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ORIGINAL PAPER

The association between race and prostate cancer risk on initial biopsy in an equal access, multiethnic cohort

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Abstract
Purpose Population-based studies have established a link between race and prostate cancer (PC) risk, but whether race predicts PC after adjusting for clinical characteristics is unclear. We investigated the association between race and risk of low- and high-grade PC in men undergoing initial prostate biopsy in an equal access medical center.
Methods We conducted a retrospective record review of 887 men (48.6 % black, 51.4 % white) from the Durham Veterans Affairs Medical Center who underwent initial prostate biopsy between 2001 and 2009. Multivariable logistic regression analysis of race and biopsy outcome was conducted adjusting for age, body mass index, number of cores taken, prostate-specific antigen (PSA), and digital rectal examination findings. Multinomial logistic regression was used to test the association between black race and PC grade (Gleason <7 vs. ≥ 7).
Results Black men were younger at biopsy (61 vs. 65 years, $p < 0.001$) and had a higher pre-biopsy PSA (6.6 vs. 5.8 ng/ml, $p = 0.001$). A total of 499 men had PC on biopsy (245 low grade; 254 high grade). In multivariable analyses, black race was significantly predictive of PC overall [odds ratio 1.50, $p = 0.006$] and high-grade PC [relative risk ratio (RRR) 1.84, $p = 0.001$], but was not significantly associated with low-grade PC (RRR 1.29, $p = 0.139$).
Conclusion In an equal access healthcare facility, black race was associated with greater risk of PC detection on initial biopsy and of high-grade PC after adjusting for clinical characteristics. Additional investigation of mechanisms linking black race and PC risk and PC aggressiveness is needed.

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Abbreviations

BMI	Body mass index (kg/m^2)
CI	Confidence interval
DRE	Digital rectal exam
DVAMC	Durham Veterans Affairs Medical Center
IQR	Interquartile range
OR	Odds ratio
RRR	Relative risk ratio
P	*p* value
PC	prostate cancer
PSA	prostate specific antigen (ng/mL)

Introduction

In Western society, prostate cancer (PC) is the most frequently diagnosed non-skin malignancy in men, with black men twice as likely to die from PC as white men [1]. The increased risk of aggressive disease could be due to increased risk of having PC on biopsy [2, 3], higher risk of aggressive disease at diagnosis [4], poorer outcomes after treatment [5], or a combination thereof.

Existing data are inconsistent as to whether race is associated with poor outcomes [6–8]. However, a key question is whether factors such as stage, grade, and other clinical parameters (i.e., "all clinical features being equal") can explain poorer outcomes in black men. One approach to accomplishing this uses data from an equal access medical center, wherein differences in access to care are minimized and detailed clinical characteristics are available and can be accounted for [9]. In an equal access setting, black men are more likely to have a prostate-specific antigen (PSA) recurrence after radical prostatectomy even after controlling for clinical characteristics [10], supporting the hypothesis that black race is linked with PC aggressiveness.

There are several possible ways that black men could have higher mortality from PC: increased risk of having PC on biopsy (i.e., higher incidence) or increased risk of PC tumor aggressiveness. Population studies of black men have not thoroughly included clinical risk factors to assess increased PC incidence. Moreover, individual studies showed inconsistent results as to whether black race is a risk factor for PC at the time of biopsy [11, 12]. In addition, studies examining tumor aggressiveness as a function of race have been inconclusive [13, 14]. This study seeks to understand whether race can independently account for these differences after accounting for clinical features.

A leading provider of national health care, the Durham (North Carolina) Veterans Affairs Medical Center (DVAMC) is an equal access hospital that serves more than 200,000 veterans in a 26-county area [15]. Honorably discharged veterans who meet financial eligibility criteria receive medical care at little or no cost, minimizing financial access to care issues [16]. Minority populations are overrepresented in lower socioeconomic groups and are overrepresented in the armed forces, comprising ~25 % of the force [17, 18]. As such, given that the DVAMC maintains comprehensive electronic medical records, it is an ideal environment in which to assess the effects of race on PC outcomes [19, 20].

As such, we examined the association between black race and biopsy outcomes in men undergoing initial prostate biopsy at the DVAMC. We hypothesized that in this population of veterans where poor access to care is minimized, black race is associated with increased PC risk and disease severity.

Materials and methods

Study participants

After obtaining institutional review board approval, we conducted a retrospective review of 1,277 men who underwent an initial prostate needle biopsy between 2001 and 2009 at the DVAMC. Enrollment methods have been described previously [21]. Participants were referred for biopsy through the urology clinics, typically due to elevated PSA or abnormal digital rectal examination (DRE) findings. Participants who qualified were then encouraged to schedule a biopsy. Upon returning for their biopsy, a repeat pre-biopsy PSA test was performed to confirm the indication. We excluded 19 men who were missing data on race and whose race was neither black nor white. Although missing data can be imputed, to minimize bias, we elected to exclude men missing data on pre-biopsy serum PSA ($n = 43$), DRE findings ($n = 127$), body mass index (BMI) ($n = 88$), total number of biopsy cores ($n = 112$), and Gleason score ($n = 1$). When men who were missing data were included in analysis, the associations between race and biopsy outcomes (PC status and PC grade) did not change. Thus, our final study population consisted of 887 subjects (69.5 %) with complete data available for analysis.

Data collection

From participant records, we abstracted age at biopsy, race, BMI, DRE, pre-biopsy PSA, prostate volume, year of biopsy, total number of biopsy cores, and biopsy findings

Table 1 Clinical characteristics and biopsy outcomes for men undergoing an initial prostate biopsy at the DVAMC, 2001–2009 with complete data for all variables ($n = 887$)

	Black $n = 431$ Median (IQR)	White $n = 456$ Median (IQR)	p*
Year of biopsy	2005 (2002–2006)	2004 (2002–2006)	0.29
Total number of cores taken	11 (8–12)	11 (8–12)	0.84
Age at biopsy (years)	61 (57–68)	65 (60–70)	<0.001
PSA (ng/ml)	6.6 (4.7–12.1)	5.8 (4.4–8.4)	0.001
Total number of positive cores among men with cancer	4 (2–6)	3 (1–6)	0.08
	No. (%)	No. (%)	p^+
BMI (kg/m^2)			0.32
25	110 (25.5)	100 (21.9)	
25–29.99	160 (37.1)	188 (41.2)	
30–34.99	114 (26.5)	109 (23.9)	
≥35	47 (10.9)	59 (12.9)	
Abnormal DRE	109 (25.3)	141 (30.9)	0.06
Biopsy outcome			0.001
No cancer	164 (38.1)	224 (49.1)	
Any cancer	267 (61.9)	232 (50.9)	
Gleason grade distribution			0.001
Low grade (<7)	122 (28.3)	123 (27.0)	
High grade (≥7)	145 (33.6)	109 (23.9)	

BMI body mass index, *DRE* digital rectal examination, *DVAMC* Durham Veteran Affairs Medical Center, *IQR* interquartile range, *p p* value, *PSA* prostate-specific antigen

Statistical analyses: * rank-sum test; $^+$ chi-squared test

Table 2 Crude and adjusted models of Black race as an independent predictor of cancer on initial biopsy at the DVAMC, 2001–2009 with complete data for all variables ($n = 887$)

	Model 1: adjusted			Model 2: unadjusted		
	OR	95 % CI	p	OR	95 % CI	p
Black race[a]	1.50	1.12–2.00	0.006	1.57	1.20–2.05	<0.001
Age (years)	1.00	0.98–1.02	0.90			
Total number of cores taken	0.94	0.89–1.00	0.07			
BMI (kg/m^2), relative to <25 kg/m^2						
25–29.99	0.57	0.39–0.83	0.02[#]			
30–34.99	0.58	0.39–0.88				
≥35	0.68	0.41–1.13				
Abnormal DRE	2.25	1.61–3.15	<0.001			
PSA (ng/ml)	2.03	1.61–2.55	<0.001			

Statistical analysis: logistic regression for cancer on biopsy

Models adjusted for age, log-PSA, BMI, DRE, and total number of cores taken

OR odds ratio for black race versus white race, *CI* confidence interval, *p p* value

[#] Likelihood ratio test

[a] Reference group was white race

(benign vs. malignant and Gleason score, if positive). Prostate volume, an established predictor of PC risk, was unavailable for the majority of men and was not included in the analyses.

Statistical analysis

Our primary and secondary outcomes were PC risk on initial biopsy and PC grade, respectively. PC risk on initial biopsy was measured based on cancer status indication derived from medical record pathology reports. PC grade was defined as no PC (reference group), low-to-intermediate-risk PC (Gleason Score <7), and high-risk PC (Gleason Score ≥7) [22]. Race, the primary exposure variable, was based on self-report. Continuous variables that were not normally distributed (age, year of biopsy, total number of biopsy cores, prostate volume, and pre-biopsy PSA) were compared using the Wilcoxon rank-sum test. Categorical variables such as DRE (normal/abnormal) and BMI (<25, 25–29.9, 30–34.9, and ≥35 kg/m^2) were compared using the chi-squared test.

We evaluated the risk of PC diagnosis by race using odds ratios (ORs) in multivariable logistic regression models. In analysis where PC grade (low-grade Gleason <7 vs. no PC, high-grade Gleason ≥7 vs. no PC) was examined, multinomial logistic regression was used. The models were adjusted for age, BMI, total number of cores, PSA (logarithmically transformed), biopsy calendar year, and DRE.

All statistical analyses were performed using Stata 12.1 (StataCorp, College Station, TX, USA). Two-tailed p values of ≤0.05 were considered statistically significant.

Results

There were similar proportions of black ($n = 431$, 48.6 %) and white men ($n = 456$, 51.4 %) in this cohort. Black men were younger at biopsy (median age: 61 vs. 65, $p < 0.001$, Table 1) and had higher pre-biopsy PSA values (6.6 vs. 5.8 ng/ml, $p = 0.001$). Black men were also less likely to have an abnormal DRE ($p = 0.06$). Black and white men had comparable BMI ($p = 0.32$), total number

Table 3 Crude and adjusted models of Black race as an independent predictor of cancer grade on initial biopsy at the DVAMC, 2001–2009 with complete data for all variables ($n = 887$)

Cancer outcome: low grade (<7)	Model 1: adjusted			Model 2: unadjusted		
	RRR	95 % CI	*p*	RRR	95 % CI	*p*
Black race[a]	1.29	0.92–1.80	0.14	1.35	0.98–1.87	0.06
Age (years)	0.99	0.97–1.01	0.26			
BMI (kg/m^2), relative to <25 kg/m^2			0.02[#]			
25–29.99	0.58	0.38–0.90				
30–34.99	0.63	0.40–1.01				
≥35	0.62	0.34–1.12				
Total number of cores taken	0.92	0.86–0.99	0.02			
Abnormal DRE	1.45	0.97–2.16	0.07			
PSA (ng/ml)	1.37	1.04–1.80	0.02			
Cancer outcome: high grade (≥7)	Model 1: adjusted			Model 2: unadjusted		
	RRR	95 % CI	*p*	RRR	95 % CI	*p*
Black race[a]	1.84	1.28–2.66	0.001	1.82	1.32–2.50	<0.001
Age (years)	1.01	0.99–1.04	0.30			
BMI (kg/m^2), relative to <25 kg/m^2						
25–29.99	0.55	0.34–0.86	0.02[#]			
30–34.99	0.53	0.32–0.88				
≥35	0.77	0.42–1.41				
Total number of cores taken	0.97	0.90–1.05	0.52			
Abnormal DRE	3.41	2.29–5.07	<0.001			
PSA (ng/ml)	2.91	2.21–3.83	<0.001			

Statistical analysis: multinomial logistic regression for cancer grade on biopsy

Models adjusted for age, log-PSA, BMI, DRE, and total number of cores taken

RRR relative risk ratio for no cancer versus low grade versus high grade, *CI* confidence interval, *p* *p* value

[#] Likelihood ratio test

[a] Reference group was white race

of biopsy cores ($p = 0.84$), and year of biopsy ($p = 0.29$) (Table 1).

Of the 887 men, 499 had PC on biopsy (56.3 %, Table 1). Black men (61.9 %) were significantly more likely to have PC on biopsy than white men (50.9 %, $p \leq 0.001$). This association changed minimally after adjusting for age, total number of cores, BMI, DRE, and PSA ($p = 0.006$, Table 2). Of the men with a positive biopsy, high-grade PC was more common in black men than in white men (54.3 vs. 47.0 %), although the difference was not statistically significant ($p = 0.10$). Table 3 shows the association between race and PC grade on initial biopsy. Compared to white race, unadjusted analysis showed black race was more strongly linked to high-grade PC ($p < 0.001$) than low-grade PC ($p = 0.06$). After adjusting for age, PSA, BMI, DRE, and total number of cores taken, race was not significantly associated with low-grade PC ($p = 0.14$), but remained significantly associated with high-grade PC ($p = 0.001$).

Among 81 (70.4 %) black men and 34 (29.6 %) white men under the age of 55 years ($n = 115$), black men had higher pre-biopsy PSA (5.5 vs. 5.1 ng/ml, $p = 0.12$) and a greater proportion had PC (black 60.5 % vs. white 47.1 %, $p = 0.17$) though the differences were not statistically significant. Further, multivariable logistic regressions for PC on biopsy ($p = 0.40$), and multivariable multinomial logistic regression analyses (relative to no PC, low grade: $p = 0.68$, high grade: $p = 0.26$) suggested positive associations between race and PC, and race and PC grade in this subset, though the associations were not statistically significant (data not shown).

Discussion

Accounting for almost 10 % of cancer deaths in American men, PC remains the most prevalent form of cancer in men with an estimated 238,590 new cases diagnosed in 2013

[23]. Black men have a 67 % higher incidence of PC than white men [23]. While population-level studies have consistently shown that the incidence and mortality burden is highest among black men, whether this can be explained by inadequate access to care has remained unclear [24, 25]. Our key finding is that in an equal access setting with analyses adjusted for baseline clinical characteristics, black men have an increased risk of PC on initial biopsy. This association was stronger among men with high-grade PC. This supports the hypothesis that black race is integrally linked with more aggressive PC grade at diagnosis and differences in incidence and mortality are unlikely due to access to care alone.

Overall data on whether race predicts PC diagnosis after adjusting for clinical characteristics are inconsistent. The magnitude of the positive association between black race and increased PC risk (50 % increased risk) is consistent with that of population-level findings from both SEER (67 % increased risk) and the PC Prevention Trial (40 % increased risk) [1, 26]. Although the association between black race and increased PC risk is well documented, several studies found that race is not a predictor of PC risk in populations on repeat biopsy [27, 28], or in men who had fewer than 12-core biopsy taken [12]. Such findings suggest that both screening and biopsy method (12 vs. 6 cores) may have an effect on PC risk in these populations, given that men who are more frequently screened are more likely to be diagnosed. In addition, a 12-core biopsy increases the likelihood of detecting PC on biopsy [21]. Furthermore, one study found that race was not associated with increased PC risk after adjusting for socioeconomic status and literacy [27]. However, the study may have lacked power to detect a significant association due to a small sample size ($n = 212$) [29]. Collectively, while these studies suggest that race is not an independent predictor of PC risk, none also examined race in relation to PC grade or controlled for other clinical factors that may influence risk on initial biopsy.

To our knowledge, our study is the first conducted in a contemporary cohort that reports black race is an independent predictor of total and high-grade PC on initial biopsy. Moreover, our results that black race is linked to increased PC risk are consistent with those of earlier studies, which included non-contemporary cohorts of men and did not examine the relationship between race and PC grade as an outcome [11]. While our study did not account for psychosocial and behavioral barriers, a notable feature of our cohort is that the DVAMC is an equal access hospital, which minimizes the effects of barriers to care related to financial status. Our findings point to a need for additional studies aimed at understanding the molecular underpinnings for this phenomenon and support the idea that black men should be targeted more aggressively for initial PC early detection efforts.

There has always been controversy surrounding the benefits and risks of PSA screening and this has intensified recently with the publication of the US Preventive Services Task Force (USPSTF) guidelines on PSA screening and the American Urological Association (AUA) guidelines on PC screening. Specifically, the USPSTF suggests the risks of PSA screening outweighs the harms [30], while the AUA suggests shared decision making, but only for men between the ages of 55 and 69 [31]. Notably, the AUA guidelines discuss recommendations for men at "average risk" of PC. However, based upon the current data and those of others, it is clear that black men are not average: they have greater than average risk.

While one could argue that black men are in most need of screening and early detection, a counterargument could be made that given the inherent aggressiveness of PC tumors in black men, that screening alone may not be beneficial. During the era of PSA screening, PC deaths declined to a greater extent in black men than white men [23], suggesting that screening is beneficial for black men. Therefore, if further studies support our findings that even at initial diagnosis, black men present at younger age and with more aggressive disease, this would strongly support targeted screening approaches for all men of African ancestry—even below the AUA guidelines age limits of 55.

In our analysis of men younger than 55 years, there were a larger number of black men, they had higher PSA levels, and, relative to white men, had more aggressive PCs. While an association between race and PC on biopsy and race and PC grade is evident, there were no statistically significant associations in these models, which could be the result of small numbers and low power within men in this subset with complete data from our dataset. Thus, while this subset analysis does not show that early and aggressive screening helps black men, it still provides evidence that in conjunction with population-level data, screening younger black men will identify more PCs at an earlier stage, which is a prerequisite for screening to improve outcomes.

As with any retrospective study, there are limitations regarding the outcomes reported and generalizability investigated here. One potential source for information bias lies in the fact that we are evaluating data from a VA patient population. It has been suggested that VA populations are not representative of the general North Carolina population [32, 33] that may seek treatment at a university-sponsored hospital [34] nor of the general US population as they have lower rates of cancer mortality relative to the whole [35]. Furthermore, patients at VA hospitals are typically different than people in the generalized population. These men have healthcare access, have a relatively standardized level of education, have access to preventive care measures, and have encountered the healthcare system [36]. VA patients also have, in general, poorer health status

and lower socioeconomic status than the general population [19]. Thus, while the findings of our DVAMC study suggest a role in race and PC incidence and tumor aggressiveness, future studies that can deconstruct and evaluate the social components of "race" and evaluate them independently along with PC risk and aggressiveness are needed.

Furthermore, because we had access to data from a single equal access facility, our sample size of men with complete data is relatively small. Additionally, exclusions are high, as the data were not uniformly collected for all patients, though similar trends were noted in men who did not have complete data available for analysis. Even though controlling for socioeconomic status would have been informative, we did not have data on components that describe this variable (i.e., zip codes, income, education), even though some data suggest that when controlling for socioeconomic factors, black race remains an independent predictor of disease recurrence and/or PC mortality [37]. Moreover, family history data were not available for men included in this cohort. Additionally, in this study, race was self-reported and not measured by ancestral markers. Given the heterogeneity of ancestral markers among individuals who self-report as black, race becomes less a biological phenomenon due to the inability to determine at which point one is considered "black" or not. Race, therefore, in self-report cases, becomes a social phenomenon that is hard to quantify and measure. As a result, important social and cultural structures may not be controlled for or measured that influence results [38]. However, self-reported race lends insight into the cultural and social indices that people use to self-identify and provides valuable insight beyond mere genetics. It is clear that race is not the biological classification strata historically represented in research and other studies, thus further studies to fully understand the link among black race, PC risk, and PC tumor aggressiveness are needed. Nonetheless, future studies should examine the combination of ancestral markers, genetic mapping, and ethnicity to determine the exact relationship between genetic racial identification, social racial identification, and adherence to cultural norms with respect to PC predictability on biopsy. Given that genetics only accounts for approximately 5–42 % [39] of the biological differences between races, it is clear we need to develop a better understanding of the non-genetic drivers of racial differences as it relates to PC risk.

Conclusion

In an equal access medical center, we found that black race was positively associated with an increased risk of overall and high-grade PC risk on initial prostate biopsy even after adjusting for key clinical characteristics. This suggests that black men are at a heightened risk for PC, which should be taken into account when considering whether to screen black men given current PC screening guidelines are based on average-risk men.

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Conflict of interest The authors have no actual or potential conflict of interest in relation to this manuscript.

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