The Lifetime Risk of Developing Prostate Cancer in White and Black Men

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Abstract

Two factors help explain increases in the lifetime risk of developing cancer: (*a*) decreasing overall mortality rates such that people are now living to older ages when cancer rates rise rapidly; and (*b*) increasing numbers of cancer cases discovered by new medical procedures, screening tests, and changes in the population risk factors. Prostate cancer lifetime risk estimates are particularly influenced by improved mortality rates and increased detection of asymptomatic disease. In this study, we report trends in lifetime risk estimates of developing prostate cancer in white and black men in the United States, from 1975 to 1993, and focus on the effects of changing mortality and screening. For the study period 1975–1977 to 1991–1993, the lifetime risk of developing invasive prostate cancer increased from 7.3 to 19.6% for whites and from 8.5 to 18.6% for blacks. When we recalculated these estimates using age-specific incidence trends from 1975 through 1989 (thereby controlling for the effect of prostate-specific antigen serum testing on prostate cancer incidence rates), the lifetime risk estimates in 1991–1993 fell to 13.8% for whites and 12.5% for blacks. When we made an additional assumption, basing lifetime risk estimates on higher 1975–1977 mortality rates, the lifetime risk estimates in 1991–1993 became 11.3% for whites and 11.8% for blacks. It is also shown that although mortality rates have improved for white and black men over the study period, they are much larger for blacks than whites in younger age groups, when the prevalence of prostate cancer is relatively low. As a result, fewer blacks survive to older ages when age-specific prostate cancer rates are large. It is of note that blacks have higher incidence rates for prostate cancer than do whites at every age-specific interval. Hence, increasing trends in lifetime risk of prostate cancer suggest, in large part, longer life expectancy and better detection methods.

Received 12/19/96; revised 4/28/97; accepted 5/2/97.

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Introduction

Perhaps no single cancer statistic has received more attention in the popular press than lifetime risk. In recent months, newspapers across the country have reported the lifetime risk of developing prostate cancer: The Arizona Republic, The Atlanta Journal and Constitution, The Dallas Morning News, The Seattle Times, The Toronto Star, and the Washington Times, to name a few (1–6). Lifetime risk estimates also appear in annual reports by the American Cancer Society and the National Cancer Institute (7–8). Lifetime risk has been used to aid in the positive aspects of cancer awareness (*e.g.*, encouraging increased use of screening and clinical exams), in influencing the distribution of resources for research and health care services, and in comparing the cancer risk for subgroups of the population (*e.g.*, between whites and blacks, between males and females, and over calender time; Refs. 9 and 10). Calculations of lifetime risk are based on current age-specific incidence and mortality rates in the population applied to a hypothetical cohort (11). The result is an estimate of the risk of developing cancer for a newborn today and is based on the assumption that the current incidence and mortality rates in the population used in the computation remain stationary over the baby's lifetime. Lifetime risk does not take into account individual risk profiles but reflects the average condition in the population (12).

In recent decades, mortality rates from all causes have decreased such that people are now living to older ages when cancer rates are high. In addition, early detection efforts have increased, improving our ability to identify cancers when small and (hopefully) nonfatal. These changes are likely to have important effects on lifetime risk estimates for prostate cancer. In this study, we assess the effects of changing mortality and screening through PSA[2] serum testing on the lifetime risk of developing prostate cancer in black and white men in the United States, from 1975 to 1993. We show that improved overall mortality and PSA screening explain much of the observed increase in the lifetime risk estimates of developing prostate cancer.

Materials and Methods

The standard methodology for computing lifetime risk estimates of developing cancer has been presented elsewhere (11). This methodology involves a double decrement life table derived by applying age-specific incidence and mortality rates from cross-sectional data to a hypothetical cohort of 10 million live births. For each age interval a (0–4, 5–9, . . . , 90–94, 95+), the following are determined: (*a*) the number alive and cancer free, n_a; (*b*) the number of newly developed cancers, $n_a(c)$; and (3) the number of noncancer deaths, $n_a(d)$, among the cancer-free population. Through the life table, the number alive

[2] The abbreviations used are: PSA, prostate-specific antigen; SEER, surveillance, epidemiology, and end results; TURP, transurethral resection of the prostate.

and cancer free are computed as $n_{a+5} = n_a - n_a(c) - n_a(d)$. The cumulative probability of developing prostate cancer from birth to a given age is derived by summing the number of newly developed cancers through that age interval and dividing by 10 million. Being diagnosed with cancer for the first time and death due to causes other than cancer without having developed the cancer are mutually exclusive and exhaustive events. We assume that the death rate due to non-prostate cancer causes is the same in the total population as it is in the cancer-free population. The incidence rates reflect only the first primary cancer for the disease. Also, an adjustment is made based on age-specific cancer prevalence, so that the risk estimates in each 5-year age interval only represent the cancer-free population.

Incidence and mortality data used in this analysis come from the nine standard tumor registries in the SEER Program of the National Cancer Institute. Population values from the United States Bureau of the Census are applied to this data to compute rates. The nine tumor registries that entered the SEER Program between 1973 and 1975 represent approximately 10% of the United States population and are located in San Francisco/Oakland, Connecticut, metropolitan Detroit, Hawaii, Iowa, New Mexico, Utah, Seattle/Puget Sound, and metropolitan Atlanta. Reported incidence rates are age-adjusted within 10-year age categories per 100,000 person-years using the 1970 standard population. Differences in prostate cancer incidence rates between whites and blacks were tested using Poisson regression techniques (13).

Lifetime risk estimates are computed using the National Cancer Institute's computer program for developing cancer, DEVCAN (14). The risk estimates are presented as the percentage of males in SEER developing invasive prostate cancer from birth, based on cross-sectional data from 1975–1977, 1978–1980, 1981–1983, 1984–1986, 1987–1989, and 1991–1993. We use 1991–1993 rather than 1990–1992 as the final period to avoid using overlapping data and to use the most recent available data.

To account for the effect of PSA screening on lifetime risk estimates of developing prostate cancer, 1991–1993 lifetime risk estimates were calculated based on trends in the age-specific incidence rates from 1975 through 1989. Previous reports have identified 1989 as the period in which PSA screening began to perturb prostate cancer incidence rates above their secular trend (15). This rise in rates reflects, at least in part, cases being detected earlier through screening (16). In each 5-year age group, a linear trend was fit to the incidence rates through 1989. The estimated annual percentage changes were computed by regressing the natural logarithm of the incidence rates (r) on calendar year (y) (*i.e.*, $\ln r = my + b$) and equal $100 \times (e^m - 1)$. The fitted linear trends were then assumed to represent the age-specific secular trend in the absence of PSA screening. It was also assumed that the age-specific trends would have continued through 1991–1993.

Table 1 Estimated annual percentage change over 1975–1989 in prostate cancer incidence rates by age group and race[a]

Age (yrs)	Whites	Blacks
50–54	3.1	2.5
55–59	3.9	1.0
60–64	3.9	2.9
65–69	4.3	2.7
70–74	3.9	2.0
75–79	2.9	2.0
80–84	1.8	1.8
85+	0.7	1.2

[a] Source, SEER.

To account for declining mortality due to causes other than prostate cancer (mostly associated with declines in cardiovascular mortality) since 1975–1977, we performed calculations in the remaining time periods using the corresponding observed prostate cancer incidence rates, but holding mortality due to other causes constant at 1975–1977 levels.

Results

Estimated annual percentage changes for the period 1975–1989, before the rapid rise in prostate cancer incidence rates, are presented by age group and race in Table 1. These annual percentage changes increase for both blacks and whites, being somewhat smaller for blacks through age group 75–79. Prostate cancer incidence rates are reported in Table 2 by age group, race, and time period. The incidence rates rise with age and time period, with the most noticeable increase in the age groups 50–59 and 60–69 and after 1989. The results from our Poisson regression model, controlling for age and calender time, indicate that blacks have 1.5 (confidence interval, 1.48–1.52) times the prostate cancer incidence rates of whites. The difference in rates between whites and blacks significantly decreases with older age groups and later time intervals. In addition, of the black men identified with prostate cancer from 1975–1993, 31% were diagnosed at age 75 or later. A somewhat higher corresponding percentage was observed for whites, 40%.

The percentages of men developing prostate cancer from birth are reported over time for white and black males in Fig. 1, *a* and *b*. The effects of changing mortality and perturbed incidence due to PSA screening on the lifetime risk estimates of developing prostate cancer are reported. A large PSA screening effect on the risk estimates is shown for both whites and blacks. However, the mortality effect on the risk estimates, although noticeable for whites (Fig. 1*a*), is much smaller and trivial for blacks (Fig. 1*b*).

Table 2 Prostate cancer incidence rates by age group, race, and time period[a]

	40–49 yrs		50–59 yrs		60–69 yrs		70–79 yrs		80+ yrs	
	Whites	Blacks	Whites	Blacks	Whites	Blacks	Whites	Blacks	Whites	Blacks
1975–1977	3	10	46	100	244	456	626	957	1048	1314
1978–1980	4	9	47	105	260	452	681	1074	1070	1412
1981–1983	4	9	53	103	283	511	728	1136	1122	1481
1984–1986	4	5	57	107	312	565	776	1115	1127	1532
1987–1989	4	8	72	124	406	612	969	1271	1250	1551
1991–1993	9	13	153	282	754	1109	1477	2014	1452	1962

[a] Rates are age-adjusted within age categories per 100,000 person-years using the 1970 standard population. Source, SEER.

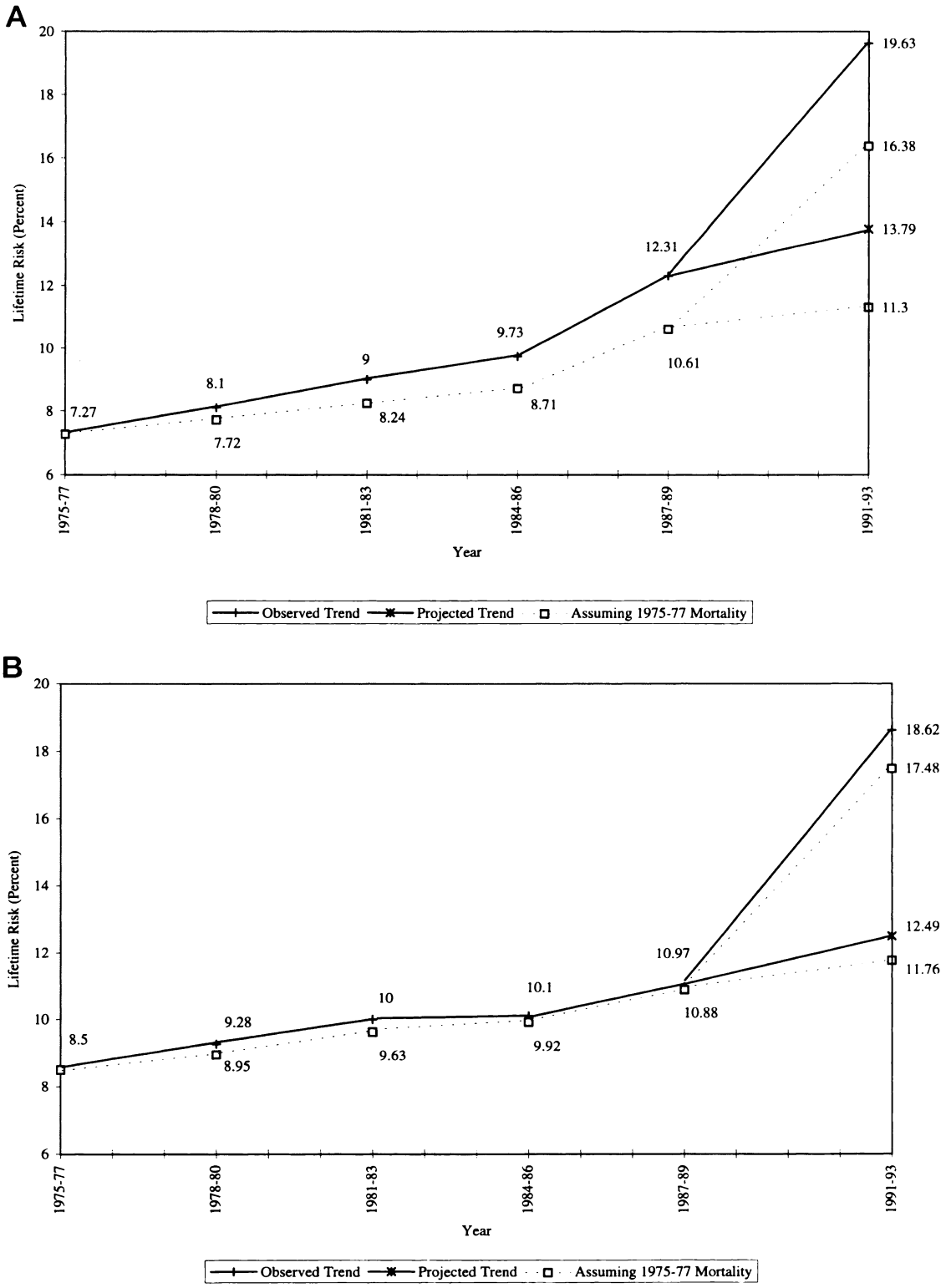

Fig. 1. A, lifetime risk of developing prostate cancer in white men by time period. *B*, lifetime risk of developing prostate cancer in black men by time period. For *A* and *B*, the projected trend through 1993 is based on years 1975–1989. It assumes that increases in PSA screening after 1989, which would inflate prostate cancer risk, did not occur. +, observed trend; X, projected trend; □, assuming 1975–1977 mortality.

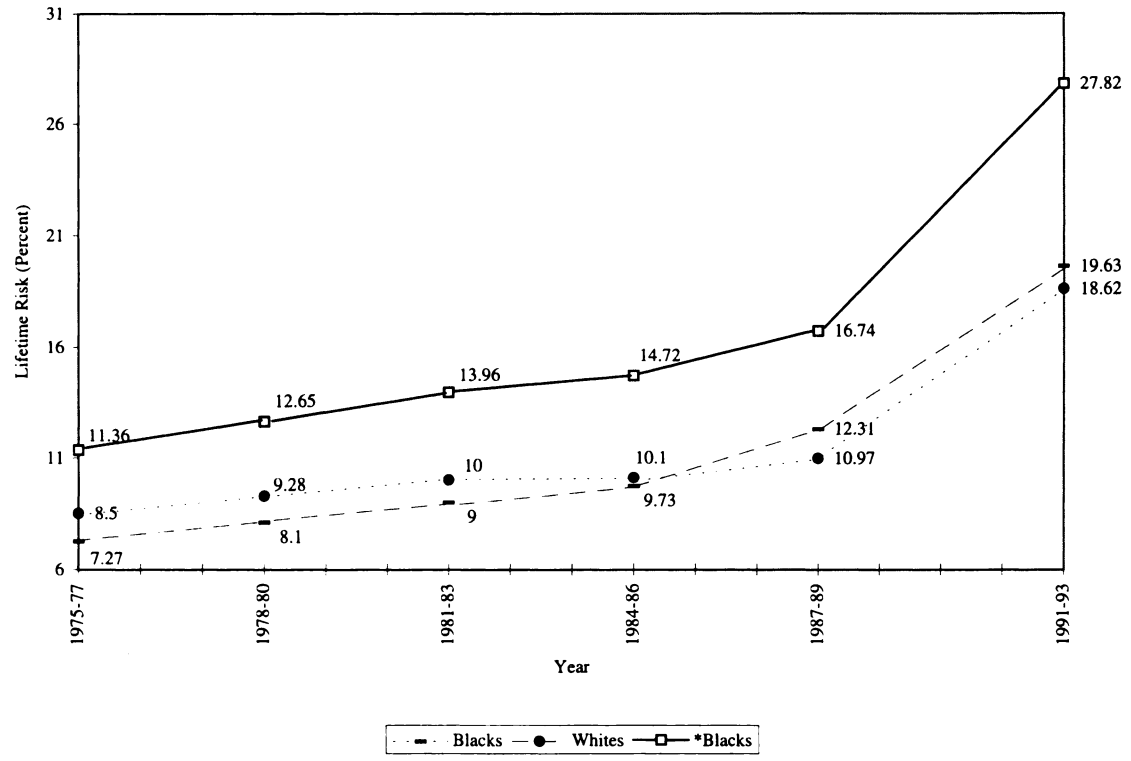

Fig. 2. Lifetime risk of developing prostate cancer by time period and race. ■, blacks; ●, whites; □, blacks, assuming blacks have the same noncancer mortality rates as whites.

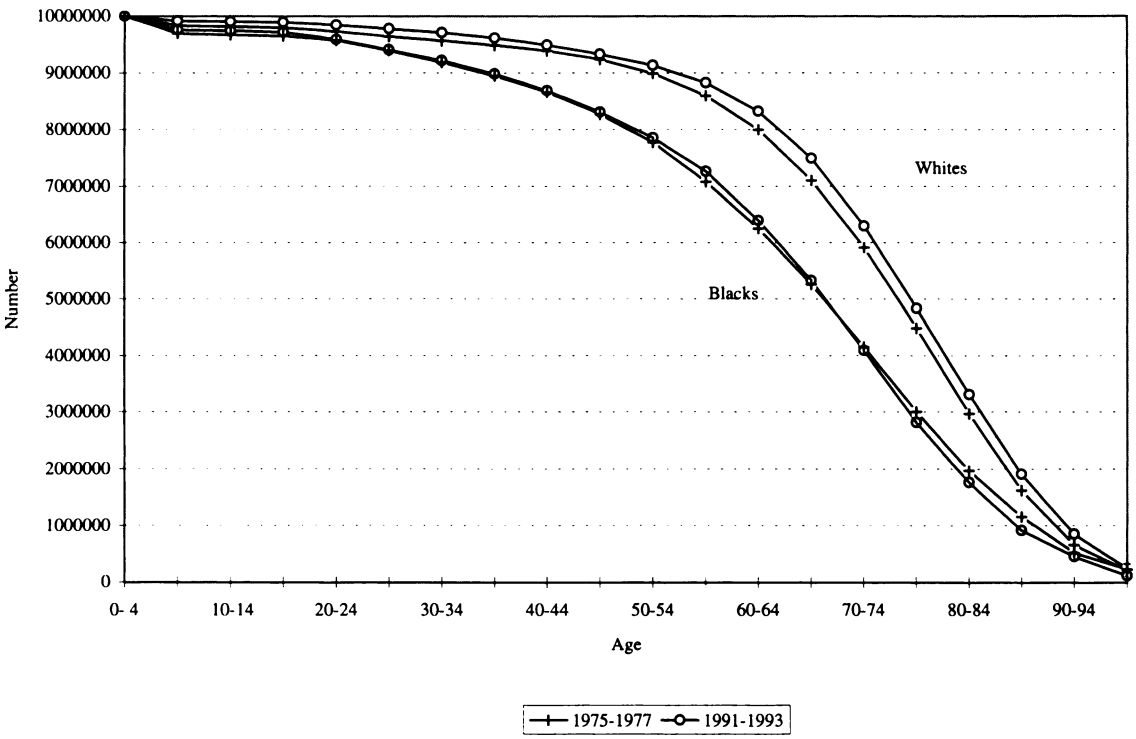

Fig. 3. Total alive and prostate cancer free at the beginning of each age interval by time period and race. +, 1975–1977; ○, 1991–1993.

By reporting the unadjusted lifetime risk estimates for whites and blacks in the same graph (Fig. 2), we see a crossover after 1984–1986 in magnitude of lifetime risk estimates for blacks and whites. The *solid line* represents the trend in lifetime risk estimates that occur for blacks if we assume they have the same mortality rates as whites for causes other than prostate cancer.

Fig. 3 shows for hypothetical cohorts of whites and blacks the total number alive and cancer free at the beginning of each age interval from 1975–1977 and 1991–1993. We see here that fewer blacks survive to older ages when age-specific rates for prostate cancer become large. Also, whereas whites have had improvements in mortality at all ages, blacks have experienced little improvement in mortality at any age over the study period.

Discussion

The increasing lifetime risk estimates for both blacks and whites can be attributed to increasing incidence rates and lower mortality rates due to non-prostate cancer, such that men are living to older ages when prostate cancer incidence rises.

Autopsy studies consistently find high percentages of prostate cancer in older men who have died of causes other than prostate disease (17–18). Carter *et al.* (19) report that the prevalence of latent prostate cancer at autopsy in United States men ranges from 3% for ages 40–44 to 72% for ages 85+. It follows that, because of the large number of subclinical prostate disease in the population, an increase in the number of workups of the prostate gland will result in increased detection rates. Indeed, widespread use of TURP between 1973 and 1986 to treat benign prostatic hypertrophy was shown to be highly correlated with increasing prostate cancer incidence rates (20). More aggressive physician case findings may also explain some of the gradual increase in incidence during the 1970s and 1980s (20). A sharp increase in prostate cancer incidence rates in the late 1980s and early 1990s has been attributed to the use of PSA screening (21). However, after a sharp increase in prostate cancer incidence rates from 1989 to 1992, there is now evidence that the incidence rates are returning to the secular trend existing before PSA screening (22), supporting the notion that the rapid rise and subsequent fall in rates represent a screening artifact.

Our analysis assumes that the age-specific trends in prostate cancer incidence rates from 1975 to 1989 would have continued through 1991–1993 had PSA screening not been introduced. However, it is difficult and perhaps impossible to know, given the complex dynamic of detection methods, access to medical care, stage, and age migration, the extent to which this assumption holds. Nevertheless, the time period over which the age-specific secular trends are assumed to continue in the absence of PSA screening is fairly short.

Differences in lifetime risk estimates between whites and blacks may be due in part to disparity in incidence rates, inasmuch as they reflect differential detection and screening practices. Researchers at the National Cancer Institute are currently assessing PSA and TURP procedure rates using physician and hospital claims data from 1986–1994 for men 65 years of age and older who are entitled to Medicare, are not enrolled in a health maintenance organization, are in one of the nine SEER registries, and received the procedure before a prostate cancer diagnosis. A preliminary finding suggests that racial disparities in PSA and TURP procedure rates are too small to explain any differences in incidence and, consequently, lifetime risk estimates (data not shown).

In view of the larger age-group-specific prostate cancer incidence rates in blacks, the higher lifetime risk estimates for whites since 1987 seem to be a contradiction, which can be understood by examining the age distribution of prostate cancer and of mortality due to other causes. Less than 1% of incident cases occurred before age 50 in blacks and whites in the SEER areas considered from 1975–1993. As reported in Fig. 3, mortality due to causes other than prostate cancer is higher for blacks in younger age groups, when the prevalence of prostate cancer is relatively low. Consequently, fewer blacks in the hypothetical cohort survive to older ages when age-specific rates for prostate cancer become large. Although mortality rates improve over the study period, they do so more for whites than blacks. Consequently, lower mortality rates from other causes seem to be the primary reason why the lifetime risk of whites exceeds that of blacks in the more recent time periods.

Although this study has shown that the hypothetically constructed lifetime risk measure is sensitive to changes in mortality and detection, changes in other potential risk factors (*e.g.*, sexual practices, diet, occupation, and comorbid disease) may also play a role. The extent to which the sharply increasing lifetime risk estimates have resulted from prostate cancer risk factors rather than from changes in mortality and detection is unclear. However, after accounting for changing mortality and PSA screening, the increase in lifetime risk estimates over the study period is relatively small (as shown in Fig. 1, *a* and *b*). The remaining increase may reflect "real" risk factors, an increasing amount of detected latent prostate disease, or a combination of the two. Nevertheless, it is important that the observed rising lifetime risk estimates not be interpreted as an increase in the amount of progressive prostate disease in the population, thereby causing an unnecessarily heightened level of concern (23). Rather, increasing trends in lifetime risk estimates of prostate cancer suggest, in large part, longer life expectancy and new and increasingly sensitive detection methods.

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