Components of Depression in HIV-1 Infection: Their Differential Relationship to Neurocognitive Performance

STEVEN A. CASTELLON[1,2], **DAVID J. HARDY**[1], **CHARLES H. HINKIN**[1,2], **PAUL SATZ**[1], **PHILLIP K. STENQUIST**[1], **WILFRED G. van GORP**[3], **HECTOR F. MYERS**[4], and **LARRY MOORE**[5]

[1] Department of Psychiatry & Biobehavioral Sciences, University of California Los Angeles, Los Angeles, CA, USA

[2] Greater Los Angeles VA Healthcare System, Los Angeles, CA, USA

[3] Weill Medical College of Cornell University, Athaca, NY, USA

[4] Department of Psychology, University of California Los Angeles, Los Angeles, CA, USA

[5] Oregon Health Sciences University, Portland, OR, USA

Abstract

Both depression and neurocognitive compromise are commonly observed among persons infected with the Human Immunodeficiency Virus (HIV). To date, the majority of studies have failed to find a consistent relationship between mood and cognition among HIV-seropositive (HIV+) individuals, suggesting that these constructs are independent of one another. However, depression is a multi-dimensional syndrome and its measurement often utilizes multi-factorial instruments containing cognitive, affective, somatic, and motivational components. The degree to which various symptoms or dimensions of depression might be related to neuropsychological performance in HIV-1 infection is not typically explored and was a main objective of the current study. A sample of 247 HIV+ persons completed both a comprehensive neurocognitive battery and the Beck Depression Inventory (BDI) as part of a standard clinical evaluation at a major community hospital. To examine the dimensionality of the BDI, a principal components analysis was conducted which suggested a three-factor solution comprised of factors representing Self-Reproach (SR), Mood-Motivation Disturbance (MM), and Somatic Disturbance (SOM). The relationship between each of these three factors and neurocognitive performance was examined using both regression and analysis of variance techniques. These analyses showed the MM factor, more so than either the SR or SOM factors, to be associated with several aspects of neurocognitive performance, including verbal memory, executive functioning, and motor speed. These findings suggest that certain items on depression rating scales may be more indicative of central nervous system (CNS) involvement than others. The association between disturbance in mood and motivation and neurocognitive compromise may suggest that each are sequelae of disease specific mechanisms.

Introduction

It is well established that neuropsychological compromise can occur secondary to HIV infection (Becker et al., 1997; Bornstein et al., 1993a; Grant & Martin, 1994; Heaton et al., 1995; Hinkin, Castellon, van Gorp, & Satz, 1998; Maj et al., 1994a, 1994b; Martin et al., 1992; Stern et al., 1991; van Gorp, Miller, Satz & Visscher, 1989; von Giesen, Baecker, Hefter, & Arendt, 2001). When present, HIV-related neurocognitive impairment can range from subtle deficits of unknown clinical significance to frank dementia syndromes that profoundly disrupt an individual's functioning and activities of daily living. The neurocognitive deficits typically associated with HIV-1 infection include decrements in motor and information-processing speed, divided attention, memory retrieval processes, and executive functioning. This pattern of cognitive deficits is consistent with the known sites of neuropathology in HIV/AIDS, which include the basal ganglia and deep white matter tracks (Aylward et al., 1993; Brew, Rosenblum, & Price, 1988; Dal Pan et al., 1992; Grant & Martin, 1994; Navia, Cho, Petito, & Price, 1986).

Depression, like neurocognitive dysfunction, is also commonly observed among a subset of HIV-infected persons (Atkinson et al., 1988; Bing et al., 2001; Dew et al., 1997; Judd & Mijch, 1996; Lesserman, 2003; Maj et al., 1994a; Ciesla & Roberts, 2001; Perkins, Stern, Golden, Murphy, Naftolowitz, & Evans, 1994; Prado et al., 2004; Rabkin, 1996; Satz et al., 1997; Stern, 1994; Summers et al., 1995; Williams, Rabkin, Remien, Gorman, & Ehrhardt, 1991). Relative to population base rates, both syndromal depression (i.e., Major Depressive Disorder (MDD)) and depressive symptomatology are highly elevated among HIV-seropositive (HIV+) persons. Because elevated rates of depression are also observed in HIV/AIDS high-risk groups such as gay/bisexual men and injection drug users (Bing et al., 2001; Dausey & Desai, 2003; Johnson, Rabkin, Lipsitz, Williams, & Remien, 1999; Satz et al., 1997), clearly mood disturbance may antedate HIV infection. Still, it is generally accepted that HIV infection can lead to mood disturbance through both direct (e.g., primary effect of the virus in the CNS) and indirect pathways (e.g., response to increased medical/financial/social stressors, mortality issues).

Although depression has been linked to neurocognitive compromise in numerous studies of both neurologic and psychiatric populations (see reviews by Burt et al., 1995; Cassens, Wolf, & Zola, 1991), the vast majority of studies have found the neurocognitive and psychiatric sequelae of HIV/AIDS to be largely independent of one another (Bix et al., 1995; Bornstein et al., 1993b; Goggin et al., 1997; Grant et al., 1993; Hinkin et al., 1992; Mapou et al., 1993; Mason et al., 1998; von Giesen et al., 2001). Most studies that have failed to find an association between cognition and depression in HIV have used rating scales that typically survey the frequency and intensity of depressive symptomatology over the past 1 to 2 weeks and do not yield clinical diagnoses of depression (e.g., MDD). However, those few studies that have used diagnostic interviews to evaluate the relationship between MDD and cognition in HIV/AIDS have similarly failed to find a relationship (Bix et al., 1995; Goggin et al., 1997; Richardson et al., 1999), suggesting that the lack of association between mood and cognition is not merely an artifact of mood disturbance severity.

If both depression and cognitive performance are subserved by similar CNS substrates, then the identification of depression associated with CNS involvement might be useful in differentiating those HIV+ individuals with disease-associated depression from those without such neuropsychiatric disturbance. Historically, several attempts have been made to subtype depression, using phenomenological differences in symptom patterns to identify individuals with more or less "biological/organic/cerebral" involvement. Subtyping systems such as "reactive vs. non-reactive", "endogenous vs. non-endogenous", or "melancholic vs. non-melancholic", have been used with varying degrees of success to form more homogenous and meaningful subgroups of depressed individuals. A major tenet guiding most of these subtyping

attempts is that certain profiles of symptoms are more likely to be associated with cerebral dysfunction than are others, explaining, at least in part, why some but not all depressed individuals show prominent cognitive deficits. In the current study, we sought to determine if certain items on depression rating scales consistently cluster together, and, if so, whether these might represent meaningful factors that would be differentially related to cognitive function in HIV/AIDS.

We have suggested in earlier work with HIV+ individuals, that a primary disturbance of motivation and goal-directed behavior, captured by the construct of apathy, may be a marker of HIV-associated CNS involvement (Castellon, Hinkin, & Myers, 2000; Castellon, Hinkin, Wood, & Yarema, 1998). While the constructs of apathy and depression significantly overlap, our group demonstrated that in a sample of HIV-infected individuals they are dissociable domains and apathy, but not depression, is more likely to be associated with working memory deficits and executive dysfunction (Castellon et al., 1998, 2000). These findings are not unique to HIV/AIDS, as apathy is a common consequence of CNS disease or disorder that overlaps with depression to varying degrees in several neurologic populations (Cummings, 1993; Duffy & Kant, 1997; Marin, 1991; Marin, Firinciogullari, & Biedrzycki, 1994; Starkstein et al., 1992), and in many of these populations apathy is associated with cognitive compromise (Andersson & Bergedalen, 2002; Kuzis, Sabe, Dorrego, & Starkstein, 1999; Levy et al., 1998; Starkstein et al., 1992). If apathy is indeed a potential indicator of CNS pathology, we should pay careful attention to its presence and its correlates in affected individuals. Given the significant overlap between depression and apathy, it may be that a dimension of depression similar to the construct of apathy is more likely to be associated with other measures of CNS dysfunction.

The following study sought to explore the degree to which meaningful components of depression might be identified on a popular depression rating scale, the Beck Depression Inventory (BDI: Beck, 1987), and, if so, the degree to which these components might be differentially associated with cognitive performance among HIV-infected individuals. The BDI is one of the most widely used self-report instruments for assessing depressive symptoms and has enjoyed considerable popularity in neuropsychological studies. However, it was originally constructed by selecting items that discriminated between depressed and non-depressed *psychiatric* patients. The degree to which mood disturbance may manifest differently in primary psychiatric versus primary neurologic populations has not been sufficiently studied. In fact, to our knowledge, in HIV/AIDS, there have been *no* attempts using sufficient sample size to examine the factor structure of the BDI in spite of indications that cognitive/affective items and somatic/vegetative items may be measuring different phenomenon in this group (Kalichman, Rompa et al., 2000). However, several factor studies of the BDI in non-HIV+ samples have noted that items reflective of disturbance in motivation have consistently clustered together (Startup, Rees & Backham, 1992; Steer et al., 1987; Tanaka & Huba, 1984). These five items (decreased work initiative, decreased social initiative, anhedonia, difficulty making decisions, and anergia) bear a striking similarity to items found on apathy scales, including the Apathy Evaluation Scale (Marin, 1991) and the apathy subscale of the Neuropsychiatric Inventory (Cummings et al., 1994).

We wished to determine if meaningful dimensions of depression could be identified on the BDI among a sample of HIV-infected individuals. If so, would "apathy" items load on the same factor? Also, do the somatic/vegetative symptoms of depression cluster together and might they be more common with advanced disease stage? Finally, and most theoretically interesting, do different dimensions of depression have a differential association with neurocognitive performance among HIV-infected individuals. The following study sought to address these issues by 1) performing a factor analysis of the BDI among a large sample of HIV-infected

individuals, and 2) comparing the extent to which different BDI factors were associated with both overall neurocognitive performance as well as functioning in specific cognitive domains.

Method

Participants

This study examined the data from 247 HIV+ individuals who received comprehensive neuropsychological assessment and completed a BDI between the years of 1989 and 1995 at a major community hospital and medical center in the Los Angeles area. Data from a subset of these individuals have been reported in previous studies by our group, which have primarily been aimed at exploring age group differences in neurocognitive performance (Hardy et al., 1999; van Gorp, 1994). These individuals were typically referred for neuropsychological evaluation as part of a standard work-up following diagnosis with HIV, and any individual with history or evidence of learning disability or other neurologic condition was excluded from analysis. Further, any participant showing evidence of opportunistic infection or lymphoma of the central nervous system (CNS) was excluded. All subjects were tested as outpatients. Selected demographic characteristics for these participants are presented in Table 1, and as can be seen, the average age for this sample was 37.6 years and mean level of education was 15.3 years. Most participants were self-identified gay or bisexual men and approximately 21% of the entire sample was on some form of work disability at the time of testing. Approximately 25% ($n = 62$) had a diagnosis of AIDS at the time of assessment, and among this subgroup, 42% were on disability ($n = 26$).

Neurocognitive Measures Administered

The Stroop Color Word Interference Test (Stroop, 1935) assesses multiple functions including color-naming and word-reading speed, sustained attention, and, in the interference condition, selective attention and the ability to inhibit a habitual response in favor of a more overlearned one. Dependent variables yielded by this task include number of trials completed in 45 seconds for each condition. The Trailmaking Test (Reitan, 1969) is a brief test of visual-motor function, attention, and psychomotor speed. Part A of this task requires subjects to connect numbers in ascending sequence as quickly as possible while Part B of this task involves switching conceptual set (alternating between connecting numbers AND letters in ascending sequence). For both parts of this task, the dependent variable of interest is completion time in seconds. The Symbol Digit Modalities Test (Smith, 1991) is a test of mental proficiency, visual scanning/tracking, and processing speed. It involves rapidly completing as much of a symbol-substitution task as is possible in the course of 90 seconds. The dependent variable is number of items correctly completed. The Controlled Oral Word Association Test (Benton, Hamsher, Varney & Spreen, 1983) assesses semantic and phonemic verbal fluency. Over the course of four, one-minute trials, the subject must name as many words as they can starting with a given letter ("F", "A", and "S") and, in the final trial, name as many types of animals as possible. The Grooved Pegboard (Lezak, 1995) task measures speed and dexterity of upper extremity fine motor movements. Subjects must rapidly place small pegs that require subtle manipulations to properly fit in their respective pegholes. Two trials were typically administered, one for the dominant and one for the non-dominant hand. Completion time in seconds was the dependent variable of interest. The Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983) assesses the ability of the subject to name pictured objects. Dependent variable was the number of items correctly named without cueing. The Finger Tapping Test (Reitan, 1969) measures motor speed by assessing the number of times a subject can depress a keyboard key using the index finger of their dominant and non-dominant hands. The Logical Memory subtest from the Wechsler Memory Scale-Revised (Wechsler, 1987) measures auditory verbal memory by assessing immediate and delayed recall of details of short stories. The Visual Reproduction subtest from the Wechsler Memory Scale-Revised (Wechsler,

1987) measures non-verbal memory by assessing the immediate and delayed recall of simple geometric designs. The Rey Auditory Verbal Learning Test (Lezak, 1995) assesses several aspects of verbal learning and memory (including immediate memory span, new learning, susceptibility to interference, and recognition memory). The subject is presented with a 15-item list of words and their ability to recall list words is tracked over multiple trials. Immediate and 20-minute delayed recall, as well as delayed recognition are assessed. Multiple dependent variables are yielded from this task, including total number of list items recalled, number of items recalled at both immediately and after a 20 minute delay, and number of items recognized.

Beck Depression Inventory (BDI)

The BDI (Beck, 1987) is a 21-item self-report rating scale containing questions pertaining to the presence and intensity of various cognitive, affective, and somatic symptoms and signs of depression over the prior one-week period. Scores on each item can range from zero (symptom absent) to 3 (presence of symptom is pronounced), yielding a potential range of scores from 0 to 63.

Data Analysis

The Statistical Package for the Social Sciences, Version 8.0 (1997) was used for all data analyses. After assessing the normality of the BDI data, principal components factor analysis with varimax rotation was used to determine the number of meaningful components represented on the BDI. Both simple correlation and regression analyses were used to assess the relationship between BDI factors and neurocognitive variables. To determine the correlation between normally distributed continuous variables, Pearson's r was calculated; for any correlation using a non-normally distributed variable, Spearman's r was used. After running basic diagnostics to explore for violations of important regression assumptions, hierarchical multiple regression was conducted to examine the relationship and contribution of each depression factor to neurocognitive criterion variables (determination of neurocognitive summary variables is described below). For each regression analysis, demographic variables (e.g., age, education) showing a zero-order correlation with the neurocognitive criterion variable of interest were entered in the initial block of the regression equation while the three factor scores were entered, simultaneously, in the following block. Standardized beta coefficients were tested for significance, as was the overall regression model.

Results

The mean BDI score for these participants was 9.9 (SD = 7.7) with scores ranging from 0 to 37. Using the cut-off scores empirically established by Kendall and colleagues (1987), approximately 57% of the sample scored in the range typically considered as non-depressed (0–9), 21% in the range considered dysphoric (10–15), and 22% in the range frequently labeled as depressed ($\geq$16). Table 2 shows means and standard deviations for each BDI item as well as the prevalence with which each BDI item was positively endorsed (i.e., a non-zero response). Mean ratings for individual items ranged from a low of .18 for Item 6 (Self-punishment) item to a high of 1.01 for Item 20 (Somatic Preoccupation). The most frequently endorsed items included Anergia/Fatigue (Item 17), which was endorsed by 68% of the sample, Somatic Preoccupation (Item 20), endorsed by 65% of participants, and Irritability (Item 11), which was positively endorsed by 49% of respondents. In this sample, there was no difference between the BDI scores of patient's with and those without AIDS [t (240) = .24, p = 81].

Principal Components Analysis of the BDI

Principal components analysis with varimax rotation was performed on the BDI data from the 247 participants. In choosing a factor rotation method we decided upon an orthogonal rotation to determine how many independent components are measured by the BDI and increase the

simplicity of reporting results (Tabachnick & Fidell, 1996), although findings are often quite similar whether orthogonal or oblique rotation methods are used (Welch, Hall, & Walkey, 1990). Following Bryant & Yarnold (1995) we used a combination of scree plot analysis and eigenvalue examination to determine how many factors to retain, with these methods suggesting a three-factor solution that accounted for 47.5% of the common variance (eigenvalues of 5.3, 2.1, and 1.3). The rotated factor solution is depicted in Table 3 with factor loadings greater than or equal to an absolute value of .40 judged to be significant and indicated by bold typeface. Coefficient alpha for each of the first two factors was acceptable (both α > . 75) but was considerably lower for the third factor (α = .59), as indicated in Table 3. Communalities for each BDI item ranged between .40 and .66.

As illustrated in Table 3, the first factor, which explained approximately 21% of the common BDI variance, contained items representing negative cognitions and was labeled "self-reproach". The second factor contained all five of the "apathy" items that have previously been found to load together (Startup et al., 1992;Steer et al., 1987;Tanaka & Huba, 1984) and, in this sample, these apathy items loaded together with irritability and sad mood. This factor, which we labeled "mood-motivation disturbance", accounted for approximately 17% of total BDI variance. Finally, a third factor, comprised wholly of items representing "somatic disturbance" explained the remaining 9% of the common variance.

Relationship between BDI Factors and Neurocognitive Performance

The neuropsychological evaluation administered was an extensive one, including multiple performance measures of several cognitive domains (e.g., memory, psychomotor speed). In order to reduce the number of neurocognitive outcome measures, we selected *a priori*, component tasks to represent each of seven neurocognitive factors. A similar strategy, aimed at reducing the problem of multiple comparisons, has been employed by other groups employing extensive neuropsychological batteries (e.g. Heaton et al., 1995; Stern et al., 1995). These factors represented Verbal Memory (RAVLT, Total Trials 1–5, immediate and delayed recall; Logical Memory, immediate and delay), Visual Memory (Rey-Osterreith Complex Figure, Delay; WMS-R Visual Reproduction), Psychomotor Speed (Trails A completion time; Stroop color-naming and word-naming completion times; Symbol Digit Modalities Test), Language (Boston Naming Test; WAIS-R Vocabulary), Spatial (Rey-Osterreith Complex Figure, Copy; WAIS-R Block Design), Motor Function (Grooved Pegboard, dominant and non-dominant hand completion time; Grip Strength, dominant and non-dominant), and Executive Function (Trails B, completion time; Stroop Interference completion time). To calculate factor scores for each patient, each component score was first transformed into a z-score based on the mean and standard deviation of test scores for all participants. For all neurocognitive factor scores, a positive z score was indicative of better performance (i.e., for timed tests, the direction of the z-score was changed as necessary). Factor scores were then calculated as the average of the z scores for measures within each factor.

Table 4 shows the bivariate correlation coefficients between the three BDI and the seven neurocognitive factors. As can be seen, the Mood-Motivation Disturbance factor was associated with poorer performance on the Verbal Memory ($r = -.27$, $p < .01$), Executive ($r = -.22$, $p < .01$), and Motor ($r = -.23$, $p < .01$) factors. Of note, neither the Self-Reproach nor the Somatic Disturbance factors were significantly related to any of these three factors. In contrast, the Self-Reproach factor was significantly correlated with both the Spatial ($r = -.16$, $p < .05$) and the Visual Memory ($r = -.16$, $p < .05$) factors. While the correlation coefficients obtained here are relatively small (albeit significant), bivariate correlations between depression factors and several of the component tasks comprising each factor were often of much greater magnitude (several between .3 and .5). However, to ascertain the importance of each BDI factor *after* controlling for the effects of age and education, which were each significantly correlated

with several of the neurocognitive variables, we used hierarchical multiple regression. After entering relevant demographic predictor variables (e.g., age, education), we simultaneously entered the three BDI factor scores and examined the amount of variance in the neurocognitive criterion variables explained by each BDI factor. Neurocognitive criterion variables used in this set of regression analyses included only those neurocognitive factors showing a significant bivariate correlation with one or more of the BDI factors.

Table 5 presents the standardized beta coefficients, the t-statistic associated with each standardized beta, and the F statistic associated with each step of the regression model for each of five regressions. Thus, the ΔF for Step 2 suggests whether a statistically significant amount of the variance in the neurocognitive criterion variable of interest is accounted for by the addition of the three BDI predictor variables. The standardized beta weights indicate the relative importance of each predictor variable. As reflected in Table 5 by the significant F statistic observed at Step 1 of each regression model, the combination of age and education was significantly associated with performance across all five factors (increasing age and lower levels of education related to poorer performance). Looking at the contribution of BDI factor information to explaining variance in neurocognitive performance above and beyond the influence of demographic characteristics, the ΔF statistic associated with Step 2 of the regression model was significant in three of the five analyses. A statistically significant amount of variance in performance on the Verbal Memory, Executive, and Motor factors was accounted for by the addition of the three BDI factors. Of primary interest to the current study however, was the pattern of standardized coefficients observed. The Mood-Motivation Disturbance factor, but not the Self-Reproach or the Somatic Disturbance factors, was associated with performance in these neurocognitive domains. Of note, although the Self-Reproach factor showed a significant bivariate correlation with Visual Memory and Spatial performance (see Table 4), after accounting for the effects of age and education, this factor did not explain a significant amount of variance in these two neurocognitive domains.

Relationship of BDI Factors to Diagnosis of AIDS

Finally, of interest in the current study was the degree to which more advanced disease, in this case diagnosis of AIDS at time of assessment, was associated with each of the three BDI factors. Table 6 shows the proportion of the BDI total score accounted for by each BDI factor as a function of disease stage group. Of note, although there was a slightly higher prevalence of somatic disturbance and mood-motivation disturbance among patients with AIDS (and therefore, greater endorsement of self-reproach items among participants without AIDS), these differences were not statistically significant.

Discussion

The results of this study confirm that depression, at least as it is reflected by scores on the BDI, is a *multidimensional* construct in HIV/AIDS and, perhaps more importantly, these various dimensions show a differential relationship to neurocognitive performance. More specifically, a BDI factor reflecting disturbance in mood and motivation was more strongly associated with neurocognitive performance than were factors representing either negative self-view or somatic disturbance. The neurocognitive domains most closely associated with the mood and motivation disturbance factor were verbal memory, executive functioning, and motor performance, all of which have been shown to be affected by HIV-infection (Bornstein et al., 1993a; Heaton et al., 1995; Stern et al., 1991).

These findings suggest that certain items from depression rating scales may be more indicative of central nervous system (CNS) dysfunction than other items. We have argued elsewhere (see Castellon et al., 1998; Castellon, Hinkin, & Myers, 2001) that depression in HIV/AIDS is a heterogeneous construct with multiple potential etiologies and that while the mood disturbance

of some HIV+ individuals may be a direct consequence of the virus in the CNS, many others may be depressed without showing HIV-associated CNS involvement. As such, these results are consistent with findings reported by our group showing that apathy, more so than depression, is related to divided attention and working memory deficits, suggestive of executive dysfunction (Castellon et al., 1998, 2000). It may be that the mood and motivation disturbance found in the current study to associated with various cognitive deficits are both indicative of HIV-associated CNS disruption. Possible neurochemical and neuroanatomical mechanisms of both mood disturbance and neurocognitive impairment may be chronic abnormalities in the hypothalamic-pituitary-adrenal (HPA) axis and the resultant increase in cortisol levels (McAllister-Williams, Ferrier, & Young, 1998), and/or changes in dopaminergic regulation in HIV/AIDS (Berger & Arendt, 2000; Lopez, Smith, Meltzer, & Becker, 1999).

To our knowledge, this study represents the first factor analysis of the BDI among an exclusively HIV+ sample in which an adequate sample size was employed. The findings from the current study may partially explain why past research has failed to find an association between depression and cognitive performance in HIV-1 infection (Bornstein et al., 1993b; Grant et al., 1993; Hinkin et al., 1992). In the current study, only the BDI-MM factor was consistently related to neurocognitive performance, while the BDI-SD and BDI-SOM factors were largely unrelated. The vast majority of studies have used *total* scores on depression rating scales as their measure of depression, a practice that may obfuscate any relationship between a particular component of depression and neurocognitive performance. We believe that it is of considerable importance to demonstrate the psychometric properties of depression measures such as the BDI among patients with HIV-1 infection as these properties may vary considerably as a function of the population studied (Beck, Steer, & Garbin, 1988; Dozois, Dobson, & Ahnberg, 1998; Steer, Ball, Ranieri, & Beck, 1999; Volk, Pace, & Parchman, 1993). Most of the depression measures traditionally used with neurologic populations were originally constructed and validated using samples of patients with idiopathic etiologies of their mood disturbance. There may be important phenomenological differences between the expression of mood disturbance among individuals with diseases or disorders known to affect the central nervous system and those whose mood disturbance if of idiopathic (e.g., psychiatric) etiology.

The factor structure yielded in the current study of HIV-infected persons is actually quite similar to several prior factor analyses with other populations (Startup et al., 1992; Steer et al., 1987; Tanaka & Huba, 1987). In fact, as noted by Beck and colleagues (1988) in their review of 25 years of BDI research, a three-factor solution including "Negative Attitudes Toward Self", "Performance Impairment" and "Somatic Disturbance" is among the most common finding of BDI factor studies. The Performance Impairment factor mentioned by these authors bears similarity to the Mood-Motivation Disturbance factor in the current study and the Negative Attitudes Toward Self factor is extremely similar to our Self-Reproach factor.

Finally, it will be important to further investigate the relevance and significance of these depression factors in independent samples of HIV-infected participants as well as those from other neurologic populations. We have presented preliminary data from an independent sample of HIV-infected individuals that show these factors do indeed share differential relationship to aspects of memory performance in HIV/AIDS (Castellon et al., 1999). Mood and motivation disturbance, more so than either somatic disturbance or self-reproach, was associated with both episodic and procedural memory whereas self-reproach was associated with subjective complaints of memory deficit among 46 HIV+ individuals (Castellon et al., 1999). Also, among individuals diagnosed with Chronic Fatigue Syndrome, a diagnostic entity involving both neurologic and psychiatric factors, investigators from our research team and others have found that mood and motivation disturbance, more so than somatic disturbance or self-reproach, is associated with neurocognitive performance (Satz, personal communication). These promising results support our thesis that there is an aspect of depression, similar to apathy, which is more

likely to reflect potential neurologic involvement than do global ratings of depressive symptoms. Further study is needed to begin to clarify the relationship between dimensions of depression and other biological and/or neuroanatomical or neurophysiological markers (e.g., brain metabolism) with potential implications for intervention efforts if it proves to be that mood and motivation disturbance are markers of potential CNS involvement.

Acknowledgments

References

Andersson S, Bergedalen AM. Cognitive correlates of apathy in traumatic brain injury. Neuropsychiatry, Neuropsychology, & Behavioral Neurology 2002;15:184–191.

Atkinson JH, Grant I, Kennedy CJ, Richman DD, Spector SA, McCutchan JA. Prevalence of psychiatric disorders among men infected with human immunodeficiency virus: a controlled study. Archives of General Psychiatry 1988;45:859–864. [PubMed: 3415427]

Aylward EH, Brettschneider PD, McArthur JC, Harris GJ, Schlaepfer TE, Henderer JD, Barta PE, Tien AY, Pearlson GD. Magnetic resonance imaging measurement of gray matter volume reductions in HIV dementia. American Journal of Psychiatry 1993;152:987–994. [PubMed: 7793469]

Basso MR, Bornstein RA. Neuropsychological deficits in psychotic versus non-psychotic unipolar depression. Neuropsychology 1999;13:69–75. [PubMed: 10067778]

Beck, AT. Beck Depression Inventory: Manual. San Antonio, TX: Psychological Corporation; 1987.

Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty five years of evaluation. Clinical Psychology Review 1988;8:77–100.

Becker JT, Sanchez J, Dew MA, Lopez O, Dorst SK, Banks G. Neuropsychological abnormalities among HIV-infected individuals in a community-based sample. Neuropsychology 1997;11:592–601. [PubMed: 9345703]

Benton, AL.; Hamsher, K.; Varney, NR.; Spreen, O. A clinical manual. New York: Oxford University Press; 1983. Contributions to neuropsychological assessment.

Berger JR, Arendt G. HIV dementia: The role of the basal ganglia and dopaminergic systems. Journal of Psychopharmacology 2000;14:214–221. [PubMed: 11106299]

Berger JR, Kumar K, Kumar A, Fernandez JB, Levin B. Cerebrospinal fluid dopamine in HIV-1 infection. AIDS 1994;8:67–71. [PubMed: 8011238]

Bing EG, Burnam MA, Longshore D, Fleishman JA, Sherbourne CD, London AS, Turner BJ, Eggan F, Beckman R, Vitiello B, Morton SC, Orlando M, Bozzette SA, Ortiz-Barron L, Shapiro M. Psychiatric disorders and drug use among human immunodeficiency virus-infected adults in the United States. Archives of General Psychiatry 2001;58:721–728. [PubMed: 11483137]

Bix BC, Glosser G, Holmes W, Ballas C, Meritz M, Hutelmyer C, Turner J. Relationship between psychiatric disease and neuropsychological impairment in HIV seropositive individuals. Journal of the International Neuropsychological Society 1995;1:581–588. [PubMed: 9375246]

Bornstein RA, Nasrallah HA, Para MF, Pace P, Whitacre C, Rosenberger P, Fass RJ. Neuropsychological performance in symptomatic and asymptomatic HIV infection. AIDS 1993a;7:519–524. [PubMed: 8507418]

Bornstein RA, Pace P, Rosenberger P, Nasrallah HA, Para MF, Whitacre C, Fass RJ. Depression and neuropsychological performance in asymptomatic HIV infection. American Journal of Psychiatry 1993b;150:922–927. [PubMed: 8494070]

Brew BJ, Rosenblum M, Price RW. AIDS dementia complex and primary HIV brain infection. Journal of Neuroimmunology 1988;20:133–140. [PubMed: 3058741]

Bryant, F.; Yarnold, PR. Principal components analysis and exploratory and confirmatory factor analysis. In: Grimm, P.; Yarnold, PR., editors. Reading and understanding multivariate statistics. Washington, D.C: American Psychological Association Press; 1995.

Burack JH, Barrett DC, Stall RD, Chesney MA, Ekstrand ML, Coates TJ. Depressive symptoms and CD4 lymphocyte decline among HIV-infected men. Journal of the American Medical Association 1993;270:2568–2573. [PubMed: 7901433]

Burt DB, Zembar MJ, Niederehe G. Depression and memory impairment: a meta-analysis of the association, its pattern, and specificity. Psychological Bulletin 1995;117:285–305. [PubMed: 7724692]

Cassens G, Wolfe L, Zola M. The neuropsychology of depressions. Journal of Neuropsychiatry and Clinical Neurosciences 1990;2:202–213. [PubMed: 2136077]

Castellon SA, Hardy D, Stenquist P, Hinkin CH, van Gorp WG, Moore I, Satz P. Apathy and non-apathy items on the Beck Depression Inventory: Their neurocognitive correlates in HIV-1 infection. [abstract]. Journal of the International Neuropsychological Society 1999;5:154.

Castellon SA, Hinkin CH, Myers HF. Neuropsychiatric disturbance is associated with executive dysfunction in HIV-1 infection. Journal of the International Neuropsychological Society 2000;6:336–347. [PubMed: 10824505]

Castellon SA, Hinkin CH, Myers HF. Neuropsychiatric alterations and neurocognitive performance in HIV/AIDS: A response to Bungener & Jouvent. Journal of the International Neuropsychological Society 2001;7:776–777.

Castellon SA, Hinkin CH, Wood S, Yarema K. Apathy, depression and cognitive performance in HIV-1 infection. Journal of Neuropsychiatry and Clinical Neurosciences 1998;10:320–329. [PubMed: 9706540]

Centers for Disease Control and Prevention. Morbidity and Mortality Weekly Report. Vol. 41. 1992. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults; p. 1-19.

Ciesla JA, Roberts JE. Meta-Analysis of the relationship between HIV infection and risk for depressive disorders. American Journal of Psychiatry 2001;158:725–730. [PubMed: 11329393]

Cummings JL. Frontal-subcortical circuits and human behavior. Archives of Neurology 1993;50:873–880. [PubMed: 8352676]

Cummings J, Mega M, Gray K, Rosenberg-Thompson S, Carusi DA, Gornbein J. The Neuropsychiatric Inventory: Comprehensive assessment of psychopathology in dementia. Neurology 1994;44:2308–2314. [PubMed: 7991117]

Dal Pan GJ, McArthur JH, Aylward E, Selnes OA, Nance-Spronson TE, Kumar AJ, Mellits ED, McArthur JC. Patterns of cerebral atrophy in HIV-1 infected individuals: Results of a quantitative MRI analysis. Neurology 1992;42:2125–2130. [PubMed: 1436522]

Dausey DJ, Desai RA. Psychiatric comorbidity and the prevalence of HIV infection in a sample of patients in treatment for substance abuse. Journal of Nervous and Mental Disease 2003;191:10–17. [PubMed: 12544594]

Dew MA, Becker JT, Sanchez J, Caldararo R, Lopez OL, Wess J, Dorst SK, Banks G. Prevalence and predictors of depressive, anxiety, and substance use disorders in HIV-infected and uninfected men: A longitudinal evaluation. Psychological Medicine 1997;27:395–409. [PubMed: 9089832]

Dozois D, Dobson K, Ahnberg J. A psychometric evaluation of the Beck Depression Inventory-II. Psychological Assessment 1998;10:83–89.

Duffy JD, Kant R. Apathy secondary to neurologic disease. Psychiatric Annals 1997;27:39–43.

Goggin KJ, Zisook S, Heaton RK, Atkinson JH, Marshall S, McCutchan JA, Chandler JL, Grant I. the HNRC Group. Neuropsychological performance in HIV-1 infected men with major depression. Journal of the International Neuropsychological Society 1997;3:457–464. [PubMed: 9322405]

Grant, I.; Martin, A. Neuropsychology of HIV infection. New York: Oxford University Press; 1994.

Grant I, Olshen RA, Atkinson JH, Heaton RK, Nelson J, McCutchan JA, Weintrich JD. Depressed mood does not explain neuropsychological deficits in HIV-infected persons. Neuropsychology 1993;7:53–61.

Hardy DJ, Hinkin CH, Satz P, Stenquist PK, van Gorp WG, Moore LH. Age differences and neurocognitive performance in HIV-infected adults. New Zealand Journal of Psychology 1999;28:94–101.

Harker JO, Satz P, Jones FD, Verma RC, Gan MP, Poer HL, Gould BD, Chervinsky AB. Measurement of depression and neuropsychological impairment in HIV-1 infection. Neuropsychology 1995;9:110–117.

Heaton RK, Grant I, Butters N, White DA, Kirson D, Atkinson JH, McCutchan JA, Taylor MJ, Kelly MD, Ellis RJ, Wolfson T, Velin R, Marcotte TD, Hesselink JR, Jernigan TL, Chandler J, Wallace M, Abramson I. the HNRC Group. The HNRC 500 — Neuropsychology of HIV infection at different disease stages. Journal of the International Neuropsychological Society 1995;1:231–251. [PubMed: 9375218]

Hinkin CH, van Gorp WG, Satz P, Weisman JD, Thommes J, Buckingham S. Depressed mood and its relationship to neuropsychological test performance in HIV-1 seropositive individuals. Journal of Clinical and Experimental Neuropsychology 1992;10:79.

Hinkin, CH.; Castellon, SA.; van Gorp, WG.; Satz, P. Neuropsychological features of HIV infection. In: van Gorp, WG.; Buckingham, SU., editors. A Mental Health Practitioner's Guide to Neuropsychiatric Complications of HIV/AIDS. New York: Oxford University Press; 1997.

Johnson JG, Rabkin JG, Lipsitz JD, Williams JBW, Remien RH. Recurrent major depressive disorder among human immunodeficiency virus (HIV) – positive and HIV-negative intravenous drug users: Findings of a 3-year longitudinal study. Comprehensive Psychiatry 1999;40:31–34. [PubMed: 9924874]

Judd FK, Mijch AM. Depressive symptoms in patients with HIV infection. Australian and New Zealand Journal of Psychiatry 1996;30:104–109. [PubMed: 8724332]

Kalichman SC, Rompa D, Cage M. Distinguishing between overlapping somatic symptoms of depression and HIV disease in people living with HIV/AIDS. Journal of Nervous and Mental Disease 2000;188:662–670. [PubMed: 11048815]

Kalichstein AD, Hinkin CH, van Gorp WG, Castellon SA, Satz P. Depression predicts procedural but not episodic memory in HIV-1 infection. Journal of Clinical and Experimental Neuropsychology 1998;20:529–535. [PubMed: 9892056]

Kaplan, EF.; Goodglass, H.; Weintraub, S. The Boston Naming Test. Philadelphia: Lea & Febiger; 1983.

Kemeny ME, Dean L. Effects of AIDS-related bereavment on HIV progression among New York City gay men. AIDS Education & Prevention 1995;7(suppl):36–47. [PubMed: 8664097]

Kendall PC, Hollon SD, Beck AT, Hammen CL, Ingram RE. Issues and recommendations regarding the use of the Beck Depression Inventory. Cognitive Therapy and Research 1987;11:289–299.

Ketzler S, Weis S, Haug H, Budka H. Loss of neurons in the frontal cortex in AIDS brains. Acta Neuropathologica 1990;80:92–94. [PubMed: 2360420]

Kuzis G, Sabe L, Dorego F, Starkstein SE. Neuropsychological correlates of apathy and depression in patients with dementia. Neurology 1999;52:1403–1407. [PubMed: 10227625]

Law WA, Martin A, Salazar AM, Mapou RL. Symptoms of depression in HIV-infected individuals: Etiological considerations. Neuropsychiatry, Neuropsychology, and Behavioral Neurology 1993;6:181–186.

Lesserman J. HIV disease progression: Depression, stress, and possible mechanisms. Biological Psychiatry 2003;54:295–306. [PubMed: 12893105]

Levin B, Kumar M, Fernandez B, Duncan R. Cerebrospinal fluid dopamine levels in HIV-1 infection: Correlations with immune function and neuropsychological profile. Neurological and Neuropsychological Complications of HIV Infection 1991;2:122. (abstract).

Levy ML, Cummings JL, Fairbanks LA, Masterman D, Miller BL, Craig AH, Paulsen JS, Litvan I. Apathy is not depression. Journal of Neuropsychiatry and Clinical Neurosciences 1998;10:314–319. [PubMed: 9706539]

Lezak, M. Neuropsychological assessment. 3. NewYork: Oxford University Presss; 1995.

Lopez O, Smith G, Meltzer C, Becker JT. Dopamine systems in human immunodeficiency virus-associated dementia. Neuropsychiatry, Neuropsychology, & Behavioral Neurology 1999;12:184–192.

Lyketsos CG, Hoover DR, Guccione M, Senterfitt W, Dew MA, Wesch J, vanRaden MA, Treisman GJ, Morgenstern H. Depressive symptoms as predictors of medical outcomes in HIV infection. Journal of the American Medical Association 1993;270:2563–2567. [PubMed: 7901432]

Maj M, Janssen R, Starace F, Zaudig M, Satz P, Sughondhabirom B, Luabeya M, Riedel R, Ndetei D, Calil H, Bing E, St Louis M, Sartorius N. WHO neuropsychiatric AIDS study, cross-sectional phase I: Study design and psychiatric findings. Archives of General Psychiatry 1994a;51:39–49. [PubMed: 8279928]

Maj M, Satz P, Janssen R, Zaudig M, Starace F, D'Elia L, Sughondhabirom B, Mussa M, Naber D, Ndetei D, Schulte G, Sartorius N. WHO neuropsychiatric AIDS study, cross-sectional phase II: Neuropsychological and neurological findings. Archives of General Psychiatry 1994b;51:51–61. [PubMed: 8279929]

Mapou RL, Law WA, Martin A, Kampen D, Salazar AM, Rundell JR. Neuropsychological performance, mood, and complaints of cognitive and motor difficulties in individuals infected with the human immunodeficiency virus. Journal of Neuropsychiatry and Clinical Neurosciences 1993;5:86–93. [PubMed: 8428141]

Marin RS. Apathy: A neuropsychiatric syndrome. Journal of Neuropsychiatry and Clinical Neurosciences 1991;3:243–254. [PubMed: 1821241]

Marin RS, Firinciogullari S, Biedrzycki RC. Group differences in the relationship between apathy and depression. Journal of Nervous and Mental Disease 1994;182:235–239. [PubMed: 10678322]

Martin EM, Robertson LC, Edelstein H, Jagust W, Sorensen DJ. Performance of patients with early HIV-1 infection on the Stroop task. Journal of Clinical and Experimental Neuropsychology 1992;14:857–868. [PubMed: 1474150]

Mason KI, Campbell A, Hawkins P, Madhere S, Johnson K, Takushi-Chinen R. Neuropsychological functioning in HIV-positive African-American women with a history of drug use. Journal of the National Medical Association 1998;90:665–674. [PubMed: 9828581]

Mayne TJ, Vittinghoff E, Chesney MA, Barrett DC, Coates TJ. Depressive affect and survival among gay and bisexual men infected with HIV. Archives of Internal Medicine 1996;156:2233–2238. [PubMed: 8885823]

McAllister-Williams RH, Ferrier IN, Young AH. Mood and neuropsychological function in depression: the role of corticosteroids and serotonin. Psychological Medicine 1998;28:573–584. [PubMed: 9626714]

Navia B, Cho ES, Petito C, Price R. The AIDS dementia complex: II. Neuropathology. Annals of Neurology 1986;19:525–535. [PubMed: 3014994]

Perkins DO, Stern RA, Golden RN, Murphy C, Naftolowicz D, Evans D. Mood disorders in HIV infection: Prevalence and risk factors on a non-epicenter of the AIDS epidemic. American Journal of Psychiatry 1994;151:233–236. [PubMed: 8296895]

Perkins D, Leserman J, Stern R, Baum S, Liao D, Golden R, Evans D. Somatic symptoms and HIV infection: Relationship to depressive symptoms and indicators of HIV disease. American Journal of Psychiatry 1995;152:1776–1781. [PubMed: 8526245]

Prado G, Feaster DJ, Schwartz SJ, Pratt IA, Smith L, Szapocznik J. Religious Involvement, Coping, Social Support, and Psychological Distress in HIV-Seropositive African American mothers. AIDS & Behavior 2004;8:221–235. [PubMed: 15475672]

Rabkin J. Prevalence of psychiatric disorders in HIV illness. International Review of Psychiatry 1996;8:157–166.

Rabkin JG, Goetz RR, Remien RH, Williams JB, Todak G, Gorman JM. Stability of mood despite HIV illness progression in a group of homosexual men. American Journal of Psychiatry 1997;154:231–238. [PubMed: 9016273]

Reitan, RM. Manual for administration of neuropsychological test batteries for adults and children. Indianapolis: 1969.

Richardson MA, Satz P, Myers HF, Miller EN, Bing EG, Fawzy F, Maj M. Effects of depressed mood versus clinical depression on neuropsychological test performance among African American men impacted by HIV/AIDS. Journal of Clinical and Experimental Neuropsychology 1999;21:769–783. [PubMed: 10649533]

Satz, P.; Myers, HF.; Maj, M.; Fawzy, F.; Forney, DL.; Bing, EG.; Richardson, MA.; Janssen, R. Depression, substance use, and sexual orientation as co-factors in HIV-1 infected men: Cross-cultural comparisons. In: Onkin, LS.; Blaine, JD.; Genzer, S.; Horton, AM., editors. NIDA Research Monograph. Vol. 172. Rockville, MD: National Institute on Drug Abuse; 1997.

Smith, A. Symbol Digit Modalities Test. Los Angeles: Western Psychological Services; 1991.

Starkstein S, Mayberg HS, Preziosi TJ, Andrezejewski P, Leiguarda R, Robinson RG. Reliability, validity, and clinical correlates of apathy in Parkinson's disease. Journal of Neuropsychiatry and Clinical Neurosciences 1992;4:134–139. [PubMed: 1627973]

Startup M, Rees A, Barkham M. Components of major depression examined via the Beck Depression Inventory. Journal of Affective Disorders 1992;26:251–259. [PubMed: 1479138]

Statistical Package for Social Sciences for Windows, Version 8.0. Chicago, IL: SPSS Inc; 1997.

Steer RA, Beck AT, Riskind JH, Brown G. Relationships between the Beck Depression Inventory and Hamilton Psychiatric Rating Scale for Depression in depressed outpatients. Journal of Psychopathology and Behavioral Assessment 1987:327–339.

Steer RA, Ball R, Ranieri WF, Beck AT. Dimensions of the Beck Depression Inventory-II in clinically depressed outpatients. Journal of Clinical Psychology 1999;55:117–128. [PubMed: 10100838]

Stern R. Neuropsychiatric aspects of HIV, with a note on pyschoneuroimmunology. Advances 1994;10:28–31.

Stern Y, Liu X, Marder K, Today G, Sano M, Ehrhardt A, Gorman J. Neuropsychological changes in a prospectively followed cohort of homosexual and bisexual men with and without HIV infection. Neurology 1995;45:467–472. [PubMed: 7898698]

Stern Y, Marder K, Bell K, Chen J, Dooneief G, Goldsten MA, Mindry D, Richards M, Sano M, Williams J, Gorman J, Ehrhardt A, Mayeux R. Multidisciplinary baseline assessment of homosexual men with and without the human immunodeficiency virus infection. Archives of General Psychiatry 1991;48:131–138. [PubMed: 1671199]

Stroop JR. Studies of interference in serial verbal reactions. Journal of Experimental Psychology 1935;18:643–662.

Summers J, Zisook S, Atkinson JH, Sciolla A, Whitehall W, Brown S, Patterson T, Grant I. Psychiatric morbidity associated with acquired immune deficiency syndrome-related grief resolution. Journal of Nervous and Mental Disease 1995;183:384–389. [PubMed: 7798087]

Tabachnick, BG.; Fidell, LS. Using multivariate statistics. 3. New York: Harper Collins College Publishers; 1996.

Tanaka JS, Iluba GJ. Confirmatory hierarchical factor analyses of psychological distress measures. Journal of Personality and Social Psychology 1984;46:621–635.

van Gorp WG, Miller EN, Marcotte TD, Dixon W, Paz D, Selnes O, Wesch J, Becker JT, Hinkin CH, Mitrushina M, Satz P, Weisman JD, Buckingham SL, Stenquist PK. The relationship between age and cognitive impairment in HIV-1 infection: Findings from the Multicenter AIDS Cohort Study and a clinical cohort. Neurology 1994;44:929–935. [PubMed: 8190299]

van Gorp WG, Miller EN, Satz P, Visscher B. Neuropsychological performance in HIV-1 immunocompromised patients: A preliminary report. Journal of Clinical and Experimental Neuropsychology 1989;11:763–773. [PubMed: 2808663]

Volk RJ, Pace TM, Parchman ML. Screening for depression in primary care patients: Dimensionality of the short form of the Beck Depression Inventory. Psychological Assessment 1993;5:173–181.

von Giesen HJ, Baecker R, Hefter H, Arendt G. Depression does not influence basal ganglia-mediated psychomotor speed in HIV-1 infection. Journal of Neuropsychiatry & Clinical Neurosciences 2001;13:88–94. [PubMed: 11207334]

Wallace J, Pfohl B. Age-related differences in the symptomatic expression of major depression. Journal of Clinical Psychology 1995;46:817–827.

Wechsler, D. Wechsler Memory Scale-revised. San Antonio, TX: The Psychological Coroporation; 1987.

Welch G, Hall A, Walkey F. The replicable dimensions of the Beck Depression Inventory. Journal of Clinical Psychology 1990;46:817–827. [PubMed: 2286677]

Williams JBW, Rabkin JG, Remien RH, Gorman JM, Ehrhardt AE. Multidisciplinary baseline assessment of homosexual men with and without human immunodeficiency virus infection. Archives of General Psychiatry 1991;48:124–130. [PubMed: 1671198]

Table 1

Demographic characteristics of the total sample and for the patients with diagnoses of AIDS

Variable	Total sample		AIDS only	
	Mean	SD	Mean	SD
Age (in years)	37.5	7.9	38.4	8.6
Education (# of years)	15.3	2.2	15.5	2.0
BDI total score	9.8	7.8	10.1	6.3
CD4 cell count (cells/mm^3)	281.9	224.5	145.1	209.3
Receiving disability	20.6%		47.0%	

Table 2

Means, standard deviations, and prevalence of the 21 items on the BDI

BDI item	Mean	*SD*	% Endorsed
1. Depressed mood	.45	.72	33
2. Pessimism regarding future	.53	.72	42
3. Sense of failure	.24	.65	15
4. Anhedonia	.51	.72	40
5. Guilty feelings	.26	.62	19
6. Sense of punishment	.18	.48	14
7. Self-dislike	.37	.66	29
8. Self-accusation	.57	.72	45
9. Suicidal ideation	.32	.53	28
10. Crying	.36	.63	29
11. Irritability	.54	.65	49
12. Social withdrawal	.48	.88	30
13. Difficulty making decisions	.44	.91	25
14. Distorted body image	.43	.81	29
15. Decreased initiative	.66	.92	44
16. Sleep disturbance	.63	.86	45
17. Anergia/fatigue	.86	.81	68
18. Loss of appetite	.23	.54	19
19. Weight loss	.31	.72	19
20. Somatic concerns	1.01	.99	65
21. Loss of libido	.69	.99	43

Table 3

Item loadings for rotated component matrix

Item	Factor/component		
	1	**2**	**3**
1. Depressed mood	.29	**.54**	.21
2. Pessimism regarding future	**.46**	.30	.21
3. Sense of failure	**.67**	.12	.01
4. Anhedonia	.20	**.66**	.15
5. Guilty feelings	**.69**	.15	−.01
6. Sense of punishment	**.75**	.00	.00
7. Self-dislike	**.77**	.02	00
8. Self-accusation	**.52**	.01	.20
9. Suicidal ideation	**.50**	.18	.24
10. Crying	**.49**	.09	.17
11. Irritability	.34	**.41**	.29
12. Social withdrawal	.26	**.71**	−.11
13. Difficulty making decisions	.12	**.71**	.00
14. Distorted body image	.32	.22	.32
15. Decreased initiative	.03	**.73**	.05
16. Sleep disturbance	.14	.13	**.50**
17. Anergia/fatigue	.09	**.58**	.04
18. Loss of appetite	.23	.07	**.66**
19. Weight loss	.25	−.03	**.51**
20. Somatic concerns	.14	.17	**.64**
21. Loss of libido	.01	.39	.37
Coefficient alpha	.79	.80	.59

Note: Factor 1 "Self-Denigration", Factor 2 "Mood-Motivation Disturbance", Factor 3 "Somatic Disturbance". Loadings which are noted in bold type indicate assignment of items to factors. Coefficient alpha for items comprising factor only.

Table 4

Bivariate correlation between seven neurocognitive factors and three BDI factors

Neurocognitive factor	BDI factor			
	SR	MM	SOM	BDI
Verbal memory ($N = 220$)	−.08	−.27**	−.06	−.18*
Visual memory ($N = 218$)	−.16*	−.14	−.06	−.14
Psychomotor ($N = 227$)	−.06	−.10	−.13	−.05
Executive ($N = 200$)	−.04	−.22**	−.05	−.16*
Motor ($N = 230$)	−.05	−.23**	−.06	−.11
Spatial ($N = 200$)	−.16*	−.04	−.02	−.06
Language ($N = 211$)	−.11	−.08	−.01	−.08

Note: SR = "Self-Reproach", MM = "Mood-Motivation Disturbance", SOM = "Somatic Disturbance", BDI = Total Score for Beck Depression Inventory. For each analysis, negative correlation suggests that higher score on the BDI factor of interest is associated with worse (variables transformed as necessary) performance on the variables comprising that neurocognitive factor. Sample size varied per analysis as not all 247 participants were given all neurocognitive measures.

*
$p < .05$.

**
$p < .01$.

Table 5

Standardized Beta coefficients for BDI factors predicting performance on selected neurocognitive factors

Regression model	β	t	ΔF
Verbal memory			
Step 1			6.37**
Age		−2.95*	
Education		−2.73*	
Step 2			4.39*
Age	−.21	−2.97*	
Education	−.17	−2.37*	
BDI-MM	−.26	−3.45**	
BDI-SR	−.03	−0.38	
BDI-SOM	−.02	−0.22	
Visual memory			
Step 1			6.87*
Age	−.25	−2.95*	
Education	−.14	−2.73*	
Step 2			2.60
Age	−.28	−3.87**	
Education	−.11	−1.51	
BDI-MM	−.07	−0.91	
BDI-SR	−.14	−1.81	
BDI-SOM	−.03	−0.38	
Executive			
Step 1			4.28*
Age	−.17	−2.29*	
Education	−.17	−2.36*	
Step 2			3.12*
Age	−.17	−2.22*	
Education	−.16	−2.20*	
BDI-MM	−.23	−2.86*	
BDI-SR	−.08	−0.93	
BDI-SOM	−.03	−0.33	
Motor			
Step 1			3.20*
Age	−.18	−2.50*	
Education	−.08	−1.12	
Step 2			5.40**
Age	−.18	−2.54*	

Regression model	β	t	ΔF
Education	−.07	−0.99	
BDI-MM	−.29	−3.78**	
BDI-SR	−.15	−1.83	
BDI-SOM	−.05	−0.54	
Spatial			
Step 1			6.88**
Age	−.02	−0.27	
Education	−.25	−3.49**	
Step 2			1.12
Age	−.01	−3.87**	
Education	−.23	−1.51	
BDI-MM	−.04	−0.48	
BDI-SR	−.15	−1.82	
BDI-SOM	−.03	−0.39	

Note. BDI-SR = BDI Self-Reproach factor, BDI-MM = BDI Mood-Motivation Disturbance factor, BDI-SOM = BDI Somatic Disturbance factor. ΔF represents the significance of each step of the model.

*
$p < .05$.

**
$p < .01$.

Table 6

Percentage of total BDI score accounted for by each of the three BDI factors as a function of AIDS diagnosis

BDI Factor	Pre-AIDS	AIDS
Self reproach	33%	24%
Mood-Motivation	46%	49%
Somatic disturbance	21%	27%