RACIAL DIFFERENCES IN HEPATITIS B AND HEPATITIS C AND ASSOCIATED RISK BEHAVIORS IN VETERANS WITH SEVERE MENTAL ILLNESS

Marian I. Butterfield, MD, MPH; Hayden B. Bosworth, PhD; Karen M. Stechuchak, MS; Richard Frothingham, MD; Lori A. Bastian, MD; Keith G. Meador, MD, MPH; Marvin Swartz, MD; and Ron D. Horner, PhD

Durham, North Carolina and Bethesda, Maryland

Racial differences in the seroprevalence of and risks for hepatitis B (HBV) and hepatitis C (HCV) were examined in military veterans with severe mental illnesses (SMI). Participants (376; 155 Caucasian, 221 African American) were inpatients at a Veterans Affairs (VA) psychiatric unit in Durham, N.C., from 1998 to 2000. Prevalence rates of HBV and HCV were 21.3% and 18.9%, respectively. African Americans had a higher HBV seroprevalence than did Caucasians: 27.6% versus 12.3%; odds ratio (OR) 2.73; 95% confidence interval (CI)=1.55, 4.79. Although not statistically significant, HCV seroprevalence was also higher for African Americans than it was for Caucasians: 21.3% versus 15.5%; OR=1.47; 95% CI=0.86, 2.53. No racial difference was observed for injection drug use (IDU), the strongest risk indicator for both HBV and HCV. Multivariable analyses indicated that African-American race, IDU, and multiple sex partners in the past six months were related to an increased risk of HBV, whereas IDU and smoking crack cocaine were both independently related to an increased risk of HCV. Thus, veterans with SMI—particularly African-American veterans—have high rates of HBV and HCV infection. African-American veterans have significantly higher rates of HBV than do Caucasian veterans, which persist after controlling for prominent risk behaviors. (*J Natl Med Assoc.* 2004;96:43–52.)

 From Health Services Research and Development, Durham VAMC (Butterfield, Bosworth, Stechuchak, Bastian, Horner); Department of Psychiatry and Behavioral Science, Duke University (Butterfield, Bosworth, Meador, Swartz); Department of Medicine, Division of General Medicine, Duke University (Bosworth, Frothingham, Bastian, Horner); Center for Aging and Human Development, Duke University (Bosworth, Meador); Epidemiologic Research and Information Center, Durham VAMC (Frothingham, Horner); Medical Service, Durham VAMC (Frothingham, Bastian); Mental Health Service, Durham VAMC (Butterfield, Meador); and National Institute of Nervous Disorders and Stroke, Bethesda, MD (Horner). Send correspondence and reprint requests for *J Natl Med Assoc.* 2004;96:43–52 to: Marian I. Butterfield, MD, HSR&D, Building 16, Room 70, Veterans Affairs Medical Center (152), 508 Fulton St., Durham, NC 27705; phone: (919) 286-6936; fax: (919) 416-5836; e-mail: mimi@duke.edu, mimi@acpub.duke.edu

Key words: race ♦ hepatitis B ♦ hepatitis C ♦ veterans ♦ mental illness

Vulnerable subpopulations with high rates or risks of hepatitis-B virus (HBV) and hepatitis-C virus (HCV) infection may warrant focal points for public health efforts to reduce or eliminate the transmission of these diseases. Although four million persons in the United States—or 1.8% of the population—are thought to be infected with HCV, the estimated U.S. rates for non-Hispanic African Americans are as high as 3.2%.[1] For HBV, the estimated rates for the United States are approximately 5%,[2] whereas the rates for non-Hispanic African Americans are as high as 12%.[3] Recently, persons with severe mental illness (SMI), including veter-

ans, were specifically defined as another high-risk subpopulation for both HBV and HCV infection, with rates of 20% and 23%, respectively.[4-6] SMI criteria include the presence of a major mental illness, chronicity, and pervasive impairment of function. SMI diagnoses include schizophrenia, bipolar disorder, depression, and, for veterans, PTSD because of its severe and disabling effect in this population.

Military veterans, particularly those accessing the Department of Veterans Affairs (VA) health services, represent a significant proportion of the homeless and deinstitutionalized mentally ill.[7] High rates of these infections have been reported in veteran cohorts: as many as 30% of homeless, impoverished veterans are HBV-infected,[8] and 10–35% of veterans using VA health services are HCV-infected.[9,10] Combat and medical work have been described as risk factors for HCV in veterans.[1,11] Among veterans with HCV infection, high rates of SMI have been reported.[6] Collectively, these individuals constitute an overlapping subpopulation at high risk for both HBV and HCV infection.

The extent to which race/ethnicity factors contribute to the heightened risk is not known. The risks for minorities may be underestimated, because these infections are under-recognized among persons belonging to disenfranchised risk groups (i.e., homeless or deinstitutionalized persons) of which minorities are disproportionately represented. Furthermore, veterans with SMI are often unaware of their hepatitis infections,[4] which raises concerns about the risk of continued transmission. Race/ethnicity may be linked to several social contextual factors (e.g., poverty, homelessness) that place members at greater risk for exposure to and subsequent development of HBV and HCV. Thus, it is crucial to characterize this high-risk population further and to understand if there is a role of race/ethnicity in the risks of these infections.

In this study, we examine racial differences in the constellation of risk behaviors associated with HBV and HCV infection among veterans with SMI. Specifically, we address two questions: 1) Does the prevalence of HBV and HCV risk behaviors differ by race in a sample of veterans with SMI? and 2) Do racial differences in risk behaviors (e.g., combat exposure, injection drug use [IDU], sharing needles, and unprotected sex for drugs) account for any observed racial differences in seroprevalence for HBV and HCV?

PATIENTS AND METHODS

Study Design

This is a cross-sectional study of risk behaviors and the prevalence of HBV and HCV infection among veterans with SMI. We recruited 399 consecutive patients admitted to the Durham VA inpatient psychiatric unit between March 1998 and June 2000. The study setting was a VA psychiatric ward with 26 inpatient beds and an average length of stay of eight days.

Patient Population

All participants were psychiatric inpatients who met diagnostic criteria for SMI, provided informed consent, and were between the ages of 20 and 80. The Durham VA's Institutional Review Board approved the study and the manner by which informed consent was obtained from participants. Most participants were from the surrounding catchment area of North Carolina and southern Virginia, a mixed urban/rural region. Primary diagnoses were schizophrenia, schizoaffective disorder, bipolar-I disorder, and post-traumatic stress disorder (PTSD). Psychiatric diagnoses were based on clinical diagnoses, record review to confirm that participants met the diagnostic criteria of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV),[12] and consensus review of the diagnosis by two attending psychiatrists. Confirmation by the Structured Clinical Interview for DSM-IV (SCID) was also completed in a subset of 25 patients; in all patients, psychiatric diagnoses were concordant with the clinical diagnoses.[13] A PTSD diagnosis was additionally confirmed through VA records indicating that a patient was receiving compensation for disability related to PTSD or had exacerbation of PTSD symptoms rendering the hospital admission and through a confirmation using the PTSD Checklist (PCL).[14] Only 3% of eligible persons refused to participate in the study. Participants were paid a participation fee of $25.

Data Collection

After giving informed consent, each study participant completed a standardized, hour-long, structured risk interview conducted by two trained interviewers who were experienced with this population and had good agreement on inter-rater reliability tests. The risk interview was adapted from

established instruments for assessing HIV risk behavior, prevalence, and associated variables of relevance in populations with SMI.[15] The domains surveyed on the interview included demographic characteristics and HIV-associated risks and behaviors, including sex-risk behaviors. Study participants also received pretest counseling for HIV and provided blood specimens.

Outcomes

The primary outcome variables were HBV and HCV serostatus, which were determined through a blood specimen collected within 72 hours of the informed consent and the interview. HBV seropositivity was defined as having HBV core antibody as assessed by the Abbott Corzyme test according to the manufacturer's criteria. HCV seropositivity was defined as having antibodies to HCV, indicating current or past HCV infection. Antibodies to HCV were determined in sera samples by the Abbott HCV enzyme immunoassay 2.0. Initially reactive results were repeated in duplicate, and immunoblot (RIBA, Ortho, Raritan, NJ) or polymerase chain reaction (Roche, Indianapolis, IN) confirmation was performed on HCV enzyme immunoassay reactives. All serological tests were performed under well-controlled conditions by laboratory staff at the Durham VA clinical microbiology laboratory, which is accredited by the College of American Pathologists.

Explanatory Variables

The primary explanatory variable was self-identified racial background. Because our sample was composed predominantly of African-American and Caucasian veterans, we were interested in the effects of race. We excluded from the analysis 19 individuals who reported other racial background categories such as Hispanic, American Indian, Pacific Islander, Alaskan Native, and "other" because of small sample sizes in these categories. We also excluded four white individuals who did not have hepatitis test results.We assessed risk behaviors with the AIDS Risk Inventory, which assesses nearly all HBV and HCV risk behaviors. The AIDS Risk Inventory is a structured interview for assessing risk of HIV infection. It probes risk behaviors associated with drug use and sexual practices and assesses both lifetime risk behaviors and behaviors in the preceding six months. In general, we report lifetime risks, except

Table 1. Demographic Characteristics of Study Population, Shown by Race

Variable	Caucasians (n=155) Percent	African Americans (n=221) Percent	*P*
Age 50 or over	56.77	31.22	<0.0001
Male	90.97	90.05	0.8594
Currently married	38.06	18.18	<0.0001
Children: at least one	72.90	72.73	1.0000
Education level			0.4937
<High school	12.90	9.95	
High school	20.65	24.89	
>High school	66.45	65.16	
Residence			0.0195
Independent or with family	87.10	81.00	
Institutionalized or supervised	1.29	6.33	
Homeless	3.23	7.24	
Other	8.39	5.43	
Psychiatric diagnosis			<0.0001
Schizophrenia	17.42	48.87	
Schizoaffective	9.68	9.50	
Bipolar	25.81	9.50	
PTSD	47.10	32.13	

for multiple sex partners during the past six months. Lifetime risk was analyzed because it is a better indication of exposure than is past-six-month risk. Compared with other risk interviews, the AIDS Risk Inventory is a more sensitive instrument, is able to discriminate between high- and low-risk HIV behaviors, and reliably assesses behavior patterns associated with a high risk of HIV infection.[15] We previously used this instrument successfully in examining HCV risk factors.[4]

Analysis

In addition to determining HBV and HCV serostatus, we analyzed the risk behaviors queried on the AIDS Risk Inventory, including IDU, unprotected sex (vaginal, anal, oral), crack use, and sniffing or snorting drugs. Racial differences for demographic and risk factors were assessed by chi-squared statistics.

Multiple logistic regression analyses were performed to examine the association of hepatitis with race, risk factors, and other clinically relevant variables. Variables were selected for inclusion in the models on the basis of clinical relevance, known associations with these infections (e.g., IDU, combat, homelessness), or to adjust for significant differences in the racial distribution of our variables. Because Vietnam-era veterans are at risk for these infections and are over age 50, we used a cut-off value of 50 years or older as a variable. The variables included in the final models were: psychiatric diagnosis, marital status, living independently or with family, age over 50, combat exposure, lifetime risks of IDU, smoking crack cocaine, sniffing or snorting drugs, unprotected sex for drugs, multiple sex partners in the past six months, and the presence of an alcohol use disorder. All analyses were conducted with SAS version 8.1. All statistical tests were two-tailed and conducted at a 0.05 significance level.

RESULTS

Population Characteristics

The final sample was 376 persons with SMI: 155 Caucasian and 221 African-American military veterans, of whom over 90% were men (see Table 1). Compared with Caucasians, African Americans were younger, a smaller proportion were currently married, and a greater proportion were institutionalized or homeless. There were no racial differences in educational status or having at least one child. A greater proportion of African Americans had a diagnosis of schizophrenia spectrum disorders and drug use disorders, fewer were diagnosed with PTSD or bipolar disorder, and a smaller proportion had experienced combat exposure. Over 40% of the study cohort had an alcohol use disorder, with no racial difference observed.

HBV and HCV Infection Rates and Risk Factor Prevalence

For HBV, the overall seroprevalence rate was 21.3%. It was a new diagnosis for 65% (52 of 80) of the HBV-positive patients. African Americans had a higher HBV rate than did Caucasians: 27.6% versus 12.3%; OR=2.73; 95% CI=1.55, 4.79 (Table 2). The overall HCV seroprevalence was 18.9%, and, for 56% (40 of 71) of the HCV-positive patients, it was a new diagnosis. The HCV rate was also higher in African Americans; although this difference was not statistically significant, a trend was observed: 21.3% versus 15.5%; OR=1.47; 95% CI=0.86, 2.53.

African Americans were also more likely to have a self-report of a drug use disorder than were Caucasians. No racial differences in lifetime IDU and sharing needles were observed. African Americans were significantly more likely than Caucasians to report smoking crack and sniffing or snorting drugs over their lifetime.

Among sexual risk behaviors, African Americans were more likely to report engaging in sex for drugs and were more likely than Caucasians to report having multiple sex partners in the past six months.

Multivariable Analysis

After adjusting for other important factors, we found that African Americans continued to have a higher risk of testing positive for HBV: OR=2.79; 95% CI=1.41, 5.53 (see Table 3). Notably, the multivariable analysis did not eliminate or weaken the association for race. Other factors also significantly associated with HBV seropositivity were IDU (OR=4.54; 95% CI=2.28, 9.04) and having multiple sex partners in the last six months (OR=2.01; 95% CI=1.06, 3.78). In contrast, in the multivariable analysis, race was not significantly related to serostatus for HCV (OR=1.52; 95% CI=0.61, 3.79), but IDU (OR=40.53; 95% CI=16.72, 98.24) and use of crack cocaine (OR=5.20; 95% CI=1.91, 14.17) remained independent risk factors for HCV seropositivity.

DISCUSSION

This is the first study to assess racial differences in HBV and HCV infection rates and self-reported behavioral risks among veterans with SMI, a very high-risk subpopulation. Overall, the prevalence of both these infections was high. Over half of these infections were newly diagnosed; thus, most veterans were unaware of the infection. African Americans had higher rates of both HBV and HCV than did Caucasians. This difference was statistically significant for HBV, and a similar trend was observed for HCV. There was no significant racial difference observed in this sample for lifetime IDU. However, several drug- and sex-risk behaviors were reported significantly more often in African Americans than in Caucasians, specifically, smoking crack, sniffing or snorting drugs over their lifetime, unprotected sex for drugs in their lifetime, and multiple sex partners in the past six months. These observations are consistent with findings on high-risk sexual behavior reported for the general population.[16,17] The rates of hepatitis infections in veterans with SMI are much higher than those reported in the general population for both races and across similar age groupings.[1-4]

After controlling for potential confounding sex- and drug-risk variables, as well as psychiatric diagnosis, combat exposure, and selected demographics variables, we found that African-American race continued to be a significant factor for HBV infection. In addition, IDU and multiple sex partners also remained associated with HBV infection. African-American race was not significantly associated with HCV seropositivity after we adjusted for other variables. Of the sex- and drug-risk variables, crack and IDU remained significantly associated with HCV seropositivity in the logistic regression.

The racial difference in psychiatric diagnosis observed in our cohort (e.g., higher rates of schizophrenia in African-American veterans and of PTSD in Caucasian veterans) warrants discussion, although it does not account for the racial differ-

Table 2. Hepatitis B and C Prevalence Rates and Risk Factors, Shown by Race

Variable	Caucasians (n=155) Percent	African Americans (n=221) Percent	Odds Ratio	95% Confidence Interval
HBV infected	12.26	27.60	2.73***	1.55, 4.79
HCV infected	15.48	21.27	1.47	0.86, 2.53
Combat exposure	62.99	51.13	0.61*	0.40, 0.94
Alcohol use disorder	40.65	42.99	1.10	0.73, 1.67
Drug use disorder	17.42	32.13	2.24**	1.36, 3.71
Drug/needle risks, lifetime IV drug use	18.06	17.65	0.97	0.57, 1.66
Shared needles	11.61	13.57	1.20	0.64, 2.23
Smoked crack	25.16	61.82	4.82***	3.06, 7.58
Sniffed/snorted drugs	47.74	63.35	1.89**	1.25, 2.87
Type of unprotected sex, lifetime				
Any type of sex	97.42	99.55	5.83	0.65, 52.65
Vaginal sex	97.42	99.10	2.90	0.52, 16.04
Anal sex	24.03	21.82	0.88	0.54, 1.44
Oral sex	70.78	68.78	0.91	0.58, 1.42
Sex for drugs	7.79	19.91	2.94**	1.50, 5.78
Sex for money/gifts	17.11	23.08	1.45	0.86, 2.46
Multiple sex partners past six months	16.88	36.36	2.81***	1.70, 4.65

*p<0.05; **p<0.01; ***p<0.001

ences in hepatitis infection rates. The racial differences in diagnosis may be due to differences in treatment-seeking patterns for the veterans in our service area and may reflect the largely urban and minority characteristic of the surrounding community. The area the Durham VA serves is urban, and over half the patients seeking services are African Americans. A large study of HCV-infected veterans found that 86% had a comorbid psychiatric or substance use disorder; PTSD was a risk factor for HCV in these veterans, whereas psychosis was not.[6] Thus, we would expect that, if there were a racial bias in our diagnosis, it would minimize the racial difference in these infections rather than increase it. Of note, there were higher rates of combat exposure among Caucasians in this sample, and PTSD diagnoses were confirmed by the self-report symptom checklist. Alternatively, racial bias in diagnosing psychotic spectrum disorders more frequently in African Americans than in Caucasians can occur in clinical and research settings.[18-20] It is hypothesized that clinician or screening-instrument bias contributes to a misdiagnosis of SMIs in African Americans.[21] Errors in psychiatric diagnoses may also be related to cultural insensitivities in traditional mental health systems, clinician prejudice, or to ethnic differences in the ways that patients express emotional distress and psychotic symptoms.[19,22-24] African Americans with PTSD may endorse more psychotic symptoms without higher rates of primary psychosis than do Caucasians, leading to an underdiagnosis or misdiagnosis of PTSD in African-American veterans.[25] Rates of service connection for veterans with PTSD are substantially lower for African Americans than they are for other veterans, a finding that persists after the authors adjusted for differences in PTSD severity and functional status.[26] This racial disparity may lead to less access to psychiatric services for African Americans with PTSD and contribute to fewer African Americans in the PTSD group in this study, which warrants further research.

Our results are consistent with studies that report higher rates of HCV infection[1,27] and HBV infection[2,28] in minority populations, although we noted only a trend toward higher rates of HCV infection. In our model, race continued to be significantly related to an increase in HBV-positive serostatus after we adjusted for other known risk factors. Similarly, our results lend support to research linking African-American race to a number of independent risk factors for HBV infection, such as increased number of sexual partners and cocaine use.[2] HBV risks, however, could be mediated through social and contextual factors such as poverty, neighborhood exposure, or lifestyle choices not measured here and perhaps not attributable to the construct of race. A plausible reason for the higher rates in African Americans is a potential difference in baseline prevalence rates of HBV within social networks; hence, exposure rates could vary between different groups. For example, we did not evaluate the study participants on the basis of the neighborhoods in which they reside, which has been shown to explain some of the racial variance in crack use.[29] Alternatively, some undefined risk factor may account for the higher rate in African-American veterans. There may be racial differences in susceptibility to HBV, which has been reported for other infections, e.g., tuberculosis,[30] malaria,[31] and for the clearance of HCV.[32] In summary, the relative frequencies and patterns of HBV risks in the different racial groups and the baseline prevalence rates among exposure groups may explain the prevalence differences that we observed.

Although the prevalence of several risk behaviors demonstrates clear racial differences, the actual risks of HCV transmission were similar for African Americans and Caucasians. Overall, IDU posed the greatest risk for HCV in this cohort, which supports the findings of other studies[4,10,33] and extends those findings to a high-risk subpopulation, veterans with SMI. Our ability to control for several factors—particularly IDU—is an important strength of our study, compared with earlier work.[1] The significant risk of smoking crack cocaine and HCV infection, however, remained after we adjusted for IDU in the HCV model. Although this finding is divergent from prior studies,[34,35] it is consistent with recent reports.[36,37] One of those reports of crack cocaine users, most of whom were African-American men, also found high rates of HCV (33%). Over a third of the HCV-positive patients, however, denied any lifetime IDU. The authors concluded that other cocaine-related factors outside of its parenteral use might be related to the high rates observed in their cohort.[36] A higher prevalence of crack cocaine use among African Americans than among Caucasians is consistent with other population studies of patterns of drug use in the United States.[38,39]

Crack cocaine use has physical, social, and biological effects that may explain why it remained an

independent risk factor for HCV in our study. Physically, when crack cocaine is smoked, individuals can suffer mucosal burns and ulcerations, which could enhance the likelihood of HCV viral transmission. Crack has neurochemical effects that make it uniquely addictive and, with chronic use, causes strong cocaine craving and a desire for frequent use.[40] In addition to a desire to use crack frequently, crack's neurochemical effects can result in increased sexual risk behaviors.[41] Our multivariate analyses, however, showed that the analyzed sex-risk behaviors did not contribute independently to HCV infection. Thus, the social and contextual factors that underlie the enhanced use of crack cocaine among African Americans are complex, and the biological effects of this drug may also underlie other high-risk behaviors, including sex-risk behaviors.

In contrast, having multiple sex partners was related to HBV in the multivariate model. Although epidemiological evidence suggests that IDU—not sexual behavior[1,2]—is the predominant mode of transmission for both these infections, our findings highlight that HBV can also be spread sexually and that it is transmitted more efficiently by sex than HCV is.[42,43] HBV and HCV have unique aspects in both mode of and risk factors for transmission. Both viruses may be transmitted by percutaneous or permucosal exposure to infected body fluids.[44-46] The HCV model is better characterized in this regard than that of HBV. Nevertheless, there likely are routes of transmission of HBV besides the traditional drug and sex routes that need to be further examined and, as noted, may explain the racial difference we observed.

There are other limitations in this study that temper interpretation of the findings. Whereas IDU was the single most important risk factor predicting HBV and HCV seropositivity, the temporal relationship of risk behaviors to the acquisition of these infections is uncertain, as are the duration and frequency of these behaviors. In addition, as to specific high-risk behaviors queried on the risk

Table 3. Hepatitis B and C Logistic Regression Models

	Hepatitis B		Hepatitis C	
Selected Risk Factors	**Adjusted Odds Ratio**	**95% Confidence Interval**	**Adjusted Odds Ratio**	**95% Confidence Interval**
African-American race	2.79**	1.41, 5.53	1.52	0.61, 3.79
Combat exposure	1.30	0.63, 2.67	1.10	0.41, 2.94
Psychiatric diagnosis[†]				
Bipolar	0.59	0.22, 1.60	0.80	0.22, 2.97
PTSD	1.34	0.66, 2.72	2.06	0.78, 5.40
Currently married	1.67	0.83, 3.40	1.35	0.52, 3.48
Independent or live with family[††]	0.82	0.39, 1.72	0.55	0.21, 1.44
IV drug use	4.54***	2.28, 9.04	40.53***	16.72, 98.24
Smoked crack cocaine	1.18	0.55, 2.54	5.20**	1.91, 14.17
Sniffed/snorted drugs	1.06	0.51, 2.21	0.68	0.25, 1.88
Unprotected sex for drugs	1.25	0.60, 2.61	0.97	0.38, 2.48
Multiple sex partners in past 6 months	2.01*	1.06, 3.78	0.75	0.31, 1.81
Alcohol use disorder	0.93	0.52, 1.69	1.24	0.57, 2.70
Age 50+	1.27	0.67, 2.39	1.64	0.69, 3.95

*p<0.05; **p<0.01; ***p<0.001
Hepatitis B model: c statistic=0.74
Hepatitis C model: c statistic=0.91
[†] Schizophrenia spectrum disorder reference group
[††] Homeless, Institutionalized, or Other reference group

interview, there may be respondent bias or a resistance to disclose socially stigmatized behaviors in a personal interview format. Of note, we did not query lifetime reports of multiple sexual partners but instead inquired about the six months prior to the study interview. This may decrease recall bias but limit a longitudinal assessment of sex-exposure risk. Another limitation is that blood transfusion and tattooing, which have been found to be related to a small percentage of HCV infections in veterans,[11] were unavailable as risk factors in this data set. Prior to 1990, before the national blood supply was screened for HCV, the incidence of transfusion-associated HCV was relatively high, particularly in older persons.[47] Although our study found that older age was related to both HBV and HCV infection, the association was not statistically significant. Finally, this study was conducted on veterans who were psychiatric inpatients and may not be generalizable to outpatient veterans with SMI. Overall, veterans with SMI warrant study because the VA is the largest integrated mental health delivery system in the country, providing services to over 700,000 veterans annually.[48]

In summary, risk factors for HBV were African-American race, IDU, and having multiple sex partners in the past six months. Risk factors for HCV were IDU and crack cocaine use, but not race. The overall prevalence rates of HBV and HCV were very high for this cohort with SMI, but especially high for African-American veterans. Given that the incidence rates of HBV have not changed from 1976 to 1943 and that persons with HCV for the most part remain infective, screening for both viruses should be done in veterans with SMI. Our study suggests that there may be an enhanced vulnerability of individuals with mental illness to particular domains of risk behaviors, such as crack cocaine use for HCV and multiple sex partners for HBV. Increased time spent by mental healthcare providers in assessing these risks may be warranted.

There are important clinical implications for the high rates of hepatitis among SMI persons—particularly African Americans. HCV has been associated with depression,[49] cognitive impairment,[50] emotional distress,[51] and an adverse impact on psychiatric illness. These viruses—particularly chronic HCV—can impair hepatic function, which is relevant to the care of persons with mental disorders who are taking hepatically metabolized drugs.[52] Disparities may exist in accessing newer effective HCV treatments for SMI persons—particularly those who are African American—as has been reported for newer antipsychotics.[53] Further, the drugs used to treat hepatitis (e.g., alpha interferon) have been associated with depression,[54] suicidality,[55] and mania and psychosis.[56] There is a suggestion in the literature that depression—including that induced by HCV treatment—may be prevented by the SSRI antidepressants.[57,58] Further, newer drug therapies—such as peg interferon alpha-2a-plus ribovirin—may have a better side effect profile for patients with mental illnesses (e.g., less depression).[59] Collectively, these are pressing issues that need to be assessed and fully appreciated by mental health providers who treat persons with SMI, particularly those who provide mental health services to African-American people.

ACKNOWLEDGEMENTS

This research is supported in part by the Department of Veterans Affairs, Veterans Health Administration, HSR&D Service, through Program 824 Funds and the Cooperative Studies Program (CSP 706D), and through a VA research career development award to Dr. Butterfield (RCD-0019-2). Dr. Butterfield is an HSR&D core research investigator in the HSR&D Field Program at the VA Medical Center in Durham, NC. The views expressed in this article are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs. The authors wish to thank their collaborating colleagues in the five-site study of HIV in SMI funded by the National Institute of Mental Health (principal investigators at the five sites were Dr. S.D. Rosenberg, Dr. D. Steinwachs, Dr. M.S. Swartz, Dr. S. Woods, and Dr. S.M. Essock).

The findings of this work were presented at the VA Health Services Research 2002 Annual Meeting, February 13–15, 2002, Washington, DC.

We wish to acknowledge the editorial assistance of Patricia M. Spivey, HSR&D, Durham VA Medical Center.

REFERENCES

1. Alter MJ, Kruszon-Moran D, Nainan OV, et al. The prevalence of hepatitis-C virus infection in the United States, 1988 through 1994. *N Engl J Med.* 1999; 341:556-562.

2. McQuillan GM, Coleman PJ, Kruszon-Moran D, et al. Prevalence of hepatitis-B virus infection in the United States: The National Health and Nutrition Examination Surveys, 1976 through 1994. *Am J Public Health.* 1999;89:14-18.

3. Coleman PJ, McQuillan GM, Moyer LA, et al. Incidence of hepatitis-B virus infection in the United States, 1976–1994: Esti-

mates from the National Health and Nutrition Examination Surveys. *J Infect Dis.* 1998;178:954-959.

4. Rosenberg SD, Goodman LA, Osher FC, et al. Prevalence of HIV, hepatitis B, and hepatitis C in people with severe mental illness. *Am J Public Health.* 2001;91:31-37.

5. Brau N, Bini EJ, Shahidi A, et al. Prevalence of hepatitis C and coinfection with HIV among U.S. veterans in the New York City metropolitan area. *Am J Gastroenterol.* 2002;97:2071-2078.

6. El-Serag HB, Kunik M, Richardson P, et al. Psychiatric disorders among veterans with hepatitis-C infection. *Gastroenterology.* 2002;123:476-482.

7. Rosenheck R, Seibyl CL. Homelessness: health service use and related costs. *Med Care.* 1998;36:1256-1264.

8. Gelberg L, Robertson MJ, Leake B, et al. Hepatitis B among homeless and other impoverished U.S. military veterans in residential care in Los Angeles. *Public Health.* 2001;115:286-291.

9. Pueschel M. VHA hepatitis C costs placed at $250 million. *US Med.* 1999;35:52.

10. Cheung R. Epidemiology of hepatitis-C virus infection in American veterans. *Am J Gastroenterol.* 2000;95:740-747.

11. Briggs ME, Baker C, Hall R, et al. Prevalence and risk factors for hepatitis-C virus infection at an urban Veterans Administration medical center. *Hepatology.* 2001;34:1200-1205.

12. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th edition. Washington, DC: American Psychiatric Association, 1994.

13. First MB, Spitzer RL, Gibbon M, et al. Structured Clinical Interview for DSM-IV Axis I Disorders. Washington, DC: American Psychiatric Press, Inc., 1997.

14. Blanchard EB, Jones-Alexander J, Buckley TC, et al. Psychometric properties of the PTSD Checklist (PCL). *Behav Res Ther.* 1996;34:669-673.

15. Chawarski MC, Pakes J, Schottenfeld RS. Assessment of HIV risk. *J Addict Dis.* 1998;17:49-59.

16.Anderson JE, Dahlberg LL. High-risk sexual behavior in the general population. Results from a national survey, 1988–1990. *Sex Transm Dis.* 1992;19:320-325.

17. Leigh BC, Temple MT, et al. The sexual behavior of U.S. adults: results from a national survey. *Am J Public Health.* 1993;83:1400-1408.

18. Neighbors HW, Trierweiler SJ, Munday C, et al. Psychiatric diagnosis of African Americans: diagnostic divergence in clinician-structured and semistructured interviewing conditions. *J Natl Med Assoc.* 1999;91.

19. Coleman D, Baker FM. Misdiagnosis of schizophrenia in older, black veterans. *J Nerv Ment Dis.* 1994;182:527-528.

20. Trierweiler SJ, Neighbors HW, Munday C, et al. Clinician attributions associated with the diagnosis of schizophrenia in African-American and non-African-American patients. *J Consult Clin Psychol.* 2000;68:171-175.

21. Bell CC, Mehta H. The misdiagnosis of black patients with manic depressive illness. *J Natl Med Assoc.* 1980;72:141-145.

22. Neighbors HW. Socioeconomic status and psychologic distress in adult blacks. *Am J Epidemiol.* 1986;124:779-793.

23. Strakowski SM, Flaum M, et al. Racial differences in the diagnosis of psychosis. *Schizophr Res.* 1996;21:117-124.

24. Adebimpe VR, Klein HE, et al. Hallucinations and delusions in black psychiatric patients. *J Natl Med Assoc.* 1981;73:517-520.

25. Frueh BC, Hamner MB, Bermat JA, et al. Racial differences in psychotic symptoms among combat veterans with PTSD. *Depress Anxiety.* 2002;16:157-161.

26. Murdoch M, Hodges J, Cowper D, et al. Racial disparities in VA service connection for post-traumatic stress disorder disability. *Med Care.* 2003;41:536-549.

27. National Institute of Health. Management of hepatitis C: NIH consensus statement. 1997;15:1-41.

28. Kelen GD, Green GB, Purcell RH, et al. Hepatitis B and hepatitis C in emergency department patients. *N Engl J Med.* 1992;326:1399-1404.

29. Lillie-Blanton M, Anthony JC, et al. Probing the meaning of racial/ethnic group comparisons in crack cocaine smoking. *JAMA.* 1993;269:993-997.

30. Stead WW, Senner JW, Reddick WT, et al. Racial differences in susceptibility to infection by Mycobacterium tuberculosis. *N Engl J Med.* 1990;322:422-427.

31. Kwiatkowski D. Genetic susceptibility to malaria getting complex. *Current Opinion in Genetics and Development.* 2000; 10:320-324.

32. Thio CL, Thomas DL, Goedert JJ, et al. Racial differences in HLA class-II associations with hepatitis-C virus outcomes. *J Infect Dis.* 2001;184:16-21.

33. Butterfield MI, Bosworth HB, Meador KG, et al. Gender differences in hepatitis-C infection and risks in persons with severe mental illness. *Psychiatric Services.* 2003;54:848-853.

34. Thorpe LE, Ouellet LJ, Levy JR, et al. Hepatitis-C virus infection: prevalence, risk factors, and prevention opportunities among young injection drug users in Chicago, 1997–1999. *J Infect Dis.* 2000;182:1588-1594.

35. Murphy EL, Bryzman SM, Glynn SA, et al. Risk factors for hepatitis-C virus infection in U.S. blood donors. *Hepatology.* 2000;31:756-762.

36. Harsch HH, Pankiewicz J, Bloom AS, et al. Hepatitis-C virus infection in cocaine users—a silent epidemic. *Community Mental Health J.* 2000;36:225-233.

37. Osher FC, Goldberg R, McNary S, et al. Substance abuse and the transmission of hepaititis C among persons with severe mental illness. *Psychiatric Services.* 2003;54:842-847.

38. Hwang LY, Ross MW, et al. Prevalence of sexually transmitted infections and associated risk factors among populations of drug abusers. *Clin Infect Dis.* 2000;31:920-926.

39. Braun BL, Murray D, Hannan P, et al. Cocaine use and characteristics of young adult users from 1987 to 1992: the CARDIA study. *Am J Public Health.* 1996;86:1736-1741.

40. Wallace B. Treating crack cocaine dependence: the critical role in relapse prevention. *J Psychoactive Drugs.* 1992;24:213-222.

41. Kim A, Galanter M, Casteneda R, et al. Crack cocaine use and sexual behavior among psychiatric inpatients. *Am J Drug Alcohol Abuse.* 1992;18:235-246.

42. Fiscus SA, Kelly WF, Battigelli DA, et al. Hepatitis-C virus seroprevalence in clients of sexually transmitted disease clinics in North Carolina. *Sexually Transmitted Dis.* 1994;21:155-160.

43. Center for Disease Prevention. Recommendations for prevention and control of hepatitis-C virus (HCV) infection and HCV-related chronic disease. *MMWR.* 1998;47:1-36.

44. Holland CA, Ma Y, Moscicki B, et al. Seroprevalence and risk factors of hepatitis B, hepatitis C, and human cytomegalovirus among HIV-infected and high-risk uninfected adolescents. *Sex Transmitted Dis.* 2000;27:296-303.

45. Hollinger F. Hepatitis B virus. In: Fields BN, Knipe DM, Howley PM, eds. Fields virology. Philadelphia:Lippincott-Raven, 1995:2738-2807.

46. Houghton M. Hepatitis-C virus. In: Fields BN, Knipe DM, Howley PM, eds. Fields virology. Philadelphia:Lippincott-Raven, 1995:1035-1058.

47. Vamvakas E, Taswell HF. Epidemiology of blood transfusion. *Transfusion.* 1994;34:464-470.

48. Rosenheck R, Greenberg G. Department of Veterans Affairs national mental health program performance monitoring system: fiscal year 2001 report. West Haven, CT: Department of Veterans Affairs Northeast Program Evaluation Center, VA Connecticut Healthcare System.

49. Dwight MM, Kowdley KV, Russo JE, et al. Depression, fatigue, and functional disability in patients with chronic hepatitis C. *J Psychosomat Res.* 2000;49:311-317.

50. Forton DM, Thomas HC, Murphy CA, et al. Hepatitis C and cognitive impairment in a cohort of patients with mild liver disease. *Hepatology.* 2002;35:433-439.

51. Fontana RJ, Hussain KB, Schwartz SM, et al. Emotional distress in chronic hepatitis-C patients not receiving antiviral therapy. *J Hepatol.* 2002;36:401-407.

52. Carvajal GP, Garcia D, Sanchez SA, et al. Hepatotoxicity associated with the new antidepressants. *J Clin Psychiatry.* 2002;63:135-137.

53. Kuno E, Rothbard AB. Racial disparities in antipsychotic prescription patterns for patients with schizophrenia. *Am J Psychiatry.* 2002;159:567-572.

54. Bonaccorso S, Marino V, Puzella A, et al. Increased depressive ratings in patients with hepatitis C receiving interferon-alpha-based immunotherapy are related to interferon-alpha-induced changes in the serotonergic system. *J Clin Psychopharmacol.* 2002;22:86-90.

55. Ademmer K, Beutel M, Bretzel R, et al. Suicidal ideation with IFN-alpha and ribavirin in a patient with hepatitis C. *Psychosomatics.* 2001;42:365-367.

56. Kanno A, Yamada M, Abe M, et al. A case of interferon alpha-induced manic psychosis in chronic hepatitis C. *Tohodu J Exp Med.* 1999;187:79-82.

57. Gleason OC, Yates WR, Isbell MD, et al. An open-label trial of citalopram for major depression in patients with hepatitis C. *J Clin Psychiatry.* 2002;63:194-198.

58. Kraus MR, Schafer A, Faller H, et al. Paroxetine for the treatment of interferon-alpha-induced depression in chronic hepatitis C. *Aliment Pharmacol Ther.* 2002;16:1091-1099.

59. Fried MW, Shiffman ML, Reddy KR, et al. Peginterferon alfa-2a plus ribavirin for chronic hepatitis-C virus infection. *N Engl J Med.* 2002;347:975-982.

We Welcome Your Comments

The *Journal of the National Medical Association* welcomes your Letters to the Editor about articles that appear in the JNMA or issues relevant to minority healthcare.

Address correspondence to ktaylor@nmanet.org.