

Determinants of Mortality Following a Diagnosis of Prostate Cancer in Veterans Affairs and Private Sector Health Care Systems

Vincent L. Freeman, MD, MPH, Ramon Durazo-Arvizu, PhD, Ahsan M. Arozullah, MD, MPH, and LaShon C. Keys, MEd, RD

Prostate cancer remains the most commonly occurring cancer and the second most common cause of cancer-related mortality among US males.¹ The American Cancer Society has projected that 220 900 new cases and 28 900 prostate cancer–related deaths will occur in the year 2003 alone.² One of the most striking features of the disease in the United States is the marked and persistent racial/ethnic disparity in incidence and mortality. Compared with their White counterparts, Black men with prostate cancer tend to have shorter survival times even after we accounted for age, stage, and histological differences at the time of diagnosis.³ In the United States, race/ethnicity is a proxy for social and demographic forces that influence health care access, utilization, and treatment patterns contributing to group differences in outcomes of chronic disease in general. Therefore, the most prominent hypotheses put forth to explain US racial disparities in prostate cancer have been patient and provider behavioral barriers and health care system access failure.^{4–8}

The Veterans Health Administration (VHA), the largest integrated health care system in the United States, attempts to equalize access by significantly reducing financial barriers to health-related services.⁹ This setting seems particularly well suited to an examination of the impact of this parity in access on racial disparities in survival among men with prostate cancer. Therefore, the primary focus of this study was to compare the relation of race and cause-specific mortality in the VHA health care system with that in the private sector. A secondary purpose was to compare the determinants of mortality among prostate cancer patients in these health care settings to shed light on social and clinical characteristics that may contribute to or modify racial differences in mortality.

Objectives. We compared patterns of mortality among men with prostate cancer at 2 Department of Veterans Affairs (VA) and 2 private-sector hospitals in the Chicago area.

Methods. Mortality rates for 864 cases diagnosed between 1986 and 1990 were estimated using Cox proportional hazards models that incorporated age; income; cancer stage, differentiation, and treatments; and baseline comorbidity.

Results. Race tended to associate with all-cause mortality irrespective of health care setting (Blacks vs Whites: hazard rate ratio [HRR]=1.68 [95% confidence interval (CI)=1.06, 2.67]; $P < .001$ in the private sector; HRR=1.50 [95% CI=0.94, 2.38]; $P = .088$ in the VA). However, comorbidity determined risk in the VA, whereas age and income predicted risk in the private sector.

Conclusions. Determinants of all-cause mortality in men with prostate cancer vary according to health care setting. (*Am J Public Health.* 2003;93:1706–1712)

METHODS

Study Design and Patient Population

We used a retrospective cohort design. The cohort consisted of all cases of adenocarcinoma of the prostate (*International Classification of Diseases, Ninth Revision, Clinical Modification [ICD-9-CM] code 187.0*¹⁰) diagnosed among Black and White men at 2 Department of Veterans Affairs (VA) hospitals and 2 private university medical centers between January 1, 1986, and December 31, 1990. Of the 1163 cases of adenocarcinoma of the prostate originally identified in the tumor registry of each hospital, 1007 (87%) had medical records available for detailed review.

Baseline Characteristics

Inpatient and outpatient medical records were abstracted on-site by 2 trained reviewers. The data abstracted from each record included demographics (name, race, date of birth, Social Security number, zip code), tumor characteristics (stage, differentiation, Gleason sum), processes of diagnosis and management (indication for diagnostic evaluation, method of diagnosis, clinical date of diagnosis, pathological date of diagnosis, metastatic evaluation, first-course treatments), comorbidities present at the time of diagnosis,

and subsequent health outcomes (disease recurrence/progression date and location, vital status, and cancer status).^{11–13} Staging was based on all of the evidence available in the original patient record, and assignments were made according to the American Joint Committee on Cancer TNM (tumor, nodes, metastasis) staging system.¹¹ Comorbid conditions present at the time of diagnosis was measured with the index developed by Charlson and others.¹⁴

Agreement between the 2 reviewers was monitored for race, clinical and pathological diagnosis date, tumor differentiation, stage, treatment modality, treatment date, comorbidities, Charlson scoring, last contact date, and vital status in a 20% random sample of all records reviewed. The level of agreement was high ($\kappa = 0.95–0.98$).

Exclusions

Ninety of the cases were excluded because cancers were T1a-stage lesions, which are believed to be clinically insignificant, and 53 others were excluded because of incomplete data. This exclusion left 864 records (385 in the VA, 479 in the private sector) for analysis. Blacks made up 38.8% of the analytic cohort, and 64% of Black patients were diagnosed at one of the VA hospitals. Of the records not

available for detailed review, 42% were for Black patients.

Outcomes and Their Ascertainment

Follow-up ended on December 31, 2000, with death from prostate cancer and death from other causes as the primary outcomes of interest. The tumor registry at each hospital was our primary source for vital status ascertainment. We also conducted multiple searches of the National Death Index of the National Center for Health Statistics and the VA Beneficiary Identification and Record Locator System over a 24-month period beginning January 1, 2000. Causes of death were established on the basis of death certificate review performed by an independent physician—reviewer blinded to the study's hypotheses. Causes of death were coded according to *ICD-9-CM*.¹⁰

Statistical Methods

All analyses were performed for the entire cohort and within each health care setting. Baseline demographic and clinical characteristics of Blacks were compared with those of Whites using the 2-sample *t* test for continuous traits and χ^2 analysis for categorical ones. Kaplan–Meier survival distributions were computed for subgroups of men defined by race (Black, White), tumor differentiation (well, moderate, poor), stage (local/regional, distant), and Charlson score (0–1, 2–3, ≥ 4), and these distributions were compared using the Mantel–Haenszel statistic.¹⁵

Local-/regional-stage tumors are defined as those confined to the prostate gland or extraprostatic extension, with or without regional lymph node involvement (grouping=T1b–3 any, N0–3, M0). Distant-stage tumors are defined as distant metastases, including disease that has spread beyond the pelvis to areas including bones, liver, lungs, and brain (grouping=T4, NX–0, M0, or T any, N any, M1). In this study, local- and regional-stage cases were combined because they would be candidates for aggressive curative therapy. Unstaged cases were combined with distant cases because their respective survival distributions were not significantly different ($\chi^2_1=0.30$; $P=.58$). The 3 histological subgroups used in our analyses—well, moderately, and poorly differentiated tumors—gen-

erally corresponded to Gleason sums of 2–4, 5–6, and 7–10, respectively. Charlson comorbidity scores were available for all but 2.6% of patients. These values were imputed by a multiple regression imputation method in which the available comorbidity score was regressed on age at diagnosis, race, hospital of origin, stage, differentiation, and comorbid diseases or conditions (coronary artery disease, hypertension, tobacco and alcohol usage).

A stratified Cox proportional hazards regression model was used to account for the baseline hazard associated with the hospital where the case originated.¹⁶ The regression model included age, race (Black, White), Charlson comorbidity score, tumor differentiation (well, moderately, poorly differentiated), stage (local/regional, distant), first-course treatment (surgery, radiation, diethylstilbestrol, castration, observation) and mean household income per capita by race for zip code of residence according to the 1990 US Census.^{17,18} Potential interactions between comorbidity with stage and race were evaluated by including comorbidity-by-stage and comorbidity-by-race terms in the regression model. Ninety-five percent CIs for hazard rates of death from any cause for Blacks relative to Whites by comorbidity level (Charlson scores=0, 1, 2, 3, 4, and ≥ 5) were computed with a bias-corrected bootstrap approach based on 2500 bootstrap samples.¹⁹ Different regression models were considered before we reached the final models. The proportional hazards assumption in each was tested with time-dependent coefficients and Schoenfeld residuals.²⁰ No violations of the proportional hazards assumptions were observed ($P=.23$ to $.97$). The statistical analyses were performed with Stata Version 7.0.²¹

RESULTS

Baseline Characteristics

Table 1 shows a comparison of baseline sociodemographic and clinical characteristics of Blacks and Whites in both health care systems. Blacks tended to reside in areas of lower household income per capita (mean income=\$8508 vs \$15 782 for VA patients, \$9852 vs \$20 689 for private-sector patients; $P<.001$), were significantly more likely

to present with distant-stage disease (37.1% vs 29.1% for VA patients, 48.7% vs 23.3% for private-sector patients), and were significantly less likely to receive aggressive curative therapy for local-/regional-stage prostate cancer (62.9% vs 82% for VA patients, 73.3% vs 85.6% for private-sector patients). Within each system, Blacks and Whites had similar levels of comorbidity, but the levels of VA patients appeared to be greater than those of patients in the private sector (mean Charlson score=1.3 for VA patients vs 2.2 for private-sector patients; $P<.001$). In the private sector, Blacks were older (mean=70.7 years vs 68.9 years; $P=.044$), had a higher crude all-cause mortality rate (72.7% vs 48.9%; $P<.001$), and had shorter survival times (mean=4.1 years vs 6.1 years; $P<.001$) compared with their White counterparts. However, significant racial differences in age, crude all-cause mortality, and survival times were not observed in the VA.

Overall Survival by Race and Comorbidity Level

Figure 1 presents Kaplan–Meier survival distributions by race and by comorbidity level. Blacks in the private sector fared significantly worse than did Whites, with a median crude survival time of approximately 3.5 years for Blacks and 8.2 years for Whites ($\chi^2=37.6$; $P<.001$). However, no racial differences in survival were found in the VA, where the median survival for both groups was 5.7 years. Results by comorbidity level were more nuanced. In the private sector, Charlson score groupings of 0–1, 2–3, and 4 or higher yielded significantly distinct survival distributions (median survival=8.3 years, 5.9 years, and 2.7 years, respectively; $\chi^2=53.3$; $P<.001$). However, stratification of VA cases in the same manner did not result in as great a contrast in overall survival. Median survival distribution for Charlson score groupings of 0–1, 2–3, and 4 or higher was 7.0 years, 6.8 years, and 3.2 years, respectively ($\chi^2=25.4$; $P<.001$; for 0–1 vs 2–3 only, $\chi^2=1.44$; $P<.230$).

Predictors of Mortality

Predictors of all-cause and cause-specific mortality in each health care setting are shown in Table 2. In the VA, tumor differentiation (poor vs well), stage at diagnosis

TABLE 1—Baseline Characteristics of Sample Populations: 1986–1990

	Department of Veterans Affairs (n = 385)			Private Sector (n = 479)		
	Blacks	Whites	P	Blacks	Whites	P
Sample size, n (%)	210 (55)	175 (45)	...	117 (24)	362 (76)	...
Mean age, y (SE)	68.5 (0.5)	68.5 (0.5)	.967	70.7 (0.9)	68.9 (0.4)	.044
Mean household income per capita, \$ (SE)	8508 (199)	15 782 (390)	<.001	9852 (250)	20 689 (512)	<.001
Differentiation, n (%)			.025			.033
Well	63 (30.0)	76 (43.4)	...	17 (14.5)	79 (21.8)	...
Moderate	70 (33.3)	46 (26.3)	...	33 (28.2)	125 (34.5)	...
Poor	77 (36.8)	53 (30.3)	...	67 (57.3)	158 (43.7)	...
Stage, n (%)			.044			<.001
Local	92 (43.8)	99 (56.6)	...	40 (34.2)	206 (56.9)	...
Regional	40 (19.1)	25 (14.3)	...	20 (17.1)	72 (19.9)	...
Distant/unstaged ^a	78 (37.1)	51 (29.1)	...	57 (48.7)	84 (23.3)	...
Surgery and/or radiation for local-/regional-stage cancer, n (%)	83 (62.9)	102 (82.2)	<.001	44 (73.3)	238 (85.6)	.033
Mean Charlson score (SE) ^b	2.3 (0.2)	2.1 (0.2)	.234	1.5 (0.2)	1.3 (0.1)	.329
Charlson score group, n (%)			.424			.710
0–1	94 (44.8)	89 (50.9)	...	76 (65.0)	247 (68.2)	...
2–3	73 (34.8)	51 (29.1)	...	27 (23.0)	79 (21.8)	...
≥ 4	43 (20.5)	35 (20.0)	...	14 (12.0)	36 (10.0)	...
Deaths, n (%)	137 (65.2)	108 (61.7)	...	85 (72.7)	177 (48.9)	...
Time to death mean, y (SE)	5.3 (0.2)	5.7 (0.3)	.293	4.1 (0.3)	6.1 (0.2)	<.001

^aPresumed to be distant stage by the treating physician but not staged for 3 Black patients and 2 White patients.

^bScores ranged from 0 to 14.

with risk of death from prostate cancer accounted for their independent relation with all-cause mortality. However, comorbidity as measured with the Charlson index did not predict mortality from any cause in the private sector after adjustment for race, age, income, and histological differences at baseline.

Racial Differences in Survival in Relation to Initial Comorbidity

Figure 2 shows all-cause mortality rates for Blacks relative to Whites by Charlson score after adjustment for age, stage, tumor differentiation, and treatment. The relative excess all-cause mortality experienced by Blacks was inversely related to initial comorbidity. This association was more pronounced in the private sector, where excess mortality rates among Blacks relative to Whites tended to be statistically significant (HRRs=2.26 [95% CI=1.40, 3.45], 2.07 [95% CI=1.45, 2.97], 1.89 [95% CI=1.31, 2.85], 1.73 [95% CI=1.07, 3.07], 1.58 [95% CI=0.84, 3.42], and 1.44 [95% CI=0.65, 3.91], respectively, for Charlson scores 0, 1, 2, 3, 4, and ≥ 5; $P<.05$). We found a parallel trend in the VA system, but none of the relative hazard estimates reached statistical significance (HRRs=1.24 [95% CI=0.82, 1.92], 1.14 [95% CI=0.80, 1.59], 1.05 [95% CI=0.75, 1.37], 0.96 [95% CI=0.67, 1.26], 0.88 [95% CI=0.56, 1.20], and 0.81 [95% CI=0.45, 1.20], respectively).

DISCUSSION

With few exceptions, evidence of racial/ethnic disparities in health and health care is fairly consistent across a broad range of illnesses and health care services.⁴ Some studies suggest that these disparities are either reduced or eliminated in the health care systems of the US Department of Defense and the VA.^{22–26} This has been observed more consistently in studies of the Department of Defense system, which ensures universal access, than in the VA system, which significantly reduces the financial barriers to health care for veterans. Consequently, investigators often make the following inferences regarding racial differences in health outcomes detected in such health care systems: (1) their absence is likely the result of reduc-

(local/regional vs distant) and Charlson score predicted risk of death from any cause (hazard rate ratios [HRRs]=1.68 [95% confidence interval (CI)=1.19, 2.38], 0.26 [95% CI=0.15, 0.44], and 1.16 [95% CI=1.04, 1.29], respectively; $P<.001$ to .003). The relations of tumor characteristics (stage, differentiation) and all-cause mortality appeared to be attributable to significant associations with risk of death from prostate cancer, whereas the relation between Charlson score and all-cause mortality was explained by its significant positive association with risk of death from other causes (HRR=1.36 [95% CI=1.18, 1.56] per unit increase; $P<.001$). Blacks had a 25% to 50% excess in mortality relative to Whites, depending on the cause of death, but these differences did not reach statistical significance.

By contrast, race was a significant predictor of all-cause mortality in the private sector,

even after we accounted for sociodemographic and clinical differences at baseline (Blacks vs Whites: HRR=1.68 [95% CI=1.06, 2.67]; $P<.001$). This association was attributable to racial disparities in risk of death from prostate cancer (Blacks vs Whites: HRR=2.68 [95% CI=1.36, 5.27]; $P<.001$) but not from other causes. Increasing age (in 5-year increments) was also associated with increasing risk of death from any cause (HRR=1.17 [95% CI=1.07, 1.28]; $P=.001$), whereas increasing income (per \$1000 per capita for household) appeared to be protective (HRR=0.98 [95% CI=0.96, 0.99]; $P=.025$). In both cases, significant associations with risk of death from other causes explained the relation with all-cause mortality (HRRs=1.40 [95% CI=1.23, 1.60] and 0.94 [95% CI=0.91, 0.97] for age and income, respectively; $P<.001$). Similarly, the association of cancer stage and differentiation

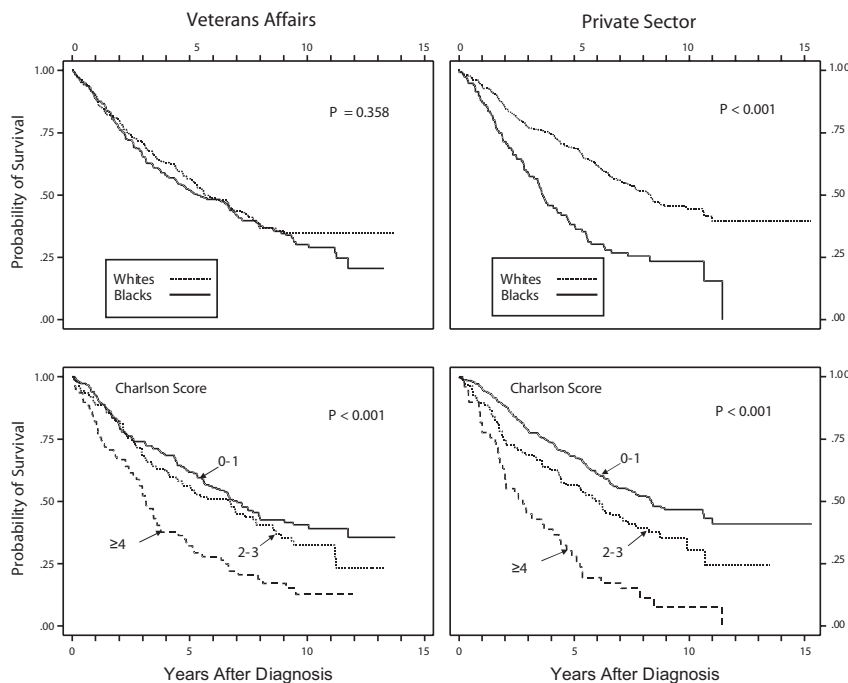


FIGURE 1—Kaplan-Meier survival distributions by race and by comorbidity level.

ing the economic pressures associated with chronic disease control and health maintenance; and (2) their persistence reflects residual confounding due to differences in access to health services, utilization patterns, and processes of care or differences in disease severity over time. Indeed, racial/ethnic disparities in health status and health care, which usually are associated with socioeconomic differences, tend to remain even after control for socioeconomic status and other health care access-related factors.^{27,28} This pattern may be the case in our cohort of prostate cancer patients.

After adjustment for age, income, tumor characteristics, treatment, and comorbidities at baseline, all-cause mortality rates among Blacks relative to Whites were 1.68 (95% CI=1.06, 2.67; $P<.001$) in the private sector and 1.50 (95% CI=0.94, 2.38; $P<.088$) in the VA. Because the rate ratio for race in the VA subset would probably reach statistical significance in a larger sample, application of a statistically based dichotomous interpretation of significance (Neyman-Pearson hypothesis testing) could misinterpret this result.²⁹ There-

fore, we conclude that at least a trend toward excess all-cause mortality among Blacks relative to Whites existed in the VA hospitals.

The relationship between race and disparities in survival among men with prostate cancer is well established in the private sector.¹ However, findings within the VA and other health care settings assumed to offer equal access to care are mixed.³⁰⁻³³ Our results differ from those findings in that we attempted to account for the simultaneous contributions of age, prostate cancer stage and histopathological severity, treatments, comorbidity burden, and income. Moreover, adjustment for these factors narrowed racial differences in mortality in the private sector but exposed them in the VA.

Clinical factors also predicted mortality in our cohort, but with different degrees of cause specificity. For example, tumor differentiation predicted risk of death from prostate cancer but not from other causes. On the other hand, early-stage diagnosis (i.e., cancer confined to the prostate gland or extraprostatic extension with or without ipsilateral lymph node involvement) not only correlated with a

lower risk of death from prostate cancer but also tended to correlate inversely with risk of death from other causes (for VA patients: HRR=0.48 [95% CI=0.23, 1.01]; $P=.052$; for private-sector patients: HRR=0.54 [95% CI=0.30, 0.96]; $P=.056$). Also contrary to expectation was the lack of an association between comorbidity at time of diagnosis and subsequent mortality in the private sector. The absence of racial disparities in risk of death from other causes in the private sector is also inconsistent with observations made by many others.³⁴⁻³⁶ However, excess all-cause mortality of Blacks in the private-sector subset seemed to derive from risk of death from prostate cancer. This pattern suggests that biases in cause-of-death attribution may have partly accounted for the lack of association of race with death from other causes.

What do these data say about determinants of mortality and potential sources of racial disparities in survival after a diagnosis of prostate cancer? First, determinants of all-cause and cause-specific mortality vary according to health care setting, with sociodemographic factors such as age and income playing a role in the private sector but not in the VA system. Second, the association between certain aspects of prostate cancer diagnosis and management with mortality from other causes suggests the presence of more general patient, provider, and health care system characteristics that impact health status and its prognosis in general. These characteristics, in turn, can contribute to or modify racial disparities in survival. For example, diagnosis of prostate cancer at a clinically early stage (i.e., local or regional) tended to be associated with a lowered risk of death not only from prostate cancer but also from other causes. However, Blacks were significantly more likely to present with distant-stage disease. Undergoing surgery for prostate cancer also tended to correlate with a lowered risk of death from other causes. However, Blacks in our cohort were significantly less likely than Whites to receive surgery or other aggressive curative therapy for clinically early-stage disease. Such racial differences in stage at presentation and aggressive primary therapy where indicated have been reported by others.^{8,11} Factors that lead to diagnosis and treatment differences in prostate cancer may

TABLE 2—Relative Hazard Ratios for Death, by Cause,^a for 864 Black and White Men Diagnosed with Prostate Cancer: 2 Veterans Affairs and 2 Private Sector Hospitals, Chicago Area, 1986–1990

	Department of Veterans Affairs (n = 385)						Private Sector (n = 479)					
	All Causes (n = 245)		Prostate Cancer (n = 102)		Other (n = 143)		All Causes (n = 262)		Prostate Cancer (n = 113)		Other (n = 149)	
	HRR (95% CI)	P ^b	HRR (95% CI)	P	HRR (95% CI)	P	HRR (95% CI)	P	HRR (95% CI)	P	HRR (95% CI)	P
Race (Black vs White)	1.50 (0.94, 2.38)	.088	1.36 (0.62, 2.96)	...	1.25 (0.66, 2.35)	...	1.68 (1.06, 2.67)	<.001	2.68 (1.36, 5.27)	<.001	1.03 (0.54, 1.97)	...
Age (5-y increments)	1.07 (0.97, 1.17)	...	1.03 (0.90, 1.19)	...	1.06 (0.93, 1.21)	...	1.17 (1.07, 1.28)	.001	0.99 (0.87, 1.12)	...	1.40 (1.23, 1.60)	<.001
Income (per \$1000) ^c	1.00 (0.97, 1.05)	...	1.02 (0.96, 1.08)	...	1.00 (0.96, 1.04)	...	0.98 (0.96, 0.99)	.025	1.00 (0.98, 1.02)	...	0.94 (0.91, 0.97)	<.001
Differentiation (vs well)												
Moderate	1.24 (0.88, 1.76)	...	2.23 (1.15, 4.29)	.017	0.91 (0.59, 1.41)	...	1.54 (1.02, 2.31)	.040	2.49 (1.06, 5.86)	.037	1.18 (0.73, 1.90)	...
Poor	1.68 (1.19, 2.38)	.003	3.41 (1.79, 6.47)	<.001	1.28 (0.82, 1.98)	...	1.83 (1.23, 2.73)	.003	4.58 (2.06, 10.2)	<.001	1.06 (0.65, 1.73)	...
Stage (local/regional vs distant)	0.26 (0.15, 0.44)	<.001	0.18 (0.08, 0.43)	<.001	0.48 (0.23, 1.01)	.052	0.31 (0.21, 0.48)	<.001	0.19 (0.10, 0.36)	<.001	0.56 (0.31, 1.02)	.056
Treatment												
Surgery	0.66 (0.42, 1.04)	.074	0.40 (0.17, 0.96)	.041	0.72 (0.41, 1.25)	...	0.49 (0.31, 0.77)	.001	0.45 (0.21, 0.97)	.041	0.54 (0.30, 0.96)	.035
Radiation	1.00 (0.67, 1.48)	...	1.25 (0.83, 1.90)	...	0.90 (0.53, 1.52)	...	1.18 (0.84, 1.68)	...	1.80 (1.09, 2.98)	.022	0.92 (0.58, 1.45)	...
DES	1.09 (0.61, 1.95)	...	1.81 (0.84, 3.88)	...	0.50 (0.18, 1.42)	...	1.88 (0.98, 3.60)	.057	1.25 (0.44, 3.60)	...	3.08 (1.34, 7.08)	.008
Castration ^d	0.89 (0.55, 1.43)	...	1.14 (0.57, 2.29)	...	0.72 (0.37, 1.41)	...	1.38 (0.94, 2.04)	...	1.91 (1.10, 3.31)	.021	1.14 (0.63, 2.04)	...
Observation	1.51 (0.90, 2.55)	...	0.47 (0.14, 1.66)	...	2.04 (1.10, 3.79)	.024	1.14 (0.62, 2.09)	...	0.61 (0.14, 2.21)	...	1.26 (0.62, 2.55)	...
Charlson score ^e	1.16 (1.04, 1.29)	.008	0.98 (0.80, 1.20)	...	1.36 (1.18, 1.56)	<.001	1.01 (0.89, 1.15)	...	1.06 (0.89, 1.26)	...	1.02 (0.84, 1.22)	...

Note. HRR = hazard rate ratio; CI = confidence interval; DES = diethylstilbestrol.

^aRelative hazard ratios were calculated using a stratified Cox proportional hazards regression model, which accounted for the baseline hazard associated with the hospital where the case originated and is specified as follows: $\lambda = \lambda_0(t) \exp(\beta_1 \text{race} + \beta_2 \text{age} + \beta_3 \text{moderate diff} + \beta_4 \text{poorly diff} + \beta_5 \text{loc_regstg} + \beta_6 \text{surgery} + \beta_7 \text{radiation} + \beta_8 \text{DES} + \beta_9 \text{castration} + \beta_{10} \text{observation} + \beta_{11} \text{score} + \beta_{12} \text{black*score} + \beta_{13} \text{loc_regstg*score})$.

^bOnly *P* values below .10 are reported in this table.

^cMean household income per capita for zip code of residence at time of diagnosis.

^dOrchiectomy or leuprolide and/or flutamide.

^eScores ranged from 0 to 14.

affect the recognition and prognosis of other health conditions. These include (1) racial/ethnic differences in patient preferences and care-seeking behaviors and attitudes,^{37,38} (2) provider bias against minorities and greater clinical uncertainty in interactions with minority patients, resulting in differences in disease detection and treatment,^{35,39–43} and (3) structural arrangements for services (geography, centralization of services) that fragment the delivery of care.^{44–47}

Finally, the effect of race on all-cause mortality varied by comorbidity level at diagnosis in each health care setting. This variation is indicated by the inverse relation between relative hazards of death (Blacks vs Whites) and Charlson score, as shown in Figure 2. The coefficient for the race-by-comorbidity interaction term incorporated into our Cox proportional hazards model was statistically significant in the cohort overall ($P < .001$). These observations suggest either modifica-

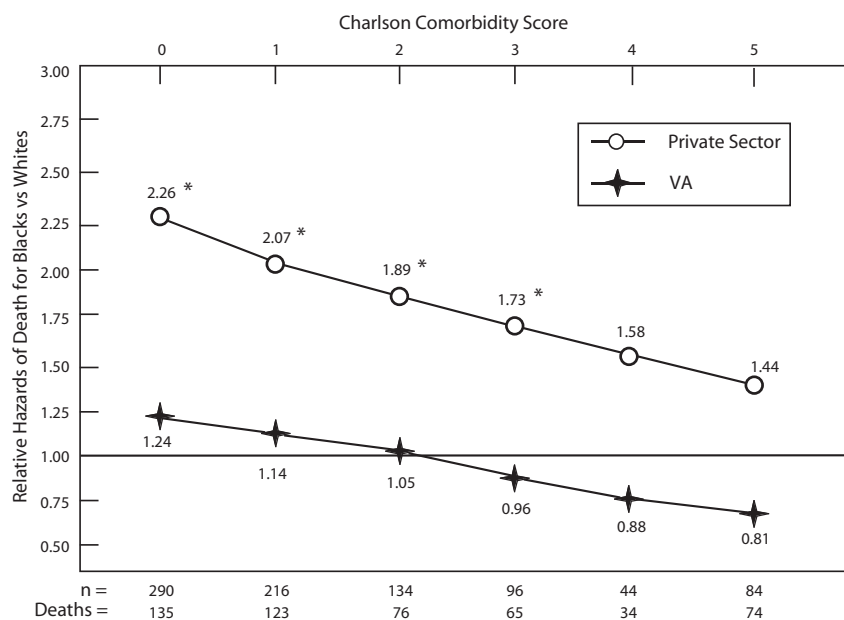
tion of the prognostic significance of racial factors by comorbidity or, conversely, modification of the prognostic significance of comorbidity by racial factors. Intrinsic biological differences between Blacks and Whites seem an unlikely explanation, especially because the association between relative all-cause mortality rates and Charlson score is inverted. Therefore, modification of the prognostic significance of comorbidity by racial factors seems the more likely hypothesis.

Among men with local-/regional-stage prostate cancer, these race-specific estimates for the risk of death from any cause per unit increase in Charlson score were observed after adjustment for age, tumor differentiation, and treatments: for VA patients (Whites vs Blacks): HRR (95% CI) = 1.20 (1.09, 1.43) vs 1.10 (1.00, 1.22); for private sector patients: HRR (95% CI) = 1.28 (1.19, 1.51) vs 1.17 (0.97, 1.56). Hence, comorbidity at the time of diagnosis tended to be less predictive of all-

cause mortality among Blacks. This trend may reflect racial differences in the prognostic performance of the Charlson index. Possible mechanisms for such differential performance include less precise ranking of the severity of comorbidities among Blacks and a lower sensitivity in detecting prognostically important conditions (which may be more likely to go unrecognized or be less fully characterized in Blacks compared with Whites^{44,48,49}). In light of these possibilities, the results shown in Figure 2 suggest that absence of a significant pre-existing medical diagnosis is a risk factor for excess all-cause mortality among Black men with prostate cancer.

Limitations

Prostate-specific antigen (PSA) is an important marker of prostate cancer disease activity and prognosis.⁵⁰ However, the results of PSA testing were not available for approximately 15% of our cohort, and levels were not ac-



Note. VA = Department of Veterans Affairs. Estimates are adjusted for age, tumor stage, tumor differentiation, and treatment. * $P < .05$.

FIGURE 2—Bootstrap confidence intervals for all-cause mortality rates of Blacks relative to Whites, by Charlson comorbidity score.

counted for in our analyses. Racial and ethnic minorities are more likely than Whites to be enrolled in “lower-end” health plans. Such plans are characterized by higher per-capita resource constraints and stricter limits on covered services that may affect health care utilization.⁴⁶ However, data on payment sources in the private sector were not available for this analysis.

In conclusion, determinants of all-cause mortality in men with prostate cancer vary between the Veterans Affairs and private-sector health care systems. More detailed analysis of the sociodemographic and clinical characteristics of patients, provider behavior, and structural arrangements for services in each health care setting in relation to cause-specific mortality should provide important insights into the causes of racial/ethnic disparities in survival in this population and into strategies to eliminate these disparities. ■

About the Authors

Vincent L. Freeman is with the Edward Hines Jr VA Hospital, Hines, Ill. and with the Dept of Medicine and

Urology, Loyola University Medical Center, Maywood, Ill. Ramon Durazo-Arvizu is with the Feinberg School of Medicine, Northwestern University, Chicago, Ill. and with the West Side VA Medical Center, Chicago, Ill. Ahsan M. Arozullah is with the University of Illinois at Chicago. LaShon C. Keys is with the Illinois Department of Human Services, Tinley Park.

Requests for reprints should be sent to Vincent L. Freeman, MD, MPH, Edward Hines Jr VA Hospital, PO Box 5000 (151H), Hines, IL 60141 (e-mail: freem595@aol.com).

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Contributors

V.L. Freeman, R. Durazo-Arvizu, and L. C. Keys designed and implemented the study, including developing the hypotheses, conducting sample size and statistical power analyses, designing the data collection instrument, supervising data collection, and performing quality controls. V.L. Freeman and R. Durazo-Arvizu conducted the data analyses. All of the authors interpreted the data and drafted or revised the article.

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Human Participant Protection

Appropriate institutional review board approval was obtained at each hospital.

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