

## Is there a differential deficit in fronto-executive functioning in Huntington's Disease?

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### Abstract

Three measures of executive functioning, initial-letter verbal fluency (e.g., Lezak, 1995), the Modified Card Sorting Test (MCST; Nelson, 1976) and the Cognitive Estimates Test (CET; Shallice & Evans, 1978), were administered to a sample of patients with Huntington's disease (HD) ( $N=23$ ) and a healthy sample ( $N=22$ ) matched for age, sex, years of education and social class. The HD sample exhibited highly significant deficits on all three tasks ( $p<.001$ ). To determine if these qualified as *differential* deficits, the performance of the two samples on the Verbal Scale and Full Scale of the WAIS-R was also examined. These latter variables, which are relatively insensitive to the effects of frontal lesions, were used as measures of general intellectual functioning against which to compare the executive tasks. Using the method of testing for differences in non-independent correlations (Baron & Treiman, 1980), there was no evidence of differential deficits on the executive tasks; i.e. the executive deficits did not significantly exceed the deficits on the WAIS-R.

Hierarchical regression analysis was employed to determine whether executive performance could account for between-group variance in verbal learning performance as measured by recall on trial five of the California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan, & Ober, 1987). A composite of the executive tasks accounted for 91% of the total variance in CVLT performance and all but a trivial proportion (0.003%) of the between-group variance. Executive task performance remained a highly significant predictor of memory performance after controlling for WAIS-R scores. It is concluded that executive deficits in HD do not qualify as differential deficits but nevertheless make a specific and very substantial contribution to poor verbal memory performance in HD.

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## Introduction

Huntington's Disease (HD) is a progressive neurodegenerative disease that is transmitted genetically via an autosomal dominant mechanism with complete lifetime penetrance. The most prominent neuropathological features are neuronal loss and gliosis in the striatum, particularly the caudate nucleus. The behavioural syndrome is characterized by uncontrollable choreiform movements, cognitive deterioration and changes in personality and affect (Brandt, 1991; Lishman, 1987).

Although impairment in many cognitive domains has been documented (see Brandt, 1991) it has commonly been suggested that executive deficits associated with prefrontal dysfunction (e.g. planning, shifting cognitive set, self-monitoring) is a particularly striking feature of HD (Caine, Hunt, Weingartner, & Ebert, 1978; Lezak, 1995; Starkstein et al., 1988). Caine et al. (1978) for example suggest that, "patients with Huntington's disease demonstrate a loss of cortical, executive function similar to that found in classical frontal lobe patients" (p. 384) and they refer to the existence of the "Huntington's disease frontal syndrome" (p. 384). Similarly, Lawrence, Sahakian, Hodges, Rosser, Lange and Robbins (1996), suggest that HD should be characterised as a "fronto-striatal" dementia (Robbins et al., 1994). These executive hypotheses stem from the following observations. Firstly, the cortical connections of the caudate nucleus are primarily with prefrontal cortex. As the caudate is the principal site of pathology in HD, the frontal-caudate loop system will be compromised (Starkstein et al., 1988). There is also some evidence that the prefrontal regions may exhibit greater atrophy than other cortical regions (Lezak, 1995). Secondly, clinical observation and patient and carer reports have been interpreted as revealing a behavioural picture similar to that seen following frontal lesions (Caine et al., 1978). Thirdly, formal neuropsychological testing has provided some evidence of deficits in HD on executive tasks that lesion studies have shown to be sensitive to focal prefrontal damage. For example, there are reports of deficits on tests of verbal fluency (Butters, Wolfe, Granholm, & Martone, 1986), card sorting tasks (Josiasen, Curry, & Mancall, 1983; Lange, Sahakian, Quinn, Marsden, & Robbins, 1995), and planning tasks such as the Tower of London (Lange et al., 1995; Lawrence et al., 1996). However, it remains the case that the quantitative evidence for executive deficits is modest. As Lawrence, Sahakian and Robbins (1998) point out, "The notion that HD represent some form of dysexecutive syndrome has some ecological validity, given that many HD describe difficulties with organising their day-to-day activities, and appear behaviourally inflexible. It is somewhat surprising then that the literature on executive dysfunction is relatively sparse" (p. 381).

The aim of the present study is to contribute to an evaluation of the executive hypothesis in HD. As a first step in this evaluation we seek to confirm the presence of deficits on executive tasks. The task selected for this purpose were the Modified Card Sorting Test (MCST; Nelson, 1976), initial-letter Verbal Fluency (VF; e.g., Lezak, 1995) and the Cognitive Estimates Test (CET; Shallice & Evans, 1978). The first two of these tasks are amongst the best-validated measures of cognitive dysfunction following prefrontal damage (e.g., Parker & Crawford, 1992). The final test is one of the few tasks to be generated a priori by a theory of prefrontal function and is impaired following focal frontal damage (Shallice & Evans, 1978).

Confirmation of a deficit on executive tasks would, in isolation, provide only limited support for an executive hypothesis. HD patients exhibit impairment on many cognitive tasks that are not associated with prefrontal dysfunction (Brandt, 1991; Lange et al., 1995). Therefore, a more stringent test of the executive hypothesis requires an attempt to demonstrate that deficits on executive tasks are significantly greater than deficits on tasks that do not impose a high executive load. The Wechsler Adult Intelligence Scale - Revised (WAIS-R; Wechsler, 1981) was employed in the present study as a comparison standard for the executive tasks. The WAIS-R can be viewed as well suited to this purpose as it has impressive psychometric properties (Kaufman, 1990), samples a wide range of cognitive abilities (Kaufman, 1990), and yet is relatively insensitive to the effects of prefrontal dysfunction (Dempster, 1992; Lezak, 1995; Stuss & Benson, 1984; Walsh, 1991). As an example of this latter feature, Miller (1984) reported a substantial verbal fluency deficit coupled with preserved performance on the Verbal scale of the WAIS in a sample of patients with focal frontal lesions. Although the insensitivity of the WAIS-R and its predecessors to prefrontal dysfunction may have been exaggerated (Parker & Crawford, 1992; Shallice, 1988), the present rationale does not require that it should be entirely insensitive; simply that it is less sensitive than validated executive tasks. In the present study we used the Full Scale IQ as an index of general cognitive functioning. In addition, as at least two of the executive tasks (verbal fluency and Cognitive Estimates) have substantial verbal components, we also used the Verbal Scale as an index of general verbal functioning.

A further issue concerning executive deficits is the extent to which such deficits are responsible for the well-documented impairments of verbal learning in HD (see Brandt, 1991). A number of authors have suggested that impairment of verbal learning and memory in HD stems from an inability to initiate systematic search strategies for newly stored information (e.g., Brandt, 1985; Butters, 1984; Caine et al., 1978). This hypothesis arose from the consistent finding that a severe deficit in free recall is coupled with relatively normal recognition memory in HD. More direct empirical support is provided by Brandt (1985) who demonstrated problems with metacognitive control processes. In the present study this hypothesis will be examined by testing whether between-group (HD vs. controls) variance in verbal memory performance is accounted for by performance on the executive tasks.

Five hypotheses will be tested in the present study; (1) HD cases will exhibit statistically significant deficits on the three executive tasks when compared with matched controls; (2) These deficits will qualify as *differential deficits*, i.e. they will be significantly greater than the deficits on a measure of general intellectual functioning (the WAIS-R); (3) The HD sample will exhibit significantly greater error rates on the executive tasks than matched controls; (4) Executive task performance will account for the between-group variance in memory performance; and (5) Executive task performance will remain a significant predictor of memory performance after controlling for level of WAIS-R performance.

Testing for the presence of differential deficits is problematic (Chapman & Chapman, 1973). Many of the commonly employed statistical methods for their identification have been ruled as inappropriate (Strauss & Allred, 1987). The present study employs Baron & Treiman's

(1980) method which relies on the application of a test for a significant difference between non-independent correlations. In this method the correlation between the dichotomous variable of group membership (i.e., clinical versus control case) and a task hypothesized to expose a differential deficit is compared with the correlation obtained for a control task. If the former correlation is significantly higher than the latter, this would be consistent with the presence of a differential deficit (to determine significance the correlation *between* the tasks must be also be computed). Strauss & Allred's (1987) review of methods for identifying differential deficits endorsed this technique as avoiding major problems associated with other approaches. An example of the application of this method can be found in Crawford, Johnson, Mychalkiw and Moore (1997). These authors reported that, in a comparison of head-injured and healthy participants, the head-injured sample exhibited a differential deficit on the attention / concentration (or working memory) factor of the WAIS-R when compared with the deficits on summary IQs (FSIQ, VIQ and PIQ) and the other WAIS-R factors (verbal and perceptual organisation).

## Method

### Participants

The HD sample consisted of 23 individuals (8 males, 15 females). All cases met Folstein, Leigh, Parhad & Folstein's (1986) criteria for definitive HD, namely (1) chorea or the characteristic impairment of voluntary movement, which was not present at birth, was insidious in onset, and which had become gradually worse, and (2) a family history of at least one other member with these symptoms. Onset of symptoms was estimated to have occurred between 0 and 3 years prior to participation in the study for six of the HD cases, between 3-6 years for 11 cases and greater than six years for the remainder.

A healthy control sample, screened by interview for the absence of neurological and psychiatric disorder was recruited to match the HD sample in terms of age, years of education, social class, and sex ratio ( $N = 22$ ; 8 males, 14 females). As can be seen in Table 1,  $t$ -tests revealed that the mean age and the mean years of education in the HD and control samples did not differ significantly.  $F$  tests also revealed that the variances of these variables did not differ significantly between samples. Social class was coded from a participant's occupation, or previous occupation, using the Classification of Occupations (Office of Population, 1980). None of the HD or control participants were coded as social class 1. Because of small expected frequencies, participants coded as social class 2 and 3 were combined ( $n = 14$  for HD sample,  $n = 11$  for controls) as were those coded as social class 4 or 5 ( $n = 9$  for HD sample,  $n = 11$  for controls). A Chi-Square test revealed that the two samples did not differ significantly;  $\chi^2(1) N = 45 = 0.54, p > 0.1$ .

**Table 1. Mean age and years of education in HD and control samples**

	HD		Controls		<i>t</i>	<i>F</i>
	Mean	<i>SD</i>	Mean	<i>SD</i>		
Age	52.6	11.60	52.0	12.16	0.17, <i>p</i> >.05	0.08, <i>p</i> >.05
Education (years)	9.57	0.66	9.86	0.71	1.46, <i>p</i> >.05	0.14, <i>p</i> >.05

### Tests and Procedure

All participants completed a full-length WAIS-R according to standard procedures. Two measures were derived; the sum of scaled scores on the Verbal Scale and the sum of scaled scores on the Full Scale. These measures, rather than the IQs, were used in the interest of consistency as none of the other cognitive measures employed were age-corrected.

Initial letter verbal fluency was administered by asking participants to produce in ninety seconds, as many words as possible beginning with each of three letters (F, A and S) in turn. Participants were informed that any words were acceptable with the exception of proper nouns, numbers and the same words ending with different suffixes (e.g. fast, faster). To ensure comprehension, a practice trial with the letter C was administered with the examiner providing examples and asking participants to provide their own. Examples of incorrect words were also provided. Two variables were derived from this procedure, the sum of legitimate words produced across the three trials and the sum of rule break errors and perseverative errors (e.g. repeating a previously generated word).

The MCST (Nelson, 1976) was administered and scored according to standard procedures. This test is a variant on the Wisconsin Card Sorting Test (Grant & Berg, 1948; Heaton, 1981) and requires participants to sort cards according to three attributes (colour, form and number of items depicted). The sorting rule changes throughout the administration thereby assessing participants' ability to shift cognitive set. The MCST differs from the original Wisconsin in that (a) the cards to be sorted share a maximum of one attribute with a key card and (b) participants are told when the sorting rule is changed. Two variables were derived from this task; the number of cards sorted correctly and the number of card sorts that were classified as perseverative (a perseverative response is defined as an incorrect sort which follows the previous sorting rule). Following Nelson's (1976) directions, this latter measure was expressed as a percentage of the total number of incorrect sorts.

The CET was administered according to standard procedures. It consists of 15 questions for which precise answers are unavailable (e.g. what is the length of an average person's spine?). Therefore, in order to generate an appropriate response, participants must formulate a cognitive plan, execute it and check the reasonableness of their output. Responses on each item are scored using a four point scale derived from control data in which a score of zero represents normality and three is classified as "very extreme" (Shallice & Evans, 1978). In the standard scoring scheme high scores therefore indicate poor performance. In the interests of consistency and ease of interpretation these scores were reflected so that low scores indicated poor performance.

In order to study executive function as a predictor of memory performance the three executive tasks were combined into a single, equally weighted composite by converting scores on each to  $z$  scores and summing the  $z$  scores for each participant.

The California Verbal Learning Test (CVLT; Delis et al., 1987) was administered and scored according to standard procedures. This is a verbal list learning task in which free recall is examined over five presentation of the same list. This can be followed by recall of a new list and a number of tests of delayed recall and recognition of the original list. A large number of measures can be derived from the CVLT which, if all were used, raises the danger of either losing statistical power by controlling for inflation of the Type I error rate, or capitalizing on chance if such a control were not introduced. Therefore one measure was selected *a priori* for use as the dependent measure of verbal learning; recall performance on trial five.

### Statistical Analysis

William's (1959) significance test for differences between non-independent correlations was used to evaluate the hypothesis of differential deficits on the executive tasks. This test, which has been widely endorsed (Dunn & Clark, 1971; Howell, 1997; Steiger, 1980) yields a statistic that is distributed as  $t$  on  $N-3$  degrees-of-freedom; significance is assessed as for any  $t$ .

The hypothesis concerning memory performance was tested using hierarchical multiple regression models. In the first model the executive composite was entered first followed by the dummy variable of group membership. This tested whether executive performance was a significant predictor of CVLT performance and whether group membership predicted a significant proportion of additional variance. In the second model these two variables were entered in the same order but were preceded by the sum of scaled scores on the WAIS-R. In the third model the order of entry for the two cognitive measures was reversed; i.e. the WAIS-R was entered first followed by the executive composite, then group membership.

## Results

### Comparison of HD executive task performance with controls

Mean scores and  $SD$ s for the HD and control samples on the three executive tasks and on the two WAIS-R scales are presented in Table 2. The correlations between these five measures and group membership (HD vs. control) are presented in Table 3. It can be seen from Table 3 that the correlations of the executive tasks with group membership were all highly significant ( $p < .001$ ); thus the HD sample exhibited significant deficits on all three tasks. However statistically significant deficits ( $p < .001$ ) were also exhibited on the sum of scaled scores for the two WAIS-R scales.

**Table 2. Means and SDs for HD and control samples on three executive tasks and on the sum of scaled scores for the WAIS-R Verbal and Full Scales**

	Verbal Fluency	MCST correct	Cognitive Estimates	Verbal Scale	Full Scale
HD	12.91 (9.61)	15.70 (10.89)	12.43 (7.69)	33.17 (13.17)	50.83 (20.74)
Controls	38.27 (9.58)	33.73 (5.65)	5.36 (3.06)	57.77 (8.27)	98.41 (15.12)

**Table 3. Correlations of executive tasks with group membership and with the sum of scaled scores on the Verbal and Full Scale of the WAIS-R**

	Verbal Fluency	MCST Correct	Cognitive Estimates	Verbal Scale WAIS-R	Full Scale WAIS-R
Group membership	.81	.73	.52	.75	.80
Verbal	.84	.80	.72	-	-
Full	.88	.84	.72	-	-

Note: All correlations significantly different from zero ( $p < .001$ )

### Executive deficits as differential deficits

To determine if the deficits on the executive tasks qualified as differential deficits, the correlation between each of the executive tasks and group membership were compared with the correlation between the two WAIS-R indices and group membership. To determine if these correlations differ significantly it is necessary to factor in the correlation between the executive tasks and the WAIS-R indices. The latter correlations form the second and third rows of Table 3.

**Table 4. Results of testing for a differential deficit on executive tasks using a significance test for non-independent correlations.**

	Verbal Fluency		MCST		Cognitive Estimates	
	<i>t</i>	<i>p</i>	<i>t</i>	<i>p</i>	<i>t</i>	<i>p</i>
$r_{\text{exec}}$ vs. $r_{\text{viq}}$	1.04	ns	.041	ns	2.96	<.01
$r_{\text{exec}}$ vs. $r_{\text{fsiq}}$	0.07	ns	1.45	ns	3.99	<.001

Note: Degrees of freedom =  $(N - 3) = 42$  for all comparisons

The results of applying William's (1959) test for non-independent correlations are presented in Table 4. It can be seen that the correlations of the verbal fluency test and MCST with group membership did not differ significantly from the WAIS-R correlations with group membership. Significant differences were obtained for the Cognitive Estimates task but these were in the opposite direction to that which would be predicted from the hypothesis of differential deficits on executive tasks; i.e., the WAIS-R indices were significantly more sensitive to group differences than the Cognitive Estimates task.

### Error rates in HD and control samples

In the HD sample the mean number of errors on verbal fluency was 1.48 ( $SD = 1.59$ ) compared to a mean of 1.36 ( $SD = 1.47$ ) in controls. A Mann-Whitney  $U$  test revealed that the samples did not differ significantly ( $U = 241.5$ ,  $p > .1$ ). The mean percentage of perseverative errors on the MCST in the HD sample was 40.8 ( $SD = 37.57$ ) with a median of 29.0, compared to a mean of 21.6 ( $SD = 19.68$ ) and median of 19.0 in controls. A Mann-Whitney  $U$  test revealed that this difference was not significant ( $U = 183.0$ ,  $p > .1$ ).

**Table 5. Results of hierarchical multiple regression analyses examining the ability of executive performance to account for between-group variance in memory performance with (Model 2) and without (Model 1) prior entry of WAIS-R sum of scaled scores**

Variables	$R^2$	$R^2$ change	$F$ for $R^2$ change	$p$
<i>Model 1</i>				
Executive composite	.91	.9072	410.36	<.0001
Group (HD vs Control)	.91	.0003	0.14	ns
<i>Model 2</i>				
WAIS-R SSS	.68	.6821	90.11	<.0001
Executive composite	.91	.2313	109.55	<.0001
Group (HD vs Control)	.91	.0013	0.61	ns
<i>Model 3</i>				
Executive composite	.91	.9072	410.36	<.0001
WAIS-R SSS	.91	.0063	2.97	ns
Group (HD vs Control)	.91	.0013	0.61	ns

### Executive dysfunction as a predictor of between-group variance in memory

The mean score on trial 5 of the CVLT was 5.5 ( $SD = 2.87$ ) in the HD sample compared to 12.5 ( $SD = 2.43$ ) in controls. An independent samples  $t$ -test revealed that this difference was highly significant;  $t(44) = 8.92$ ,  $p < .001$ . The results from the three hierarchical multiple

regression analyses are presented in Table 5. It can be seen from the results for Model 1 that the executive composite predicted a very substantial proportion of the variance in memory performance (91%). Entry of group membership into the model did not produce a significant increase in the percentage variance predicted (0.003%). Therefore, all but a trivial proportion of the between group variance in memory performance is accounted for by executive performance. In the second model the sum of scaled scores on the WAIS-R was entered prior to entry of these former variables. WAIS-R performance accounted for a significant proportion of the variance (68%). However, the executive composite accounted for a significant proportion of additional variance (23%). In contrast, when the WAIS-R was entered after the executive composite (Model 3), it did not account for a significant proportion of the variance (0.06%).

## Discussion

### Executive deficits as differential deficits

The pathology of HD and clinical observations suggest that fronto-executive dysfunction may be a prominent feature of the behavioural and cognitive problems seen in HD (Caine et al., 1978). Support for this view was obtained in the present study as the correlations between the three executive tasks and group membership were highly significant, indicating the presence of severe executive deficits in the HD sample. The present results are therefore consistent with previous findings of impaired performance in HD on executive tasks that have been shown to be sensitive to dysfunction in the prefrontal cortex in focal lesion studies (e.g., Butters et al., 1986; Josiassen et al., 1983; Lawrence et al., 1996).

As some readers may not be familiar with the use of correlation coefficients to detect significant mean differences between groups, it should be stressed that the results obtained are the same as would be obtained if ANOVAs or *t*-tests were applied to the data; i.e., the probability that *r* is significantly different from zero is identical to the probability value that would be obtained for the *F* ratio or *t* value generated by these latter tests. Although not the primary reason for their use in the present study, it is worth noting that correlation coefficients have the advantage that, unlike the results of the equivalent *t*-test, they simultaneously provide a significance test and an index of effect size. For example, the correlation between group membership and verbal fluency was 0.81. Therefore we know that 0.66 of the variance ( $.81^2$ ) in fluency performance was between-group variance.

Correlations were used in preference to more conventional tests on means as they permitted examination of the further issue of whether the deficits on the executive tasks qualified as *differential* deficits. This additional issue has not, to our knowledge, been previously examined. However, patients with HD exhibit impairments on a wide range of tasks which are not associated with fronto-executive dysfunction (Brandt, 1991; Lange et al., 1995). Thus, it could be argued that deficits in executive function should be shown to exceed deficits in other cognitive domains if they are to be afforded the special significance that Caine et al. (1978) and other commentators (e.g., Lezak, 1995; Martin, 1987) have suggested. In the present study the WAIS-R was used to provide a comparison standard for the executive tasks as it samples a wide range of cognitive domains but is, as noted, relatively insensitive to the effects of executive

problems arising from frontal lesions. Using Baron & Treiman's (1980) method the deficits on the three executive tasks were found to be no greater than the deficits on the WAIS-R and thus all failed to qualify as differential deficits.

Reflecting the rarity of HD, the sample size in the present study was relatively modest. Therefore, it might be argued that a differential deficit in executive functioning would have been detected in a larger sample with consequently greater statistical power. However, the pattern of results makes this unlikely. It can be seen from Table 3 that both WAIS-R indices had higher correlations with group membership than either the MCST or CET; indeed in the latter case the power was sufficient to reveal a significant difference in favor of the WAIS-R indices. Verbal fluency had a higher correlation with group membership than either of the WAIS-R indices but in the case of the Full Scale IQ this difference was trivial (.81 vs. .80).

It could also be argued that a differential deficit would have been detected had alternative measures of executive dysfunction been employed. Although dissociations between the current measures and IQ have been demonstrated in other conditions in which executive dysfunction is a feature (see next section but one), this argument deserves serious attention. It would be useful to test it empirically using other well-established tests such as the Tower of London / Tower of Hanoi (e.g., Lawrence et al., 1996; Morris, Miotto, Feigenbaum, Bullock, & Polkey, 1997; Shallice, 1982) or more recent but promising tests, such as the Behavioural Assessment of the Executive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie, & Evans, 1996), or the Brixton Test (Burgess & Shallice, 1997).

### **The Baron and Treiman (1980) method of testing for differential deficits**

The Baron & Treiman (1980) method of testing for differential deficits is based on a psychometric / individual differences approach rather than through the application of statistical techniques associated with the experimental tradition. Although the former techniques are more widely employed for this purpose, a number of methodologists have noted that they are often inappropriate in clinical research generally (e.g., Cook & Campbell, 1979), in clinical neuropsychological research in particular (Clark, 1986), and specifically where the aim of an investigation is to identify differential deficits (Strauss & Allred, 1987). For example, analysis of covariance (ANCOVA) is often used to test for differential deficits; in the present case this would have involved determining if a significant difference between groups on the executive task was obtained when WAIS-R performance was used as a covariate. However, among the assumptions underlying the use of ANCOVA are that subjects have been randomly allocated to groups and that the regression of the task of interest on the comparison or control task are equivalent across groups. This application of ANCOVA has therefore been heavily criticized. Strauss and Allred (1987) for example have noted that "the use of ANCOVA in the presence of systematic group differences (on the covariate) is inappropriate in almost all instances" (p. 91).

The most sophisticated and rigorous approach to testing for differential deficits is that developed by Loren Chapman and colleagues (e.g., Chapman & Chapman, 1973; Chapman & Chapman, 1978; Chapman, Chapman, Curran, & Miller, 1994). However, this method is time-consuming and arduous as it involves a number of stages; test development (or test modification); piloting (to find the manipulations required to equate measures for test

difficulty); and collection of standardisation data. After these stages are completed the crucial comparison between a clinical and control sample can be performed.

In contrast, the Baron and Treiman (1980) method used in the present study can be readily applied using standard neuropsychological measures and we therefore suggest it can be usefully employed as part of an ongoing process of evaluating executive hypotheses in other clinical disorders. One minor obstacle to the use of Baron and Treiman's method is that the calculations involved in performing William's (1959) test are tedious and prone to error (i.e., they include, among other things, calculating the determinant of the 3 x 3 correlation matrix). Therefore, we have written a program to perform this test and installed it on the first author's web site<sup>1</sup>. The program requires the correlation between group membership (clinical case versus control case) and scores on the task hypothesised to qualify as a differential deficit, the correlation between group membership and the control task, the correlation between the two measures, and the total sample size (i.e., clinical plus control cases). In coding up the group membership of the participants, the values assigned to represent a clinical or control case are arbitrary but coding a clinical case as 0 and a control case as 1 would be simplest (if you wanted to assert your individualism you could code the clinical cases as -1.32 and the control cases as 520, the results would be the same).

The emphasis in the present paper is on testing for differences between tasks considered to measure *different* cognitive functions. However, the method outlined is just as applicable to comparing competing measures of the *same* construct. For example, suppose that a neuropsychologist wishes to evaluate two measures of sustained attention (measures A and B) for use in clinical practice or research. Many features of the measures should be considered in conducting such an evaluation, but one important criterion would be the extent to which they are sensitive to differences between healthy controls and clinical cases for whom there would be strong grounds for expecting a sustained attention deficit. If the correlation between group membership and measure A was significantly higher than that for measure B, this would indicate superior (criterion) validity for the former measure.

### **Verbal Fluency versus Verbal IQ in HD and other disorders**

It is informative to compare the present results on verbal fluency and its relationship to general verbal ability in HD with that reported in other neurological conditions. On present evidence, the HD pattern is unlike that seen in focal frontal lesions. Miller (1984) found a highly significant deficit on verbal fluency accompanied by preserved performance on the Verbal Scale of the WAIS in a sample of 30 focal frontal cases. The HD pattern also differs from that seen in two other conditions in which a differential deficit in fronto-executive function has been hypothesised, namely closed head injury (e.g., see Mattson & Levin, 1990; Stuss, 1987; Walsh, 1991) and schizophrenia (e.g., see Goldberg & Weinberger, 1988; Liddle, 1987; Pantelis & Nelson, 1994; Winn, 1994). In both these conditions evidence for the presence of a differential deficit using the same measures has been obtained in that, although WAIS-R VIQ was significantly lower in the patient groups than in their respective controls, the verbal fluency deficit was significantly greater than the VIQ deficit (Crawford, Obonsawin, & Bremner, 1993; Crawford, Wright, & Bate, 1995).

<sup>1</sup>The program can be downloaded from the following address:  
<http://www.psyc.abdn.ac.uk/homedir/jcrawford/differential-deficit.htm>.

Miller (1985) examined the frontal hypothesis in Parkinson's disease by comparing verbal fluency with verbal intelligence in a sample of 20 patients. No evidence of a differential deficit was obtained as the PD sample did not differ significantly from the control sample on either measure. In this case it could be argued that evidence for a differential deficit could still be forthcoming from samples in which the disease had progressed sufficiently to produce cognitive changes. Such an argument is not available in the case of HD as the present results demonstrate highly significant deficits on both tasks. The HD pattern is identical to that reported by Miller (1984) in dementia of the Alzheimer type (DAT). DAT cases exhibited significant deficits on both fluency and VIQ but the fluency deficits did not significantly exceed the VIQ deficits.

To summarize, the evidence suggests a differential deficit in verbal fluency performance is a feature of focal frontal cases and at least two conditions in which, like HD, there were grounds for hypothesising that such a differential deficit would be found. In contrast, the fluency deficit in HD is no greater than would be predicted from their current general level of verbal functioning, a result identical to that found in DAT.

### **Errors of commission in HD**

Problems in self-monitoring and utilization of feedback are characteristic consequences of focal frontal lesions (Lezak, 1995; Parker & Crawford, 1992; Stuss & Benson, 1984). These executive problems can reveal themselves in perseverative responding on card sorting tasks (e.g., Nelson, 1976) and perseverative and rule break errors on verbal fluency (e.g., Walsh, 1994). In line with the claim that HD be viewed as a frontal or fronto-striatal syndrome (Caine et al., 1978) we hypothesized that the HD sample would exhibit raised error rates on these two tasks. This hypothesis was not supported, as error rates in the HD sample were not significantly raised above control rates. It is possible that, with a larger sample, significant differences would have been obtained as the trends were in that direction. However, the failure to find increased error rates on the verbal fluency task is consistent with other studies of HD (see Butters, 1984; Butters, Salmon, Heindel, & Granholm, 1988).

### **The contribution of executive dysfunction to memory impairment in HD**

The indications that executive deficits in HD are no greater than the averaged level of cognitive deficit on the WAIS-R does not preclude the possibility that, as suggested by a number of researchers (e.g. Brandt, 1991), they may nevertheless make a specific contribution to the well documented memory deficits in this condition. In the present study this was vividly illustrated in the results of the hierarchical regression analysis in which verbal learning was the criterion variable. Ninety one percent of the variance in verbal learning was accounted for by the executive composite. As a proportion of the variance in the predictor and criterion variables would be error variance, this suggests that close to all of the *explainable* variance in verbal learning was predicted by executive task performance. It will be noted also that there was no danger of over modeling in the present analysis as the executive measures were formed into a single, unweighted, composite for entry into the models. The hypothesis that executive deficit would explain all of the *between-group* variance in verbal learning was confirmed. Despite there being a highly significant difference between the groups in verbal learning ( $p < .001$ ), entry of

group membership into the model did not lead to a significant increase in the variance explained; indeed the variance added was vanishingly small, i.e., 0.003% (see Model 1, Table 5). Thus, the present results add to a body of converging evidence which suggests that the memory and learning problems observed in HD largely stem from deficits in executive control processes (e.g., Brandt, 1991).

Similar suggestions have been offered for the memory deficits observed in other conditions. For example, it has been argued that memory deficits in idiopathic Parkinson's disease (PD) are attributable to fronto-executive dysfunction (Bondi, Kaszniak, Bayles, & Vance, 1993; Della Sala, 1988). Support for this argument was provided by Bondi et al. (1993) who reported that differences between a PD sample and controls in memory performance were no longer significant after controlling for performance on a battery of executive tasks which included verbal fluency and the Wisconsin Card Sorting Test. A similar approach was adopted by Troyer, Graves & Cullum (1994) to test the hypothesis that memory deficits associated with normal aging are attributable to executive dysfunction (e.g., Dempster, 1992). Regression analysis revealed that in a sample ranging between 60 and 91 years of age, age did not account for a significant proportion of the variance in memory performance after controlling for performance on a battery of executive tasks. Crawford, Bryan, Luszcz, Obonsawin, and Stewart (in press) reported the same result for verbal free recall in a sample ranging in age from 16 to 75.

One major issue for these studies and the present one is whether the direction of causality may be the opposite of that assumed. Specifically it could be argued that the impairment of verbal learning observed in the present HD sample was responsible for, or at least contributed to, the poor performance on the executive tasks; e.g., poor memory for the task instructions or for previous responses led to impaired performance. Evidence against this possibility is provided by demonstrations that densely amnesic subjects can perform at average or above-average levels on measures of executive function. For example, Dall'Ora, Della Sala & Spinnler (1989) report a number of amnesic cases with normal or above average performance on fluency tests and sorting tasks. Similarly, Van der Linden, Bredart, Depoorter & Coyette (1996) report that case AC, a severe amnesic, performed within normal limits on Nelson's (1976) MCST and on initial letter verbal fluency. Thus it would appear that these tasks can be performed adequately in the face of severe deficits in verbal learning.

Another possible alternative explanation for the results obtained in this study and those of Bondi et al. (1993), Troyer et al. (1994), and Crawford et al. (in press) is that performance on the memory and executive tasks were simply independent indicators of the general severity of cerebral dysfunction; i.e., these tasks co-vary but are not causally related. Although this possibility was not addressed in Bondi et al. (1993) or Troyer et al. (1994), the present pattern of results do not support it in the case of HD. William's tests showed that the deficits on the executive tasks were no greater than the deficits on the WAIS-R, thus these two sets of measures can be regarded as equally sensitive indicators of overall severity of dysfunction. Thus, if covariance between these two sets of measures and the verbal learning measure are simply reflecting overall severity, the former measures should not differ in their ability to predict between-group variance in the latter. However, regression Model 2 revealed that, although

WAIS-R performance did predict a significant proportion of CVLT variance, the executive composite predicted a highly significant proportion of additional unique variance. In contrast when the executive composite was entered first (Model 3), general intellectual functioning did not significantly increase the variance predicted. A similar pattern of results was obtained by Crawford et al. (in press) in their study of the executive hypothesis of normal ageing.

### Conclusions and future research

To summarize, the present results indicate that, contrary to Caine et al's. (1978) suggestion that HD can be characterized as a fronto-executive syndrome, deficits on executive tasks in HD are no greater than the general level of deficit observed on the WAIS-R. However, the finding that executive performance nevertheless explained all of the between-group variance in verbal learning does support Caine et al's. (1978) general hypothesis and the specific hypothesis (e.g., Brandt, 1991; Butters, 1984) that memory problems in HD stem from deficits in executive control processes.

Finally, fronto-executive hypotheses have been offered to account for the cognitive and behavioural disturbances seen in a vast array of neurological and psychiatric disorders. If we are to avoid engaging in what David (1992) has termed "Frontal Lobology: Psychiatry's new pseudoscience", we need to apply rigorous standards of proof when evaluating these hypotheses. The demonstration of deficits on executive tasks is only a pre-requisite in such an evaluation given that, as is the case in HD, generalized deficits are a feature of many of these other disorders. Therefore, again just as in HD, an important stage in evaluating an executive hypothesis in any disorder, is to test whether any observed deficits on executive tasks qualify as differential deficits (Crawford et al., in press).

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