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Social support, neuropsychological performance, and depression in HIV infection

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Abstract

The present study was designed to examine the impact of neuropsychological performance on the relationships between stress, social support, and depression in 217 HIV-infected men. Using path analysis, the contributions of four domains of cognitive functioning (memory, attention, executive function, and psychomotor speed), IQ, and relevant psychosocial variables to depression were evaluated. In the model which best fit the data, cognitive domains did not contribute directly to depression, but contributed significantly to psychosocial variables which affected levels of depression. Attention and executive function contributed to reduced illness-related disability; while higher IQ was associated with fewer stressful life events. Number of stressful life events and level of illness-related disability were associated with depressive symptoms. Higher IQ led to greater numbers of social contacts, which was associated with fewer reported symptoms of depression. These findings suggest that better neuropsychological performance may lead to reduced stress and perceived disability, and more available social contacts. By these multiple paths, different domains of cognitive ability contribute indirectly to ameliorating depression in HIV-infected men. (*JINS*, 2002, *8*, 436–447.)

Keywords: HIV, Neuropsychological performance, Depression, Social support

INTRODUCTION

Symptoms of depression are common among persons with HIV infection and may substantially affect feelings of well-being and quality of life (Stober et al., 1997). Social support is a widely studied construct which has been associated with lower levels of depressive symptoms in a variety of populations (Cohen & Wills, 1985). The beneficial effects of social support on depression have been confirmed among HIV-positive individuals (Hays et al., 1992; Lackner et al., 1993b). Factors which may contribute to this relationship have not, however, been carefully examined in the context of HIV infection. HIV has been shown to have detrimental effects on cognitive function, as measured by performance on neuropsychological tests, even in early stages of infection (White et al., 1995). It is not known whether cognitive dysfunction influences the ability to access available social supports. Furthermore, it is unclear whether deficits in particular cognitive domains are associated with ability to obtain or use social support.

HIV-positive individuals have higher rates of depressive syndromes than are found in the general population. This increased risk is also reported among those who report engaging in behaviors which would put them at risk for contracting HIV (Maj, 1996). Among men at risk for AIDS, depressive symptoms are higher than in the population at large, and those who are HIV-positive have been found to have a small but significant elevation in depressive symptoms when compared to HIV-negative at-risk men (Ostrow et al., 1989; Rosenberger et al., 1993).

Several factors have been identified which may predict increased depression in HIV-infected persons. These include number of years living with an HIV-positive diagnosis (Kalichman et al., 1995), avoidant *versus* active coping style (Wolf et al., 1991), and excessive rumination about health-related problems (King, 1989). In addition, numerous studies have examined the relationship between social support and depression in this population. Perry (1994) identified past depression, personality disorder, and low levels of social support as the strongest predictors of HIV-related

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depression. Our preliminary data have identified lower IQ as a potential risk factor for depression (Bornstein et al., 1993).

Social support has become one of the most widely studied concepts pertaining to psychological distress and well-being (Veiel & Baumann, 1992). Inverse correlations between social support and depression have been found in numerous samples of HIV-positive individuals (Nott et al., 1995; Ostrow et al., 1991; Siegel et al., 1994). Further, longitudinal studies have found that social support directly contributes to later mental health in this population (Hays et al., 1992; Lackner et al., 1993a, 1993b; Siegel et al., 1997). Social support has beneficial effects on self-reported quality of life (Friedland et al., 1996), overall well-being (Ostrow et al., 1991), and adjustment to AIDS (Rodgers, 1995), and is related to lower levels of loneliness (Gant & Ostrow, 1995), hopelessness (Zich & Temoshok, 1987), and general distress (Wolcott et al., 1986). Support has also been associated with fewer physical symptoms, better perceived health, and improved immune function (Namir et al., 1989; Pakenham et al., 1994; Wolcott et al., 1986). Very few studies have examined potential mediators between social support and depression or controlled for factors which may impact this relationship.

Social support has been defined in many diverse ways, however, the majority of support measures can be classed into two broad categories. Quantitative, also known as structural or social network, measures provide an indication of how many and what type of relationships individuals have. The qualitative or functional perspective, often termed perceived social support, includes measures of the quality of support received, or whether particular social needs are fulfilled. Such measures indicate a person's perception of the functions filled by their social contacts, regardless of the number of people they are in contact with or their relationship to the subject (Cohen & Syme, 1985). There have been two primary approaches to examine the relationship between social support, however it is conceived, and psychological functioning. The buffering model proposes that social support moderates stressful life events to protect individuals against depression or other psychological distress. The main effects model of support proposes that support has a direct, positive effect on mental health. When stress is included in studies using the main effects model, it is thought to have a detrimental effect on psychological well-being, independent from the impact of social support.

Cohen and Wills (1985) examined the empirical support for each of the two models. They reviewed studies of diverse psychological and health-related outcomes, which predominantly included measures of psychological distress. In this review, studies were classified based on whether the support measures used were functional (perceived support) or structural (social network) measures. These authors reported that social network measures generally showed main effects, suggesting that the degree to which a person has contact with a large network of individuals predicts overall well-being. Studies using functional measures produced less consistent results, but tended to support the stress-buffering hypothesis. The authors concluded that both buffering and main effects models can be supported, and that the domain of support measured influences the type of relationship between support and well-being. Further, they found little evidence to support a model in which both a main effect and a buffering effect are present.

Early in the HIV/AIDS epidemic, it became evident that some portion of patients experienced substantial cognitive changes related to the disease. The term HIV-1-associated dementia complex (HAD) was adopted to describe the most severe form of cognitive impairment, in which patients demonstrate moderate to severe impairment in work or activities of daily living (Working Group of the American Academy of Neurology AIDS Task Force, 1991). Patients with HAD may experience forgetfulness, difficulty concentrating, loss of balance, motor slowing, and apathy or social withdrawal. HAD has been considered a "subcortical dementia" because it primarily involves motor slowing and rarely involves typical "cortical" symptoms such as aphasia or apraxia (Navia et al., 1986). It is estimated that HAD occurs in 5–15% of HIV-infected persons (Kelly et al., 1996). A second cognitive manifestation of HIV-infection, more common and less severe than HAD, has been termed HIV-1 associated minor cognitive/motor disorder (MCMD). MCMD is associated with neuropsychological changes such as decreased attention and concentration, mental slowing, or memory problems, which produce mild difficulty in work or daily activities (Working Group of the American Academy of Neurology AIDS Task Force, 1991). In addition to these disorders, it appears that some portion of asymptomatic, HIV-infected persons may demonstrate mild neuropsychological deficits. White et al. (1995) reviewed 57 studies of neuropsychological performance in HIV-positive, asymptomatic persons and found that the median rate of cognitive impairment was 35% in HIV-positive, asymptomatic participants, but only 12% in HIV-negative controls.

In those HIV-infected patients who are cognitively impaired, several specific domains of neuropsychological performance are typically affected. In a study of 389 nondemented HIV-positive patients at various stages of infection, Heaton et al. (1995) found that two domains, attention/speed of information processing and learning, were affected in over half of patients rated as cognitively impaired. Other authors have reported deficits in verbal memory (Delis et al., 1995), executive function (Sahakian et al., 1995), and working memory (Stout et al., 1995).

It is evident that many HIV-infected persons experience symptoms of depression and that social support may help mitigate against depression. The precise relationship between support and depression in this population, however, is not clear. Studies using a wide variety of support measures have found that HIV-infected patients with higher levels of social support experience fewer depressive symptoms, however, little research has examined whether there are additional factors which contribute to this relationship. Understanding of specific characteristics which may lead to

symptoms of depression and enhance or attenuate support may enable identification of individuals who are at especially high risk for depression.

The question of whether neuropsychological performance can affect ability to seek and use social support and, thus, reduce depression has not been explored in any population. This question may be particularly relevant for individuals who experience a wide range of cognitive impairment, as is the case with HIV-infected people. The current study was conducted to examine how cognitive ability may influence the relationship between social support and depression in a group of HIV-positive gay or bisexual men. Consistent with the findings of Cohen and Wills (1985), this study used a measure of network support (number of available social contacts) to test the main effects hypothesis that social support and stress contribute independently to depression. Furthermore it was of interest to determine whether specific cognitive domains were of particular relevance in moderating or mediating this relationship.

In order to achieve these goals path analysis was employed to test and modify a specific theoretical model. In addition to social support, neuropsychological performance, and depression, the model included relevant psychosocial and disease-related factors including stress, illness-related dysfunction, socioeconomic status, immune function, and intelligence.

METHODS

Research Participants

Participants were 217 homosexual or bisexual HIV positive men enrolled in a study of the effects of HIV infection on cognitive performance. Those who had histories of intravenous drug use, head injury with unconsciousness greater than 1 hr, or neurologic disease were excluded from participation. Their average age was 34.0 years ($SD = 7.3$), mean education was 14.0 years ($SD = 2.4$), and 92.2% ($n = 200$) were White. The majority of participants ($n = 129$, 59.4%) were medically asymptomatic other than generalized lymphadenopathy. An additional 58 (26.7%) were symptomatic but did not meet Centers for Disease Control (CDC) criteria for AIDS. Thirty individuals (13.8%) had AIDS. The average CD4 cell count among the 189 participants for whom this information was available was 404.7 ($SD = 266.6$). These data were collected prior to the advent of protease inhibitors and highly active antiretroviral treatment regimens.

Neuropsychological Measures

The battery of neuropsychological tests was chosen to represent a broad range of functions and to include tasks shown to be sensitive to the effects of HIV infection. The instruments used included measures of overall ability, executive function, learning and memory, attention, and psychomotor speed. General ability was measured using the Wechsler Adult Intelligence Scale–Revised (WAIS–R; Wechsler, 1981). Measures of executive function included the Wisconsin Card Sorting Test (Heaton et al., 1993), Verbal Concept Attainment Test (VCAT; Bornstein & Leason, 1985), Figural Fluency Test (Ruff et al., 1987), Trail Making Test, Part B (Reitan & Davidson, 1974), and Verbal Fluency Test (Borkowski et al., 1967). Learning and memory were measured using the Selective Reminding Test (Buschke & Fuld, 1974). Attention was measured using the Visual Span Forward and Backward subtests from the Wechsler Memory Scale–Revised (Wechsler, 1987) and the Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977). Psychomotor function was measured with the Grooved Pegboard Test (Kløve, 1963), the Trail Making Test, Part A (Reitan & Davidson, 1974), and measures of simple and choice reaction time. This battery includes at least one test from each of the nine neuropsychological domains recommended by the NIMH Workshop on Neuropsychological Assessment Approaches for assessment of HIV-related cognitive dysfunction (Butters et al., 1990).

In order to measure domains of cognitive function, each individual test score was transformed into a *Z* score based on the means of the HIV-positive subjects in the study. This was done to create a common metric so that scores on various tests could be easily compared and mathematically combined. These *Z* scores were then averaged to create four domain scores (executive function, memory, attention, and psychomotor function) for each subject. Each domain score represents the mean of the *Z* scores for the individual measures in that domain.

Psychosocial and Health-Related Measures

Depression was measured using the Cognitive–Affective subscale of the Beck Depression Inventory (Beck et al., 1961). Previous research has suggested that items on this measure that represent somatic symptoms of depression are highly correlated with physical symptoms of HIV. In contrast, items that represent cognitive–affective symptoms of depression have been shown to better reflect psychological distress in persons with HIV (Kalichman et al., 1995).

Social support was measured using the Social Network Index summary score for number of potential social contacts (Cohen, 1991). This score is the total number of individuals a participant has regular contact with, across 11 possible social roles such as friends, family members, neighbors, and church members. Socioeconomic status was determined using the Hollingshead ratings of occupation, based on the usual occupation participants pursued when gainfully employed (Hollingshead, 1975). Illness-related dysfunction was measured using the Sickness Impact Profile (SIP; Bergner et al., 1976). The SIP is a self-report instrument which provides an indication of level of impairment across 12 categories including physical functioning, psychosocial behavior, and daily activities. This instrument, sometimes referred to as a measure of quality of life, is sensitive to different stages of HIV infection (Ragsdale &

Morrow, 1990). Stress was measured using a 58-item version of the Psychiatric Epidemiology Research Interview (PERI; Dohrenwend et al., 1978), which provides a count of the number of notable life events experienced by each participant in the past year. This method of scoring this instrument has been associated with level of distress, and weighting of the impact of individual events experienced does not alter the relationship between stressful life events and depression, therefore weighted totals were not used (Kale & Stenmark, 1983). A separate measure of stress requested that participants provide a subjective rating of the stress in their lives, on a 1 to 10 scale.

Data Analysis

Data were maintained and all basic calculations and analyses were performed using the SPSS Statistical Package, version 7.0 for Windows. Variables to be used in path analytic models were transferred into the SYSTAT 8.0 statistical package and path analysis was conducted using the RAMONA procedure. RAMONA is based on the McArdle and McDonald Reticular Action Model for path analysis, and allows for causal interpretations of the relationships among variables (McArdle & McDonald, 1984). The estimation method was Maximum Wishart likelihood and analyses were performed on correlation matrices, derived by SYSTAT from raw data. All analyses used a significance level of $p < .05$.

RESULTS

Examination and Transformation of Data

Initial examination revealed that the distribution of three variables (depression, life events, and SIP total score) deviated notably from normal. Overall, participants tended to have low rates of depression, few stressful life events, and low rates of illness-related disability. Square root transformations resulted in normal distributions; when these variables are referred to from this point forward, the score described is the transformed score. These transformations may slightly increase the likelihood that significant results involving these variables are due to chance, however, if untransformed, the low rates of the variables of interest would most likely lead to a failure to detect any existing relationships in the data. In addition, the statistical assumptions of the tests used necessitated transformation of the variables so that they were normally distributed. Table 1 shows descriptive statistics for each variable included in the model. In addition, original scores for those variables which were transformed and total Beck Depression Inventory scores are provided in this table. Correlations between study variables are shown in Table 2.

Table 1. Descriptive statistics for study and related variables

Variable	*n*	*M*	*SD*
Total depression[a]	216	12.7	9.3
Affective depression	217	6.7	6.1
Square root[b]	217	2.3	1.2
Executive function[bc]	217	0.03	0.53
Memory[bc]	217	0.02	0.64
Attention[bc]	217	0.02	0.73
Motor function[bc]	217	0.00	0.71
Full scale IQ[b]	217	103.2	13.5
Social contacts[b]	217	18.1	8.2
Subjective stress[b]	217	6.4	2.2
Sickness Impact Profile total	217	9.0	9.2
Square root[b]	217	2.5	1.6
CD4 percentage[bd]	189	22.2	11.1
Total life events	217	8.8	4.5
Square root[b]	217	2.9	0.8
Positive life events	217	3.9	2.4
Square root	217	1.9	0.7
Negative life events	217	3.6	2.6
Square root	217	1.7	0.8
Occupation[b]	217	4.6	2.3

[a]One participant neglected to complete somatic items.
[b]Included in initial model.
[c]Mean of *Z* scores for all measures within domain.
[d]Immune data was not available for 28 participants.

Specification and Evaluation of Theoretical Model

In the initial theory-based model, shown in Figure 1, the four domains of cognitive function were predicted to covary with each other and with intelligence. All four cognitive domains were predicted to be associated with depression such that lower levels of cognitive performance would be associated with higher levels of depression. Higher levels of executive function, memory, and attention and greater full scale IQ scores were predicted to lead to more social contacts. Motor function was not predicted to affect number of social contacts. Although motor function (e.g., ambulation) could have such an effect, the tests were not related to this type of motor function. Social support was expected to have a direct, inverse effect on depression such that larger numbers of social contacts would result in lower levels of depression.

In order to account for as much variance as possible and to better predict depression, the subjective stress rating, total number of stressful life events (PERI), level of illness-related dysfunction (SIP), socioeconomic status (occupation), and CD4 percentage were also included in the model. Consistent with the main effects theory of the effect of social network size on depression, stress was included as a separate predictor of depression, independent from social contacts. CD4 percentage, occupation, illness-related dysfunction, and life events were predicted to covary with one another. Lower occupation and CD4 percentage scores and higher illness-related dysfunction and life events scores were expected to be causally related to subjective stress. Illness-related dysfunction and life events were both predicted to contribute causally to subjective stress, but not to correlate directly with each other. Subjective stress, illness-related

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Table 2. Correlations among study variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Depression[b]	—										
2. Executive function	−.19[a]	—									
3. Memory	−.02	.29[a]	—								
4. Attention	−.24[a]	.53[a]	.28[a]	—							
5. Motor function	−.06	.50[a]	.24[a]	.36[a]	—						
6. Full scale IQ	−.32[a]	.65[a]	.39[a]	.54[a]	.36[a]	—					
7. Social contacts	−.35[a]	.13	.12	.23[a]	.08	.28[a]	—				
8. Subjective stress	.45[a]	−.13	.06	−.15[a]	−.05	−.20[a]	−.09	—			
9. SIP[bc]	.50[a]	−.36[a]	−.20[a]	−.35[a]	−.23[a]	−.34[a]	−.35[a]	.35[a]	—		
10. CD4 percentage	.05	.01	.09	.09	.13	.05	.08	.02	−.08	—	
11. Life events[b]	.27[a]	.02	.14[a]	−.04	.03	−.09	.07	.32[a]	.12	.03	—
12. Occupation	−.10	.24[a]	.15[a]	.13	.01	.27[a]	.22[a]	−.04	−.12	−.09	−.04

[a] $p < .05$.
[b] Square root.
[c] Sickness Impact Profile.

dysfunction, and life events were all expected to have a direct, causal effect on depression, with higher levels of each resulting in greater depression. Occupation was predicted to covary with full scale IQ.

Upon analysis, the resulting value of the root mean square error of approximation (RMSEA) for this model was .11, indicating an inadequate fit with the data. RMSEA is a function of the difference between the sample correlation matrix and the population correlation matrix and takes into account the sample size and degrees of freedom of the model. A RMSEA of 0 indicates that a model fits perfectly with the data, a value of .05 is considered a close fit with the data, and values less than or equal to .08 are considered to represent a reasonable fit of the model to the data (Browne &

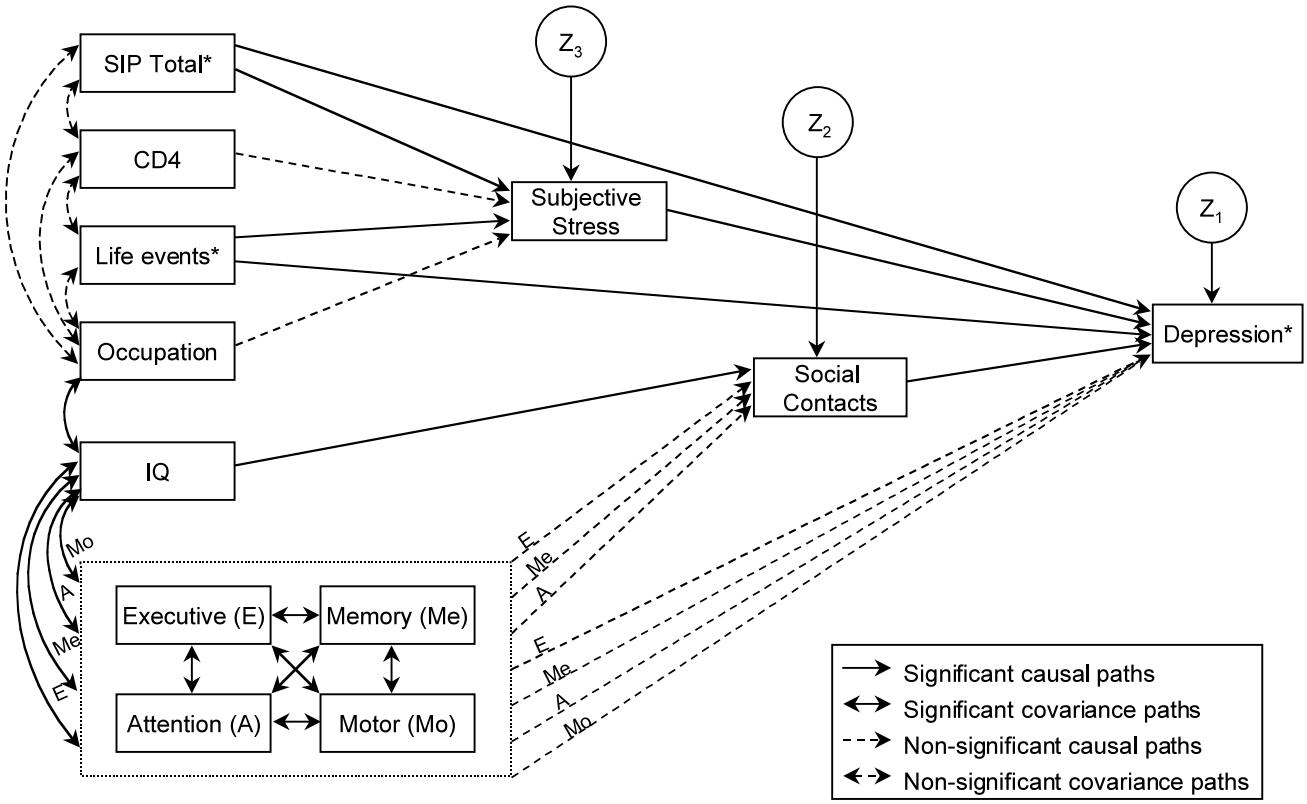

Fig. 1. Theory-based model of factors contributing to depression. *Square root. SIP = Sickness Impact profile.

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Table 3. RMSEA values for all models tested

Model	RMSEA	90% confidence interval
Theoretical Model	.11	.09, .13
Model 2	.11	.09, .14
Model 3	.09	.07, .11
Model 4	.00	.00, .05
Model 5	.04	.00, .07
Model 6	.02	.00, .06
Model 7	.02	.00, .06
Final Model	.00	.00, .05
Positive stress	.07	.04, .09
Negative stress	.07	.05, .10

Mels, 1997). The RMSEA value and 90% confidence interval for this model (Theoretical Model) as well as for all subsequent models tested are shown in Table 3.

Modification of Theoretical Model

Given the inadequate fit of the theoretical model to the data and the exploratory nature of the study, the theoretical model was modified carefully and systematically in order to find a data-driven model which provided an optimal fit with the data. Three guidelines were used to find pairs of relationships which might not have been correctly specified in the original model. First, paths which were not significant were removed. Second, as suggested by Billings and Wroten (1978), pairs of variables with residual correlations above .05 were considered to be potentially misspecified. For each such pair, a path was added to the model. Finally, in order for a path to be added, it could not be theoretically improbable or inconsistent with previous research.

As can be seen in Figure 1, none of the paths between CD4 percentage and other variables in the model were significant. In an attempt to keep this variable in the model, the residual correlation matrix was examined to find other variables for which a value of .05 or greater suggested the presence of a relationship with CD4 percentage. The previous paths from and between CD4 percentage were eliminated and six new paths were added to the model. In this new model (Model 2) only one path, the covariance between CD4 percentage and motor function, was significant. Therefore, for the next iteration of the model (Model 3) all other paths related to CD4 percentage were removed. Also in this step, all remaining nonsignificant paths from the initial theoretical model were removed.

In the next step, pairs of variables which had not previously been associated and which had residual correlations greater than .05 were added to the model. In order for the model to make theoretical sense and be consistent with the assumptions of the statistical technique, it was necessary to reconceptualize illness-related dysfunction and life events as endogenous variables, that is, caused by other variables in the model. This model (Model 4) included 21 new paths.

In the next model (Model 5), 14 nonsignificant paths were removed. At this point, no significant paths remained for CD4 percentage, so it was dropped from the model entirely. This resulted in an addition of 28 subjects to the analysis, as immunologic data was unavailable for this subset of participants. The final sample size, therefore, was 217. In the next model (Model 6), 4 paths were added, including paths which had been marginally significant ($p < .10$) in any of the previous models. The resulting model contained two nonsignificant paths, both of which were removed in the next iteration. The model which resulted (Model 7) was found to have a very good fit with the data (RMSEA = .02). As a final test, all pairs of variables with residual correlations greater than .05 were examined and paths between them were entered into the model one at a time, then removed if not significant. Only one path was added, resulting in the final, data-driven model (Final Model), shown in Figure 2. This model had an excellent fit to the data (RMSEA = 0.0, 90% confidence interval = 0, .05) and contained only significant paths. Table 4 provides path or covariance coefficients for all paths in the model. As suggested by Hatcher (1994), a test of the difference between the chi-squared values for each model using the difference between the degrees of freedom of the initial theoretical and final data-driven models was conducted. This analysis confirmed that the final data-driven model differed significantly from the theoretical model [$\chi^2(7) = 86.45$]. This difference, as well as χ^2 values for each model are shown in Table 5.

Description and Evaluation of Data-Driven Model

The model shown in Figure 2 was based on a theoretical model then modified to fit the data as closely as possible. This model contains numerous significant causal paths, each representing the unique relationship between two variables, while controlling for all other variables in the model. The factors which directly produced increased levels of depression were lower numbers of social contacts, higher perceived stress, higher numbers of stressful life events, and higher levels of illness-related dysfunction. In addition to increasing depression, stressful life events contributed to increased subjective stress levels and illness-related dysfunction. Greater perceptions of illness-related dysfunction also contributed to higher subjective stress levels. Greater numbers of social contacts were not only associated with decreased depression, they also resulted in reduced levels of illness-related dysfunction. Intelligence contributed to both increased social contacts and decreased stressful life events. Higher occupational levels resulted in increased numbers of social contacts. Better executive function and attention scores resulted in reduced levels of illness-related dysfunction. Better memory scores were associated with increased numbers of stressful life events. In this model, there were also numerous significant covariance relationships, that is, simple correlations between pairs of vari-

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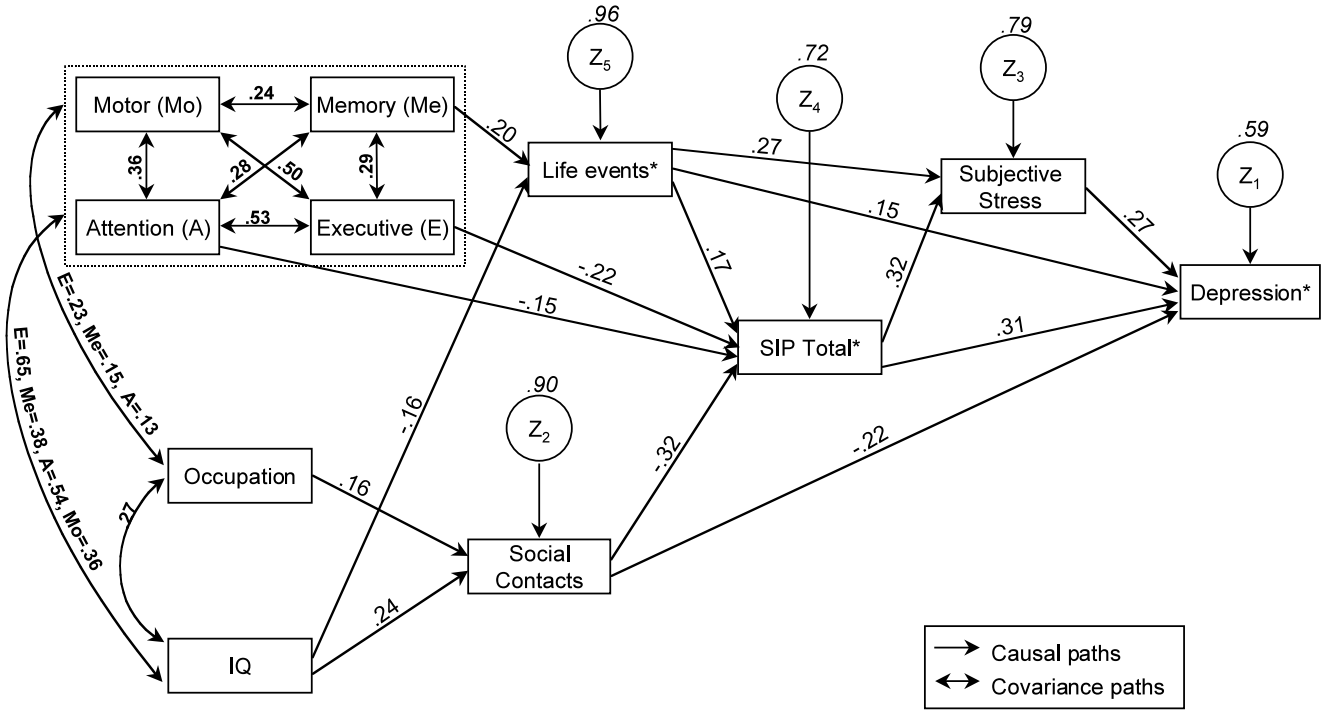

Fig. 2. Data-based model of factors contributing to depression. *Square root. SIP = Sickness Impact profile. All paths shown are significant, $p < .05$.

ables. Intelligence, all neuropsychological performance scores, and occupation were all significantly related to one another with one exception: Motor function was not related to occupation.

Follow-Up Analyses

In order to clarify how different types of stressful life events might contribute to depression and other variables, two variations of the data-driven model were tested. The events listed on the PERI were divided into positive life events (gains) and negative life events (losses), as specified by Dohrenwend et al. (1978). This resulted in a positive subscale containing 19 items and a negative subscale containing 29 items. Ten items could not be clearly classified as positive or negative and were omitted. For each of the supplementary analyses, the PERI subscale was added to the model between total life events and the variables which contribute to it, memory and IQ. The model using the PERI negative subscale was found to have a reasonable fit to the data (RMSEA = .07), as was the model using the PERI positive subscale (RMSEA = .07). RMSEA values and 90% confidence intervals are shown in Table 3. Before examining each model further, they were examined to determine if they differed significantly from the final model. Both the positive and negative models were significantly different from the final model [$\chi^2(10) = 47.18$ and 55.03, respectively; see Table 5]. Because both positive and negative models had the same number of degrees of freedom, they could not be compared to each other. In the positive model, all paths were significant except from IQ to positive life events. In the negative model, all paths were significant except from memory to negative life events.

DISCUSSION

The present study was intended to clarify how social support, neuropsychological performance, and other psychosocial variables contribute to symptoms of depression in persons with HIV infection. A primary hypothesis was that better neuropsychological performance would be associated with larger numbers of social contacts which would, in turn, result in lower levels of depression. This was confirmed for the relationship between IQ and social contacts. Higher IQ was associated with greater numbers of social contacts, which were associated with lower levels of depression. This primary hypothesis was not, however, upheld for the four composite domains of cognitive function. In the first model tested, neuropsychological performance in three different domains was predicted to contribute to increased levels of social support, however, none produced this effect. Neither IQ nor any of the four domains of neuropsychological performance contributed directly to reduction of depression. Rather, in addition to the direct effect of IQ on social contacts, domains of neuropsychological performance contributed indirectly to depression by four dif-

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Table 4. Path and covariance coefficients for final model

Path	Standardized path coefficient	Covariance coefficient (correlation)	Confidence interval (90%)
Contacts to depression	−.22		−.31, −.13
Life events to depression	.15		.06, .24
SIP to depression	.31		.21, .40
Subjective stress to depression	.27		.18, .37
IQ to contacts	.24		.13, .34
Occupation to contacts	.16		.05, .27
SIP to subjective stress	.32		.22, .42
Life events to subjective stress	.27		.18, .37
Executive to SIP	−.22		−.33, −.11
Attention to SIP	−.15		−.26, −.04
Life events to SIP	.17		.07, .26
Contacts to SIP	−.32		−.41, −.23
Memory to life events	.20		.09, .32
IQ to life events	−.16		−.28, −.05
Occupation with executive		.23	.14, .32
Occupation with memory		.15	.04, .25
Occupation with attention		.13	.02, .23
IQ with occupation		.27	.17, .36
IQ with executive		.65	.58, .71
IQ with memory		.38	.28, .47
IQ with attention		.54	.46, .61
IQ with motor		.36	.26, .45
Executive with attention		.53	.44, .60
Executive with motor		.50	.41, .56
Executive with memory		.29	.18, .39
Motor with attention		.36	.26, .45
Motor with memory		.24	.13, .34
Attention with memory		.28	.17, .38

Note. All paths are significant, $p < .05$. SIP = Sickness Impact Profile (illness-related disability).

ferent pathways. Better attention and executive function were found to contribute significantly to reduction of participants' perceptions of illness-related dysfunction. Perceptions of greater disability were related to increased feelings of depression. Higher IQ was associated with lower numbers of stressful life events, which were related to reduced levels of depression. Memory was also found to have an indirect effect on depression through life events, although this may have been an artifact of the study methods.

None of the neuropsychological domains were found to have a direct effect on depression levels in this population. This is consistent with some studies on the relationship between neuropsychological performance and depression. For example, Harker et al. (1995) found no significant associations between Beck Depression Inventory affective items and scores on nine neuropsychological tests. The present results do provide evidence that there is a relationship between neuropsychological performance and depression, but that it is an indirect relationship. Lower levels of both attention and executive function contributed to increased levels of illness-related dysfunction. Higher perceived disability, in turn, resulted in higher levels of depression. Thus, illness-related dysfunction can be seen to mediate between these two domains of neuropsychological performance and depression. Although the current study

Table 5. Chi-square values and differences for all models tested

Model	χ^2	χ^2 diff (*df*)
1. Theoretical	111.51	
2. Final	25.06	
Difference between final & theoretical		86.45[a] (7)
3. Positive stress	72.24	
Difference between positive stress & final		47.18[a] (10)
4. Negative stress	80.09	
Difference between negative stress & final		55.03[a] (10)

[a] $p < .05$

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examined symptoms of depression as opposed to depressive syndromes, this finding may have implications for intervention with HIV-infected persons. If those who experience greater cognitive dysfunction, specifically in the areas of attention and executive function, can be helped to approach daily activities more effectively, their feelings of depression might be reduced.

The inverse relationship between attention and executive function and illness-related dysfunction may be due to the specific cognitive abilities required to effectively complete many daily activities. These tasks often require planning and problem solving abilities or ability to concentrate and remain focused. Declines in attention and executive function could, therefore, contribute to problems in such areas as home care, communication, social activity, and alertness, all of which are domains of illness-related dysfunction measured by the SIP. It is interesting that the domains of memory and motor function were not associated with illness-related dysfunction. While forgetting or taking longer to complete tasks may be frustrating for those who experience these difficulties, they do not hinder the completion of activities typically impacted by physical illness, as measured on the SIP.

An alternate explanation for the relationship between neuropsychological performance and illness-related disability is that both are indicators of the effects of HIV on the brain and body. However, such an explanation would predict that declines in other cognitive domains, such as memory or motor function, would also be related to increased illness-related dysfunction, as these domains have been shown to be affected by HIV (Delis et al., 1995; Law et al., 1995). In addition, this explanation would predict that an indicator of the effects of HIV on the body, such as CD4 percentage, would be associated with greater levels of illness-related dysfunction. Therefore, as relationships were not found between memory, motor function, or CD4 percentage and illness-related disability it is unlikely that the relationships between attention and executive function, and illness-related disability are due to the physiological effects of HIV.

Memory was found to have an indirect effect on depression through a different mediating variable. Better memory scores were associated with increased numbers of stressful life events, which were related to higher levels of depression. It is possible that this finding may be an artifact of the study methods. Those with better memory performance may report a higher proportion of the stressful life events they experienced over the past year due to an ability to recall more events, even those which they did not feel impacted them substantially. If this is the case, the relationship between memory and life events is of questionable meaning. However, the effect may be genuine, that is, persons with better memories both report more life events and experience the stress associated with enduring greater numbers of life events. In this case, worse memory function would result in reduced feelings of stress as stressful life events would more easily fade from consciousness.

The follow-up analyses regarding positive and negative life events clarify the relationship between memory and stressful life events. When positive life events were included in the model, better memory was significantly associated with greater numbers of stressful life events, which indicates that positive life events are less likely to be recalled by participants with worse memory performance. In contrast, when negative life events were included, memory did not have a significant effect on PERI scores, that is, negative life events are recalled equally by all participants. Distressing events do not appear to be subject to differential recall by participants in this study. These findings support the hypothesis that the relationship between memory and stressful life events is related to the study methodology rather than indicating a true indirect relationship between memory and depression.

Intelligence was found to predict depression indirectly through two paths. Higher full-scale IQ scores were associated with greater numbers of social contacts, which were associated with fewer depressive symptoms. A second mechanism by which higher IQ scores were indirectly associated with depression was through stressful life events. Higher IQ resulted in fewer stressful life events, which were associated with greater levels of depression. This suggests that more intelligent individuals are perhaps better able to avoid the stressful events which contribute to feelings of depression.

The present results are consistent with work by Bornstein et al. (1993). These authors reported results which indicated a direct relationship between depression and IQ in HIV-positive, asymptomatic men. After controlling for the effects of education, they found that individuals reporting high levels of depression, defined as scores greater than or equal to 15 on the Beck Depression Inventory (BDI), had lower verbal IQ scores than individuals with lower BDI scores. The magnitude of the verbal IQ difference was 8.5 points, a value which could be deemed clinically significant. The indirect relationships found in the present study may translate into a direct relationship when social support and life events are not measured or accounted for and raises the possibility that it is specifically verbal, rather than nonverbal or general, intelligence which protects against depression or stressful events, or enables individuals to access more social contacts.

The finding that IQ was associated with increased numbers of social contacts, which then contributed to reduced depressive symptoms, is consistent with the initial hypotheses. This result suggests that individuals with higher levels of general cognitive ability are, in some way, better able to seek out or maintain social contacts. The exact nature of this relationship, however, is not clear. While it is possible that this relationship may be attributable to socioeconomic status, this is more likely explained by the significant, unique contribution of occupation to social contacts. In the final model, the significant contribution of IQ to social contacts is independent of the contribution of occupation to social contacts.

It is unlikely that more intelligent people have an enhanced ability to avoid all of the life events measured by the PERI. However, there may be a subset of life events which are more easily avoided by those with greater cognitive resources. The relationship between intelligence and stressful life events was further examined in the follow-up analyses of positive and negative stressors. In the model containing positive stressful life events, the effect of IQ on stress was no longer significant, while in the model containing negative stressful life events, the effect of IQ remained significant. This suggests that negative events such as problems in school, being accused of a crime, or not getting an expected raise, might, indeed, be less common in populations of more intelligent individuals. In contrast, the occurrence of positive events such as deciding to live with a partner, acquiring a pet, or taking a vacation is not likely to be related to the IQ of the participant.

The purpose of this study was to clarify the contribution of cognitive function to the relationship between social support and depression in persons with HIV infection. Neuropsychological performance was found to contribute significantly to this relationship through four indirect pathways. Two domains of neuropsychological performance, attention and executive function, were shown to indirectly attenuate depression by producing decreased illness-related dysfunction. Higher IQ scores were associated with fewer numbers of stressful life events and with greater numbers of social contacts, both of which were, in turn, related to lower levels of depression. In addition, the direct effect of social support on depression and physical distress was supported. Finally, it was confirmed that illness-related dysfunction, stress, and social contacts contributed directly to depression. These results suggest that cognitive factors play an important role in the prediction of psychosocial factors which lead to increased depression and underscore the importance of considering neuropsychological, psychosocial, and physical functioning when attempting to identify individuals at greatest risk for depression. Because the current study was intended to build a new model based on available data, it will be important to extend these findings by replicating the model in another sample.

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