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ORIGINAL PAPER

Patterns of meat intake and risk of prostate cancer among African-Americans in a large prospective study

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Abstract
Objective Given the large racial differences in prostate cancer risk, further investigation of diet and prostate cancer is warranted among high-risk groups. The purpose of this study was to examine the association between type of meat intake and prostate cancer risk among African-American men.
Methods In the large, prospective NIH-AARP Diet and Health Study, we analyzed baseline (1995–1996) data from African-American participants, aged 50–71 years. Incident prostate cancer cases ($n = 1{,}089$) were identified through 2006. Dietary and risk factor data were ascertained by questionnaires administered at baseline. Cox models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) within intake quantiles.
Results Neither white nor processed meat intake was associated with prostate cancer, regardless of meat-cooking method. Red meats cooked at high temperatures were associated with an increased risk of prostate cancer (HR = 1.18, 95% CI = 1.00–1.38 and HR = 1.22, 95% CI = 1.03–1.44, for the upper two intake tertiles). Intake of the heterocyclic amine (HCA), 2-amino-3,4,8-trimethylimidazo[4,5-*f*] quinoxaline (DiMeIQx) was positively associated with prostate cancer (HR = 1.30; 95% CI = 1.05–1.61, $p = 0.02$). No associations were observed for intake of other HCAs.
Conclusion Red meats cooked at high temperatures were positively associated with prostate cancer risk among African-American men. Further studies are needed to replicate these findings.

Keywords Prostate cancer · Cohort studies · Diet · Meat consumption · Racial disparities · Men

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Introduction

Prostate cancer is the most common incident cancer in men and the second most common cause of cancer death in the United States [1]. Over the past two decades, an increase in incidence has been observed which has been attributed, in part, to an increase in prostate-specific antigen screening. With regard to natural history, prostate cancer varies considerably with indolent disease not becoming life-threatening and aggressive disease leading to high morbidity and death. Established risk factors include race, with the highest rates being among African-Americans, age, and family history.

The reason(s) for the African-American–Caucasian disparity in prostate cancer is not clear. One possible contributory factor may be differences in dietary patterns [2–4]. African-Americans may have different dietary preferences based on cultural influences [5]. For example, African-American men consume higher amounts of fried fatty meats than other men in the south [6]. Given the large racial differences in prostate cancer risk, further

investigation of diet is warranted. To date, few studies have explored this high-risk group with specific regard to meat consumption. Recently published findings suggest that certain types of meat intake (e.g., high red and processed meat consumption) increase the risk of prostate cancer [7]; however, this finding was based on a population mostly comprised of white participants and may therefore not apply to individuals with different lifestyles and ethnic backgrounds. Meat is a potential source of multiple mutagens and carcinogens, including heterocyclic amines (HCAs), polycyclic aromatic hydrocarbons, and *N*-nitroso compounds. The purpose of the present study was to examine the association between meat intake and meat-cooking methods and subsequent risk of total and advanced prostate cancer among African-American men.

Materials and methods

Study population

From 1995 through 1996, men and women between the ages of 50–71 years residing in one of six US states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) or two metropolitan areas (Atlanta, Georgia, and Detroit, Michigan) were recruited to participate in the National Institutes of Health (NIH)-AARP Diet and Health Study, a large cohort study examining the relation between diet and health [8]. The NIH-AARP Diet and Health Study was approved by the Special Studies Institutional Review Board of the US National Cancer Institute (NCI).

Our baseline cohort of 566,401 persons included 9,304 African-American men. We excluded those whose questionnaire was completed by someone else on their behalf ($n = 320$), subjects who reported having end-stage renal disease or previous cancer ($n = 968$), and subjects reporting extreme daily total energy intake defined as more than two inter-quartile ranges above the 75th percentile or below the 25th percentile ($n = 66$). Men who were later diagnosed with in situ cancer were excluded ($n = 1$). After exclusions, our final analytic cohort was 7,949 African-American men.

Study measures

Incident cases

Our case ascertainment method has been previously described [9]. Briefly, cancer cases were identified through linkage with the cancer registry databases of the states of residence with the addition of Arizona and Texas, which were certified by North American Association of Central Cancer Registries as being at least 90% complete within 2 years of cancer occurrence and the National Death Index Plus. For the present study, prostate cancer cases with information on cancer stage and histology were identified during follow-up through 31 December 2006. Advanced cases were defined as those with clinical stages of T3, T4, N1, or M1 according to the American Joint Committee on Cancer 1997 tumor-node-metastasis classification system [10]. Information on Gleason sum was not available.

Demographics and behavioral risk factors

Information on demographic and behavioral factors, including dietary intake, smoking, and physical activity, was ascertained at baseline through a mailed questionnaire sent to members of the cohort in 1995–1996. Physical activity was assessed by asking subjects how often they participated in physical activities at work or home, including exercise, sports, and activities such as carrying heavy loads during a typical month in the prior 12 months: vigorous physical activity was defined as activity $\geq$20 min (that increased breathing or heart rate, or worked up a sweat) for five or more times per week. A validated 124-item food frequency questionnaire was used to assess a participant's usual dietary intake in the prior 12-months as well as to estimate energy and nutrient intakes. Self-reported body weight (lbs) and height (ft-inches) were used to derive body mass index [weight (kg)/height (m^2)]. We categorized dietary intake of meat groups (e.g., red, white, processed) based on definitions previously reported in this cohort [11] for which "red meat" included all types of beef and pork, "white meat" included chicken, turkey, and fish, and "processed meats" included bacon, sausage, luncheon meats, cold cuts, ham, regular hot dogs, and low-fat hot dogs made from poultry. Daily intake of food was energy-adjusted using the nutrient density method [12].

A follow-up questionnaire that assessed cooking methods and doneness of meats was mailed to participants who reported being cancer-free, within 6 months of completing the baseline questionnaire. Levels of HCAs (e.g., 2-amino-3,4,8- trimethylimidazo[4,5-*f*] quinoxaline (DiMeIQx), 2-amino-3,8-dimethylimidazo[4,5-*f*] quinoxaline (MeIQx), and 2-amino-1-methyl-6-phenylimidazo[4,5-*b*] pyridine (PhIP)), and polycyclic aromatic hydrocarbons (e.g., benzo[a]pyrene (B[a]P)) from meats were ascertained by linking data to the NCI CHARRED database (http://charred.cancer.gov). Heme iron intake was estimated using laboratory-measured values from samples of meat cooked using varying methods and degrees of doneness. For processed meats, nitrate and nitrite intake was ascertained by linking data to an NCI database containing nitrate and nitrite values for 10 (90%) types of processed meats consumed in the United States [13].

Statistical methods

Descriptive statistics were calculated for baseline characteristics of participants. Multivariable Cox proportional hazard regression models were used to assess the associations between meat intake and prostate cancer with time since entry into the study as the underlying time metric. Participants were followed from the date the baseline questionnaire was returned to the date of death, moving out of the study area, or the end of 2006, whichever came first. The proportional hazards assumption was assessed by modeling cross-product terms of meat intake and time. The hazard ratio (HR) and 95% confidence interval (CI) were calculated for each variable in the multivariable Cox models. Results for non-advanced disease were not markedly different than those for any prostate cancer, which was not surprising given that non-advanced disease represents 90% of the cases; therefore, results for the latter are reported. All models summed to total meat consumption (e.g., red and white meat were included in the same model). Fully adjusted models included age (continuous), education, marital status, family history of prostate cancer, history of diabetes, body mass index (<18.5, 18.5 to <25, 25 to <30, ≥30 kg/m^2), smoking (never, former ≤20 cigarettes/d, former >20 cigarettes/d, current ≤20 cigarettes/d, current >20 cigarettes/d, missing), self-reported health status (excellent/great, good, fair/poor), alcohol intake (none, 0 to <5, 5 to <15, 15 to <30, ≥30 g/d), and fruits (g/1,000 kcal categorized into quintiles), which altered risk estimates by 10% or more. Although quintiles were used for most variables, tertiles were used for meats by cooking temperatures variables because of a smaller range of intake. Quantiles were based on the distributions among non-cases. Tests for trend were based on quantile-specific median values entered as a continuous term in the regression model. Statistical significance was based on two-sided *p*-values of <0.05. Data were analyzed using SAS® (version 9.2, SAS Institute Inc., Cary, NC).

Results

Subject characteristics were examined by cancer status (Table 1). In the present study, approximately 7,949 participants were African-American men, 1,089 of who developed prostate cancer (including 108 advanced prostate cancers, 22 of which were fatal). Additional information from a follow-up risk factor questionnaire was available for 3,903; 541 of who developed prostate cancer and 47 of these were advanced. In general, those with prostate cancer were more likely to be married and have a family history of prostate cancer, less likely to report having a history of diabetes or perception of poor health.

Overall, there was no association for red, white, or processed meat intake and prostate cancer (Table 2). However, among African-American men, the risk of developing non-advanced prostate cancer was approximately 20% higher for those with higher consumption of red meats cooked at high temperatures (HR = 1.22, 95% CI = 1.03–1.44; *Ptrend* = 0.04) (Table 2). A similar pattern was observed between red meats cooked at high temperatures and risk of developing advanced disease, albeit the observed associations were not statistically significant. There was a suggested protective association between red meats cooked at low temperatures and risk of developing non-advanced disease (*Ptrend* = 0.05).

African-American men who consumed meats with high levels of DiMeIQx (upper tertile) were 30% more likely (HR = 1.30, 95% CI = 1.05–1.61; *Ptrend* = 0.02) to develop prostate cancer compared to African-American men who consumed low levels of DiMeIQx (lowest tertile) (Table 3); this association was not evident for advanced disease. The major contributing meats to DiMeIQx intake in our sub-cohort of African-American men was steak (43.5%) and hamburger (41.1%). When examining meats that contain high amounts of DiMeIQx, we observed that African-American men who consumed steak had an increased risk (HR = 1.36, 95% CI = 1.08–1.72; *Ptrend* = 0.03) (data not shown) when comparing the upper tertile of intake to the lowest; no association was observed for hamburger (*Ptrend* = 0.27). There was no significant association between B[a]P, heme iron, or nitrite/nitrate intake and risk of prostate cancer.

Discussion

This study is the largest prospective study to examine the association of meat and HCA intake on prostate cancer risk in African-American men to date. There was no association found for total red, white, or processed meat consumption; however, those consuming a lot of red meats cooked at high temperatures and DiMeIQx had an increased risk of non-advanced prostate cancers. We found that African-American men in the highest intake quantile of red meat cooked at high temperatures had a 22% higher risk of prostate cancer over a 10-year period than men in the lowest-consumption quantile. There was no increase risk seen for advanced prostate cancers.

The majority of previous prospective studies examining the relation between meat and prostate cancer have been in primarily Caucasian populations, and results have been inconsistent [7, 14–20]. Rodriguez et al. [21] examined meat consumption among black and white men separately in the Cancer Prevention Study II (CPS-II) Nutrition Cohort and found a significant increased risk for prostate

Table 1 Characteristics of African-American men by prostate cancer status in the NIH-AARP Diet and Health Study ($n = 7{,}949$)

Characteristic	Non-cases	Prostate cancer	Advanced prostate cancer
Baseline questionnaire, *n*	6,860	1,089	108
Age, years	61.8 (0.06)	61.8 (0.15)†	61.3 (0.50)
BMI, kg/m^2	28.1 (0.06)	27.9 (0.14)	27.7 (0.44)
Family history of prostate cancer, %	7.8	12.2*	11.1
History of diabetes, %	19.2	14.7*	14.8
College graduate or postgraduate study, %	32.3	34.2	30.6
Currently married, %	70.7	74.8*	77.8
Smoking status, %			
Never	29.1	31.5	25.5
Former ≥1 year	51.7	51.1	53.9
Current or former <1 year	19.2	17.4	20.6
Vigorous physical activity ≥5 times/week, %	17.5	18.0	17.6
Use of ≥1 vitamin supplement/month, %	48.2	49.8	46.3
Self-perceived health, fair/poor %	19.2	14.2*	8.5*
Dietary intake			
Energy, kcal/d	2,080 (12.7)	2,060 (31.9)	2,131 (113.5)
Alcohol, g/d	13.7 (0.48)	15.6 (1.3)	16.3 (4.2)
Cholesterol, mg/d	242 (1.9)	233 (4.7)	258 (16.3)
Protein, g/d	73.1 (0.49)	71.1 (1.2)	73.8 (4.2)
Tomatoes, srv/1,000 kcal	0.25 (0.003)	0.24 (0.01)	0.24 (0.02)
Fruit, srv/1,000 kcal	2.0 (0.02)	2.2 (0.05)†	2.0 (0.13)
Vegetables, srv/1,000 kcal	1.8 (0.01)	1.8 (0.04)	1.9 (0.12)
Saturated fat, g/1,000 kcal	10.2 (0.04)	9.9 (0.09)†	10.1 (0.29)
α-Linolenic acid, g/1,000 kcal	0.71 (0.003)	0.70 (0.01)	0.74 (0.03)
Total meat, g/1,000 kcal	69.0 (0.43)	66.4 (1.0)†	67.7 (3.1)
Red meat, g/1,000 kcal	32.6 (0.26)	30.8 (0.63)†	32.1 (2.0)
Cooked at high temperature	14.4 (0.15)	14.1 (0.37)	14.3 (1.1)
Cooked at low temperature	18.2 (0.17)	16.7 (0.37)†	17.8 (1.2)
White meat, g/1,000 kcal	36.4 (0.34)	35.6 (0.81)	35.6 (2.4)
Processed meat, g/1,000 kcal	12.6 (0.14)	11.8 (0.32)†	13.1 (1.1)
Total calcium, mg/d	782 (4.4)	786 (11.5)	767 (34.5)
Total vitamin E, μg/d	57.6 (1.1)	57.3 (2.7)	48.1 (7.7)
Total iron, mg/d	21.7 (0.12)	21.9 (0.31)	20.4 (0.89)
Total zinc, mg/d	15.5 (0.10)	15.6 (0.25)	14.4 (0.69)
Total selenium, μg/d	97.2 (0.30)	95.8 (0.76)	96.2 (2.2)
Follow-up risk factor questionnaire, *n*	3,361	542	46
DiMeIQx, ng/1,000 kcal	0.85 (0.03)	0.93 (0.08)	0.83 (0.19)
MeIQx, ng/1,000 kcal	13.4 (0.31)	13.8 (0.9)	10.9 (1.8)
PhIP, ng/1,000 kcal	61.6 (1.7)	63.0 (4.7)	59.3 (14.8)
BaP, ng/1,000 kcal	11.2 (0.4)	11.2 (0.9)	12.2 (3.3)
Heme iron, μg/1,000 kcal	168 (2.1)	163 (5.1)	169 (17.4)
Nitrite, μg/1,000 kcal	96.2 (1.7)	91.3 (3.8)	108.9 (15.5)
Nitrate, μg/1,000 kcal	199 (3.0)	199 (7.6)	220 (32.2)

Mean (SE) reported unless otherwise indicated

Total meat consisted of red meat (beef and pork), white meat (poultry and fish), and processed meat

Significance indicated for non-case comparisons

* Significant proportional difference

† Significant mean difference

cancer for men whose consumption of cooked processed meat was in the highest quartile. In that study, the association between HCAs and prostate cancer was not examined. Further, the limited number of prostate cancer cases ($n = 85$) precluded analyses of advanced or metastatic disease among African-American men. In our study, 1,089 African-American men developed prostate cancer, 108 of whom had advanced disease.

Table 2 Associations of meat intake and prostate cancer in African-American men ($n = 7,949$)

	Meat intake quantiles					*Ptrend*
	Q1 (ref)	Q2	Q3	Q4	Q5	
Any prostate cancer ($n = 1,089$)						
Red meat						0.48
Cases (median)	244 (8.42)	225 (19.35)	226 (29.17)	213 (40.32)	181 (60.92)	
HR (95% CI)	1.00	0.99 (0.82–1.19)	1.05 (0.87–1.26)	1.01 (0.83–1.24)	0.92 (075–1.14)	
White meat						0.75
Cases (median)	246 (10.84)	194 (20.13)	218 (29.83)	214 (42.19)	217 (69.17)	
HR (95% CI)	1.00	0.81 (0.67–0.98)	0.93 (0.77–1.12)	0.92 (0.76–1.11)	0.96 (0.79–1.16)	
Processed meat						0.76
Cases (median)	244 (2.10)	223 (5.68)	205 (9.61)	226 (14.96)	191 (26.48)	
HR (95% CI)	1.00	0.97 (0.80–1.16)	0.95 (0.78–1.15)	1.07 (0.88–1.30)	0.94 (0.76–1.14)	
Red meat cooked at high temp						0.04
Cases (median)	365 (3.49)	373 (11.40)	351 (24.74)			
HR (95% CI)	1.00	1.18 (1.00–1.38)	1.22 (1.03–1.44)	–	–	
Red meat cooked at low temp						0.05
Cases (median)	405 (6.63)	368 (15.36)	316 (29.06)			
HR (95% CI)	1.00	0.91 (0.78–1.06)	0.84 (0.71–0.99)	–	–	
Advanced prostate cancer ($n = 108$)						
Red meat						0.62
Cases (median)	19 (8.42)	25 (19.35)	22 (29.17)	21 (40.32)	21 (60.92)	
HR (95% CI)	1.00	1.46 (0.80–2.69)	1.32 (0.70–2.50)	1.28 (0.66–2.49)	1.34 (0.68–2.65)	
White meat						0.72
Cases (median)	26 (10.84)	16 (20.13)	22 (29.83)	22 (42.19)	22 (69.17)	
HR (95% CI)	1.00	0.61 (0.32–1.14)	0.88 (0.49–1.57)	0.89 (0.49–1.60)	0.93 (0.52–1.69)	
Processed meat						0.95
Cases (median)	19 (2.10)	25 (5.68)	20 (9.61)	27 (14.96)	17 (26.48)	
HR (95% CI)	1.00	1.37 (0.74–2.53)	1.16 (0.60–2.22)	1.61 (0.86–3.01)	1.04 (0.52–2.08)	
Red meat cooked at high temp						0.20
Cases (median)	34 (3.49)	35 (11.40)	39 (24.74)			
HR (95% CI)	1.00	1.23 (0.74–2.06)	1.44 (0.83–2.47)	–	–	
Red meat cooked at low temp						0.47
Cases (median)	40 (6.63)	33 (15.36)	35 (29.06)			
HR (95% CI)	1.00	0.79 (0.48–1.30)	1.03 (0.50–1.43)	–	–	

Number of cases (median intake for each quantile) and HRs (95% CIs) reported. *p*-values for trend test

Tertiles were used for meats by cooking temperatures variables because of a smaller range of intake

Models adjusted for age, education, marital status, family history of prostate cancer, history of diabetes, smoking, health status, BMI, alcohol, fruit intakes

We have previously shown in the full, predominantly Caucasian NIH-AARP Study that high consumption of red or processed meat is associated with increased risk of total and advanced prostate cancer [7]. Our mean meat intake values were within the range of those reported for the full study. In the present study, however, we did not observe an increased risk with total red or processed meats or with heme iron, nitrite/nitrate, or B[a]P. Instead, in the present sub-cohort of African-American men, we observed associations with red meat cooked at high temperatures and subsequent risk of prostate cancer, which was supported by the finding that risk was also increased for men with higher intakes of DiMeIQx. We did not observe significant associations for advanced disease, which may be due in part to the small number of cases ($n = 108$) in our sub-cohort of African-American men.

Multiple mechanisms have been proposed to explain the association between increased meat intake and subsequent cancer risk. One proposed mechanism is that the contents of red meat are involved in the development of carcinogens

Table 3 Associations of HCAs, B[a]P, heme iron, nitrite, and nitrate and prostate cancer in African-American men ($n = 3{,}903$)

	Intake tertiles			*Ptrend*
	Q1 (ref)	Q2	Q3	
Any prostate cancer ($n = 541$)				
DiMeIQx				0.02
Cases (median)	165 (0.04)	180 (0.32)	196 (1.47)	
HR (95% CI)	1.00	1.15 (0.93–1.42)	1.30 (1.05–1.61)	
MeIQx				0.22
Cases (median)	189 (1.67)	168 (7.63)	184 (22.99)	
HR (95% CI)	1.00	0.97 (0.79–1.20)	1.12 (0.90–1.38)	
PhIP				0.50
Cases (median)	194 (6.20)	168 (30.76)	179 (99.27)	
HR (95% CI)	1.00	0.87 (0.71–1.08)	1.03 (0.84–1.26)	
B[a]P				0.66
Cases (median)	192 (0.47)	179 (3.01)	170 (19.89)	
HR (95% CI)	1.00	0.95 (0.77–1.16)	0.94 (0.76–1.16)	
Heme iron				0.45
Cases (median)	192 (65.95)	178 (140.31)	171 (263.55)	
HR (95% CI)	1.00	0.95 (0.77–1.18)	1.07 (0.86–1.34)	
Nitrite				0.77
Cases (median)	198 (23.67)	171 (66.19)	172 (159.76)	
HR (95% CI)	1.00	0.91 (0.74–1.13)	1.01 (0.82–1.25)	
Nitrate				0.37
Cases (median)	175 (51.05)	198 (155.89)	168 (336.49)	
HR (95% CI)	1.00	1.18 (0.95–1.45)	1.13 (0.90–1.41)	
Advanced prostate cancer ($n = 47$)				
DiMeIQx				0.66
Cases (median)	18 (0.04)	16 (0.32)	13 (1.47)	
HR (95% CI)	1.00	0.91 (0.45–1.83)	0.84 (0.41–1.74)	
MeIQx				0.52
Cases (median)	22 (1.67)	11 (7.63)	14 (22.99)	
HR (95% CI)	1.00	0.60 (0.29–1.24)	0.72 (0.35–1.47)	
PhIP				0.93
Cases (median)	17 (6.20)	17 (30.76)	13 (99.27)	
HR (95% CI)	1.00	1.18 (0.59–2.39)	1.02 (0.48–2.14)	
B[a]P				0.23
Cases (median)	18 (0.47)	18 (3.01)	11 (19.89)	
HR (95% CI)	1.00	1.03 (0.53–2.01)	0.66 (0.31–1.43)	
Heme iron				0.69
Cases (median)	19 (65.95)	13 (140.31)	15 (263.55)	
HR (95% CI)	1.00	0.83 (0.39–1.75)	1.12 (0.53–2.35)	
Nitrite				0.34
Cases (median)	18 (23.67)	12 (66.18)	17 (159.76)	
HR (95% CI)	1.00	0.83 (0.38–1.80)	1.31 (0.64–2.65)	
Nitrate				0.25
Cases (median)	18 (51.05)	12 (155.89)	17 (336.49)	
HR (95% CI)	1.00	0.78 (0.34–1.65)	1.40 (0.68–2.87)	

Number of cases (median value for each tertile) and HRs (95% CIs) reported. *p*-values for trend test

Models adjusted for age, education, marital status, family history of prostate cancer, history of diabetes, smoking, health status, BMI, alcohol, fruit intakes

that may increase the risk of disease, such as heme iron, which may cause oxidative biochemical and cellular damage [22], as well as increase endogenous formation of *N*-nitroso compounds [23]. In addition, meats cooked at high temperatures (e.g., barbecuing, grilling, and frying) form HCAs, which are genotoxic and carcinogenic

compounds thought to increase cancer risk [24–28]. The carcinogenicity of HCAs has been demonstrated in experimental studies [29]. Also, PhIP, which has estrogenic activity, has been shown to induce cancer specifically in the prostate of rats [30]. Although the exact biological effect of these compounds remains unclear, DiMeIQx and MeIQx are thought to be more potent mutagens than PhIP [31]. In the present study, we observed an association between increased DiMeIQx, but not PhIP, and risk of prostate cancer.

Among the inherent strengths of the present study is the prospective design in which diet and other health risk factors were measured prior to development of disease. Extensive data collection of information on lifestyle and medical history allowed us to control for possible confounding on a wide set of characteristics and lifestyle factors. Further, the large size of the NIH-AARP Diet and Health Study allowed us to examine potential associations among a high-risk population.

A limitation of our study is that the cohort consisted of predominantly older, upper-to-middle class participants; therefore, results may not apply to other African-American populations. The FFQ used here was not specifically developed for African-Americans and therefore may not capture unique dietary patterns not reflected in the general US population diet. The lack of specific ethnic/minority foods may have led to misclassification of dietary intakes for this analytic cohort, resulting in a bias of observed associations. The FFQ was assessed at study baseline and did not assess early life exposure; therefore we were unable to examine changes in diet. Our findings may reflect incomplete adjustment for other health risk factors not available in our study cohort, although the NIH-AARP Diet and Health Study did collect a wide range of characteristics and lifestyle factors, which we adjusted for in our analyses that are typically not available in other study populations. However, NIH-AARP Study did not assess PSA screening on the baseline questionnaire; therefore, we were unable to adjust the observed associations between meat intake and prostate cancer for screening. The number of advanced prostate cancer cases was small ($n = 108$), which may in part explain why we did not observe significant associations for advanced disease.

For African-American men, reducing and/or avoiding eating red meats cooked at high temperature may reduce one's risk of developing prostate cancer. If confirmed, our results suggest that African-Americans may be able to decrease their risk of prostate cancer by dietary modification. Further studies are needed to replicate these associations.

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Conflict of interest None.

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