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Impact of Race/Ethnicity on Survival among HIV-Infected Patients in Care

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Abstract: **Objective**. To determine the prognostic influence of race/ethnicity on survival among patients infected with HIV infection. **Background**. In the U.S., HIV infection occurs disproportionately in minority communities. Additionally, worse outcomes (including higher mortality) have been reported, particularly among African Americans. **Methods**. This was a retrospective cohort study among 870 HIV-infected patients attending a Midwestern academic medical center. The study determined individual characteristics that were predictive of survival by using log rank tests and multivariate analysis models, after adjusting for known predictors of outcome. **Results**. Low CD4 cell count (<100 cells/mm^3), high viral load (>250,000 copies/mL), age older than 30, and Black race were independently predictive of poorer outcomes among patients infected with HIV. **Conclusion**. We found a large disparity in survival, with African Americans with advanced disease more likely to die than whites. This finding was not explained by socioeconomic status or other confounders. Future prospective studies are warranted.

Key words: HIV, AIDS, survival, race, ethnicity.

The influence of gender, race, and ethnicity on outcomes in people with HIV disease remains an area of active investigation as it may explain disparities in health care. A better understanding of this influence may guide health systems change and health care policy. In the U.S., prevalence of and mortality from HIV infection are known to be higher in minority communities than among non-Hispanic Whites, particularly among African Americans.

Representing only 14% of the U.S. population, Blacks account for 50% of the HIV-infected cases.[1] In the initial years of the AIDS epidemic, several studies showed that

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African Americans had higher mortality rates than Whites.[2,3] A meta-analysis conducted in 1993 suggested that this phenomenon was associated with decreased access to health care, along with other cultural and socioeconomic factors.[4,5] Although a few more recent studies have failed to show significant differences in outcomes with regard to race,[6–8] regression analysis designed to predict country-level mortality for Black men and the corresponding Black:White male mortality ratios in 140 counties between 1999–2002 showed that Black-White disparities widened significantly after the introduction of potent combination antiretroviral therapy.[9] This was observed among all age and gender groups, but particularly among women and the elderly. Supporting these data, White men were reported to experience an 85% decline in HIV-related mortality between 1995 and 2001, in comparison with only a 65% decline among Black men.[10] A cohort study conducted by the HIV Cost and Services Utilization Consortium consisting of three interviews among 2,864 respondents from January 1996 to January 1998 found that HIV care had the least favorable pattern of use among Black and Latino persons, even after statistically adjusting the model for CD4 cell count, age, sex, and exposure group.[11] Addition of insurance coverage to the model attenuated the disparities. However, even among patients with access to antiretroviral therapy, race has been associated with differences in antiretroviral therapy prescriptions or use of less potent combinations.[12]

To add to the current body of knowledge on racial disparities in HIV infection among patients in care, we conducted a retrospective study of patients attending an HIV clinic in a Midwestern academic medical center, with the aim of determining the prognostic influence of race/ethnicity on survival, after adjusting for known predictors of outcome.

Methods

This was a retrospective cohort study designed to examine the impact of race/ethnicity on HIV disease progression and mortality. The study was performed at the HIV clinic (*HIV Clinic*) of a Midwestern academic center. The HIV Clinic is Ryan White-funded and is the only dedicated HIV clinic in the region. Individuals included in this study were all patients attending the HIV Clinic at least once from September 1, 1997 until August 31, 2007. Follow-up of patients began with their first clinic visit after September 1, 1997 and ended with the subject's death or the patient's last visit before August 31, 2007. Patients who lacked any follow-up data were excluded (n=10). Data were abstracted from the clinic database and de-identified to protect patient confidentiality. Demographic variables of interest were race/ethnicity, gender, age, and income level. The categories for race/ethnicity were obtained by self-report, and are defined below:

Hispanic: A person of Mexican, Puerto Rican, Cuban, Central or South American, or other Spanish culture or origin, regardless of race.

White: A person having origins in any of the original racial groups of Europe, North Africa, or the Middle East.

Black: A person having origins in any of the Black racial groups of Africa.

Native American: A person and who maintains affiliation or community attachment to an indigenous American tribe.

Asian: A person having origins in any of the original peoples of the Far East, Southeast Asia, or the Indian Subcontinent (including, for example, origins in Cambodia, China, India, Japan, Korea, Malaysia, Pakistan, the Philippine Islands, Thailand, or Vietnam).

Native Hawaiian or Other Pacific Islander: A person having origins in any of the original peoples of Hawaii, Guam, Samoa, Micronesia, the Northern Marianas, or other Pacific islands.

For the analysis, patients were grouped into the racial/ethnic categories of White, Black, Hispanic, and other, which included Asian and Native American patients. There were no Pacific Islanders.

Income was recorded in terms of the federal poverty level. Data on income levels are recorded in the clinic database and updated regularly. Clinical variables in this study included CD4 cell count and HIV viral load at intake, and presence of comorbidities or opportunistic infections. Hyperlipidemia was defined as total cholesterol above 200 mg/dL, LDL cholesterol above 160 mg/dL, triglycerides above 200 mg/dL, or a patient receiving lipid-lowering therapy. Hypertension was defined as blood pressure repeatedly above 140/90 mmHg or a patient receiving treatment for hypertension. Chronic active hepatitis was defined as clinical evidence of chronic liver disease, persistent elevation of liver enzymes, or detectable viremia in the setting of serology suggestive of infection by hepatitis B or C virus. Depression was included if the diagnosis was listed in the patient's medical record. All other diagnoses were also abstracted from medical records.

Combination antiretroviral therapy was offered to all patients with indication for treatment either by fulfilling criteria suggested by contemporary guidelines[13] (the reference provided here includes links to all guidelines from the U.S. Department of Health and Human Services that have been used since 1998) or by clinician's recommendation.

Causes of death were as recorded in death certificates.

The study was approved by the local Institutional Review Board.

Differences in patient characteristics by race/ethnicity were analyzed using the chi-squared test for contingency tables. Estimates of survival distributions were calculated using the Kaplan Meier method, with 95% confidence intervals calculated using Greenwood's formula. Log-rank tests were used to determine which individual characteristics were predictive of survival. The Cox proportional hazards model was then used to assess the independent prognostic influence of various factors on survival. Because of the strong influence of intake values of CD4 cell count and viral burden on survival, the proportional hazards model was also used to assess factors independently predictive of survival within two risk subsets defined by intake CD4 cell count and viral burden (a lower risk category [CD4 cell count >100 cells/mm^3 and HIV viral load $<250{,}000$ copies/mL] and a higher risk category [all the other individuals]).

Finally, generalized estimating equations (GEE) were used to model the population mean of the HIV viral load (in logarithmic scale) and CD4 cell count (regular scale)

as a function of race/ethnicity and time since intake at the HIV Clinic and to compare these distributions at intake by race/ethnic group and whether these distributions changed over the follow-up period. All p-values quoted are 2-sided.

Results

A total of 870 patients were included in this study. The patient characteristics at the time of intake appear in Table 1. Seventy-three percent of patients were male and the median age was 36 years. Fifty-three percent were White, 29% Black, and 15% Hispanic. The median CD4 cell count at intake was 313 cells/mm^3 and the median HIV viral load was 71,136 copies/mL. Depression, hyperlipidemia, and hypertension were the most common comorbidities.

The following patient characteristics differed among racial/ethnic groups at the time of intake to the clinic: age; gender; income level; history of depression, hyperlipidemia, or hypertension; CD4 cell count; and HIV viral load (Table 2). White patients were more likely to be male, to be older, and to have depression and hyperlipidemia, than patients in other racial/ethnic groups. Black patients tended to be poorer and have more hypertension than other groups. Hispanic patients had more advanced HIV disease at presentation, with the lowest CD4 cell counts and the highest HIV viral loads.

HIV viral loads were lower in Whites than in Blacks or Hispanics at both 6–12 months and 12–18 months following intake at the HIV Clinic (p values: .02 and .03 respectively). HIV viral loads were higher for Hispanics than for Blacks and Whites at 4, 5, and 6 years following intake (p values .009, .02 and .02 respectively). No statistically significant differences were seen in other time periods.

Median CD4 cell counts were higher in Whites than in Blacks and Hispanics; the difference was largely maintained over the follow-up period, although the information available beyond seven years of follow-up is small (median for White, Black, and Hispanic: 259, 199, 164, respectively). However, mean CD4 counts were higher for Whites and Blacks than for Hispanics; this difference also was largely maintained during the follow-up period (mean for Whites, Blacks, and Hispanics: 352, 358, and 188, respectively).

A total of 76 individuals died during the study period, and the most common causes of death were AIDS-related (Table 3). Lymphoma, *Mycobacterium avium* complex, fungal infections and *Pneumocystis* pneumonia were the most common recorded causes of death. Twenty seven patients died from end-stage liver disease, cardiovascular disease, and other non-AIDS related conditions. The estimated five-year survival for the entire cohort of 870 patients was 90% (95% CI: 87–92%). Although the study encompassed ten years of activity at the HIV Clinic, we used a five-year survival estimate as this is most commonly used in medicine to report diseases with a short life expectancy. The median follow-up for individual patients known to be alive at last contact was 3.6 years. There was no difference in survival when individuals were classified according to their year of intake: 1997–2000 (n=281), 2001–2003 (n=263) and 2004–2007 (n=326), p=.94.

When individual characteristics were studied as predictors of survival using the log-rank test, age at intake, lower CD4 cell count, higher viral load, and a history of

Table 1.

INDIVIDUAL CHARACTERISTICS AT INTAKE TO THE HIV CLINIC 1997–2007

Characteristic	Number	Percentage
Age (years)		
<29	237	27
30–34	162	19
35–39	187	21
>40	284	33
Gender		
Male	639	73
Female	231	27
Race		
Native American	13	1
Asian	7	<1
Black African	91	10
Black African American	167	19
Hispanic	127	15
Other	6	<1
White	459	53
Comorbidities		
Depression	176	20
Diabetes mellitus	39	4
Hepatitis B	50	6
Hepatitis C	87	10
Hyperlipidemia	121	14
Hypertension	123	14
Neurological conditions	33	4
Psychiatric conditions other than depression	81	10
CD4 cell count in cells/mm^3[a]		
<99	185	21
100–299	232	27
300–499	222	26
>500	229	26
HIV viral load in copies/mL[b]		
<14,999	186	26
15,000–74,999	177	25
75,000–249,999	166	23
250,000	185	26

[a]Two patients were missing CD4 cell count values at intake.
[b]One hundred and fifty six patients were missing viral load values at intake.

Table 2.

SIGNIFICANTLY DIFFERENT CHARACTERISTICS AMONG DIFFERENT RACIAL/ETHNIC GROUPS

Characteristic	Race/ethnicity				p-value
	White	Black	Hispanics	Others	
Age older than 40	38%	26%	22%	17%	<0.0001
Male gender	83%	55%	76%	65%	<0.0001
Income level below the federal poverty level	43%	70%	57%	58%	<0.0001
Depression	27%	14%	9%	15%	<0.0001
Hyperlipidemia	19%	14%	6%	8%	<0.0001
Hypertension	13%	19%	9%	8%	0.016
CD4 cell count above 500 cells/mm3	30%	24%	16%	27%	0.0015
HIV RNA viral load below 15,000 copies/mL	26%	30%	18%	35%	0.016

Pneumocystis pneumonia were all associated with worse outcomes (p=.009, p<.0001, p=.0001 and p=.008 respectively). Surprisingly, hyperlipidemia was associated with better survival (100% survival if hyperlipidemia was present versus 88% if absent, p=.0006). The survival outcome by race/ethnicity was not significantly different, but results in Black patients were somewhat inferior (Figure 1).

The results of a multivariate analysis including 714 patients (this is the number out of the 870 initial individuals included in the study for whom there were CD4 and virus load data available at intake), 60 of whom died, showed that CD4 cell count below 100 cells/mm^3, HIV viral load above 250,000 copies/mL, Black race, and age older than 30 were independently predictive of outcome (Table 4). A history of *Pneumocystis* pneumonia and hyperlipidemia were not independent predictors.

When the patients in this study were grouped into a low risk category (CD4 cell count >100 cells/mm^3 and HIV viral load <250,000 copies/mL) and a high risk category (all the other individuals including those with CD4 count >100 cells/mm^3 and HIV viral load >250,000 copies/mL and patients with CD4 count <100 cells/mm^3, regardless of their viral load), the former had a 95% survival rate while the latter had a survival rate of 84% (p<.001). Interestingly, for neither the low or high risk groups was there evidence that the outcome differed among patients by CD4 cell count strata (100–300, 300–500 or more than 500) or by HIV viral burden (15,000, 15,000–75,000, 75,000–250,000 and >250,000 copies/mL), however in the small number of patients with CD4 count >500 cells/mm^3 and viral load <15,000 copies/mL (n=14), no deaths were observed.

Table 3.

CAUSES OF DEATH AMONG HIV-INFECTED PATIENTS

Cause of death	Number	Percentage
AIDS-related conditions		
Lymphoma	15	9.55
Mycobacterium avium complex	14	8.92
Fungal infection (Histoplasma or Cryptococcus)	8	5.10
Pneumocystis pneumonia	7	4.46
Others	35	22.29
Non AIDS-related conditions		
End-stage liver disease	18	11.46
Cardiovascular disease	7	4.46
Infections	5	3.18
Other	7	4.46
Unknown	41	26.11
Total	157	100.00

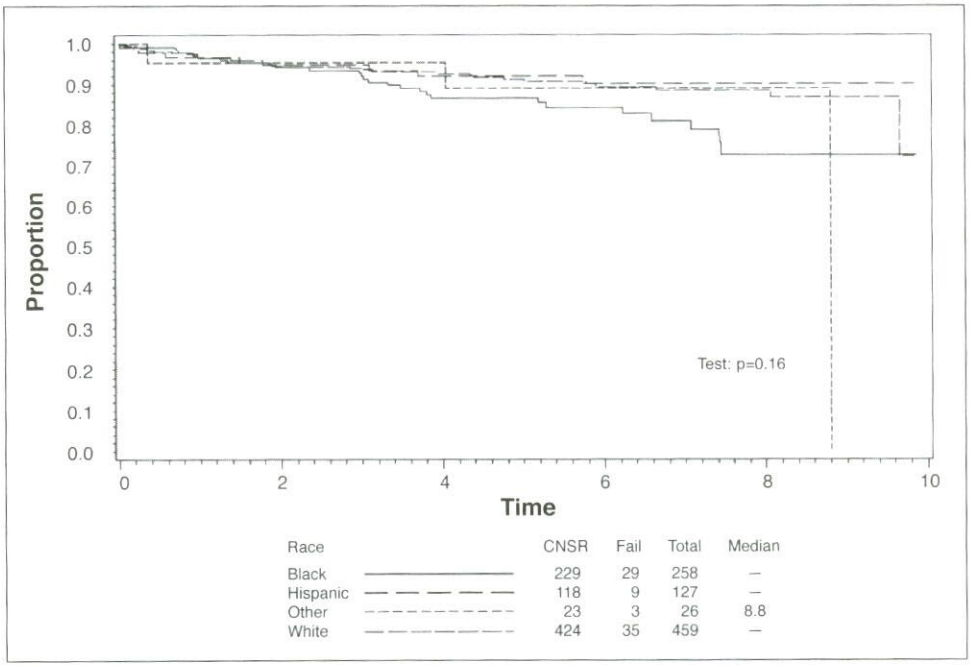

Figure 1. Survival of HIV-infected patients by race, 1997–2007.

Table 4.

INDEPENDENT PREDICTORS OF SURVIVAL OUTCOME BY MULTIVARIATE ANALYSIS

Characteristic	Hazard ratio (95% CI)	p-value
CD4 cell count <100	2.3 (1.31, 3.98)	0.003
HIV viral load >250,000	1.7 (0.98, 2.98)	0.060
Black race	1.8 (1.90, 3.11)	0.022
Age 30–34	2.5 (1.00, 6.05)	0.049
Age 35–39	3.0 (1.30, 6.93)	0.010
Age 40+	2.4 (1.07, 5.49)	0.035

CI = confidence interval

When the effect of race/ethnicity on survival was assessed, there was no evidence of a difference in survival by race/ethnicity for the low risk category (p=.35, see Figure A in the Appendix). However, for the high risk patients, survival was poorer among Black patients (73%), than among White (88%) or Hispanic (96%) patients (Figure 2).

The generalized estimating equations confirmed that White patients had statistically lower HIV viral loads at 6–12 months and at 12–18 months after intake (p=.02 and .03 respectively), and higher CD4 cell counts (maintained over the follow-up period), in comparison with Black and Hispanic patients (see Figures B, C, and D in the Appendix).

Discussion

This study examined the influence of race/ethnicity on the prognosis of HIV infection, while taking into consideration potential confounders such as socioeconomic status, presence of comorbidities, and stage of HIV infection. The main finding was that Black patients with advanced disease (CD4 cell count <100/mm^3 and/or HIV viral load >100,000 copies/mL) had poorer prognoses than other racial/ethnic groups, even after controlling for confounders.

As the earlier literature suggested racial disparity in HIV mortality whereas recent literature has not, we examined whether there was an interaction between racial disparity and time period. For this purpose we classified subjects as to when they were first seen at the HIV Clinic: 1997–2000, 2001–2003 and 2004–2007. We found no evidence of a different survival by "era" (p=.94). Other risk factors that influenced prognosis were not unexpected, including older age, lower CD4 cell counts and higher HIV viral loads.[14] Counterintuitively, hyperlipidemia was associated with better survival in the univariate analysis. As prolonged exposure to antiretroviral therapy is associated with

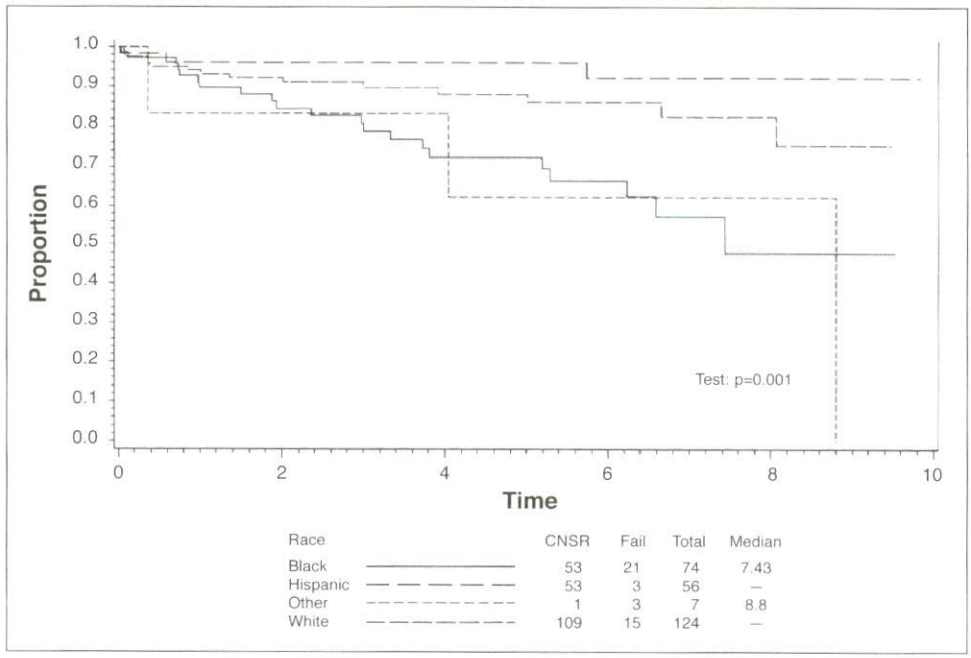

Figure 2. Survival of HIV-infected patients in the high risk group, 1997–2007.

hyperlipidemia,[15] this may represent a surrogate for adherence to antiretroviral therapy for patients receiving therapy prior to entering the cohort in September 1997.

Additionally, Hispanic patients as a group had the lowest initial CD4 cell counts and the highest HIV viral loads, but survival rates similar to White patients. This result is consistent with a previous report from our group, showing that despite presenting with more advanced disease, Hispanics had immunological recovery comparable to that of patients with less advanced disease at presentation.[5]

Although antiretroviral therapy (ART) has dramatically modified the prognosis of HIV infection,[16] survival differences among Black patients appear to persist. In a study conducted in New York from 1990 through 1999, mortality fell 63% (from 12.3 to 4.5 deaths per 100,000) among White men who have sex with men (MSM), but only 25% (from 66.2 to 49.5 per 100,000) among Black MSM.[17] In San Francisco, only 67% of African American patients survived six years after a diagnosis of AIDS, compared with 83% of Whites, among 5,007 individuals diagnosed with AIDS between 1996 and 2002.[10] A more recent study among 2,383 HIV-infected patients (U.S., 1999–2005) found a 10% survival difference (56% versus 66%) at mean follow-up of 5.9 years between Black and White patients.[18]

The reasons for survival differences in Blacks have been attributed to socioeconomic, behavioral or biological factors.[19] Also factors related to the health care delivery system, such as infrastructure, availability and quality of services and resources, and method of payment may impact the prognosis of Black patients. Black patients may be less likely to be started on antiretroviral therapy,[20] in part due to worse access to health

care.[21] Additionally, Black race may be a surrogate marker for socioeconomic status, which in turn is associated with worse AIDS-related mortality.[22] Substance abuse has been suggested as an explanation of worse prognosis among Black patients, but has not proven to be more common than among Whites.[23] High risk sexual behavior may increase risk of coinfections such as hepatitis B or C, or human papillomavirus which in turn may affect the prognosis and reduce the life-span of affected individuals. Several other behavioral factors such as the emotional disposition of patients, expectations regarding health outcomes, or the patients' trust in their health care providers have rarely been studied in patients with HIV infection and may be different among Blacks because of previous experiences with social, residential, and educational segregation.[24,25] Biological differences are also possible: the composite CCR5P1 haplotype, for example, is associated with rapid progression of the diseases in both Black and White patients, but it has a dominant effect in the former and a recessive in the latter.[26] Additionally, the cytochrome P450 2B6 gene (CYP2B6) may carry certain polymorphisms among Black patients that may be associated with different metabolic patterns, predisposing to longer half lives for efavirenz and potentially greater resistance to this drug.[27]

There are several limitations to this study, including the retrospective nature of the design and the fact that the study was conducted in a single clinic, which mean that results can not be widely generalized. In addition, we did not include alcohol or substance abuse in our analysis, as our database could not reliably differentiate between current or present, use and abuse. We used income per household (federal level of poverty) as a surrogate for poverty, but the inclusion of other characteristics such as employment, health, education, housing, and geographic access to services, may provide a better understanding of the degree of deprivation in this population Finally, it is possible that poorer results among Black patients may have been related to lack of acceptance or poor adherence to combination antiretroviral therapy. Although all patients were offered combination antiretroviral therapy if indicated, we did not have the means to track those who declined treatment, interrupted treatment for any reason, or were not fully adherent to treatment.

In summary, this retrospective study found that in a HIV clinic in the U.S. Midwest, Black patients with advanced disease (patients with CD4 counts >100 cells/mm^3 and HIV viral load $>250{,}000$ copies/mL and patients with CD4 counts <100 cells/mm^3, regardless of viral load) had shorter survival than their counterparts in other racial/ethnic groups. More research is needed into the biological, behavioral, sociocultural, and healthcare delivery reasons for persistent racial disparities in HIV infection, in order to better address them.

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Appendix

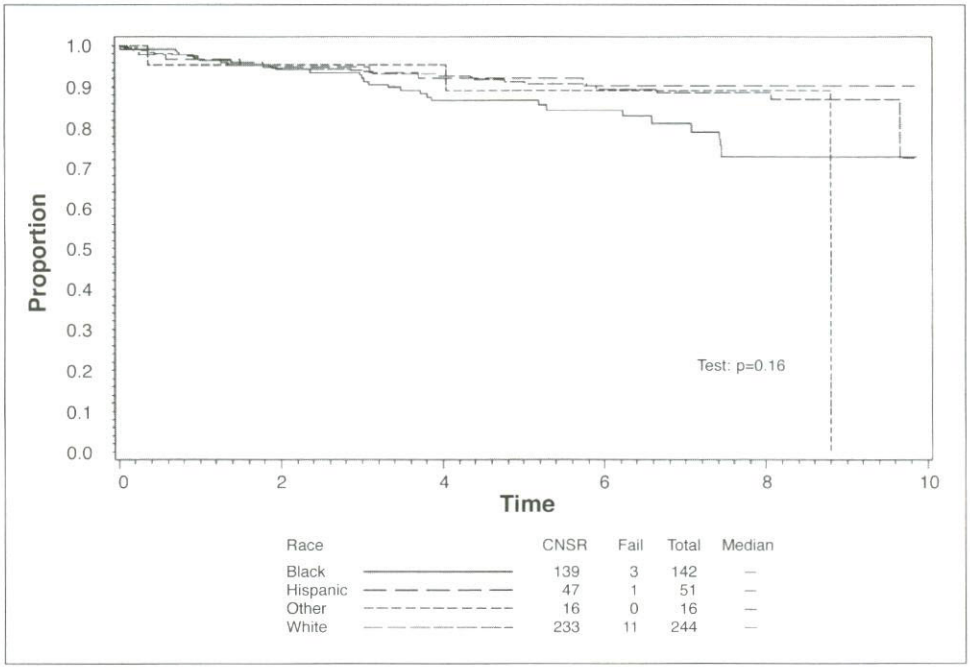

Figure A. Survival of HIV-infected patients in the low risk group, 1997–2007.

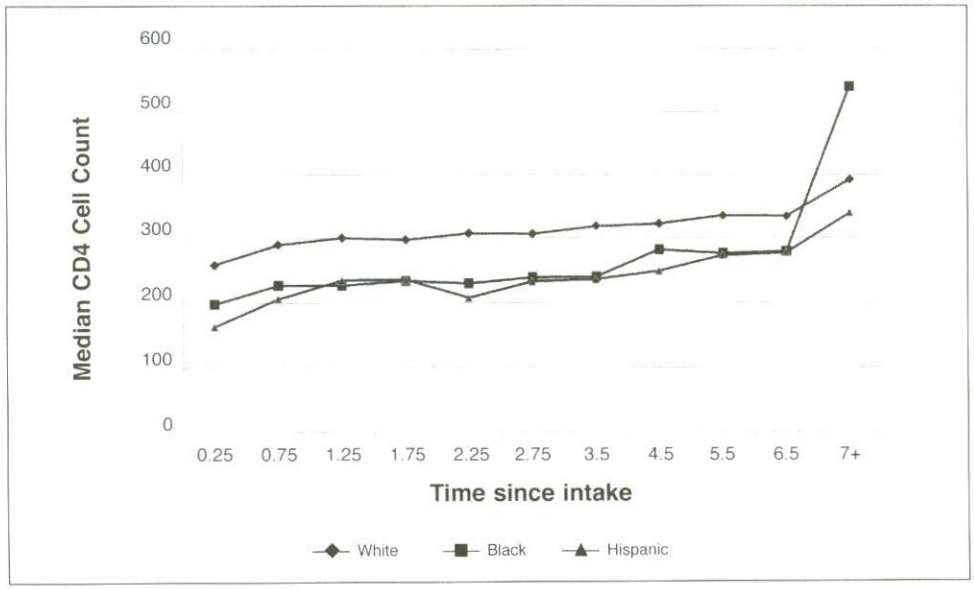

Figure B. Median CD4 cell count by race/ethnicity and time since patient intake at the HIV clinic.

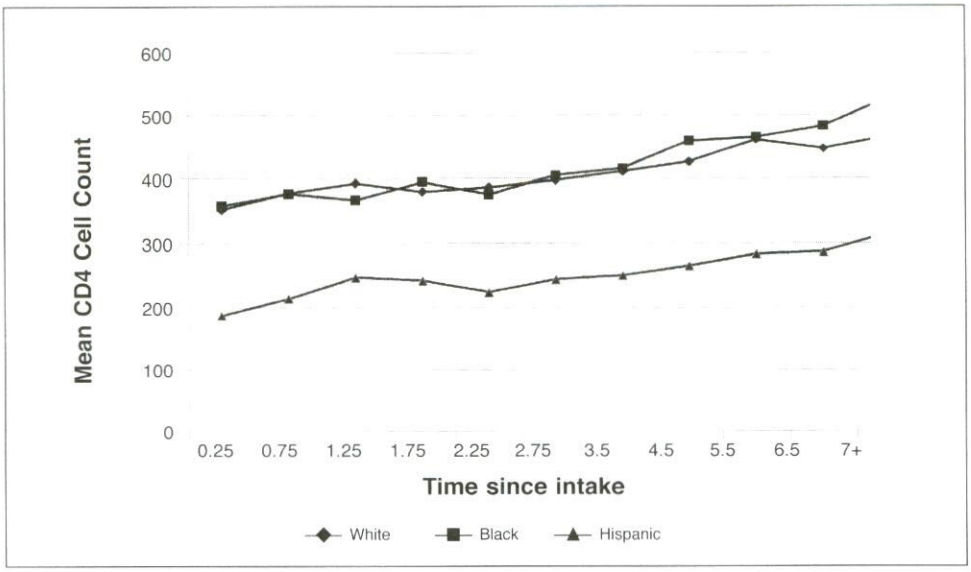

Figure C. Mean CD4 cell count by race/ethnicity and time since patient intake at the HIV clinic.

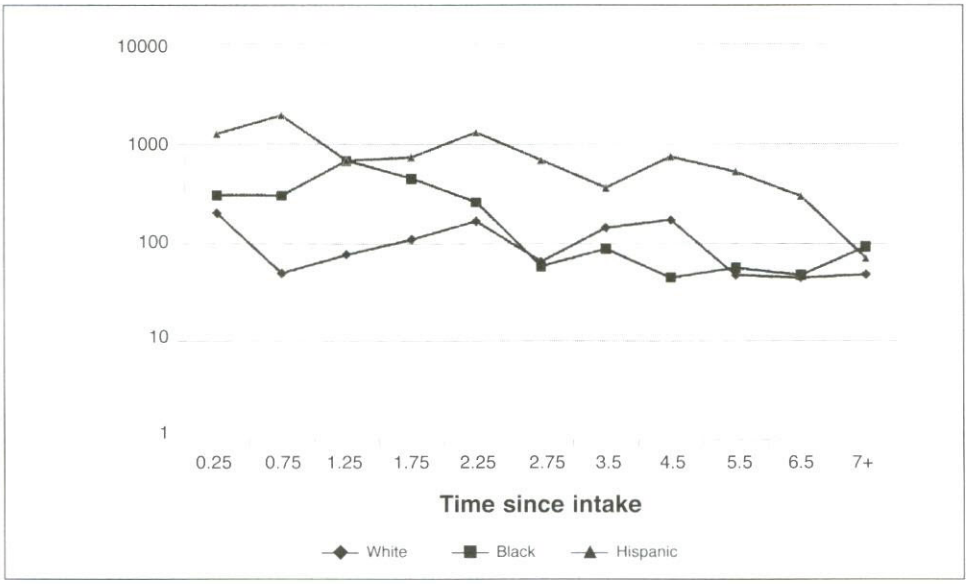

Figure D. Median HIV RNA viral load in logarithmic scale by race/ethnicity and time since patient intake at the HIV clinic.

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