Neuropsychiatric Correlates of Memory-Metamemory Dissociations in HIV-Infection*

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ABSTRACT

Ninety-one adults with HIV-infection who varied in the concordance between their subjective memory complaints (or metamemory) on the Patient’s Assessment of Own Functioning (Chelune, Heaton, & Lehman, 1986) and their memory performance on the California Verbal Learning Test (CVLT), were compared on the Beck Depression Inventory (BDI), and on neuropsychological (NP) tests of attention, language, psychomotor speed, and conceptual problem-solving. Subjects with low memory complaints and normal CVLT performance (n = 29) had low BDI scores and were normal in all other NP abilities. Subjects with high memory complaints and impaired CVLT performance (n = 20) had elevations on the BDI as well as NP impairments in psychomotor speed and category fluency. Subjects with low memory complaints but impaired CVLT performance (n = 16) had low BDI scores and were selectively impaired in conceptual problem-solving. Subjects with high memory complaints but normal CVLT performance (n = 26) had high BDI scores and normal NP functioning in all other abilities. These results suggest that there are at least two key determinants to metamemory inaccuracy in HIV-infection, namely, frontal executive impairments and mood disturbance.

Human immunodeficiency virus (HIV) Type 1, the causative agent of acquired immunodeficiency syndrome (AIDS), can be associated with varying degrees of neuropsychiatric complications (Grant & Martin, 1994; van Gorp & Buckingham, 1998). The risk for neurocognitive impairment due to primary HIV-infection (i.e., the effects of HIV-infection on the central nervous system (CNS) that is not due to CNS infections, tumors, or metabolic disorders) is known to increase with each successive stage of HIV disease (Heaton et al., 1995; Miller, Satz, & Vischer, 1991; White, Heaton, & Monsch, 1995). Using the current standard for clinical staging of HIV disease (Center for Disease Control, 1992 [CD93]), recent reviews of the literature suggest that 35% of asymptomatic (CD93-A), 44% of mildly symptomatic (CD93-B), and 55% of persons with acquired immunodeficiency syndrome (AIDS; CD93-C) exhibit neurocognitive impairments, particularly in the areas of attention, speed of information processing, learning efficiency, and psychomotor skills (Heaton et al., 1995; White et al., 1995). At least initially, the pattern of neurocognitive impairments associated with HIV-infection is characteristic of a ‘subcortical’ neurologic process (Cummings, 1990), but with progression in systemic disease and severe immunosuppression, additional disruption in executive (frontal lobe) brain systems can occur (Bornstein et al., 1993; Law et al., 1994; Marcotte et al., 1996; Sahakian

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et al., 1995; Stern, Silva, Chaisson, & Evans, 1996; Stern et al., 1995).

Up to 52% of persons with HIV-infection report neurocognitive complaints, particularly problems with memory or concentration (Mehta et al., 1995; Wilkins et al., 1991), but the degree to which these complaints have been found to reflect cerebral dysfunction has varied. Several studies have reported significant associations between neurocognitive complaints and neuropsychological test performance (Beason-Hazen, Nasrallah, & Bornstein, 1994; Mapou et al., 1993; Poutiainen & Elovaara, 1996; Stern et al., 1991), but a similar proportion found no reliable relationship between complaints and neuropsychological functioning (Hinkin et al., 1996; Moore et al., 1997; van Gorp et al., 1991; Wilkie, Eisdorfer, Morgan, Loewenstein, & Szapocznik, 1990). Common across the majority of these studies is the finding that as mood symptoms increase (even in the subclinical range or below clinical cut-offs), there is a corresponding increase in neurocognitive complaints. It is common for patients with mood disturbance, with or without HIV-infection, to report increased distractibility, decreased concentration, and memory problems but show few, if any, neuropsychological impairments that are secondary to mood. As such, it may be that as depressive symptoms increase in HIV-infected individuals, it becomes more difficult to differentiate clinically between neurocognitive complaints that may be due to an underlying depression and complaints that may reflect HIV-related brain impairments. This clinical problem notwithstanding, it is possible to show statistically that neurocognitive complaints can be associated with both depression and neuropsychological performance on measures of attention, psychomotor processing speed, and memory (Beason-Hazen et al., 1994; Mapou et al., 1993).

In a recent study in our laboratory, we confirmed that neurocognitive complaints (regardless of the specific type of complaint) are associated with depressive symptoms but also with measures of psychomotor speed and attention (Rourke, Halman, & Bassel, this issue, pp. 737-756). Our recent findings also support the suggestion by Mapou and colleagues (1993) that subjective neurocognitive complaints may reflect both mood disturbance and neuropsychological impairments. In addition, our results provide an extension of the work of Beason-Hazen et al. (1994) and Mapou et al. (1993) in that we quantified the relative contribution of depression and neuropsychological functioning to neurocognitive complaints. Specifically, we found that mood symptomatology had the most significant impact on reporting of subjective neurocognitive complaints (i.e., depressive symptoms accounted for 75 to 88% of the total variance explained in various neurocognitive complaint areas), whereas psychomotor efficiency had a minimal, although statistically significant, impact on neurocognitive complaints (i.e., psychomotor efficiency accounted for only 12-25% of the explained variance).

Most studies in the literature to date have generally addressed the concordance (and validity) of neurocognitive complaints with neuropsychological test performance by examining the linear association between these two dimensions. A second approach to examining the relationship between neurocognitive complaints and objective neurocognitive impairments is to consider that some persons with HIV-infection may be ‘accurate’ in their subjective assessment of their neurocognitive status (i.e., their neurocognitive complaints will correspond well with objective neuropsychological test performance), but other persons may be ‘inaccurate’ in their self-assessment (i.e., where there is poor correspondence between their neurocognitive complaints and their objective neuropsychological data). Hinkin and colleagues (1996) first used this subgrouping approach to look at actual versus self-reported memory dysfunction in HIV-infection (Hinkin et al., 1996). In their study, they described three subgroups: one group whose self-appraisal of their memory functioning (or metamemory) was consistent with their objective memory performance (37% of their sample), a second group whose self-reported memory complaints exceeded deficits on memory testing (another 37% of sample), and a third group who had few memory complaints or who denied impairments but exhibited significant deficits on memory testing (26% of their sample.
met this criteria). As such, there were two sub-
groups of HIV-infected subjects who were ‘in-
accurate’ in their metamemory or memory ap-
praisal (i.e., one subgroup that ‘over-reported’
cognitive complaints and a second subgroup that
‘under-reported’ or ‘minimized’ the extent of
their memory problems). Those subjects who
were classified as ‘over-reporters’ had eleva-
tions on mood inventories whereas the ‘under-
reporters’ were low on the same instruments. In
examining the clinical characteristics of the ‘in-
accurate’ groups, Hinkin and colleagues found
that those who ‘under-reported’ or minimized
their memory impairments had more advanced
HIV-infection than the other subgroups (i.e., 9
of 12 minimizers had AIDS). This latter finding
led Hinkin and colleagues to hypothesize that
‘minimization’ of neurocognitive problems may
be due to specific disruption in frontal-subcorti-
bal brain systems which could impair both mem-
ory performance and disturb awareness of mem-
ory deficits or metamemory. However, they
were not able to test this hypothesis empirically
because their study was limited to memory func-
tioning.

Determining whether there are reliable and
distinguishable subgroups of patients with HIV-
infection who vary in their estimation of their
memory functioning (as well as other cognitive
functions) has obvious clinical implications for
the assessment and treatment of HIV-related
brain impairments. As such, we designed the
following study to: (1) replicate the meta-
memory-memory subgroup findings by Hinkin
and colleagues (1996); (2) delineate the neuro-
psychological profiles of individuals who vary
in metamemory and objective memory concor-
dance; and (3) address the hypothesis that fron-
tal executive impairments may underlie under-
reporting or minimization of memory impair-
ment in HIV-infection.

METHOD

Participants
Ninety-one HIV-infected adults (87 males, 4 fe-
nales; 96% Caucasian) with no history of neuro-
logical disease (e.g., seizure disorder), significant
developmental problem (e.g., diagnosis of learning
disability), CNS opportunistic infection, head
trauma with loss of consciousness exceeding 30
min, or substance abuse or dependence in the past
2 months, participated in this study. Of the 91 par-
ticipants, 12 were asymptomatic (CDC93 A1 and
A2), 37 were mildly symptomatic (CDC93 B1 and
B2), and 42 had AIDS-defining illnesses or CD4
Lymphocyte counts less than 200 (CDC93 A3 [n =
2], B3 [n = 13] and C1-C3 [n = 27]) as defined by
the Center for Disease Control (CDC93) revised
classification system for HIV infection (Center for
Disease Control, 1992). Eighty-five percent of the
sample was on some combination of antiretroviral
therapy (i.e., 75% of the sample was on at least one
nucleoside analogue reverse transcriptase inhibitor
(NNRTI) or non-NNRTI and one protease inhibi-
tor (PI), 10% of the sample was taking only an
NNRTI, and 15% were not taking any antiretro-
viral medications).

HIV-infected subjects were recruited from a
variety of sources (research pool, primary care
HIV medical clinic, and psychiatric service) to in-
crease generalizability; these subjects represent a
subsample from an earlier study (Rourke, Halman,
& Bassel, this issue, pp. 737-756). Subjects re-
cruited from the three recruitment sources were not
significantly different (all p values > .05) in age,
education, subjective memory complaints on the
Patient’s Assessment of Own Functioning Inven-
tory, memory performance (as defined by the Cali-
ifornia Verbal Learning Test List A Trials 1-5 Total
T score), CD4 Lymphocyte count, or CDC93 stag-
ing.

The mean age and education of the sample was
40.7 (SD = 8.8) and 14.8 (SD = 2.5) years, respec-
tively. The sample had mean WAIS-R Information
and Picture Completion T scores of 49.4 (SD = 8.6)
and 49.8 (SD = 8.7), respectively (Heaton, 1992).
The mean Beck Depression Inventory score for the
sample was 16.7 (SD = 10.6) (Beck & Steer, 1993;
There were no significant differences in demo-
graphics, depressive symptoms, or total subjective
memory complaints across the three CDC93 clin-
cal stages of HIV-infection (all p values > .05).
CD4 Lymphocyte counts, as expected, were signi-
fically different across the three CDC93 categories
[F(2,87) = 30.77, p < .00001]. Mean CD4 Lym-
phocyte counts for the CDC93-A, CDC93-B, and
CDC93-C groups were 525 (SD = 181), 384 (SD =
142), and 179 (SD = 154), respectively.

Measures
Neuropsychological tests were selected according
to guidelines that emanated from the National In-
stitute of Mental Health Workshop on Neuropsychological Assessment Approaches of AIDS-related Cognitive Changes (Butters et al., 1990). The tests relevant to this study were as follows: (1) Information, Digit Span, Picture Completion, and Digit Symbol subtests from the WAIS-R (Wechsler, 1981); (2) Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983) and Letter and Category Verbal Fluency (Spreen & Strauss, 1991); (3) Trail Making Test: Parts A and B (Reitan & Wolfson, 1993) and Symbol Digit Modalities Test (Selenes et al., 1991; Smith, 1973); (4) California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987); (5) Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1993); and (6) Beck Depression Inventory (Beck & Steer, 1993).

To assess subjective memory complaints, we administered the Patient’s Assessment of Own Functioning (PAOF) Inventory (Chelune, Heaton, & Lehman, 1986). The PAOF is a 33-item self-report instrument that was “designed to elicit patient’s self-perceptions regarding the adequacy of their functioning in various everyday tasks and activities” (p. 96, Chelune et al., 1986). It contains questions about memory functioning (10 items), language and communication (9 items), sensory-motor skills (5 items), and higher-level cognitive and intellectual functions (9 items). In the present study, we used the 10-item memory scale to reflect subjective memory complaints (e.g., PAOF question # 1: ‘How often do you forget something that has been told to you within the last day or two?’; or PAOF question #9 ‘How often do you lose things or have trouble remembering where they are?’). Participants were instructed to rate the severity of each memory item on a 6-point scale (0 = almost never; 1 = very infrequently; 2 = once in a while; 3 = fairly often; 4 = very often; and 5 = almost always). Total subjective memory complaints were defined as the sum of the 10 memory items from the PAOF.

**Procedure**

All participants were informed prior to study participation that they would be asked to provide information about their developmental, neurological, and psychiatric history, fill out questionnaires about their current mood status and subjective cognitive functioning, and complete a battery of neuropsychological tests. The background interview, questionnaires, and neuropsychological tests generally took between 3-4 hrs. to complete with a short break in the middle. Recruitment and testing of participants for this study was approved by our hospital Review Ethics Board.

**Statistical Analyses**

All data analyses were carried out with SPSS for the Power Macintosh (version 6.1.1). Statistical significance was defined as $p < .05$ for all analyses. Post hoc Student-Newman-Keuls were conducted when significant one-way ANOVA results were obtained.

**Subgroup Classification**

Based on our own clinical experience, we have generally observed there to be four possible metamemory-memory subgroups associated with HIV-infection: (a) two discordant metamemory-memory subgroups similar to Hinkin and colleagues’ formulations (i.e., ‘over-reporters’ and ‘minimizers’); and (b) two subgroups that demonstrate good concordance between the level of memory complaints and objective memory test performance (i.e., one subgroup, which we refer to as the ‘accurate-impaired’ subgroup, who endorse an elevated number of memory complaints and perform poorly on formal memory testing, and a second subgroup, which we refer to as ‘accurate-normal’, who report few memory complaints and perform normally on objective memory testing). Although these two ‘accurate’ subgroups were generally collapsed into one group in the Hinkin (1996) study, we believe that it is important to make a distinction between those who are ‘accurate-impaired’ versus those who are ‘accurate-normal’.

Because the ‘memory’ impairment associated with HIV-infection is primarily a learning efficiency problem (Delis, Peavy, Heaton, Butters et al., 1995; Heaton et al., 1995; Peavy et al., 1994), we used the California Verbal Learning Test (CVLT) List A Trials 1-5 Total T score (referred to subsequently as CVLT T score) as the measure to define memory performance. ‘Normal’ memory was defined as having a CVLT T score $> 40$ whereas ‘impaired’ memory was defined as CVLT T score $< 40$. We used a score of 20 on the PAOF memory scale (median split of sample) to define ‘low’ and ‘high’ memory complaints.

Based on each participant’s performance on the CVLT T score and PAOF, the following four memory-metamemory subgroups were formed (see Fig. 1 for relationship between memory complaints, CVLT T scores, and depressive symptoms):

<table>
<thead>
<tr>
<th>Metamemory Subtype</th>
<th>Memory Complaints (PAOF)</th>
<th>CVLT T Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accurate-Normal ($n = 29$)</td>
<td>Low</td>
<td>Normal (range = 40-72)</td>
</tr>
<tr>
<td>Accurate-Impaired ($n = 20$)</td>
<td>High</td>
<td>Impaired (range = 9-38)</td>
</tr>
<tr>
<td>Minimizers ($n = 16$)</td>
<td>Low</td>
<td>Impaired (range = 9-38)</td>
</tr>
<tr>
<td>Over-Reporters ($n = 26$)</td>
<td>High</td>
<td>Normal (range = 40-72)</td>
</tr>
</tbody>
</table>
All four subgroups were similar in age, education, and on estimates of verbal and nonverbal intellectual skills, as well as in the proportion of subjects from all three referral sources (all \( p \) values > .05). There were no significant differences across metamemory subgroups in CD4 Lymphocyte counts or in the percentage of cases with diagnoses of AIDS (CDC93-C). Sample mean CVLT \( T \) score was 42.6 (\( SD = 14.3 \)). See Table 1 for demographics, symptoms, and systemic markers.

RESULTS

We grouped 91 adults with HIV-infection into four metamemory-memory subgroups according to the level of their memory complaints (i.e., ‘low’ or ‘high’) and their objective memory performance (i.e., normal or impaired) on the CVLT (Delis et al., 1987). In our study, 54% of the sample was characterized as being ‘accurate’ in their memory self-assessment (i.e., 29 out of 91 subjects or 32%) had ‘low’ memory complaints and normal CVLT \( T \) scores ['accurate-normal' subgroup], whereas 20 out of 91 subjects (or 22%) had ‘high’ memory complaints and impaired CVLT \( T \) scores ['accurate-im-
Table 1. Demographic Data (N = 91).

<table>
<thead>
<tr>
<th>Metamemory Groups</th>
<th>Accurate Groups</th>
<th>Inaccurate Groups</th>
<th>Post hoc Results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NP¹ Normal  (n = 29)</td>
<td>NP² Impaired  (n = 20)</td>
<td>Minimizers³  (n = 16)</td>
</tr>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>39.0 (8.1)</td>
<td>42.8 (10.7)</td>
<td>41.2 (10.8)</td>
</tr>
<tr>
<td>Education (years)</td>
<td>15.3 (2.1)</td>
<td>14.0 (2.1)</td>
<td>14.5 (3.0)</td>
</tr>
<tr>
<td>Information³⁵</td>
<td>49.4 (8.7)</td>
<td>47.2 (9.5)</td>
<td>51.7 (10.0)</td>
</tr>
<tr>
<td>Picture Completion³⁵</td>
<td>52.4 (9.1)</td>
<td>47.6 (7.6)</td>
<td>47.1 (9.9)</td>
</tr>
<tr>
<td>Beck Depression</td>
<td>11.6 (9.2)</td>
<td>22.2 (11.1)</td>
<td>10.5 (9.0)</td>
</tr>
<tr>
<td>Memory Complaints³⁵</td>
<td>11.4 (4.7)</td>
<td>29.7 (5.8)</td>
<td>10.4 (6.2)</td>
</tr>
<tr>
<td>Recent CD4 Count</td>
<td>286 (177)</td>
<td>297 (226)</td>
<td>261 (190)</td>
</tr>
<tr>
<td>CDC-93 (% AIDS)²</td>
<td>52</td>
<td>45</td>
<td>56</td>
</tr>
</tbody>
</table>

Note. * NP = Neuropsychological.
¹ WAIS-R Demographically-corrected T scores (Heaton et al., 1992).
² Patient’s Assessment of Own Functioning Inventory (Chelune et al., 1986).
³ Chi-Square analysis.

paired’ subgroup]). In contrast, in 46% of the sample, there was poor concordance between memory complaints and objective memory performance (i.e., 26 of 91 (or 28%) of subjects reported ‘high’ memory complaints but had normal CVLT T scores [‘over-reporters’], and 16 out of 91 subjects (or 18%) had ‘low’ memory complaints but impaired CVLT T scores [‘minimizers’]).

The total level of memory complaints were similar in the ‘accurate-normal’ and ‘minimizing’ subgroups, as well as in the ‘accurate-impaired’ and ‘over-reporters’ subgroups, and the former two subgroups had significantly less complaints than the latter two subgroups [F(3,87) = 80.66, p < .00001]. Depressive symptoms followed a similar pattern as memory complaints [F(3,86) = 10.02, p < .00001] with the ‘accurate-impaired’ and ‘over-reporters’ subgroups having similar scores on the BDI (i.e., means of 11.6 and 10.5, respectively). Finally, ‘accurate-normal’ and ‘over-reporters’ subgroups were similar on CVLT T score (i.e., means of 52.6 and 51.0, respectively) and both of these groups had significantly higher CVLT T scores [F(3,87) = 55.48, p < .00001] than the ‘accurate-impaired’ and ‘minimizing’ subgroups which were comparable with each other (i.e., mean CVLT T scores were 26.9 and 30.3, respectively).

In our next set of analyses, we used one-way ANOVAs to compare the four metamemory subgroups on neuropsychological measures of attention, psychomotor speed, language, conceptual skills and problem-solving, and on other indices of verbal learning and memory (see Table 2).

On measures of attention and psychomotor speed, significant differences were obtained on the Trail Making Test: Part B [F(3,86) = 4.90, p = .003], WAIS-R Digit Symbol raw score [F(3,86) = 2.66, p = .05], and Symbol Digit Modalities [F(3,77) = 3.20, p = .03] with post hoc analyses indicating that the ‘accurate-impaired’
Table 2. Neuropsychological (NP) Results

<table>
<thead>
<tr>
<th></th>
<th>Accurate Groups</th>
<th>Inaccurate Groups</th>
<th>Post hoc Results (p &lt; .05)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NP(^1) Normal (n = 29)</td>
<td>NP(^2) Impaired (n = 20)</td>
<td>Minimizers(^3) (n = 16)</td>
</tr>
<tr>
<td>Attention / Psychomotor Speed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAIS-R Digit Span</td>
<td>52.5 (10.3)</td>
<td>49.3 (11.8)</td>
<td>52.7 (8.2)</td>
</tr>
<tr>
<td>Trail Making Test Part A(^b)</td>
<td>49.9 (8.7)</td>
<td>44.3 (10.8)</td>
<td>50.2 (12.8)</td>
</tr>
<tr>
<td>Trail Making Test Part B(^b)</td>
<td>55.0 (8.3)</td>
<td>43.4 (11.7)</td>
<td>49.1 (12.5)</td>
</tr>
<tr>
<td>WAIS-R Digit Symbol(^b)</td>
<td>49.6 (9.5)</td>
<td>42.1 (9.4)</td>
<td>49.1 (9.6)</td>
</tr>
<tr>
<td>Symbol Digit Modalities(^c)</td>
<td>46.9 (10.0)</td>
<td>36.6 (13.1)</td>
<td>43.2 (11.9)</td>
</tr>
<tr>
<td>Language</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boston Naming Test</td>
<td>51.0 (12.4)</td>
<td>45.5 (13.8)</td>
<td>46.8 (14.9)</td>
</tr>
<tr>
<td>FAS Fluency (raw)(^b)</td>
<td>44.1 (13.1)</td>
<td>36.4 (12.8)</td>
<td>42.5 (15.9)</td>
</tr>
<tr>
<td>Category Fluency (raw)</td>
<td>53.1 (8.9)</td>
<td>42.6 (7.9)</td>
<td>47.5 (14.4)</td>
</tr>
<tr>
<td>Verbal Memory (CVLT)(^d)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial 1A</td>
<td>−0.4 (0.8)</td>
<td>−1.4 (0.8)</td>
<td>−0.9 (0.9)</td>
</tr>
<tr>
<td>Trial 5A</td>
<td>0.5 (1.0)</td>
<td>−2.5 (1.3)</td>
<td>−2.0 (1.4)</td>
</tr>
<tr>
<td>Short-Delay Free Recall</td>
<td>−0.1 (1.2)</td>
<td>−2.0 (1.0)</td>
<td>−1.9 (1.4)</td>
</tr>
<tr>
<td>Long-Delay Free Recall</td>
<td>0.0 (1.1)</td>
<td>−1.7 (0.9)</td>
<td>−1.6 (1.3)</td>
</tr>
<tr>
<td>Recognition Hits</td>
<td>−0.3 (1.4)</td>
<td>−1.5 (1.6)</td>
<td>−1.1 (1.6)</td>
</tr>
<tr>
<td>Discriminability</td>
<td>0.0 (0.5)</td>
<td>−1.0 (1.4)</td>
<td>−0.7 (0.9)</td>
</tr>
<tr>
<td>Semantic Clustering</td>
<td>0.4 (1.2)</td>
<td>−1.0 (0.8)</td>
<td>−1.2 (1.0)</td>
</tr>
<tr>
<td>Serial Clustering</td>
<td>0.0 (0.9)</td>
<td>0.5 (1.4)</td>
<td>0.4 (1.5)</td>
</tr>
<tr>
<td>Consistency</td>
<td>0.1 (0.6)</td>
<td>−0.6 (1.2)</td>
<td>−1.3 (1.1)</td>
</tr>
<tr>
<td>Total Trials 1-5 T score</td>
<td>52.6 (9.1)</td>
<td>26.9 (8.5)</td>
<td>30.3 (7.3)</td>
</tr>
<tr>
<td>Wisconsin Card Sorting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of Categories (raw)</td>
<td>5.6 (1.2)</td>
<td>4.9 (2.1)</td>
<td>3.7 (2.2)</td>
</tr>
<tr>
<td>% Conceptual Responses</td>
<td>49.5 (8.8)</td>
<td>45.0 (9.4)</td>
<td>38.9 (10.5)</td>
</tr>
<tr>
<td>Perseverative Responses</td>
<td>51.8 (8.7)</td>
<td>45.1 (10.3)</td>
<td>38.4 (9.9)</td>
</tr>
<tr>
<td>Total Errors</td>
<td>49.2 (9.0)</td>
<td>44.7 (9.2)</td>
<td>37.8 (10.0)</td>
</tr>
</tbody>
</table>

\(^a\) All measures are demographically-corrected T scores (Heaton et al., 1991, 1992; Selnes et al., 1991) unless noted

\(^b\) n = 90.

\(^c\) n = 81.

\(^d\) CVLT Standard Scores (Exception: CVLT Total Trials 1-5 = T score).
subjects performed significantly worse than the ‘accurate-normal’ subgroup. There were no significant differences on the Trail Making Test: Part A \(F(3,86) = 1.41, p = .25\) or WAIS-R Digit Span \(F(3,87) = 0.49, p = .69\).

Significant differences in category fluency were obtained \(F(3,87) = 4.69, p = .004\) with the ‘accurate-impaired’ subgroup performing worse than both the ‘accurate-normal’ and ‘over-reporters’. No significant differences were found on the Boston Naming Test \(F(3,87) = 1.51, p = .22\) or on phonemic (FAS) fluency \(F(3,86) = 1.79, p = .15\).

On selected CVLT indices, the ‘accurate-impaired’ group performed significantly worse than the ‘accurate-normal’ and ‘over-reporters’ on Trial 1 \(F(3,87) = 5.80, p = .001\) and on recognition discriminability \(F(3,87) = 5.48, p = .002\); in addition, the ‘minimizers’ performed significantly worse than the ‘accurate-normal’ group on recognition discriminability. Both the ‘accurate-impaired’ and ‘minimizers’ performed significantly worse than the ‘accurate-normal’ and ‘over-reporters’ on Trial 5 \(F(3,87) = 39.99, p < .0001\), short-delay free recall \(F(3,87) = 20.44, p < .0001\) and long-delay free recall \(F(3,87) = 20.18, p < .0001\), semantic clustering \(F(3,87) = 10.52, p < .0001\), and recall consistency \(F(3,87) = 10.91, p < .0001\), although the ‘minimizers’ also performed worse than the ‘accurate-impaired’ on the consistency measure. There were no significant differences in serial clustering across the four subgroups \(F(3,87) = 0.76, p = .52\).

Finally, on selected indices of the Wisconsin Card Sorting Test, significant differences were obtained in the number of categories achieved \(F(3,87) = 6.88, p = .0003\), the percentage of conceptual responses \(F(3,87) = 5.82, p = .001\), the number of perseverative responses \(F(3,87) = 6.97, p = .0003\), and in the total number of errors \(F(3,87) = 6.70, p = .0004\), with the ‘minimizers’ performing significantly worse than all other subgroups, except in the case of perseverative responses, where the ‘minimizers’ performed significantly worse than the ‘accurate-normal’ and ‘over-reporters’.

**DISCUSSION**

This study replicates the metamemory-memory subtype findings of Hinkin and colleagues (1996) that some individuals with HIV-infection are ‘accurate’ in the self-appraisal of their memory ability (i.e., their memory complaints correspond well with objective memory test results), whereas other persons are ‘inaccurate’ in their self-assessment (i.e., where the level of memory complaints do not correspond well with objective memory testing). We found that there was good correspondence between memory complaints or metamemory and objective memory performance in 54% of our sample, where 32% had normal CVLT performance and low memory complaints and 22% had significant memory complaints and definite impairment on the CVLT. Consistent with the findings of Hinkin and colleagues (1996), we obtained similar proportions of HIV-infected individuals who were ‘inaccurate’ in their memory self-assessment. In our sample, 28% ‘over-reported’ or complained of significant memory problems but performed normally on the CVLT (37% of Hinkin’s sample fell into this category), and another 18% in our sample minimized or under-reported the extent of their memory problems (26% of Hinkin’s sample fell into this category).

The other main objectives of this study were to delineate whether the metamemory-memory subgroups differed in other neuropsychological abilities and to determine whether frontal executive impairments may underlie under-reporting or minimization of memory impairment in HIV-infection.

There were two subgroups of HIV-infected individuals that we classified as ‘accurate’ in their memory self-assessment or metamemory who varied in the level of their depressive symptoms, memory complaints, and neuropsychological profiles. The ‘accurate-normal’ group had low depressive symptoms and low memory complaints and performed within normal limits (relative to established normative data) on tests of attention, language, psychomotor efficiency, executive skills, and verbal learning and memory (see Table 2). The ‘accurate-impaired’ group, in contrast, had ‘moderate’ depressive
symptoms on the Beck Depression Inventory (Beck & Steer, 1993), significant memory complaints, neuropsychological impairments in verbal learning and memory, psychomotor speed, and verbal fluency, but relatively normal problem-solving and conceptual skills. This neuropsychiatric profile is consistent with a ‘subcortical’ disease process that has been found in other neurological disorders (Cummings, 1990).

The two ‘inaccurate’ subgroups also varied in the level of their memory complaints, depressive symptoms, and neuropsychological test performance. The ‘over-reporters’, despite having significant memory complaints and ‘moderate’ depressive symptoms (Beck & Steer, 1993), performed within normal limits (relative to established normative data) on tests of attention, language, psychomotor efficiency, executive skills, and verbal learning and memory, and were comparable in neuropsychological functioning to the ‘accurate-normal’ group (see Table 2). On the other hand, the ‘minimizers’ had significantly lower depressive and memory complaints than the ‘over-reporters’, but they exhibited neuropsychological impairments that tend to implicate selectively more disruption in frontal than in subcortical brain systems. We did not find, as Hinkin and colleagues (1996) reported, that minimization was associated with more advanced HIV-infection. One possibility for this discrepancy may be related to the recent development of combination therapy with antiretrovirals and protease inhibitors that may have changed: (1) the natural history or development of HIV-related neuropsychological impairments, and (2) the degree to which systemic markers of HIV-infection (e.g., CD4 lymphocyte counts, plasma viral load, and AIDS-defining illnesses) are associated with elevated risks for HIV-related neuropsychological impairments.

Our current findings suggest that there are at least two determinants that can lead to poor metamemory accuracy, namely, disruption in frontal lobe processing systems and mood disturbance. By comparing the neuropsychological profiles of the ‘accurate-impaired’ and ‘minimizers’ subgroups, we found that a selective impairment in conceptual and problem-solving skills (abilities that are thought to be mediated by frontal executive processing systems), appears to be a necessary condition for neuropsychologically-based metamemory disturbance. As such, our current findings provide support to Hinkin and colleagues’ (1996) prediction that minimization of memory impairment in HIV-infection may be due to dysfunction in frontal-subcortical pathways. More specifically, however, our findings and those of other groups (Janowsky, Shimamura, & Squire, 1989; McGlynn & Schacter, 1989; Shimamura & Squire, 1986), suggest that the integrity and processing systems of the frontal lobes play a critical role in the awareness and ability to accurately gauge the adequacy of one’s memory and other cognitive abilities.

A second determinant that appears to be important in metamemory assessment accuracy in HIV-infection is mood disturbance. Despite the fact that our ‘accurate-impaired’ and ‘over-reporters’ had similar elevations in depressive symptoms on the Beck Depression Inventory, we found that subjects in the ‘over-reporting’ subgroup tended to have more evidence of chronic mood disturbance and characterological features than the ‘accurate-impaired’ subgroup. As such, this suggests that elevations in depressive symptoms alone are not sufficient to produce metamemory inaccuracy. Rather, for metamemory inaccuracy to occur, there must also be other long-standing or complicating psychiatric features. Support for the hypothesis that personality or characterological features may be implicated in ‘over-reporting’ is consistent with the results obtained by Chelune and colleagues (1986) who found that MMPI clinical scales contributed a significant amount of variance to the scales of the Patient’s Assessment of Own Functioning Inventory.

In summary, our results and those of Hinkin and colleagues (1996) suggest that one major reason for the discrepant results in the HIV literature thus far regarding the concordance between neurocognitive complaints and neuropsychological test performance may be that investigators have failed to consider the existence of distinct subgroups within a sample of HIV-infected individuals who vary in the ‘accuracy’ of the self-appraisal of their neurocognitive status.
By examining neuropsychiatric profiles across these distinct subgroups, we were able to isolate at least two key determinants to metamemory inaccuracy, namely, frontal executive impairment and mood disturbance. In terms of the application of our findings, we can only generalize to Caucasian samples of well-educated, gay men who are medically stable and are free from other neurological confounds (e.g., significant head trauma, epilepsy, CNS opportunistic infections) and who are not actively using significant amounts of alcohol or illicit drugs. However, given the broad recruitment source of our sample, these findings would appear to be generalizable to both referred patients (medical and psychiatric) and to those who participate as volunteers. Further studies are needed to examine how our findings may generalize to other sociodemographic and referral samples, including women, other ethnic groups, those with other risk factors for HIV-infection (e.g., HIV-infection acquired through blood products and IV drug use), and in persons with concurrent neuromedical and drug comorbidities (e.g., traumatic head injury and current substance use).

As we follow-up this HIV-infected cohort in our laboratory, we will be better able to address: (1) the essential characterological and psychiatric features that may lead to over-reporting of neurocognitive complaints; (2) whether ‘over-reporters’ may in fact be responding to a neuropsychological impairment that we were not able to detect with the measures selected for this study; (3) whether elevations in depressive symptoms in those characterized as ‘accurate-impaired’ may represent early signs of a developing mood disorder that has not progressed to the point where cognitive distortions have become salient; and (4) the clinical characteristics of our two neuropsychologically impaired groups (‘minimizers’ and ‘accurate-impaired’) and whether these subgroups have different risk factors for morbidity and mortality.

REFERENCES


