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Verbal fluency deficits in Parkinson's disease: A meta-analysis

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Abstract

A meta-analysis of 68 studies with a total of 4644 participants was conducted to investigate the sensitivity of tests of verbal fluency to the presence of Parkinson's disease (PD) relative to healthy controls. Both phonemic and semantic fluency were moderately impaired but neither deficit qualified as a differential deficit relative to verbal intelligence or psychomotor speed. However, PD patients were significantly more impaired on semantic relative to phonemic fluency ($rs = .37$ *vs.* .33, respectively), and confrontation naming, a test of semantic memory that imposes only minimal demands upon cognitive speed and effortful retrieval, was associated with a deficit that was of a comparable magnitude to the deficits upon each of these types of fluency. Thus, the disorder appears to be associated with particular problems with semantic memory. Tests that impose heavy demands upon switching may also be disproportionately affected. Demented and non-demented PD patients differ quantitatively but not qualitatively in terms of the relative prominence of deficits on tests of phonemic and semantic fluency. However, patients with dementia of the Alzheimer's type and demented PD patients can be differentiated from one another by the relative magnitude of deficits upon these two measures. (*JINS*, 2004, *10*, 608–622.)

Keywords: Parkinson's, Fluency, Semantic memory, Switching

INTRODUCTION

In addition to motor abnormalities, it has long been recognised that Parkinson's disease (PD) is also associated with a number of cognitive deficits, of which it has been suggested that executive dysfunction is particularly prominent (Della Sala, 1988). Executive functioning is considered to be responsible for the more complex, or supervisory aspects of cognition such as self-directed planning and strategy formation, future-orientated, goal-directed and non-habitual behavior (Crawford & Henry, in press; Phillips, 1997; Shallice, 1988; Stuss & Benson, 1986). It has been argued that PD is associated with a number of specific executive deficits, including problems with effortful processing (Weingartner et al., 1984), the use of internal attentional cues (Brown & Marsden, 1988), cognitive set-shifting (Zec et al., 1999), and self-directed strategy formation (Taylor et al., 1986). Moreover, Della Sala (1988) has suggested that executive dysfunction can account for all of the cognitive deficits associated with non-demented PD.

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Consistent with this possibility, it has been found that patients with PD often perform poorly on tests designed to capture executive dysfunction, including the Wisconsin Card Sorting Test (WCST; Gotham et al., 1988; Tsai et al., 1994) tests of verbal fluency (Auriacombe et al., 1993; Cooper et al., 1991; Flowers et al., 1995; Matison et al., 1982) and the Stroop interference test (Hanes et al., 1996a). Moreover, neuropathologically, PD is associated with neuronal loss in the substantia nigra that leads to dopamine depletion in the nigro-striatal projection. This, in turn, leads to functional abnormalities in the subcorticofrontal circuits (De Long & Georgopolis, 1981). Since there is a great deal of evidence that executive processes rely heavily upon the intact functions of frontal structures (see, e.g., Shallice, 1988; Stuss & Benson, 1986) the presence of frontal abnormalities would therefore suggest that deficits in this aspect of cognition should be especially marked.

However, patients with PD may be impaired upon virtually all measures of cognitive function, and this includes tests presumed to make only minimal demands on executive processes. As Miller (1984) and others (Crawford & Henry, in press; Laws, 1999) have pointed out, a deficit on an executive measure is not by itself sufficient to infer the

presence of a differential executive deficit; instead, it must be shown that the executive deficit is in excess of the averaged performance deficit across a range of other cognitive tasks that are not considered to impose heavy executive demands. Moreover, it has been shown that putative measures of executive functioning such as the WCST, Stroop interference task and verbal fluency can be dissociated in PD (Gurd, 1995; Van Spaendonck et al., 1996), and thus, it may be that only certain aspects of executive functioning are differentially impaired.

Verbal Fluency Performance in PD

In an attempt to resolve whether PD is associated with a differential executive deficit, verbal fluency performance has been studied extensively. Tests of verbal fluency require time-restricted generation of multiple response alternatives under constrained search conditions, and involve associative exploration and retrieval of words based on phonemic or semantic criteria (known as phonemic or letter fluency, and semantic or category fluency, respectively). Both measures are thought to require efficient organisation of verbal retrieval and recall, as well as self-monitoring aspects of cognition (the participant must keep track of responses already given), effortful self-initiation, and inhibition of responses when appropriate (Crawford & Henry, in press; Perret, 1974; Phillips, 1997; Ruff et al., 1997).

However, whilst some studies have reported significant deficits on measures of phonemic fluency in non-demented PD (Azuma et al., 1997; Flowers et al., 1995) others have failed to do so (Auriacombe et al., 1993; Caltagirone et al., 1989; Goldman et al., 1998; Ivory et al., 1999; Levin et al., 1989; Matison et al., 1982; Miller, 1985). Indeed, at least one study reported that PD patients performed better than their respective control group on this task (Taylor et al., 1986). Preserved semantic fluency has also been reported (Gabrieli et al., 1996; Troyer et al., 1998) but the most consistent finding is impaired performance (Auriacombe et al., 1993; Cooper et al., 1991; Flowers et al., 1995; Matison et al., 1982). Moreover, a number of studies have reported significant deficits on semantic, but not phonemic fluency (Auriacombe et al., 1993; Matison et al., 1982).

A number of different explanations have been proposed to explain why patients with PD are impaired on tests of verbal fluency. Flowers et al. (1995) for instance, have argued that the verbal fluency deficit associated with PD reflects a mental bradyphrenia that parallels patients' motor bradykinesia. Flowers et al. (1995) found that non-demented PD patients were impaired on both semantic and phonemic fluency, but that verbal intelligence alone could not account for poor performance, and other word-production characteristics did not differ from healthy controls.

However, in a meta-analytic review of the verbal fluency performance of patients with focal cortical lesions, Henry and Crawford (2004) found that focal frontal lobe injuries were associated with equivalent phonemic and semantic fluency deficits (rs = .52 and .54 respectively) suggesting that phonemic and semantic fluency impose comparable demands upon executive processes. It may therefore be that a pattern of comparable impairment upon tests of phonemic and semantic fluency for patients with PD reflects executive dysfunction. However, to support this hypothesis, it would also be necessary to demonstrate that for patients with PD, as for frontal patients (but not for non-frontal patients), the deficit in verbal fluency qualifies as a differential deficit relative to current VIQ and psychomotor speed. Although Flowers et al. (1995) found that the phonemic and semantic fluency deficits could not be explained by level of VIQ, no assessment was made of cognitive speed, and thus it is not clear whether comparable deficits for the two measures reflects the presence of a bradyphrenia as Flowers et al. (1995) suggested, or executive dysfunction.

However, as noted earlier, many studies have found that semantic fluency is more impaired than phonemic fluency, and this finding has typically been attributed to the presence of a specific deficit in semantic memory. Consistent with this interpretation, whilst Henry and Crawford (2004) found that focal frontal injuries were associated with equivalent phonemic and semantic fluency deficits, semantic fluency was more impaired following focal temporal damage (r = .61), and the deficit was significantly larger than the corresponding phonemic fluency deficit (r = .44). Since there is a great deal of evidence that temporal structures are the neural substrates particularly responsible for semantic memory, this was presumed to reflect the greater reliance of semantic fluency upon the integrity of semantic memory.

Thus, Raskin et al. (1992a), for instance, found that non-demented PD patients did not differ from healthy controls on a test of phonemic fluency, but were significantly impaired on a test of semantic fluency in which specific cues were provided. Raskin et al. (1992a) suggested that PD patients possess intact storage systems, but that there may be a specific deficit in the *retrieval* of semantic information. Auriacombe et al. (1993) also found that semantic but not phonemic fluency was significantly impaired in PD. However, this was attributed to a more specific retrieval deficit in accessing the verbal labels, or phonological shapes, associated with category exemplars; i.e., not a problem with the retrieval of semantic information *per se*, but in lexical retrieval.

It has also been suggested that PD is associated with a specific deficit in the executive control mechanisms responsible for the consecutive inhibition and disinhibition of algorithms. This derives from the fact that some studies have reported that measures of verbal fluency in which participants must alternate or shift between naming exemplars that belong to more than one different category, or according to more than one type of phonemic criteria, are also impaired. However, whilst performance on the WCST is often disrupted (Gotham et al., 1988; Lees & Smith, 1983; Tsai et al., 1994), deficits on tests of alternating fluency may not be disproportionate to those associated with the single fluency condition (Cooper et al., 1991; Gurd, 1995). Downes et al. (1993) argue that an important factor when

assessing alternating fluency performance is whether intra- or extra-dimensional shifting is required, as it was found that whilst PD patients' ability to alternate between probes of the same domain (i.e., semantic–semantic, or phonemic–phonemic) was intact, generation of exemplars from different fluency domains (i.e., phonemic–semantic) was selectively compromised.

Thus, different researchers have advanced different interpretations of the nature of the cognitive impairment associated with PD, and that is presumed to underlie deficits on tests of verbal fluency, and this at least partially reflects discrepancies between studies in terms of the relative magnitude of deficits on different cognitive measures. It has been suggested that such discrepancies may be attributable to substantive differences between studies in terms of the PD patients sampled, and in particular, PD is associated with a high incidence of dementia.

Verbal Fluency Performance in Demented PD

It remains unclear whether dementia in PD is analogous to dementia of the Alzheimer's type (DAT), or reflects a clinical/neuropsychological syndrome that Albert (1978) has named subcortical dementia. It has been claimed that cortical dementias such as DAT are typified by a pattern of worse semantic relative to phonemic fluency performance, and subcortical dementias by the opposite deficit profile. However, whilst research involving patients with DAT, Huntington's disease and progressive supranuclear palsy has found evidence consistent with this distinction (Hodges et al., 1990; Rosser & Hodges, 1994), the cortical–subcortical dissociation has not been consistently upheld. Suhr and Jones (1998) for instance, found the pattern of semantic and phonemic fluency deficits to be comparable for patients with Alzheimer's, Huntington's and Parkinson's dementias. Questions also remain with respect to the relationship between cognitive deficits in demented and non-demented PD. Azuma et al. (1997) found that as mental state decreases, PD is associated with a reduced ability to use sub-category structure to facilitate retrieval, suggesting that there is a qualitative difference, and that semantic fluency should be disproportionately impaired relative to phonemic fluency as the dementia progresses. However, this hypothesis has not yet been rigorously tested.

Aims

To the present authors' knowledge, the current paper is the first to apply meta-analytic techniques to compare performance upon tests of phonemic and semantic fluency in PD. One of the most important advantages of this methodology is that corrections can be implemented for sampling error, and thus it will be possible to assess whether discrepancies between studies reflect the influence of substantive factors such as dementia status, or artifactual variance. In addition, using meta-analysis an effect's generalisability can be subjected to a level of scrutiny not possible in a single study, and with a level of objectivity and methodological consistency that is difficult to achieve in non-quantitative reviews (Stanley, 2001).

The first aim was to derive effect size estimates for phonemic and semantic fluency for patients with PD relative to healthy controls. Comparison of the relative magnitude of each will help to resolve the inconsistencies noted in the literature, and permit an assessment of whether the verbal fluency deficit associated with PD predominantly reflects executive dysfunction, or problems with semantic memory (Henry & Crawford, 2004).

However, as noted, the presence of a deficit on a test of phonemic or semantic fluency does not by itself provide evidence of executive or semantic memory dysfunction, respectively. Thus, the second aim was to estimate effect sizes for other cognitive measures in order to assess to what extent fluency deficits in PD qualify as *differential* deficits.

Premorbid intelligence as estimated by the National Adult Reading Test (NART; Nelson, 1982) and the reading subtest of the Wide Range Achievement Test (WRAT; Jastak & Wilkinson, 1984) was included to address the possibility that if a phonemic fluency deficit is present, it reflects the fact that PD patients have not been successfully matched to their controls for premorbid ability. However, of particular importance was to address the possibility that phonemic and semantic fluency deficits simply reflect a current general impairment in verbal abilities (see Miller, 1984). Thus, the *pattern* of deficits across fluency versus verbal intelligence as measured by the WAIS (Wechsler, 1955; 1981) Verbal and Vocabulary scales (VIQ) will be compared.

We will also assess whether deficits on tests of phonemic and semantic fluency are in excess of deficits on the WAIS Digit Symbol test (Wechsler, 1955; 1981), a widely used measure of psychomotor speed (Salthouse, 1992). This will address the possibility that deficits on tests of verbal fluency simply reflect generalised slowing rather than executive dysfunction. Performance on tests of phonemic and semantic fluency will also be compared with the Boston Naming Test (BNT; Kaplan et al., 1983), a measure of semantic memory that imposes only minimal demands upon effortful retrieval and cognitive speed.

The third aim is to compare the magnitude of phonemic and semantic fluency deficits with performance on tests of alternating fluency. Effect sizes will also be calculated for the number of categories completed and perseverative errors upon the Wisconsin Card Sorting Test (WCST CC and WCST PE respectively; Heaton, 1981) as this measure also imposes demands upon cognitive set-shifting (Miyake et al., 2000).

A fourth issue relates to whether the relative magnitude of the deficits for phonemic and semantic fluency are comparable for demented and non-demented sub-groups, and for demented PD patients relative to patients with DAT. The magnitude of these deficits will therefore be quantified for each of these sub-groups. Data for patients with DAT will

be taken from an independent meta-analysis (Henry et al., in press). PD patients were only permitted to contribute to the 'demented' or 'non-demented' analyses where the dementia status of the patients was specifically indicated in the study.

METHODS

Sample of Studies

A computer-based search involving the *Web of Science*, *Psych Lit CD-ROM*, and *Science Direct* databases was undertaken, using the following terms as search parameters; *letter fluency*, *FAS*, *semantic fluency*, *category fluency*, *controlled oral word association*, *COWA(T)*, *word fluency verbal fluency*, *oral fluency*, *phonemic fluency*, *executive test*, and *frontal test*. In addition, a manual search of most issues of the journals *Journal of the International Neuropsychological Society*, *Brain*, *Neuropsychology*, *Clinical Neuropsychologist*, *Neuropsychologia*, *Neuropsychiatry, Neuropsychology and Behavioural Neurology*, *Journal of Neuropsychiatry and Clinical Neurosciences*, and the *Journal of Clinical and Experimental Neuropsychology* was conducted. These journals were selected as they were considered to be the most relevant to the current area of research (i.e., neuropsychological deficits in Parkinson's disease). The fact that most but not all issues were searched manually unfortunately reflects the very real problem that the libraries accessible to us had only incomplete collections of certain journals, and in particular very early copies of certain journals were often not available. A systematic method of search through these journals was adopted, with every page checked for references to measures of verbal fluency. The search was completed in October 2002.

The inclusion criteria were (1) the patient group had to consist entirely of adults with PD; (2) the study had to include a healthy control group free from neurological or psychiatric disease; and (3) a measure of phonemic, semantic, intra- or extra-alternating fluency. Effect size estimates for premorbid IQ, current VIQ, Digit Symbol, BNT, WCST CC and WCST PE were derived from studies that also reported verbal fluency results. For inclusion, the study must also have presented *precise* statistics convertible to effect size *r* (i.e., the *M* and *SD* for the patient and control group separately, or precise statistical test results, *F*, *t*, or Z). Since an effect size expresses a directional relationship, only statistical test results based on 1 degree of freedom could be used to derive effect sizes (Rosenthal, 1994). Imprecise statistical test results were also not included (i.e., where it was simply stated that $p < .05$ or $p < .01$, etc.; Le Bras et al., 1999; Oyebode et al., 1986). Finally, studies had to have been published in English in a journal.

Statistical Analysis

Meta-analysis is a rigorous, quantitative alternative to the traditional review process, as it involves statistical integration of results. The basis of this methodology is the effect size, a standardised statistic that quantifies the magnitude of an effect. Two basic types of metric exist that can be used to quantify effect size, known as the *r*- and the *d*-families. Although mathematically equivalent, they are associated with different interpretations of what the effect size represents. Whilst exemplars of the *r* family characterise the degree of correlation between two variables, e.g. the point-biserial correlation between group membership (i.e., presence or absence of PD), and the variable of interest (i.e., performance on the cognitive measure of interest), *d* family members exemplify this relationship in terms of the standardized difference between these two variables calibrated in terms of the standard deviation. As a consequence of its greater generality of interpretation, consistency of meaning and more salient practical meaning, *r* is the more useful effect size estimate (see Rosenthal & DiMatteo, 2001), and thus, in the present study this effect size was employed.

It should be noted that because the correlation coefficient is associated with a slight bias, Fisher (1928) derived a transformation of *r* that Snedecor and Cochran (1989) have recommended should be employed during statistical analyses in preference to *r*. However, this transformed estimate is itself associated with a bias, and in a Monte Carlo analysis, Field (2001) reported that for random effects meta-analytic models, transformed effect-size estimates produced substantial upward biases of a larger magnitude than the corresponding downward biases associated with untransformed correlation coefficients. Thus, in the present study, untransformed correlation coefficients have been employed for statistical analyses.

For each construct, effects were pooled to derive an estimate of the mean, with each effect weighted for sample size to correct for sampling error. To do so, the random effects meta-analytic model was selected in preference to the more commonly employed fixed effects model as it yields more generalisable parameter estimates. This is because, in the fixed effects model, the mean is presumed to reflect a *common* underlying effect parameter that gives rise to the sample observations. However, in the random effects model the mean represents a hyperparameter, as it allows for substantive differences beyond sampling error that differentiate the effects contributing to each respective mean (Raudenbush, 1994).

Statistically, the crucial difference between these methodologies is in the calculation of standard errors and confidence intervals, which for the random effects model are typically larger. The National Research Council (1992) argues that the fixed effects model should be the exception rather than the rule, as it may lead to inappropriately strong conclusions. Thus, although more technically demanding, it was considered important to use the random effects model in the present work.

To estimate the degree of heterogeneity of the effects contributing to each mean, the homogeneity statistic *Q* and the random effects variance (σ_θ^2) were estimated, as well as the *SD* of random effects, and the 95% confidence intervals

(*CI*) within which random effects can be expected to fall. *Q* quantifies within-group heterogeneity (i.e. the degree to which the studies contributing to each respective mean can be regarded as homogenous). If the *Q* statistic associated with a mean effect is significant, this suggests that there are substantive differences between the studies contributing to that particular mean. In contrast, a non-significant estimate of *Q* suggests that once sampling error has been removed, no substantive differences between the studies contributing to the respective mean in question remain (i.e. the null hypothesis of homogeneity of effects cannot be rejected).

It was also important to test whether the difference in the magnitude of mean effects between, for instance, phonemic versus semantic fluency, was statistically significant. However, there is no agreed method for statistically comparing mean effects using the random effects meta-analytic model. A particular difficulty is whether the degrees of freedom (*df*) in such analyses should be based on *N* (the number of participants) or *K* (the number of studies). In the present work, a relatively large number of studies were included, and therefore, *t* tests were computed using the more conservative *K* as the *df*.

Since dementia status will moderate the magnitude of deficits across individual studies, for each statistical comparison, only studies that assessed *both* variables of interest were included. For example, although in total 80 PD groups were tested on phonemic fluency, and 66 PD groups on semantic fluency, since only 50 groups were assessed on both phonemic and semantic fluency, when conducting inferential statistics to compare phonemic and semantic fluency, *only* data from these 50 groups were permitted to contribute to the analyses. This ensured that the participants being compared upon the two measures were equated for dementia severity (i.e., it is exactly the same participants being compared upon each of these measures).

It should also be noted that because the same participants were compared upon each measure, paired *t* tests were employed for all statistical comparisons. Mean effects were also calculated for each of the non-fluency variables identified (premorbid IQ, current VIQ, Digit Symbol, BNT, WCST CC and WCST PE) and compared with the corresponding effects for phonemic and semantic fluency. Again, to ensure that dementia severity was controlled for, only studies that assessed *both* the fluency and non-fluency variable of interest were included in each comparison.

Finally, the null hypothesis that the mean effect size is zero was tested with the statistic *Z*; if the value of *Z* exceeds 1.96, this indicates that the mean effect differs significantly from zero at the .05 level. To interpret how important a particular effect was in practical terms, Cohen's (1977) guidelines were adopted. These suggest that a correlation of .1 should be regarded as representing a small effect, .3 as medium, and .5 as large. In addition, squares of the effect size multiplied by 100 were also presented as these latter quantities represent the percentage of the variance accounted for (*PVAF*) by group membership (i.e., the presence of PD versus being a member of the healthy adult population) on a measure of interest. It should be noted that for inferential statistics comparisons were made using the *PVAF* by group membership upon each of the measures of interest because the difference between effect sizes is non-linear as *r* increases and thus *PVAF* is the more appropriate index when comparing variables.

RESULTS

Participant Characteristics

Sixty-eight studies published between 1983 and 2002 met the inclusion criteria specified, and in total, data from 2644 PD patients and 2000 controls contributed to these analyses. References for the 68 studies included in this meta-analysis are provided in the Appendix. Patients and controls did not differ significantly in terms of age ($M = 65.01$, $SD = 6.97$ *vs.* $M = 63.16$, $SD = 8.13$ respectively) or education ($M = 12.90$, $SD = 2.14$ *vs.* $M = 12.80$, $SD = 2.23$, respectively). However, a significantly higher proportion of the patient group were male (63.27% *vs.* 47.85% male, respectively, $p < .001$). For patients with PD, the mean Hoehn and Yahr (1967) score, an index of disease severity that categorises level of disability according to stages between 1 (*mild disability*) and 5 (*complete invalidism*), was 2.32 ($SD = 0.52$). The mean duration of illness was 5.66 ($SD = 2.83$) years.

Effect Sizes for Patients With PD Relative to Healthy Controls

Table 1 presents estimates of the mean effects for phonemic and semantic fluency, their variability, and practical importance in terms of the *PVAF* for studies that include *both* of these measures. In addition, mean effects are presented for premorbid IQ, current VIQ, Digit Symbol, BNT, WCST CC and WCST PE, calculated using only those studies that included the particular non-fluency measure of interest *in addition* to phonemic or semantic fluency. As noted previously, this methodology ensures that exactly the same participants are contributing to the mean effects for the two variables of interest. This is particularly important given that, as expected, the magnitude of the deficits for phonemic and semantic fluency in terms of the *PVAF* were both substantially and significantly related to dementia severity as measured by the Mini Mental State Examination (MMSE; Folstein et al., 1975); phonemic: $r = -.77$, $K = 41$, $p < .001$, semantic: $r = -.67$, $K = 29$, $p < .001$.

Thus, it can be seen in Table 1 that for *each* non-fluency measure, for instance premorbid IQ, *two* mean effects have been calculated; one for studies that also assess phonemic fluency ($r = .14$; $K = 19$), and one for studies that also assess semantic fluency ($r = .08$, $K = 17$). Each fluency mean effect was also re-calculated for these comparisons. For comparisons with premorbid IQ, current VIQ, Digit Symbol, BNT, WCST CC and WCST PE the mean effects

Table 1. Performance on phonemic fluency (PF), semantic fluency (SF), and other cognitive measures for PD patients *versus* healthy controls

					95% *CI*s of mean							95% *CI*s of mean effects		
	M	*K*	*N***	*SE*	Lower	Upper	*Z*	*PVAF*	*Q*	ϕ_θ^2	*SD*	Lower	Upper	
Studies with PF														PF *M*
Semantic fluency	*.37*	50	1603	.037	.30	.44	10.1*	13.7	316.7*	.053	.229	−.08	.82	*.33* (*K* = 50)
Premorbid IQ	*.14*	19	468	.045	.05	.23	3.1*	1.9	35.0*	.018	.133	−.12	.40	*.31* (*K* = 19)
Current VIQ	*.24*	25	608	.070	.10	.37	3.4*	5.5	173.4*	.098	.314	−.38	.85	*.24* (*K* = 25)
Digit Symbol	*.39*	8	204	.063	.27	.52	6.2*	15.5	15.9*	.017	.130	.14	.65	*.24* (*K* = 8)
BNT	*.34*	28	659	.048	.25	.43	7.2*	11.7	122.3*	.047	.216	−.08	.76	*.31* (*K* = 28)
WCST CC	*.35*	23	778	.030	.29	.40	11.7*	12.0	30.4	.005	.072	.21	.49	*.20* (*K* = 23)
WCST PE	*.33*	17	418	.031	.27	.39	10.4*	10.8	6.6	–	–	–	–	*.23* (*K* = 17)
Studies with SF														SF *M*
Phonemic fluency	*.33*	50	1603	.036	.26	.40	9.1*	10.6	274.2*	.049	.222	−.11	.76	*.37* (*K* = 50)
Premorbid IQ	*.08*	17	461	.068	−.05	.21	1.2	0.6	71.4*	.058	.241	−.39	.55	*.37* (*K* = 17)
Current VIQ	*.23*	21	507	.080	.07	.38	2.9*	5.2	162.2*	.110	.332	−.42	.88	*.30* (*K* = 21)
Digit Symbol	*.42*	4	64	.085	.26	.59	5.0*	18.0	4.0	.007	.085	.26	.59	*.43* (*K* = 4)
BNT	*.37*	21	535	.059	.25	.48	6.3*	13.6	113.3*	.057	.238	−.10	.83	*.38* (*K* = 21)
WCST CC	*.31*	21	685	.033	.25	.38	9.4*	9.8	28.7	.006	.080	.16	.47	*.33* (*K* = 21)
WCST PE	*.30*	19	432	.031	.24	.36	9.7*	9.2	7.5	–	–	–	–	*.37* (*K* = 19)

*$p < .05$.
***N* refers to the patient group
– indicates that the random effects variance has been estimated to be zero.
Note. For conducting inferential statistics, the mean effects for PF and SF were recalculated for each comparison of interest. For example, only eight studies included both PF and Digit Symbol. In addition to calculating the mean effect for Digit Symbol from these eight studies ($r = .39$), the mean effect for PF was also recalculated based *only* on these eight studies (i.e. $r = .24$). Thus, in each comparison exactly the same participants have been tested upon each of the measures of interest, 'controlling' for any substantive differences between studies, such as in level of dementia severity.

for phonemic fluency were estimated to be .31 ($K = 19$), .24 ($K = 25$), .24 ($K = 8$), .31 ($K = 28$), .20 ($K = 23$) and .23 ($K = 17$) respectively; the corresponding *semantic* fluency mean effects were .37 ($K = 17$), .30 ($K = 21$), .43 ($K = 4$), .38 ($K = 21$), .33 ($K = 21$) and .37 ($K = 19$), respectively.

With the exception of premorbid IQ for studies that also include semantic fluency ($r = .08$), all the mean effects are significantly different from zero, and in terms of practical importance, at least small in magnitude. The *PVAF* by group membership ranges from 0.6% to 18.0%. All the mean effects are associated with significant heterogeneity with the exception of WCST CC and WCST PE (for studies that include phonemic fluency), and WCST CC, WCST PE and Digit Symbol (for studies that include semantic fluency).

The deficit for semantic fluency ($r = .37$) is significantly larger than the deficit for phonemic fluency ($r = .33$; $t = 2.53$, $df = 49$, $p = .015$). Although the difference in the absolute magnitude of these mean effects is not striking, it is important to emphasize that the *p* value is a conservative one due to the use of *K* rather than *N* for the degrees of freedom. Moreover, although the studies that contributed to each of these statistics were heterogeneous as indexed by the statistic *Q*, the small difference between these two mean effects cannot be attributed to the presence of a few outliers, as no outliers contributed to the mean effects for either phonemic or semantic fluency. However, to provide a more rigorous test of the possibility that the difference between phonemic and semantic fluency reflects the influence of extreme values, the studies contributing the 10 most extreme phonemic fluency effect sizes were omitted, and the mean effects for phonemic and semantic fluency re-calculated from the 40 remaining studies. The studies contributing the 10 most extreme semantic fluency effect sizes were then omitted, and the mean effects for phonemic and semantic fluency re-calculated from the remaining 40 studies. For both these analyses, the mean effect for phonemic fluency was calculated to be .33, and the semantic fluency mean .37. Thus, whilst the absolute difference between phonemic and semantic fluency may be regarded as relatively small, it also appears to be robust and not attributable to the presence of a few outlying studies or extreme values.

The effect sizes for phonemic and semantic fluency are both significantly larger than for *premorbid* IQ ($rs = .31$ *vs.* .14; $t = 2.21$, $df = 18$, $p = .040$; $rs = .37$ *vs.* .08; $t = 2.83$, $df = 16$, $p = .012$, respectively). However, the phonemic fluency deficit did not differ significantly from *current* VIQ ($rs = .24$ *vs.* .24; $t = 0.18$, $df = 24$, $p = .857$), and was significantly smaller than the deficit for Digit Symbol ($rs = .24$ *vs.* .39; $t = 2.65$, $df = 7$, $p = .033$). The semantic fluency deficit does not significantly differ from the deficits for VIQ ($rs = .30$ *vs.* .23; $t = 1.38$, $df = 20$. $p = .183$) or Digit Symbol ($rs = .43$ *vs.* .42; $t = 0.64$, $df = 3$, $p = .953$). The magnitude of the deficit for the BNT also does not differ

from the deficits for phonemic or semantic fluency (rs = .34 *vs.* .31; t = 0.63, df = 27, p = .535; rs = .37 *vs.* .38; t = 0.12, df = 20, p = .904, respectively).

Deficits in Shifting in PD

The WCST CC is significantly more impaired than phonemic fluency (rs = .35 *vs.* .20; t = 2.65, df = 22, p = .015), whilst WCST PE is also substantially more impaired than phonemic fluency, although this difference failed to attain significance (rs = .33 *vs.* .23; t = 1.66, df = 16, p = .117). Relative to semantic fluency, the deficits for WCST CC and WCST PE do not differ significantly (rs = .33 *vs.* .31; t = 0.25, df = 20, p = .607; rs = .37 *vs.* .30, t = 1.09, df = 18, p = .291, respectively).

For studies that assess both semantic intra-alternating fluency and standard semantic fluency (K = 6), the deficit for the former is substantially larger; rs = .34 *vs.* .20. However, for studies that assess both phonemic intra-alternating fluency and standard single-condition phonemic fluency (K = 4), there is virtually no difference in the mean effect sizes (rs = .12 and .13, respectively). Finally, for studies that assess both *extra*-alternating and semantic fluency (K = 3), the deficit for the latter is slightly larger (rs = .36 *vs.* .29); this is also true of extra-alternating relative to phonemic fluency in studies that assess both of these measures (K = 3; rs = .36 *vs.* .32).

Demented *Versus* Non-Demented PD and DAT

The *difference* in terms of the *PVAF* (Δ*PVAF*) by group membership upon semantic versus phonemic fluency is not significantly related to mean scores on the MMSE (r = −.02, K = 25, p = .920). Thus, this suggests that the relative prominence of semantic memory storage and executive dysfunction is equivalent in demented and non-demented PD.

To provide a more visual illustration of the relationship between dementia severity and deficits upon tests of phonemic and semantic fluency, in Table 2 mean effects have been presented for phonemic and semantic fluency, stratified according to dementia status. It can be seen that for the mixed dementia, non-demented, and demented groups, the *relative* magnitude of the phonemic and semantic fluency deficits are comparable (i.e., rs = .33 *vs.* .37, .21 *vs.* .24 and .64 *vs.* .67 for phonemic and semantic fluency, respectively). Although the heterogeneity associated with the mean effects for the demented and non-demented sub-groups is substantially reduced relative to the mixed dementia group, all estimates of Q remain significant.

The *PVAF* by phonemic and semantic fluency for non-demented and demented PD patients is illustrated in Figure 1 alongside the corresponding values for patients with DAT. For the DAT analyses, data is taken from Henry et al.'s (in press) meta-analysis, in which 153 studies with a total of 15,990 participants contributed. It can be seen that demented and non-demented PD patients differ quantitatively but not qualitatively, as the lines for the two groups are parallel, indicating that the relative prominence of phonemic and semantic fluency deficits are comparable for the two groups. However, relative to patients with DAT, demented PD patients are substantially more impaired on phonemic fluency, but substantially less impaired on semantic fluency.

Table 2. Mean fluency effect sizes for mixed-dementia status, non-demented, and demented PD patients, and for patients with DAT

					95% *CI*s of mean							95% *CI*s of mean effects	
Patient type	*M*	*K*	*N**	*SE*	Lower	Upper	*Z***	*PVAF*	Q**	σ_θ^2	*SD*	Lower	Upper
Mixed PD													
Phonemic fluency	*.33*	50	1603	.036	.26	.40	9.1	10.6	274.2	.049	.222	−.11	.76
Semantic fluency	*.37*	50	1603	.037	.30	.44	10.1	13.7	316.7	.053	.229	−.08	.82
Non-demented PD													
Phonemic fluency	*.21*	28	1028	.030	.15	.27	7.1	4.4	44.0	.009	.092	.03	.39
Semantic fluency	*.24*	28	1028	.029	.19	.30	8.2	5.9	45.0	.009	.094	.06	.43
Demented PD													
Phonemic fluency	*.64*	11	207	.033	.57	.70	19.2	40.8	17.2	.005	.069	.50	.78
Semantic fluency	*.67*	11	207	.035	.60	.73	19.0	44.3	21.7	.007	.081	.51	.82
DAT													
Phonemic fluency	*.57*	70	2674	.024	.52	.62	23.8	32.6	600.3	.033	.180	.22	.92
Semantic Fluency	*.73*	70	2674	.017	.69	.76	42.7	52.7	630.6	.016	.128	.47	.98

*N refers to patient group.
**All values of Q and Z significant (i.e., ps <.05).
Note. DAT data taken from Henry et al. (in press).

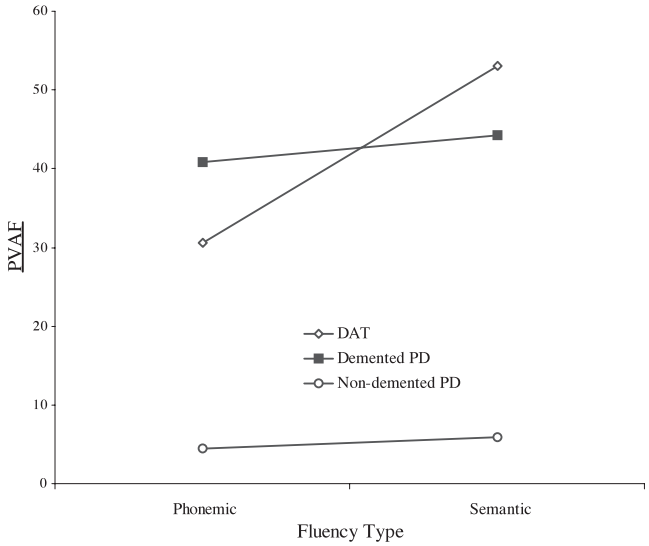

Fig. 1. PVAF in performance on phonemic *versus* semantic fluency by the presence of non-demented PD, demented PD and DAT (DAT data taken from Henry et al., in press).

Assessing the Possibility of Publication Bias

A number of validity threats have been identified that may lead to imprecise conclusions in both non-quantitative and meta-analytic reviews. Particularly problematic is the "file drawer problem," which refers to the fact that significant results are more likely to be published than non-significant results (Easterbrook et al., 1991). To assess whether this bias posed a threat to the results of the present study, funnel plot diagrams were constructed for each of the fluency and non-fluency measures of interest. In these diagrams, sample size is plotted against the corresponding study-level effect; if statistically non-significant results have been discriminated against, there should be a relative absence of studies with small sample sizes that report weak effects. For none of the variables was there evidence of this bias operating.

DISCUSSION

Quantifying Verbal Fluency Deficits in PD

Although the presence of PD was associated with deficits upon tests of phonemic and semantic fluency that were significantly different from zero (both ps $< .05$), and moderate in practical importance according to Cohen's (1977) criteria, for studies that assessed both measures, the semantic fluency deficit was significantly larger, consistent with the possibility that PD is associated with particular difficulties with semantic memory (see Henry & Crawford, 2004).

Relative to current VIQ, the deficits for phonemic and semantic fluency did not differ significantly, suggesting that poor verbal fluency performance may simply reflect a general impairment of verbal abilities. Moreover, the phonemic fluency deficit was significantly smaller than the deficit for Digit Symbol, whilst the deficits for semantic fluency and Digit Symbol did not differ. Unfortunately conclusions relating to the possibility that bradyphrenia or cognitive slowing underlies many of the cognitive deficits associated with PD must necessarily be tempered by the fact that a relatively small number of studies contributed to the analyses involving Digit Symbol. Nevertheless, the current findings are consistent with this possibility.

However, as noted previously, the phonemic fluency deficit was significantly smaller than the deficit for semantic fluency. Since these measures have been found to impose comparable demands upon executive processes (Henry & Crawford, 2004), and are also presumably equivalent in terms of their relative dependence upon cognitive speed, this suggests that PD may be associated with a particular deficit in semantic memory. Indeed, confrontation naming is considered to be very sensitive to semantic memory (Hart, 1988), but imposes only minimal demands upon speed and effortful retrieval. In the present study, confrontation naming was associated with comparable deficits relative to both phonemic and semantic fluency. Thus, even when the requirements for speed and effortful retrieval have been almost entirely removed, there is still evidence of a deficit in semantic memory.

However, a semantic memory deficit may reflect *either* a degradation in the integrity of semantic memory (i.e. semantic representations may be abnormally organized or lost) as is thought to be the case in DAT, or instead, specific difficulties with the retrieval of semantic information. Whilst there is evidence that phonemic and semantic fluency impose equivalent demands upon *effortful* retrieval processes (Henry & Crawford, 2004), it is probable that the retrieval of semantic items in semantic fluency tasks may depend on additional retrieval mechanisms that are different to those required in phonemic retrieval (e.g., if depth of encoding differs, retrieval mechanisms may differ as well).

For patients with DAT, there is a great deal of evidence that a degradation of the semantic store underlies the semantic memory deficit observed. Nebes (1989), for instance, points out that semantic tasks that impose similar demands upon the retrieval of semantic information are not always comparably impaired. Moreover, multidimensional scaling has revealed DAT to be associated with a distortion in semantic space (Chan et al., 1993) indicating that there is a fundamental difference in the organisation of DAT patients' semantic representations relative to healthy elderly. Thus, in addition to the finding that semantic fluency is substantially more impaired than phonemic fluency (Henry et al., in press), a great deal of other evidence has accumulated that suggests that DAT is associated with a disorganization or a degradation of semantic representational knowledge.

In contrast, the most prominent position in the literature is that the semantic memory deficit in PD reflects a problem with the *retrieval* of information from semantic memory. Raskin et al. (1992a) and Auriacombe et al. (1993), for

instance, have suggested that whilst PD patients possess intact storage systems, access to these semantic representations is disrupted, although they differ with regard to the level of specificity of this retrieval deficit. Thus, whilst Auriacombe et al. (1993) suggests that this retrieval deficit is the level of accessing phonological shapes, Raskin et al. (1992a) have argued that it is at the level of semantic information in general. Other evidence that a retrieval deficit underlies the semantic memory deficit in DAT has been presented by Troyer and Moscovitch (1996), who found that patients with PD, but not patients with DAT, produced clusters of a normal size on tests of phonemic and semantic fluency. In addition, PD patients switched between clusters significantly less often than healthy controls. Thus, although it is not possible to rule out the possibility that PD is also associated with a degradation of the semantic store, the present authors favour the interpretation that the semantic memory deficit reflects a retrieval deficit because there is a great deal of other evidence that is consistent with this possibility.

However, it should be noted that in interpreting data of these sort, there is the possibility of identity fallacies. Thus, whilst it is suggested that a similar pattern in phonemic and semantic fluency effect sizes for patients with PD and patients with temporal lobe damage provides evidence that PD patients are like temporal patients and therefore have greater difficulties with semantic memory than with executive control processes, other interpretations of these data are possible, and a phonemic deficit cannot be ruled out. Relatedly, if we were to find a profile of phonemic and semantic fluency deficits in PD that paralleled the profile for frontal patients, this would not constitute evidence of either frontal or executive dysfunction, but could only be regarded as consistent with this possibility.

In addition, it is important to note that in the present study there was a significant difference between the gender distribution of patients and controls, and thus a caveat is that there may have been a confounding by gender in the patients-*versus*-controls comparisons. Indeed, previous literature has indicated that performance on different cognitive measures including tests of phonemic and semantic fluency may be influenced by gender. However, the direction of these effects has not proven consistent, and typically any effects observed are small in magnitude (see Lezak, 1995).

Performance on Measures that Impose Demands Upon Switching

Relative to phonemic fluency, performance on the WCST CC was significantly more impaired, and the WCST PE was also substantially more impaired than phonemic fluency, although this difference failed to attain significance. Relative to semantic fluency, the deficits for WCST CC and WCST PE did not differ significantly, consistent with there being particular difficulties with both semantic memory and in switching. However, since performance on the WCST may be impaired for reasons unrelated to difficulties with task shifting (i.e. as with all cognitive measures, the WCST is multifactorial), it was also important to assess whether fluency measures that additionally impose substantial demands upon shifting were more impaired relative to standard fluency measures.

The present study found that tests of extra-alternating fluency are associated with slightly larger deficits than standard measures of fluency, whilst measures where switching is based solely on phonemic criteria (i.e., intra-alternating phonemic fluency) were not associated with larger deficits than standard tests of phonemic fluency. However, semantic intra-dimensional shifting was substantially more impaired than standard tests of semantic fluency. Thus, it may be that it is not the ability to shift *per se* that is disproportionately impaired, nor as Downes et al. (1993) has suggested, the ability to shift extra-dimensionally. Instead, the present results suggest that it is the requirement to shift between different semantic dimensions, again suggesting that for patients with PD there are particular problems with tests that impose substantial demands upon semantic memory. This would also be consistent with the deficits on the WCST observed, since this measure requires participants to shift between different semantic concepts. However, it is important to stress that relatively few studies assessed both alternating and standard measures of fluency, and thus contributed to these particular analyses, and this points to an important area of future research.

Demented PD *Versus* Non-Demented PD and DAT

It was also important to address whether the relative prominence of phonemic and semantic fluency deficits differs as the disease progresses, and thus whether the relative contributions of executive versus semantic memory deficits are comparable at each stage of the disease. As noted previously, it has been suggested that dementing pathology in PD is associated with a qualitative change in cognition, and in particular, as mental state deteriorates, a reduction in the ability to use sub-category structure to facilitate retrieval (Azuma et al., 1997).

In the present study it was found that for demented PD patients, both semantic and phonemic fluency were associated with deficits large in magnitude whilst for non-demented PD patients both these deficits were small to moderate. However, for both groups, the deficit for semantic fluency was the larger of the two (rs = .67 *vs.* .64, and rs = .24 *vs.* .21, respectively), with the relative prominence of the deficits upon the two measures equivalent for the two groups (i.e. as was shown in Figure 1, the deficits for demented and non-demented PD patients upon these measures were parallel).

Indeed, although dementia severity was significantly related to the magnitude of the deficits associated with measures of both phonemic and semantic fluency in terms of the *PVAF* (rs = $-.77$ and $-.67$ respectively, both

*p*s $< .001$), the *difference* in the *PVAF* by semantic and phonemic fluency was not related to mean scores on the MMSE ($r = -.02$, $p = .92$). Thus, the present results indicate that demented and non-demented PD patients differ quantitatively but not qualitatively in terms of the relative prominence of deficits upon the two types of fluency, suggesting that the relative prominence of deficits in executive functioning and semantic memory may also be equivalent.

Questions have also been raised with respect to the comparability of dementing pathology in PD and DAT. Relative to "cortical" dementias such as DAT, "subcortical" dementias such as PD are thought to be associated with more pronounced subcortical neuropathological abnormalities (Albert, 1978; Cummings, 1990). It remains a central issue whether such etiologically distinct forms of dementia can be differentiated in terms of their deficit profiles. A common assertion is that whilst cortical dementias are typified by a pattern of worse semantic relative to phonemic fluency performance, subcortical dementias are typified by the opposite deficit profile, and evidence consistent with this perspective has been found in studies involving DAT, HD and progressive supranuclear palsy patients (Hodges et al., 1990; Rosser & Hodges, 1994).

As noted, the present results indicate that as with DAT, demented PD patients are more impaired on semantic fluency than fluency based on orthographic criteria. However, Henry et al. (in press) found that for patients with DAT, the mean difference in terms of the *PVAF* by phonemic and semantic fluency was 20.8%, substantially larger than the corresponding difference for demented PD (3.9%). Thus, although demented patients with PD and DAT are both relatively more impaired on semantic than phonemic fluency, for the latter group the distinction between the two fluency types is substantially more pronounced. Thus, although patients with PD appear to experience particular difficulties with tasks reliant upon semantic memory, for patients with DAT the corresponding difficulties can be regarded as substantially greater.

Substantive Versus Artefactual Variance

One of the aims of the present study was to quantify the degree to which the presence of PD is related to performance on tests of phonemic and semantic fluency using meta-analytic techniques. The present results are particularly useful because they emphasise the magnitude of the effects. Although researchers are strongly encouraged to report effect sizes for their individual studies (American Psychological Association, 2001), this is rarely done so in practice, yet is far more informative than simply reporting whether a particular effect is significant or not. Moreover, because using meta-analysis it is possible to integrate effects across studies that differ in both the participants sampled and methodology employed, the effects reported can be considered to be very reliable, robust estimates of the corresponding parameters of interest, which is particularly important given the inconsistencies noted in the primary studies contributing to these analyses.

Meta-analysis however, has not escaped criticism. In particular, problems of heterogeneity have been raised since meta-analysts average across studies in which the variables of interest are not uniform. Thus, it may be that by collapsing across different studies, important differences are obscured. It has also been suggested that in the calculation of mean effects, bad studies as well as good studies contribute, and that where there is "garbage in" there will be "garbage out." However, as Strube et al. (1985) note, reviews based on non-quantitative methodology are equally susceptible to both of these criticisms. Moreover, in contrast to traditional narrative reviews, using meta-analysis it is possible to quantify the heterogeneity of the effects contributing to each particular mean, with the influence of sampling error removed, and identify bad or outlying studies using rigorous statistical methodology.

In the present study the mean effects for almost all of the cognitive measures assessed in the present study were associated with significant heterogeneity ($p < .05$). Since sampling error, the most serious source of artefactual variance, had been removed by weighting for sample size, this suggests that substantive differences between studies remain. It is probable that a great deal of the variance reflects differences in dementia severity. As noted, patients' mean MMSE score was significantly and substantially correlated with the magnitude of the deficits for both phonemic and semantic fluency.

It might be argued that for many of the mean effects significant heterogeneity was almost inevitable given that a relatively large number of individual effects contributed. As Hedges and Olkin (1985) point out, in such circumstances, relatively minor differences in the values of each effect may be associated with a significant homogeneity statistic. However, the 95% *CI*s of the random effects for the majority of the mean effects calculated can be considered to be large. Moreover, although sub-dividing patients according to dementia status removed a considerable amount of variability, significant heterogeneity remained for both measures of fluency in the non-demented and demented PD groups. It is therefore important to reiterate that whilst patients with PD are *generally* more impaired on semantic relative to phonemic fluency, it remains plausible that for certain sub-groups, the reverse, or a pattern of comparable impairment may emerge, and indeed this has been reported in some studies (McDonald et al., 1996; Piatt et al., 1999; Reid et al., 1989).

Thus, the present results do not rule out the possibility that distinct sub-types exist that differ with respect to their level of executive impairment. It has, for instance, been suggested that PD patients with major depression are disproportionately more impaired upon measures of executive functioning (Starkstein et al., 1989), whilst Flowers et al. (1995) found that disease severity is an important moderator of the magnitude of deficits upon tests of both phonemic and semantic fluency. However, much of this variance will

be bundled up *within*, rather than between studies, and thus the specific influence of each cannot be explored in the present study (for example, there were insufficient studies that were restricted only to mild *vs.* severe patients, etc.). The heterogeneity statistic *Q* quantifies the degree of heterogeneity between studies but cannot address the degree of heterogeneity within each of the studies contributing to a mean. However, it is recommended that if future primary research breaks down their samples more fully, meta-analysis should be conducted to address which variables moderate performance on tests of verbal fluency.

SUMMARY AND CONCLUSIONS

PD patients were significantly more impaired on semantic relative to phonemic fluency. Neither of these deficits qualified as differential deficits relative to measures of psychomotor speed or verbal intelligence, and thus patients with PD do not appear to perform poorly on these tasks as a consequence of executive dysfunction. However, since the deficit for the BNT, a measure that imposes only minimal demands upon cognitive speed and effortful retrieval, was equivalent in magnitude to the deficits upon these two types of fluency, it is suggested that PD is associated with a particular deficit in semantic memory. There is also some evidence that patients with PD may suffer a specific deficit in cognitive set-shifting. Finally, the difference between demented and non-demented PD patients in terms of the relative prominence of deficits upon these two measures is quantitative but not qualitative, but for DAT the difference in terms of the *PVAF* between these two types of fluency was substantially greater than for PD patients with concomitant dementia.

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APPENDIX

Articles included in the meta-analysis:

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