Journal of Urban Health: Bulletin of the New York Academy of Medicine, Vol. 82, No. 4,
doi:10.1093/jurban/jti123
Published by Oxford University Press 2005.
Advance Access publication October 19, 2005

# Trends in Predictors of Death due to HIV-Related Causes Among Persons Living with AIDS in New York City: 1993–2001

Denis Nash*, Monica Katyal*, and Sarita Shah

**ABSTRACT** *To examine trends in predictors of HIV-related mortality among cohorts of persons living with AIDS (PLWA) in New York City (NYC), nine calendar year-specific cohorts of PLWA were created from 1993 to 2001. Cohorts were defined as persons who had been alive at any time during that year and had been diagnosed with AIDS before the end of that year. Predictors of death because of HIV-related causes of death were assessed by examining year-specific, stratified death rates per 1,000 PLWA and adjusted relative risks (RRs) from proportional hazards models. We conducted an analysis of AIDS surveillance data PLWA in NYC between 1993 and 2001. Univariate and multivariate Cox proportional hazards models were constructed for each calendar year cohort to evaluate trends in the RR of HIV-related death over the subsequent 5 years, adjusting for sex, race/ethnicity, age, transmission risk, borough of residence, category of AIDS diagnosis [opportunistic illness (OI) or CD4 count <200 cells/μL], time since AIDS diagnosis, and CD4 count at time of AIDS diagnosis. Death rates due to all causes and HIV-related causes declined substantially during 1993–1997 and then stabilized in all subgroups of PLWA between 1998 and 2001. Beginning in 1995, differences in survival emerged in some subgroups, such that by 2001 (1) injecting drug users (IDUs) had poorer survival compared with men who have sex with men (MSM) [$RR_{2001}$ = 2.1, 95% confidence intervals (95% CI) = 1.8–2.4]; (2) black and Hispanic PLWA had a significantly higher risk of death than white PLWA ($RR_{2001}$ = 1.4, 95% CI = 1.2–1.6, $RR_{2001}$ = 1.2, 95% CI = 1.1–1.4, respectively, and (3) PLWA aged 60 and above had poorer survival compared with younger persons ($RR_{2001}$ = 2.4, 95% CI = 1.9–3.0), after adjustment for other factors. The observed disparities that began to emerge in 1995 may be attributable to differential effects of, access to, or usage of highly active antiretroviral therapy (HAART). More targeted studies are needed to determine why such disparities have emerged.*

**KEYWORDS** *HAART, HIV, Mortality, Survival analysis, Trends.*

## INTRODUCTION

Many studies of survival among persons living with AIDS (PLWA) have focused on cohorts of persons diagnosed with AIDS during a defined calendar period, controlling for factors known to affect survival.[1,2] Others have examined relative survival time following AIDS diagnosis, controlling for stage of disease.[3,4] Such approaches

Dr. Nash and Ms. Katyal were with The New York City Department of Health and Mental Hygiene, HIV Epidemiology Program, 346 Broadway, New York, New York; and Dr. Shah was with the Division of General Medicine, Columbia University College of Physicians and Surgeons, New York, New York.

Correspondence: Denis Nash, PhD, MPH, Mailman School of Public Health, Columbia University, 722 W, 168th Street, 7th Floor, New York, NY 10032. (E-mail: dn2145@columbia.edu)

*Present address: Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, New York.

are useful for gaining insight into the natural history of disease progression. However, from a public health standpoint, it is critical to understand the factors that determine survival among all PLWA at a given point in time, despite the heterogeneity that exists with regard to the year of AIDS diagnosis, disease stage, and treatment experience. Furthermore, knowledge of how factors associated with survival among PLWA change temporally, demographically, and geographically can provide useful information on emerging issues to public health agencies for use in developing more effective prevention and treatment services, as well as in evaluating public health interventions aimed at reducing morbidity and mortality among PLWA.

With nearly 60,000 PLWA and over 5,000 new AIDS diagnoses each year, New York City (NYC) continues to be a major epicenter for HIV in the United States. NYC's HIV epidemic is continually evolving with regard to demographics and risk factors.[5–9] Despite dramatic declines in the number of deaths following the availability of highly active antiretroviral therapy (HAART) in 1995,[2,10–14] HIV remains the leading cause of death among New Yorkers aged 25–44 years and the third leading cause of death among non-Hispanic black and Hispanic New Yorkers of all ages.[15] The number of HIV-related deaths in NYC in 1998 was 1,563 and has not since declined appreciably, whereas the number of PLWA is increasing steadily.

To examine trends in the predictors of survival among PLWA, we used data from the HIV/AIDS Reporting System on nine successive annual cohorts of PLWA (1993–2001) in NYC.

## METHODS

Active and passive surveillance for AIDS has been in place in NYC since 1981. Data presented in this report reflect events occurring through December 31, 2002 and reported to the NYC Department of Health and Mental Hygiene through August 31, 2003.

### AIDS Case Definition

Persons were classified as having AIDS according to case definitions established by the Centers for Disease Control and Prevention.[16,17] HIV-infected persons with a documented Centers for Disease Control and Prevention AIDS-defining event [i.e., diagnosis of an opportunistic illness (OI) or CD4 count <200 cells/μL] were categorized as having been diagnosed with AIDS in the calendar year of the earliest known AIDS-defining event.[17] The completeness of AIDS case reporting in NYC was systematically evaluated immediately before the study period and found to be highly complete (85%–90%).[18]

### Study Population

The study population consisted of all PLWA who were alive at any time during 1993–2001 in NYC (n = 93,585). The following PLWA groups were excluded from analysis: (1) nonresident PLWA who were diagnosed in NYC (n = 4,188), (2) PLWA under age 13 at AIDS diagnosis (n = 1,322), (3) PLWA of Asian/Pacific Islander or other/unknown race/ethnicity (n = 1,079), (4) persons whose risk transmission category was transfusion-related or perinatal/presumed perinatal (n = 1,947) on the basis of insufficient sample size in any one calendar year, (5) PLWA surviving less than 30 days after AIDS diagnosis (n = 4,431) as they may represent previously diagnosed but unreported AIDS cases, and (6) PLWA with unknown vital status (n = 116). The final study population consisted of 82,243 PLWA.

*Year-Specific PLWA Cohorts* Nine calendar year-specific cohorts of PLWA were defined as persons who had been alive at any time during a given calendar year (1993–2001) and had been diagnosed with AIDS before the end of that year. The cohorts were not mutually exclusive, as PLWA in one calendar year were also members of cohorts in subsequent years until their death.

### Covariates

*Demographics* Information on sex, race/ethnicity, date of birth, zip code, and borough of residence at the time of HIV/AIDS diagnosis were collected for all reported cases.

*HIV Transmission Risk* Information was recorded on the likely mode of HIV transmission when available from the medical record or from diagnosing providers. Persons with multiple risks factors were categorized into one of the above groups based on the following hierarchy: parenteral [injecting drug user (IDU), pre-1985 transfusion], perinatal, and sexual [men who have sex with men (MSM), heterosexual]. Similar hierarchies have been used and discussed by other investigators.[19,20]

### HIV-Related Laboratory Data

Since 1993, all clinical laboratories have been required to report CD4 lymphocyte counts <200 cells/μL or <14% of the total lymphocyte count on NYC residents. Reporting of HIV-related laboratory tests was expanded in New York State in June 2000 to include positive HIV western blot tests, detectable HIV viral loads, and CD4 counts <500 cells/μL or <29% of the total lymphocyte count.[21]

*Stage of Disease at AIDS Diagnosis* Persons diagnosed with AIDS were classified as having been diagnosed on the basis of an AIDS-defining OI or immunologic status (CD4 < 200 cells/μL). The first CD4 count within 9 months of the AIDS diagnosis date was used as a marker for the stage of disease at diagnosis.

### Vital Status and Cause of Death

The vital status and underlying cause of death of persons reported to the HIV/AIDS Reporting System are updated through quarterly matches with the NYC Vital Statistics registry. Vital status for the study population was current as of December 31, 2002.

Routine (every 2–3 years) matching with the National Death Index is done to ascertain vital status and underlying cause of death on persons diagnosed in NYC who may have subsequently moved and/or died elsewhere in the United States. The last National Death Index match was conducted in March 2002 for deaths through 1999. Deaths were classified as being due to HIV/AIDS or other causes using the International Classification of Diseases, 9th Revision (for deaths in 1993–1999) and 10th Revision (for deaths in 2000–2001).

### Statistical Methods

For each year-specific cohort, the time-to-event variable was number of years from beginning of a given calendar year (or date of diagnosis if this occurred during that year) to death from an HIV-related cause. Persons who died from causes other than HIV were censored as of their date of death. To be consistent with previous investigations[22] and due to concern about incompleteness of vital status among long-term survivors, persons surviving more than 5 years following AIDS diagnosis were right censored. Cox proportional hazards models were constructed for each

cohort to evaluate trends in predictors of mortality over time. Models included sex, race/ethnicity, age at AIDS diagnosis, transmission risk category, borough of residence, stage of disease at AIDS diagnosis, time since AIDS diagnosis, and first CD4 count following AIDS diagnosis. Thus, the maximum follow-up for persons in each cohort ranged from 2 years (2001 cohort) to 5 years (1993–1998 cohorts). The overall median follow-up time was 3.7 years (range, 0.003–5.0). The proportional hazards assumption was tested and met for each year-specific cohort. Statistical analyzes were carried out using SAS software (Version 8.0, Cary, North Carolina).

## RESULTS

### Epidemiology of PLWA in NYC, 1993–2001

The number of AIDS diagnoses in NYC peaked at 12,658 in 1993. In 1993, 27,381 PLWA were reported. By 2001, this number had grown by 78% to 48,742 PLWA, including 5,199 new AIDS diagnoses. Characteristics of each year-specific cohort of PLWA are shown in Table 1. With each successive cohort, females made up a growing proportion of PLWA (1993, 24%; 2001, 29%; $P < .05$). In all cohorts, most of the PLWA were black and Hispanic, with whites accounting for a decreasing proportion over time. The median age at time of AIDS diagnosis was 37 years in 1993 and did not change appreciably over the study period. IDU PLWA remained the largest transmission risk group over the entire period (1993, 53.1%; 2001, 36.6%). However, heterosexuals and PLWA with unknown risk factor information comprised a growing proportion of each successive cohort ($1993_{\text{heterosexual}}$, 11.4%; $2001_{\text{heterosexual}}$, 20.2%; $1993_{\text{unknown}}$, 4.5%;

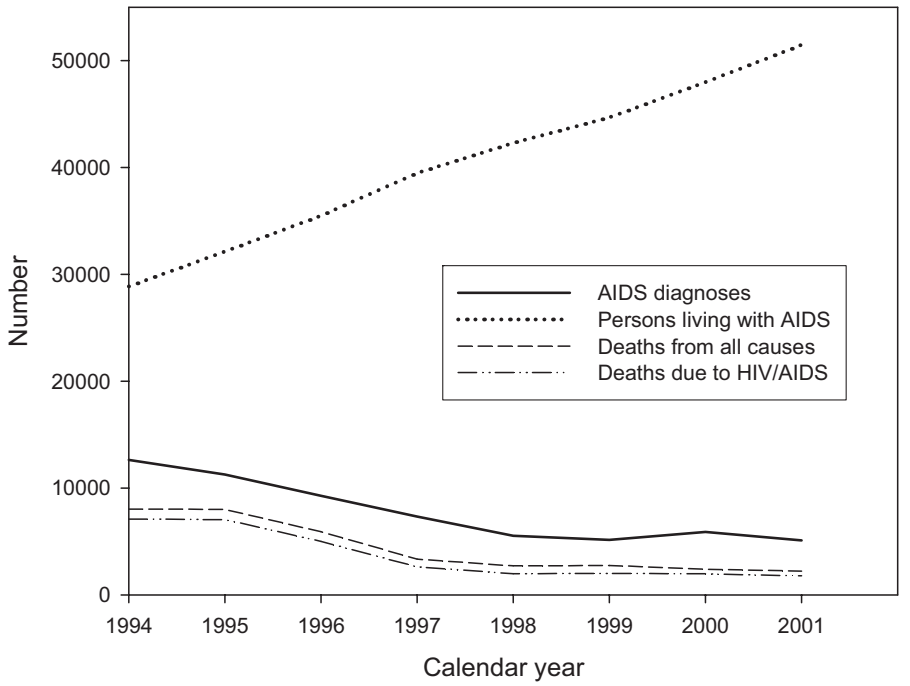


**FIGURE 1.** AIDS diagnoses, persons living with AIDS (PLWA), and deaths among PLWA from all causes and deaths due to HIV/AIDS, New York City (NYC) 1994–2001.

**TABLE 1. Descriptive characteristics of persons living with AIDS (PLWA) by calendar-year cohort, New York City (NYC), 1993–2001**

| | 1993 [N = 27,381 (%)] | 1994 [N = 32,554 (%)] | 1995 [N = 35,653 (%)] | 1996 [N = 37,039 (%)] | 1997 [N = 38,615 (%)] | 1998 [N = 40,740 (%)] | 1999 [N = 43,065 (%)] | 2000 [N = 46,233 (%)] | 2001 [N = 48,742 (%)] |
|---|---|---|---|---|---|---|---|---|---|
| Sex | | | | | | | | | |
| Male | 76.3 | 75.4 | 74.7 | 73.8 | 73.0 | 72.4 | 72.1 | 71.7 | 71.3 |
| Female | 23.7 | 24.6 | 25.3 | 26.2 | 27.0 | 27.6 | 27.9 | 28.3 | 28.7 |
| Race/ethnicity | | | | | | | | | |
| Black | 42.1 | 42.3 | 42.3 | 43.3 | 43.9 | 44.1 | 44.3 | 44.9 | 45.4 |
| Hispanic | 32.5 | 33.6 | 34.2 | 34.4 | 34.5 | 34.8 | 34.9 | 34.6 | 34.4 |
| White | 25.4 | 24.1 | 23.5 | 22.3 | 21.6 | 21.2 | 20.8 | 20.5 | 20.2 |
| Age at AIDS diagnosis | | | | | | | | | |
| 13–19 | 0.4 | 0.4 | 0.4 | 0.4 | 0.5 | 0.5 | 0.5 | 0.6 | 0.6 |
| 20–29 | 14.8 | 14.3 | 13.9 | 13.7 | 13.6 | 13.4 | 13.2 | 12.9 | 12.8 |
| 30–39 | 45.2 | 44.6 | 44.2 | 43.9 | 43.4 | 43.2 | 43.0 | 42.4 | 42.0 |
| 40–49 | 29.6 | 30.3 | 31.3 | 31.6 | 31.7 | 31.6 | 31.6 | 32.0 | 32.1 |
| 50–59 | 7.9 | 8.1 | 8.0 | 8.3 | 8.6 | 9.0 | 9.0 | 9.4 | 9.8 |
| 60+ | 2.2 | 2.3 | 2.2 | 2.2 | 2.3 | 2.4 | 2.6 | 2.7 | 2.7 |
| Transmission risk | | | | | | | | | |
| IDU | 53.1 | 51.5 | 49.2 | 47.0 | 44.8 | 42.9 | 41.3 | 38.9 | 36.6 |
| MSM | 31.0 | 29.8 | 28.3 | 26.8 | 26.0 | 25.7 | 25.5 | 25.3 | 25.2 |
| Heterosexual | 11.4 | 13.2 | 15.3 | 17.0 | 18.3 | 19.4 | 20.0 | 20.2 | 20.2 |
| Unknown | 4.5 | 5.5 | 7.2 | 9.2 | 11.0 | 11.9 | 13.1 | 15.6 | 18.1 |
| Borough of residence* | | | | | | | | | |
| Manhattan | 39.3 | 38.0 | 37.3 | 36.4 | 35.8 | 34.9 | 34.5 | 34.3 | 33.9 |
| Brooklyn | 24.9 | 25.6 | 26.1 | 26.3 | 26.2 | 26.4 | 26.4 | 26.8 | 26.9 |
| Bronx | 19.8 | 20.0 | 20.3 | 20.8 | 21.5 | 22.1 | 22.4 | 22.2 | 22.5 |
| Queens | 13.9 | 14.1 | 14.2 | 14.3 | 14.4 | 14.7 | 14.7 | 14.7 | 14.8 |
| Staten Island | 2.2 | 2.2 | 2.2 | 2.1 | 2.0 | 2.0 | 1.9 | 2.0 | 2.0 |

**TABLE 1.** ***Continued***

| | 1993 [N = 27,381 (%)] | 1994 [N = 32,554 (%)] | 1995 [N = 35,653 (%)] | 1996 [N = 37,039 (%)] | 1997 [N = 38,615 (%)] | 1998 [N = 40,740 (%)] | 1999 [N = 43,065 (%)] | 2000 [N = 46,233 (%)] | 2001 [N =48,742 (%)] |
|---|---|---|---|---|---|---|---|---|---|
| Category of AIDS diagnosis† | | | | | | | | | |
| Opportunistic illness | 70.4 | 59.5 | 52.8 | 47.2 | 43.4 | 41.9 | 40.6 | 37.6 | 34.9 |
| CD4 <200 cells/μL or <14% | 29.6 | 40.5 | 47.2 | 52.8 | 56.6 | 58.1 | 59.4 | 62.4 | 65.1 |
| Time since AIDS diagnosis (years) | | | | | | | | | |
| Median (range) | 5.0 (0.003–5.0) | 5.0 (0.003–5.0) | 5.0 (0.003–5.0) | 5.0 (0.003–5.0) | 5.0 (0.003–5.0) | 5.0 (0.003–5.0) | 4.0 (0.003–4.0) | 3.0 (0.003–3.0) | 2.0 (0.003–2.0) |
| CD4 at AIDS diagnosis‡ | | | | | | | | | |
| Missing | 52.3 | 40.1 | 34.3 | 30.6 | 28.5 | 27.3 | 26.1 | 25.1 | 24.8 |
| 0–49 | 17.7 | 21.0 | 21.8 | 20.4 | 19.3 | 19.2 | 19.3 | 20.0 | 20.3 |
| 50–99 | 8.5 | 10.4 | 11.0 | 11.2 | 11.3 | 11.5 | 11.5 | 11.5 | 11.3 |
| 100–149 | 7.9 | 10.1 | 11.3 | 12.4 | 13.1 | 13.3 | 13.7 | 13.8 | 13.7 |
| 150–199 | 10.5 | 13.7 | 15.5 | 17.6 | 18.8 | 19.3 | 19.9 | 20.3 | 20.6 |
| 200+ | 3.2 | 4.6 | 6.2 | 7.8 | 9.0 | 9.4 | 9.5 | 9.4 | 9.3 |

IDUs, injecting drug users; MSM, men who have sex with men.

*Borough of patient residence at the time of AIDS diagnosis.

†Based on the 1993 AIDS case definition of the Centers for Disease Control and Prevention.

‡Within 9 months of AIDS diagnosis.

$2001_{unknown}$, 18.1%). Median CD4 count at time of AIDS diagnosis increased from 85 cells/μL among those with available data (48% of PLWA) in 1993 to 122 in 2001 (75%).

### Trends in Mortality Rates Among PLWA

The number of deaths from all causes among PLWA peaked in 1994 at 8,043 and decreased by 66% to 2,707 in 1998 (Fig. 1). Most of the decline occurred in HIV-related deaths, following the introduction of HAART. This downward trend slowed by 1998 and has not changed appreciably since that time. Table 2 summarizes the annual mortality rate per 1,000 PLWA for all and HIV only-related causes among each year-specific cohort. Both all-cause and HIV-related mortality rates decreased from 1993 to 2001, with a sharp decline beginning in 1995. HIV-related death rates declined at a similar rate for males and females (Fig. 2a) and for each race/ethnicity category (Fig. 2b) over time. Declines in HIV-related mortality rates occurred in every age group, most notably after 1995 (Fig. 2c), but PLWA aged 60+ had consistently higher death rates than younger PLWA. In 1993, MSM PLWA had higher death rates per 1,000 PLWA than IDU and heterosexual PLWA, for both all-cause and HIV-related mortality. Although HIV-related death rates subsequently declined in all three transmission risk groups, the decline was greatest among MSM (Fig. 2d). Early disparities in mortality rates based on AIDS diagnosis category converged over time such that there were no apparent differences in death rates by 2001 (Fig. 2f).

### Trends in the Adjusted Relative Hazards for Death due to HIV-Related Causes

Table 3 summarizes cohort-specific adjusted relative risks (ARR) for HIV-related death among selected subgroups. Females were at significantly increased risk compared with males over the latter half of the study period ($ARR_{1994} = 1.1$, $ARR_{2001} = 1.2$) (Fig. 3a). Beginning in 1996, black and Hispanic PLWA had a significantly higher risk of death than white PLWA, respectively ($ARR_{2001} = 1.4$, $ARR_{2001} = 1.1$) and these differences increased with time (Fig. 3b).

Compared to PLWA aged 20–29 years, PLWA in older age groups were at significantly increased risk of death. This difference remained relatively steady over time (Fig. 3c), except among PLWA aged 60+ whose risk appeared to increase ($ARR_{1993} = 1.7$, $ARR_{2001} = 2.4$).

Transmission risk category was also a significant predictor of survival, with emerging disparities beginning in 1996 and growing over time (Fig. 3d). Compared with MSM PLWA, IDU, heterosexual, and PLWA with unknown transmission risk had poorer survival even after covariate adjustment. For example, in 1995, survival experiences among IDU PLWA were similar to MSM PLWA ($ARR_{1995} = 1.1$). However, by 2001, IDU PLWA were twice as likely to die ($ARR_{2001} = 2.1$) when compared with MSM.

PLWA diagnosed with AIDS by OI had consistently poorer survival from 1993 to 2001 compared with those diagnosed by immunologic criteria ($ARR_{1993} = 1.7$, $ARR_{2001} = 1.4$) (Fig. 3f). CD4 count at time of AIDS diagnosis was an important predictor of survival in 1993. However, though still statistically significant, differences in survival by initial CD4 count diminished with time.

Increasing time since AIDS diagnosis was consistently a significant predictor of survival. Before 1997, persons diagnosed with AIDS 1– <4 years prior had significantly poorer survival than persons diagnosed within 1 year. After 1997, PLWA who had been diagnosed for more than one year tended to have significantly better survival experiences than those diagnosed within the year. In each cohort, PLWA

**TABLE 2. Death rates from all causes and from HIV-related causes per 1,000 persons living with AIDS (PLWA) by calendar-year cohort, New York City (NYC), 1993–2001**

| | Calendar year | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1993 (N = 27,381) | | 1994 (N = 32,554) | | 1995 (N = 35,653) | | 1996 (N = 37,039) | | 1997 (N = 38,615) | | 1998 (N = 40,740) | | 1999 (N = 43,065) | | 2000 (N = 46,233) | | 2001 (N =48,742) | |
| | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes |
| Sex | | | | | | | | | | | | | | | | | | |
| Male | 199.1 | 171.6 | 187.3 | 169.7 | 175.9 | 161.0 | 121.5 | 108.0 | 64.5 | 52.7 | 50.0 | 36.5 | 47.1 | 35.5 | 41.8 | 31.4 | 38.5 | 26.9 |
| Female | 179.1 | 153.2 | 202.7 | 180.2 | 176.8 | 161.6 | 131.9 | 116.1 | 74.9 | 60.5 | 56.2 | 43.2 | 56.1 | 43.3 | 49.8 | 37.8 | 46.7 | 35.1 |
| Race/ethnicity | | | | | | | | | | | | | | | | | | |
| Black | 195.8 | 165.7 | 206.9 | 186.5 | 184.9 | 167.8 | 137.5 | 120.2 | 80.0 | 64.7 | 60.9 | 46.1 | 58.1 | 44.6 | 51.4 | 39.7 | 46.9 | 34.4 |
| Hispanic | 194.4 | 168.5 | 184.8 | 165.4 | 180.6 | 165.8 | 122.1 | 111.3 | 62.2 | 52.5 | 48.6 | 35.5 | 48.9 | 37.7 | 42.4 | 32.0 | 40.5 | 28.8 |
| White | 192.0 | 148.8 | 172.0 | 157.0 | 153.9 | 142.6 | 101.5 | 88.8 | 49.8 | 38.4 | 37.8 | 27.0 | 32.6 | 22.8 | 30.8 | 21.2 | 27.7 | 18.5 |
| Age at death | | | | | | | | | | | | | | | | | | |
| 13–19 | 47.6 | 38.1 | 66.1 | 57.9 | 31.0 | 31.0 | 13.1 | 6.5 | 16.3 | 10.9 | 0.0 | 0.0 | 12.9 | 8.6 | 3.8 | 3.8 | 6.9 | 6.9 |
| 20–29 | 101.4 | 92.3 | 92.1 | 85.9 | 78.5 | 75.1 | 47.1 | 42.7 | 22.7 | 21.0 | 14.3 | 12.7 | 13.0 | 10.9 | 9.5 | 8.5 | 7.9 | 6.6 |
| 30–39 | 174.1 | 153.3 | 166.2 | 152.4 | 152.5 | 143.1 | 97.4 | 89.3 | 48.4 | 42.0 | 34.3 | 27.4 | 27.6 | 22.9 | 24.2 | 20.4 | 21.3 | 17.1 |
| 40–49 | 239.5 | 203.6 | 235.9 | 212.4 | 218.6 | 197.6 | 166.5 | 145.9 | 91.9 | 72.9 | 70.6 | 52.7 | 69.3 | 52.3 | 60.1 | 45.5 | 51.9 | 38.3 |
| 50–59 | 279.5 | 229.5 | 294.7 | 259.2 | 267.5 | 234.4 | 204.5 | 176.4 | 112.9 | 86.6 | 100.3 | 66.3 | 113.1 | 80.7 | 100.8 | 67.3 | 105.4 | 67.1 |
| 60+ | 345.8 | 263.0 | 358.3 | 282.0 | 354.6 | 304.5 | 249.7 | 203.0 | 191.8 | 138.1 | 157.1 | 93.4 | 146.6 | 97.7 | 143.0 | 94.5 | 142.8 | 85.9 |
| Transmission risk | | | | | | | | | | | | | | | | | | |
| IDU | 201.8 | 167.9 | 204.3 | 180.6 | 187.4 | 168.7 | 140.5 | 122.7 | 80.9 | 62.5 | 63.8 | 45.5 | 63.6 | 47.1 | 59.4 | 43.8 | 55.5 | 37.9 |
| MSM | 199.8 | 182.2 | 180.0 | 169.1 | 167.9 | 156.3 | 104.9 | 96.4 | 47.6 | 41.0 | 33.9 | 26.5 | 30.0 | 23.4 | 25.3 | 19.8 | 22.8 | 15.8 |
| Heterosexual | 160.9 | 141.1 | 185.3 | 168.8 | 171.4 | 160.3 | 121.3 | 109.6 | 65.9 | 57.0 | 53.4 | 42.7 | 49.0 | 39.3 | 44.4 | 35.3 | 39.7 | 30.9 |
| Unknown | 154.3 | 122.7 | 140.6 | 119.3 | 141.6 | 130.8 | 102.5 | 87.2 | 61.0 | 52.2 | 44.0 | 31.4 | 44.4 | 33.1 | 35.9 | 25.9 | 37.7 | 28.6 |
| Borough of residence* | | | | | | | | | | | | | | | | | | |
| Manhattan | 189.0 | 165.4 | 176.7 | 159.2 | 164.1 | 149.4 | 108.7 | 97.9 | 61.5 | 51.8 | 45.0 | 33.6 | 42.6 | 31.8 | 38.8 | 28.5 | 35.0 | 25.3 |
| Brooklyn | 196.4 | 166.1 | 202.9 | 183.8 | 186.6 | 172.3 | 135.1 | 119.0 | 78.9 | 64.5 | 58.2 | 43.6 | 55.9 | 41.6 | 52.5 | 41.0 | 45.5 | 31.7 |

**TABLE 2.** ***Continued***

| | Calendar year | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1993 (N = 27,381) | | 1994 (N = 32,554) | | 1995 (N = 35,653) | | 1996 (N = 37,039) | | 1997 (N = 38,615) | | 1998 (N = 40,740) | | 1999 (N = 43,065) | | 2000 (N = 46,233) | | 2001 (N =48,742) | |
| | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes | All causes | HIV-related causes |
| Bronx | 201.1 | 171.2 | 200.6 | 178.5 | 190.8 | 173.8 | 139.7 | 124.4 | 70.5 | 56.4 | 58.2 | 43.5 | 57.9 | 45.6 | 49.6 | 37.3 | 49.1 | 36.1 |
| Queens | 191.7 | 163.7 | 189.0 | 171.9 | 163.6 | 149.7 | 116.7 | 101.3 | 55.5 | 43.1 | 44.9 | 32.1 | 42.7 | 32.8 | 32.7 | 24.0 | 32.4 | 23.2 |
| Staten Island | 225.2 | 200.0 | 229.4 | 208.8 | 202.3 | 188.1 | 154.4 | 131.3 | 72.6 | 49.3 | 63.0 | 40.7 | 42.2 | 34.9 | 43.7 | 32.8 | 47.4 | 29.9 |
| Category of AIDS diagnosis† | | | | | | | | | | | | | | | | | | |
| Opportunistic illness | 230.2 | 201.0 | 213.1 | 194.2 | 198.8 | 183.7 | 142.0 | 127.3 | 74.8 | 62.9 | 56.6 | 43.5 | 55.9 | 42.9 | 47.3 | 36.2 | 39.7 | 30.2 |
| CD4 <200 cells/μL or <14% | 109.4 | 87.1 | 158.7 | 140.1 | 150.8 | 136.0 | 108.3 | 94.9 | 61.6 | 48.6 | 48.2 | 34.7 | 45.3 | 34.1 | 42.2 | 31.4 | 41.5 | 28.7 |
| Earliest CD4 after AIDS diagnosis‡ | | | | | | | | | | | | | | | | | | |
| Missing | 223.1 | 193.2 | 184.7 | 165.4 | 150.3 | 134.2 | 88.5 | 76.3 | 46.1 | 34.3 | 43.1 | 30.8 | 41.1 | 29.2 | 34.8 | 23.7 | 29.3 | 19.9 |
| 0–49 | 252.4 | 223.9 | 293.6 | 274.3 | 293.2 | 277.3 | 223.3 | 208.6 | 108.9 | 96.9 | 78.9 | 66.2 | 71.2 | 61.5 | 55.0 | 45.6 | 49.9 | 38.4 |
| 50–99 | 173.0 | 145.9 | 232.1 | 206.5 | 208.3 | 194.7 | 166.9 | 146.0 | 92.3 | 77.4 | 62.8 | 47.8 | 53.9 | 42.4 | 55.5 | 44.6 | 50.5 | 38.9 |
| 100–149 | 110.2 | 88.0 | 144.6 | 129.0 | 153.5 | 136.3 | 112.0 | 97.0 | 66.5 | 52.1 | 53.3 | 36.3 | 51.1 | 37.7 | 47.5 | 34.3 | 45.6 | 31.8 |
| 150–199 | 72.9 | 54.7 | 101.1 | 84.2 | 105.7 | 94.1 | 85.8 | 72.0 | 55.6 | 44.1 | 39.1 | 26.4 | 43.3 | 30.0 | 39.9 | 28.9 | 41.7 | 28.8 |
| 200+ | 67.1 | 51.2 | 55.9 | 45.9 | 67.4 | 55.6 | 48.4 | 39.2 | 39.7 | 27.0 | 31.6 | 19.6 | 34.7 | 23.1 | 35.7 | 26.0 | 31.4 | 20.0 |
| Time since AIDS diagnosis (years) | | | | | | | | | | | | | | | | | | |
| <1 | 127.7 | 106.7 | 116.9 | 103.7 | 115.1 | 104.3 | 81.1 | 68.0 | 60.2 | 48.4 | 52.5 | 40.0 | 57.5 | 44.8 | 41.4 | 31.6 | 42.9 | 31.7 |
| 1–<2 | 278.8 | 243.3 | 268.3 | 241.2 | 230.6 | 214.3 | 163.7 | 146.6 | 79.5 | 63.9 | 62.7 | 46.0 | 65.5 | 49.0 | 62.6 | 49.0 | 57.4 | 44.0 |
| 2–<3 | 263.9 | 230.4 | 261.6 | 239.2 | 242.2 | 221.9 | 160.9 | 146.1 | 78.2 | 66.8 | 54.1 | 39.4 | 54.6 | 41.8 | 49.8 | 36.6 | 39.3 | 28.5 |
| 3–<4 | 212.2 | 184.4 | 207.3 | 189.5 | 188.4 | 171.6 | 137.8 | 124.3 | 77.5 | 62.7 | 59.2 | 45.0 | 52.0 | 40.6 | 47.5 | 36.8 | 44.1 | 31.5 |
| 5+ | 97.0 | 79.3 | 79.7 | 70.6 | 73.2 | 62.6 | 60.8 | 50.0 | 39.2 | 30.7 | 36.1 | 25.8 | 38.7 | 28.4 | 37.8 | 27.6 | 36.1 | 25.0 |

IDUs, injecting drug users; MSM, men who have sex with men.

*Borough of patient residence at the time of AIDS diagnosis.

†Based on the 1993 AIDS case definition of the Centers for Disease Control and Prevention.

‡Within 9 months of AIDS diagnosis.

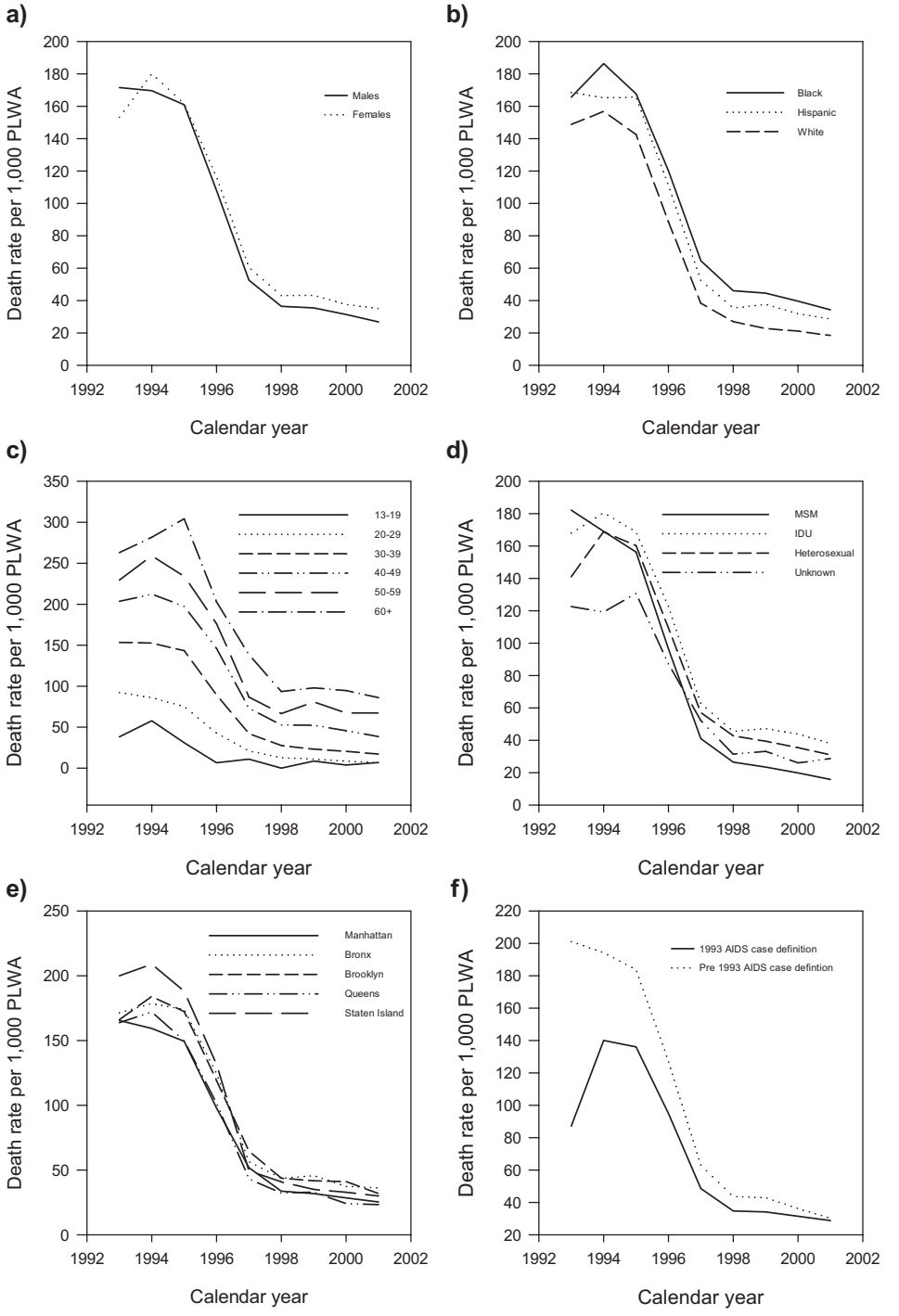


**FIGURE 2.** Mortality among persons living with AIDS (PLWA) due to HIV-related causes by (**a**) sex, (**b**) race/ethnicity, (**c**) age, (**d**) transmission risk, (**e**) borough of patient residence, and (**f**) AIDS diagnosis category, 1993–2001, New York City (NYC).

**TABLE 3. Adjusted relative risks [95% confidence intervals (95% CI)] for death from HIV-related causes among persons living with AIDS (PLWA) in each calendar year cohort, New York City (NYC), 1993–2001**

| | Adjusted relative risks (95% CI) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 | 2001 |
| Sex | | | | | | | | | |
| Male (referent) | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| Female | 1.1 (1–1.1) | 1.1 (1.1–1.2) | 1.1 (1.1–1.2) | 1.2 (1.1–1.2) | 1.2 (1.1–1.2) | 1.2 (1.1–1.3) | 1.2 (1.1–1.2) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) |
| Race/ethnicity | | | | | | | | | |
| Black | 1 (0.99–1.1) | 1.1 (1–1.1) | 1.1 (1.1–1.2) | 1.3 (1.2–1.4) | 1.4 (1.3–1.5) | 1.4 (1.3–1.5) | 1.4 (1.3–1.5) | 1.4 (1.3–1.5) | 1.4 (1.2–1.6) |
| Hispanic | 1 (0.98–1.1) | 1 (0.98–1.1) | 1.1 (1–1.1) | 1.1 (1.1–1.2) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.4) | 1.2 (1.1–1.3) | 1.2 (1.1–1.4) |
| White | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| Age at AIDS diagnosis | | | | | | | | | |
| 13–19 | 0.8 (0.58–1.1) | 0.77 (0.55–1.1) | 0.7 (0.48–1) | 0.93 (0.63–1.4) | 1.1 (0.75–1.6) | 1.1 (0.71–1.6) | 1 (0.67–1.6) | 0.82 (0.48–1.4) | 0.69 (0.34–1.4) |
| 20–29 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| 30–39 | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1–1.3) | 1.1 (1–1.3) | 1.2 (1.1–1.4) |
| 40–49 | 1.3 (1.2–1.4) | 1.3 (1.2–1.4) | 1.3 (1.3–1.4) | 1.4 (1.3–1.5) | 1.4 (1.3–1.5) | 1.4 (1.2–1.5) | 1.4 (1.3–1.6) | 1.4 (1.2–1.5) | 1.4 (1.2–1.7) |
| 50–59 | 1.5 (1.4–1.6) | 1.6 (1.5–1.7) | 1.5 (1.4–1.6) | 1.6 (1.4–1.7) | 1.5 (1.4–1.7) | 1.6 (1.4–1.8) | 1.5 (1.4–1.8) | 1.5 (1.3–1.8) | 1.7 (1.4–2) |
| 60+ | 1.7 (1.6–2) | 1.8 (1.6–2.1) | 1.9 (1.7–2.2) | 2.2 (1.9–2.5) | 2.5 (2.1–2.9) | 2.3 (2–2.7) | 2.5 (2.1–3) | 2.5 (2–2.9) | 2.4 (1.9–3) |
| Transmission risk | | | | | | | | | |
| IDU | 0.91 (0.88–0.95) | 0.99 (0.95–1) | 1.1 (1–1.1) | 1.3 (1.2–1.3) | 1.5 (1.4–1.6) | 1.7 (1.6–1.9) | 1.8 (1.7–2) | 2 (1.8–2.2) | 2.1 (1.8–2.4) |
| MSM | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| Heterosexual | 0.83 (0.77–0.88) | 0.87 (0.82–0.93) | 0.93 (0.87–0.99) | 0.97 (0.9–1.1) | 1.1 (1–1.2) | 1.2 (1.1–1.3) | 1.3 (1.1–1.4) | 1.3 (1.2–1.5) | 1.4 (1.2–1.6) |
| Unknown | 0.65 (0.6–0.72) | 0.72 (0.66–0.78) | 0.82 (0.75–0.88) | 0.85 (0.78–0.93) | 0.96 (0.87–1.1) | 0.98 (0.88–1.1) | 1.1 (0.94–1.2) | 1.1 (0.99–1.3) | 1.3 (1.1–1.5) |
| Borough of residence* | | | | | | | | | |
| Manhattan | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| Brooklyn | 1.1 (1.1–1.1) | 1.1 (1–1.1) | 1.1 (1–1.1) | 1.1 (1–1.1) | 1.1 (1–1.1) | 1.1 (1–1.2) | 1.1 (1–1.2) | 1.1 (1–1.2) | 1.1 (0.95–1.2) |
| Bronx | 1.1 (1–1.1) | 1.1 (1–1.1) | 1.1 (1.1–1.2) | 1.1 (1–1.2) | 1.1 (1–1.2) | 1.1 (1.1–1.2) | 1.1 (1.1–1.2) | 1.1 (1–1.2) | 1.1 (1–1.2) |
| Queens | 1.1 (1–1.1) | 1 (0.96–1.1) | 0.95 (0.89–1) | 0.89 (0.83–0.95) | 0.83 (0.77–0.9) | 0.85 (0.78–0.93) | 0.83 (0.76–0.92) | 0.78 (0.7–0.87) | 0.8 (0.7–0.91) |
| Staten Island | 1.3 (1.1–1.4) | 1.2 (1.1–1.3) | 1.1 (0.99–1.3) | 1 (0.91–1.2) | 0.93 (0.78–1.1) | 1 (0.85–1.2) | 1 (0.84–1.3) | 1 (0.82–1.3) | 1 (0.76–1.3) |

**TABLE 3.** ***Continued***

| | Adjusted relative risks (95% CI) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 | 2001 |
| Category of AIDS diagnosis† | | | | | | | | | |
| Opportunistic illness | 1.7 (1.6–1.7) | 1.6 (1.5–1.6) | 1.7 (1.6–1.7) | 1.7 (1.6–1.7) | 1.6 (1.5–1.6) | 1.4 (1.3–1.5) | 1.5 (1.4–1.6) | 1.4 (1.3–1.6) | 1.4 (1.3–1.6) |
| CD4 <200 cells /μL or <14% | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| Earliest CD4 after AIDS diagnosis‡ | | | | | | | | | |
| Missing | 2.7 (2.5–2.9) | 2.6 (2.5–2.8) | 2.4 (2.3–2.6) | 2.1 (2–2.3) | 1.7 (1.5–1.8) | 1.5 (1.4–1.6) | 1.4 (1.3–1.5) | 1.2 (1.1–1.4) | 1.2 (1.1–1.4) |
| 0–49 | 2 (1.8–2.1) | 1.9 (1.8–2) | 1.8 (1.7–1.9) | 1.6 (1.5–1.8) | 1.5 (1.4–1.6) | 1.4 (1.3–1.5) | 1.3 (1.2–1.5) | 1.3 (1.2–1.5) | 1.3 (1.2–1.5) |
| 50–99 | 1.4 (1.3–1.5) | 1.3 (1.2–1.4) | 1.3 (1.2–1.4) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.2 (1.1–1.3) | 1.1 (1–1.3) | 1.2 (1–1.3) |
| 100–149 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| 150–199 | 0.64 (0.56–0.74) | 0.6 (0.54–0.68) | 0.59 (0.53–0.66) | 0.63 (0.56–0.71) | 0.7 (0.62–0.78) | 0.79 (0.7–0.88) | 0.81 (0.72–0.91) | 0.81 (0.71–0.93) | 0.81 (0.69–0.96) |
| 200+ | 1.3 (1.2–1.4) | 1.1 (1–1.2) | 0.92 (0.86–0.99) | 0.76 (0.7–0.82) | 0.71 (0.65–0.78) | 0.74 (0.67–0.82) | 0.64 (0.58–0.72) | 0.58 (0.52–0.66) | 0.59 (0.51–0.68) |
| Time since AIDS diagnosis (years) | | | | | | | | | |
| <1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 |
| 1–<2 | 1 (0.99–1.1) | 1.2 (1.2–1.3) | 1.3 (1.2–1.4) | 1.3 (1.3–1.4) | 1 (0.95–1.1) | 0.92 (0.84–1) | 0.87 (0.78–0.98) | 0.76 (0.67–0.86) | 0.89 (0.76–1) |
| 2–<3 | 0.93 (0.88–0.98) | 1.1 (1–1.1) | 1.3 (1.3–1.4) | 1.4 (1.3–1.5) | 1.1 (1–1.2) | 0.93 (0.85–1) | 0.84 (0.75–0.93) | 0.68 (0.6–0.78) | 0.58 (0.49–0.7) |
| 3–<4 | 0.62 (0.58–0.66) | 0.84 (0.79–0.89) | 0.99 (0.93–1) | 1.2 (1.1–1.2) | 1 (0.94–1.1) | 0.95 (0.87–1) | 0.88 (0.8–0.97) | 0.72 (0.65–0.8) | 0.62 (0.54–0.72) |
| ≥5 | 0.24 (0.22–0.26) | 0.3 (0.27–0.33) | 0.4 (0.36–0.44) | 0.58 (0.53–0.64) | 0.58 (0.53–0.64) | 0.61 (0.56–0.68) | 0.66 (0.6–0.73) | 0.6 (0.54–0.67) | 0.58 (0.51–0.67) |

IDUs, injecting drug users; MSM, men who have sex with men.

* Borough of patient residence at the time of AIDS diagnosis.

†Based on the 1993 AIDS case definition of the Centers for Disease Control and Prevention.

‡Within 9 months of AIDS diagnosis.

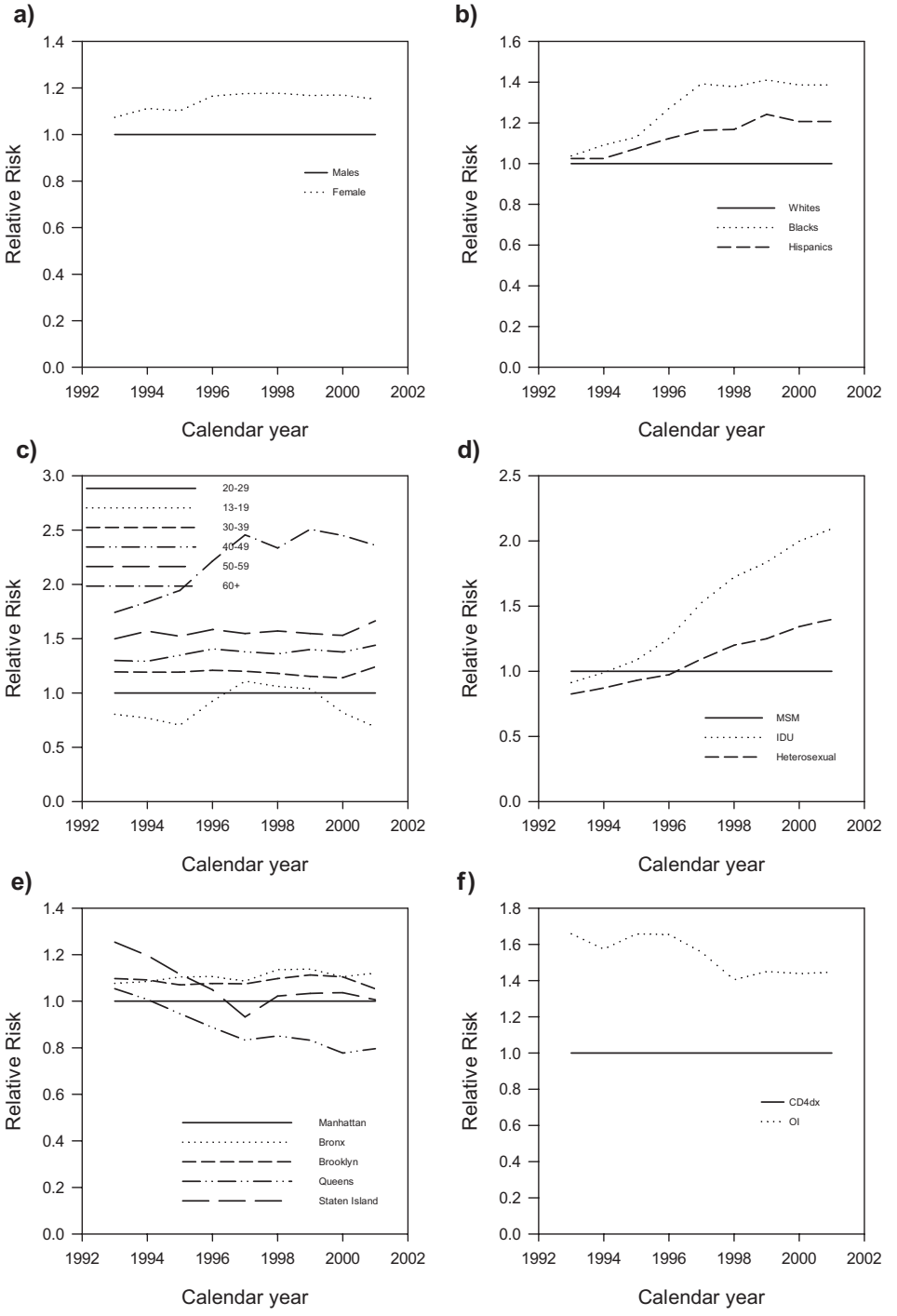


**FIGURE 3.** Adjusted relative risk of death due to HIV-related causes by (**a**) sex, (**b**) race/ethnicity, (**c**) age, (**d**) transmission risk, (**e**) borough of patient residence, and (**f**) AIDS diagnosis category among AIDS case-patients alive in each calendar year, 1993–2001, New York City (NYC).

diagnosed with AIDS 5 or more years prior had significantly better survival than persons diagnosed within 1 year.

## DISCUSSION

Our analysis indicates that, in the context of marked improvement in both mortality and survival among all PLWA, significant disparities have emerged among specific subgroups coincident with the widespread availability of HAART in 1995–1996. Disparities increased such that as of 2001, females, persons aged 60+, persons of black or Hispanic race/ethnicity, and persons with a history of IDU or heterosexual transmission risk had significantly poorer survival than their counterparts. Disparities in survival appeared to be widening not because death rates were increasing in any one group but primarily because they were not declining proportionally across subgroups. These disparities were seen to be independent of differences in age at AIDS diagnosis, disease stage, CD4 count at diagnosis, or time since diagnosis.

Our investigation evaluated trends in the predictors of survival using cohorts of PLWA alive in a given year, rather than cohorts based on stage of disease, year of AIDS diagnosis, or date of HIV seroconversion. This approach provides an alternative method for public health officials to characterize predictors of survival in a population of PLWA at a given point in time, which may be more relevant for some applications since it is an easily definable group in which to follow trends and target interventions. Many differences observed in our study population intensified over time, suggesting that elucidation of the underlying causes of these disparities among subgroups will be necessary to implement effective interventions aimed at reducing such disparities in the near future.

The findings are likely to reflect individual- and population-level differences in a variety of factors that affect survival in persons with HIV/AIDS that have become more apparent in the post-HAART era. Before HAART, the major determinants of survival in HIV patients were age and time since seroconversion.[23] After 1996, factors reported to affect survival include socioeconomic status,[24,25] access to care, timely initiation of HAART,[26] physicians' experience with caring for HIV/AIDS patients,[27] and adherence to complex medical regimens that may render viral strains therapy-resistant. In a cohort of HIV patients receiving HAART, one report found that neither age nor sex was associated with mortality,[28] suggesting that our results may indeed reflect differential initiation or usage of HAART in older persons and females. Such disparities in utilization of appropriate therapy for HIV have been previously reported[29,30] and are further suggested from the results of our analysis.

Reports are conflicting about sex differences in the natural history of HIV and response to HAART. Some studies have found no sex difference in response to HAART,[31,32] whereas others suggest that females do better.[33,34] Our investigation found that females are more likely to die compared with males and is inconsistent with both of these findings. Perhaps female PLWA in NYC are less likely than males to be on HAART.

Among persons with HIV, there has been an overall increase in proportion of patients receiving HAART,[26] however, disparities in therapy initiation or usage among groups based on sociodemographic factors have been reported, with blacks, IDUs, female heterosexuals, persons with lower levels of education, and the uninsured or Medicaid-insured less likely to receive timely therapy or any therapy at all.[26,35–37] A study of HIV-positive patients receiving HAART in NYC demonstrated that black race/ethnicity, older age, and low CD4 count had a significant, indepen-

dent negative effect on mortality,[38] supporting results observed in other studies that initiation of therapy may be delayed in these groups. Given reports that triple-therapy regimens uniformly decrease HIV RNA levels independent of sex, age, race, or transmission risk group,[28] the differences in survival that we observed may reflect differential access to, usage of, or initiation by providers of these life-prolonging regimens. The pronounced emergence of poorer survival among IDU relative to MSM, independent of other differences controlled for, could be attributed to a combination of a lower likelihood of HAART use or deleterious interaction between illicit drugs and HAART, both of which have been observed elsewhere.[39]

Limitations of our analysis must be considered. Differences in outcomes among subgroups might result from biases and confounders not accounted for in our model. We were unable to examine specific factors, such as health insurance, access to and quality of care, or health-related behavior that may account for these disparities on an individual and/or population level. Another limitation is the large proportion of persons missing a CD4 count in the 1993 PLWA cohort. We found in multivariate analysis that missing a CD4 count was independently associated with a higher risk of death. This finding and RRs for the other CD4 count levels in 1993 should be interpreted cautiously as it is possible that no available CD4 count is a marker for other factors such as more severe AIDS defining illness. Following the implementation of HIV (non-AIDS) reporting in June 2000,[21] the completeness of OI reporting may have been reduced, possibly resulting in some misclassification. Perhaps the largest limitation with our analysis is that information on treatment and clinical events, such as HAART initiation or development of an intercurrent illness, was not available for our study population. Finally, the methodological use of successive non-independent year-specific cohorts of PLWA limits the ability to apply statistical tests for trends and differences in RRs from year to year. However, AIDS case reporting in NYC was shown to be highly complete during the study period,[18] and the study population likely includes nearly all PLWA. Therefore, the observed trends may be interpreted with less reliance on statistical inference.

In summary, our analysis documents the emergence of significant disparities in survival among specific subgroups of PLWA in NYC over time, even after adjusting for multiple possible confounding factors. We attribute the observed differences to real differences in survival based on sex, race/ethnicity, and transmission risk group that likely reflect differential access to or utilization of medical services. The results of this investigation are highly generalizable to PLWA in NYC, given the high completeness of AIDS case reporting[18] and the large number of persons included in our analysis. Given the societal burden of HIV/AIDS in years of potential life lost,[40] future research and interventions must be targeted toward continued reduction of HIV/AIDS-related mortality and more definitive research studies regarding the specific factors that contribute to survival disparities are needed. Such efforts should be directed toward mitigating external factors that impact health and access to medical care and treatment.

## REFERENCES

1. Moore RD, Chaisson RE. Natural history of HIV infection in the era of combination antiretroviral therapy. *AIDS*. 1999;13:1933–1942.
2. Perez-Hoyos S, del Amo J, Muga R, et al. Effectiveness of highly active antiretroviral therapy in Spanish cohorts of HIV seroconverters: differences by transmission category. *AIDS*. 2003;17:353–359.

3. Baillargeon J, Borucki M, Black SA, Dunn K. Determinants of survival in HIV-positive patients. *Int J STD AIDS*. 1999;10:22–27.
4. Baillargeon J, Grady J, Borucki MJ. Immunological predictors of HIV-related survival. *Int J STD AIDS*. 1999;10:467–470.
5. Fordyce EJ, Blum S, Shum R, Singh TP, Chiasson MA, Thomas P. The changing AIDS epidemic in New York City: a descriptive birth cohort analysis of AIDS incidence and age at diagnosis. *AIDS*. 1995;9:605–610.
6. Fordyce EJ, Shum R, Singh TP, Forlenza S. Economic and geographic diversity in AIDS incidence among HIV exposure groups in New York City. *AIDS Public Policy J*. 1998 Fall;13:103–114.
7. Fordyce EJ, Williams RD, Surick IW, Shum RT, Quintyne RA, Thomas PA. Trends in the AIDS epidemic among men who reported sex with men in New York City. *AIDS Educ Prev*. 1995;7(suppl 5):3–12.
8. New York City Department of Health HIV Epidemiology Group. 25 years of HIV in New York City: lessons from surveillance. *J Urban Health*. 2001;78:669–678.
9. Des Jarlais DC, Friedman SR, Novick DM, et al. HIV-1 infection among intravenous drug users in Manhattan, New York City, from 1977 through 1987. *JAMA*. 1989;261:1008–1012.
10. Mocroft A, Vella S, Benfield TL, et al. Changing patterns of mortality across Europe in patients infected with HIV-1. EuroSIDA Study Group. *Lancet*. 1998;352:1725–1730.
11. Egger M, Hirschel B, Francioli P, et al. Impact of new antiretroviral combination therapies in HIV infected patients in Switzerland: prospective multicentre study. Swiss HIV Cohort Study. *BMJ*. 1997;315:1194–1199.
12. Palella FJ Jr, Delaney KM, Moorman AC, et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. HIV Outpatient Study Investigators. *N Engl J Med*. 1998;338:853–860.
13. Hogg RS, Heath KV, Yip B, et al. Improved survival among HIV-infected individuals following initiation of antiretroviral therapy. *JAMA*. 1998;279:450–454.
14. Detels R, Munoz A, McFarlane G, et al. Effectiveness of potent antiretroviral therapy on time to AIDS and death in men with known HIV infection duration. Multicenter AIDS Cohort Study Investigators. *JAMA*. 1998;280:1497–1503.
15. Schwartz S, Li W. *Summary of Vital Statistics 2001: The City of New York*. New York, NY: New York City Department of Health and Mental Hygiene; 2003.
16. Centers for Disease Control and Prevention. Guidelines for national human immunodeficiency virus case surveillance including monitoring for human immunodeficiency virus infection and acquired immunodeficiency syndrome. *MMWR Recomm Rep*. 1999;48:1–27, 29–31.
17. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. *MMWR Recomm Rep*. 1992;41:1–19.
18. Greenberg AE, Hindin R, Nicholas AG, Bryan EL, Thomas PA. The completeness of AIDS case reporting in New York City. *JAMA*. 1993;269:2995–3001.
19. CDC. *HIV/AIDS Surveillance Report*. Vol 13. Atlanta, GA: Centers for Disease Control and Prevention; 2001. No. 2.
20. Schmidt MA, Mokotoff ED. HIV/AIDS surveillance and prevention: improving the characterization of HIV transmission. *Public Health Report*. 2003;118:197–204.
21. CDC. Implementation of named HIV reporting — New York City, 2001. *MMWR Morb Mortal Wkly Rep*. 2004;52:1248–1252.
22. Lee LM, Karon JM, Selik R, Neal JJ, Fleming PL. Survival after AIDS diagnosis in adolescents and adults during the treatment era, United States. *JAMA*. 2001;285:1308–1315.
23. Survival after introduction of HAART in people with known duration of HIV-1 infection. The CASCADE collaboration. Concerted action on seroconversion to AIDS and death in Europe. *Lancet*. 2000;355:1158–1159.
24. Rapiti E, Porta D, Forastiere F, Fusco D, Perucci CA. Socioeconomic status and survival of persons with AIDS before and after the introduction of highly active antiretroviral therapy. Lazio AIDS Surveillance Collaborative Group. *Epidemiology*. 2000;11:496–501.

25. McFarland W, Chen S, Hsu L, Schwarcz S, Katz M. Low socioeconomic status is associated with a higher rate of death in the era of highly active antiretroviral therapy, San Francisco. *J Acquir Immune Defic Syndr*. 2003;33:96–103.
26. Cunningham WE, Markson LE, Andersen RM, et al. Prevalence and predictors of highly active antiretroviral therapy use in patients with HIV infection in the United States. HCSUS consortium. HIV cost and services utilization. *J Acquir Immune Defic Syndr*. 2000;25:115–123.
27. Kitahata MM, Koepsell TD, Deyo RA, Maxwell CL, Dodge WT, Wagner EH. Physicians' experience with the acquired immunodeficiency syndrome as a factor in patients' survival. *N Engl J Med*. 1996;334:701–706.
28. Hogg RS, Yip B, Chan KJ, et al. Rates of disease progression by baseline CD4 cell count and viral load after initiating triple-drug therapy. *JAMA*. 2001;286:2568–2577.
29. Shapiro MF, Morton SC, McCaffrey DF, et al. Variations in the care of HIV-infected adults in the United States: results from the HIV Cost and Services Utilization Study. *JAMA*. 1999;281:2305–2315.
30. Moore RD, Stanton D, Gopalan R, Chaisson RE. Racial differences in the use of drug therapy for HIV disease in an urban community. *N Engl J Med*. 1994;330:763–768.
31. Moore AL, Kirk O, Johnson AM, et al. Virologic, immunologic, and clinical response to highly active antiretroviral therapy: the gender issue revisited. *J Acquir Immune Defic Syndr*. 2003;32:452–461.
32. Chaisson RE, Keruly JC, Moore RD. Race, sex, drug use, and progression of human immunodeficiency virus disease. *N Engl J Med*. 1995;333:751–756.
33. Prins M, Robertson JR, Brettle RP, et al. Do gender differences in CD4 cell counts matter? *AIDS*. 1999;13:2361–2364.
34. Junghans C, Ledergerber B, Chan P, Weber R, Egger M. Sex differences in HIV-1 viral load and progression to AIDS. Swiss HIV Cohort Study. *Lancet*. 1999;353:589–590, author reply 590–591.
35. Easterbrook PJ, Keruly JC, Creagh-Kirk T, Richman DD, Chaisson RE, Moore RD. Racial and ethnic differences in outcome in zidovudine-treated patients with advanced HIV disease. Zidovudine Epidemiology Study Group. *JAMA*. 1991;266:2713–2718.
36. del Amo J, del Romero J, Barrasa A, et al. Factors influencing HIV progression in a seroconverter cohort in Madrid from 1985 to 1999. *Sex Transm Infect*. 2002;78:255–260.
37. Sackoff JE, McFarland JW, Shin SS. Trends in prescriptions for highly active antiretroviral therapy in four New York City HIV clinics. *J Acquir Immune Defic Syndr*. 2000;23:178–183.
38. Messeri P, Lee G, Abramson DM, Aidala A, Chiasson MA, Jessop DJ. Antiretroviral therapy and declining AIDS mortality in New York City. *Med Care*. 2003;41:512–521.
39. Lucas GM, Cheever LW, Chaisson RE, Moore RD. Detrimental effects of continued illicit drug use on the treatment of HIV-1 infection. *J Acquir Immune Defic Syndr*. 2001;27:251–259.
40. Obiri GU, Fordyce EJ, Singh TP, Forlenza S. Effect of HIV/AIDS versus other causes of death on premature mortality in New York City. *Am J Epidemiol*. 1998;147:840–845.