Published in final edited form as:

Cancer Causes Control. 2004 September ; 15(7): 647–655. doi:10.1023/B:CACO.0000036172.63845.d4.

Lifestyle and prostate cancer among older African-American and Caucasian men in South Carolina

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Abstract

Objective—We investigated the association between lifestyle and prostate cancer risk among Caucasian and African– American men, separately.

Methods—This population-based case–control study of prostate cancer among men aged 65–79 years was conducted between 2000 and 2002 in South Carolina. Telephone interviews were completed with 416 incident prostate cancer cases ascertained through the South Carolina Central Cancer Registry, and 429 controls identified through the Health Care Financing Administration Medicare beneficiary file (with respective response rates of 71% and 64%).

Results—Caucasian men working in production, transportation, and material moving had increased prostate cancer risk (odds ratio [OR]=2.04, 95% confidence interval [CI] 1.17–3.54), while African-American men in the military had reduced prostate cancer risk (OR=0.19, 95% CI 0.05–0.76). Having five or more prostate specific antigen (PSA) tests within the past five years was associated with prostate cancer among Caucasian men; however, African-American men with prostate cancer tended to have fewer PSA tests. Increasing lycopene consumption was associated with a reduced risk of prostate cancer among Caucasian men ($p=0.03$), but not among African– American men.

Conclusions—In this population-based case–control study conducted in South Carolina we did not find marked differences in lifestyle factors associated with prostate cancer by race.

Keywords

case–control studies; lifestyle; prostate cancer

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Introduction

Prostate cancer is the most frequently diagnosed cancer in the US, and the second leading cause of cancer deaths among men. From 1996 to 2000, the age-adjusted incidence was 65% higher in African-American men than in white men (276.8 vs. 167.5 per 100,000) and mortality was 140% higher (73.0 vs. 30.2 per 100,000) [1]. From 1992 to 1999, African-American men were 5% less likely to survive beyond five years after diagnosis than white men [1]. This disparity in survival continues to exist at each stage of disease, even when financial barriers have been removed [2, 3].

Little is understood about the etiology of prostate cancer nor do we know what factors might explain why African-American men are at greater risk relative to white men. Age is the strongest risk factor for prostate cancer, and the occurrence of prostate cancer before age 45 is rare [1, 4]. Family history of prostate cancer has been identified as a risk factor for prostate cancer in both case-control studies [5, 6] and cohort studies [7, 8]. There is conflicting evidence on the relation between prostate cancer and risk factors such as obesity, diet, and physical inactivity [4]. Few studies have had sufficient numbers of African-American men to examine risk factors for prostate cancer by race [3, 5, 6, 9–13]. The purpose of this analysis was to investigate the association between lifestyle and prostate cancer risk among African-American and Caucasian men, separately.

Materials and methods

This population-based case-control study was conducted in South Carolina from 2000 to 2002. Cases diagnosed with primary invasive prostate cancer between October 1999 and September 2001 were identified through the South Carolina Central Cancer Registry (SCCCR). To ensure that a majority of cases were identified during the study period, a rapid case ascertainment procedure was developed and implemented between hospitals, pathology laboratories, and the SCCCR. Eligible cases were South Carolina residents, aged 65–79, whose prostate cancer was histologically confirmed, and whose physicians had given permission for research staff to contact their patient. During the study period, a total of 755 Caucasian men and 384 African-American men with localized disease (stages I and II), and 144 Caucasian men and 81 African-American men with advanced disease (stages III and IV) were reported to the SCCCR. Of these, 551 Caucasian men and 245 African-American men with localized disease, and 98 Caucasian men and 70 African-American men with advanced disease met the eligibility criteria. We selected all eligible cases with advanced disease, and a random sample of men with localized disease within five-year age group (42% of Caucasian men and 83% of African-American men). A total of 426 prostate cancer cases (70.6% of eligible cases) completed a standardized telephone interview. Of potentially eligible cases, 71 refused (11.8%), 24 died prior to the interview (4.0%), 59 were not located (9.8%), and 23 were too sick to participate (3.8%). A greater percentage of Caucasian cases (75.8% localized and 71.4% advanced) than African-American cases (68.5% localized and 45.7% advanced) completed the interview.

Control subjects were randomly sampled from the 1999 Health Care Financing Administration (HCFA) Medicare beneficiary file. Controls were frequency matched to

cases on age (five-year age groups), race (Caucasian, African-American), and geographical region (western, middle and eastern third of the state). Eligible controls were South Carolina residents, aged 65–79, with no history of prostate cancer. A total of 482 control subjects (63.8%) completed the interview. Of potentially eligible controls, 108 refused (14.3%), 22 died prior to the interview (2.9%), 112 were not located (14.8%), and 32 were too sick to participate (4.2%). Caucasian controls (69.6%) were more likely than African-American controls (52.2%) to complete the interview.

After eliminating 59 subjects (7 cases and 52 controls) who had prevalent prostate cancer and three subjects (2 cases and 1 control) who completed fewer than five questions, the final sample size was 845 subjects (416 cases, 429 controls). Of those, 11 cases and 36 controls completed questions on age, race, education, cancer history, history of benign prostatic hyperplasia, and prostate-specific antigen and digital rectal exam screening only, resulting in 407 cases and 393 controls for most analyses.

Institutional Review Boards of the University of South Carolina, the Centers for Disease Control and Prevention, and the National Cancer Institute approved this project's data collection procedures. Cases and controls were recruited through mailings that described the study and informed the potential participant that an interviewer would call them soon. Since the HCFA file does not contain telephone numbers, controls whose telephone numbers could not be located through directory assistance, telephone or reverse directories were sent an additional letter asking for a preferred contact number.

Interviewing began in June 2000 and was completed in August 2002. Trained interviewers from the University of South Carolina Survey Research Laboratory conducted computer-assisted telephone interviews with subjects who provided consent with the understanding that written consent would be obtained. The questionnaire collected information on demographic characteristics, socioeconomic status, stress, coping, alcohol and tobacco use, physical activity, diet, medical history, family history of cancer, history of sexually transmitted infections, and farm-related work activities and exposures. Most exposures pertained to the period prior to a reference date, the date of diagnosis for cases and an assigned date for controls comparable to the date of diagnosis for the cases. The telephone interview took approximately 30–40 min to complete. We pilot tested the study protocol and questionnaire on 20 cases and 20 controls.

We used unconditional logistic regression to estimate the relative risk of prostate cancer associated with lifestyle factors while controlling for potential confounding factors [14]. Race, age, geographical region, educational level, annual income, marital status, occupation, family history of prostate cancer, body mass index (BMI), prostate cancer screening history, diet, physical activity, and alcohol and tobacco use as categorized in Tables 1–4 were evaluated as confounders of lifestyle factor-prostate cancer relations. Usual occupation, based on the longest paying job since respondents were age 14 years, was aggregated into major groups using the 1998 Standard Occupational Classification [15]. BMI, defined as self-reported weight (kg) before reference date divided by the square of self-reported height (m^2), was categorized using quartile distributions among controls. Diet was assessed in a 20-item food frequency questionnaire and pertained to foods consumed at least once a year.

Although none of the men indicated their diet had changed since the reference date, they were asked to recall their diet for the period immediately prior to the reference date. Foods on the questionnaire that contributed to the animal fat food group were eggs, whole milk, cheese, ice cream, beef, stew, mixed meat dishes, hot dogs, luncheon meats, bacon, other pork, liver, and chicken; to the dairy food group were whole milk, cheese, and ice cream, and to the lycopene food group were raw tomatoes, cooked tomatoes, and watermelon. Servings per week of food groups were categorized into quartiles among controls. Foods chosen for the food frequency questionnaire were based on those utilized in a prostate cancer study conducted in the late 1980's by Hayes *et al.* [16] among Caucasian and African-American men in three metropolitan areas of the US. Their 60-item food frequency questionnaire was developed by analyzing 24-hour recalls of Caucasian and African-American participants in the National Health and Nutrition Examination Survey I. The present study used 13 of the 17 foods used by Hayes *et al.* for animal fat (missing foods were salt pork, gravy, and half and half; chicken was not separated into baked and fried) and three of the five foods used for lycopene (missing foods were tomato sauce or spaghetti sauce, and tomato juice). Engaging in strenuous or moderate leisure-time physical activity for an average of one or more hours a week since age 18 years were categorized as none and as tertiles within the active group. Using a 10% change between unadjusted and adjusted odds ratios as evidence of confounding, analyses were adjusted for age, geographic region and family history of prostate cancer. The combined analysis was also adjusted for race. Interaction terms between race and lifestyle factors were included to examine whether there was evidence of effect measure modification. Linear trend was assessed by treating categorical variables as continuous variables; for BMI, diet and physical activity, scores were assigned to the median value within quartiles.

Results

Table 1 compares demographic factors of cases and controls separately by race. Although there was no statistical evidence of effect measure modification, analyses are presented separately by race since the effect of some factors on prostate cancer was on either side of the null value. Compared to controls prostate cancer cases were more likely to be younger and have a lower household income. Caucasian cases tended to be better educated, while African-American cases tended to be less educated.

The odds ratios (ORs) and 95% confidence intervals (CIs) for prostate cancer associated with demographic and medical factors for all cases and controls, and among Caucasian and African-American men separately are shown in Table 2. The distribution of factors by race differed for occupation (African-Americans were more likely to work in production), BMI (African-Americans were more likely to be in the upper quartile), prostate cancer screening (African-Americans were less likely to have had a PSA test or digital rectal exam), and sexually transmitted infections (African-American men were more likely to have a history of gonorrhea or syphilis). Caucasian men employed in a production occupation had an increased prostate cancer risk (OR=2.04, 95% CI 1.17–3.54), but African-American men who worked in the military were at reduced risk of prostate cancer (OR=0.19, 95% CI 0.05–0.76). More than two-fold elevations in risk were seen for family history of prostate cancer in a first-degree relative for all men (OR=2.34; 95% CI 1.57–3.48), and for Caucasian

(OR=2.29; 95% CI 1.36–3.85) and African–American (OR=2.40; 95% CI 1.28–4.51) men. An increased prostate cancer risk was seen for Caucasian men who had five or more PSA tests (OR=2.14, 95% CI 1.20– 3.83) and African-American men who had one–two PSA tests (OR=2.37, 95% CI 1.14–4.90) and three–four PSA tests (OR=2.10, 95% CI 1.02–4.34) in the past five years.

Risk of prostate cancer associated with diet and physical activity is presented in Table 3. The distribution of factors by race differed for lycopene (African-American were more likely to be in the lowest quartile) and physical activity (African-Americans were less likely to have engaged in strenuous or moderate activity). Consumption of animal fat, dairy or lycopene among all men was not related to prostate cancer risk. Caucasian men in the highest quartile of lycopene consumption had a 45% reduction in prostate cancer risk (95% CI 0.31–0.98), and there was a trend of decreasing prostate cancer risk with increasing lycopene consumption ($p=0.03$). Neither strenuous nor moderate physical activity was associated with prostate cancer risk for all men. Among Caucasian men, the reduced risk of prostate cancer seen for two or fewer hours per week of strenuous physical activity did not hold for more hours of strenuous physical activity.

Table 4 presents prostate cancer risk associated with alcohol and tobacco use. The distribution of factors by race differed for drinking and smoking (African-Americans were less likely to be current drinkers and less likely to be former smokers). There were no significant findings for drinking or smoking and prostate cancer risk in our study.

Discussion

Using broad groups to categorize usual occupation we saw an increased risk for prostate cancer among Caucasian men employed in production, transportation and material moving, and a decreased risk among African-American men who worked in the military. We also saw a non-significant increase in prostate cancer risk for Caucasian men employed in service (OR=2.64, 95% CI 0.82–8.48). Krstev *et al.* [17] reported elevated prostate cancer risks among African-American men, but not Caucasian men, for plant and system operators (OR=4.06) and other laborers (OR=1.37) (which are within our production occupations category), and African- American and Caucasian men employed in service (OR=1.41). They attributed these findings to potential exposure to polycyclic aromatic hydrocarbons. The reduced risk we saw associated with military occupation for African-American men has not been seen elsewhere; [18] however, the numbers of men in this category were quite small (3 cases, 14 controls).

In agreement with several studies, we found more than a two-fold elevation in prostate cancer risk associated with a first-degree (father, brothers, sons) family history of prostate cancer among all men, and among Caucasian and African-American men [5–8]. As was the case with two previous case–control studies, the increase in risk did not differ by race [5, 6]. It should be noted that 70% of cases did not have a family history of prostate cancer; thus, this risk factor affected relatively few men.

We failed to find an association between BMI and prostate cancer as has been the case in the majority of case-control and cohort studies that investigated this relation [19]. Only three of these studies included African-American men, [9, 10, 20] and the two that reported associations for African-American men separately found no association [9, 20]. Although weight and height were based on self-report, it is interesting to note that twice the percentage of African-American controls (35.7%) were in the highest quartile of BMI compared with Caucasian controls (17.7%). This is consistent with the higher prevalence of obesity in African-American men [21].

Our finding of increased risk of prostate cancer among Caucasian men who had five or more PSA tests in the past five years was not unexpected since these men may have undergone work-ups for symptoms related to benign prostate hyperplasia (BPH) [22]. We examined Caucasian cases who had five or more PSA tests by history of BPH; the percentage with a history of BPH (58.9%) did not differ substantially from the percentage without a history of BPH (38.4%), which would argue against detection bias. In contrast, we saw elevated prostate cancer risks among African-American men who had one-four PSA tests in the past five years. A possible explanation for this finding is that African-American men may have been less likely to undergo annual screening due to issues of health care access. When we restricted our analysis to men age 70 years and over who would have been eligible for Medicare for the previous five years, the associations were more pronounced. Thus, in agreement with other studies, African-American men in our study were less likely to be screened regardless of the availability of health insurance [11].

We found no association between a history of sexually transmitted infections and prostate cancer risk. This is in contrast to a meta-analysis that reported significantly elevated pooled odds ratios for gonorrhea and syphilis [23]. Hayes *et al.* [24] reported elevated risks of prostate cancer associated with a history of gonorrhea or syphilis that were similar for African-American and Caucasian men. Although the percentage of Caucasian men in our study reporting a history of STI was comparable to that reported by Hayes *et al.*, the percentage of African-American men was lower indicating there may have been underreporting of this sensitive topic.

Although we saw no relation between consumption of animal fat or dairy and prostate cancer risk, we saw a reduced risk among Caucasian men who consumed the highest quartile of lycopene and a significant trend of decreasing risk with increasing consumption. Findings from case-control and cohort studies on animal fat [25] and lycopene [26] have been inconsistent; however, dairy has been linked to increased prostate cancer risk in the majority of studies on this topic [27]. In the case-control study we used to design our food frequency questionnaire, Hayes *et al.* [16] assessing Caucasian and African-American men separately, reported significant trends and elevations in risk in the highest quartile of animal fat intake among African-Americans, but not Caucasians. After restricting the analysis to advanced cancer, significant trends and elevations were seen for both groups. Hayes *et al.* [16] failed to find an association between consumption of dairy or lycopene and prostate cancer risk. Restriction of our analysis to advanced cancers did not change our results (data not shown).

Among Caucasian men we found a decreased prostate cancer risk associated with two or fewer hours per week of strenuous physical activity that was not seen for men engaging in more hours per week of strenuous physical activity. Evidence for a protective effect of occupational and/or recreational physical activity on prostate cancer risk has been weak [28]. One case-control study reported a borderline significant reduced risk of prostate cancer associated with physical activity among Caucasian men, but not among African-American men [12], while another study found no association [20]. Although we provided examples of different types of strenuous (*e.g.*, moving heavy things, digging, running, playing basketball) and moderate (*e.g.*, brisk walking, fishing, gardening, playing baseball) physical activity, the time period was since age 18 years, which may have introduced misclassification.

We found no association between ever use, amount and duration of alcohol use and prostate cancer risk. This finding agreed with most studies that reported no association [29]. The one study that investigated the relation separately by race reported a positive association between consumption of large quantities of alcohol and prostate cancer risk that was similar for African-American and white men, and was independent of smoking [30].

In agreement with the majority of studies on this topic, [31] we saw no association between current smoking and prostate cancer risk. Two cohort studies have shown an elevated risk of advanced disease associated with smoking [32, 33]. When we investigated the association between smoking and advanced prostate cancer we still saw no effect (data not shown). The two studies investigating race as an effect modifier of the relation between smoking and prostate cancer were split, with the first case-control study showing no association in either race [34], and the second cohort study showing similarly elevated risks for both races [13].

This study was not without limitations. Our response rates were lower than desired, especially among African-Americans, somewhat limiting the generalizability of our results. The refusal rates by race were not different yet the proportion that could not be located was higher among African-American than Caucasian cases and controls. The range of months from time of diagnosis to interview was 1.8–25.2 (median =7.2 months). This may have led to misclassification, especially for the 20% of men who recalled events that occurred more than one year ago. Another source of misclassification was the memory problems common in men age 65 years and over. Although our food frequency questionnaire was based on the one utilized by Hayes et al. [16] that was designed to capture the Caucasian and African-American diets, we used fewer foods which may have led to misclassification and prevented us from performing energy adjustment. It is likely that this misclassification was nondifferential, thereby reducing our ability to identify weak associations.

Our study power was limited for some main effects and due to small numbers, we were unable to assess effect modification by family history or stage of disease within race. Our study is the first population-based case-control study of prostate cancer conducted in a rural Southeastern state that included both Caucasian and African-American men. South Carolina has had one of the highest incidence rates of prostate cancer in recent years [35]. Given that apart from age, family history would appear to be the only established risk factor for prostate cancer, [5–8], studies of genetic factors are the logical next step. It is likely that gene-environment interactions will be important in explaining prostate cancer risk [36].

Acknowledgments

This research was supported by funding to Dr. Maureen Sanderson from the Association of Schools of Public Health/Centers for Disease Control and Prevention and the National Cancer Institute. Dr. Sanderson was partially supported by career development award DAMD-17-00-1-0340 from the US Army Medical Research and Materiel Command.

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Table 1

Comparison of cases and controls for demographic factors by race

	Caucasian		African-American	
	Cases (n = 241) N (%)	Controls (n = 227) N (%)	Cases (n = 166) N (%)	Controls (n = 166) N (%)
Stage ^a				
I/II	175 (71.4)		139 (81.3)	
III/IV	70 (28.6)		32 (18.7)	
Age (years) ^a				
65–69	110 (44.9)	111 (43.0)	82 (48.0)	75 (43.9)
70–74	79 (32.2)	73 (28.3)	55 (32.2)	52 (30.4)
75–79	56 (22.9)	74 (28.7)	34 (19.9)	44 (25.7)
Geographical region ^a				
Eastern	146 (59.6)	155 (60.1)	89 (52.1)	88 (51.5)
Middle	49 (20.0)	46 (17.8)	58 (33.9)	46 (26.9)
Western	50 (20.4)	57 (22.1)	24 (14.0)	37 (21.6)
Educational level ^a				
Elementary education	27 (11.2)	24 (9.3)	79 (46.5)	65 (38.0)
Some high school	23 (9.5)	30 (11.6)	32 (18.8)	39 (22.8)
High school graduate	71 (29.3)	71 (27.5)	30 (17.7)	31 (18.1)
Some college or technical school	39 (16.1)	59 (22.9)	15 (8.8)	18 (10.5)
College graduate	82 (33.9)	74 (28.7)	14 (8.2)	18 (10.5)
Missing	3	0	1	0
Annual income				
<\$20,000	43 (19.6)	38 (19.0)	79 (58.5)	68 (51.9)
\$20,000–\$29,999	49 (22.3)	34 (17.0)	23 (17.0)	24 (18.3)
\$30,000–\$39,999	31 (14.1)	35 (17.5)	11 (8.2)	19 (14.5)
\$40,000–\$49,999	18 (8.2)	27 (13.5)	7 (5.2)	9 (6.9)
\$50,000	79 (35.9)	66 (33.0)	15 (11.1)	11 (8.4)
Missing	21	27	31	35
Marital status				
Single/separated/divorced/widowed	34 (14.2)	36 (15.9)	39 (24.2)	44 (27.3)
Married/living as married	206 (85.8)	191 (84.1)	122 (75.8)	117 (72.7)
Missing	1	0	5	5

^aConsists of 245 Caucasian cases and 258 Caucasian controls, and 171 African-American cases and 171 African-American controls.

Table 2

Odds ratios for prostate cancer associated with demographic and medical factors

	Cases (n = 407)	Controls (n = 393)	Combined OR ^a	95% CI	OR Caucasian ^b	OR African-American ^b
Occupation						
Management, professional and related	96	95	1.00	Referent	1.00	1.00
Service	21	13	1.65	0.76, 3.58	2.64	1.10
Sales and office	45	70	0.67	0.42, 1.08	0.56	1.07
Natural resources, construction and maintenance	74	74	1.06	0.68, 1.65	0.98	1.04
Production, transportation, and material moving	134	96	1.60	1.06, 2.40	2.04 ^c	1.18
Military	24	39	0.60	0.33, 1.08	0.84	0.19 ^c
Missing	13	6				
Family history						
None	278	329	1.00	Referent	1.00	1.00
First-degree	86	43	2.34	1.57, 3.48	2.29 ^c	2.40 ^c
Second-degree	34	17	2.33	1.27, 4.26	4.44 ^c	0.90
Missing	9	4				
Body mass index (quartiles)						
<24.4	90	90	1.00	Referent	1.00	1.00
24.4–27.2	114	101	1.11	0.74, 1.66	1.02	1.27
27.3–29.8	96	96	0.95	0.63, 1.44	0.93	0.98
29.9	96	96	0.92	0.61, 1.41	1.10	0.79
Missing	11	10				
<i>p</i> for trend			0.98		0.67	0.61
Number of prostate specific antigen tests in past 5 years ^d						
0	61	98	1.00	Referent	1.00	1.00
1–2	68	64	1.62	0.99, 2.65	1.22	2.37 ^c
3–4	67	66	1.49	0.91, 2.42	1.15	2.10 ^c
5	157	110	2.16	1.46, 3.33	2.14 ^c	1.79
Unknown	63	90	1.03	0.63, 1.66	0.71	1.37
Missing	0	1				

	Cases (n = 407)	Controls (n = 393)	Combined OR ^a	95% CI	OR Caucasian ^b	OR African-American ^b
<i>p</i> for trend			0.26		0.30	0.55
Number of digital rectal exams in past 5 years ^d						
0	47	58	1.00	Referent	1.00	1.00
1–2	66	93	0.73	0.43, 1.24	0.70	0.75
3–4	80	84	0.96	0.57, 1.62	1.09	0.88
5	205	169	1.27	0.79, 2.06	1.25	1.44
Unknown	18	23	0.96	0.43, 2.13	0.56	1.33
Missing	0	2				
<i>p</i> for trend			0.06		0.18	0.10
History of gonorrhea						
No	358	356	1.00	Referent	1.00	1.00
Yes	43	33	1.27	0.77, 2.08	1.62	1.20
Missing	6	4				
History of syphilis						
No	397	383	1.00	Referent	1.00	1.00
Yes	3	4	0.60	0.13, 2.82	–	0.70
Missing	7	6				

^a Adjusted for race, age, geographic region and family history of prostate cancer.

^b Adjusted for age, geographic region and family history of prostate cancer.

^c $p < 0.05$.

^d Consists of 416 cases and 429 controls.

Table 3

Odds ratios for prostate cancer associated with diet and physical activity

	Cases (n = 407)	Controls (n = 393)	Combined OR ^a	95% CI	OR Caucasian ^b	OR African-American ^b
Consumption of animal fat (quartiles of servings per week)						
12.7	90	82	1.00	Referent	1.00	1.00
12.8–18.95	100	79	1.15	0.75, 1.76	1.53	0.75
18.96–26.7	95	83	1.00	0.65, 1.53	1.11	0.84
26.8	68	78	0.78	0.49, 1.20	0.69	0.82
Missing	54	71				
<i>p</i> for trend			0.24		0.16	0.84
Consumption of dairy (quartiles of servings per week)						
2.2	105	91	1.00	Referent	1.00	1.00
2.3–5.0	102	102	0.84	0.56, 1.24	0.82	0.86
5.1–8.5	75	72	0.83	0.53, 1.28	0.60	1.29
8.6	94	86	0.93	0.61, 1.40	0.84	1.04
Missing	31	42				
<i>p</i> for trend			0.59		0.21	0.57
Consumption of lycopene (quartiles of servings per week)						
2.6	98	86	1.00	Referent	1.00	1.00
2.7–4.5	97	85	0.98	0.64, 1.49	0.88	1.01
4.6–8.0	96	80	1.04	0.68, 1.59	0.98	1.08
8.1	76	84	0.71	0.46, 1.08	0.55 ^c	0.99
Missing	40	44				
<i>p</i> for trend			0.32		0.03	0.36
Strenuous physical activity (tertiles among active in hours per week)						
None	218	189	1.00	Referent	1.00	1.00
2.0	40	56	0.64	0.41, 1.02	0.50 ^c	1.02
2.1–4.0	42	54	0.70	0.44, 1.11	0.87	0.40
4.1	83	60	1.18	0.80, 1.76	0.94	1.87
Missing	24	34				
<i>p</i> for trend			0.60		0.48	0.86

	Cases (n = 407)	Controls (n = 393)	Combined OR ^a	95% CI	OR Caucasian ^b	OR African-American ^b
Moderate physical activity (tertiles among active in hours per week)						
None	85	86	1.00	Referent	1.00	1.00
3.0	70	67	0.95	0.59, 1.51	0.68	1.47
3.1–6.0	109	103	0.97	0.64, 1.49	0.88	0.98
6.1	102	94	1.01	0.66, 1.56	0.88	1.12
Missing	41	43				
<i>p</i> for trend			0.39		0.71	0.37

^a Adjusted for race, age, geographic region and family history of prostate cancer.

^b Adjusted for age, geographic region and family history of prostate cancer.

^c *p* < 0.05.

Table 4

Odds ratios for prostate cancer associated with alcohol and tobacco use

	Cases (n = 407)	Controls (n = 393)	Combined OR ^a	95% CI	OR Caucasian ^b	OR African-American ^b
Alcohol use						
Never	130	110	1.00	Referent	1.00	1.00
Former	154	139	0.96	0.67, 1.36	0.76	1.24
Current	117	138	0.69	0.47, 1.00	0.60	0.86
Missing	6	6				
Drinks per day						
0	130	110	1.00	Referent	1.00	1.00
1–2	77	65	1.01	0.66, 1.55	0.95	1.09
3–4	33	47	0.60	0.35, 1.01	0.64	0.53
5	141	135	0.86	0.60, 1.24	0.66	1.43
Missing	26	36				
<i>p</i> for trend			0.29		0.78	0.15
Drinking duration (years)						
0	130	110	1.00	Referent	1.00	1.00
<25	75	74	0.84	0.55, 1.28	0.84	0.86
25–45	94	87	0.96	0.64, 1.43	0.62	1.84
>45	87	93	0.79	0.53, 1.18	0.69	0.95
Missing	21	29				
<i>p</i> for trend			0.96		0.55	0.35
Smoking status						
Never	106	116	1.00	Referent	1.00	1.00
Former	229	208	1.15	0.83, 1.60	1.20	1.10
Current	67	64	1.13	0.73, 1.75	1.20	1.08
Missing	5	5				
Cigarettes per day						
0	106	116	1.00	Referent	1.00	1.00
<20	146	118	1.32	0.91, 1.90	1.48	1.17
20	78	79	1.03	0.68, 1.57	1.10	0.98

	Cases (n = 407)	Controls (n = 393)	Combined OR ^a	95% CI	OR Caucasian ^b	OR African-American ^b
>20	69	64	1.08	0.70, 1.69	1.10	1.17
Missing	8	16				
<i>p</i> for trend			0.79		0.63	0.71
Smoking duration (years)						
0	106	116	1.00	Referent	1.00	1.00
<25	116	109	1.12	0.77, 1.64	1.29	0.93
25–45	103	93	1.11	0.75, 1.65	1.07	1.25
>45	75	67	1.24	0.81, 1.90	1.33	1.17
Missing	7	8				
<i>p</i> for trend			0.55		0.93	0.29

^a Adjusted for race, age, geographic region and family history of prostate cancer.

^b Adjusted for age, geographic region and family history of prostate cancer.