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HIV-1, cocaine, and neuropsychological performance in African American men

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Abstract

The purpose of this study was to examine the independent and interactive effects of HIV-1 serostatus and cocaine on neuropsychological (NP) performance in a sample of 237 gay and bisexual urban-dwelling African American men. Consistent with current evidence, it was expected that the greatest neuropsychological performance deficits would be evident (1) in the symptomatic seropositives (SSPs), especially in domains affected by HIV (i.e., memory and psychomotor speed), and on tests that are sensitive to subtle slowing; (2) in those who are recent and frequent cocaine abusers; and (3) in those who are both HIV seropositive and cocaine abusers. Multivariate analyses controlling for age and alcohol use confirmed expectations, with symptomatic seropositives (SSPs) evidencing significantly poorer psychomotor speed than the seronegatives (SNs), and slower reaction time and poorer nonverbal memory than the asymptomatic seropositives (ASPs). Moderate to heavy recent cocaine use was associated with slower psychomotor speed. However, contrary to expectations, no interaction of serostatus and cocaine was noted for any NP domain, and the expected serostatus and cocaine effects on verbal memory and frontal systems were not obtained. Level of alcohol consumption exacerbated the detrimental effects of HIV-1 on a computerized reaction time test which is especially sensitive to subtle slowing. This study provides one of the first descriptions of the neuropsychological effects of HIV–AIDS in a noninjection drug-using community sample of gay and bisexual African American men. (*JINS*, 2000, *6*, 322–335.)

Keywords: HIV infection, Cocaine abuse, Neuropsychological performance, African American men

INTRODUCTION

The direct central nervous system involvement and effects of human immunodeficiency virus (HIV) have been documented by numerous sources (Brew et al., 1988; Ho et al., 1985; Levy et al., 1985; Navia et al., 1986). A progressive decline in neuropsychological functioning is observed in a large proportion of individuals, with 20 to 30% of patients with AIDS developing dementia before death (McArthur, 1994). The picture of neuropsychological decline in individuals with HIV is characterized by slowed performance on speeded tests, particularly tests with a psychomotor or motor component, attentional deficits, and memory impairment (Grant et al., 1987; Grant & Heaton, 1990; Heaton et al., 1995; Miller et al., 1990; van Gorp et al., 1993). Language, visuospatial, and global cognitive functioning are typically intact in most individuals evidencing early signs of HIV-associated CNS dysfunction, and substantial deficits in these domains are seen only in late stage illness (van Gorp et al., 1993).

In general, studies examining more severely immunocompromised individuals, particularly those with AIDS, have found greater neuropsychological impairment in those with

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advanced disease compared to seronegative controls. Mild, clinically nonsignificant neurobehavioral declines are generally observed in infected persons in the early stages of the disease (Goethe et al., 1989; Janssen et al., 1989; McArthur et al., 1989; Miller et al., 1990; Miller et al., 1991; van Gorp et al., 1989), unless other co-factors such as low education, substance use, and other premorbid CNS risk factors are also present (Satz et al., 1993).

Cocaine Use and the Central Nervous System

Habitual and frequent cocaine use is implicated in CNS disturbances including cerebrovascular accidents and seizures (Levine et al., 1987; Pascual-Leone et al., 1991a, Volkow et al., 1991) and cerebral atrophy (Pascual-Leone et al., 1991b), with the most striking effects noted in frontotemporal regions, and basal ganglia (Holman et al., 1991; Pascual-Leone et al., 1991a; Strickland et al., 1993; Tumeh et al., 1990). In recently abstinent individuals with a minimum of 6 months of regular cocaine use (abstinence ranging from 14–35 days), deficits in verbal memory, attention and concentration, abstraction and problem solving, psychomotor speed, and information processing and reaction time have been described (Ardila et al., 1991; Beatty et al., 1995; Berry et al., 1993; O'Malley et al., 1988, 1992). Less consistent results were obtained on tests assessing visuospatial abilities and language.

The current literature suggests that cocaine use may have to exceed a certain threshold before any measurable NP impairment is observed (Ardila et al., 1991; Beatty et al., 1995). However, because of differences in time and dose effects on the brain and patterns of use, this threshold would be expected to be lower for crack cocaine than for powder cocaine (Wilkinson et al., 1980).

Overall, the literature on substance use and HIV is largely limited to injection drug users, typically opiate users and methadone maintenance patients. These findings may not be generalizable to studies examining HIV positive cocaine abusers, since cocaine has been shown to be more neurotoxic than opiates, particularly heroin (Hartman, 1995). Additionally, regional patterns of drug use, with West Coast users largely using cocaine, suggest that the existing literature may not satisfactorily address the issue of drug use, HIV, and NP performance for populations in certain regions. This study, with its large proportion of cocaine users, was designed to explore the impact of HIV and cocaine use on NP performance.

The current study is also of considerable import to the field because of its focus on African American men. To date, although a large literature examining the neuropsychological sequelae of HIV has developed, inadequate attention has been given to ethnically diverse samples in the United States, despite the growing overrepresentation of ethnic minorities among those infected by HIV/AIDS in the U.S. For example, over half of AIDS cases among men reported to the Centers for Disease Control (CDC) in 1998 were members of racial–ethnic minority groups (CDC, 1998). African American men constitute the ethnic minority group most affected by the epidemic in recent years, accounting for 33% of the AIDS cases through 1998, and the rate of AIDS among African American men (125.2/100,000) far surpasses all other groups (CDC, 1998). Further, racial differences in disease progression and severity have been documented, with greater severity of AIDS as indexed by CD4, CD4/CD8 ratio, and incidence of certain opportunistic infections among African Americans compared to Whites (Cruse et al., 1989). Given that most of the work done to date has been conducted with affluent and educated gay White males, such racial differences on immunologic dimensions necessitate independent investigation of the neuropsychology of HIV/AIDS in African American men.

Therefore, the present study provides an opportunity to extend the investigation of the neurobehavioral sequelae of HIV to the population of greatest risk, African American men who have sex with men, as well as to investigate whether HIV and cocaine use produce independent neurocognitive effects or whether these agents interact to produce greater NP deficits than they produce by acting independently. Specifically, the proposed study addresses three questions: (1) Is there an effect of HIV serostatus on neuropsychological performance in African American gay and bisexual men similar to that observed in previous studies of gay White males? (2) Is heavier and/or more frequent cocaine use associated with more marked NP deficits than is lighter and/or more infrequent cocaine use? (3) Do HIV serostatus and cocaine use interact to yield greater NP deficits than both acting alone?

Consistent with current evidence, it was expected (1) that symptomatic seropositives (SSPs) would evidence poorer NP performance than seronegatives (SNs) and asymptomatic seropositives (ASPs), especially in domains affected by HIV (i.e., memory, psychomotor speed, and frontal systems), and on tests that are sensitive to subtle slowing; (2) that those who were currently using cocaine as indexed by both pattern of use and toxicology results would perform more poorly than those with no current or past use; and (3) that HIV status and cocaine use would interact to yield poorer NP performance, such that the difference in NP performance between cocaine users and nonusers will be greater in the seropositives than in the seronegatives.

METHODS

Research Participants

U.S.-born African American men between 18 and 50 years of age were recruited from Los Angeles County to participate in the African American Health Project (AAHP; Myers et al., 1997). The AAHP was a NIDA-funded, multidisciplinary, cross-sectional study that examined medical, psychiatric, neurological, neuropsychological, and psychosocial aspects of HIV–AIDS and substance abuse in a community

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sample of English speaking African American men, and all testing was conducted in English. All men in the sample described their race–ethnicity as "Black." In order to minimize potential confounding, men with low educational attainment or functional illiteracy (8th grade education or less and/or impaired reading or comprehension), with non-HIV-related physical or sensory difficulties, or with acute medical, neurological or psychiatric conditions that might interfere with their ability to tolerate the intensive evaluation were excluded. In order to ensure a sample that was generally representative of the community of interest, men were not excluded if they had a history of head injury or loss of consciousness. In order to encourage participation, all participants were compensated for their time and provided with transportation and refreshments. No participants were tested if they appeared to be acutely intoxicated. Participant recruitment is described at length in Myers et al. (1997).

For purposes of this study, the hypotheses were tested on the subsample of 237 behaviorally identified gay and bisexual men for whom complete data on the variables of interest were available. As shown in Table 1, this sample included 75 HIV seronegatives (SN), 67 asymptomatic seropositives, and 95 symptomatic seropositives. Sero–symptom status was categorized on the following basis: (1) HIV-positive participants with no history of opportunistic illness and no history of constitutional symptoms of 2 weeks duration or longer were categorized as *asymptomatic*; (2) HIV-positive participants with a history of opportunistic illness or a history of constitutional symptoms of 2 weeks duration or longer were categorized as *symptomatic*; (3) diagnosis of AIDS on the basis of CD4 count less than 200 in the absence of opportunistic illness OR constitutional symptoms of 2 weeks duration or longer did not result in participants being categorized as symptomatic. Thus, while the categorization of participants was based in part on CDC (1993) criteria, the grouping of symptomatic participants was comprised solely of participants with histories of opportunistic illness or constitutional symptoms. Participants were relatively young ($M = 34.8$ years), moderately well educated ($M = 13.7$ years of education), and with a median annual income of $15,711. However, only 29.7% were employed full or part time. Tests for group differences indicated that the serostatus groups did not differ on age or education, but differed on alcohol use (M number of drinks/week; one-way ANOVA, $F(3, 233) = 3.6$, $p = .03$) and employment status ($\chi^2(2) = 22.6$, $p = .00002$). The SNs and SSPs consumed more alcohol on average than the ASPs, and a greater percentage of SNs were employed (51%) compared to both ASPs and SSPs [25% and 16% respectively; $\chi^2(2) = 22.6$; $p < .001$]. The high rates of unemployment among the SNs is in part attributable to a recessionary economy in Southern California, which was particularly pronounced within minority communities such as South Los Angeles and Compton.

The sample was also stratified by cocaine use using two different categorizations: (1) pattern of use, and (2) recency of use. Pattern of use was grouped as follows: (1) cocaine naïve (no reported lifetime use); (2) past user only (more than 12 months ago); (3) infrequent current user (less than 1/week); and (4) frequent current user (more than 1/week). Recency of use was grouped as follows: (1) cocaine naïve (those with no reported lifetime cocaine use); (2) those with past use only (more than 12 months ago); (3) those who currently use cocaine but were toxicology negative; and (4) those who currently use cocaine and were toxicology positive. Participants were distributed equally across all levels of serostatus and cocaine use (Table 2).

More detailed information on pattern and frequency of cocaine use was ascertained on a smaller subset of the sample. Only a small percentage of the sample endorsed intravenous use of crack or cocaine; thus these figures represent only a fraction of the sample (less than 10%). Among those who endorsed using crack or powder cocaine less than once per week during the past year, 85% reported never injecting crack or cocaine. Eight percent reported injecting cocaine once during the past year, and 8% reported injecting cocaine once per month during the past year. Among those who reported using crack or cocaine once per week or more

Table 1. Gay–bisexual sample characteristics by serostatus

Variable	Whole sample ($N = 237$)	SN ($N = 75$)	ASP ($N = 67$)	SSP ($N = 95$)
Mean age (*SD*)	34.83 (6.8)	33.88 (7.7)	34.22 (6.8)	36.01 (6.0)
Mean education (years) (*SD*)	13.66 (2.2)	14.07 (2.3)	13.70 (1.8)	13.32 (2.4)
Alcohol use (*SD*)	13.4 (23.2)	17.7 (25.8)	7.6 (11.1)	14.2 (26.6)**
CES–D score (*M, SD*)	17.9 (11.4)	16.3 (10.5)	17.3 (10.5)	19.7 (12.4)
Mean SCL score (*M, SD*)	65.1 (40.2)	59.5 (34.7)	76.1 (45.8)	67.8 (41.6)*
Estimated *Mdn* Ann. Income	15711.00	19831.00	14077.00	13575.00
Employment (% employed)	29.7%	50.6%	25%	16.2%***
Hx of neurological trauma (% with hx)	62.3%	46.5%	63.4%	58.2%
Hx of other substance use, past 12 months (% with hx)	12%	10%	10%	11%

*$p < .05$.
**$p < .01$.
***$p < .001$.

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Table 2. Stratification of sample by serostatus and cocaine use

HIV serostatus × recent cocaine use								
	SN ($N = 75$)		ASP ($N = 67$)		SSP ($N = 95$)		Total sample ($N = 237$)	
Cocaine use	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)
Naïve (no lifetime use)	27	(36.0%)	17	(25.4%)	24	(25.3%)	68	(28.7%)
Past user only (>12 months ago)	17	(22.7%)	20	(30.0%)	27	(28.4%)	64	(27.0%)
Current user–toxicology negative	19	(25.3%)	19	(28.3%)	20	(21.0%)	58	(24.5%)
Current user–toxicology positive	12	(16.0%)	11	(16.3%)	24	(25.3%)	47	(19.8%)
Totals	75	(100%)	67	(100%)	95	(100%)	237	(100%)
HIV serostatus × pattern of cocaine use (*N* and percent)								
	SN		ASP		SSP		Total sample	
Cocaine use	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)
Naïve (no lifetime use)	27	(36.0%)	17	(25.4%)	24	(25.2%)	68	(28.7%)
Past user only (>12 months ago)	17	(22.7%)	20	(29.9%)	27	(28.4%)	64	(27.0%)
Current user–> 1/week	13	(17.3%)	20	(29.9%)	26	(27.4%)	59	(24.9%)
Current user–< 1/week	18	(24.0%)	10	(14.8%)	18	(19.0%)	46	(19.4%)
Totals	75	(100%)	67	(100%)	95	(100%)	237	(100%)

often during the past year, over 85% reported never injecting crack or cocaine, 8% reported injecting cocaine less than once per week, 3% reported injecting crack cocaine once per week, and 8% reported injecting cocaine 2 to 3 times per week. Again, because of the very small sample for which such data is available, these percentages often represent only 1 or 2 participants.

A far larger percentage of crack and cocaine users in this sample endorsed snorting or smoking cocaine or crack. Data for cocaine use is available for 37 participants. Among those who endorsed cocaine use of less than once per week 81% reported using powder cocaine once per month or less, while 19% endorsed using cocaine less than once per week but greater than once per month. Among those who endorsed cocaine use of greater than once per week, 57% reported use of powder cocaine of once per month or less frequently, 19% reported using cocaine less than once per week, 13% reported using once per week, and 11% reported using twice a week or more often.

Data for those who endorsed smoking crack is available for 101 participants. Among those who endorsed crack–cocaine use of less than once per week, 68% reported using crack cocaine once per month or less, while 32% reported using crack cocaine less than once per week but greater than once per month. Among those who endorsed crack–cocaine use greater than once per week, 16% reported using crack cocaine once per week, 48% reported using crack 2 to 6 times per week, 12% reported using once per day, and 24% reported crack use of once per day or more often. Unfortunately, because this data was available on a small percentage of participants, this information provides only a suggestion at the drug use patterns of this sample.

One-way ANOVA and chi-square analyses revealed differences among the cocaine groups on education ($F(3, 233) = 9.3$, $p = .002$), alcohol use ($F(3, 233) = 15.6$, $p = .0001$), annual income ($F(3, 233) = 10.9$, $p = .0001$), and employment status [$\chi^2(2) = 24.4$, $p = .00002$]. As expected, current and more frequent cocaine users reported less education, lower income, were less likely to be employed, and consumed more alcohol than nonusers or past users.

Measures and Procedures

All participants completed comprehensive assessments conducted by a team of trained interviewers and examiners, most of whom were African American. The assessment battery included demographic information, medical history, measures of psychiatric history and status, neurologic and neuropsychological examinations, detailed description of past and current sexual practices, and measures of a range of psychosocial factors that could mediate the effects of substance use and HIV–AIDS. Blood and urine samples were obtained and tested for comprehensive serology, HIV, and toxicology testing by technicians who were blind to serostatus and substance use histories. Specifically, qualitative urine toxicology (Roche Abuscreen) was used in conjunction with standard laboratory toxicology to test for the presence of amphetamines, barbiturates, cannabis, cocaine, opiates, and phencyclidine (PCP). In addition, a detailed structured interview to assess duration, recency, and chronicity of use of each of these substances was conducted (see Richardson et al., 1997, for a detailed description of these assessment measures).

Reliance on self-reports of drug use is often criticized, and there is general agreement that reliability diminishes the greater the time difference between assessment and use (i.e., reports of recent use are more reliable than reports of use in the past year or beyond; Carroll, 1995). However,

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reliability is improved when the assessment is conducted as a structured interview by trained interviewers, when there is little secondary gain, and when the self-reports are verified by collateral reports or laboratory testing (Carroll, 1995). Examination of self-reported cocaine use and toxicology data in this sample revealed that only 10% (22 participants) of the sample evidenced a discrepancy between self-reported cocaine use and toxicology results (e.g., self-report of no cocaine use in the last 12 months while obtaining a positive toxicology result).

HIV serostatus was determined by enzyme-linked immunoabsorbent assay (ELISA) and confirmed by Western Blot. A serology battery was also run to assess immune function as indexed by CD4 and CD8 count and CD4/CD8 ratio.

Neuropsychological assessment was conducted using the UCLA–WHO Neuropsychology Battery (Maj et al., 1993), which is a comprehensive battery comprised of measures included in the World Health Organization's cross cultural multicenter studies of HIV-associated neuropsychiatric disorders and supplemented by traditional clinical NP tests that are widely used in the United States. The UCLA–WHO battery assesses multiple NP domains including attention, concentration, visuoconstructive abilities, psychomotor and motor speed, verbal and nonverbal memory, verbal fluency, and intellectual functioning. This battery was also supplemented with computerized measures of reaction time. Several of the domains assessed by this battery have been found to be preferentially affected by HIV infection and cocaine use. The specific measures included (1) Color Trails 1 and 2 (D'Elia et al., 1994), (2) Color Figure Mazes 1–2–3 (D'Elia & Satz, 1989), (3) the Block Design and Digit Symbol subtests of the Escala de Inteligencia Wechsler para Adultos (EIWA; Wechsler et al., 1968), (4) Grooved Pegboard Test (Matthews & Kløve, 1964), (5) Trail Making Test Parts A and B (Reitan & Wolfson, 1985), (6) the WHO–UCLA Auditory Verbal Learning Test (Satz et al., 1993), (7) the WHO–UCLA Picture Memory and Interference Test (Satz & Chervinsky, 1993), (8) Verbal Fluency–Names & Animals (Benton & Hamsher, 1977), and (9) the California Computerized Assessment Package (CalCAP; Miller, 1991), which provides measures of simple, choice, and sequential reaction times.

For purposes of analyses, tests were grouped by functional domain, and groupings were confirmed empirically using principal components analysis with oblique rotation. Oblique rotation was employed because NP test scores are correlated. This analysis identified seven stable, reliable, and interpretable NP domains. As shown in Table 3, the NP domains identified were (1) Verbal Memory, (2) Psychomotor Speed I (traditional measures of psychomotor speed such as the Trail Making Test); (3) Psychomotor Speed II (psychomotor speed measures that capture higher order abilities such as perceptual organization and planning), (4) Reaction Time, (5) Nonverbal Memory, (6) Motor Speed, and (7) Verbal Fluency. All NP test scores were tested for skewness, kurtosis, and normality, and no transformations were judged to be necessary.

Data Analyses

To control for other factors that could confound the effects of serostatus on NP performance, age (in years), education (in years), history of neurologic trauma, depressed mood (sum CES-D score; Radloff, 1977), general psychological distress (sum score on all except the depression items of the SCL–90–R; Derogatis et al., 1977), alcohol use (number of standard drinks consumed per week); and other substance use (indexed as frequency–history of and toxicology for amphetamine, barbiturate, opiate, and PCP use) were identified and tested for inclusion as covariates. Other substance use was treated as a 2-level variable indexing whether or not the respondent used any other substances during the past year. Data for all variables are presented on Table 1.

A series of ANOVA, chi-square, and correlational analyses were conducted to determine appropriate inclusion of covariates in the MANCOVA analyses used to test the hypotheses. Results indicated that with the exception of age, no other covariate was found to be consistently correlated with the dependent variables. In addition and contrary to expectations, age was not associated with measures of reaction time or nonverbal memory, possibly because all participants were between ages 18 and 50 years. Alcohol use was associated with two of the five measures of the Psychomotor Speed I factor. Thus, age and alcohol were entered as covariates in relevant analyses.

A series of 3×4 MANCOVAs, with three levels of serostatus and four levels of cocaine use, were run to test for the hypothesized differences on test scores in each of the NP domains identified. Two series of analyses were conducted: one examining Serostatus $\times$ Pattern of Cocaine Use and one examining Serostatus $\times$ Recency of Cocaine Use. When significant multivariate findings are obtained, univariate tests were examined. Neuropsychological test scores were treated as dependent variables, with raw scores for tests comprising an NP domain employed as dependent variables (seven test groupings in total), and all scores were treated as continuous variables. When a case was missing data for any of the tests in a given factor, the case was omitted from that particular analysis (cell sizes are provided in Table 2).

RESULTS

HIV Serostatus Effects

Mean test scores are presented in Tables 4 and 5. As shown in Table 6, MANCOVAs testing for differences as a function of serostatus and controlling for age and alcohol identified significant serostatus differences on (1) traditional measures of psychomotor speed [Psychomotor Speed I; ($F(2,218) = 2.14$, $p = .02$], with this effect evident on Trail Making Test Part B [univariate $F(2,218) = 5.36$, $p = .005$]; (2) measures of reaction time [$F(2,214) = 2.26$, $p = .04$], which was most evident on Sequential Reaction Time I [univariate $F(2,214) = 4.63$, $p = .01$] and Sequential Reaction Time II [univariate $F(2,214) = 5.48$, $p = .005$]; and (3) non-

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Table 3. Factor analysis of neuropsychological test variables

	Factors						
Measure–domain	1	2	3	4	5	6	7
Auditory Verbal Learning Test (Verbal Memory)							
Total Trials 1–5	.91						
Immediate Recall	.87						
Trial 5	.82						
Delayed Recall	.85						
Delayed Recognition	.61						
Trial 1	.59						
Mazes/Simple Reaction Time–Block Design (Psychomotor Speed II)							
Color Figure Maze 3		.81					
Color Figure Maze 2		.80					
EIWA Block Design		.68					
Color Figure Maze 1		.76					
Simple Reaction Time		.55					
Digit Symbol–Trail Making (Psychomotor Speed I)							
Color Trails 1			.82				
Color Trails 2			.80				
Trails A			.63				
Trails B			.67				
EIWA Digit Symbol			.64				
Choice–Sequential Reaction Time							
Median Sequential Reaction Time 1				.85			
Median Choice Reaction Time				.81			
Median Sequential Reaction Time 2				.73			
Picture Memory Interference (Nonverbal Memory)							
Book C Recognition					.80		
Book B Recognition					.74		
Book A Recognition					.65		
Delayed Recall Book A–C					.56		
Grooved Pegboard (Motor Speed)							
Dominant Hand						.92	
Nondominant Hand						.91	
Verbal Fluency							
Animals							.75
Names							.69

verbal memory ($F(2,223) = 2.49$, $p = .04$], with differences most evident on Picture Memory Interference Test Book C [univariate $F(2,223) = 4.63$, $p = .01$], and Book D [univariate $F(2,223) = 6.35$, $p = .002$]. A significant main effect for cocaine use on traditional measures of psychomotor speed (Psychomotor Speed I) [$F(3,218) = 1.79$, $p = .03$] was also obtained.

Specific contrasts between serostatus groups probing the significant serostatus effects on traditional measures of psychomotor speed (Psychomotor Speed I) using 2-way MANCOVAs revealed that SSPs evidenced significantly slower performance on the Psychomotor Speed I measures than the SNs [$F(1,155) = 3.45$, $p = .006$]. These differences were specific to Trail Making Test Part B [univariate $F(1,155) = 10.59$, $p = .001$]. No significant differences between SNs and ASPs or between ASPs and SSPs, either overall or specific to Trails B were obtained.

Planned comparisons probing the significant serostatus effect on reaction time indicated that the SSPs also evidenced slower reaction times than the ASPs [$F(1,145) = 3.85$, $p = .01$] on both sequential reaction time measures; Sequential Reaction Time I [$F(1,145) = 6.96$, $p = .009$] and Sequential Reaction Time II [$F(1,145) = 9.83$, $p = .002$]. No differences between SNs and either ASPs or SSPs emerged on either of these measures.

Planned comparisons probing the significant serostatus effects on nonverbal memory using 2-way MANCOVAs revealed that ASPs had significantly higher scores on the nonverbal memory tests than SSPs [$F(1,152) = 4.16$, $p = .003$], with univariate effects once again noted on Picture Memory Interference Test Books C [univariate $F(1,152) = 8.68$, $p = .004$] and D [univariate $F(1,152) = 12.14$, $p = .001$]. No significant differences on nonverbal memory between the SNs and ASPs or between the SNs and the SSPs were observed.

Post-hoc analyses testing for differences in characteristics that might impact NP performance including age, education, alcohol use, and depression were conducted com-

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Table 4. Test scores (unadjusted) by serostatus groups

	Total sample		SN		ASP		SSP	
Test	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)
Verbal Memory (AVLT)								
Total Words	47.4	(8.7)	49.6	(8.0)	47.3	(8.3)	45.7	(9.1)
Immediate Recall	9.7	(2.7)	10.2	(2.6)	9.7	(2.7)	9.3	(2.6)
Delayed Recall	9.4	(2.7)	10.0	(2.7)	9.3	(2.8)	9.0	(2.6)
Delayed Recognition	14.0	(1.4)	14.3	(1.1)	14.1	(1.1)	13.8	(1.8)
Psychomotor Speed II								
CFM1 (s)	54.3	(21.5)	49.8	(16.7)	52.1	(18.8)	59.4	(25.6)
CFM2 (s)	114.3	(47.7)	107.2	(47.7)	111.0	(43.5)	122.4	(50.0)
CFM3 (s)	135.4	(48.3)	129.1	(47.0)	127.6	(45.2)	146.7	(50.1)
Block Design (raw score)	33.9	(8.5)	34.8	(7.9)	33.6	(9.7)	33.3	(8.1)
Simple RT (ms)	436.2	(240.5)	420.3	(226.9)	411.7	(206.3)	466.2	(271.0)
Psychomotor Speed I								
Color Trails 1 (s)	38.4	(18.6)	35.8	(14.9)	35.8	(17.1)	42.4	(21.5)
Color Trails 2 (s)	85.8	(32.9)	83.2	(27.0)	78.1	(29.8)	93.3	(37.8)
Trails A (s)	28.9	(14.1)	28.6	(15.3)	26.5	(9.0)	30.8	(15.9)
Trails B (s)	78.5	(42.5)	67.2	(33.9)	74.9	(40.8)	90.5	(47.3)
Digit Symbol (raw score)	60.5	(14.5)	63.3	(14.1)	61.5	(13.2)	57.6	(15.3)
Reaction Time								
Seq. RT I	557.3	(133.0)	535.6	(121.7)	539.6	(118.3)	587.2	(146.5)
Seq RT II	647.8	(184.6)	621.9	(182.8)	609.3	(169.0)	695.9	(187.8)
Choice RT	399.9	(65.4)	393.9	(52.2)	389.1	(64.8)	412.3	(73.5)
Nonverbal Memory								
Pic. Mem. A	18.1	(2.2)	18.0	(2.6)	18.6	(1.7)	17.9	(2.1)
Pic. Mem. B	14.9	(3.3)	14.7	(3.3)	15.8	(2.8)	14.6	(3.7)
Pic. Mem. C	15.3	(3.6)	15.4	(3.5)	16.2	(3.1)	14.6	(3.9)
Pic. Mem. D	7.7	(2.2)	7.9	(2.3)	8.2	(2.0)	7.2	(2.1)
Motor Speed								
Pegboard (Dom. hand)	70.1	(16.2)	68.0	(14.8)	68.6	(15.0)	72.8	(17.9)
Pegboard (Nondom.)	68.2	(16.1)	69.1	(11.9)	69.8	(12.1)	74.6	(24.2)
Verbal Fluency								
Names	25.7	(6.8)	26.9	(6.4)	25.9	(6.4)	25.2	(7.5)
Animals	21.1	(7.8)	22.3	(9.7)	21.9	(8.3)	19.5	(5.1)

paring the SNs and ASPs to ascertain the nature of any systematic differences between these two groups. One-way ANOVAs (with alpha set at .01 to account for multiple comparisons) revealed a significant difference on level of alcohol use, with the SNs reporting significantly heavier alcohol consumption in the past year compared to the ASPs [17.7 drinks/week *vs.* 7.6; $F(1,141) = 8.83$, $p = .004$]. No other differences between the groups emerged. These findings indicate that while recruitment efforts aimed at achieving demographic comparability across groups succeeded, these groups still differed in alcohol use. This may partially account for the findings, and because of the differential distribution of alcohol across the three groups statistical controls are not adequate to control for the contribution of alcohol to these findings.

The contribution of alcohol use to the unexpected pattern of results was also tested using *post-hoc* analyses to examine serostatus groups dichotomized by amount of alcohol use (7 drinks/week or less; 7 drinks/week or more). Two-way (3×2) ANOVAs were conducted for those tests in which ASPs evidenced better performance than SSPs (Picture Memory Interference Books C and D and Sequential Reaction Time I and II). Serostatus was treated as a three-level variable (SN, ASP, SSP) and alcohol use was dichotomized as described above. An interaction between alcohol and serostatus was found for Sequential Reaction Time I [$F(2,220) = 6.68$, $p = .002$], with SSPs categorized as heavy drinkers evidencing poorer performance than all other groups (SN and ASP, heavy and light drinkers).

Finally, to further elucidate the lack of differences between SNs and SSPs on these measures another series of one-way ANOVAs was conducted comparing only the SNs and SSPs within levels of alcohol use. To more precisely delineate this, three levels of alcohol use were employed: *light* (0–7 drinks/week); *moderate* (7–21 drinks/week) and *heavy* (21 or more drinks per week; this three-level grouping could not be employed with the ASP participants as only 8 ASP participants endorsed 21 or more drinks per week). As expected, within the heavy drinking group SSPs performed more poorly than SNs on Sequential Reaction Time

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Table 5. Test scores (unadjusted) by cocaine use groups

	Cocaine naïve		Past users		Current infrequent user		Current frequent user		Current user toxicology negative		Current user toxicology positive	
Test	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)
Verbal Memory (AVLT)												
Total Words	47.7	(9.3)	46.5	(8.7)	47.7	(8.6)	47.9	(8.1)	48.1	(8.7)	47.5	(7.9)
Immed. Recall	9.8	(2.7)	9.5	(2.7)	9.5	(2.7)	10.1	(2.5)	9.8	(2.7)	9.8	(2.5)
Delayed Recall	9.5	(2.8)	9.2	(2.8)	9.4	(2.7)	9.7	(2.7)	9.4	(2.8)	9.7	(2.5)
Delayed Recog.	14.0	(1.5)	13.9	(1.4)	14.0	(1.6)	14.3	(.95)	14.2	(.90)	14.0	(1.8)
Psychomotor Speed II												
CFM 1 (s)	53.1	(20.6)	53.7	(24.6)	55.8	(21.2)	55	(19.0)	54.4	(19.8)	56.8	(20.8)
CFM 2 (s)	113	(47.3)	108.3	(47.0)	119.8	(44.5)	117.1	(53.7)	118.3	(45.3)	119	(52.7)
CFM 3 (s)	130.8	(48.8)	126.4	(48.0)	149.4	(50.4)	136.1	(42.0)	146.1	(47.1)	141.2	(47.9)
Block Design (raw score)	35.0	(7.9)	33.7	(9.2)	32.6	(9.1)	34.2	(7.9)	32.7	(8.6)	34.1	(8.5)
Simple RT	433.3	(284.8)	428.6	(209.6)	447.4	(229.2)	437.1	(233.3)	397.8	(177.7)	504.1	(276.7)
Psychomotor Speed I												
Color Trails 1	37.0	(18.1)	37.8	(19.4)	42.9	(17.7)	35.6	(18.7)	40.9	(20.9)	38.3	(14.8)
Color Trails 2	82.1	(28.9)	87.8	(36.7)	90.1	(31.4)	82.8	(35.2)	88.5	(33.9)	84.9	(32.4)
Trails A	27.8	(12.2)	31.7	(20.5)	28.3	(8.8)	27.2	(11.0)	28.1	(10.9)	27.4	(8.3)
Trails B	75.9	(44.0)	75.5	(42.1)	85.1	(42.6)	77.6	(41.2)	78.7	(39.6)	86.0	(44.9)
Digit Symbol	64.4	(15.5)	60.1	(14.6)	56.7	(11.9)	60.3	(14.9)	56.9	(13.3)	60.0	(13.3)
Reaction Time (ms)												
Sequential RT I	541.7	(118.2)	561.2	(128.7)	570.2	(136.1)	557.8	(156.4)	558.8	(140.9)	572.9	(150.9)
Sequential RT II	629.0	(183.3)	672.3	(190.5)	640.5	(181.8)	649.7	(183.6)	636.0	(181.4)	656.0	(183.7)
Choice RT	405.4	(70.6)	394.4	(60.4)	405.2	(68.6)	392.9	(60.6)	389.9	(55.0)	413.4	(75.5)
Nonverbal Memory (Picture Memory Interference Test, correct recognitions)												
Book A	18.4	(2.1)	17.8	(2.1)	18.4	(1.8)	17.9	(2.8)	17.9	(2.6)	18.5	(1.9)
Book B	14.9	(2.9)	15.1	(3.5)	14.8	(3.4)	14.9	(3.8)	14.8	(3.5)	14.9	(3.6)
Book C	15.9	(3.4)	15.3	(3.2)	15.2	(3.4)	14.4	(4.7)	14.5	(4.1)	15.4	(3.8)
Book D	8.1	(2.1)	7.3	(2.1)	7.9	(2.1)	7.4	(2.2)	7.6	(2.0)	7.8	(2.1)
Motor Speed												
Pegboard–Dom. Hand	68.2	(16.1)	69.1	(11.9)	69.8	(12.1)	74.6	(24.2)	72.4	(22.5)	71.2	(11.7)
Pegboard–Nondom. Hand	75.1	(17.9)	76.4	(17.4)	81.4	(18.2)	80.4	(21.9)	81.4	(22.4)	80.5	(16.1)
Verbal Fluency												
Names	27.4	(7.5)	25.2	(5.4)	25.4	(7.6)	24.0	(6.1)	25.5	(8.0)	24.0	(5.5)
Animals	22.1	(8.5)	21.4	(8.3)	20.2	(8.0)	20.1	(5.3)	19.9	(4.9)	20.4	(8.9)

I [$F(1,38) = 14.6$, $p < .001$) and Sequential Reaction Time II [$F(1,38) = 8.9$, $p = .005$]. Also consistent with expectations, within the group of light drinkers, SSPs also performed worse than SNs on both measures of sequential reaction time [Sequential Reaction Time I: $F(1,101) = 4.1$, $p = .04$] [Sequential Reaction Time II: $F(1,101) = 4.4$, $p = .04$]. Contrary to expectations, when we examined the moderate drinkers (7–21 drinks per week) SNs performed worse than SSPs on Sequential Reaction Time I [$F(1,26) = 8.7$, $p = .007$].

Cocaine Effects

No main effects for pattern of cocaine use were obtained. When participants were grouped by recency of cocaine use, performance on traditional indices of psychomotor speed (Psychomotor Speed I) was found to vary as a function of recent cocaine exposure. A series of *post-hoc* tests probing the main effect of cocaine use on the Psychomotor Speed I measures were conducted by comparing nonusers to past users, nonusers to tox-negative current users, and past users to tox-positive current users. Two-way MANCOVAs controlling for age and alcohol use indicated that nonusers evidenced better performance on traditional measures of psychomotor speed than tox-negative current cocaine users [$F(2,118) = 3.13$, $p = .01$], with a significant univariate effect on the EIWA Digit Symbol subtest [$F(2,118) = 5.62$, $p = .02$].

Contrary to expectations, there were no significant main effects for serostatus or cocaine use on verbal memory, the tests comprising the Psychomotor Speed II factor, motor speed, or verbal fluency, and no HIV × Cocaine Use interactions were obtained on any NP domain.

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Table 6. Multivariate Analysis of Covariance and Variance: Sero–symptom Status × Cocaine Use

		Serostatus × Cocaine Toxicology			Serostatus × Pattern of Cocaine Use		
NP domain	Effect	Wilks's Lambda	*F*	*p*	Wilks's Lambda	*F*	*p*
Verbal Memory ($N = 229$)	C × S	.90	.92	.58	.91	.87	.64
	Cocaine	.97	.48	.93	.97	.55	.88
	Serostatus	.96	1.10	.39	.96	1.07	.38
Psychomotor Speed II ($N = 212$)	C × S	.85	1.04	.41	.87	.95	.54
	Cocaine	.93	.92	.54	.94	.76	.73
	Serostatus	.95	1.11	.35	.94	1.19	.29
Psychomotor Speed I ($N = 232$)	C × S	.89	.86	.68	.87	1.00	.46
	Cocaine	.88	1.79	.03	.90	1.59	.07
	Serostatus	.91	2.14	.02	.92	1.90	.04
Reaction Time ($N = 226$)	C × S	.93	.84	.66	.94	.70	.81
	Cocaine	.96	1.07	.38	.96	.90	.52
	Serostatus	.94	2.26	.04	.95	2.00	.06
Nonverbal Memory ($N = 235$)	C × S	.90	1.03	.42	.91	.89	.62
	Cocaine	.94	1.19	.29	.94	1.18	.30
	Serostatus	.92	2.49	.01	.93	2.12	.03
Motor Speed ($N = 235$)	C × S	.96	.74	.71	.98	.33	.98
	Cocaine	.99	.50	.81	.96	1.57	.16
	Serostatus	.98	1.03	.39	.98	.96	.43
Verbal Fluency ($N = 236$)	C × S	.94	1.09	.37	.94	1.08	.39
	Cocaine	.96	1.44	.20	.97	1.27	.27
	Serostatus	.97	1.50	.20	.98	1.13	.34

DISCUSSION

The present study was designed to examine neuropsychological (NP) performance as a function of serostatus and cocaine use in a sample of gay and bisexual African American men. This project is the most comprehensive study to date conducted on the neurobehavioral sequelae of HIV–AIDS in a non-IDU community sample of African American men. NP performance was examined in multiple domains, with an emphasis on those that are preferentially affected by both HIV infection and cocaine use. Consistent with previous studies on primarily White gay samples, the results reveal a pattern of diminished NP performance among symptomatic seropositives (SSPs) compared to both seronegatives (SNs) and asymptomatic seropositives (ASPs), especially on tests assessing psychomotor speed, reaction time, and nonverbal memory. Contrary to expectations, however, cocaine use, either independently or in interaction with serostatus, did not contribute to poorer NP performance in this sample. The one exception was slower psychomotor speed among recent cocaine users compared to nonusers.

Several factors are considered in accounting for these results, including the nature of the sample, the potentially greater impact of alcohol rather than cocaine on NP performance, the relatively modest level of reported cocaine use in the sample, and issues related to the NP measures used.

Serostatus Effects

Consistent with previous evidence and as predicted, SSPs evidenced significantly poorer performance on tests of psychomotor speed than SNs. This finding is congruent with the characterization of psychomotor slowing as a hallmark of HIV-related cognitive decline and has been documented by numerous studies (Bornstein et al., 1993a,b; Heaton et al., 1995; Miller et al., 1990; Saykin et al., 1988; van Gorp et al., 1989).

Several of the findings observed across serostatus groups only partially supported the study hypotheses. While differences in nonverbal memory were obtained in this sample, nonverbal memory performance has been mixed in previous studies. The stronger nonverbal memory performance of ASPs relative to SSPs is consistent with the extant literature that supports the absence of clinically significant levels of HIV associated cognitive decline prior to the advent of symptomatic illness (McArthur et al., 1989; Selnes et al., 1990). This finding, is however, inconsistent with findings reported by other studies that describe NP decline in ASPs (Heaton et al., 1995; Maj et al., 1994; Stern et al., 1991). That the SNs did not demonstrate better nonverbal memory performance relative to either group was unexpected. Unexpected null findings were also obtained with measures of reaction time even though these measures have been shown in previous studies to tap subtle slowing which

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may be evidenced early in the infection (Martin et al., 1992; Wilkie et al., 1990, 1992). Finally, the null findings for both verbal memory and the tests that comprised the complex psychomotor speed factor were unexpected given that serostatus differences in these domains have been well documented by other authors (Heaton et al., 1995; Stern et al., 1991).

The slightly stronger performance of the ASPs relative to SSPs in the absence of differences between the SNs and the SSPs on tests of nonverbal memory and reaction time was unexpected. While other studies have reported slightly lower performance in SNs compared to ASPs (Miller et al., 1990), the fact that the SNs did not outperform the SSPs in this study is puzzling, and may in part be explained by alcohol use. *Post-hoc* analyses revealed that SSPs with heavy and light alcohol use performed worse than SNs; however, SNs with moderate levels of alcohol use actually performed worse than SSPs. Among those with light alcohol consumption, it is likely that serostatus explains the difference between the SSPs; however the opposite effect among the moderate drinking SNs and SSPs is unexpected. Unfortunately, the absence of historical data on alcohol use limits our ability to draw any conclusions regarding the contribution of chronicity of alcohol use to these findings among the moderate drinking SNs and SSPs.

The poorer performance of the SSPs with heavier alcohol consumption suggests that heavy alcohol use may have eclipsed the contribution of HIV serostatus or that it may confer an additional risk that is magnified among sicker individuals. The discovery of this synergistic effect of alcohol use and HIV on sequential reaction time suggests that alcohol use may render an HIV-positive individual vulnerable to more pronounced cognitive decline with the onset of constitutional symptoms. While little work examining the neuropsychological effects of alcohol and HIV has been conducted, studies employing magnetic resonance spectroscopy have described an additive effect of HIV and alcohol use on white matter concentrations of phosphodiester and phosphocreatine suggesting that HIV related CNS changes are exacerbated by alcohol use (Meyerhoff et al., 1995). In addition, alcohol abuse and HIV infection have also been implicated in frontal lobe function as tested with auditory P3A evoked potential, with alcohol abuse augmenting HIV associated decrements in frontal lobe function (Fein et al., 1995, 1998). Our observation of a decrement in performance among SSPs with heavier alcohol use on a test of sequential reaction time is consistent with the findings obtained by Fein et al., and this result in combination with work by other researchers in this area highlight the need for further work examining the neuropsychiatric sequelae of HIV and alcohol use.

The amount of variability in alcohol use among the SN controls raises some interpretive concerns. First, the SNs were consuming significantly more alcohol than the ASPs, a factor which may explain some of the findings obtained. This study endeavored to ascertain an HIV-seronegative sample that was comparable to the HIV seropositive sample on demographic factors, sexual orientation, and drug use, and thus, the somewhat high levels of drug and alcohol use were not altogether unanticipated. Nonetheless, an HIV-seronegative sample characterized by levels of alcohol use at or higher than the seropositive group may have actually obscured some of our findings, since this sample as a whole may be experiencing cognitive problems secondary to alcohol use. Also, the alcohol use variable only indexes alcohol use during the past 12 months, and does not provide a lifetime index. Given that alcohol use is believed to impact NP performance through numerous mechanisms and over a longer duration than 12 months (Grant, 1987), this variable may not be adequately gauging long-term patterns of use that may better elucidate the likelihood of alcohol related cognitive impairments in this sample. Future work focusing on a subset of seronegative individuals endorsing lower levels of alcohol consumption will be conducted to better address this issue.

Our NP test battery included both traditional and newer measures developed specifically for the WHO international collaborative HIV studies (Maj et al., 1993). In a pilot study designed to test the sensitivity of the newly developed tests in the WHO test battery, the WHO–AVLT and Color Trails 1 and 2 were found to discriminate between symptomatic seropositives and seronegative controls in samples collected in Kinshasa and Munich, with symptomatic seropositives evidencing poorer performance on both measures (Maj et al., 1993). Greater NP impairment as indexed by scores on the WHO battery was observed among symptomatic seropositives as compared to seronegatives in all of the pilot testing sites worldwide, while poorer performance between asymptomatic seropositives and seronegatives were observed in the Sao Paolo and Kinshasa sites (Maj et al., 1994). Finally, recent work conducted using Color Trails 1 and 2 and the WHO–AVLT has revealed that the both tests were sensitive to HIV sero and symptom status (Starace et al., 1998). Thus, the sensitivity of tests comprising this test battery to HIV effects has been demonstrated internationally, and have shown cross-cultural utility in assessing domains preferentially affected by HIV. While several of the tests comprising the WHO have shown good sensitivity, lack of data on some of the other measures (e.g., Color Figure Mazes) has limited our ability to compare these findings to those obtained by other investigators.

Cocaine Effects

Unlike previous studies that reported significant neurocognitive effects of cocaine, we obtained only one significant group difference as a function of level of cocaine use. Specifically, as expected, participants who never used cocaine evidenced better psychomotor speed as measured by the EIWA Digit Symbol test than current users who tested toxicology negative.

While this significant finding for cocaine was ultimately in the expected direction, the overall absence of findings as a function of pattern and recency of cocaine use was gen-

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erally unexpected. These results are attributable in part to the relatively modest level of cocaine use in the present sample. The extant literature examining the NP effects of cocaine use has been variable, with most studies of heavy users reporting decreased NP performance relative to nonusers (Ardila et al., 1991; O'Malley et al., 1992). In general, however, the literature on cocaine use has also been somewhat inconsistent with regard to types of NP impairments obtained, and the types of issues (e.g., measurement error, heterogeneity in use) faced in this study may be in part responsible for the types of findings obtained herein. Specifically, our ability to capture group differences may have been limited given that our measure only tapped the pattern of cocaine use during the past 12-month period, and the potential heterogeneity of the groups. For example, the group labeled *past user only* may have included individuals who used frequently up until 1 year ago, as well as individuals who have merely experimented with cocaine on a few limited occasions. Sample sizes and power limit our ability to further distinguish these groups. Furthermore, we did not account for amount of use, a variable that is difficult to capture given intraindividual variability in patterns of use and availability of resources to obtain drugs. Accurate amount estimates were not captured in the present study; thus, it was not possible to include this quantitation variable. Finally, other variables such as duration of use were also examined and not found to have an impact on NP performance in this sample.

It is possible that there is a threshold level and duration of cocaine use that must be crossed before NP impairment is observed. In the case of our sample, while mean duration of use was higher than that reported in at least three studies that obtained significant cocaine effects (i.e., Ardila et al., 1991; Beatty et al., 1995; O'Malley et al., 1992), level of use in this sample was substantially lower on average. Thus, it appears that our sample included primarily self-reported light to moderate users who have a long and variable history of use (i.e., recreational users).

One methodological caveat regarding the toxicology data must also be noted. All participants were instructed to refrain from drug use for 72 hr prior to the testing. If participants tested as toxicology positive on the day of testing, it indicated that either (1) they did not comply with the study instructions and used drugs within the 72-hr period before testing (but were not acutely intoxicated at the time of the study); or (2) they did comply with the study request of 72 hr abstinence but registered a positive toxicology result for some other reason. Thus, participants were asked to deviate from their typical pattern of use for the period immediately prior the testing. It is possible that this forced change in drug use patterns may explain the unanticipated finding of toxicology-negative current users evidencing the poorest performance.

Our results provide some evidence that even light to moderate cocaine users can experience detrimental neurocognitive effects on psychomotor speed, though these deficits were seen on only one of the measures of this functional domain. The absence of appreciable differences in all other domains may be attributable to the heterogeneity of cocaine use within the sample. It is also possible that cocaine in light to moderate levels of use may exert only subtle central nervous system effects and the deficits described by some studies are attributable to significantly heavier use or to other cerebrovascular sequelae of cocaine (Levine et al., 1987; Pascual-Leone et al., 1991a). Additional research testing this threshold hypothesis is needed in order to clarify these findings.

Joint Effects of HIV and Cocaine

We hypothesized that cocaine use would exacerbate the neurocognitive effects of HIV-1 infection. Contrary to expectations, however, no interaction effects were obtained. Issues such as the potentially confounding effects of alcohol use, a sample characterized by largely recreational drug users, and heterogeneity within subgroups with regard to substance use histories may have contributed to these negative findings. However, these results are not entirely incongruent with the results from other studies of substance abuse, most of which have studied HIV-positive opiate abusers (Bornstein et al., 1993a,b; Concha et al., 1992; Handelsman et al., 1992; Selnes et al., 1992, 1997). Those studies also failed to support a synergistic effect between HIV and substance use on NP performance. Additional research with HIV positive and negative chronic cocaine abusers will be needed to provide a more sensitive test of this hypothesis.

Because there is little information about the neurocognitive effects of HIV infection in non-IDU, gay, and bisexual African American men, it is not clear whether our results are atypical. Previous studies conducted with cohorts of White gay males and IDUs have reported findings similar to these in the domain of psychomotor speed, and the comparable performance of ASPs and SNs is consistent with other studies as well (Goethe et al., 1989; Janssen et al., 1989; McAllister et al., 1992; McArthur et al., 1989; Miller et al., 1990; Selnes et al., 1990).

However, the fact that many of our findings do not closely correspond to studies of White gay males or African American IDUs may be attributable to differences in the samples studied. For example, most of the previous neuropsychological studies that include HIV-positive African American men have primarily studied injection drug users or hospital-based samples, and the deficits observed in those studies may be a function of the health risks secondary to injection drug use and/or to chronic drug and alcohol abuse and their sequelae. While the WHO studies employed diverse international samples, this sample is judged to be quite different from those groups on the basis of both cultural and educational factors (Maj et al., 1993, 1994). The present sample was infected primarily via sexual contact and not injection drug use, and their drug use contributed only indirectly to their infection by increasing the likelihood of engaging in risky sexual behavior. Thus, this study, by focusing on non-IDUs, provides a description of the neurobehavioral se-

quelae of African American men whose risk profile is more comparable to that of the gay White males on whom most of what we know about the neurobehavioral sequelae of HIV-1 is based. Given the current epidemiologic evidence that identifies African American men who have sex with men as the group at highest risk for HIV–AIDS, there is considerable need for more studies to be conducted testing current hypotheses about the NP effects of HIV in this population. Such studies must also take into account the potential contributions of other cofactors such as drug abuse and other psychosocial factors that might contribute to ethnic differences in neurocognitive vulnerability and disease progression.

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