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Socioeconomic Status, Race, and Long-Term Outcomes After Radical Prostatectomy in an Equal Access Health System: Results from the SEARCH Database

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Abstract

Introduction: We previously found racial differences in biochemical recurrence (BCR) after radical prostatectomy (RP) persisted after adjusting for socioeconomic status (SES) while SES did not predict BCR. The impact on long-term prostate cancer (PC) outcomes is unclear. We hypothesized higher SES would associate with better long-term outcomes regardless of race.

Methods: Among 4,787 black and white men undergoing RP from 1988 to 2015 in the SEARCH Database, poverty (primary SES measure) was estimated by linking home ZIP-code to census data. Cox models were used to test the association between SES adjusting for demographic, clinicopathological features, and race with BCR, castration-resistant PC (CRPC), metastases, PC

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specific mortality (PCSM), and all-cause mortality (ACM). Interactions between race and SES were tested.

Results: Median follow-up was 98 months (IQR: 54–150 months). There were no interactions between race and SES for BCR. Black men had 10–11% increased BCR risk ($p<0.06$) while SES was unrelated to BCR. There were interactions between SES and race for CRPC ($p=0.002$), metastasis ($p=0.014$), and PCSM ($p=0.004$). Lower SES was associated with *decreased* CRPC ($p=0.012$), metastases ($p=0.004$), and PCSM ($p=0.049$) in black, but not white men (all $p\geq0.22$). Higher SES was associated with decreased ACM in both races.

Conclusions: In an equal-access setting, lower SES associated with decreased CRPC, metastases, and PCSM in black but not white men. If confirmed, these findings suggest a complex relationship between race, SES, and PC with further research needed to understand why low SES in black men decreased risk for poor PC outcomes after RP.

1. Introduction

Relative to white men, black men have higher risk of prostate cancer (PC) incidence and worse outcomes[1–4]. Although these health disparities often persist despite adjusting for socioeconomic status (SES), SES is important as it relates to access to care[5]. Given black race and lower SES are correlated, the degree to which race vs. SES explains worse outcomes is unclear. Few studies examined SES; fewer still examined the intersection between race, SES and PC outcomes.

SES includes factors such as income, education, and employment. Previous studies of SES and PC are mixed. One study showed lower SES patients presented with higher grade disease, and black race, but not SES, was associated with post-radical prostatectomy (RP) biochemical recurrence (BCR)[6]. In contrast, a retrospective case-control study found lower SES increased BCR risk[7]. A Norwegian study found higher education was associated with increased PC risk, while another study suggested lower education levels influenced the association between obesity and BCR[8, 9]. Contrarily, another study found greater income and education did not improve PC outcomes among black men[10]. A review assessing socioeconomic differences in PC survival rates found 75% of studies showed lower SES was associated with worse survival[11]. Although these studies suggest complicated relationships between SES and PC, few examine the influence of race *and* SES on long-term PC outcomes.

We previously studied race and SES on short-term outcomes after RP in an equal-access setting. We found racial disparities in BCR persisted after adjustment for SES, and higher SES was associated with less positive margins[12]. More recently, we found race was unrelated to long-term outcomes after RP, though we did not account for SES[13]. In this study, we tested the influence of SES on long-term outcomes after RP and how this differs by race. We hypothesized men with higher SES would have better long-term outcomes, such as lower rates of castration-resistant PC (CRPC) and PC-specific mortality (PCSM), regardless of race.

2. Materials and Methods

2.1 Study Design

After obtaining Institutional Review Board approval, data from patients undergoing RP between 1998 and 2015 at six Veteran Affairs Medical Centers (West Los Angeles, San Diego, and Palo Alto, CA; Augusta, GA; Durham and Asheville, NC) were combined into the SEARCH database[14]. Patients treated with preoperative hormonal therapy or radiotherapy were excluded. Patients were followed for BCR, CRPC, metastases, PCSM, and all-cause mortality (ACM). BCR was defined as a single PSA >0.2ng/mL, two concentrations at 0.2ng/mL or secondary treatment for an elevated PSA. CRPC was defined as a PSA rise of ≥2 and ≥25% from the post-ADT nadir while being castrate, defined as testosterone <50ng/dL, bilateral orchiectomy, or continuous receipt of luteinizing hormone releasing hormone agonist or antagonist. Metastases were determined radiographically as evidence of PC outside of the prostate, seminal vesicles, or pelvic nodes. PCSM was defined as metastatic, progressive CRPC at time of death with no obvious indication of another cause of death. ACM included death from any cause. Initiation of secondary treatments, including XRT and ADT were at the discretion of the treating physician.

Race was abstracted from the electronic medical records and is generally self reported. Current or last known home address zip code was collected and matched with zip code-level estimates of SES measures from the 2014 American Community Survey, including poverty (percent of individuals above the poverty level), median household income, education (percent of adults age ≥25 with high school or higher education), and employment (percent of civilian labor force employed)[15]. Each socioeconomic measure (household income, education, employment and poverty) was categorized into tertiles, with higher tertiles signifying higher SES. Poverty was considered the primary SES measure, while the other SES measures were considered secondary.

Of 5,515 patients in SEARCH, we excluded patients missing SES variables (n=16), preoperative PSA (n=168), or follow-up (n=117), and patients with race other than black or white (n=184). Our study cohort included 4,787 patients.

2.2 Statistical Analysis

Cox proportional hazards were used to test whether race and poverty were associated with BCR after RP. In Model 1, we adjusted for demographic and clinical characteristics, including age at surgery (continuous), surgery year (continuous), BMI (<25 kg/m^2, 25–29.0 kg/m^2, ≥30 kg/m^2, missing), PSA (continuous; log-transformed), surgical center (categorical), biopsy grade group (1, 2–3, 4–5, missing), and clinical stage (T1, T2–T4, missing). In Model 2, we adjusted for the same characteristics as in Model 1 but without biopsy grade or clinical stage but rather included pathological grade group (1, 2–3, 4–5, missing), extracapsular extension (yes, no, missing), seminal vesicle invasion (yes, no, missing), and positive margins (yes, no, missing). **After Bonferroni correction for analyzing seven different outcomes, there were no significant interactions between center and race for predicting any outcome. Therefore, results are not shown stratified by center.** The interaction between race and poverty was tested by including a cross product

term and main effects in the model including the Model 2 covariates. Additional analyses tested time from surgery to ADT, XRT, CRPC, metastases, ACM, and PCSM. Analyses were repeated testing the secondary SES measures of income, education, and employment.

Since we observed statistically significant interactions between race and poverty for the later outcomes, we ran models with a 6-tiered variable that accounted for both poverty and race (i.e. white and poverty tertile 1, black and poverty tertile 1, white and poverty tertile 2, etc.). For this analysis, we used forward selection with an entry criterion of alpha=0.05 to select covariates for each outcome to ensure models were not overfit.

3. Results

3.1 Patient characteristics

There were 3,083 (64%) white and 1,704 (36%) black patients in our cohort (Table 1). White patients were older, had earlier surgery year, lower PSA, higher clinical stage, fewer positive margins, more extracapsular extension, and less seminal vesicle invasion. White race was associated with higher SES on all four SES factors: poverty, income, employment, and education. Poverty, our primary SES measure, was defined as percent living above the poverty level. When split into tertiles, the first tertile ranged from 35.8% to 78.7%, the second from 78.8% to 85.5%, and the third from 85.6% to 100%. **Within each race, there were only modest differences in baseline characteristics among poverty tertiles.**

3.2 Poverty, Race, and Risk of PC and non-PC Outcomes

Median follow-up after surgery was 8.2 years (IQR: 4.5–12.5). During follow-up, 1,733 men developed a BCR, 1,045 received XRT, 806 received ADT, 161 developed CRPC, 218 developed metastases, 1,476 died of which 121 died from PC. **The 5-year rate for BCR was 43%, ADT was 13%, and XRT was 20%. The 10 year rates for CRPC, metastases, ACM, and PCSM were 3.5%, 4.6%, 24%, and 2.5%, respectively. Rates were similar between races, except there was a higher 5-year rate of receiving XRT in black men (25% vs. 18%).** On multivariable analysis controlling for demographic and clinical characteristics, there was a suggestion that black men were at increased BCR risk, though this did not reach significance (p=0.06). Beyond this one finding, neither race nor poverty were independently associated with BCR, receipt of ADT, receipt of XRT, CRPC, metastasis, or PCSM (Table 3). The highest poverty tertile (i.e. lowest poverty rate and highest SES) was associated with 20% decreased ACM versus the lowest tertile (p=0.001). However, race was not independently associated with ACM. Results were similar after adjusting for pathological features (Table 2, Model 2).

3.3 Poverty × Race Interactions and Risk of PC and non-PC Outcomes

There were statistically significant interactions between race and poverty in predicting CRPC (p=0.002), metastasis (p=0.014), and PCSM (p=0.004). Specifically, among white men, SES was unrelated to any of these outcomes (all $p \geq 0.22$) (Table 3). In contrast, among black men, SES was significantly related to all PC outcomes (all $p < 0.05$), though in general the lowest SES tertile was associated with the *best* outcomes. In both races, greater SES was

associated with lower ACM, reaching significance in the 3rd tertile vs. 1st tertile, though overall trends were only suggestive and did not reach statistical significance.

3.4 Secondary analysis of other SES measures

Results for other SES measures – income, education, and employment – are shown in supplementary tables 1–4. Results were similar in that black race was suggestively, but not significantly linked with BCR. While both race and SES were unrelated to any outcomes except ACM, which was lower with higher SES, results did not always reach significance (Supplementary Table 5). Moreover, similar to the analysis of poverty, though there were no significant interactions between SES and race for any outcome, black men in the lowest income tertile were at lower CRPC risk (HR 0.54, 95%CI 0.29–0.99) and PCSM (HR 0.46, 95%CI 0.23–0.95) compared to the lowest income white men (Supplementary Table 5). Otherwise, SES was unrelated to the outcomes studied in both black and white men.

4. Discussion

Many studies examined the relationship between race and PC outcomes, but few examined the interplay between race and SES[13, 16]. To address this gap, we looked at interactions between race and SES in relation to long-term PC outcomes including BCR, CRPC, and PCSM among men undergoing RP at multiple equal-access hospitals. We found that although SES alone was not associated with worse long-term outcomes, there were interactions between race and SES in predicting CRPC, metastasis, PCSM.

Specifically, black men from high poverty (i.e. lower SES) areas had a *lower* risk of CRPC, metastasis, and PCSM, while poverty was unrelated to PC outcomes among white men. This is a somewhat surprising finding given most research suggests lower SES is associated with *worse* access to resources such as screening and healthcare[5, 17]. While our findings require validation in other datasets, if confirmed, these results suggest in an equal-access setting, long-term PC outcomes may be better for low-SES black men then low SES white men. Our findings raise questions about the reasons black men of lower SES may experience better long-term outcomes than white men of similar SES. This carries clinical significance not only for men with PC, but for clinicians who should be aware of complicated risk factors that may be influencing patient outcomes. **However, whether these findings hold in other practice settings requires confirmation.**

Many prior studies regarding PC and SES focused on PC diagnosis. They found complex relationships between SES and PC incidence, treatment and outcomes[5, 11, 18]. While some studies found no association between SES and PC incidence, others linked increased education and income with increased risk, or lower income with increased risk of distant-stage PC and worse PCSM[8, 9, 17, 19, 20]. This may be explained by access to care and screening in some settings, but does not explain our findings that low SES black men are less likely to experience CRPC, metastasis, and PCSM than low SES white men. Because the VA is equal-access, the effects of reduced access to healthcare from low SES should be lessened. However, our previous study showed effects of SES on PC outcomes, even in an equal-access setting[12].

Our analysis found black men with lower SES had decreased risk of CRPC, metastasis, and PCSM. This was surprising, since we hypothesized that, even in an equal-access setting, lower SES men would experience worse outcomes, regardless of race. The better outcomes in lower income black men suggests there may be factors other than access that complicate outcomes. This disparity cannot be explained by differences in treatment after RP, since we found black and white men as well as different SES levels were equally likely to receive XRT. Few prior studies found worse survival in low SES white men compared to low SES black men. A study of geographical differences in cancer survival in Michigan found that when looking at small geographic units, predominantly white areas with chronic poverty had worse PC survival rates than black areas, however these results are difficult to generalize[21]. This finding may be the result of a systemic pattern in how patients are diagnosed, treated, and/or followed-up. It is possible our findings are caused by other confounding factors such as black men being less likely to follow-up at the VA and progressing and dying outside the VA, and therefore we miss these poor outcomes. The issue of long-term follow-up habits in an equal-access setting has not been well-studied; however, a study at the Nebraska-Western Iowa VA Healthcare System showed black patients were more than twice as likely as white patients to miss primary care appointments[22]. In our study, median follow-up for white patients was 102.1 months, while median follow-up for black patients was 89.7 months ($p<0.001$). When stratified by poverty, median follow-up was not significantly different ($p=0.347$). **Furthermore, when using number of PSA tests as a surrogate for follow-up quality, number of PSAs did not vary by race (p=0.86) or poverty tertile (p=0.81),** suggesting differential follow-up **or quality of follow-up** may not fully explain our results.

Although the results were not statistically significant ($p<0.06$), we found black men had 10–11% increased BCR risk after adjusting for SES. This is similar to our previous SEARCH studies, which found black men had 20% increased BCR risk[12]. Moreover, the lack of differences in long-term outcomes by race are also consistent with our prior study. Thus, while race alone may not be prognostic, the complex interplay between SES and race may hold important prognostic information into PC.

Consistent with findings that lower SES is correlated with lower life expectancy and higher mortality, we found lower poverty (higher SES) was associated with reduced ACM[23]. Some of the excess mortality from low SES may be related to differences in health behavior, however this only partially accounts for excess mortality[24]. Unfortunately, our database lacked information on health behaviors and thus we could not include this.

Our study has several limitations. Since it was retrospective, patient treatment and follow-up was as the discretion of the treating physician. **Furthermore, we did not have data on exposures or behaviors that may have confounded our results such as diet and exercise information.** We only examined men who underwent an RP at the VA, so we may have missed patients who could differ from this cohort in some way, such as having worse disease at diagnosis or those not treated at equal-access centers. Previous studies suggest lower SES may be linked to less frequent use of RP, which may have influenced the cohort[25]. **We were unable to account for patients (possibly the higher SES patients) who may receive care outside the VA after their RP.** Though we measured follow-up time **and number of PSA tests as a surrogate of follow-up quality**, measuring **other metrics of** quality

(number, frequency, and length) of follow-up visits was beyond the scope of this study. We used zip codes to match patients to SES estimates; however, this method has been criticized by some as an imperfect measure of SES because zip codes are primarily administrative divisions and may not be as homogenous in their "population characteristics, economic status, and living conditions" as a census tract[26]. **Moreover, we did not have detailed information on whether patients moved over time and/or their SES changed.** Nonetheless, other studies also found zip code-based estimates of SES are associated with cancer outcomes[18, 27]. **Lastly, as 97% of men in our database are black or white, we did not have sufficient number to examine other races.**

5. Conclusions

Among men undergoing RP in across multiple equal access medical centers, we found black men with lower SES had decreased risk of CRPC, metastasis and PCSM after RP, relative to all other groups (higher SES black men and white men regardless of SES). If validated in future studies **and in other practice settings**, these better long-term outcomes for low-SES black men suggest there may be complex societal factors affecting outcomes. Further research is needed to understand the interplay between SES, race, and long-term PC outcomes **across various practice settings**.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Black race, but not SES, had increased risk of biochemical recurrence vs white men
- There were interactions between SES and race for CRPC, metastasis, and PCSM
- Lower SES was associated with *decreased* CRPC, metastases, and PCSM in black men only
- Higher SES was associated with decreased ACM in both

Table 1.

Baseline patient characteristics by race and poverty tertile

	Black men			White men		
	Tertile 1 (N=856)	**Tertile 2 (N=497)**	**Tertile 3 (N=351)**	**Tertile 1 (N=777)**	**Tertile 2 (N=1058)**	**Tertile 3 (N=1248)**
Age						
Median	61	61	61	64	64	64
Q1, Q3	56, 65	56, 65	57, 65	60, 68	60, 68	60, 68
Year of surgery						
Median	2007	2006	2006	2003	2004	2004.5
Q1, Q3	2001, 2011	2001, 2011	2001, 2010	1997, 2009	1997, 2009	1998, 2010
BMI						
<25	204 (24%)	108 (22%)	80 (23%)	163 (21%)	185 (17%)	247 (20%)
25–29.9	348 (41%)	196 (39%)	143 (41%)	294 (38%)	395 (37%)	536 (43%)
>30	266 (31%)	167(34%)	110 (31%)	214 (28%)	304 (29%)	315 (25%)
Missing	38 (4%)	26 (5%)	18 (5%)	106 (14%)	174 (16%)	150 (12%)
PSA (ng/mL)						
Median	7.3	6.7	7.0	6.3	6.2	6.1
Q1, Q3	5.0, 11.3	5.1, 10.5	4.8, 11.4	4.7, 10.1	4.6, 9.1	4.4, 9.2
Clinical stage						
T1	550 (64%)	344 (69%)	224 (64%)	371 (48%)	484 (46%)	626 (50%)
T2–T4	273 (32%)	129 (26%)	117 (33%)	331 (43%)	426 (40%)	520 (42%)
Missing	33 (4%)	24 (5%)	10 (3%)	75 (10%)	148 (14%)	102 (8%)
Biopsy Gleason score						
2–6	382 (45%)	226 (45%)	153 (44%)	353 (45%)	510 (48%)	559 (45%)
7	333 (39%)	203 (41%)	142 (40%)	242 (31%)	352 (33%)	393 (31%)
8–10	117 (14%)	55 (11%)	42 (12%)	97 (12%)	97 (9%)	177 (14%)
Missing	24 (3%)	13 (3%)	14 (4%)	85 (11%)	99 (9%)	119 (10%)
Pathological Gleason sc ore						
2–6	235 (27%)	128 (26%)	101 (29%)	230 (30%)	379 (36%)	421 (34%)
7	512 (60%)	313 (63%)	204 (58%)	378 (49%)	474 (45%)	564 (45%)
8–10	97 (11%)	45 (9%)	34 (10%)	99 (13%)	133 (13%)	172 (14%)
Missing	12 (1%)	11 (2%)	12 (3%)	70 (9%)	72 (7%)	91 (7%)
Positive surgical margins	385 (45%)	221 (44%)	149 (42%)	301 (39%)	345 (33%)	445 (36%)
Extracapsular extension	156 (18%)	79 (16%)	66 (19%)	181 (23%)	202 (19%)	230 (18%)
Seminal vesicle invasion	104 (12%)	52 (10%)	43 (12%)	91 (12%)	98 (9%)	101 (8%)
Employment						
Tertile 1	488 (57%)	173 (35%)	73 (21%)	393 (51%)	287 (27%)	149 (12%)
Tertile 2	301 (35%)	216 (43%)	96 (27%)	280 (36%)	409 (39%)	330 (26%)
Tertile 3	67 (8%)	108 (22%)	182 (52%)	104 (13%)	362 (34%)	769 (62%)
Income						
Tertile 1	697 (81%)	76 (15%)	8 (2%)	545 (70%)	277 (26%)	32 (3%)
Tertile 2	153 (18%)	328 (66%)	70 (20%)	212 (27%)	556 (53%)	285 (23%)

	Black men			White men		
	Tertile 1 (N=856)	**Tertile 2 (N=497)**	**Tertile 3 (N=351)**	**Tertile 1 (N=777)**	**Tertile 2 (N=1058)**	**Tertile 3 (N=1248)**
Tertile 3	6 (1%)	93 (19%)	273 (78%)	20 (3%)	225 (21%)	931 (75%)
Education						
Tertile 1	528 (62%)	167 (34%)	24 (7%)	473 (61%)	337 (32%)	65 (5%)
Tertile 2	302 (35%)	210 (42%)	64 (18%)	245 (32%)	513 (48%)	251 (20%)
Tertile 3	26 (3%)	120 (24%)	263 (75%)	59 (8%)	208 (20%)	932 (75%)
Follow-up						
Median	86.5	89.5	92.9	105.1	103.0	97.8
Q1, Q3	45.6, 140.6	49.2, 142.1	52.5, 145.5	61.7, 158.4	55.9, 156.4	57.2, 155.3
Number of PSA tests						
Median	14	15	15	15	14	14
Q1, Q3	8, 23	9, 23	9, 23	9, 23	9, 21	9, 22

‘PSA=prostate-specific antigen, Q1=25th percentile, Q3=75th percentile, PC=prostate cancer, RP=radical prostatectomy’

Table 2:

Hazard ratios for the associations between both race and poverty and each of the outcomes after radical prostatectomy

	Model 1		Model 2		Interaction with race
	HR (95% CI)	**P**	**HR (95% CI)**	**P**	
Biochemical recurrence					
Black vs. white	1.11 (0.99–1.24)	0.060	1.11 (0.99–1.25)	0.061	0.69
Poverty tertile 1	*Ref.*	0.83	*Ref.*	0.55	
Poverty tertile 2	1.01 (0.90–1.14)		1.05 (0.93–1.18)		
Poverty tertile 3	0.99 (0.87–1.11)		1.04 (0.92–1.17)		
Receipt of ADT					
Black vs. white	0.98 (0.84–1.16)	0.87	0.97 (0.82–1.14)	0.70	0.90
Poverty tertile 1	*Ref.*	0.062	*Ref.*	0.21	
Poverty tertile 2	0.99 (0.83–1.17)		1.00 (0.84–1.19)		
Poverty tertile 3	0.84 (0.70–1.00)		0.89 (0.74–1.06)		
Receipt of XRT					
Black vs. white	1.04 (0.90–1.19)	0.63	1.09 (0.95–1.26)	0.21	0.55
Poverty tertile 1	*Ref.*	0.88	*Ref*	0.54	
Poverty tertile 2	1.03 (0.88–1.20)		1.09 (0.94–1.27)		
Poverty tertile 3	0.99 (0.84–1.15)		1.04 (0.89–1.22)		
CRPC					
Black vs. white	1.10 (0.75–1.59)	0.63	1.05 (0.72–1.53)	0.81	7 0.002
Poverty tertile 1	*Ref.*	0.83	*Ref*	0.38	
Poverty tertile 2	1.11 (0.75–1.66)		1.17 (0.79–1.75)		
Poverty tertile 3	1.04 (0.70–1.54)		1.19 (0.80–1.78)		
Metastases					
Black vs. white	1.24 (0.90–1.70)	0.20	1.20 (0.87–1.66)	0.26	0.014
Poverty tertile 1	*Ref.*	0.63	*Ref.*	0.84	
Poverty tertile 2	1.27 (0.91–1.77)	y	1.33 (0.96–1.85)		
Poverty tertile 3	0.91 (0.64–1.29)		1.02 (0.71–1.45)		
All-cause mortality					0.62
Black vs. white	0.98 (0.87–1.12)	0.81	0.98 (0.86–1.11)	0.72	
Poverty tertile 1	*Ref*	0.001	*Ref.*	0.004	
Poverty tertile 2	0.95 (0.84–1.08)		0.95 (0.84–1.08)		
Poverty tertile 3	0.80 (0.70–0.91)		0.82 (0.72–0.94)		
PSCM					0.004
Black vs. white	0.91 (0.58–1.43)	0.69	0.92 (0.59–1.45)	0.73	

	Model 1		Model 2		Interaction with race
	HR (95% CI)	**P**	**HR (95% CI)**	**P**	
Poverty tertile 1	*Ref.*	0.26	*Ref.*	0.63	
Poverty tertile 2	0.92 (0.59–1.43)		1.01 (0.65–1.58)		
Poverty tertile 3	0.77 (0.49–1.21)		0.89 (0.56–1.41)		

Model 1 is adjusted for race, SES measure, age, year, BMI, PSA, surgical center, biopsy Grade group, and clinical stage

Model 2 is adjusted for race, SES measure, age, year, BMI, PSA, surgical center, pathological Grade group, extracapsular extension, seminar vesicle invasion, positive surgical margins, and prostate weight

Table 3:

Hazard ratios for CRPC, metastases, all-cause mortality, and prostate cancer-specific mortality after radical prostatectomy stratified by race

	HR (95% CI)	Race-specific p-value[5]
CRPC[1]		
Poverty tertile 1, white	*Ref.*	0.29
Poverty tertile 2, white	0.68 (0.42–1.10)	
Poverty tertile 3, white	0.83 (0.53–1.30)	
Poverty tertile 1, black	0.45 (0.25–0.80)	0.012
Poverty tertile 2, black	1.23 (0.73–2.09)	
Poverty tertile 3, black	1.03 (0.55–1.92)	
Metastases[2]		
Poverty tertile 1, white	*Ref.*	0.61
Poverty tertile 2, white	0.89 (0.59–1.35)	
Poverty tertile 3, white	0.81 (0.54–1.22)	
Poverty tertile 1, black	0.62 (0.38–1.01)	0.004
Poverty tertile 2, black	1.59 (1.01–2.51)	
Poverty tertile 3, black	0.98 (0.55–1.75)	
All-cause mortality[3]		
Poverty tertile 1, white	*Ref.*	0.068
Poverty tertile 2, white	0.95 (0.81–1.11)	
Poverty tertile 3, white	0.84 (0.72–0.98)	
Poverty tertile 1, black	1.02 (0.85–1.22)	0.15
Poverty tertile 2, black	0.98(0.79–1.21)	
Poverty tertile 3, black	0.76 (0.59–0.98)	
PSCM[4]		
Poverty tertile 1, white	*Ref.*	022
Poverty tertile 2, white	0.73 (0.43–1.22)	
Poverty tertile 3, white	0.64 (0.39–1.08)	
Poverty tertile 1, black	0.40 (0.21–0.78)	0.049
Poverty tertile 2, black	1.00 (0.53–1.88)	
Poverty tertile 3, black	0.85 (0.40–1.78)	

[1] CRPC model is adjusted for seminal vesicle invasion, pathological grade group, year of surgery, and PSA

[2] Metastases model is adjusted for seminal vesicle invasion, pathological grade group, year of surgery, surgical center, and extracapsular extension

[3] All-cause mortality model is adjusted for age, seminal vesicle invasion, year of surgery, obesity, pathological Grade group, extracapsular extension, and surgical center

[4] Prostate-cancer specific mortality model is adjusted for seminal vesicle invasion, pathological Grade group, obesity, positive surgical margins, year of surgery, and extracapsular extension

[5] Race-specific p-value is calculated using the Wald test of the three categories pertaining to that race