion/exclusion criteria. Forty participants with PD who met the exclusion/inclusion criteria were available to participate in the study. All patients were on anti-Parkinsonian medication and were tested while on optimal levels of medication (self-report and examiner observation).

*Controls.* Controls were recruited from a number of sources including a previously established database, advertisements at local clubs (bowling, hiking, and table tennis), and businesses. All controls were given a brief outline of the study on first phone contact. In addition to adequate or corrected hearing and vision (self-report and checked by the examiner) and being aged between 50 and 80 years of age, the same exclusion criteria listed participants with PD also applied to the control group.

## Procedure

Assessments were carried out at the University of Canterbury. Written consent was obtained from all participants at the beginning of testing after the study had been explained. Additional information pertinent to the inclusion/exclusion criteria was obtained from all participants using a semistructured interview. All tests were conducted according to standardised procedures. Each PD patient was individually matched to a healthy control in terms of age, premorbid IQ, and current mental status.

## Measures

### *Clinical and demographic information*

The NART was used to estimate premorbid IQ. Words were scored 0 for incorrect and 1 for correct pronunciation (Lezak, 1995). The BDI-II was used to assess mood. This test consists of 21 items; each question was rated

0–3 with higher scores indicating greater intensity of symptoms (Beck et al., 1996). The BDI-II has been validated for use with PD patients, with a cutoff of 16/17 being recommended (Leentjens, 2004). The MMSE provided information regarding current cognitive status (Folstein, Folstein, & McHugh, 1975). Two additional measures were used for patients with PD to provide information regarding motor impairment: (1) Unified Parkinson's Disease Rating Scale (UPDRS) motor section (Fahn & Elton, 1987) and (2) the Hoehn and Yahr, which was used to rate the stage of the disease (Hoehn & Yahr, 1967).

### *Language and cognitive assessment*

*Pragmatic language.* Pragmatic language was assessed using the Test of Language Competence–Expanded Edition Level 2 (TLC-E). Five scores were generated using scoring instructions provided in the test manual (Wiig & Secord, 1989): (1) a total score (maximum = 189) and a score for each of the following subtests; (2) ambiguous sentences, used to assess the participant's ability to recognise lexical and structural ambiguities of a sentence (possible scores ranged from 0 to 39); (3) listening comprehension (making inferences) assessed the participants' ability to identify inferences in a series of short paragraphs (possible scores ranged from 0 to 36); (4) oral expression (recreating sentences) evaluated the ability to formulate a grammatically complete sentence that was appropriate for a given situation (possible scores ranged from 0 to 78); (5) figurative language designed to assess the ability to interpret metaphoric expressions (possible scores range from 0 to 36). For each of the subtests, a discontinue rule of failure to respond to three consecutive items was used.

*Attentional set-shifting*. This skill was assessed using the Intradimensional/Extradimensional Shift (ID/ED) from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Owen, Roberts, Polkey, Sahakian, & Robbins, 1991). This test uses computer-generated visual stimuli to assess the individuals' ability to maintain attention to different examples within a particular perceptual dimension (e.g., shapes) while ignoring irrelevant information (e.g., lines). Without prior warning, the participant must then shift attention away from this relevant dimension and attend to a previously redundant dimension. In this present study all sections of the test were administered, but analysis of the results was based on the ID + ED trails as outlined by Hutton et al. (1998).

*Working memory.* The Daneman and Carpenter Reading Span test (Daneman & Carpenter, 1980) was used to assess verbal working memory (Waters & Caplan, 1996). This test requires the participant to read sentences,

judge their veracity, and then retain the final word in the sentence. The number of sentences presented prior to recall of words varied from two to six. The reading span was assessed as the maximum number of words remembered, with at least two out of three trials correctly recalled (possible scores ranged from 1 to 6). The test was discontinued if a participant was unable to remember the last word from any of the sentences in a trial set.

*Processing speed.* Word naming and colour naming from the Delis Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001) were used to assess this skill. Age-adjusted scores were used with a mean of 10 and standard deviation of 3. Scores for colour naming and word reading were then averaged to provide a single score for speed of mental processing.

## RESULTS

Differences in demographic and clinical characteristics were examined using *t*-tests and $\chi^2$ as appropriate. Patients were well matched to healthy controls in terms of age and premorbid IQ (see Table 1), but differed in terms of symptoms consistent with low mood (as measured by the BDI-II) and current mental status (as measured by the MMSE), but no patient met the criteria for a depressive episode or dementia (DSM-IV criteria).

Table 2 shows the comparison between PD patients and matched controls on measures of language, working memory, processing speed, and attentional set-shifting using *t*-tests. PD patients performed more poorly in terms of their overall TLC-E score and for three out of the four subtests. Deficits

TABLE 1
Clinical and demographic characteristics, Parkinson's disease group versus controls

| | *Parkinson's disease (n = 40)* | | *Control group (n = 40)* | | | |
|---|---|---|---|---|---|---|
| | *Mean* | *SD* | *Mean* | *SD* | *t-value* | *p-level* |
| NART[1] | 109.05 | (10.13) | 111.20 | (10.30) | 0.94 | >.30 |
| Education (years)[2] | 13.94 | (2.56) | 13.76 | (2.57) | −0.30 | >.75 |
| Age | 66.15 | (6.65) | 66.58 | (5.47) | 0.31 | >.75 |
| MMSE[3] | 28.65 | (1.42) | 29.58 | (0.71) | 3.67 | <.001 |
| BDI-II[4] | 7.59 | (4.34) | 4.13 | (3.39) | −3.96 | <.001 |
| PD onset[5] | | | | | | |
| DRS-II[6] | 10.06 | (2.60) | 11.14 | (2.40) | | |
| UPDRS[7] | 28.46 | (9.49) | | | | |

[1]National Adult Reading Test; [2]Total number of years formal education; [3]Mini-Mental Status Exam; [4]Beck Depression Inventory; [5]Years since diagnosis of Parkinson's disease; [6]Demential Rating Scale–II; [7]Unified Parkinson's Disease Rating Scale (motor score component).

TABLE 2
Comparisons between Parkinson's disease group and matched controls on measures of language functioning, information processing speed, working memory, and attention

| | *PD patients* | *Controls* | *t* | *p* |
|---|---|---|---|---|
| TLC-E total[1] | 155.95 (19.12) | 167.28 (16.22) | 2.86 | <.01 |
| Subtests | | | | |
| Ambiguous sentence | 31.18 (6.47) | 32.35 (5.63) | 0.87 | >.35 |
| Making inferences | 25.73 (4.87) | 29.73 (4.64) | 3.76 | <.001 |
| Oral expression | 68.70 (8.22) | 72.78 (6.07) | 2.52 | <.02 |
| Figurative language | 30.25 (5.48) | 33.10 (3.79) | 2.71 | <.01 |
| Information processing speed[2] | 9.66 (1.87) | 11.34 (1.47) | 4.46 | <.0001 |
| Reading span | 1.66 (0.57) | 2.46 (0.67) | 5.73 | <.0001 |
| Attentional set-shifting | 10.23 (2.13) | 10.95 (2.25) | 1.48 | >.10 |

[1]Test of Language Competence–Expanded, total score; [2]age-adjusted scores (mean = 10, $SD = 3$).

were also evident for tests of processing speed and working memory. However, there was no significant difference between the groups on attentional set-shifting or for ability to interpret ambiguous sentences.

Table 3 shows the correlations (using Pearson correlations) for the combined sample (i.e., PD patients and controls) between pragmatic language functioning and processing speed, working memory, and attentional set-shifting. Significant positive correlations were found between each of the different aspects of cognition and pragmatic language. Significant negative correlations were also found between disease state (i.e., PD patients vs. controls) and measures of cognition and pragmatic language functioning, confirming the deficits shown by PD patients in Table 2.

Next, a series of path analyses were conducted to determine whether the pragmatic language deficits observed in the PD patients might be a secondary effect of deficits in cognitive functioning. Specifically, whether working memory, processing speed and attentional set-shifting might mediate the relationship between disease state and pragmatic language functioning evidenced in Table 3. TLC-E total score was used as the measure of pragmatic language functioning.

Four basic steps were followed in the models described here (Shrout & Bolger, 2002). First, the direct path in which the independent variable (in this case disease state) caused a change in another dependent variable (in this case pragmatic language) was calculated (represented by the solid arc in Figures 1 and 2). The relationship between disease state and a potential mediating variable (attentional set-shifting, processing speed and working memory) was then tested. Next, the relationship between the proposed

TABLE 3
Correlations between measures of language functioning, processing speed memory, and attentional set-shifting for the combined Parkinson's disease and control group ($n = 80$)

| | *TLC-E total* | *Ambiguous sentences* | *Making inferences* | *Oral expression* | *Figurative language* | *Processing speed* | *Reading span task* | *Attentional set-shifting* |
|---|---|---|---|---|---|---|---|---|
| TLC-E total | | | | | | | | |
| Ambiguous sentences | 0.72*** | | | | | | | |
| Making inferences | 0.73*** | 0.39*** | | | | | | |
| Oral expression | 0.78*** | 0.42*** | 0.42** | | | | | |
| Figurative language | 0.82*** | 0.45*** | 0.60*** | 0.51*** | | | | |
| Processing speed | 0.51*** | 0.27* | 0.54*** | 0.48*** | 0.42*** | | | |
| Reading span | 0.36*** | 0.26* | 0.35** | 0.22 | 0.40*** | 0.39*** | | |
| Attentional set-shifting | 0.44*** | 0.23* | 0.37*** | 0.37*** | 0.35** | 0.27* | 0.15 | |
| PD vs. control | −0.31** | −0.10 | −0.40*** | −0.28* | −0.29** | −0.45*** | −0.54*** | −0.09 |

*$p < .05$; **$p < .01$; ***$p < .001$.

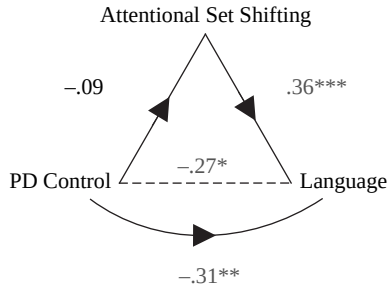


**Figure 1.** Path diagram where the intervening variable is attentional set-shifting.

mediating variable and pragmatic language was assessed. Finally, the change in the relationship between disease state and pragmatic language was calculated when the mediator was included. The resulting effect is the indirect path, signified by a dotted line in Figures 1 and 2. Sobel's $z$-test was used (Baron & Kenny, 1986) to test whether the difference in the relationship between disease state and pragmatic language when the mediator was included reflected a significant change.

As can be seen in Figure 1, there was no significant association between PD disease state and attentional set-shifting ability. Further, although performance on attentional set-shifting was significantly associated with language performance, there was no evidence of a significant mediating effect (Sobel's $z = 0.80$, $p > .40$) and the relationship between disease state and pragmatic language remained significant even after attentional set shifting ability was controlled for (see Table 4 for beta weights).

Figure 2 shows that there was a significant change in the relationship between disease state and pragmatic language functioning with the inclusion of either working memory or processing speed as a mediator (see Table 4 for full results). For both these models, the indirect pathway between disease

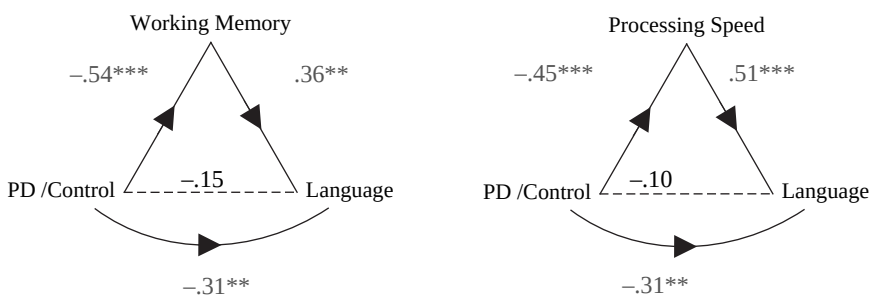


**Figure 2.** Path diagram where the intervening variable is working memory and separately processing speed.

TABLE 4
Regression coefficients for mediating variables

| | *Step 1 (criterion: Lang)* | | *Step 2 (criterion: PS)* | | *Step 3 (criterion: Lang)* | |
|---|---|---|---|---|---|---|
| *Variable* | *ß* | *t* | *ß* | *t* | *ß* | *t* |
| Group | −.31 | −2.86** | −.45 | −4.46*** | −.10 | −0.88 *ns* |
| Processing speed | | | | | .47** | 4.32 |
| | | | *Step 2 (criterion: WM)* | | *Step 3 (criterion: Lang)* | |
| Group | | | −.54 | −5.73*** | −.16 | −1.27 *ns* |
| Working memory | | | | | .27 | 2.16* |
| | | | *Step 2 (criterion: ATT )* | | *Step 3 (criterion: Lang)* | |
| Group | | | −.09 | −0.81 *ns* | −.27 | −2.47** |
| Attentional set-shifting | | | | | .41 | 4.19*** |

Lang = language functioning; PS = processing speed; WM = working memory; ATT = attentional set-shifting.

state and pragmatic language functioning was no longer significant when the mediating variable was included, and the drop in association between the direct and indirect pathways was significant (Sobel's $z = 2.90$, $p < .01$ and $z = 3.4$, $p < .001$ for working memory and processing speed, respectively). Thus, results of the path analyses suggest that both working memory and processing speed, but not attentional set-shifting, can explain the deficits in pragmatic language functioning shown by the PD patients.

Because measures of working memory and processing speed were correlated, (see Table 3), multiple regression analysis was used to enable us to examine these two factors separately so that the effect of each could be estimated. We specifically wanted to test whether one of these variables might be primarily responsible for the pragmatic language deficits associated with PD. For these analyses, group (PD versus control) and verbal working memory (or processing speed) were entered at the first step, and processing speed (or verbal working memory) was entered at the second step. When group and verbal working memory were entered in the first step, processing speed was significantly related to the TLC-E ($\beta = .44$, $R^2$ change $= .15$, $p = .001$). However, when processing speed and group were entered on the first step, verbal working memory was not significantly related to the TLC-E ($\beta = .18$, $R^2$ change $= .02$, $p = .12$). These results suggest that processing speed is a stronger determiner of performance on the TLC-E than verbal working memory, and hence that the pragmatic language deficits associated with PD are best understood as being mediated by deficits in processing speed.

## DISCUSSION

The goal of this study was to assess pragmatic language functioning in patients with PD compared to healthy older adults, and to examine the degree to which deficits that were observed could be explained by other cognitive processes. These processes included working memory, processing speed and attentional set-shifting. Patients with PD were found to perform significantly more poorly on pragmatic language tasks than healthy controls. Performances on pragmatic language tasks were significantly correlated with processing speed, verbal working memory, and attentional set-shifting. These three cognitive skills accounted for 13–26% of the variance on the TLC-E. Whereas path analyses indicated that both verbal working memory and processing speed mediated the relationship between disease status and higher order language functioning, multiple regressions confirmed that information processing speed was a stronger determiner of pragmatic language performance than verbal working memory. Overall, these results suggest that the pragmatic language deficits in PD are secondary to deficits in processing speed, more than likely reflecting the severe depletion of dopamine in the basal ganglia and subsequent deterioration of frontostriatal circuits that has been associated with deficits in executive functioning (Owen, 2004).

Our findings may indicate that processing speed declines more rapidly than working memory in individuals with PD compared to healthy older people, hence the stronger association between speed of processing and deficits in pragmatic language abilities. It is generally accepted that processing speed and working memory are interdependent, as processing speed plays a major role in working memory efficiency (Salthouse, 1994; Verhaeghen & Salthouse, 1997). For example, if processing speed is slowed, a higher demand is placed on the storage system of the working memory system. Therefore, impaired speed of processing ability could be viewed as underlying the cognitive deficits seen in PD.

However, as Verhaeghen and Salthouse (1997) point out, speed of processing and working memory share a high proportion of variance in different cognitive abilities, and neither appears to be a single determiner of age related declines. It therefore it seems likely that both processing speed and working memory contribute to complex cognitive activities such as the interpretation of pragmatic language in PD.

Another possible reason for our findings may be that the reading span task used was not a pure measure of working memory. Reading span tasks have been used as tests of working memory because they require active manipulation of information and concurrent item retention (Just & Carpenter, 1992). However, reading span tasks have been found to rely on many of the same processes as reading comprehension tasks (Engle,

Tuholski, Laughlin, & Conway, 1999), which makes it difficult to draw any strong conclusions in terms of the mediating value of working memory for pragmatic language skills. However, a working memory task that was not highly dependent on reading comprehension may have resulted in a different outcome to those reported in this study.

The comparisons between PD patients and matched controls in the present study are consistent with previous studies that have documented deficits in processing speed and working memory in PD patients (Berry, Nicolson, Foster, Behrmann, & Sagar, 1999; Lewis, Cools, et al., 2003; Lewis, Dove, et al., 2003; Lewis, Slabosz, Robbins, Barker, & Owen, 2005; Pillon et al., 1989). Further, PD patients have consistently been reported as experiencing difficulty in different components of language, particularly understanding complex sentences (Grossman et al., 2002; Hochstadt et al., 2006). Deficits in working memory and processing speed have previously been reported as affecting the accuracy of patients with PD with regard to understanding of complex sentences (Angwin et al., 2005; Grossman et al., 2002; Hochstadt et al., 2006). Given this evidence and the findings from our own investigations, it seems likely that both working memory and speed of processing contribute the effective understanding of pragmatic language.

Although relatively few studies have investigated outcomes for PD patients in terms of pragmatic language functioning, those that have reported deficits particularly for those experiencing cognitive problems (Lewis et al., 1998; McNamara et al., 1996; Monetta & Pell, 2007). Of particular interest is the study conducted by Lewis et al. (1998), which, contrary to this study, found evidence for deficits in the understanding of ambiguous sentences. However, patient characteristics may account for this apparent discrepancy. Lewis et al. did not explicitly exclude PD patients with dementia, and it is likely that interpretation of ambiguity is more impaired for these patients.

Pragmatic language functioning required in everyday communication is complex, with a considerable degree of novelty. It would be expected that these types of language interactions would rely more heavily on other cognitive skills such as of information processing working memory. The method of assessment used here enabled the examination of skills that closely resemble those used in everyday communication and their relationship with other cognitive skills. Nonetheless, it is pertinent to consider the limitations of this study. Although this study endeavoured to recruit a representative sample, patients were self-selected. Further, only nondemented patients with no illnesses apart from PD were included. It is likely that pragmatic language deficits would be more severe in patients with greater cognitive decline. Moreover, we individually matched participants in terms of age, education, and premorbid IQ, but we did not assess socioeconomic status, although it is highly correlated with cognitive ability.

Pragmatic language used in everyday communication requires the ability to understand meanings that extend beyond the actual words spoken. Understanding of ambiguity and inference of what is intended by the speaker is also required. Even subtle deficits in these areas of language may serve to increase isolation of PD patients from normal social interaction intensifying their reduced quality of life (Miller, Noble, Jones, & Burn, 2006). It is also likely that these types of difficulties may cause frustration for caregivers who may not understand the changes in comprehension and interpretation that are occurring for the patient. Understanding the exact nature of cognitive deficits, or intact skills, that could facilitate learning could potentially provide a means of intervention to ease any language problems. For example, education for professionals and caregivers regarding how to present information in an appropriate way to enhance communication could ease the frustration of caring for an individual who has difficulty communicating. Patients could be instructed in the use of strategies to clarify misunderstandings. Further, because effective communication appears to be linked with intact cognition, professionals could screen for cognitive decline as a marker for communication problems and take steps to intervene early in the disease process.

## REFERENCES

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Angwin, A. J., Chenery, H. J., Copland, D. A., Murdoch, B. E., & Silburn, P. A. (2005). Summation of semantic priming and complex sentence comprehension in Parkinson's disease. *Cognitive Brain Research*, *25*, 78–89.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory* (2nd ed.). New York: Psychological Corporation.

Berry, E. L., Nicolson, R. I., Foster, J. K., Behrmann, M., & Sagar, H. J. (1999). Slowing of reaction time in Parkinson's disease: The involvement of the frontal lobes. *Neuropsychologia*, *37*(7), 787–795.

Bodis-Wollner, I., & Jo, M. Y. (2006). Getting around and communicating with the environment: Visual cognition and language in Parkinson's disease. *Journal of Neural Transmission Supplement*, *70*, 333–338.

Cooper, J. A., Sagar, H. J., Jordan, N., Harvey, N. S., & Sullivan, E. V. (1991). Cognitive impairment in early, untreated Parkinson's disease and its relationship to motor disability. *Brain*, *114*(5), 2095–2122.

Daneman, M., & Carpenter, P. (1980). Individual differences in working memory and reading. *Journal of Verbal Learning and Verbal Behavior*, *19*, 450–466.

Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). *Delis Kaplan Executive Function System*. New York: Psychological Corporation.

Engle, R. W., Tuholski, S. W., Laughlin, J. E., & Conway, A. R. A. (1999). Working memory, short-term memory, and general fluid intelligence: A latent-variable approach. *Journal of Experimental Psychology: General*, *128*(3), 309–331.

Fahn, S., & Elton, R. L. (1987). *Unified Parkinson's Disease Rating Scale* (Vol. 2). Florham Park, NJ: Macmillan Health Care Information.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*(3), 189–198.

Frank, M. J. (2005). Dynamic dopamine modulation in the basal ganglia: A neurocomputational account of cognitive deficits in medicated and nonmedicated Parkinsonism. *Journal of Cognitive Neuroscience*, *17*(1), 51–72.

Gabrieli, J. D. E., Singh, J., Stebbins, G. T., & Goetz, C. G. (1996). Reduced working memory span in Parkinson's disease: Evidence for the role of a frontostriatal system in working and strategic memory. *Neuropsychology*, *10*(3), 322–332.

Gernsbacher, M. A., Keysar, B., Robertson, R. R. W., & Werner, N. K. (2001). The role of suppression and enhancement in understanding metaphors. *Journal of Memory and Language*, *45*, 433–450.

Glucksberg, S. (2003). The psycholinguistics of metaphor. *Trends in Cognitive Neuroscience*, *7*(2), 92–96.

Grossman, M. (1999). Sentence processing in Parkinson's disease. *Brain Cognition*, *40*(2), 387–413.

Grossman, M., Carvell, S., Gollomp, S., Stern, M. B., Vernon, G., & Hurtig, H. I. (1991). Sentence comprehension and praxis deficits in Parkinson's disease. *Neurology*, *41*(10), 1620–1626.

Grossman, M., Carvell, S., Stern, M. B., Gollomp, S., & Hurtig, H. I. (1992). Sentence comprehension in Parkinson's disease: The role of attention and memory. *Brain and Language*, *42*(4), 347–384.

Grossman, M., Cooke, A., DeVita, C., Lee, C., Alsop, D., Detre, J., et al. (2003). Grammatical and resource components of sentence processing in Parkinson's disease: An fMRI study. *Neurology*, *60*(5), 775–781.

Grossman, M., Zurif, E., Lee, C., Prather, P., Kalmanson, J., Stern, M. B., et al. (2002). Information processing speed and sentence comprehension in Parkinson's disease. *Neuropsychology*, *16*(2), 174–181.

Hochstadt, J., Nakano, H., Lieberman, A., & Friedman, H. (2006). The roles of sequencing and verbal working memory in sentence comprehension deficits in Parkinson's disease. *Brain and Language*, *97*, 243–257.

Hoehn, M. M., & Yahr, M. D. (1967). Parkinsonism: Onset, progression and mortality. *Neurology*, *17*(5), 427–442.

Hutton, S. B., Puri, B. K., Duncan, L. J., Robbins, T., Barnes, T. R. E., & Joyce, E. M. (1998). Executive function in first-episode schizophrenia. *Psychological Medicine*, *28*, 463–473.

Jorgensen, C., Barrett, M., Huistingh, R., & Zachman, L. (1981). *The WORD Test: A test of expressive vocabulary and semantics.* Moline, IL: LinguiSystems, Inc.

Jurica, P. J., Leitten, C. L., & Mattis, S. (2001). *DRS-2 Dementia Rating Scale-2: Professional manual.* Psychological Assessment Resources, Inc.

Just, M. A., & Carpenter, P. A. (1992). A capacity theory of comprehension: Individual differences in working memory. *Psychological Review*, *99*(1), 122–149.

Leentjens, A. F. (2004). Depression in Parkinson's disease: Conceptual issues and clinical challenges. *Journal of Geriatric Psychiatry and Neurology*, *17*(3), 120–126.

Lewis, S. J., Cools, R., Robbins, T. W., Dove, A., Barker, R. A., & Owen, A. M. (2003). Using executive heterogeneity to explore the nature of working memory deficits in Parkinson's disease. *Neuropsychologia*, *41*(6), 645–654.

Lewis, S. J., Dove, A., Robbins, T. W., Barker, R. A., & Owen, A. M. (2003). Cognitive impairments in early Parkinson's disease are accompanied by reductions in activity in frontostriatal neural circuitry. *Journal of Neuroscience*, *23*(15), 6351–6356.

Lewis, S. J., Lapointe, L. L., Murdoch, B. E., & Chenery, H. J. (1998). Language impairment in Parkinson's disease. *Aphasiology*, *12*(3), 193–206.

Lewis, S. J., Slabosz, A., Robbins, T. W., Barker, R. A., & Owen, A. M. (2005). Dopaminergic basis for deficits in working memory but not attentional set-shifting in Parkinson's disease. *Neuropsychologia*, *43*(6), 823–832.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). Oxford, UK: Oxford University Press.

Lieberman, P., Friedman, J., & Feldman, L. S. (1990). Syntax comprehension deficits in Parkinson's disease. *Journal of Nervous and Mental Disease*, *178*(6), 360–365.

Martin, I., & McDonald, S. (2003). Weak coherence, no theory of mind, or executive dysfunction? Solving the puzzle of pragmatic language disorders. *Brain and Language*, *85*, 451–466.

McNamara, P., & Durso, R. (2003). Pragmatic communication skills in patients with Parkinson's disease. *Brain and Language*, *84*, 414–423.

McNamara, P., Krueger, M., O'Quin, K., Clark, J., & Durso, R. (1996). Grammaticality judgments and sentence comprehension in Parkinson's disease: A comparison with Broca's aphasia. *International Journal of Neuroscience*, *86*(1–2), 151–166.

Middleton, F. A., & Strick, P. L. (2000). Basal ganglia and cerebellar loops: Motor and cognitive circuits. *Brain Research and Brain Research Review*, *31*(2–3), 236–250.

Miller, M., Noble, E., Jones, D., & Burn, D. (2006). Life with communication changes in Parkinson's disease. *Age and Ageing*, *35*, 235–239.

Monetta, L., Ouellet-Plamondon, C., & Joanette, Y. (2006). Simulating the pattern of right-hemisphere-damaged patients for the processing of the alternative metaphorical meanings of words: Evidence in favour of a cognitive resources hypothesis. *Brain and Language*, *96*, 171–177.

Monetta, L., & Pell, M. D. (2007). Effects of verbal working memory deficits on metaphor comprehension in patients with Parkinson's disease. *Brain and Language*, *101*, 80–89.

Murdoch, B. E. (2001). Subcortical brain mechanisms in speech and language. *Folia Phoniatrica*, *53*(5), 233–251.

Muslimovic, D., Post, B., Speelman, J. D., & Schmand, B. (2005). Cognitive profile of patients with newly diagnosed Parkinson disease. *Neurology*, *65*(8), 1239–1245.

Owen, A. M. (2004). Cognitive dysfunction in Parkinson's disease: The role of frontostriatal circuitry. *Neuroscientist*, *10*(6), 525–537.

Owen, A. M., Roberts, A. C., Polkey, C. E., Sahakian, B. J., & Robbins, T. W. (1991). Extra-dimensional versus intra-dimensional set shifting performance following frontal lobe excisions, temporal lobe excisions or amygdalo-hippocampectomy in man. *Neuropsychologia*, *29*(10), 993–1006.

Perkins, M. R. (2005). Pragmatic ability and disability as emergent phenomena. *Cinical Linguistics and Phonetics*, *19*(5), 367–377.

Pillon, B., Boller, F., Levy, R., & Dubois, B. (2001). Cognitive deficits and dementia in Parkinson's disease. In F. Boller & S. F. Cappa (Eds.), *Handbook of neuropsychology* (2nd ed., Vol. 6, pp. 311–371). New York: Elsevier Science BV.

Pillon, B., Dubois, B., Bonnet, A. M., Esteguy, M., Guimaraes, J., Lhermitte, F., et al. (1989). Cognitive slowing in Parkinson's disease fails to respond to levodopa treatment: The 15-objects test. *Neurology*, *39*, 762–768.

Prutting, C. A., & Kirchner, D. M. (1987). A clinical appraisal of the pragmatic aspects of language. *Journal of Speech and Hearing Disorders*, *52*(2), 105–109.

Royall, D. R., Lauterbach, E. C., Cummings, J. L., Reeve, A., Rummans, T. A., Kaufer, D. I., et al. (2002). Executive control function: A review of its promise and challenges for clinical research.

A report from the Committee on Research of the American Neuropsychiatric Association. *Journal of Neuropsychiatry and Clinical Neuroscience*, *14*(4), 377–405.

Salthouse, T. A. (1994). The aging of working memory. *Neuropsychology*, *8*(4), 535–543.

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, *7*(4), 422–445.

Ullman, M. T. (2001). The declarative/procedural model of lexicon and grammar. *Journal of Psycholinguist Research*, *30*(1), 37–69.

Verhaeghen, P., & Salthouse, T. (1997). Meta-analyses of age-cognition relations in adulthood: Estimates of linear and nonlinear age effects and structural models. *Psychological Bulletin*, *3*, 231–249.

Waters, G. S., & Caplan, D. (1996). The capacity theory of sentence comprehension: Critique of Just and Carpenter (1992). *Psychological Review*, *103*(4), 761–772.

Wiig, E. H., & Secord, W. (1989). *Test of Language Competence–Expanded Edition*. New York: Psychological Corporation.