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BRIEF COMMUNICATION

Neurobehavioral functioning in asymptomatic HIV-1 infected women

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Abstract

Numerous reports have assessed the neuropsychological functioning of medically asymptomatic HIV-1 infected men. However, to date there have been no published studies of the neuropsychological functioning of asymptomatic HIV-1 infected women, even though women represent the fastest-growing demographic group of HIV-1 infected individuals. In this investigation, 31 women (17 asymptomatic HIV-1 seropositive, 14 seronegative) were administered a battery of neurocognitive and neuropsychiatric instruments. Participants in both groups were matched for age, education, months since injection drug use, and substance use. Group comparisons revealed no significant differences in any of the neurocognitive or neuropsychiatric measures. The results of this preliminary study suggest that clinically significant differences in neurobehavioral function are unlikely in medically asymptomatic HIV-1 infected women compared to seronegative controls. However, additional studies are needed with larger sample sizes and with careful attention to possible confounding or masking variables. (*JINS*, 1998, *4*, 172–178.)

Keywords: Women, HIV-1, AIDS, Neuropsychological tests, Psychiatric

INTRODUCTION

Neurobehavioral functioning of men infected with human immunodeficiency virus–type 1 (HIV-1) has been well described. Most recent literature reviews indicate that subtle neurocognitive changes—most often affecting speeded information processing and fine motor function—are present in a subgroup of HIV-1 seropositive men (White et al., 1995). However, there are no published reports to date of similar studies in women, even though women comprise the fastest growing demographic group of individuals infected with HIV-1 (Karon et al., 1996).

HIV-1 seropositive women may be at heightened risk for the development of neuropsychological complications compared with other seropositive groups. HIV-1 infected women are typically less educated and receive antiretroviral therapy less frequently than seropositive males (Ickovics & Rodin, 1992), variables that appear to protect against the neurobehavioral effects of HIV-1 infection (e.g., Stern et al., 1996). Further, HIV-1 seropositive women are significantly more likely than seropositive males to be living in poverty (Ickovics & Rodin, 1992), with consequent greater economic barriers to health care.

The purpose of the present preliminary study was to describe the nature and extent of neurobehavioral dysfunction in women in the early stages of HIV-1 infection. To our knowledge, this represents the first report of a controlled study to examine the neuropsychological and psychiatric functioning of asymptomatic, HIV-1 seropositive women.

METHODS

Research Participants

Participants were 31 women, including 17 asymptomatic HIV-1 seropositive (HIV+), and 14 HIV-1 seronegative

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(HIV−) controls. All HIV+ participants met criteria for Clinical Categories A (Centers for Disease Control and Prevention, 1992) as determined by their treating physician, who provided confidential documentation to the research team. Participants were between the ages of 24 and 52 years and spoke English as their primary language. Exclusion criteria included (1) mental retardation (documented IQ < 70); (2) disorders of the central nervous system (CNS); (3) loss of consciousness greater than 30 min secondary to drug overdose; (4) significant learning disability, including known diagnosis or self-report of profound spelling deficits; (5) being perimenopausal or postmenopausal; or (6) traumatic brain injury, including closed or open head injury. For traumatic brain injury, participants were excluded if they had (1) a loss of consciousness greater than 20 min, (2) any permanent motor, sensory, or cognitive sequelae, or (3) hospitalization greater than 24 hr for neurological complications. Participants in both groups were recruited from HIV clinics, drug treatment programs, women centers, newspaper advertisements, and word-of-mouth. This study was approved by the responsible institutional review board. All participants provided informed consent prior to testing and were given small financial incentives for their participation. Financial reimbursements were also provided for transportation and childcare needs. Tables 1 and 2 detail demographic information for the sample.

As can be seen in Table 1, the HIV+ and HIV− groups were matched on age, education, months since injection drug use, and current alcohol and substance use. The mean age and education level (i.e., number of years of education) of the HIV− and HIV+ groups were consistent (± 1.5 *SD*) with the age and educational levels of HIV-1 seronegative and asymptomatic HIV-1 seropositive male samples included in previous similar studies (e.g., Martin et al., 1992; Stern et al., 1992). At the time of participation, 47% of the asymptomatic HIV+ group were taking antiretroviral drugs, including zidovudine (AZT) or dideoxyinosine (ddI) monotherapy, or as part of a clinical trial.

Instrumentation

Each neurocognitive and neuropsychiatric instrument was selected based on one or more of the following criteria: (1) it was recommended by the National Institute of Mental Health (NIMH) work group on neurobehavioral assessment in HIV (Butters et al., 1990); (2) it has previously been found to be sensitive to the neurobehavioral impairments exhibited in HIV-1 infected men in studies by our groups (Martin et al., 1992; Stern et al., 1992) and others; or (3) it is believed to be culturally unbiased, including measures suggested by the World Health Organization (WHO; Maj et al., 1993). Neurocognitive instruments included measures of attention, information processing speed–reaction time, executive functioning, fine motor speed and dexterity, visuoconstructive skills, and learning and memory. Neuropsychiatric instruments included measures of apathy, fatigue, and depression–mood. Table 3 lists all of the measures used in the study.

Statistical Analyses

Observed differences in neurocognitive and/or neuropsychiatric functioning were assessed using univariate, independent *t* tests. Overall, family-wise error rates (FW_{α}) were set to .05 for each functional domain (e.g., information processing speed–reaction time) using the Bonferroni adjustment procedure, in order to reduce the possibility of Type I error. Measures of treatment effect [omega squared (ω^2)] were calculated to determine whether clinically meaningful differences exist between the HIV− and HIV+ groups. The ω^2 index was chosen as it is a relatively unbiased measure of population treatment effect and because it is unaffected by variations in sample size (Carroll & Nordholm, 1975). Omega squared values range from 0.0 to 1.0, with scores of .01, .06, and greater than .15 representing *small*, *medium*, and *large* treatment effects, respectively. Because of the possibility that measures of central tendency may not reveal

Table 1. Age, years of education, months since injection drug use, severity of alcohol use, and severity of current substance use

	HIV-1 seronegative ($N = 14$)	Asymptomatic HIV-1 seropositive ($N = 17$)	
Variable	*M* (*SD*)	*M* (*SD*)	*t*
Age	34.7 (6.1)	35.7 (6.8)	0.43
Years of education	13.1 (1.3)	12.4 (0.9)	−1.82
Months since injection drug use	26.6 (20.8)	39.1 (36.7)	0.85
Severity of current alcohol use*	9.2 (4.8)	11.9 (7.4)	1.16
Severity of current substance use*	12.1 (7.8)	14.9 (8.9)	0.90

All group comparisons were nonsignificant ($p > .05$).
*Each based on a 7-item scale of self-reported behavior over the previous week, with a range of 7 (*least severe*) to 35 (*most severe*).

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Table 2. Sample demographics

Variable	Percent HIV-1 seronegative	Percent asymptomatic HIV-1 seropositive
Race		
White	57.1	58.8
African American	21.4	41.2
Hispanic	14.3	0.0
Other	7.1	0.0
Current work status		
Full time	14.3	17.6
Part time	0.0	11.8
Unemployed–seeking work	7.1	5.9
Unemployed–not seeking work	0.0	5.9
Receiving disability	14.3	41.2
Student	14.3	5.9
Homemaker	42.9	11.8
Other	7.1	0.0
Previous psychiatric history (based on self-report)		
Never treated	50.0	29.4
Psychotherapy only	14.3	0.0
Medication	21.4	58.8
Hospitalization (1 admission)	7.1	5.9
Hospitalization (> 1 admission)	7.1	5.9
Previous psychiatric diagnosis (based on self-report)		
None	64.3	29.4
Depression	7.1	52.9
Anxiety	0.0	5.9
Psychosis	0.0	0.0
Other	28.6	11.8
Previous drug use		
Sedative–hypnotics, anxiolytics	84.6	66.7
Cannabis	92.3	86.7
Stimulants	76.9	60.0
Opioids	69.2	80.0
Cocaine	100.0	93.3
Hallucinogens	53.8	66.7
Other	38.5	46.7
Multiple drug use (includes cases from other drug use categories)	100.0	93.3
Previous injection drug use		
No	50.0	47.1
Yes	50.0	52.9
CDC staging		
A1 (CD4 $\geq 0.5 \times 10^9$/L)	0.0	41.7
A2 (CD4 = 0.2–0.499 $\times 10^9$/L)	0.0	41.7
A3 (CD4 = 0–0.199 $\times 10^9$/L)	0.0	16.7

group differences in situations where subtle differences may exist or in a small sample study, impairment rates were also calculated. For those tests with available normative data, the percentage of each group (i.e., HIV− and HIV+) scoring in the impaired range was calculated. For the purpose of this study, an impaired score was defined as 1 standard deviation or more worse than the normative mean, or as falling below a published cutoff score (e.g., Hamilton Depression Rating Scale; Hamilton, 1960). Group differences in the frequency of impaired performance were assessed using the χ^2 statistic.

RESULTS

There were no significant differences between the HIV− and HIV+ samples on any of the measures of neurocognitive and neuropsychiatric functioning (Table 4). Furthermore, there were no significant differences in the number

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Table 3. Neurocognitive and neuropsychiatric instruments

Neurocognitive instruments
Attention
Adaptive Rate Continuous Performance Test (ARCPT; Cohen & Fisher, 1989)
Performance Assessment Battery (PAB; Thorne et al., 1985)
Letter-matching
Stroop
Time-wall
Information processing speed–reaction time
Computerized simple and choice reaction time tasks (Martin et al., 1992)
Paced Auditory Serial Addition Test (PASAT; Gronwall & Sampson, 1977)
Executive functioning
Boston Qualitative Scoring System (BQSS) for the Rey–Osterrieth Complex Figure (Stern et al., 1994; June 1996 revision); Fragmentation and Planning scores
Color Trails 1 & 2 (D'Elia & Satz, 1996)
Fine motor speed and dexterity
Grooved Pegboard (Matthews & Kløve, 1964)
Visuoconstructive skills
BQSS Presence and Accuracy score
Learning and memory
BQSS immediate and delayed retention scores
California Verbal Learning Test (CVLT; Delis et al., 1987)
Neuropsychiatric instruments
Apathy
Apathy Evaluation Scale (Marin et al., 1991)
Fatigue
Fatigue Assessment Scale (Cohen & Fisher, 1989)
Daily Fatigue Diary (Cohen & Fisher, 1989)
Depression–mood state
Center for Epidemiological Studies–Depression Scale (CES–D; Radloff, 1977)
Hamilton Depression Rating Scale (HDRS; Hamilton, 1960)
Visual Analog Mood Scales (VAMS; Stern, 1997)

of impaired individuals in each group, though for several variables the percentage of participants in *both* groups scoring in the impaired range was high. Treatment effect estimates suggest that observed mean group differences in neurocognitive and neuropsychiatric functioning may be of little clinical importance. Omega-squared values were quite small, ranging from .01 to .03 for all neurocognitive and neuropsychiatric functional domains.

DISCUSSION

In this preliminary investigation, we found no differences between medically asymptomatic HIV-1 infected women and cohort-matched seronegative controls in either neuropsychological performance or neuropsychiatric symptoms. These results contrast with previous reports in which asymptomatic HIV-1 infected men were studied. Our negative findings may possibly be due to methodological limitations in the present study or may represent sex differences in the manifestation of HIV-1 in the central nervous system.

Inadequate statistical power may have played a role in the absence of detectable differences in neurobehavioral functioning between our HIV− and HIV+ groups. *Post-hoc* power analyses indicated that the current sample size would have been sufficient to detect clinically meaningful group differences (e.g., at $\omega^2 = .15$) but was too small to detect subtle differences in neurobehavioral functioning. However, differences in neuropsychological performance have been demonstrated in studies of gay men with sample sizes comparable to ours (White et al., 1995) which further suggests that sample size alone is an insufficient explanation for the current negative results.

With regard to detection of *subclinical* deficits, it is possible that a variety of confounding variables masked any underlying HIV-related group differences. For example, it should be noted that over one-third of the HIV-1 seropositive group in the present study were taking AZT, an antiretroviral medication that has been shown to improve some aspects of neurocognitive performance in HIV-1 infected men. In addition, a larger proportion of the HIV+ group reported having some form of psychiatric history and treatment than the HIV− group, though both groups reported similarly high levels of previous substance use. These demographic findings are generally consistent with large natural history studies of HIV-1 infection in women, such as the Women's Interagency HIV Study (WIHS), which have documented high rates of psychopathology, substance abuse, and histories of domestic violence and childhood abuse in both HIV-1 seropositive and high-risk seronegative women (Deamant et al., 1996). Given these potential confounds, detecting subtle differences between these groups in current neuropsychological function is likely to be extremely difficult. In fact, we found that, for several variables, there was a high percentage of participants in *both* groups who exhibited impaired performance.

Studies of HIV-1 seropositive injection drug users have shown that neuropsychological findings from the literature on gay men cannot always be generalized to substance abusers. For example, substance abusers are impaired regardless of serostatus when tested with speeded psychomotor tasks (e.g., Martin et al., 1995b), measures that detect HIV-related mental slowing reliably in homosexual and bisexual men (e.g., van Gorp et al., 1989). In other words, it is likely that no single neuropsychological profile is common to different HIV-1 infected populations, and studies of asymptomatic HIV-1 infected women might benefit from emphasis on assessment procedures with demonstrated sensitivity to HIV-related cognitive deficits in substance abusers, such as measures of working memory (Martin et al., 1995b).

The present study also found no significant differences between the HIV− and HIV+ groups on any of the neuropsychiatric instruments, including measures of depression, apathy, fatigue, and internal mood state. Furthermore, both groups had a large number of individuals who would be considered impaired on some measures of depression symp-

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Table 4. Group differences on tests of neurocognitive and neuropsychiatric functioning

Measure	HIV-1 seronegative ($N = 14$)		Asymptomatic HIV-1 seropositive ($N = 17$)				
	M (*SD*)	% Impaired	*M* (*SD*)	% Impaired	*t*	ω^2	χ^2
Attention						**.00**	
Continuous Performance Test							
Hits (total)	49.3 (4.1)	n/a	48.6 (4.3)	n/a	−0.44	.00	n/a
Performance Assessment Battery							
Letter							
Errors	0.6 (1.3)	n/a	0.3 (0.5)	n/a	−0.75	.00	n/a
Stroop							
Errors—incongruent stimuli	2.8 (5.7)	n/a	2.4 (2.9)	n/a	−0.25	.00	n/a
Errors—congruent stimuli	0.3 (0.5)	n/a	0.4 (0.8)	n/a	0.28	.00	n/a
Time Wall							
Estimated reaction time	9.5 (1.0)	n/a	9.2 (0.7)	n/a	−1.00	.00	n/a
Information processing speed–reaction time						**.02**	
Paced Auditory Serial Addition Test							
Trial B	33.3 (7.7)	42.9	28.6 (7.3)	37.5	−1.65	.06	0.09
Computerized reaction time							
Simple reaction time	318.0 (68.4)	n/a	316.0 (68.8)	n/a	−0.08	.00	n/a
Choice reaction time	453.3 (74.1)	n/a	499.2 (105.4)	n/a	1.36	.03	n/a
Executive functioning						**.02**	
Color Trails 1 & 2							
Time on Trial 1	27.1 (12.2)	7.1	30.8 (10.5)	5.9	0.90	.00	0.02
Time on Trial 2	65.3 (21.5)	0.0	70.4 (20.8)	11.8	0.67	.00	1.76
BQSS							
Copy planning score	2.6 (1.0)	35.7	2.4 (0.7)	52.9	−0.71	.00	0.92
Copy fragmentation score	2.5 (1.0)	14.3	1.8 (1.1)	41.2	−1.73	.06	2.70
Fine motor speed and dexterity						**.00**	
Grooved Pegboard							
Nondominant hand time	81.6 (18.5)	16.7	81.7 (14.3)	41.2	0.02	.00	1.97
Dominant hand time	64.5 (8.1)	46.2	68.1 (9.7)	41.2	1.09	.01	0.07
Visuoconstructive skills						**.00**	
BQSS							
Copy presence and accuracy	15.9 (2.2)	28.6	15.8 (1.9)	29.4	−0.14	.00	0.00
Learning and memory						**.03**	
California Verbal Learning Test							
Trials 1–5 total	55.6 (8.6)	64.3	58.5 (8.1)	35.3	0.99	.00	2.58
Short delay free recall	11.3 (3.5)	57.1	12.5 (2.4)	41.2	1.17	.01	0.78
Long delay free recall	11.6 (3.3)	71.4	12.9 (2.6)	58.8	1.19	.01	0.53
Discriminability	97.3 (4.7)	7.1	96.4 (4.2)	17.6	−0.58	.00	0.75
BQSS							
Immediate retention	−35.8 (14.5)	21.4	−40.6 (13.3)	23.5	−0.96	.00	0.02
Delay retention score	−0.1 (14.2)	7.1	3.8 (23.5)	23.5	0.55	.00	1.52
Apathy						**.00**	
Apathy Evaluation Scale	56.4 (6.8)	n/a	56.4 (10.1)	n/a	−0.02	.00	n/a
Fatigue						**.03**	
Fatigue Assessment Inventory	57.4 (33.2)	n/a	65.6 (42.5)	n/a	0.55	.00	n/a
Daily Fatigue Diary	32.4 (3.4)	n/a	29.0 (7.0)	n/a	−1.57	.06	n/a
Depression–mood state						**.00**	
Hamilton Depression Rating Scale	14.4 (9.6)	35.7	16.8 (8.8)	64.7	0.74	.00	2.58
CES–D	21.6 (11.5)	57.1	24.1 (14.2)	64.3	0.51	.00	0.15
Visual Analog Mood Scales							
Afraid	17.6 (20.5)	21.4	18.1 (20.4)	23.5	0.06	.00	0.02
Confused	39.5 (29.4)	50.0	33.5 (30.8)	52.9	−0.55	.00	0.03
Tired	52.1 (29.7)	28.6	57.9 (35.1)	41.2	0.49	.00	0.53
Sad	21.3 (26.4)	14.3	36.8 (36.3)	41.2	1.33	.02	2.70

All *t* tests and χ^2 were nonsignificant ($p > .05$, two tailed with a Bonferroni Adjustment). Bold ω^2 values represent the average treatment effect for each functional domain. n/a = not applicable; percent impaired calculated only for tests with available normative data.

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tomatology. These findings are similar to many similar reports of asymptomatic HIV-1 infected gay men. For example, controlled studies have shown that the prevalence of major depression and other mood disorders is higher in asymptomatic HIV-1 infected gay men compared to the prevalence in the general population, but is similar to the prevalence in HIV-1 seronegative gay men (e.g., Perkins et al., 1994). In spite of these similarities, findings from psychiatric studies of HIV-1 seropositive men may have limited generalizability to the female HIV-1 infected population. Initial studies of psychiatric status and psychosocial stressors in HIV-1 seropositive women have shown significant gender differences for HIV-1 seropositive persons (Brown & Rundell, 1993; Semple et al., 1993). For example, a recent study reported that depression was significantly more prevalent in seropositive compared to seronegative male injection drug users (IDUs; 33 *vs*. 16%) but almost equally prevalent in female IDUs, regardless of serostatus (26 *vs*. 30%; Lipsitz et al., 1995). Another recent study reported that seropositive men's self-reported somatic symptoms of depression correlated significantly with self-reported psychological distress; however, HIV-1 seropositive women's self-reported somatic symptoms correlated significantly and inversely with CD4 lymphocyte counts but *not* with psychological distress (Martin et al., 1995a).

It is possible that the results of this preliminary study provide evidence of an actual gender difference in the early effects of HIV-1 infection on the CNS. Although this conclusion is premature due to the small sample size in this study and due to the inconsistent findings in studies of HIV-1 infected men, previous investigations have indicated other gender differences in the manifestation of HIV-1 and AIDS. For example, Phillips et al. (1994) reported that HIV-1 infected women were at increased risk for toxoplasmosis and herpes simplex viral ulceration. Similarly, Melnick et al. (1994) found an increased incidence of bacterial pneumonia in women compared with men, even after controlling for injection drug use. If the findings of the present study are replicated with larger samples and with direct comparisons between men and women, then future research examining potential underlying mechanisms would be warranted. For example, the relationship between gender-related differences in CNS endocrine milieu and the differential effects on viral load and HIV-related neuropathogenicity could be investigated. Other, gender-related psychoneuroimmunological phenomena could also be studied.

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