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Neuropsychological test performance in the acquired immunodeficiency syndrome: Independent effects of diagnostic group on functioning

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Abstract

Individuals infected with the acquired immunodeficiency syndrome (AIDS) are at risk for developing cognitive impairment. The extent to which the impairment represents the results of a single factor accounting for a wide degree of cognitive dysfunction, or is the result of the combined effects of multiple factors, has not been determined. In the present study, we analyzed data from 134 patients with AIDS and 105 HIV− controls using a recently developed analytical procedure. The results revealed that, by and large, the test variables shared a significant amount of variance related to disease status. Hence the AIDS-related influences on cognition are shared and thus cannot be considered independent. Two tests, Digit Symbol Substitution, and the primacy measure of verbal free recall, had a direct relationship with the group variable (AIDS *vs.* controls). These results suggest that a single factor is sufficient to account for a large proportion of the AIDS-related variance on a wide variety of neuropsychological tests. (*JINS*, 1999, *5*, 41–47.)

Keywords: HIV, Cognition, AIDS, Neuropsychology

INTRODUCTION

Individuals infected with human immunodeficiency virus (HIV) are at increased risk to develop cognitive impairment at some point during the natural history of the disease. Although a relatively unlikely occurrence early in the infection, the risk of cognitive deficits rises as an individual becomes immunocompromised and develops the acquired immunodeficiency syndrome (AIDS; McArthur & Grant, 1998). There appear to be two forms of disorder: a milder minor cognitive motor disorder (MCMD) and the more severe HIV-associated dementia (HAD; American Academy of Neurology, 1991). MCMD can appear anytime after infection, but HAD is most common after the onset of AIDS, and death usually follows a diagnosis of HAD by approximately 6 months (McArthur, 1987; McArthur et al., 1993).

Descriptions of the nature of the cognitive defects reported in AIDS patients usually emphasize the psychomotor slowing. Indeed, the neuropsychological syndrome associated with AIDS has been likened to that seen in patients with progressive disease involving the basal ganglia, including Parkinson's and Huntington's diseases, and progressive supranuclear palsy (Martin, 1994). AIDS patients can become slow, both motorically and cognitively, and this slowing accounts for some of the poor performance on other neuropsychological tests (Becker et al., 1997). What is not clear, is whether psychomotor slowing represents the core feature of the syndrome, or is merely a manifestation of multiple mild cognitive defects. This is important because if cognitive slowing were, in fact, at the center of the MCMD and thus was a strong predictor of performance on other cognitive measures, then changing cognitive speed would affect performance on a variety of cognitive operations. Were a therapeutic intervention shown to affect psychomotor slowing it might, in turn, be expected to have a broad effect on performance in many cognitive tasks.

We have previously reported that composite scores reflecting memory, word fluency, spatial skills, and frontal system function were significantly and independently predicted by measures reflecting premorbid skills and psychomotor speed (Becker et al., 1997). Of note, however, was the fact that the measure of HIV-related health (i.e., CD4+ cell count)

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was related only to the speed, and not the verbal composite score. This suggests that basic verbal skills are unaffected in mildly impaired (and nondemented) individuals and do not change with advancing immuno-suppression. By contrast, changes in infection status (as indexed by T-cell counts) will affect psychomotor speed, which in turn affects performance on other cognitive functions.

These findings are, of course, consistent with the hypothesis that psychomotor slowing or (impaired) speed of cognitive operations is at the core of the neuropsychological syndrome in HIV infection and AIDS. However, an alternative means of investigating the nature of AIDS-related influences involves examining relations among the test outcome variables and the grouping variable (i.e., AIDS *vs.* HIV−) to determine the extent to which there are independent and unique group-related effects on the outcome variables.

The analytical procedure we employed consists of investigating a structural equation model similar to that portrayed in Figure 1. There are three important points to note about this figure. First, the circle labeled "common factor" corresponds to the variance shared by all variables, and thus it represents what all the variables have in common. Second, the relation from the group variable to the common factor represents the group-related effects on what all the variables have in common. And third, the dotted lines from the group variable to the individual outcome variables represent relations of group status on the variables that are independent of the effects of group shared among all variables. The procedure is conceptually analogous to a combination of principal components analysis and hierarchical regression analysis. That is, the first principal component in a principal components analysis could be used to represent what the variables have in common, and then it could be entered before the group variable in a hierarchical regression analysis predicting each outcome variable. The advantage of the current single common factor analysis (SCFA) procedure is that these various steps can be carried out simultaneously in the context of a structural equation model, and in the process indicate the degree to which there are independent or distinct AIDS-associated effects on particular outcome variables. Because many diseases such as AIDS have effects on a wide range of neuropsychological test variables, some means is needed for determining whether the effects on different variables are all independent of one another, or whether many of those effects are shared and possibly mediated through a common mechanism. The SCFA procedure can be used for this purpose because the disease-related effects through the common factor represent the effects that are shared across variables, and the direct relations from the group variable to the outcome variables represent disease-related effects that are independent of those on other variables.

For these reasons, SCFA lends itself well to the study of the neuropsychological impairments associated with AIDS. To the extent that there is a single underlying factor that accounts for many impairments, then SCFA should reveal moderate to high loadings of all test variables on the common factor. There should also be little or no association between group status and test variables after accounting for

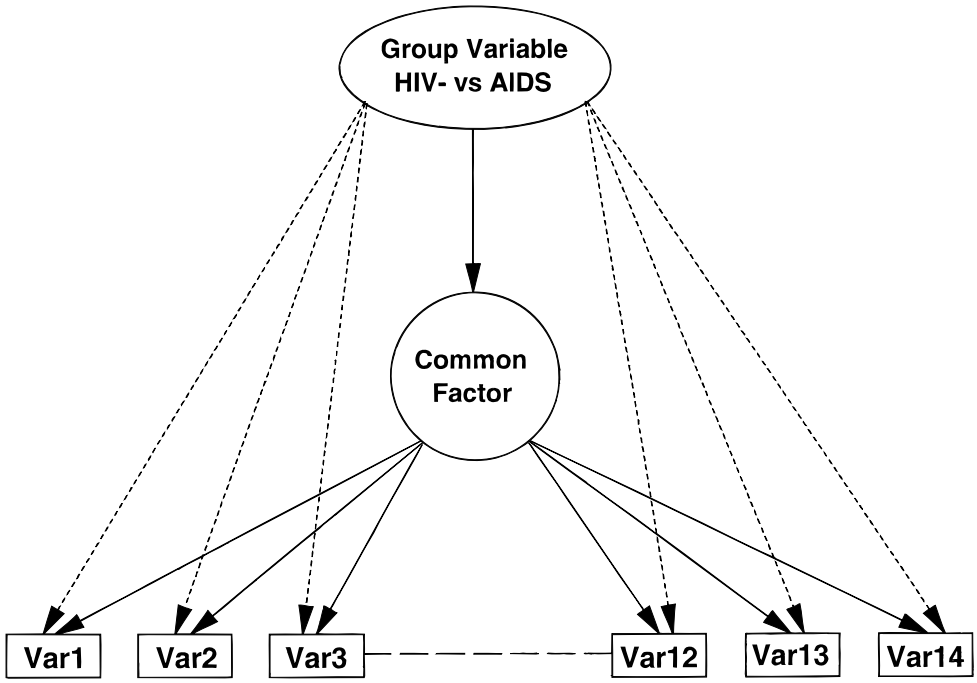

Fig. 1. Conceptual model slowing how diagnostic group (HIV− *vs.* AIDS) is related (1) to a construct (common factor) that represents what all variables have in common, and (2) possibly to each individual variable. The example shows 14 variables as in the present study, but may be any number of test scores. See text for details.

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the relation of group status to the shared variance. By contrast, to the extent that there are multiple factors underlying the performance impairments, then there should be several sets of variables with independent group-related effects. In order to provide the strongest test of the hypothesis, we focused our attention on the patients in the study sample with AIDS. We did so because these patients are at the highest risk to display cognitive impairment, and because our previous study (Becker et al., 1997) had found no significant difference between HIV− and HIV+/non-AIDS participants on neuropsychological measures.

METHODS

Research Participants

The data for this study were taken from the Allegheny County Neuropsychiatric Survey, a study of the neuropsychological, neurological, and psychosocial consequences of HIV infection and AIDS (Becker et al., 1997). Of particular relevance is that these participants were drawn from primary care physicians' offices, and thus represent a cross-section of the HIV-infected individuals within Allegheny County (PA). One hundred thirty-four participants meeting the 1993 CDC criteria for AIDS (Centers for Disease Control, 1992) provided data, and for comparison purposes 105 HIV seronegative (HIV−) individuals were also enrolled in the study during the same period. HIV serostatus for all participants was verified by enzyme-linked immunosorbent assay with Western blot confirmation.

Table 1 presents data on demographic characteristics for all study participants. There were significantly more women among the HIV− controls relative to the AIDS patients, and the controls were less likely to have met criteria for alcohol abuse–dependence prior to joining the study. The AIDS patients were more likely to be men who had sex with men, use injectable drugs, and the controls reported more risky heterosexual contact (e.g., sex with intravenous drug user). Otherwise, the two groups were comparable. No person was excluded from the study based on the presence of neurobehavior signs or symptoms. Nine of the AIDS patients (6.7%) had encephalopathy at study entry.

Neuropsychological Evaluation

The neuropsychological test battery was designed to permit both quantitative and qualitative analysis of neuropsychological function. The instruments were selected based on the recommendations of the NIMH-sponsored workgroup (Butters et al., 1990) and optimized the trade off between time and depth of the evaluations. Each was tested by a trained examiner with experience in assessing physically ill, cognitively impaired adults.

The performance of the participants on the neuropsychological test variables used in the analysis is shown in

Table 1. Characteristics of study sample

Characteristic	HIV−	AIDS	Statistic[4]
N	105	134	
Age	38.2 (13.9)	39.7 (8.4)	−1.05
Race (% White)[2]	91 (86.7)	112 (83.5)	4.52
Sex (% Male)[2]	63 (60.0)	115 (85.8)	20.6**
Education[2]			
High school or less	24 (22.9)	41 (30.6)	3.72
College	62 (59.0)	75 (56.0)	
Postgraduate	19 (18.1)	18 (13.4)	
Risk group[2]			
Men sex with men	48 (45.7)	82 (61.20)	
IV Drug Use	4 (3.8)	16 (11.9)	
MSM + IVDU	5 (4.8)	22 (16.4)	
Heterosexual contact	20 (19.0)	8 (6.0)	
Transfusion	0 (0)	2 (1.5)	
None reported	19 (18.1)	1 (0.7)	
Unknown	9 (8.6)	3 (2.2)	
CD4+ Cell Count[1]	n/a	217.05 (222)	—
HIV RNA[1] ($\log_{10}$ Copies)	n/a	4.56 (.867)	
Alcohol abuse–lifetime[2,3]	26 (32.1)	59 (44.7)	3.32*
Cocaine abuse–lifetime[2,3]	17 (21.0)	37 (28.0)	1.31
Major depression–lifetime[2,3]	37 (35.1)	58 (43.3)	1.59
Generalized anxiety–lifetime[2,3]	13 (11.4)	14 (10.4)	0.06

*$p < .05$; **$p < .001$.
[1]$M \pm SD$.
[2]Count (percent)
[3]DSM–IV diagnosis
[4]χ^2 for proportions, t for means

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Table 2, broken down by group status (i.e., HIV− *vs.* AIDS). A series of *t* tests were run on all of the variables, and 17 were found to have significant effects associated with group status and were thus retained in the subsequent analyses (a full list of variables and performance scores may be found in Becker et al., 1997). This initial screening of variables prior to their use in the analysis was necessary in order to maximize the utility of the SCFA. We did not want to include those tests that did not differentiate the patients and controls since they would provide little useful information. Further, we adopted a liberal criterion for entry (i.e., no correction for multiple comparisons) to ensure that we included all measures that could possibly differentiate between groups and perhaps have group effects independent of the common factor.

The measures retained in the analysis included components of the Wechsler Adult Intelligence Scale–Revised (Wechsler, 1981; Block Design, Digit Symbol), the Wechsler Memory Scale–Revised (Wechsler, 1987; Visual Reproductions, Digit Span), Controlled Oral Word Association (Benton et al., 1983), Booklet Category Test (DeFilippis & McCampbell, 1991), and Trail Making (Reitan, 1958). A 12-word verbal free recall task, consisting of six independent lists of words, was also presented. Each participant was read a list of 12 words, and asked to recall (immediately) as many words as possible, without regard for serial order (see, e.g., Glanzer & Cunitz, 1966). Measures of three segments of the serial position function—primacy, asymptote, and recency—were obtained by aggregating recall across the first four, middle four, and last four input positions, respec-

Table 2. Performance by AIDS patients and HIV− controls on neuropsychological test battery

Measure	HIV−	AIDS	*t* test
Verbal			
Arithmetic–WAIS–R	12.30 (3.99)	12.04 (3.65)	0.53
Information–WAIS–R	19.84 (5.45)	20.33 (5.35)	−0.70
Vocabulary–WAIS–R	54.30 (12.40)	52.04 (11.05)	1.49
Memory			
Memory spans			
Digits Forward	7.08 (0.97)	6.88 (1.04)	1.49
Digits Backward	5.36 (1.35)	4.87 (1.20)	2.95*
Words	5.72 (1.04)	5.30 (0.86)	3.46*
Recall			
Logical Memory			
Immediate	25.75 (7.33)	23.46 (7.11)	2.45*
Delayed	22.05 (7.45)	19.11 (7.81)	2.94*
Visual Reproductions			
Immediate	37.35 (4.24)	35.39 (5.30)	3.10*
Delayed	35.08 (6.64)	32.01 (8.00)	3.17*
Verbal Free Recall			
Primacy	3.63 (1.08)	2.98 (1.04)	4.73*
Asymptote	1.82 (0.92)	1.40 (0.69)	4.05*
Recency	4.17 (1.10)	4.00 (0.97)	1.29
Supraspan Learning			
Span + 1	4.41 (2.14)	4.90 (2.37)	−1.67
Span + 2	6.20 (2.75)	7.37 (3.06)	−3.04*
Visuospatial			
Visual Copy	39.92 (2.29)	39.14 (3.68)	1.91
Block Design–WAIS–R	34.29 (10.60)	31.05 (10.39)	2.30*
Word generation			
Letter Fluency	45.56 (12.00)	41.66 (11.23)	2.60*
Category Fluency	22.10 (5.87)	20.54 (5.59)	2.09*
Executive functions			
Trail Making Test			
Part A	27.80 (11.28)	28.83 (13.06)	−0.64
Part B	59.32 (30.52)	74.84 (39.45)	−3.32*
Booklet Category Test	47.70 (31.89)	59.02 (31.96)	−2.72*
Comprehension–WAIS–R	23.92 (5.89)	23.29 (4.27)	0.97
Attention–psychomotor speed			
Digit Symbol–WAIS–R	61.48 (12.24)	52.76 (12.90)	5.34*
Choice Reaction Time	807 (247)	886 (264)	−1.95

*$p < .05$

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tively. In a separate verbal learning task (*Supra-Span Learning*) the participants were first tested for their word span. After the word span task, the participants were then tested for their ability to learn lists of words one and two words above their span. Each individual was given 11 trials to learn and recall each list (i.e., span + 1, span + 2) without error.

Data Analysis

A two-step procedure was followed for each analysis using the raw data from individuals with no missing values for the variables included in the analysis. The first step consisted of estimating the common-variable and group–common relations using maximum likelihood techniques with the EQS Structural Equation Package (Bentler, 1996). In the second step, the parameters from the first step were fixed to the estimated values, and then the EQS program was used to estimate the group-variable relations for each variable. Relations between group and the individual variable were retained in the final model when the coefficients differed from zero by at least 2 standard errors.

RESULTS

The results of the SCFA are shown in Table 3. The signs of the variables that represent error scores (e.g., Booklet Category) or trials to criterion (e.g., Supra-Span Learning) are reversed so that all group–variable correlations are negative, and all common-variable loadings are positive. Overall, there was a moderate negative relation (−.315) between the group factor and the common factor. This indicates that the AIDS group was moderately lower than the HIV− controls on the factor representing the variance shared by all of the test variables. Second, all of the neuropsychological test variables had moderate-to-high loadings on the common factor. These loadings ranged from .311 to .754, with a mean of .599. This indicates that these test variables shared a significant amount of variance, and thus cannot be assumed to be independent. Third, only two variables (Digit Symbol Substitution Task, and the Primacy measure from Free Recall) had independent group variable effects. These effects are small (less than .14), but are comparable to those found in our earlier study of Alzheimer's disease patients (Salthouse & Becker, 1998). The direction of the relationship indicates that the group effect on these variables was underestimated by the influence of the common factor. This finding indicates that in addition to the effects of HIV shared by all of the test variables, there were independent HIV-related effects reflected in the DSST and free recall tasks.

Because of the differences between groups in the proportion of women, we reran the analysis with sex as a covariate (see Table 3). For this analysis the negative relation between the group factor and the common factor was slightly larger ($r = -.38$), but the pattern of loadings, and the mean loading (.600), was similar to that for the entire group (.600). The only exception was that the independent relation from group to the Digit-Symbol measure was not significantly different from zero after controlling for sex. Similar patterns were evident when the analyses were repeated using age, education, and race as covariates and the results did not differ from those reported for the group as a whole.

Table 3. Results of single common factor analysis

Group–Common = −.315	All participants		Controlling for sex	
Variables	Common	Group	Common	Group
Block Design	.718		.706	
Digit Symbol	.717	−.110	.748	
Booklet Errors	.647		.641	
Digits Backward	.497		.500	
Category Fluency	.585		.578	
Logical Memory–Immediate	.621		.630	
Logical Memory–Delayed	.589		.599	
Primacy Effect	.516	−.138	.518	−.117
Asymptote Effect	.531		.533	
Trails B	.680		.681	
SupraSpan + 2	.311		.309	
Visual Memory–Immediate	.722		.711	
Visual Memory–Delayed	.754		.743	
Word Span	.508		.505	

Note: The Group–Common relationship corresponds to the top arrow in Figure 1. The entries in the Common–Variable column correspond to the arrows with solid lines in Figure 1, and the Group–Variable entries to those with dotted lines.

DISCUSSION

The principal finding of this study is that a large component of the effects of AIDS on cognitive test performance variables is shared among those variables. That is, only a small proportion of the effects of AIDS on those test variables is independent of the effects on other variables. In the present study, only 2 of 17 variables had such independent effects. Even after controlling for age, education, or race, the results were unaffected. Although when sex was entered as a covariate there was a slight change in the number of variables with significant independent effects, the overall pattern was very similar. Thus, it appears that a single factor may, indeed, serve as the core of the neuropsychological syndrome in nondemented AIDS patients. This means that at least among largely nondemented subjects most of the cognitive deficits can be accounted for by a single factor.

Although the present study does not address the question of the nature of the common factor directly, in our previous publication (Becker et al., 1997), we described two factors that accounted for the scores on four neuropsychological composite scores. Only one of these predictor variables—a measure of psychomotor speed—was correlated with HIV-related clinical status. Therefore, we suggest that it is the construct of speed (or "slowing") that is at the root of the cognitive defects in nondemented AIDS patients, and as

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noted above, there is a substantial body of evidence to suggest that psychomotor slowing is an important factor in understanding the neuropsychological impairment in HIV (see Martin, 1994; McArthur & Grant, 1998, for reviews). This possibility might be investigated in the context of a structural model in which a latent speed variable (based on multiple indicators) is hypothesized to function as a mediator of the group-related differences in other variables, as has been done in several studies of aging (Salthouse, 1996). Whether psychomotor slowing represents the core feature of the syndrome, or is merely a manifestation of multiple mild cognitive defects is an important issue. If such slowing were, in fact, at the core of the MCMD and thus was a strong predictor of performance on other cognitive measures, then changing cognitive speed would affect performance on a variety of cognitive operations. Thus, were a therapeutic intervention shown to affect psychomotor slowing, this would, in turn, have a broad affect on performance.

In terms of the two tests with independent group-variable effects (DSST, Verbal Free Recall) it is interesting to speculate which feature of these tasks results in the relation to the AIDS variable. Both require some degree of short-term memory function, although this is obviously more pronounced for the free recall task that the DSST. However, both tasks also require a certain degree of multitasking. In the case of the DSST, the participant must perform visual scanning, writing, and (for best performance) visual–verbal learning. In the case of the free recall test, the component with independent effects—the primacy region of the serial position curve—can be influenced by interference from both intra- and extra-list items. Thus, it may be the case that factors associated with cognitive resources allocation that distinguish these tasks from those without such independent group-variable effects.

These data raise an important point concerning the possible existence of subgroups of HIV-infected subjects based on patterns of neuropsychological impairment (e.g., Becker et al., 1995; Cantlay et al., 1996; Zelkowicz et al., 1997). Indeed, research studies that focus on different variables (e.g., verbal *vs.* motor learning; Becker et al., 1995) may not necessarily be studying separate and distinct influences of AIDS. Because a large proportion of the variables shared AIDS-related effects, this suggests that there may be few unique or distinct influences of AIDS on a variable (or variables). Thus, researchers searching for subgroups or for functional dissociations in nondemented AIDS patients need to attend carefully to the issue of the interrelatedness of the study variables.

Although this analysis stresses the shared properties among these measures, it must be emphasized that our conclusions do not extend into the more severe cognitive impairments associated with HAD. This is clearly an important group to study using SCFA since they may be more likely to have independent group-variable effects, and may be more likely to express distinct subgroups based on the pattern of impairment. However, for the SCFA to be meaningful, there must be a substantial number of such cases (at least 150 for the present test battery) and thus we were precluded from that analysis.

The present study is among the first to use the new technique of SCFA to examine the relationships among test variables in clinical populations. These types of analyses can lead naturally to several questions. For example, comparing common factors across disease groups should be possible, and this will be particularly important in examining similarities and differences between MCMD and HAD. Learning why there are differences in the sizes of the effects of AIDS on different variables should also be possible, and perhaps lead to the identification of the mechanism for such differences. To the extent that there are multiple AIDS-related effects due to AIDS on cognition, then identifying such effects should also be possible, as well as examining the relationships among such effects. The sample of participants in the ACNS, and the breadth of the neuropsychological evaluation, should lend itself well to these questions.

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