

Cancer Risk in Elderly Persons With HIV/AIDS

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Context: Cancer risks in persons with AIDS are increased, but risks in elderly persons with AIDS (EPWAs) have not been previously described.

Objective: To determine the profile of cancer risks in EPWAs.

Data Sources and Analysis: Using AIDS data from 1981–1996, 8828 EPWAs were identified (60+ years old) and their records were linked to data in local cancer registries, finding 1142 cases. Expected case numbers were derived from the cancer incidence in the population matched for age, sex, race, calendar year, and registry.

Results: Compared with the general population, the relative risk (RR) for Kaposi sarcoma was 545 (95% CI, 406–717) in the 2 years after AIDS onset. For non-Hodgkin lymphoma, the RR was 24.6 (7.5–80.3). No cervical cancers were reported in this interval. From 60 months before to 27 months after AIDS onset, the RR of non-AIDS-defining cancers (n = 548) was 1.3 (1.2–1.4). The cancer types occurring at significant excess during this period were similar to those in younger adults with AIDS: Hodgkin lymphoma (RR: 13.1), anal cancer (8.2), liver cancer (3.9), multiple myeloma (2.7), leukemia (2.4), and lung cancer (1.9). However, none was significantly elevated in the 2 years after the AIDS onset. Prostate cancer risk was low overall (RR: 0.8; 0.6–0.9).

Conclusions: The profile of cancer risks in EPWAs generally resembled that in younger adults with AIDS, although RRs were lower because of higher background incidence rates. We speculate that prostate cancer risk was low because of reduced screening for this cancer in EPWAs.

Key Words: HIV/AIDS, cancer, Kaposi sarcoma, nonHodgkin lymphoma, elderly persons

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Adults with AIDS have an increased risk of cancer. Kaposi sarcoma (KS) and non-Hodgkin lymphoma (NHL) are so frequent that they are considered manifestations of AIDS in a person with HIV infection.¹ Invasive cervical cancer is also considered to be AIDS-defining in women with HIV,¹ although evidence of its association with immunodeficiency is less conclusive.² Other cancers may also occur excessively in persons with AIDS, such as Hodgkin lymphoma, seminoma, multiple myeloma, and cancers of the anus, brain, and lung.^{3–7} Some of the increased incidence may be due to co-factors, such as sexually acquired viruses or smoking, rather than to HIV infection or immunodeficiency, per se, but KS and lymphoma were related to immunodeficiency.⁸

Registry-linkage studies of cancer in persons with HIV/AIDS have focused on children, adolescents, and adults up to age 69 years, finding evidence that KS and NHL, but not most cancers in these age groups, were related to immunodeficiency.^{2–7} Cancer risks in elderly persons with AIDS have not been systematically studied, because relatively few elderly persons have developed AIDS. However, in the elderly, cancer incidence rises rapidly,^{9,10} and the distribution of cancer types changes with age. We investigated cancer risks in persons at least 60 years old at the time of AIDS diagnosis, a group we define as elderly persons with AIDS (EPWAs), to determine whether HIV/AIDS-related immunosuppression affects the profile of cancer risk in the elderly.

METHODS

In the AIDS-Cancer Match Registry Study, we linked cases of AIDS from 6 states (Connecticut, Florida, Illinois, Massachusetts, New York, New Jersey) and 5 metropolitan areas (Atlanta, Los Angeles, San Diego, San Francisco, Seattle) to cancer registry records in these regions. These areas were selected because they had large numbers of AIDS cases and cancer registries encompassing the AIDS epidemic period. The linkage used AutoMatch, versions 3.0 and 4.1 (MatchWare Technologies, Inc., Burtonsville, MD). Details of the design and matching process have been previously re-

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ported in detail.^{2,5,11} AIDS reporting has been legally required since early in the AIDS epidemic, which began in 1981. However, cancer registries in study areas differed in their years of operation. We used only data from those years that the local cancer registries considered registration to be at least 95% complete, which ended between 1994 and 1996, depending on the registry. Thus, this database was assembled before highly active antiretroviral therapy changed the incidence of the AIDS-defining cancers.¹²

For this study, we focused on cancer risk in 8828 EPWAs. In some analyses, however, we display incidence trends across a wider age range, including data from all 304,411 adults 15–69 years old with AIDS.^{2,5} To classify the cancers, we used the International Classification of Disease for Oncology, 2nd ed.¹³ Codes for AIDS-defining cancers were KS (morphology code 9140), NHL (9590-9595 and 9670-9714), and invasive cervical cancer (site code 53.0-53.9). After excluding AIDS-defining cancers, other cancer types were evaluated by site rather than morphology, unless otherwise indicated (eg, Hodgkin lymphoma). Cancers with ill-defined morphology were defined as having histology codes 8000–8004. In situ cancers were not analyzed because the data were considered incomplete.

The overall period of evaluation was from 60 months before to 27 months after AIDS onset. Within the overall period, there were several critical periods. We defined the AIDS onset period as 6 months before to 3 months after AIDS diagnosis, to accommodate variation in reporting of cancers diagnosed around the time of AIDS diagnosis. For AIDS-defining cancers, which by definition do not occur before AIDS onset, we compare the proportions of individuals in various groups who were diagnosed in the AIDS onset period, using the χ^2 test. The post-AIDS onset period was defined as 4–27 months after AIDS diagnosis. This period was used to determine KS and NHL incidence, and incidence was compared across demographic groups using Poisson regression stratified by age, including data on cancer risk in 50–59 PWAs for comparison. We also calculated relative risk (RR) in the post-AIDS onset period, comparing the cancer risk in EPWAs to that in the general population. To measure RR, the expected number of cancer cases was computed according to age group-, sex-, race-, and calendar year-specific incidence rates obtained from the local cancer registry data, using previously described methods.^{2,5,11}

For non-AIDS-defining cancers, we compared the observed number of cases in the overall observation period with the number expected, based on local cancer registry incidence data. Calculations of expected cancer numbers in the pre-AIDS onset period were complicated because some HIV-infected persons would have died before they developed AIDS. To account for this loss in computing the RR, we used a previously described survival-conditioned approach.^{5,11} This method adjusted the expected number of cancers to account for

postcancer survival, using registry survival data. RRs were calculated by dividing the observed cases by the number expected, and 95% CIs were calculated assuming a Poisson distribution.

Finally, for non-AIDS-defining cancers, we tested whether RR increased over time relative to AIDS onset, to determine whether risk increased with progressive HIV-related immunosuppression. Specifically, we applied 3 previously described criteria for AIDS-associated cancers.⁵ First, in PWAs, the RR in the overall period should be significantly increased. Second, the RR in the post-AIDS onset period should be significantly increased. And third, the RR should increase as AIDS onset approaches and passes. Here, we used a trend test for the RR in 3 periods: 60–25 months before AIDS diagnosis; 24–7 months before AIDS diagnosis; and 4–27 months after AIDS diagnosis. The AIDS onset period was excluded from trend analyses to avoid ascertainment bias due to increased medical attention at the time of AIDS onset.

RESULTS

Demographic characteristics and mode of HIV transmission for 8828 EPWAs are shown in Table 1. In most (84.1% of men and 72.3% of women), AIDS was diagnosed between 60–

TABLE 1. Demographic Characteristics and Route of HIV Transmission Among 8828 Elderly Persons With AIDS

Characteristics	Men n = 7356 (83.3%)	Women n = 1472 (16.7%)
Age at AIDS diagnosis (y)		
60–69	6187 (84.1%)	1064 (72.3%)
70–79	1060 (14.4%)	354 (24.1%)
80+	109 (1.5%)	54 (3.7%)
Median (range)	64 (60–99)	65 (60–90)
Year of diagnosis, median	1992	1992
Ethnic group		
White	3805 (51.7%)	563 (38.3%)
Black	2403 (32.7%)	670 (45.5%)
Hispanic	1075 (14.6%)	217 (14.7%)
Other/unknown	73 (1.0%)	22 (1.5%)
Route of HIV acquisition		
Homosexual contact	3667 (49.9%)	—
Homosexual contact + IV drug use	104 (1.2%)	—
Heterosexual contact	602 (8.2%)	548 (37.2%)
IV drug use	895 (12.2%)	151 (10.3%)
Transfusion or transplantation	707 (9.6%)	402 (27.3%)
Hemophilia/other bleeding disorders	95 (1.3%)	10 (0.7%)
Unknown	1286 (17.5%)	361 (24.5%)

69 years of age. The EPWAs were predominately male (83.3%), among whom 51.7% were white and 32.7% were black. In women, 45.5% of EPWAs were black and 38.3% were white. In men, HIV exposure occurred through homosexual contact, with or without IV drug use (51.3%), other known routes (31.3%), and unknown routes (17.5%). Excluding men with homosexual exposure and those with unknown exposure routes, men still comprised 67.4% of EPWAs, largely because of the excess of men exposed to HIV from IV drug use. For female EPWAs, the dominant exposure route was a male sex partner (37.2%). Among EPWAs, 1142 (12.9%) developed cancer in the overall period, including 594 AIDS-defining cancers (52%) and 548 non-AIDS-defining cancers (48%).

Kaposi Sarcoma

In the AIDS onset period, 209 cases of KS were diagnosed in EPWAs, all but 3 in men (Table 2). Most cases (83.7%) were men whose HIV exposure route was known to be

homosexual, among whom 5.6% developed KS in the AIDS onset period. In contrast, among men exposed by other routes, only 0.9% developed KS in this period. In homosexual men, KS was more common in whites (6.9%) than blacks (2.6%) or Hispanics (3.6%) (white vs. nonwhite, $P < 0.0001$). In men exposed by other routes, the differences between whites (1.4%), blacks (0.4%), and Hispanics (1.1%) were not significant (white vs. nonwhite, $P = 0.10$). In male homosexual EPWAs, the proportion developing KS decreased with age in the AIDS onset period ($P_{\text{trend}} = 0.04$).

During the post-AIDS onset period, we observed 51 KS cases, including 2 in women. For homosexual men, the incidence was 2.4 per 100 person-years in whites, 1.0 in blacks, and 0.9 in Hispanics. The corresponding RRs were also higher in white than in black or Hispanic homosexual men (1056, 379, and 394, respectively). There were only 2 cases in men exposed by other routes (incidence: 0.2 per 100 person-years) and 2 cases in women (0.3 per 100 person-years). Figure 1 displays the incidence in homosexual men, men exposed by

TABLE 2. Distribution of Kaposi Sarcoma in 6744 Elderly Persons With AIDS, by HIV Exposure Route (for Men Only) and Time From AIDS Diagnosis*

	Number	AIDS Onset Period (0–+3 months)		Post-AIDS Onset Period (4–27 months)			
		Observed	%	Observed	Incidence†	RR	95% CI
Men							
Homosexual exposure							
All	3125	175	5.6	45	2.0	836	610–1119
White	2127	146	6.9	39	2.4	1056	751–1444
Black	605	16	2.6	4	1.0	379	103–970
Hispanic	360	13	3.6	2	0.9	394	47.7–1422
Other exposure							
All	1668	15	0.9	2	0.2	76.2	9.2–273
White	650	9	1.4	0	0	0	—
Black	721	3	0.4	2	0.4	156	18.8–561
Hispanic	275	3	1.1	0	0	0	—
Unknown exposure							
All	856	16	1.9	2	0.4	178	21.5–642
White	289	7	2.4	2	1.2	513	62.0–1850
Black	362	4	1.1	0	0	0	—
Hispanic	189	5	2.6	0	0	0	—
Women							
All	1095	3	0.3	2	0.3	880	106–3174
White	449	1	0.2	2	0.7	1668	202–6022
Black	456	0	—	0	0	0	—
Hispanic	171	2	1.2	0	0	0	—

*Florida data were excluded from analysis of KS data because of uncertainty about the criteria for KS diagnoses in registry data. Not shown are 90 subjects whose race group was other/unknown (no KS cases).

†Incidence per 100 person-years.

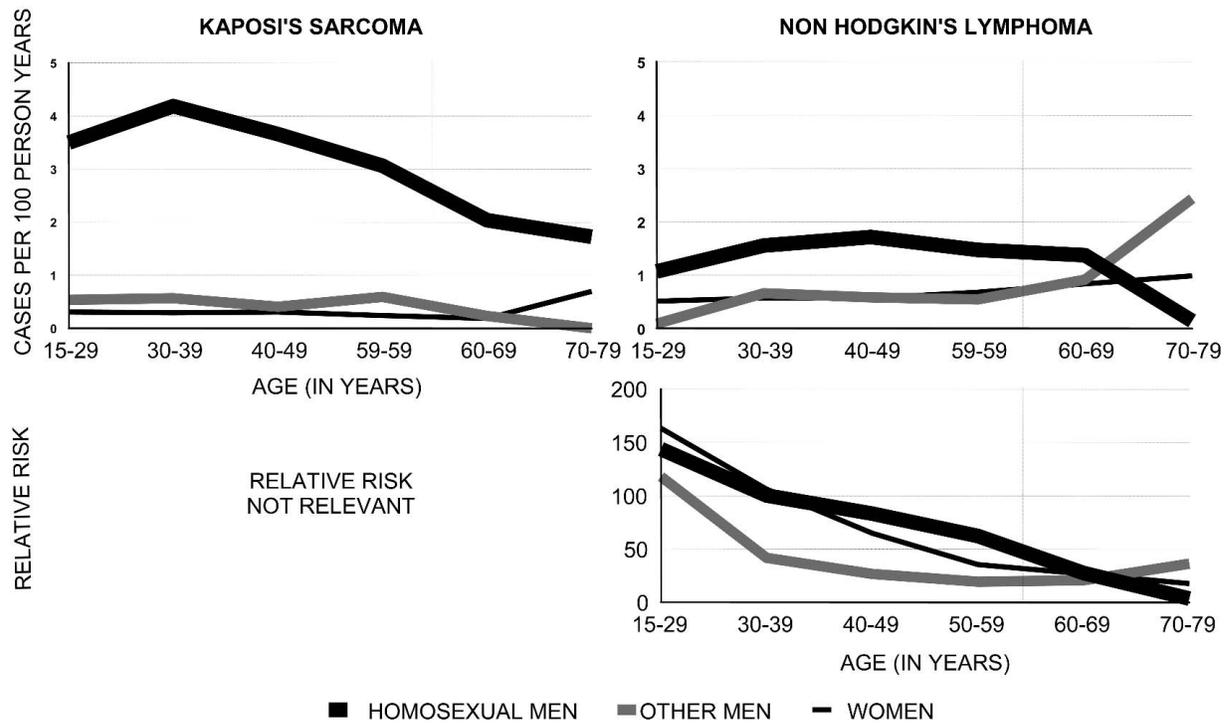


FIGURE 1.

other routes, and women, by age and HIV exposure group. Changes in incidence in the post-AIDS onset period (Fig. 1) were not statistically significant in any of the 3 EPWA groups. Data for these subjects have been previously presented as grouped data.¹⁴

Non-Hodgkin Lymphoma

In the AIDS onset period, 258 EPWAs (2.9%) had NHL (Table 3). More men (3.1%) than women (2.2%) were diagnosed with NHL during the AIDS onset period ($P = 0.09$). The proportion in whites (4.1%) was significantly higher than in blacks (1.6%; $P < 0.0001$) or Hispanics (2.5%; $P = 0.008$), and the proportion in Hispanics was higher than in blacks ($P = 0.04$). Similar differences by race were also seen separately among homosexual men, other men, and women (Table 3).

During the post-AIDS onset period, 70 EPWAs (0.8%) developed NHL, the incidence being 1.2 per 100 person-years (RR, 24.6; 95% CI, 19.2–31.1). Incidence and RR patterns for NHL in the post-AIDS onset period differed by racial groups. In white EPWAs, the incidence was 1.7 per 100 person-years, while blacks and Hispanics had lower incidences (0.5 and 1.2, respectively). The RR of NHL for whites (RR, 28.9; 95% CI, 21.4–38.2) was also higher than that for blacks (RR, 13.4; 95% CI, 6.2–25.5), while Hispanics were intermediate (RR, 22.4; 95% CI, 10.7–41.2). Similar trends were seen for homosexual men, all men, women, and all EPWAs.

Risk of NHL also differed by exposure group. In younger men of all ages, homosexual men had a higher incidence than men exposed by other routes or women (Fig. 1). However, in EPWAs, the incidence in homosexual men declined with age, whereas in the other groups, incidence was stable or increasing. Some of this decline was related to differences in racial composition between EPWAs and younger groups. However, among white homosexual men in the post-AIDS onset period, incidence declined from a peak of 2.1 per 100 in 50- to 59-year-old subjects to 1.3 in persons at least 70 years old ($P = 0.18$). In contrast, incidence continued to rise with age in white men with other exposures and in women. To exclude possible bias from differences in survival time (competing risk), we compared the number of EPWAs known dead at 28 months after AIDS diagnosis. The proportions known dead were similar (91.4% for homosexual men, 93.5% for men exposed by other routes, and 91.3% for women).

Invasive Cervical Cancer

Invasive cervical cancer was diagnosed in 6 female EPWAs (0.4%) during the AIDS onset period. There were no cases after the AIDS onset period, but only 0.26 cases were expected and the 95% upper limit of the RR was 11.7.

Other Cancers

In the overall period, non-AIDS-defining cancers were diagnosed in 548 EPWAs (1.06 cancers per 100 person-years)

TABLE 3. Non-Hodgkin Lymphoma in 8828 Elderly Persons With AIDS, by HIV Exposure Route and Time From AIDS Diagnosis*

	Number	AIDS Onset Period (0–3 months)		Post-AIDS Onset Period (4–27 months)			
		Observed	%	Observed	Incidence†	RR	95% CI
Men							
Homosexual exposure							
All	3771	145	3.8	37	1.4	26.2	18.4–36.1
White	2561	117	4.6	32	1.7	30.0	20.5–42.3
Black	704	12	1.7	1	0.2	6.5	0.2–35.4
Hispanic	472	16	3.4	3	1.0	17.6	3.6–51.4
Other exposure							
All	2299	50	2.2	17	1.2	23.6	13.8–37.8
White	874	27	3.1	7	1.4	22.1	8.9–45.6
Black	1065	14	1.3	5	0.7	18.2	5.9–42.5
Hispanic	338	8	2.4	5	2.3	40.2	13.0–93.7
Unknown exposure							
All	1286	30	2.3	8	1.1	22.0	9.5–43.4
White	370	17	4.6	6	2.6	43.4	15.9–94.6
Black	634	8	1.3	0	0	0	—
Hispanic	265	5	1.9	2	1.3	22.0	2.7–79.3
Women							
All races	1472	33	2.2	8	0.9	22.8	9.8–44.8
White	563	16	2.8	4	1.2	23.1	6.3–59.1
Black	670	14	2.1	3	0.7	27.6	5.7–80.6
Hispanic	217	3	1.4	0	0	0	—

*Not shown are 95 subjects whose race group was “other/unknown (1 NHL case).

†Incidence per 100 person-years.

(Table 4). The RR of cancers of all non-AIDS-defining types was 1.3 (95% CI, 1.2–1.4). The distribution of cancers in EPWAs by site was generally typical of cancer types found in elderly populations. Prostate cancer was the most common type (118 cases), followed by cancers of the lung (105 cases) and colon (43 cases). The risk of prostate cancer was significantly lower than expected in both the overall (RR 0.8; 95% CI, 0.6–0.9) and post-AIDS onset periods (RR 0.5; 95% CI, 0.3–0.8), and incidence decreased as AIDS approached and passed ($P_{\text{trend}} = 0.02$). Two of 120 prostate cancers (2%) had nonspecific histologies. Analyses of prostate cancer risk stratified by race or exposure group yielded unstable results (confidence intervals for RR including unity).

No specific cancer type occurred at significant excess in the post-AIDS onset period, and none increased significantly in incidence as AIDS diagnosis approached and passed. However, several cancer risks were significantly elevated for the overall period: Hodgkin lymphoma (RR, 13.1), anal cancer (8.2), liver cancer (3.9), multiple myeloma (2.7), leukemia (2.4), lung cancer (1.9), and stomach cancer (1.9). While stomach cancer risk was increased overall, the incidence declined

as AIDS approached and passed ($P = 0.05$). While all cancers diagnosed as KS or NHL were excluded from the site-specific assessments, a few cancers included in sites with increased overall risks had nonspecific histologies (8 lung, 8%; 1 liver, 12%) that might have been KS or NHL. However, reanalyses of site-specific rates excluding cancers with nonspecific histologies from both EPWAs and the background population did not materially change the findings (data not reported).

DISCUSSION

Despite the high cancer incidence in the elderly, AIDS-defining cancers comprised half of all cancers in EPWAs, with KS and NHL frequency being about equal. In the elderly, as among other age groups with AIDS,¹⁵ the highest KS risk was found in homosexual men. In this group, however, the proportion of EPWAs in whom KS was diagnosed decreased with age, even in the very elderly. This pattern continues a trend that we previously reported among younger homosexual men with AIDS, in whom KS incidence peaks in the 30- to 39-year-old age group.^{14–16} In contrast, in non-HIV-infected persons, KS incidence is highest in the elderly,¹⁷ albeit at a much lower

TABLE 4. Non-AIDS-Defining Cancers in 8828 Elderly Persons Who Developed AIDS During the Period from 60 Months Before to 27 Months After AIDS Diagnosis

	Observed	Expected	RR (95% CI)	<i>P</i> _{trend} *
All	548	423.5	1.3 (1.2–1.4)	0.0001 (↑)
Prostate	118	153.1	0.8 (0.6–0.9)	0.017 (↓)
Lung	105	54.5	1.9 (1.6–2.3)	0.61
Colon	43	49.5	0.9 (0.6–1.2)	
Bladder	24	25.9	0.9 (0.6–1.4)	
Breast	20	19.7	1.0 (0.6–1.6)	
Leukemia	19	7.8	2.4 (1.5–3.8)	0.21
Upper respiratory tract	19	12.4	1.5 (0.9–2.4)	
Oropharynx	19	14.3	1.3 (0.8–2.1)	
Hodgkin lymphoma	15	1.1	13.1 (7.4–21.6)	0.20
Multiple myeloma	15	5.6	2.7 (1.5–4.4)	0.11
Stomach	14	7.5	1.9 (1.0–3.1)	0.05 (↓)
Melanoma	14	9.1	1.5 (0.8–2.6)	
Rectum	11	11.9	0.9 (0.5–1.7)	
Liver	8	2.0	3.9 (1.7–7.8)	0.24
Kidney	7	10.6	0.7 (0.3–1.4)	
Esophagus	7	4.9	1.4 (0.6–2.9)	
Pancreas	7	4.8	1.4 (0.6–3.0)	
Anus	6	0.7	8.2 (3.0–17.8)	0.50
Central nervous system	4	2.5	1.6 (0.4–4.0)	
Thyroid	3	2.0	1.4 (0.3–4.2)	
Small intestine	2	1.3	1.5 (0.2–5.4)	
Uterus	2	4.3	0.5 (0.1–1.7)	
Ovary	2	1.8	1.1 (0.1–4.1)	
Musculoskeletal	2	2.2	0.9 (0.1–3.3)	
Renal Pelvis	1	1.0	1.0 (0.0–5.7)	
Penis	1	0.7	1.5 (0.0–8.1)	
Testicle	1	0.5	2.2 (0.0–12.0)	
Unknown sites	59	11.6	5.1 (3.9–6.6)	0.002 (↑)

*Trend analyses refer to incidence increasing (↑) or decreasing (↓) relative to the AIDS diagnosis date and are provided when RR excluded unity.

incidence than in EPWAs. We have previously speculated that the variation in KS incidence across demographic categories occurred in large part because of different exposures to human herpesvirus 8 (HHV8),^{12,14,16} a viral co-factor necessary for the development of KS.¹⁸

In both men and women, NHL incidence in white EPWAs was 3 times that of blacks in the post-AIDS onset period, with Hispanics being intermediate. Similar findings by race were found in comparing the proportions of NHL in the AIDS onset period. The higher incidence in men and in whites has been found in studies of younger adults both with and without AIDS.^{12,16,19} The reason for the higher incidence in whites is unknown, and it may be related to factors related to standard of living, such as exposures to oncogenic viruses or chemicals, rather than race.¹⁹ Both white and black homosexual men had

a higher incidence of NHL than did their respective strata in men exposed to HIV by other routes. As a group, homosexual men are likely to have a higher standard of living than men exposed by other routes, a group that includes a large number of IV drug users. The unexpected decline of NHL incidence with age in male homosexual EPWAs is puzzling. Unlike KS, in which HHV8 is requisite to tumor development, no virus or other co-factor is known to be essential to the development of NHL, although Epstein-Barr virus has been linked to some cases.

Comparisons based on RR need careful evaluation, because RRs are leveraged by variations of incidence in the background populations. For both men and women, the RRs of NHL were considerably lower in the elderly population than were found in previous studies of younger adults.^{5,6,16,19} However, NHL incidence in the background population rises with

age, and the lower RR in EPWAs (Fig. 1) was therefore expected. Because women have a lower background incidence, the RR in PWAs was inflated, resulting in higher RRs in women than men.^{6,20}

The proportion of women with cervical cancer in the AIDS onset period was similar to that in younger persons with AIDS.²⁻⁵ There were no cases of cervical cancer in the post-AIDS onset period, but only 0.26 were expected, so we cannot determine if risk was elevated.

The 1.3-fold excess of non-AIDS-defining cancer was statistically significant, but caution is warranted. In this group, 10.8% were cancers at unknown sites, compared with 2.7% expected on the basis of data in the general population. These cancers could have included AIDS-defining cancers with missing or miscoded histology as well as site. Similarly, in site analyses, we included a few cancers with nonspecific histologies in site analyses. However, excluding cancers at unknown sites from both observed and expected groups, the RR of non-AIDS-defining cancers in EPWAs remained elevated at 1.2-fold, and excluding the cancers with nonspecific histologies did not change the direction of the site-specific associations or render them nonsignificant.

While the overall risk of the non-AIDS-defining cancers in EPWA was small, the excess was due mainly to specific types of cancer: Hodgkin lymphoma, multiple myeloma, anal cancer, leukemia, liver cancer, lung cancer, and stomach cancer. Except for stomach cancer, these types were the same as those occurring excessively in younger PWAs,³⁻⁷ supporting the validity of earlier observations and indicating that EPWAs were at risk for similar but not additional cancer types. While an excess of stomach cancer was a new finding, it should be viewed as suspect, since it was based on relatively few cases, and in the trend analysis, the RR for stomach cancer declined as AIDS onset approached and passed.

Of interest, prostate cancer was found to have a significant negative association with AIDS, including a low overall RR, low RR in the 2 years after AIDS onset, and a declining trend in incidence. In our earlier studies of younger PWAs,⁵ we also found prostate cancer to be significantly decreased (RR in the post-AIDS onset period: 0.5; 95% CI, 0.4–0.7), but the trend test associating decreasing risk with advancing immunosuppression was only marginally significant ($P_{\text{trend}} = 0.10$) in that study. Prostate cancer risk was also decreased in another study, which used a subset (New York) of the current database.⁶ We know of no reason why HIV/AIDS-related immunosuppression would lower prostate cancer risk. In the era of this study, prostate cancer was often diagnosed through screening by prostate-specific antigen.²¹ We speculate that the lower risk of prostate cancer may be due to reduced screening or diagnostic work-up among elderly people with HIV/AIDS. In our study of younger PWAs,⁵ the risks of colon/rectum, bladder, and breast cancers were reduced in the post-AIDS on-

set period, but in the current study, their risks were not decreased.

In summary, EPWAs have increased risks of the same AIDS-defining and non-AIDS-defining cancers that also occur in excess in younger PWAs. The consistency of these findings strengthens the likelihood that these cancer types are truly AIDS-associated. The RRS in EPWAs are often lower than reported in younger PWAs because the background incidence of these cancers is higher in the elderly. Despite higher cancer incidence and different proportions of cancer types in EPWAs compared with younger PWAs, no additional cancer types were found to be AIDS associated. Prostate cancer risk was significantly lower than expected, a finding that could result from reduced screening and follow-up care in EPWAs. Thus, immunosuppression of the type occurring in AIDS had little impact on cancer risk in the elderly, other than for those cancer types that have been previously AIDS associated.

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