Original Article

Explaining Racial Differences in Prostate Cancer Mortality

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BACKGROUND: In the United States, black males have an annual death rate from prostate cancer that is 2.4 times that of white males. The reasons for this are poorly understood. **METHODS:** Using the Surveillance, Epidemiology, and End Results–Medicare database, 77,038 black and white males aged >65 years were identified with a first primary diagnosis of prostate cancer between 1995 and 2005, as well as 49,769 controls. The racial gap in mortality was decomposed to differential incidence and stage-specific prostate cancer mortality. The importance of various clinical and socioeconomic factors to each of these components was then examined. **RESULTS:** The estimated mortality gap for prostate cancer–specific mortality was 1320 more cases per 100,000 males among black than white men. This gap was due to higher prostate cancer incidence among black males (76%) and higher stage-specific mortality once diagnosed (24%). Differences in prostate-specific antigen testing, comorbidities, and income explained 29% of the difference in metastatic cancer incidence but none of the racial gap for local/regional incidence. Conditional on diagnosis, tumor characteristics explained 50% of the racial gap, comorbidities an additional 4%, choice of treatment and physician 17%, and socioeconomic factors 15%. Overall, approximately 25% of the racial gap in mortality and 86% of the gap in mortality conditional on diagnosis could be explained. **CONCLUSIONS:** More frequent prostate-specific antigen testing for black and low-income males could potentially reduce the prostate cancer mortality gap through earlier diagnosis of tumors that otherwise may become metastatic. More aggressive treatment of prostate cancer, especially in poor communities, might also reduce the gap. ***Cancer*** **2012;118:4280-9.** © *2012 American Cancer Society.*

KEYWORDS: prostate cancer, disparities, survival, race, blacks.

Prostate cancer is the leading incident cancer in the United States and the second most fatal among men.[1] Yet, mortality varies significantly by race. In 2007, black males had an age-adjusted annual death rate from prostate cancer 2.4 times that of whites (52.0 vs 21.6 per 100,000 males).[1]

The reasons for this mortality gap are poorly understood. Prostate-specific antigen (PSA) screening is more often performed in whites than in blacks,[2,3] so differential screening may contribute.[4] Still, the value of screening is unclear, and some studies suggest that PSA screening has no effect or a small effect on mortality.[5-7] Black patients also receive less treatment after diagnosis.[8-11] Although outcomes are similar with radical prostatectomy, radiation, and active surveillance for most men with local/regional disease,[12] younger men and those with more aggressive tumors may benefit from intervention.[13] Differential treatment may thus contribute to mortality differences between races.[14] Still, evidence on treatment differences by race is indeterminate,[9,15-19] and limited by a selective nature (eg, single institutions), relatively small sample sizes, or location (populations with universal health care or nongeneralizable minority populations).

Debate also has centered on the impact of differential social and medical access factors in explaining racial differences in outcomes. Some studies suggest that lower income,[20] education,[21,22] lack of health insurance,[23] and comorbidities[24,25] may lead to more advanced disease at diagnosis and higher mortality for minority groups, but this is not universally found.[26-28]

We used population-based data from across the United States to better understand the mortality gap for black versus white patients with prostate cancer, in 2 dimensions. First, we decomposed the racial gap in mortality into greater stage-specific incidence of disease among blacks and higher mortality, conditional on diagnosis. Second, we assessed how differential receipt of medical tests and treatments, access to different physicians, and differences in socioeconomic status affected each component.

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We thank Mary Beth Landrum for helpful comments and advice. The study was approved by the Institutional Review Board of the National Bureau of Economic Research, Cambridge, MA.

DOI: 10.1002/cncr.27379, **Received:** July 27, 2011; **Revised:** October 3, 2011; **Accepted:** November 10, 2011, **Published online** January 13, 2012 in Wiley Online Library (wileyonlinelibrary.com)

MATERIALS AND METHODS

Data and Patients

We used the Surveillance, Epidemiology, and End Results (SEER)-Medicare database, which links cancer registry data with Medicare claims.[29] We identified 77,038 males aged >65 years with a first primary diagnosis of prostate cancer between 1995 and 2005 and living in one of the SEER-13 regions that covered approximately 14% of the US population.[30] All individuals were white or black, enrolled in parts A and B of fee-for-service Medicare from at least 1 year before through 6 months after diagnosis, and not diagnosed by autopsy or death certificate.

To estimate prostate cancer incidence, we also identified 49,769 men without prostate cancer using the 5% Medicare file for non–prostate cancer patients living in SEER areas.

Variables

Prostate cancer–specific mortality was assessed through 2005 using SEER date and cause of death, based on National Death Index linkages. Patients not dying of prostate cancer were censored on the date of death from another cause or on December 31, 2005 (the last date for which complete data were available). Median follow-up was 3.89 years.

We defined date of diagnosis as the date of the first Medicare claim with a prostate cancer diagnosis (International Classification of Diseases, Ninth Revision [ICD-9] diagnosis 180.x) within 1 month of the SEER month of diagnosis (75% of patients). For remaining subjects, we used the 15th day of the SEER month. SEER provided tumor extension (T1-T4), stage (local/regional or metastatic), and grade (well, moderately, or poorly differentiated, or undifferentiated).

Cancer registries reported race, birth month/year, and marital status. ZIP code of residence was linked with US Census data to obtain household median income, categorized in approximate quartiles based on the black population.

From Medicare data, we used diagnosis and procedure codes to document PSA testing, comorbidities in the year before diagnosis (Charlson components),[31] and treatments received from 30 days before to 6 months after diagnosis. Treatments included surgery/radical prostatectomy (Current Procedural Terminology [CPT] 55810-55815, 55840-55845; ICD-9 procedure 60.5), radiation (CPT 77295, 77301, 77401-77418, 77750-77799, 0073T; Healthcare Common Procedure Coding System [HCPCS] G0103; ICD-9 procedure 92.24, 92.26, 92.27), androgen ablation therapy (CPT 54520-54522, 54530, 54535, 54690, 49510; HCPCS C9430, J3315, J9217-J9219, J9202, J1950; ICD-9 procedure 62.3, 62.4, 62.41, 62.42), or none of these treatments. Androgen ablation was considered adjuvant for patients with local/regional tumors who also underwent radical prostatectomy or radiation, and primary otherwise. We tested for racial differences using Pearson chi-square or student *t* test, as appropriate.

We assigned a single physician for each subject using the following hierarchy: physician performing radical prostatectomy (for patients having surgery), radiation oncologist (for patients having radiation), physician administering androgen ablation therapy, or first urologist seen on or after date of diagnosis. Regardless of treatment, we identified patients as seeing an operating surgeon if they saw a urologist, general surgeon, or surgical oncologist who performed at least 1 radical prostatectomy for a patient in the cohort in the calendar year before diagnosis.

Analysis

Contribution of incidence and mortality conditional on diagnosis to the racial gap in mortality

Prostate cancer mortality for any racial group is the product of incidence and mortality conditional on diagnosis, added across each stage.[4,32,33] Using a first-order Taylor Series expansion, the racial difference in stage-specific mortality may be approximated as a weighted average of the differences in stage-specific incidence and mortality conditional on diagnosis.[34] The racial gap in incidence affects mortality to the extent that people die of each stage tumor, and the racial gap in mortality conditional on diagnosis affects mortality based on the share of people diagnosed at each stage.

Mathematically, we estimated the contribution of the racial gap in incidence to the racial gap in mortality as black minus white incidence, times mortality conditional on diagnosis for all races. Similarly, the contribution of the racial gap in mortality conditional on diagnosis to the racial gap in mortality was black minus white mortality conditional on diagnosis, times incidence for all races. We then estimated a counterfactual mortality rate for blacks, if all additional (gap) tumors among blacks were diagnosed at the earliest (local/regional) stage. This estimate was stage-specific white incidence times stage-specific black mortality conditional on diagnosis, plus the gap in all-stage incidence times black mortality conditional on diagnosis for local/regional tumors.

Factors contributing to the racial gap

We estimated the contribution of various factors to differential incidence and differential stage-specific

survival by simulating the number of black deaths that would be avoided if stage-specific incidence (or survival) were the same for blacks as for whites, controlling for those factors. Specific calculations follow.

Factors contributing to the racial gap in incidence

For each race, we computed incidence rates across all calendar years for every age from 68 years (to allow ≥3 years of Medicare eligibility) to those aged ≥85 years, stratified by number of PSA tests in the prior 3 years, comorbidities, and income. To identify PSA screening rather than diagnostic tests, we excluded PSA tests for men with diagnosis codes for prostate hyperplasia, prostate nodules, prostatitis, or other prostate problems (ICD-9 diagnosis 600.00–600.91, 601.0–602.9). We assessed the number of PSA tests conducted at more than 1 year before each age, because most tests at cancer diagnosis are abnormal.

To estimate the proportion of the racial gap in incidence attributable to PSA testing, comorbidities, and income, we used a series of equations. First, we calculated a counterfactual incidence rate for blacks if they had the same proportion of men in each PSA testing category (tests in 0, 1, 2, or 3 of the prior 3 years) as whites. Patients without prostate cancer were weighted 20× (100% ÷ 5% random sample) and patients with prostate cancer were weighted 1×. The percent change in the racial gap between the counterfactual rate and the actual rate among blacks was computed, as an estimate of the marginal impact of differences in PSA testing on the racial gap in incidence.

Next, we repeated the analysis with a counterfactual incidence rate stratified by PSA testing category and comorbidity group (Charlson score of 0 or ≥1). When compared with the PSA-only stratification, the result estimated the marginal impact of differences in comorbidities on the racial gap in incidence.

We also considered a counterfactual incidence rate stratified by PSA testing, comorbidity, and area median income. When compared with the first 2 stratifications, this result estimated the marginal impact of differences in area income on the racial gap in incidence.

Factors contributing to the racial gap in mortality conditional on diagnosis

Among patients with prostate cancer, we simulated prostate cancer–specific mortality for blacks through 2005 if mortality within each stage was the same as that for whites but incidence and stage of diagnosis remained unchanged, using Cox proportional hazard models[35] with 2-tailed tests of statistical significance at the 5% level. The dependent variable was death from prostate cancer. Patients were followed from 31 days before diagnosis and censored upon death from other causes or on December 31, 2005.

We first estimated models relating survival to race, age, diagnosis year, and SEER registry. We then successively added independent variables in 4 categories: tumor characteristics, comorbidities, treatments, and socioeconomic status. Using the change in the hazard rate on the black race variable when successive variables were included, we estimated the proportion of the racial gap explained (eg, if the hazard rate declined from 1.72 to 1.36, the racial gap declined by 50%). We also assessed if the racial disparity for prostate cancer exceeded that for other causes of death by estimating the hazard ratio for black race on death from other causes.

Age mattered inversely with time since diagnosis; however, alternative specifications of the model that allowed for an interaction between age and time since diagnosis yielded similar results.

When estimating the effect of treatments, we were concerned about unobserved confounding, such as if patients in better health were more likely to undergo treatment.[36] Thus, we conducted an instrumental variable analysis.[37-39] The instrument, whether a patient saw an operating surgeon, was strongly associated with undergoing radical prostatectomy, but should be associated with mortality only through its association with radical prostatectomy. We used a logistic model to assess the association between seeing an operating surgeon and receipt of surgery, and used the fitted result in our proportional hazard model. Analyses were conducted using Stata statistical software (Stata, College Station, Texas).

RESULTS

Table 1 shows summary statistics for the cohort. Black patients were younger than white patients, less often married, had more comorbidities, and less often underwent treatment.

Prostate cancer incidence for blacks aged ≥68 was significantly above that for whites (20,745 per 100,000 versus 14,776 per 100,000) (Table 2). The risk is higher for all stages. Black patients also had higher prostate cancer mortality conditional on diagnosis than white patients (Table 2). However, this finding was true for local/regional and unstaged cancers; mortality did not differ significantly by race for metastatic cancers. The incidence rate for blacks is sufficiently higher than for whites, so that even if all additional (gap) tumors were diagnosed at a

Table 1. Summary Statistics for Prostate Cancer Patients, 1995-2005

	White (N = 70,139)	Black (N = 6,899)	*P* (χ^2 or t)
Age at diagnosis, y	74.5	73.6	<.001
Percent			
Marital status			
Married	71.8	53.1	<.001
Never married	6.1	17.2	
Separated	0.3	1.0	
Divorced	3.3	7.9	
Widowed	9.4	11.7	
Unknown	9.1	9.1	
ZIP code median income (Census 2000)			
<$25,000	1.8	23.3	<.001
$25,000-$29,999	3.2	18.6	
$30,000-$39,999	19.5	25.7	
≥$40,000	71.3	29.4	
Unknown	4.3	3.1	
ZIP code education (age ≥25 y) (Census 2000)			
Non–high school graduate	14.1	26.2	<.001
High school only	25.6	28.9	<.001
Some college	29.2	30.9	<.001
≥4 Years of college	31.2	14.1	<.001
Unknown	4.2	3.0	<.001
Charlson score			
0	82.9	73.7	<.001
≥1	17.1	26.3	
Screening			
PSA in 12 mo to 91 d before diagnosis	42.2	43.3	.08
Number of PSA tests in 3 y to 91 d before diagnosis (age ≥68 y) (Maximum 1 per y)			
0	30.1	31.8	<.001
1	29.7	32.5	
2	26.3	23.8	
3+	14.0	11.9	
Tumor characteristics			
T1	36.3	37.0	<.001
T2	52.4	48.9	
T3	2.2	2.0	
T4	4.1	5.9	
Unknown size	5.0	6.3	
Local/regional	91.1	88.0	<.001
Metastatic	4.2	6.1	
Unstaged	4.7	5.9	
Differentiation			
Well	5.9	4.1	<.001
Moderate	60.5	58.2	
Poor	27.6	29.5	
Undifferentiated	0.4	0.2	
Unknown	5.7	8.0	
Treatment			
Saw an operating surgeon	26.0	22.2	<.001
Radical prostatectomy	16.9	12.3	<.001
Brachytherapy	8.9	6.6	<.001
Radiation-nonbrachytherapy	29.5	30.9	.01
Androgen ablation, primary	22.0	23.4	.01
Androgen ablation, adjuvant	19.7	16.5	<.001
No primary treatment	22.4	26.6	<.001

Table 2. Prostate Cancer Incidence and Mortality Conditional on Diagnosis, 1995-2005

	Rates Per 100,000 Males					*Estimated Racial Gap in Mortality Per 100,000 Males*	
	All	White	Black	Gap	*P* (χ^2)	Number of Lives	Share
Incidence, age ≥68 y							
All	15,310	14,776	20,745	5970	<.001	1006	**76%**
Local/regional	14,288	13,831	19,097	5266		589	45%
Metastatic	426	371	810	439		346	26%
Unstaged	603	573	838	265		70	5%
Mortality conditional on diagnosis, age ≥66 y							
All	14,490	14,170	17,740	3570	.003	**314**	**24%**
Local/regional	11,193	11,020	13,020	2000		286	22%
Metastatic	78,927	79,900	72,110	−7790		−33	−3%
Unstaged	26,319	25,200	35,320	10,120		61	5%
Total							
Total		1965[a]	3367[a]	71%		**1320**	**100%**
Total if all additional (gap) tumors among blacks were diagnosed as local/regional		1965	3048	55%			

[a] Less than the actual rate, because of limited follow-up.

local/regional stage, the mortality rate for blacks would exceed that for whites by 55% (Table 2).

The right columns of Table 2 show factors contributing to differential mortality by race. We estimated 1320 excess deaths for black versus white men per 100,000, similar to the annual mortality difference in vital statistics data.[40] A total of 76% of this difference is related to greater incidence among blacks: 45% for local/regional, 26% for metastatic, and 5% for unstaged tumors. The higher mortality rate for blacks conditional on diagnosis accounts for the remaining 24% of the mortality difference. Almost all of this difference is greater mortality from local/regional disease for blacks.

Factors Associated With Differential Incidence

Metastatic and unstaged tumors

More frequent PSA testing was associated with lower incidence of metastatic cancers (Fig. 1). At ≤1 PSA test in the prior 3 years, the racial disparity in incidence of metastatic cancer at diagnosis was large (1202 black men vs 538 white men per 100,000 men with no PSA tests and 669 black men vs 289 white men per 100,000 men with 1 PSA test, both $P < .001$) (Table 3). However, with 2 PSA tests in the prior 3 years, the incidence of metastatic disease in blacks was nearly identical to that in whites (226 vs 223 per 100,000 men, $P = .93$) (Table 3). Racial differences were robust to reclassifying unstaged tumors as metastatic.

Income also had a negative relationship to metastatic incidence (Fig. 1). Black men in the poorest income quartile had a 24% to 40% higher metastatic incidence rate than those in wealthier quartiles.

Overall, 8 of the 26 percentage point (29%) adverse distribution of metastatic diagnoses in blacks would be eliminated if blacks had the same screening rates, comorbidities, and income distribution as whites (Table 4). Greater use of PSA testing in whites explains 2 points, fewer comorbidities explains 2 points (when PSA screening is held constant), and higher income explains 4 points (when PSA screening and comorbidities are held constant).

Local/regional tumors

More frequent PSA testing was associated with higher incidence of local/regional disease (data not shown). This result would suggest greater incidence of disease among whites, because whites were screened more than blacks. Accordingly, PSA testing had a slight negative contribution to the racial gap (Table 4) for local/regional tumors. Comorbidities were minimally related (adjusting for number of PSA tests), and ZIP code income was negatively associated (adjusting for number of PSA tests and comorbidities).

Overall effect of incidence on racial mortality gap

Our models explained 29% of the racial gap in incidence for metastatic tumors, but only 8% for unstaged tumors and none for local/regional tumors (Table 4).

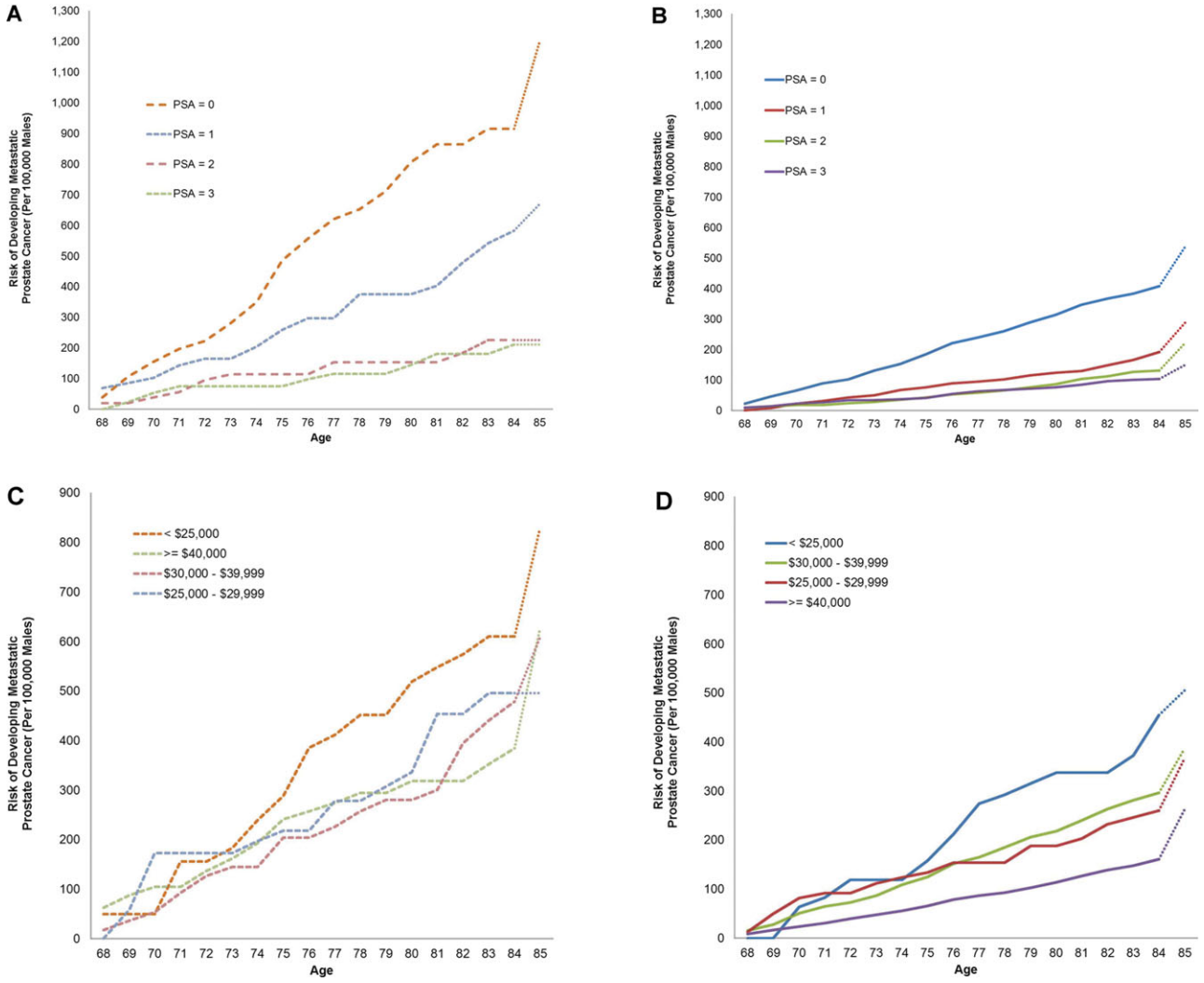

Figure 1. Cumulative risk of developing metastatic prostate cancer, per 100,000 males, is shown for (A) black males, by number of prostate-specific antigen (PSA) tests in the prior 3 years, (B) white males, by number of PSA tests in the prior 3 years, (C) black males, by ZIP code median income (Census 2000), and (D) white males, by ZIP code median income (Census 2000). The number of PSA tests was capped at 1 per year. Dotted line after age 84 indicates transition to age ≥85 (rather than just age 85).

Factors Associated With Differential Mortality Conditional on Diagnosis

In a series of models, we explained 21 of the 24 percentage points (86%) for higher mortality conditional on stage of diagnosis for blacks (Table 4). In a model including only age at diagnosis, year of diagnosis, and SEER registry, black patients were 72% more likely than white patients to die of prostate cancer (adjusted hazard ratio [aHR] = 1.72, 95% confidence interval [CI] = 1.56-1.90) (Table 5). The racial disparity exceeded that for non–prostate cancer causes of death (aHR = 1.72 vs 1.38, $P = .005$). We next included tumor characteristics: stage, size, grade, and receipt of a PSA test between 12 months and 91 days before diagnosis (as a proxy for early diagnosis). Metastatic tumors tripled the hazard rate of death (aHR = 2.87, 95% CI = 2.33-3.55), whereas having a PSA test reduced it by 23% (aHR = 0.77, 95% CI = 0.72-0.82). Results were similar among patients diagnosed in 2004 and 2005, for whom PSA values were available. Overall, tumor characteristics explained 50% of the gap in mortality conditional on diagnosis, and comorbidities explained an additional 4% (Table 5; adjusted gap = 54%, 95% CI = 45%-69%).

We next considered treatment differences. Using our instrument of whether a patient saw an operating surgeon, patients undergoing radical prostatectomy had a 22% lower hazard of death (aHR = 0.79, 95% CI = 0.69-0.90). This compared with a less realistic 60% lower

Table 3. Racial Gap in Metastatic Prostate Cancer Incidence by PSA Testing History, 1995-2005

	Rates Per 100,000 Men, Age ≥68			
	White		Black	
Number of PSA Tests in Prior 3 Years[a]	% of Men	Incidence (95% CI) of Metastatic Cancer	% of Men	Incidence (95% CI) of Metastatic Cancer
0	48	538 (529-548)	52	1202 (1147-1257)
1	20	289 (278-300)	20	669 (604-737)
2	17	223 (213-233)	16	226 (182-271)
3	15	150 (141-159)	12	211 (165-262)

CI indicates confidence interval; PSA, prostate-specific antigen.
[a] Maximum of 1 PSA test per year.

Table 4. Factors Contributing to Racial Gap in Prostate Cancer Mortality

Factor	Contribution to Total (%)			Total (%)
	Local/Regional	Metastatic	Unstaged	
Higher incidence rate	**45**	**26**	**5**	**76**
Number of PSA tests in prior 3 y	0	2	0	2
Comorbidities	0	2	0	2
Income	−4	4	0	0
Amount explained	−4 (–10%)	8 (29%)	0 (8%)	4 (5%)
Higher mortality conditional on diagnosis	**22**	**−3**	**5**	**24**
Medical factors	**14**	**−3**	**5**	**16**
Tumor characteristics	**6**	**−1**	**1**	**7**
Tumor Size	1	0	0	2
Differentiation	4	−1	1	4
Other[a]	0	0	0	1
Comorbidities	2	0	0	2
Type of treatment	3	−2	0	1
Choice of physician	4	0	3	6
Socioeconomic factors	**4**	**0**	**0**	**4**
Income	4	0	0	4
Marital status	1	0	0	1
Amount explained	19 (85%)	−3 (100%)[b]	5 (100%)[b]	21 (86%)
Total contribution to racial gap	66	24	10	**100**
Amount explained by:				
Higher Incidence	−4	8	0	4
Higher Mortality Conditional on Diagnosis	19	−3	5	21
Total amount explained	15 (23%)	5 (21%)	5 (50%)	**25 (25%)**

[a] Dummy variable for a PSA test between 12 mo and 91 d prior to diagnosis, to proxy for less advanced tumors (through earlier detection).
[b] Not meaningful, because of a small contribution to the total.

hazard rate (aHR = 0.40, 95% CI = 0.34-0.47) calculated without an instrumental variables technique. Patients undergoing nonbrachytherapy radiation (aHR = 0.84, 95% CI = 0.77-0.92) and brachytherapy (aHR = 0.58, 95% CI = 0.48-0.70) also were less likely to die of prostate cancer than patients without primary treatment. Patients who underwent primary androgen ablation had a 55% higher death rate (aHR = 1.55, 95% CI = 1.42-1.69), which may reflect poorer health. Adjuvant androgen ablation therapy was not significant. Net, primary treatment explained an additional 4% of the mortality difference between blacks and whites (Table 5; adjusted gap = 58%).

Controlling for physician who provided the primary therapy explained an additional 13% of the racial difference in mortality (Table 5; adjusted gap = 71%). This effect only existed among patients in the poorest income quartile; there was a negative contribution in each higher

Table 5. Prostate Cancer Mortality Conditional on Diagnosis[a]

Cox Proportional Hazard Rate Model	(1)	(2)	(3)	(4)	(5)	(6)
Controls Added[b]	Race and age	Tumor characteristics	Comorbidities	Treatment[c]	Physician[c]	Income and marital status[c]
Race						
Black	1.72	1.36	1.33	1.30	1.21	1.10
P	<0.001	<0.001	<0.001	<0.001	0.009	0.22
(95% CI)	(1.56-1.90)	(1.22-1.51)	(1.19-1.47)	(1.17-1.44)	(1.05-1.39)	(0.94-1.28)
Proportion of racial gap explained	0%	50%	54%	58%	71%	86%
(95% CI)		(41%-64%)	(45%-69%)	(49%-75%)	(57%-91%)	(69%-110%)

For patients diagnosed 1995-2005.
[a] Dependent variable: Died from prostate cancer, 1995-2005.
[b] Once added, controls are always used in subsequent columns.
[c] Surgery instrumented with an indicator of whether a patient saw an operating surgeon.

quartile. Measures of physician skill, such as number of prostate cancer patients seen in the prior 12 months, were not significant.

We next added socioeconomic variables, which explained an additional 15% of mortality differences conditional on diagnosis (86% in total). The wealthiest quartile of patients was less likely to die relative to the poorest quartile (aHR = 0.83, 95% CI = 0.68-1.00). Divorced and widowed patients had a 15% to 30% higher hazard of death.

Alternative Specification

It may be that poorer socioeconomic conditions earlier in life cause blacks to be in weaker health later in life. In this case, our models should order socioeconomic factors before medical characteristics. In this ordering, we attributed 39% of the racial gap in survival conditional on diagnosis to socioeconomic factors and 47% to clinical factors. Individual-level socioeconomic data would be important to explore this in more detail.

Summary

Overall, we explained 25% of the racial gap in mortality (Table 4). A small proportion (4%) was related to higher incidence for blacks than whites, and more (21%) to higher mortality conditional on diagnosis. Our models explained 86% of this gap in mortality conditional on diagnosis.

DISCUSSION

Prostate cancer is the leading cause of racial differences in mortality among males.[41] Prior reports have suggested these differences may be due to less frequent PSA screening,[2-4] less aggressive treatment,[8-11] less advanced treatment facilities,[15] and lower socioeconomic status[20-27] for black versus white men. But the combined evaluation of these different theories has not been known. Our findings suggest that approximately 76% of the mortality gap is due to higher incidence of disease in blacks—45% from local/regional, 26% from metastatic, and 5% from unstaged tumors—a portion of which was explained by differences in PSA testing and socioeconomic status. The remaining 24% of the mortality gap is due to higher mortality conditional on diagnosis.

The biggest contributors to mortality among diagnosed cases are tumor characteristics, treatments, and socioeconomic status. Because physicians cannot change socioeconomic status and tumor characteristics, from a clinical perspective, identifying effective prevention and screening strategies for black patients has the most potential for eliminating the racial gap. Although randomized trials have demonstrated no benefit or a modest benefit of PSA screening on reducing prostate cancer–specific mortality,[5-7] these studies included primarily white men of European descent. If prostate cancer is more aggressive in black than white men, black men may derive more benefit than white men from PSA screening.

Among men in our study, even occasional PSA screening (1 test in 3 years) lowered metastatic incidence substantially. The decrease was particularly large for black men, suggesting that more frequent PSA testing for black males may be desirable. This result must be tempered by knowledge that PSA testing may serve as a proxy for individual socioeconomic status.[42] In addition, more PSA screening will result in more local/regional diagnoses, leading to possible overtreatment. The risk of a local/regional diagnosis rose 2% for white males with 1 PSA test every 3 years and 6% for black males with 2 PSA tests every 3 years, versus those who had no PSA tests.

Our findings also identified area-level socioeconomic status as an important driver of disparities in

prostate cancer mortality. Controlling for differential PSA screening, men living in areas with a median income <$25,000 had higher metastatic incidence than those in the second-poorest quartile by 38% for whites and 27% for blacks. Increased screening among lower income groups would benefit both races and also reduce the racial gap, because there are more lower-income blacks. By contrast, screening, comorbidities, and income explained none of the racial gap in local/regional diagnoses. Indeed, richer, healthier communities with more PSA testing have higher local/regional incidence rates. The large, remaining gap in prostate cancer incidence may be related to biological or genetic differences or other lifestyle or exposure differences.

Most prostate cancers are diagnosed at an early stage, where 5-year relative survival rates for both races approach 100%.[1] Consequently, explaining 86% of differential mortality conditional on diagnosis explained just 21% of the total mortality gap. Thus, attempting to reduce racial differences in treatments would likely trim just 4% of the gap in survival, or 1% of the total disparity. Choice of physician is a bigger contributor to mortality differences, but this seems to matter exclusively at the bottom of the income distribution. If all patients were from the lowest income quartile, then choice of physician would contribute 9% to mortality differences, yet at higher quartiles, choice of physician has negative explanatory power. This may reflect less access to high-quality providers or more aggressive tumors among poor patients, due to a lower-quality diet and lifestyle.

Limitations

Our sample consists of men diagnosed at age >65 years enrolled in fee-for-service Medicare; whether our findings are generalizable to other populations requires further study. Second, we had no information on obesity, lifestyle factors, or family history of prostate cancer. We could not measure tumor aggressiveness well, so even with the same PSA testing intervals, black men could be diagnosed at a later stage or be more likely to die with the same stage if they had more aggressive tumors. Third, socioeconomic data were available at the area level rather than individual level. Fourth, studies disagree on how accurately death certificate data reflect cause of death.[43,44] Finally, our model assumes independence between death from prostate cancer and causes other than prostate cancer, a condition that may be violated through comorbidity.[45,46]

Conclusion

The greater prostate cancer mortality among older, insured black men is partly explained by less frequent PSA screening, more advanced tumors, and lower socioeconomic circumstances, although even when these factors are accounted for, there is a large difference between blacks and whites. More frequent PSA screening for blacks may decrease the mortality gap, albeit modestly so. Additional research is necessary to understand the potential benefits of PSA screening in black men, including whether the risk of overdiagnosis is less in black men versus white men, given differences in tumor biology.

FUNDING SOURCES

This work was funded in part by the Prostate Cancer Foundation, the National Institute on Aging, and the Dana-Farber Cancer Institute A. David Mazzone Awards Program.

CONFLICT OF INTEREST DISCLOSURE

The authors made no disclosure.

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