

# Trends in Prostate Cancer Mortality among Black Men and White Men in the United States

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**BACKGROUND.** Prostate cancer mortality rates in the United States declined sharply after 1991 in white men and declined after 1992 in black men. The current study was conducted to investigate possible mechanisms for the declining prostate cancer mortality rates in the United States.

**METHODS.** The authors examined and compared patterns of prostate cancer incidence, survival rates, and mortality rates among black men and white men in the United States using the 1969–1999 U.S. prostate cancer mortality rates and the 1975–1999 prostate cancer incidence, survival, and incidence-based mortality rates from the Surveillance, Epidemiology, and End Results (SEER) Program for the U.S. population. The SEER data represent approximately 10% of the U.S. population.

**RESULTS.** Prostate cancer incidence and mortality rates showed transient increases after 1986, when the U.S. Food and Drug Administration approved the use of prostate specific antigen (PSA) testing. The age-adjusted prostate cancer mortality rates for men age 50–84 years, however, have dropped below the rate in 1986 since 1995 for white men and since 1997 for black men. In fact, for white men ages 50–79 years, the 1998 and 1999 rates were the lowest observed since 1950. Incidence-based mortality rates by disease stage revealed that the recent declines were due to declines in distant disease mortality. Moreover, the decrease in distant disease mortality was due to a decline in distant disease incidence, and not to improved survival of patients with distant disease.

**CONCLUSIONS.** Similar incidence, survival, and mortality rate patterns are seen in black men and white men in the United States, although with differences in the timing and magnitude of recent rate decreases. Increased detection of prostate cancer before it becomes metastatic, possibly reflecting increased use of PSA testing after 1986, may explain much of the recent mortality decrease in both white men and black men. *Cancer* 2003;97:1507–16.

Published 2003 by the American Cancer Society.\*

DOI 10.1002/cncr.11212

**KEYWORDS:** prostate cancer, incidence, survival, mortality, prostate specific antigen.

**D**uring the past 20 years, there have been dynamic changes in prostate cancer incidence rates, due in large part to changing medical practices. The first of these changes occurred with the increased use of transurethral resection of the prostate (TURPS) in the early 1970s to the middle 1980s.<sup>1</sup> Subsequently, the prostate specific antigen (PSA) test gained U.S. Food and Drug Administration (FDA) approval in 1986 for use in monitoring prostate cancer recurrence and in 1994 for aiding in the detection of prostate cancer. The diagnostic use of PSA after 1986 led to similar prostate cancer incidence rate increases in white men and black men: Incidence rates rose 108% from 1986 to a peak in 1992 for white males and rose 104% from 1986

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Received July 2, 2002; revision received October 31, 2002; accepted November 4, 2002.

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to a peak in 1993 for black males.<sup>2-5</sup> Declines in distant disease incidence rates began in 1991 and occurred while localized and regional disease incidence rates were still increasing.<sup>2,3,6-11</sup> Declines in prostate cancer mortality after 1991 have been noted.<sup>10,12,13</sup>

Some have indicated that these patterns provide evidence of beneficial effects of PSA testing.<sup>10,14</sup> They note that the patterns of stage specific incidence rates and mortality rates are consistent with stage migration from distant disease to earlier stages of disease due to PSA screening. That is, the data are consistent with the hypothesis that PSA use led to the increased detection of tumors in the localized or regional stage and that some of these PSA-detected tumors would have been diagnosed clinically a few years later in the distant stage if they had not been detected earlier by PSA screening. This stage migration or stage shift is evidenced by the initial increase in localized/regional disease incidence and subsequent decline in distant disease incidence rates.<sup>10,14</sup>

Prostate cancer mortality rates among white men through 1995 showed declining trends after 1991.<sup>13</sup> However, it was suggested that the mortality decrease may have been due to errors in death certification associated with the initial large increase and subsequent sharp decrease in prostate cancer incidence rates (i.e., if a certain percentage of deaths in men diagnosed with prostate cancer but dying of another cause incorrectly are assigned prostate cancer as the cause of death, then prostate cancer mortality rates will tend to rise and fall with prostate cancer incidence rates).<sup>13,15</sup> The eventual declines in prostate cancer mortality rates below their 1986 prescreening level for white men, however, cannot be explained by death certification errors because prostate cancer incidence rates at the time of the mortality decrease in the 1990s were still well above the level of incidence rates in 1986, prior to the increased use of PSA testing.<sup>16</sup> To further our understanding of the nature of the recent decrease in prostate cancer mortality, we examined the latest prostate cancer incidence, survival, and mortality rates for black men and compared their temporal patterns with the patterns observed in white men.

## MATERIALS AND METHODS

### Data Sources and Descriptions

Incidence and survival rates were obtained from population-based data collected by the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute. Data were used on white men and black men with prostate cancer who were diagnosed from 1975 to 1999 among residents of nine geographic areas: Connecticut, Hawaii, Iowa, New

Mexico, Utah, Atlanta, Detroit, Seattle-Puget Sound, and San Francisco-Oakland. The annual incidence rates were age-adjusted to the 2000 U.S. population by direct standardization.<sup>17</sup> One-year, 3-year, and 5-year prostate cancer specific survival rates by stage at diagnosis were examined for the diagnostic years 1975-1999. Incidence and survival were calculated using the 2002 SEER\*Stat CD-ROM program.<sup>17</sup>

The categories for tumor extent (disease stage) at the time of diagnosis used in this report were localized/regional disease, distant disease, and unstaged disease. The overall incidence rates were for the total invasive tumors, including unstaged tumors but excluding in situ lesions. *Localized disease* refers to an invasive neoplasm confined entirely to the prostate. *Regional disease* refers to a neoplasm that has extended beyond the limits of the prostate directly into surrounding organs, tissues, or regional lymph nodes. *Distant disease* refers to a neoplasm that has spread to remote sites of the body. *Unstaged disease* indicates tumors for which insufficient information was available to permit accurate assignment of a stage. Since 1995, SEER reports have combined localized and regional disease in calculating incidence rates for prostate cancer.<sup>17</sup> Thus, localized and regional disease were combined for every year since 1975 in analyses of prostate cancer incidence.<sup>13</sup>

Normally, mortality data are restricted to the information on death certificates, such as race, gender, and age at death. However, in population-based SEER cancer registries, the incidence data on individuals are linked to their mortality outcomes. Therefore, it is possible to examine mortality rates by variables determined at diagnosis, such as stage at diagnosis. This special mortality measure is termed *incidence-based mortality* (IBM).<sup>18,19</sup> In this report, we examine SEER-area IBM rates by stage at diagnosis to determine which stages are responsible for recent declines in mortality rates. To prevent double counting of prostate deaths in the IBM measures, only sole primary or first primary diagnosed prostate cancer were used in the calculation of the IBM rates.

The prostate cancer mortality rates are calculated from data collected by the National Center for Health Statistics, which receives death certificates from the states and compiles mortality data by race, gender, age, year, and cause of death.<sup>20</sup> For the current study, only white men and black men in the United States who reportedly had an underlying cause of death of prostate cancer were included. The mortality rates were age-adjusted to the 2000 U.S. population by direct standardization. The age groups used for the incidence and mortality data include ages  $\geq 50$  years, 50-84 years, 50-59 years, 60-69 years, 70-79 years,

80–84 years, and  $\geq 85$  years. For white men and black men, mortality rates are reported from 1969 through 1999.

### Statistical Analysis

To allow simultaneous adjustment for age at death, calendar year of death, and year of birth, age-period-cohort models were fit to the prostate cancer mortality rates using 1-year age and calendar-period intervals.<sup>21</sup> These analyses were based on 34 1-year age intervals, ranging from age 50 years through age 83 years, and 30 1-year calendar-period intervals, ranging from 1969 through 1998. This resulted in 63 2-year birth-cohort intervals, ranging from 1885–1886 through 1947–1948. The significance of changes in the slope of the calendar-period risk curve or the birth-cohort risk curve were evaluated using linear contrasts.

A standard age-period-cohort analysis is performed using Poisson regression with a logarithmic link and linear predictor,  $\alpha_i + \pi_j + \lambda_k$ , for the rate corresponding to age group  $i$ , calendar period  $j$ , and birth cohort  $k$ .<sup>21</sup> To evaluate the change in slope of the calendar-period risk curve in 1991, the following difference between two linear contrasts was used:<sup>21</sup>

$$3\pi_{1997} + 2\pi_{1996} + \pi_{1995} - \pi_{1993} - 2\pi_{1992} - 3\pi_{1991} - (3\pi_{1991} + 2\pi_{1990} + \pi_{1989} - \pi_{1987} - 2\pi_{1986} + 3\pi_{1985}).$$

The first contrast characterizes the slope of the calendar-period risk curve between 1991 and 1997, and the second contrast characterizes the slope of the calendar-period risk curve between 1985 and 1991. A significant negative value for this parameter indicates that there was a decrease in the slope of the calendar-period risk curve in 1991. Standard errors of the parameter were adjusted for possible over-dispersion when the deviance for the age-period-cohort fit exceeded the number of residual degrees of freedom.<sup>22</sup> A more complete description of parameters to identify changes in the slope of calendar-period or birth-cohort risk curves has been reported.<sup>23</sup>

The introduction of a beneficial medical intervention usually results in a decrease in the calendar-period risk curve in age-period-cohort analyses of mortality rates, because the impact of improved early detection or improvements in treatment tends to reduce mortality in patients of all ages starting in approximately the same calendar year. Changes in exposure to risk factors usually cause changes in the birth-cohort pattern of risk in an age-period-cohort analysis. Thus, the prostate cancer age-period-cohort analysis can examine whether changes in risk factors are contributing to the declining mortality rates in the 1990s (by looking for decreases in the birth-cohort risk curve that would lead to decreasing rates after 1990). If the decrease in mortality in the 1990s is exclusively a

calendar-period phenomenon, however, then the most likely explanation for the decrease is improvement in the early detection and/or treatment of patients with prostate cancer.

### RESULTS

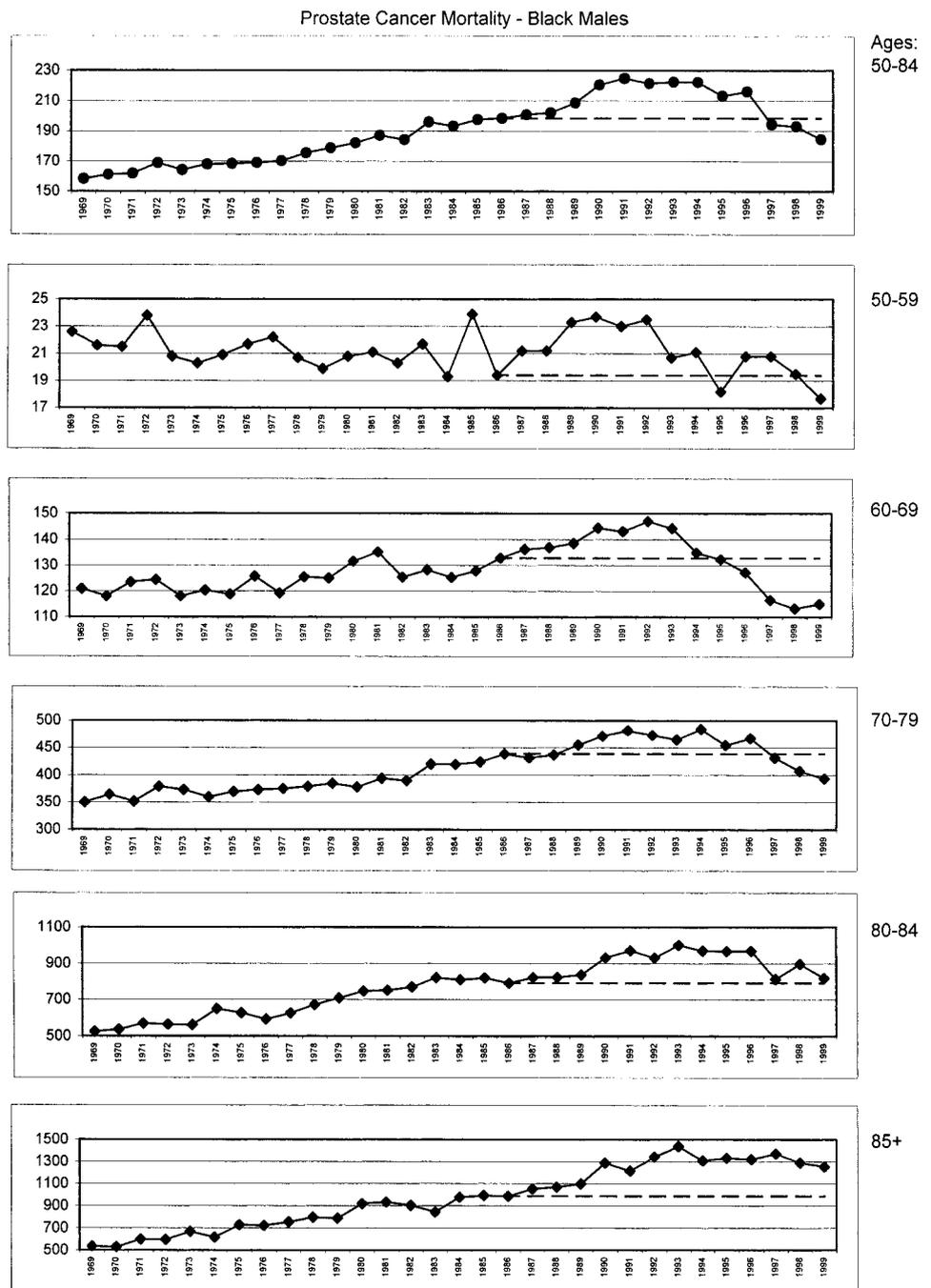
For white males, age-adjusted U.S. prostate cancer mortality rates for men ages 50–84 years, 50–59 years, 60–69 years, 70–79 years, 80–84 years, and  $\geq 85$  years are shown from 1969 through 1999 in Figure 1. The age-adjusted prostate cancer mortality rates for men age 50–84 years peaked in 1991 and declined 27% from 1991 through 1999. For men ages 50–59 years, 60–69 years, and 70–79 years, the 1998 and 1999 rates were at their lowest level since 1950. For all men age  $< 85$  years, the mortality rates after 1995 were lower compared with the rates in 1986, when the FDA first approved the use of PSA. Data on black males are reported in Figure 2, and are discussed below.

To further examine the recent decline in prostate cancer mortality rates, an age-period-cohort analysis was performed (see Figure 3). For white males, there was a significant decrease in the slope of the calendar-period effects curve in 1991 ( $P < 0.0001$ ). The only major change in the birth-cohort effects curve that would have an impact on recent prostate cancer trends for men age  $< 80$  years was an increase in the slope occurring in the 1930s ( $P = 0.02$ ). Thus, the recent decrease in prostate cancer mortality rates appears to be exclusively a calendar-period phenomenon, suggesting that the decrease reflects a change in medical practice rather than a change in prostate cancer risk factors.

For black men, age-adjusted prostate cancer rates by age are shown in Figure 2 from 1969 through 1999. The age-adjusted prostate cancer mortality rates for black men age 50–84 years leveled off around 1990 and then declined 17% from 1994 to 1999. For black men ages 50–59 years, 60–69 years, and 70–79 years, mortality rates have dropped below their levels in 1986. The rates were lower than any time since 1969 after 1997 for black men age 60–69 years and in 1999 for black men age 50–59 years (rates for black men are not available prior to 1969).

The slope of the calendar-period effects curve from the age-period-cohort analysis of prostate cancer mortality rates among black men (Fig. 3) decreased significantly in 1991 ( $P < 0.0001$ ). The birth-cohort effects curve for black men is more dynamic than the birth-cohort effects curve for white men (Fig. 3), but the major decrease in slope around the turn of the century ( $P < 0.0001$ ) occurred too early to explain the decrease in mortality rates in men age 60–79 years in the 1990s (Fig. 2). Thus, similar to white men, the recent decrease in



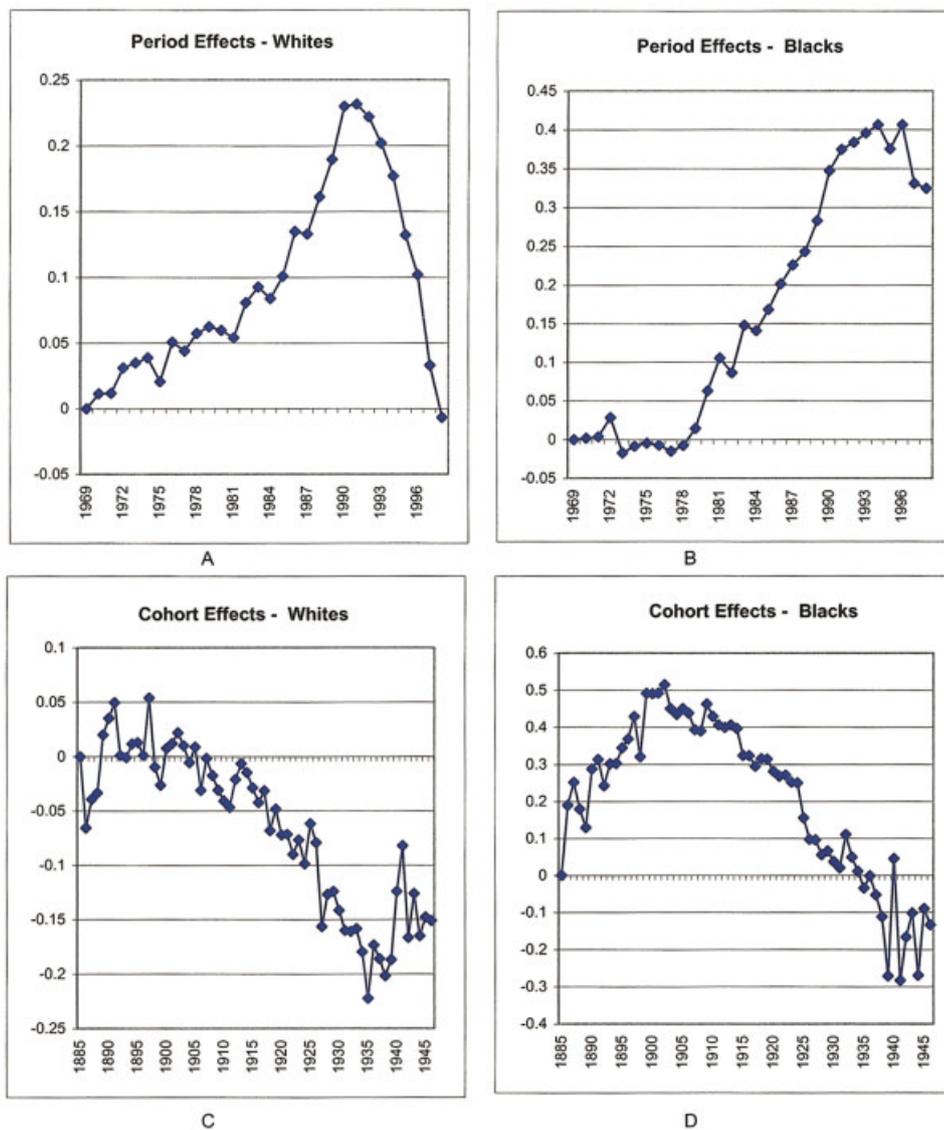


**FIGURE 2.** Age-adjusted prostate cancer mortality rates among black men in the United States between 1969–1999 by age category. The horizontal dotted line indicates the rate in 1986, the year the U.S. Food and Drug Administration first approved prostate specific antigen testing.

and/or declining distant disease incidence rates. For both white men and black men, total and localized/regional disease survival rates have been increasing since the middle 1980s (Table 1). However, distant disease survival rates have changed little for white men or black men, and have been comparable (Table 1). Distant disease is much more lethal compared with localized or regional disease. From 1992 through 1997, the 1-year and 3-year survival rates for men with distant disease were 81% and 49%, respectively, for white men and 81%

and 48%, respectively, for black men. In contrast to these low 1-year and 3-year survival rates for distant disease, the 5-year survival rates for men with localized-regional disease were 96% for white men and 93% for black men. Thus, any intervention leading to increased detection of prostate cancer before it becomes metastatic may have a dramatic and relatively rapid impact on prostate cancer mortality rates.

Panels C and D in Figure 4 show that distant disease incidence rates declined rapidly for both white

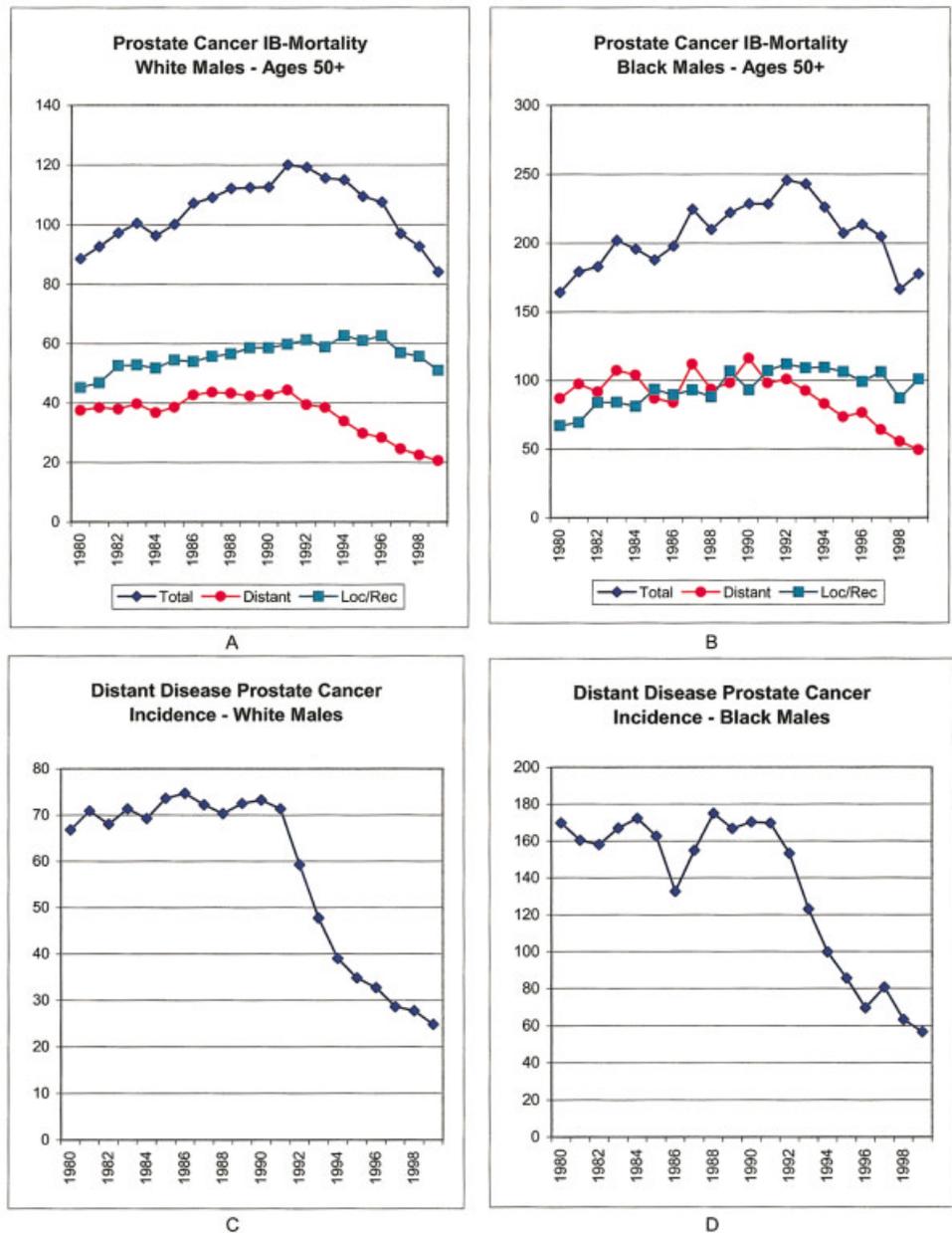


**FIGURE 3.** The calendar-period effects (A,B) and the birth-cohort effects (C,D) from the age-period-cohort analysis of the prostate cancer mortality data for white men and black men, respectively. The maximum likelihood estimates were obtained using the constraint that the most recent birth-cohort effect is 0 (this final constrained value is not plotted).<sup>21</sup>

men and black men after 1991. This decrease began 5 years after the rapid increase in localized and regional disease incidence rates began and before localized-regional disease incidence rates began to decrease (localized-regional disease rates peaked in 1992 for white men and in 1993 for black men). This pattern is consistent with a stage shift due to increased early detection (prior to 1991) in the localized-regional stage of tumors that would have been diagnosed (in the absence of early detection) after 1991 in the distant stage. The pattern is remarkably similar in black men and white men.

Although the declines in distant disease incidence rates from 1986 through 1999 are rather consistent across age groups, the changes in mortality rates are heterogeneous (Table 2). Smaller decreases or even increases in prostate cancer mortality rates were ob-

served in men age  $\geq 80$  years despite the fact that marked decreases in distant disease incidence rates and in IBM rates for distant disease were observed in these elderly men. In addition, the declines in mortality rates for black men are smaller compared with the declines for white men, even when the decreases in distant disease incidence rates are comparable. IBM rates for localized or regional disease increased in the 1990s for the oldest men, which diluted the impact of the decreasing rates of distant disease on mortality. Length of survival after a diagnosis with malignant disease depends on the quality of treatment received as well as the disease stage at the time of diagnosis. SEER records include the most aggressive surgical treatment received within 4 months of diagnosis. There is a marked decrease with age in the percentage of patients with prostate cancer who undergo radical



**FIGURE 4.** Prostate cancer incidence-based mortality (IBM) rates by race and stage at diagnosis for men age  $\geq 50$  years at the time of death by stage at diagnosis for white males (A) and by stage at diagnosis for black males (B) (diamonds, total IBM; squares, localized/regional disease IBM; circles, distant disease IBM). Age-adjusted incidence rates for prostate cancer diagnosed in the distant stage in men age  $\geq 50$  years for white males (C) and for black males (D).

prostatectomy, and prostatectomy rates are consistently lower in black men compared with white men. The percentage of patients with localized or regional disease who underwent radical prostatectomy in the years 1991–1997 dropped consistently with age, from 64% in white men and 48% in black men age 50–59 years to 0.7% in white men and 0.4% in black men age  $\geq 80$  years. Conversely, the percentage of patients for whom there was either no surgical procedure or for whom the most aggressive procedure was a biopsy or TURP increased from 30% in white men and 45% in black men age 50–59 years to 93% in both white men and black men age  $\geq 80$  years.

**DISCUSSION**

The prostate cancer mortality rates for both white men and black men showed marked declines in the 1990s. The decreasing prostate cancer mortality rates are due primarily to declining distant disease mortality rates, which coincide with declining distant disease incidence rates for both black men and white men. The mortality rate decrease began while prostate cancer incidence rates still were increasing and before the mortality rates for localized and regional disease began to fall. Examination of prostate cancer rates from 1990 through 1999 in Asian Americans and Pacific

**TABLE 1**  
Prostate Cancer Specific Survival Rates by Race and Stage at Diagnosis

Years of diagnosis <sup>a</sup> survival <sup>b</sup>	Stage at diagnosis							
	White men				Black men			
	All stages	Local/ regional	Distant	Unstaged	All stages	Local/ regional	Distant	Unstaged
1992-1997								
1 yr	98	100	81	97	98	99	81	97
3 yr	94	98	49	92	92	97	48	90
5 yr <sup>c</sup>	91	96	35	87	87	93	34	84
1981-1985								
1 yr	96	98	84	96	94	98	83	97
3 yr	85	93	50	84	78	91	47	83
5 yr	76	87	33	75	68	83	30	65

<sup>a</sup> Years of diagnosis of prostate cancer.

<sup>b</sup> Probability of not dying from prostate cancer 1 year, 3 years, or 5 years after a diagnosis of prostate cancer.

<sup>c</sup> Five-year cause specific survival rates are for patients who were diagnosed between 1992-1995.

**TABLE 2**  
Age-Adjusted Incidence Rates of Prostate Cancer with Distant Metastases at the Time of Diagnosis and Prostate Cancer Mortality Rates by Race

Age (yrs) <sup>a</sup>	Distant disease incidence rates			Prostate cancer mortality rates		
	1986	1999	Percent change	1986	1999	Percent change
White men						
50-59	9.5	4.7	-50.5	7.3	5.4	-26.0
60-69	47.9	21.5	-55.1	51.3	37.0	-27.9
70-79	140.6	41.2	-70.7	188.9	146.4	-22.5
80-84	238.9	64.0	-73.2	404.9	356.7	-11.9
≥85	229.1	76.6	-66.6	612.4	661.3	+8.0
Black men						
50-59	28.5	18.8	-34.0	19.4	17.7	-8.8
60-69	117.6	50.1	-57.4	132.9	115.1	-13.4
70-79	274.1	89.4	-67.4	438.7	393.6	-10.3
80-84	260.4	147.2	-43.5	791.7	818.8	+3.4
≥85	264.3	129.3	-51.5	986.4	1254.5	+27.2

<sup>a</sup>Age at diagnosis for incidence rates and age at death for mortality rates.

Islanders and in Hispanics also showed declines in distant disease incidence rates and significant declines in prostate cancer mortality (data not shown). These observations are consistent with the hypothesis that the decreasing prostate cancer mortality in the United States is caused by a stage shift resulting from earlier detection of cancer by PSA testing. That is, tumors that, without intervention, would be diagnosed in the lethal, distant stage are being detected early by PSA testing, so that men are diagnosed in the localized or regional stage; the resulting marked improvement in prognosis leads to decreasing mortality rates.<sup>10,14</sup>

There have been recent advances in prostate cancer treatment for patients with locally advanced disease that may be making additional contributions to declines in U.S. prostate cancer mortality rates. Beginning in 1997, a number of studies have shown that patients with locally advanced disease live longer if they receive hormone therapy earlier in the course of their disease.<sup>24-30</sup> In the SEER Program, locally advanced tumors are coded as regional disease. Thus, the improved treatments for patients with locally advanced disease cannot explain the observed decrease in mortality rates for men with distant disease. The IBM mortality rates in Figure 4A indicate that local-

ized/regional disease mortality rates did not begin to decline until 1997. Improved treatment of patients with locally advanced disease may well be contributing to these additional recent declines in prostate cancer mortality rates.

At the international level, recent trends in prostate cancer rates have shown inconsistent patterns across countries. Not all countries have trends similar to those in the United States.<sup>31,32</sup> Canada, which has shared similar patterns for breast cancer with the United States,<sup>33</sup> has had increases in incidence and subsequent declines in mortality similar to those seen in the United States.<sup>34</sup> In the United Kingdom, there have been recent declines in prostate cancer mortality rates, but there has been no prostate cancer screening program and no large increase in prostate cancer incidence.<sup>35–37</sup> Trends in survival rates in the United Kingdom do not indicate that prostate cancer treatments are affecting the declines.<sup>38</sup> Western Australia has a prostate cancer screening program but has not seen declines in prostate cancer mortality rates.<sup>39</sup> Conversely, Tyrol, Austria, where screening has been given, shows mortality rate declines similar to those in the United States as well as evidence that a stage shift has led to decreasing mortality.<sup>40</sup> In any country, comprehensive analyses of incidence and survival rates by stage at diagnosis and of mortality rates by age are required to make inferences about the possible causes for the declines or lack of declines in that country. Application of such analyses to U.S. data shows a consistent pattern for both black men and white men.

Evaluation of U.S. trends in distant disease incidence rates and prostate cancer mortality rates by age and race (Table 2) suggests that comparisons of prostatectomy rates also should be included in investigation of international trends. Comparisons of U.S. prostatectomy rates by age and race indicate that the impact of decreases in distant disease rates on overall prostate cancer mortality rates can be obscured in the absence of aggressive surgical treatment of patients with prostate cancer that has not metastasized. Thus, countries with less aggressive surgical treatment of patients with localized prostate cancer compared with the United States will observe a smaller decrease in overall prostate cancer mortality rates, even if the level of PSA screening is equal to that in the United States.

Although descriptive studies cannot provide absolute evidence of cause and effect, the analyses presented in this article show that the observed patterns of prostate cancer rates are consistent with a beneficial effect of PSA testing on prostate cancer mortality. The recent decrease in prostate cancer mortality rates has come at the cost of a very large increase in the number of men treated for prostate cancer since

1986.<sup>37</sup> With prostate cancer mortality rates in both white men and black men currently at their lowest levels in several decades for many age groups, a complete delineation of the benefits and limitations of PSA testing and subsequent treatments is needed to allow informed decisions about PSA use.<sup>41</sup> This issue is particularly important for black men in the United States who still have some of the highest prostate cancer rates in world.

## REFERENCES

1. Potosky AL, Kessler L, Gridley G, Brown CC, Horm JW. Rise in prostatic cancer incidence associated with increased use of transurethral resection [see comments]. *J Natl Cancer Inst.* 1990;82:1624–1628.
2. Brawley OW. Prostate cancer incidence and patient mortality: the effects of screening and early detection. *Cancer.* 1997;80:1857–1863.
3. Farkas A, Schneider D, Perrotti M, Cummings KB, Ward WS. National trends in the epidemiology of prostate cancer, 1973 to 1994: evidence for the effectiveness of prostate-specific antigen screening. *Urology.* 1998;52:444–448.
4. Gilliland F, Becker TM, Smith A, Key CR, Samet JM. Trends in prostate cancer incidence and mortality in New Mexico are consistent with an increase in effective screening. *Cancer Epidemiol Biomarkers Prev.* 1994;3:105–111.
5. Potosky AL, Miller BA, Albertsen PC, Kramer BS. The role of increasing detection in the rising incidence of prostate cancer. *JAMA.* 1995;273:548–552.
6. Gilliland FD, Welsh DJ, Hoffman RM, Key CR. Rapid rise and subsequent decline in prostate cancer incidence rates for New Mexico, 1989–1993. *Cancer Epidemiol Biomarkers Prev.* 1995;4:797–800.
7. Stephenson RA, Smart CR, Mineau GP, James BC, Janerich DT, Dibble RL. The fall in incidence of prostate cancer. On the down side of a prostate specific antigen induced peak in incidence—data from the Utah Cancer Registry [see comments]. *Cancer.* 1996;77:1342–1348.
8. Schwartz KL, Severson RK, Gurney JG, Montie JE. Trends in the stage specific incidence of prostate cancer in the Detroit metropolitan area, 1973–1994. *Cancer.* 1996;78:1260–1266.
9. Newcomer LM, Stanford JL, Blumenstein BA, Brawer MK. Temporal trends in rates of prostate cancer: declining incidence of advanced stage disease, 1974 to 1994. *J Urol.* 1997;158:1427–1430.
10. Smart CR. The results of prostate cancer screening in the U.S. as reflected in the surveillance, epidemiology, and end results program. *Cancer.* 1997;80:1835–1844.
11. Merrill RM, Brawley OW. Prostate cancer incidence and mortality rates among white and black men [see comments]. *Epidemiology.* 1997;8:126–131.
12. Mettlin CJ, Murphy GP. Why is the prostate cancer death rate declining in the United States [editorial]? [Published erratum appears in *Cancer.* 1998;82:1802.] *Cancer.* 1998;82:249–251.
13. Hankey BF, Feuer E, Clegg LX, et al. Cancer surveillance series: interpreting trends in prostate cancer—Part I: evidence of the effects of screening in recent prostate cancer incidence, mortality and survival rates. *J Natl Cancer Inst.* 1999;91:1017–1024.

14. Gann PH. Interpreting recent trends in prostate cancer incidence and mortality [editorial]. *Epidemiology*. 1997;8:117-120.
15. Feuer EJ, Merrill RM, Hankey BF. Cancer surveillance series: interpreting trends in prostate cancer—Part II: cause of death misclassification and the recent rise and fall in prostate cancer mortality. *J Natl Cancer Inst*. 1999;91:1025-1032.
16. Tarone RE, Chu KC, Brawley OW. Implications of stage-specific survival rates in assessing recent declines in prostate cancer mortality rates. *Epidemiology*. 2000;11:167-170.
17. National Cancer Institute Surveillance, Epidemiology, and End Results Program. SEER\*Stat version 4.2 [SEER cancer incidence public-use data base, 1973-1999]. Bethesda: National Cancer Institute, 2001.
18. Chu KC, Miller BA, Feuer EJ, Hankey BF. A method for partitioning cancer mortality trends by factors associated with diagnosis: an application to female breast cancer. *J Clin Epidemiol*. 1994;47:1451-1461.
19. Chu KC. Mortality rates by stage-at-diagnosis. *Semin Surg Oncol*. 1994;10:7-10.
20. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. National Center for Health Statistics. Hyattsville, MD: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. National Center for Health Statistics, Division of Data Services, 2002. Available at: <http://www.cdc.gov/nchs/>.
21. Tarone RE, Chu KC. Evaluation of birth cohort patterns in population disease rates. *Am J Epidemiol*. 1996;143:85-91.
22. McCullagh P, Nelder JA. Generalized linear models. London: Chapman and Hall, 1989.
23. Chu KC. Re: "Temporal trends in diabetes mortality among American Indians and Hispanics in New Mexico: birth cohort and period effects" [letter]. *Am J Epidemiol*. 1998;147:796-800.
24. Pilepich MV, Caplan R, Byhardt RW, et al. Phase III trial of androgen suppression using goserelin in unfavorable-prognosis cancer of the prostate treated with definitive radiotherapy: report of Radiation Oncology Group Protocol 85-31. *J Clin Oncol*. 1997;15:1013-1021.
25. Medical Research Council Prostate Cancer Working Party Investigators Group. Immediate versus deferred treatment for advanced prostatic cancer. Initial results of the Medical Research Council Trial. *Br J Urol*. 1997;79:235-246.
26. Bolla M, Gonzalez D, Warde P, et al. Improved survival in patients with locally advanced prostate cancer treated with radiotherapy and goserelin. *N Engl J Med*. 1999;337:295-300.
27. Roach M, Lu J, Pilepich MV, et al. Predicting long term survival, and the need for hormonal therapy: a meta-analysis of RTOG prostate cancer trials. *Int J Radiat Oncol Biol Phys*. 2000;47:617-627.
28. Messing EM, Manola J, Sarosdy M, Wilding G, Crawford ED, Trump D. Immediate hormonal therapy compared with observation after radical prostatectomy and pelvic lymphadenectomy in men with node-positive prostate cancer. *N Engl J Med*. 1999;341:1781-1788.
29. Pilepich MV, Winter K, John MJ, et al. Phase III Radiation Therapy Oncology Group (RTOG) Trial 86-10 of androgen deprivation adjuvant to definitive radiotherapy in locally advanced cancer of the prostate. *Int J Radiat Oncol Biol Phys*. 2001;50:1243-1252.
30. Hanks GE, Lu J, Machtay M, et al. RTOG Protocol 92-02: a Phase III trial of the use of long term total androgen suppression following neoadjuvant hormonal cytotoreduction and radiotherapy in locally advanced cancer of the prostate. *Int J Radiat Oncol Biol Phys*. 2000;48(Suppl 1):112.
31. Hsing AW, Tsao L, Devesa SS. International trends and patterns of prostate cancer incidence and mortality. *Int J Cancer*. 2000;85:60-67.
32. Oliver SE, May MT, Gunnell D. International trends in prostate-cancer mortality in the "PSA era." *Int J Cancer*. 2001;92:893-898.
33. Tarone RE, Chu KC, Gaudette LA. Birth cohort and calendar period trends in breast cancer mortality in the United States and Canada. *J Natl Cancer Inst*. 1997;89:251-256.
34. National Cancer Institute of Canada. Canadian cancer statistics 2001. Toronto: National Cancer Institute of Canada, 2001.
35. Oliver SE, Gunnell D, Donovan JL. Comparison of trends in prostate-cancer mortality in England and Wales and the USA. *Lancet*. 2000;355:1788-1789.
36. Oliver SE, Gunnell D, Donovan JL. Correction—comparison of trends in prostate-cancer mortality in England and Wales and the USA. *Lancet*. 2000;356:1278.
37. Tannock IF. Eradication of a disease: how we cured symptomless prostate cancer. *Lancet*. 2002;359:1341-1342.
38. Majeed A, Babb P, Jones J, Quinn M. Trends in prostate cancer incidence, mortality and survival in England and Wales, 1971-1998. *BJU Int*. 2000;85:1058-1062.
39. Threlfall TJ, English DR, Rouse IL. Prostate cancer in Western Australia: trends in incidence and mortality from 1985 to 1996. *Med J Aust*. 1998;169:21-24.
40. Bartsch G, Horninger W, Klocker H, et al. Prostate cancer mortality after introduction of prostate-specific-antigen mass screening in the federal state of Tyrol, Austria. *Urology*. 2001;58:417-424.
41. Smith RA, Cokkinides V, von Eschenbach AC, et al. American Cancer Society guidelines for the early detection of cancer. *CA Cancer J Clin*. 2002;52:8-22.