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REVIEW ARTICLE

A review of African American-white differences in risk factors for cancer: prostate cancer

Irina Mordukhovich · Paul L. Reiter · Danielle M. Backes · Leila Family · Lauren E. McCullough · Katie M. O'Brien · Hilda Razzaghi · Andrew F. Olshan

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Abstract
Objective African American men have higher prostate cancer incidence rates than White men, for reasons not completely understood. This review summarizes the existing literature of race-specific associations between risk factors and prostate cancer in order to examine whether associations differ.
Methods We reviewed epidemiologic studies published between January 1970 and December 2008 that reported race-specific effect estimates. We focused mainly on modifiable risk factors related to lifestyle and health. A total of 37 articles from 21 study populations met our inclusion criteria.
Results We found no evidence of racial differences in associations between prostate cancer and alcohol intake, tobacco use, and family history of prostate cancer. Research suggests that a modest positive association may exist between height and prostate cancer risk (all prostate cancer and advanced prostate cancer) among Whites only. No clear patterns were observed for associations with physical activity, weight/body mass index, dietary factors, occupational history, sexual behavior, sexually transmissible infections, and other health conditions.
Discussion Our results suggest few differences in prostate cancer risk factors exist between racial groups and underscore areas where additional research is needed. Future studies should enroll higher numbers of African American participants and report results for advanced prostate cancer.

Keywords Prostate cancer · Epidemiology · Review · Race · African American

Introduction

Prostate cancer is the most common malignancy affecting men in the United States (US) [1]. Following cancer of the lung and bronchus, it is the second leading cause of cancer-related death among men in the US [1]. African Americans have higher prostate cancer incidence rates than Whites. According to data from the Surveillance, Epidemiology, and End Results (SEER) program, age-adjusted prostate cancer incidence rates from 2002 to 2006 for White and African American men were 153.0 and 239.8 per 100,000 persons, respectively [2]. Incidence rates have decreased over the last few years among both White and African American men, but the large disparity between the two

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I. Mordukhovich · D. M. Backes · L. Family · L. E. McCullough · K. M. O'Brien · H. Razzaghi · A. F. Olshan (✉)
Department of Epidemiology, Gillings School of Global Public Health, University of North Carolina, CB #7435, McGavran-Greenberg Hall, Chapel Hill, NCUSA
e-mail: andy_olshan@unc.edu

P. L. Reiter · A. F. Olshan
Lineberger Comprehensive Cancer Center, University of North Carolina, Chapel Hill, NC, USA

P. L. Reiter
Department of Health Behavior and Health Education, Gillings School of Global Public Health, University of North Carolina, Chapel Hill, NC, USA

racial groups remains prominent [2]. Furthermore, a higher percentage of prostate cancers are diagnosed at advanced stages among African American men (6% at distant stages) compared to Whites (4% at distant stages) [3].

In addition to race/ethnicity, age and family history of prostate cancer are the only established risk factors for prostate cancer [4, 5]. Specifically, 63% of diagnosed prostate cancer cases occur in men aged 65 years and older [4], and men with an affected father or brother are approximately twice as likely to develop the disease themselves [6]. A wide range of additional factors have been examined as potential risk factors for prostate cancer [7, 8], but none have produced definitive evidence of an association.

Given the wide disparity that exists between White and African American men in terms of prostate cancer incidence, it is of great interest to examine how associations between possible risk factors and prostate cancer risk may differ by racial group. To our knowledge, a comprehensive review describing these potential differences does not exist. The aim of this report was to summarize the existing literature addressing race-stratified associations between risk factors and prostate cancer, focusing primarily on modifiable lifestyle and health-related factors. We examined these factors' associations with all prostate cancer, as well as advanced prostate cancer occurrence. Results will be useful for identifying potential racial differences in prostate cancer risk factors and underscoring areas in which future research is needed.

Materials and methods

We conducted a literature review using PubMed, identifying references published between January 1970 and December 2008 that reported race-specific effect estimates for potential prostate cancer risk factors. The literature search centered around the keywords of "prostate cancer" and "race" in PubMed.

Two study authors (IM and PR) independently reviewed the titles, abstracts, and articles (if appropriate) of studies identified in the search to determine if each met the following inclusion criteria: (1) report written in English; (2) study conducted in the US; and (3) study reported race-specific effect estimates (articles providing only *p*-values were excluded). We also identified and reviewed articles referenced in these studies for possible inclusion. Studies conducted among only African Americans were included, provided they met all other inclusion criteria. We excluded studies that did not include African Americans or investigated only genetic or demographic risk factors, as we chose to focus primarily on more modifiable risk factors related to lifestyle and health. We also excluded review articles and unpublished studies and abstracts.

A total of 37 articles from 21 different study populations met the inclusion criteria for this review (Table 1). The majority of articles ($n = 35$, 95%) included both African American and White men, while two reports (5%) included only African Americans. We abstracted the first author, study characteristics, race-specific sample sizes, and race-specific effect estimates for each study. Effect estimates and corresponding confidence intervals (if reported) abstracted from the reviewed articles for all prostate cancer cases are displayed in ESM Table S1, while Table 2 contains these data for advanced prostate cancers only. The definition of advanced prostate cancer differed somewhat between studies but was based on cancer stage (*i.e.*, regional/distant cases) and/or grade (*i.e.*, cells were poorly differentiated) in all instances. For the sake of simplicity, we refer to all as advanced prostate cancer cases in this review. Both tables are categorized by potential risk factor. If confidence intervals were not reported, we report *p*-values if provided or just effect estimates in cases where neither confidence intervals nor *p*-values were reported. We report covariate-adjusted effect estimates if they were provided in the original manuscript, and report unadjusted estimates otherwise.

Results

All prostate cancer cases

Anthropometric measures

Eight studies examined associations between anthropometric variables and prostate cancer and reported race-specific effect estimates [9–16]. Height was addressed in two of these studies, a population-based case–control study and a large prospective cohort study. Neither showed an association between height and prostate cancer risk among African Americans. However, both studies reported a modest increased risk among the tallest Whites, with effect estimates ranging from 1.2 to 1.7 [10, 11].

All eight studies addressed weight, body mass index (BMI), or obesity. Four of these, including a large prospective cohort study and a case–control study nested in a prospective cohort, reported no association with body mass or weight among either racial group [11, 12, 14, 16]. Two case–control studies, one hospital-based and one population-based, did report elevated risk of prostate cancer among Whites, but not African Americans, with either higher current body mass or higher body mass at their maximum weight [9, 10]. However, no clear dose–response trend was apparent in the study focusing on current body mass [9]. Clinically diagnosed obesity (ICD8 = 277; ICD9 = 278.0) was examined in a very large cohort with

Table 1 Characteristics of studies meeting inclusion criteria for review ($n = 37$)

Study	Study description	Study type	Sample size	
			African American	White
Beebe-Dimmer [37]	Michigan, US 1996–2002	Case–control	121 cases, 179 community-based controls	N/A
Beebe-Dimmer [15]	Michigan, US 1996–2002	Case–control	139 cases, 359 community-based controls	N/A
Clarke [20]	US, NHANES I baseline 1971–1975	Cohort	47 cases, 708 non-cases from cohort	154 cases, 4468 non-cases from cohort
Coker [22]	South Carolina, US 2000–2002	Case–control	166 cases, 166 population-based controls	241 cases, 227 population-based controls
Cunningham [36]	Texas, US 1993–1996	Historical cohort	126 probands	165 probands
Darbinian [23]	California, US 1964–2003	Cohort	598 cases, 5632 non-cases from cohort	2054 cases, 35068 non-cases from cohort
Friedman [24]	California, US 1964–1994	Cohort	424 cases, 7111 non-cases from cohort	1748 cases, 44397 non-cases from cohort
Govindarajan [26]	10 VA Hospitals, US 1997–2003	Cohort	14199 cohort members, race-specific number of cases not stated (3246 total cases)	50937 cohort members, race-specific number of cases not stated (3246 total cases)
Habel [11]	California, US 1964–1996	Cohort	383 cases, 8313 non-cases from cohort	1579 cases, 54282 non-cases from cohort
Habel [25]	California, US 1964–1996	Cohort	521 cases, 11,775 non-cases from cohort	1905 cases, 67981 non-cases from cohort
Hawk[27]	US, NHANES I 1971–1992	Cohort	214 cases, 4207 non-cases from cohort (race-specific numbers not stated)	214 cases, 4207 non-cases from cohort (race-specific numbers not stated)
Hayes [40]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	471 cases, 589 population-based controls	494 cases, 703 population-based controls
Hayes [18]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	479 cases, 594 population-based controls	502 cases, 721 population-based controls
Hayes [34]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	479 cases, 594 population-based controls	502 cases, 721 population-based controls
Hayes [19]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	479 cases, 594 population-based controls	502 cases, 721 population-based controls
Hayes [10]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	449 cases, 543 population-based controls	483 cases, 658 population-based controls
Hayes [43]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	479 cases, 594 population-based controls	502 cases, 721 population-based controls
Huang [45]	Multi-center study, US 1993–2001	Case–control	103 cases, 368 controls in nested case–control study	765 cases, 915 controls in nested case–control study
Jenkins [44]	Two case–control studies; one in US and Italy (1991–1994) and one in US (1986–1989)	Case–control	Study #1: 41 cases and 98 controls with benign prostatic hyperplasia study #2: 95 cases and 75 population-based controls	Study #1: 10 cases and 34 controls with benign prostatic hyperplasia study #2: 104 cases and 80 population-based controls

Table 1 continued

Study	Study description	Study type	Sample size	
			African American	White
John [41]	California, Hawaii, US and Canada 1989–1991	Case–control	1642 cases, 1636 population-based controls (race-specific numbers not stated)	1642 cases, 1636 population-based controls (race-specific numbers not stated)
Kolonel [29]	California, Hawaii, US and Canada 1989–1991	Case–control	505 cases, 519 population-based controls	510 cases, 501 population-based controls
Krstev [38]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	479 cases, 594 population-based controls	502 cases, 721 population-based controls
Meyer [39]	South Carolina, US 2000–2002	Case–control	166 cases, 167 population-based controls	239 cases, 225 population-based controls
Park [33]	California and Hawaii, US 1993–2002	Cohort	1186 cases, 9520 non-cases in cohort	924 cases, 20166 non-cases in cohort
Patel [28]	Michigan, US 1996–1998	Case–control	353 cases, 257 population-based controls	347 cases, 347 population-based controls
Rodriguez [32]	US 1992–2001	Cohort	85 cases, 608 non-cases in cohort	5028 cases, 59869 non-cases from cohort
Rosenberg [21]	New York, US 1984–1986	Case–control	78 cases, 26 hospital-based controls	216 cases, 137 hospital-based controls
Ross [17]	Two case–control studies (one among each racial group); California, US 1977–1980	Case–control	142 cases, 142 population-based controls	142 cases, 142 population-based controls
Samanic [13]	US veterans 1969–1996	Cohort	16087 cases, 816127 non-cases from cohort	49107 cases, 3619379 non-cases from cohort
Sanderson [12]	South Carolina, US 2000–2002	Case–control	166 cases, 166 population-based controls	241 cases, 227 population-based controls
Tsai [14]	California, US 1972–2000	Case–control	119 cases, 238 controls in nested case–control study	206 cases, 412 controls in nested case–control study
Vogt [30]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	99 cases, 108 population-based controls	110 cases, 120 population-based controls
Vogt [31]	Georgia, Michigan, New Jersey, US 1986–1989	Case–control	101 cases, 112 population-based controls	111 cases, 121 population-based controls
Whittemore [16]	California, Hawaii, US and Canada 1989–1991	Case–control	531 cases, 540 population-based controls	515 cases, 504 population-based controls
Whittemore [35]	California, Hawaii, US and Canada 1989–1991	Case–control	472 cases, 512 population-based controls	493 cases, 487 population-based controls
Wideroff [42]	US 1985–1991	Case–control	37 cases, 17 controls with benign prostatic hyperplasia	25 cases, 25 controls with benign prostatic hyperplasia
Yu [9]	Several US Hospitals 1969–1984	Case–control	161 cases, 320 hospital-based controls	989 cases, 2791 hospital-based controls

N/A not applicable, *US* United States, *NHANES* National Health and Nutrition Examination Survey, *VA* Veterans Affairs

Table 2 Race-specific effect estimates for potential risk factors and advanced prostate cancer

Study	Exposure	Results	
		African American	White
Adult height			
Hayes, 1999	Height (meters, category medians displayed)		
	1.67	1.0 (ref.)	1.0 (ref.)
	1.75	1.0 (0.6, 1.6)	2.2 (1.2, 4.2)
	1.80	1.1 (0.7, 1.8)	2.2 (1.2, 4.2)
	1.85	0.8 (0.5, 1.4)	2.1 (1.1, 3.9)
	p (trend)	0.66	0.03
Adult weight, BMI, obesity			
Hayes, 1999	BMI at 25 years of age (category medians displayed)		
	19.7	1.0 (ref.)	1.0 (ref.)
	21.8	1.1 (0.7, 2.0)	0.9 (0.5, 1.6)
	23.6	0.8 (0.5, 1.5)	0.8 (0.4, 1.3)
	26.5	1.8 (1.0, 3.0)	1.2 (0.7, 2.0)
	p (trend)	0.03	0.49
	BMI at usual adult weight (category medians displayed)		
	21.9	1.0 (ref.)	1.0 (ref.)
	24.3	0.8 (0.5, 1.2)	1.0 (0.5, 1.7)
	25.8	0.6 (0.3, 1.0)	1.3 (0.7, 2.2)
	28.9	0.9 (0.5, 1.4)	1.3 (0.8, 2.3)
	p (trend)	0.58	0.20
	BMI at maximum weight (category medians displayed)		
	23.9	1.0 (ref.)	1.0 (ref.)
	26.5	0.7 (0.5, 1.3)	1.2 (0.7, 2.1)
	29.1	0.7 (0.4, 1.2)	1.6 (1.0, 2.8)
	32.8	0.8 (0.5, 1.2)	1.5 (0.9, 2.7)
	p (trend)	0.30	0.09
Perceived childhood anthropometric measurements			
Hayes, 1999	Perceived childhood height		
	Short	1.0 (ref.)	1.0 (ref.)
	Somewhat short	1.1 (0.5, 2.4)	1.0 (0.4, 2.6)
	Average height	0.9 (0.6, 1.6)	1.6 (0.9, 2.7)
	Somewhat Tall	1.1 (0.5, 2.6)	2.4 (1.1, 5.4)
	Tall	1.0 (0.5, 2.0)	1.8 (0.8, 4.0)
	p (trend)	0.97	0.04
	Perceived childhood weight		
	Thin	1.0 (ref.)	1.0 (ref.)
	Somewhat thin	1.0 (0.5, 1.8)	1.6 (0.9, 2.9)
	Average weight	1.1 (0.7, 1.7)	1.6 (1.0, 2.6)
	Somewhat heavy	1.3 (0.7, 2.4)	1.1 (0.5, 2.6)
	Heavy	0.9 (0.3, 2.7)	4.0 (1.5, 10.5)
	p (trend)	0.56	0.02
Alcohol use			
Hayes, 1996	Alcohol use		
	None	1.0 (ref.)	1.0 (ref.)
	≥57 drinks per week	2.0 (1.1, 3.6)	2.1 (1.1, 3.8)

Table 2 continued

Study	Exposure	Results	
		African American	White
Physical activity			
Hayes, 1999	Occupational physical activity		
	Sedentary	1.0 (ref.)	1.0 (ref.)
	Moderate	0.9 (0.6, 1.4)	1.1 (0.7, 1.7)
	Active	1.4 (0.9, 2.3)	1.8 (1.0, 3.3)
Diabetes			
Rosenberg, 2002	Diabetes		
	No	1.0 (ref.)	1.0 (ref.)
	Yes	2.74 (0.55, 13.5)	0.27 (0.11, 0.71)
Darbinian, 2008*	Glucose tolerance (mg/dL)		
	<140	1.0 (ref.)	1.0 (ref.)
	140–159	0.85 (0.52, 1.39)	0.83 (0.58, 1.17)
	160–199	0.95 (0.64, 1.43)	1.09 (0.84, 1.42)
	≥200	1.07 (0.70, 1.64)	1.02 (0.77, 1.33)
	Diabetes	0.79 (0.36, 1.77)	0.63 (0.34, 1.20)
	p (trend)	0.97	0.96
Fruits and vegetables			
Hayes, 1999	Fruits		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.5	0.9
	Quartile 3	1.4	0.7
	Quartile 4	1.6	0.6
	p (trend)	0.12	0.06
	Vegetables		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.3	0.8
	Quartile 3	1.4	0.9
	Quartile 4	1.4	0.9
	p (trend)	0.24	0.79
	Raw tomatoes		
	0 servings	1.0 (ref.)	1.0 (ref.)
	1–3 servings per month	0.5	0.5
	1 serving per week	0.7	1.2
	2–4 servings per week	0.8	0.8
	5 + servings per week	0.5 ($p < 0.05$)	0.5
	p (trend)	0.19	0.13
	Cooked tomatoes and tomato sauces		
	0 servings	1.0 (ref.)	1.0 (ref.)
	1–3 servings per month	1.7	1.7
	1 serving per week	1.5	1.2
	2–4 servings per week	1.8	1.4
	5 + servings per week	1.9	0.7
	p (trend)	0.57	0.32
	Tomato juice		
	0 servings	1.0 (ref.)	1.0 (ref.)
	1–3 servings per month	0.8	1.1
	1 serving per week	0.9	1.1

Table 2 continued

Study	Exposure	Results	
		African American	White
	2–4 servings per week	0.9	1.3
	5 + servings per week	0.2	2.8 ($p < 0.05$)
	p (trend)	0.07	0.02
	Watermelon		
	0 servings	1.0 (ref.)	1.0 (ref.)
	1–3 servings per month	0.8	0.6 ($p < 0.05$)
	1 Serving Per Week	0.9	0.4
	2–4 Servings Per Week	1.0	0.6
	p (trend)	0.89	0.29
Meat and poultry			
Hayes, 1999	Meat		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.6	1.4
	Quartile 3	2.1 ($p < 0.05$)	1.0
	Quartile 4	2.4 ($p < 0.05$)	1.4
	p (trend)	0.002	0.56
	Red meat		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.7	1.7
	Quartile 3	1.8 ($p < 0.05$)	1.6
	Quartile 4	2.5 ($p < 0.05$)	1.5
	p (trend)	0.0008	0.34
	Poultry and fish		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	0.8	1.0
	Quartile 3	1.2	0.9
	Quartile 4	1.1	0.8
	p (trend)	0.29	0.33
Rodriguez, 2006*	Total processed plus unprocessed red meat (grams per week)		
	0– <246	–	1.0 (ref.)
	246– <408	–	0.9 (0.6, 1.4)
	408– <657	–	1.1 (0.7, 1.6)
	≥657	–	0.8 (0.5, 1.3)
	p (trend)	–	0.53
	Unprocessed red meat (grams per week)		
	0– <137	–	1.0 (ref.)
	137– <244	–	0.7 (0.5, 1.2)
	244– <423	–	0.9 (0.6, 1.4)
	≥423	–	0.8 (0.5, 1.2)
	p (trend)	–	0.39
	Processed meat (grams per week)		
	0– <59	–	1.0 (ref.)
	59– <129	–	1.2 (0.9, 1.8)
	129– <247	–	1.2 (0.8, 1.7)
	≥247	–	1.1 (0.7, 1.7)
	p (trend)	–	0.87

Table 2 continued

Study	Exposure	Results	
		African American	White
	Cooked processed meat (grams per week)		
	0– <38	–	1.0 (ref.)
	38– <87	–	1.3 (0.9, 1.8)
	87– <165	–	1.3 (0.9, 1.8)
	≥165	–	1.2 (0.7, 2.1)
	p (trend)	–	0.33
	Lunchmeat (grams per week)		
	None	–	1.0 (ref.)
	1– <33	–	0.9 (0.6, 1.4)
	33– <56	–	1.1 (0.8, 1.7)
	≥56	–	1.0 (0.7, 1.5)
	p (trend)	–	0.83
	Poultry (grams per week)		
	0– <91	–	1.0 (ref.)
	91– <164	–	0.9 (0.6, 1.3)
	164– <279	–	0.9 (0.6, 1.2)
	≥279	–	0.7 (0.4, 1.1)
	p (trend)	–	0.10
Fat intake			
Whittemore, 1995a	Dietary fat intake (g/day)		
	Quintile 1 (Median = 40.4)	1.0 (ref.)	1.0 (ref.)
	Quintile 2 (Median = 56.6)	0.53 (0.23, 1.2)	0.67 (0.31, 1.5)
	Quintile 3 (Median = 73.6)	0.62 (0.29, 1.3)	1.6 (0.77, 3.3)
	Quintile 4 (Median = 95.9)	0.68 (0.34, 1.4)	1.3 (0.64, 2.7)
	Quintile 5 (Median = 140.1)	1.1 (0.57, 2.2)	1.8 (0.89, 3.7)
	p (trend)	0.42	0.36
	Saturated fat intake (g/day)		
	Quintile 1 (Median = 11.2)	1.0 (ref.)	1.0 (ref.)
	Quintile 2 (Median = 16.7)	0.51 (0.21, 1.2)	0.91 (0.40, 2.1)
	Quintile 3 (Median = 22.8)	0.57 (0.23, 1.4)	0.94 (0.41, 2.2)
	Quintile 4 (Median = 30.1)	0.82 (0.34, 2.0)	2.0 (0.80, 4.9)
	Quintile 5 (Median = 45.5)	1.4 (0.48, 4.2)	2.4 (0.72, 7.7)
	p (trend)	0.08	0.17
Hayes, 1999	Foods high in animal fat (times/week)		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.6	1.6
	Quartile 3	4.0 ($p < 0.05$)	2.2 ($p < 0.05$)
	Quartile 4	2.4 ($p < 0.05$)	2.1 ($p < 0.05$)
	p (trend)	0.004	0.01
	Fat (g/day)		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.9 ($p < 0.05$)	0.9
	Quartile 3	2.3 ($p < 0.05$)	1.5
	Quartile 4	2.4 ($p < 0.05$)	1.6
	p (trend)	0.002	0.04
	Animal fat		
	Quartile 1 (≤33)	1.0 (ref.)	1.0 (ref.)

Table 2 continued

Study	Exposure	Results	
		African American	White
	Quartile 2 [34–46]	2.2($p < 0.05$)	2.1 ($p < 0.05$)
	Quartile 3 [47–61]	4.3 ($p < 0.05$)	2.4 ($p < 0.05$)
	Quartile 4 (≥62)	3.3 ($p < 0.05$)	2.1 ($p < 0.05$)
	p (trend)	0.0001	0.02
	Other Fat (g/day)		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.3	1.1
	Quartile 3	1.5	1.5
	Quartile 4	1.3	1.7
	p (trend)	0.18	0.07
Other nutrients, vitamins, etc.			
Hayes, 1999	Protein (g/day)		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	2.1 ($p < 0.05$)	0.7
	Quartile 3	2.9 ($p < 0.05$)	1.1
	Quartile 4	2.6 ($p < 0.05$)	1.0
	p (trend)	0.0006	0.34
	Carbohydrates (g/day)		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	0.8	1.3
	Quartile 3	1.4	1.3
	Quartile 4	1.7 ($p < 0.05$)	1.3
	p (trend)	0.002	0.71
	Vitamin A: animal sources		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.4	1.1
	Quartile 3	1.5	1.0
	Quartile 4	1.7	0.9
	p (trend)	0.25	0.62
	Vitamin A: fruit and vegetable sources		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.1	1.3
	Quartile 3	1.4	0.8
	Quartile 4	1.5	0.7
	p (trend)	0.23	0.08
	Calcium: food sources		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.0	0.7
	Quartile 3	1.1	1.0
	Quartile 4	0.8	0.9
	p (trend)	0.44	0.90
	Lycopene sources: combined food groups		
	0–3 servings/mo	1.0 (ref.)	1.0 (ref.)
	1 serving/wk	1.5	1.4
	2–4 servings/wk	1.4	1.0
	5 + servings/wk	1.0	1.0
	p (trend)	0.14	0.54

Table 2 continued

Study	Exposure	Results	
		African American	White
	Use of multivitamins		
	No	1.0 (ref.)	1.0 (ref.)
	Yes	0.8 (0.5, 1.2)	0.8 (0.5, 1.3)
Dairy, grains, miscellaneous foods			
Hayes, 1999	Breads, grains, and cereals		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.0	0.9
	Quartile 3	1.2	0.9
	Quartile 4	1.7	0.9
	p (trend)	0.03	0.63
	Dairy foods		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.2	1.4
	Quartile 3	1.5	1.2
	Quartile 4	1.1	1.7
	p (trend)	0.57	0.07
	Sweets		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	0.6	1.4
	Quartile 3	1.2	1.4
	Quartile 4	1.2	2.2 ($p < 0.05$)
	p (trend)	0.15	0.006
	Calories from food		
	Quartile 1	1.0 (ref.)	1.0 (ref.)
	Quartile 2	1.3	1.4
	Quartile 3	2.4 ($p < 0.05$)	1.6
	Quartile 4	2.2 ($p < 0.05$)	1.5
	p (trend)	0.0004	0.16
Family history			
Hayes, 1995	Family history of prostate cancer		
	No	1.0 (ref.)	1.0 (ref.)
	Yes	3.4 (1.3, 9.3)	2.1 (0.9, 4.5)

ref. referent group, *BMI* body mass index. *Dashes* (–) indicate results could not be calculated due to small numbers or were not provided in original report

Advanced prostate cancer was defined differently between studies but based on cancer stage (*i.e.*, regional/distant cases) and/or grade (*i.e.*, cells were poorly differentiated) in all instances

*Indicates cohort study; all others were case–control studies

similar weak associations found among Whites (relative risk (RR), 1.19; 95% confidence interval (CI), 1.15–1.24) and African Americans (RR, 1.12; 95% CI, 1.04–1.20) [13].

A small study including only African Americans reported that waist circumference of greater than 102 cm was positively associated with prostate cancer risk relative to waist circumference of 102 cm or smaller (odds ratio (OR), 1.84; 95% CI, 1.17–2.91) [15]. Self-reported anthropometric measurements during childhood were examined in one study, with results suggesting that White men who recalled being taller ($p_{trend} = 0.0009$) and heavier ($p_{trend} = 0.04$) during childhood had increased risk of prostate cancer [10]. Such trends were not observed among African Americans in this study, despite similar sample sizes between the racial groups.

Tobacco use

Four studies examined race-stratified associations between smoking and prostate cancer [9, 12, 17, 18]. Of these, a

small case–control study did not find an association with history of cigarette smoking (ever vs. never) among either race [17]. Two larger studies examined both former and current smoking relative to never smoking and found null or weak positive associations; results did not appear to differ by racial group [12, 18]. The fourth, a hospital-based investigation, suggested positive associations between prostate cancer and both former (OR = 1.4) and current smoking (OR = 1.7) among African Americans only [9].

Two studies examined associations between duration and intensity of cigarette smoking and prostate cancer risk [12, 18]. One study found positive associations among those who reported smoking 40 or more cigarettes per day for both African Americans (former smokers: OR, 1.1; 95% CI, 0.6–2.0; current smokers: OR, 1.9; 95% CI, 0.9–4.2) and Whites (former smokers: OR, 1.5; 95% CI, 1.0–2.2; current smokers: OR, 1.3; 95% CI, 0.7–2.4) [18]. The other, smaller study reported no associations with smoking amount or duration among either race [12]. Use of pipes, cigars, snuff, and chewing tobacco was examined in one investigation, finding a positive, but very imprecise, association between current snuff use and prostate cancer risk among African Americans (OR, 4.7; 95% CI, 0.9–24.7) [18].

Alcohol use

Four studies reported race-stratified results when examining associations between alcohol intake and prostate cancer risk [9, 12, 17, 19]. Two small case–control investigations reported no evidence of an association among either race [12, 17], and a larger study found weak associations among African Americans (OR, 1.2; 95% CI, 0.9–1.7) and Whites (OR, 1.2; 95% CI, 0.9–1.7) when comparing those who reported any alcohol intake to those who never drank [19]. A hospital-based study that looked at alcohol consumption also found weak positive associations among both races [9].

When alcohol consumption was examined in a more detailed manner, one investigation reported the odds of prostate cancer were elevated for men who had 57 or more drinks per week (African Americans: OR, 1.8; 95% CI, 1.1–3.0; Whites: OR, 2.0; 95% CI, 1.2–3.4) [19]. Similar risk patterns for increased consumption were apparent when beer, liquor, and wine were examined individually [19]. A more recent, much smaller study failed to replicate these findings when examining number of drinks per day, with the highest level of consumption defined as five or more drinks per day [12].

Physical activity

Five studies presented race-stratified results for physical activity [9, 10, 12, 16, 20]. A hospital-based investigation reported that both African Americans and Whites with low or moderate levels of physical activity had a slightly increased risk of prostate cancer compared to men who were more active [9]. Three population-based case–control studies found no association between physical activity and prostate cancer in either race [10, 12, 16]. A prospective cohort investigation reported that, among African Americans, low physical activity levels were associated with elevated prostate cancer risk (non-recreational: RR, 3.74; 95% CI, 1.66–8.44; recreational: RR, 3.17; 95% CI, 0.96–10.46) [20]. However, only 47 African American cases were included in this study and the confidence intervals for effect estimates were wide [20]. These patterns were not observed among Whites [20].

General health

Four investigations examined associations between diabetes and prostate cancer [15, 21–23]. A small hospital-based study found an inverse association among Whites (OR, 0.44; 95% CI, 0.21–0.93) and a positive effect among African Americans (OR, 3.16; 95% CI, 0.67–15.0), with the interaction between race and diabetes status reaching statistical significance ($p < 0.05$) [21]. A small case–control study found an inverse association among African Americans (OR, 0.36; 95% CI, 0.21–0.62) but not Whites (OR, 1.08; 95% CI, 0.66–1.78) [22], and another small study conducted among African Americans only found no relation between diabetes and prostate cancer occurrence (OR, 0.96; 95% CI, 0.55–1.68) [15]. Finally, a large prospective cohort investigation found some evidence of an inverse association among both African Americans (RR, 0.59; 95% CI, 0.37–0.95) and Whites (RR, 0.82; 95% CI, 0.63–1.08) [23].

Two studies examined associations between blood pressure and prostate cancer risk [15, 24]. A large prospective cohort study found an inverse association with high blood pressure among both African Americans (RR, 0.82; 95% CI, 0.66–1.02) and Whites (RR, 0.88; 95% CI, 0.78–0.98) [24]. A subsequent smaller case–control study examining only African Americans reported a positive association between self-reported high blood pressure and prostate cancer risk (OR, 2.36; 95% CI, 1.49–3.73) [15].

Heart rate [24], aspirin use [25], use of thiazolidinediones [26], male pattern baldness [27], and prostate conditions (history of benign prostatic hyperplasia (BPH) and prostatitis) [28] were examined in one study each. Among both races, use of thiazolidinediones showed a weak positive association [26], aspirin use (defined as intake of 6 or more aspirin almost every day) showed a weak, non-significant inverse association [25], and heart rate showed no evidence of an association with prostate cancer risk [24]. Male pattern baldness was correlated with prostate

cancer occurrence among both African Americans (RR, 2.10; 95% CI, 1.04–4.25) and Whites (defined as anyone who was not African American; RR, 1.42; 95% CI, 1.01–1.98) in a cohort study [27]. A history of BPH was associated with increased risk among both races in a small case–control study, but history of prostatitis showed evidence of increased risk only among African Americans [28].

Diet

A wide range of dietary factors has been examined for associations with prostate cancer risk in race-stratified analyses [10, 12, 16, 17, 29–33]. Only one study assessed the total number of calories from food and reported an elevated risk among those in the upper quartiles of daily caloric intake compared to the lowest quartile among both African Americans (OR range, 1.5–1.8) and Whites (OR range, 1.3–1.7) [10]. When looking at fruit and vegetable intake, little effect on prostate cancer occurrence has been observed [10, 17, 29].

Two studies, a case–control study and a cohort investigation, reported an increase in prostate cancer occurrence among African Americans with higher levels of red meat intake, but not among Whites [10, 32]. For example, Hayes et al. [10] reported that African American men in the highest quartile of red meat intake had 1.9 times the odds of prostate cancer compared to men in the lowest quartile ($p < 0.05$). However, a more recent prospective cohort study with more African American participants but fewer White participants failed to find associations between red meat intake and prostate cancer in either racial group [33]. African American men with higher levels of either pork (RR, 2.3; $p < 0.05$) or beef or pork kidney (RR, 2.8; $p < 0.05$) consumption had increased prostate cancer occurrence in a small case–control study [17]. Poultry intake, on the other hand, showed an inverse association among both Whites (RR, 0.4; $p < 0.05$) and African Americans (RR, 0.4; $p < 0.01$) [17], but a subsequent cohort study failed to replicate these findings [32]. The latter study, however, contained fewer African American cases.

Fat intake has also been examined in multiple studies, producing inconsistent results [10, 12, 16, 17, 33]. One case–control study reported a positive association between higher dietary fat intake and prostate cancer among both races [10]. For example, both African American (OR range, 1.5–2.0) and White (OR range, 1.2–1.7) men in quartiles 2–4 of animal fat intake had higher prostate cancer risk compared to those in the lowest quartile [10]. In a smaller study, Ross and colleagues found similar positive associations between higher dietary fat intake and prostate cancer occurrence among White and African American men, but only the association in African American men reached statistical significance [17]. Additional studies, including a large cohort study, have reported no association in either racial group when looking at dietary fat intake as a risk factor [12, 16, 33].

Many additional dietary factors have been associated with prostate cancer risk, though findings are primarily from one study only or are inconsistent among a small number of studies. For instance, higher protein intake was associated with prostate cancer among African Americans (OR range, 1.4–1.8), but not Whites, in one study [10], while a smaller study reported no association among either racial group [17]. Lycopene intake has been addressed in three studies [10, 12, 30], with one reporting reduced risk among Whites only ($\geq$8.1 servings per week vs. $\leq$2.6 servings per week: OR, 0.55; $p < 0.05$) [12]. Prostate cancer occurrence has been associated with higher consumption of β-carotene [30] and eggs [17] and lower consumption of legumes [29] among African Americans. Among Whites, higher intake levels of eggs [17] and dairy foods [10] have been positively associated with prostate cancer. Serum levels of selenium were examined in one study, with non-significant trends reported among both races [31].

Family history of cancer

Family history of prostate cancer is an established risk factor for the disease [4] and has been examined in a race-stratified manner in five investigations [12, 34–37]. All reported an increased risk of prostate cancer among those with a positive family history [12, 34–37]. Family history of prostate cancer in any first-degree blood relative has been shown to increase risk similarly for men of both racial groups [12, 34–36]. For example, both African American (OR, 3.4; 95% CI, 1.5–7.5) and White (OR, 3.1; 95% CI, 1.8–5.3) men with a family history of prostate cancer had over three times the odds of prostate cancer compared to those without an affected first-degree relative in a study by Hayes and colleagues [34].

The association between affected relative type and prostate cancer risk has been examined in more detail in some investigations, and family history of other cancers has also been assessed as a potential risk factor for prostate cancer. One study showed an increase in risk among White men with an affected second-degree relative (OR, 4.44; $p < 0.05$) [12], though a larger study did not find this association [35]. Prostate cancer occurrence was higher among men with an affected brother in a small study comprised of only African Americans (OR, 4.80; 95% CI, 2.01–11.44) [37]. Family history of other cancers has mostly produced no association with prostate cancer risk [34, 37], although one small study including only African

Americans reported that having a sister with a history of breast cancer was positively associated with prostate cancer occurrence (OR, 3.80; 95% CI, 1.57–9.22) [37].

Occupation

Multiple studies have addressed occupation as a potential risk factor for prostate cancer using various job title and industry classifications [9, 12, 17, 38, 39]. Yu and colleagues found an increased prostate cancer risk among White men with professional occupations in a hospital-based investigation (OR, 1.8; 95% CI, 1.5–2.1) [9]. Krstev et al. examined a large number of occupations but reported no clear patterns of risk when comparing racial groups or white-collar jobs to blue-collar jobs [38]. Interestingly, farming was shown to increase risk of prostate cancer among White men in two studies (ORs: 1.8–2.7) [38, 39]; the larger study also found some evidence of an association among African Americans (OR, 1.97; 95% CI, 0.91–4.25) [38].

Specific occupational exposures have also been examined in a few investigations [17, 39]. Exposure to cadmium was not associated with prostate cancer among men of either race in a small case–control study [17]. Meyer et al. focused on exposures common to farming and reported that White farmers who reported ever mixing or applying pesticides had increased odds of prostate cancer compared to non-farmers (OR, 1.8; 95% CI, 1.2–2.7) [39]. The results were also suggestive of a weakly increased risk among the smaller sample of African American men (OR, 1.2; 95% CI, 0.8–2.0) [39]. Farmers who reported never picking cotton (both races) or never harvesting tobacco (White men only) also had increased risk of prostate cancer compared to men who were not farmers in this study [39].

Sexual health and behavior

Ten studies reported race-stratified effect estimates when examining associations between sexual behaviors or sexually transmitted infections (STIs) and prostate cancer risk [12, 14, 17, 28, 40–45]. Three of these studies looked at associations with history of any STI, of which one found no relation among either race [28], one found positive associations among both Whites (RR, 2.3; $p > 0.05$) and African Americans (RR, 1.7; $p < 0.05$) [17], and one found a weak positive relation among Whites only (OR, 1.3; 95% CI, 1.0–1.6) [45]. Two studies also examined STI frequency and found no overall relation with prostate cancer occurrence [28, 45], although White men with a history of three past STIs had an increased risk compared to men with no history of STI (OR, 1.7; 95% CI, 1.1–2.6) in a nested case–control study [45].

Three studies examined associations between human papillomavirus (HPV) infection and prostate cancer risk [42, 43, 45]. HPV status was determined by either serological or viral DNA evidence of HPV infection. One of these investigations, a very small study using BPH controls and testing for HPV types 6, 11, 16, 18, 31, 33, and 45, found positive, but imprecise associations between the presence of any HPV type and prostate cancer for African Americans (OR, 1.50; 95% CI, 0.14–15.59) and Whites (OR, 1.72; 95% CI, 0.34–8.76) [42]. A larger population-based investigation (testing for antibodies to HPV type 16) reported a positive association among Whites only (OR, 1.8; 95% CI, 0.7–4.9) [43], and the third study (testing for HPV types 16 and 18) found no associations among either race [45]. Herpes virus (herpes simplex virus [HSV] 1 and 2, human herpesvirus 8) has also been examined in multiple studies with no evidence of a relation with prostate cancer among either race [28, 44, 45].

Four investigations examined associations between gonorrhea and prostate cancer [12, 28, 43, 45]. Results were either null or weakly positive with no indication of consistent racial differences [12, 28, 43, 45]. Two studies evaluated frequency of gonorrhea infection, one of which found a positive trend among African Americans ($p = 0.0003$) [43]. The other, smaller study failed to find such a trend [28]. For syphilis, one investigation found positive associations among both African Americans (OR, 2.4; 95% CI, 1.2–4.9) and Whites (OR, 2.8; 95% CI, 0.2–49.1) [43], though the latter association was imprecise. Three additional studies found no evidence of an association between syphilis and prostate cancer in either race [12, 28, 45]. In examining gonorrhea and syphilis infection as potential risk factors, studies often lacked sufficient sample size to calculate effect measures for Whites due to low prevalence of infection.

Other STIs have been examined as potential risk factors in one study each. Chancroid and cytomegalovirus infection were not associated with prostate cancer among either White or African American men [28, 45]. Serological evidence of Chlamydia (IgA) was related to prostate cancer risk among African Americans only (OR, 2.1; 95% CI, 1.2–3.6) [45].

Two case–control studies examined vasectomy with neither observing associations with prostate cancer among Whites and one finding an elevated non-significant relation for self-reported history of vasectomy among African Americans (OR, 1.6; 95% CI, 0.5–4.8) [40, 41]. No consistent patterns emerged when examining age at vasectomy or years since vasectomy [40, 41], although one study reported an increased risk among Whites who were 25–34 years old at the time of their vasectomies (OR, 2.2; 95% CI, 1.0–4.4) [40].

The following potential risk factors were examined in one study each: number of marriages, number of children, age at first sexual intercourse, frequency of sexual intercourse, number of female sexual partners, sexual intercourse with prostitutes, condom use, levels of sex hormones, and circumcision [14, 17, 43]. Circumcision was inversely associated with prostate cancer among both racial groups [17], while condom use was inversely associated only among African Americans [43]. The number of sexual encounters with prostitutes and sex hormone levels were positively associated with prostate cancer among only Whites [14, 43].

Advanced prostate cancer

Only seven studies reported race-stratified effect estimates for risk factors and advanced prostate cancer, with each risk factor addressed in only 1 or 2 studies (Table 2) [10, 16, 19, 21, 23, 32, 34]. Overall, studies tended to report comparable results for advanced prostate cancer as they did for all prostate cancer cases combined. Greater height was associated with increased risk for advanced prostate cancer among Whites (OR range: 2.1–2.2) but not among African Americans (OR range: 0.8–1.1) [10]. Positive associations involving perceived anthropometric measurements (height and weight) during childhood were also only present among Whites [10]. Heavy alcohol use (57 or more drinks per week) was correlated with increased risk of advanced prostate cancer among both African Americans (OR, 2.0; 95% CI, 1.1–3.6) and Whites (OR, 2.1; 95% CI, 1.1–3.8) [19]. For diabetes, a small hospital-based study found an inverse association among Whites (OR, 0.27; 95% CI, 0.11–0.71) and was suggestive of a positive effect among African Americans (OR, 2.74; 95% CI, 0.55–13.5) [21]. A larger cohort study did not find an association between glucose tolerance and advanced prostate cancer among either race [23]. A family history of prostate cancer was correlated with higher risk of advanced prostate cancer among both African Americans (OR, 3.4; 95% CI, 1.3–9.3) and Whites (OR, 2.1; 95% CI, 0.9–4.5) [34].

Dietary results for advanced prostate cancer also tended to be similar to those reported for all prostate cancer cases. One study found statistically significant trends involving intake of meat (OR range: 1.6–2.4), red meat (OR range: 1.7–2.5), and protein (OR range: 2.1–2.9) with advanced prostate cancer among African Americans, but not among Whites [10]. This same study reported positive associations for intake of fat and animal fat among men of both races [10]. Total number of calories was also positively associated with higher risk of advanced prostate cancer among both races, though only the results for African American men reached statistical significance [10].

Discussion

African Americans have higher prostate cancer incidence rates than Whites in the US [2]. In this report, we reviewed the existing literature addressing race-specific associations between potential risk factors and prostate cancer. These associations could vary by race due to differences in prostate tumor biology [46, 47], risk factor prevalence, and characteristics between racial groups. For example, in the US, there is a higher prevalence of obesity among African Americans relative to Whites [48]. While many studies reported race-specific associations for all prostate cancer cases, few reported such associations for advanced prostate cancer cases. It is important to examine and report results for advanced prostate cancer since a higher percentage of prostate cancers are diagnosed at advanced stages for African American men compared to Whites [3]. Findings from our review not only summarize what is currently known about racial differences in prostate cancer risk factors but also underscore areas where future research is needed.

No clear pattern of differences by racial group was observed among studies evaluating associations between prostate cancer and physical activity, weight/BMI, diet, occupational history, sexual behavior, STIs, and other health conditions. The current literature suggests that alcohol and tobacco use may not be important risk factors for prostate cancer among either African Americans or Whites, though there is some evidence of an association among both races at very high levels of exposure for both risk factors. High levels of alcohol use may also increase the risk of advanced prostate cancer among both racial groups. Consistent associations between family history of prostate cancer and risk of disease (all prostate cancer and advanced prostate cancer) were found among both racial groups. Research also suggests modest positive associations may exist between height and prostate cancer risk (all prostate cancer and advanced prostate cancer) among Whites only. It has been suggested that height may be related to prostate cancer through the insulin-like growth factor system [49].

There are a number of issues that should be considered when interpreting results from the studies included in this review. First, the statistical power to detect associations was likely lower for African Americans than Whites due to smaller sample sizes. Across all 37 manuscripts, the median number of White prostate cancer cases was 494, compared to only 166 African Americans cases. Underrepresentation of minority populations in research studies is well recognized [50, 51], and continued efforts are needed among these populations to increase their participation in future epidemiologic and clinical studies.

Detection bias may have affected studies examining associations between prostate cancer and weight-related variables, family history of prostate cancer, diabetes, blood pressure, and vasectomy. Obese individuals are likely to have other medical conditions requiring regular medical visits, which may lead to an increased chance of prostate cancer detection [52]. Men with a family history of prostate cancer are more likely to utilize prostate cancer screening [53], which could also lead to increased detection. Results concerning blood pressure and diabetes may be affected by detection bias since men undergoing increased medical care for these conditions may be more likely to be screened for prostate cancer [52]. Likewise, vasectomy may increase the chance for incidental discovery of a prostate malignancy [54].

Misclassification bias may have affected studies examining associations between prostate cancer and weight-related variables, physical activity, diet, alcohol intake, tobacco use, sexual behavior, STIs, diabetes, family history of prostate cancer, and blood pressure. This may be due to inadequate recall of relevant information that may be exacerbated by the memory problems common among older men, the group most affected by prostate cancer [4]. Blood pressure and diabetes were self-reported in some studies (1 of 2 for blood pressure, and 3 of 4 for diabetes), which may cause either differential or nondifferential misclassification [55]. Data on obesity and other body measurements were collected using various methods (self-report, trained personnel taking measurements, clinical diagnosis) and analyzed in different formats (dichotomously, quintiles, etc.). Likewise, data on STIs were obtained differently both across and within investigations (self-report, serological assessment, measurement of viral DNA in prostatic tissues). All five studies addressing family history of cancer relied on self-reported information and only two [35, 36] attempted to verify these data. This is concerning since self-reported family histories of prostate cancer have been shown to contain inaccuracies [56, 57].

It is also possible that reporting of certain health behaviors differed by racial group and that some data collection instruments were not ideal for all participants. For example, research has shown that African Americans underreport tobacco use more frequently compared to Whites [58]. Furthermore, some studies used food frequency questionnaires or diet histories that were not modified for local diet and did not incorporate racial differences in dietary habits. Both potential limitations could lead to measurement error, which may be particularly problematic for those studies that relied solely upon self-reported data.

There was inconsistency between studies in controlling for potential confounders. Age and family history of prostate cancer are two established risk factors for prostate cancer [4, 5]. Although almost all studies controlled for age, few controlled for family history of prostate cancer. Additionally, few studies accounted for prostate cancer screening history (*e.g.*, prostate-specific antigen [PSA] testing), which is important since PSA testing may differ by race [59, 60] and such testing has led to a large increase in the incidence of prostate cancer in the US [61]. Many of these detected cancers are slow-growing and would not lead to clinical manifestations of disease [62].

Our review has several strengths including the direct examination of risk factors for total and advanced prostate cancer and that almost all studies recruited both African Americans and Whites. We believe such studies allow for more direct, and possibly more valid, racial comparisons of reported associations. Our review was also limited by a number of factors. Specifically, we did not examine race-specific associations for screening patterns, genetic variants, or demographic factors. In addition, this review was qualitative in nature, as we did not perform quantitative analyses of the abstracted data. For advanced prostate cancer, we present and discuss data from various studies that used slightly different definitions of advanced prostate cancer. Finally, we did not include studies that were comprised of only Whites or did not report race-specific associations. However, the results we report for Whites appear to be consistent with the overall prostate cancer literature [1, 7].

We found no evidence of racial differences in associations between prostate cancer and alcohol intake, tobacco use, and family history of prostate cancer. Research suggests a modest positive association may exist between height and prostate cancer risk (all prostate cancer and advanced prostate cancer) among Whites only. No clear patterns were discernible among studies evaluating associations between prostate cancer and physical activity, weight/BMI, dietary factors, occupational history, sexual behavior, STIs, and other health conditions. These findings summarize what is currently known about racial differences in prostate cancer risk factors and highlight areas where future research is needed. As demonstrated by this review, enrolling higher numbers of African American participants and examining how risk factors affect both total prostate cancer occurrence and advanced prostate cancer occurrence will be extremely important in advancing this field of research.

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