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SHORT REPORT

Overall and abdominal obesity and prostate cancer risk in a West African population: An analysis of the Ghana Prostate Study

Lauren M. Hurwitz[1] | Edward D. Yeboah[2] | Richard B. Biritwum[3] | Yao Tettey[4] | Andrew A. Adjei[4] | James E. Mensah[2] | Evelyn Tay[5] | Vicky Okyne[5] | Ann Truelove[6] | Scott P. Kelly[1] | Cindy Ke Zhou[1] | Eboneé N. Butler[1] | Robert N. Hoover[1] | Ann W. Hsing[7] | Michael B. Cook[1]

[1]Division of Cancer Epidemiology and Genetics, National Cancer Institute, Bethesda, Maryland

[2]Department of Surgery, University of Ghana Medical School, Accra, Ghana

[3]Department of Community Health, University of Ghana Medical School, Accra, Ghana

[4]Department of Pathology,University of Ghana Medical School, Accra, Ghana

[5]College of Health Sciences, University of Ghana Medical School, Accra, Ghana

[6]Westat, Rockville, Maryland

[7]Department of Medicine, Stanford Prevention Research Center and Cancer Institute, Stanford University School of Medicine, Stanford, California

Correspondence
Lauren M. Hurwitz, Division of Cancer Epidemiology and Genetics, National Cancer Institute, 9609 Medical Center Drive, Rockville, MD 20850.
Email: lauren.hurwitz@nih.gov

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Abstract

Obesity has been associated with an increased risk of advanced prostate cancer. However, most studies have been conducted among North American and European populations. Prostate cancer mortality appears elevated in West Africa, yet risk factors for prostate cancer in this region are unknown. We thus examined the relationship between obesity and prostate cancer using a case-control study conducted in Accra, Ghana in 2004 to 2012. Cases and controls were drawn from a population-based sample of 1037 men screened for prostate cancer, yielding 73 cases and 964 controls. An additional 493 incident cases were recruited from the Korle-Bu Teaching Hospital. Anthropometric measurements were taken at enrollment. We used logistic regression to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for associations between body mass index (BMI), waist circumference (WC), waist-hip ratio (WHR) and prostate cancer, adjusting for potential confounders. The mean BMI was 25.1 kg/m^2 for cases and 24.3 kg/m^2 for controls. After adjustment, men with BMI ≥ 30 kg/m^2 had an increased risk of prostate cancer relative to men with BMI < 25 kg/m^2 (OR 1.86, 95% CI 1.11-3.13). Elevated WC (OR 1.76, 95% CI 1.24-2.51) and WHR (OR 1.46, 95% CI 0.99-2.16) were also associated with prostate cancer. Associations were not modified by smoking status and were evident for low- and high-grade disease. These findings indicate that overall and abdominal obesity are positively associated with prostate cancer among men in Ghana, implicating obesity as a potentially modifiable risk factor for prostate cancer in this region.

KEYWORDS

body-mass index, obesity, prostate cancer, West Africa

Abbreviations: BMI, body mass index; CI, confidence interval; CUP, Continuous Update Project; DRE, digital rectal exam; OR, odds ratio; PSA, prostate-specific antigen; SES, socioeconomic status; WC, waist circumference; WHR, waist-hip ratio.

1 | INTRODUCTION

Prostate cancer is one of the leading causes of cancer incidence and mortality worldwide.[1] Rates vary substantially across geographic region and by race/ethnicity, likely due to differences in underlying genetic predisposition, lifestyle factors and access to screening and treatment.[2]

Obesity, often defined by a high body mass index (BMI), waist circumference (WC) or waist-hip ratio (WHR), is one modifiable lifestyle factor that has been linked with prostate cancer.[3] Despite extensive research, however, the obesity-prostate cancer relationship remains to be fully elucidated, in part due to several complexities. First, obesity appears to exert differential effects on aggressive and nonaggressive disease. Specifically, while there is no conclusive evidence linking obesity to overall prostate cancer risk,[3] obesity appears to be associated with an increased risk of prostate cancer that is advanced or high-grade at diagnosis, prostate cancer recurrence and prostate cancer-specific mortality.[3,4] Further clarification of the association with aggressive, clinically relevant prostate cancer is needed, yet difficult in settings with routine prostate-specific antigen (PSA) testing and the consequent predominant detection of early-stage prostate cancer.

Second, there is evidence to suggest that the association between obesity and prostate cancer risk varies by race. For example, a prospective US-based study observed an inverse association between obesity and low-grade prostate cancer among non-Hispanic white men but a positive association among African-American men.[5] While obesity was associated with increased risk of high-grade prostate cancer for both racial groups, the association was stronger for African-Americans.[5] Other studies have reported no racial differences in associations between obesity and prostate cancer risk.[6,7] In general, associations between obesity and prostate cancer have been understudied in African-American or African populations, despite the fact that these men are at higher risk of prostate cancer incidence and mortality.[2]

To address these challenges, we examined the relationship between both overall obesity, defined by BMI, and abdominal obesity, defined by WC and WHR, and prostate cancer risk in a case-control study conducted in West Africa, where the majority of prostate cancers are advanced or symptomatic at diagnosis[8] and thus clinically relevant.

What's new?

Despite high rates of prostate cancer mortality in West Africa, risk factors for prostate cancer in African populations remain understudied. In this case-control study, the authors examined relationships between obesity and prostate cancer in cancer patients and healthy controls in Accra, Ghana. Analyses uncovered associations between increased prostate cancer risk and both overall obesity and abdominal obesity. Unlike in Western populations, obesity was associated with both low- and high-grade prostate cancer. The findings identify obesity as an important prostate cancer risk factor in West Africa and shed light on the complexity of the obesity-prostate cancer relationship in diverse populations.

2 | MATERIALS AND METHODS

2.1 | Study design

The Ghana Prostate Study was designed to study the detection and treatment of prostate cancer in the Accra region of Ghana. The study included both a population-screening component and a clinical component. For the population component, a probability sample of approximately 1000 individuals ages 50 to 74 were selected from the 2000 Ghana Population and Housing Census data. There were 1049 men sampled between 2004 to 2006, of which 1037 were ultimately enrolled (a response rate of 98.8%). In-person interviews and clinical examinations of enrolled men were conducted at the Korle-Bu Teaching Hospital. This included a PSA test and a digital rectal examination (DRE) to screen for prostate cancer. Men with a PSA concentration ≥2.5 ng/mL or a positive DRE were referred for prostate biopsy. Seventy-three men were diagnosed with histologically confirmed prostate cancer from this screening study.[9]

For the clinical component, an additional 676 prostate cancer patients were recruited from the Korle-Bu Teaching Hospital from 2008 to 2012. Korle-Bu is Ghana's largest hospital and the only hospital in the nation where treatment for prostate cancer is available; as a result, it is estimated that almost all prostate cancer patients diagnosed in Accra and the majority of prostate cancer patients diagnosed within Ghana are referred to the Korle-Bu Hospital for treatment. For the recruited cases, information on demographic characteristics, lifestyle factors, urinary symptoms and healthcare utilization were ascertained via an in-person interview, while clinical variables such as clinical stage and Gleason grade were abstracted from medical records.

For the current analysis, only clinical cases diagnosed within 1 year of enrollment (n = 493) were included to reduce potential reverse causation introduced by prostate cancer diagnosis. These cases were combined with the 73 cases from the population screening component and compared to controls, defined as men from the screening component who were not diagnosed with prostate cancer (n = 964). The Ghana Prostate Study was approved by institutional review boards at the University of Ghana and the U.S. National Cancer Institute and all participants provided informed consent.

2.2 | Anthropometric measurements

As part of the in-person interviews, a trained interviewer measured each participant's standing height (cm), weight (kg), WC (cm) and hip circumference (cm). Each measurement was taken twice, and if the difference between measurements was found to exceed a

TABLE 1 Selected characteristics of cases and controls in the Ghana Prostate Study

Characteristic	Controls n (%)[a]	Cases n (%)[a]
Total n	964 (100)	566 (100)
Age at enrollment, mean (SD)	60.1 (7.1)	69.1(8.6)
Ethnicity, n (%)		
Asanta	84 (9)	67 (12)
Akwapim	52 (5)	46 (8)
Fante	104 (11)	77 (14)
Other Akan	73 (8)	82 (14)
Ga/Adangbe	369 (38)	141 (25)
Ewe	169 (18)	96 (17)
Guan/Mole-Dagbani/Gr	60 (6)	25 (4)
Other/Unknown	53 (5)	32 (6)
Highest education, n (%)		
Less than Secondary	608 (63)	247 (44)
Secondary or more	307 (32)	299 (53)
Unknown	49 (5)	20(4)
Marital status, n (%)		
Yes	809 (84)	459 (81)
No	154 (16)	104 (18)
Unknown	1 (0)	3 (1)
Insurance status, n (%)		
Yes	39 (4)	372 (66)
No	905 (94)	188 (33)
Unknown	20 (2)	6 (1)
Smoking status, n (%)		
Never	521 (54)	368 (65)
Former	271 (28)	174 (31)
Current	132 (14)	16 (3)
Unknown	40 (4)	8 (1)
Occupation, n (%)		
Management	77 (8)	136 (24)
Military	17 (2)	41 (7)
Other/Unknown	870 (90)	389 (69)
Method of ascertainment, n (%)		
Population-based	964 (100)	73 (13)
Hospital-based	—	493 (87)
Clinical stage		
T1	—	91 (16)
T2	—	300 (53)
T3	—	76 (13)
T4	—	54 (10)
Unknown	—	45 (8)
Gleason score		
≤6	—	162 (29)
7	—	180 (32)
≥8	—	189 (33)
Unknown	—	35 (6)

[a]Percentages are column percentages.

prespecified tolerance limit, a third measurement was also recorded. For this analysis, the average value per participant for each measurement was used. BMI was calculated as weight divided by height in meters squared (kg/m^2), and WHR was calculated as WC divided by hip circumference.

2.3 | Statistical analysis

Unconditional logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for associations between anthropometric measurements and prostate cancer. Anthropometric variables were analyzed continuously as well as categorically using either study-specific quartiles based on the control distribution or recommended cut points[10,11] when available. In the primary analyses, each anthropometric variable was analyzed individually, while secondary analyses modeled BMI and WC or WHR simultaneously to examine their independent effects. Concordance between the measures was too high to compute results cross-classified by BMI and WC or WHR. Models were adjusted for potential confounders specified a priori, including age at enrollment (as a continuous variable), ethnicity (categorized as Asanta, Akwapim, Fante, Other Akan, Ga/Adangbe, Ewe, Guan/Mole-Dagbani/Grussi/Gruma/Hausa, other), educational attainment (less than secondary, secondary or more, unknown), health insurance status (yes, no, unknown) and smoking status (never, former, current, unknown). Given prior findings in our study population of an increased risk of prostate cancer among men in management and military occupations,[12] models were adjusted for these occupations (management, military, other/unknown) as well. We used missing indicators to retain men with missing covariate information (representing <5% of the study population) in the models. To assess whether smoking may be an effect measure modifier of the relationship between BMI and prostate cancer risk,[13,14] analyses were stratified by smoking status (ever, never), with statistical interaction evaluated using likelihood ratio tests. Multinomial logistic regression was used to examine associations separately for low-grade (Gleason score ≤7) and high-grade (Gleason score ≥8) prostate cancer. All analyses were conducted in SAS 9.4.

3 | RESULTS

There were 566 prostate cancer cases and 964 controls included in this analysis. Selected characteristics by case-control status are displayed in Table 1 and Table S1. The mean age of cases was 69 years

and the mean age of controls was 60 years. Cases were more likely than controls to have a secondary education and health insurance, to be in management occupations, and to have never smoked. Of the cases, 31% were Gleason score ≤6, 34% were Gleason score 7 and 36% were Gleason score ≥8.

The mean BMI of at the time of study enrollment was 25.1 kg/m^2 for cases and 24.3 kg/m^2 for controls. Based on BMI, 13% of cases and 9% of controls were obese (BMI ≥ 30 kg/m^2), and 35% of cases and 31% of controls were overweight (BMI 25 to <30 kg/m^2, Table 2). The prevalence of abdominal obesity, defined as WC > 94 cm, was 37% in cases and 22% in controls (Table 2). Correlation between BMI and WC was high (r = .83), with 93% of men with an obese BMI also exhibiting abdominal obesity assessed by WC.

After multivariable adjustment, increasing BMI was associated with prostate cancer risk, both when comparing obese men with normal weight/underweight men (HR: 1.86, 95% CI: 1.11-3.13, Table 2) and when assessing BMI continuously (HR: 1.23, 95% CI: 1.01-1.49 per 5 kg/m^2 increase). WC and WHR were also positively associated with prostate cancer. Specifically, a WC >94 cm was associated with a 76% increased risk (HR: 1.76, 95% CI: 1.24-2.51), while a WHR >0.90 was associated with a 46% increased risk (HR: 1.46, 95% CI: 0.99-2.16). Associations were generally retained in models mutually adjusted for both BMI and WC or WHR (Table S2). Associations were also similar in magnitude when analyses were restricted to individuals without health insurance, though associations for WC and WHR were no longer significant (Table S3). Adult height was not associated with prostate cancer risk, either when analyzed continuously or as a categorical variable based on quartiles (Table 2).

Results remained largely unchanged when stratified by smoking status. Obesity—as defined by either BMI ≥ 30 kg/m^2 or WC > 94 cm—was associated with an approximately twofold increase in prostate cancer risk among both never smokers and current/former smokers (Table S4). High WHR was also positively though nonsignificantly associated with prostate cancer risk across category of smoking status (Table S4).

When results were examined by prostate cancer grade at diagnosis, high BMI (≥30 kg/m^2) was more strongly associated with low-grade prostate cancer (HR: 2.36, 95% CI: 1.32-4.21, Table 3) than high-grade prostate cancer (HR: 1.51, 95% CI: 0.73-3.12, P-heterogeneity < .01). In contrast, elevated WC and WHR were associated with risk of both low-grade and high-grade prostate cancer (Table 3). Results were similar when we classified Gleason score 7 tumors with the high-grade tumors as opposed to the low-grade tumors (Table S5).

TABLE 2 Associations between anthropometric measures and prostate cancer in the Ghana Prostate Study

	Controls n (%)	Cases n (%)	Age-adjusted OR (95% CI)	Multivariable-adjusted[a] OR (95% CI)
Total n	964 (100)	566 (100)		
BMI (kg/m^2)				
Per 5 kg/m^2 increase[b]	—	—	1.38 (1.20, 1.60)	1.23 (1.01, 1.49)
<25	571 (60)	292 (53)	1.00 (ref)	1.00 (ref)
25 to <30	292 (31)	193 (35)	1.37 (1.05. 1.78)	1.14 (0.80, 1.64)
≥30	82 (9)	70 (13)	2.16 (1.44, 3.21)	1.86 (1.11, 3.13)
Height (cm)				
Per 5 cm increase[b]	—	—	0.95 (0.87, 1.04)	0.93 (0.83, 1.05)
<164[c]	259 (27)	176 (32)	1.00 (ref)	1.00 (ref)
164 to <168.5[c]	239 (25)	152 (27)	1.14 (0.83, 1.58)	1.04 (0.67, 1.61)
168.5 to <173[c]	223 (24)	139 (25)	1.21 (0.87, 1.69)	1.33 (0.85, 2.07)
≥173[c]	224 (24)	88 (16)	0.94 (0.66, 1.33)	0.86 (0.54, 1.39)
Waist circumference (cm)				
Per 5 cm increase[b]	—	—	1.14 (1.08, 1.21)	1.07 (1.00, 1.15)
≤94[d]	750 (78)	354 (63)	1.00 (ref)	1.00 (ref)
>94[d]	213 (22)	204 (37)	2.13 (1.63, 2.78)	1.76 (1.24, 2.51)
Waist-hip ratio				
≤0.9[d]	282 (29)	99 (18)	1.00 (ref)	1.00 (ref)
>0.9[d]	680 (71)	459 (81)	1.64 (1.23, 2.20)	1.46 (0.99, 2.16)

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.

[a]Adjusted for age, ethnicity, education, insurance status, smoking status and occupation.

[b]Extreme outliers excluded (ie, observations more than 3*IQR above Q3 or below Q1).

[c]Cut points are based on the quartiles of the height distribution of controls.

[d]Cut points are based on the International Diabetes Federation's sex- and ethnicity-specific cut points for central obesity[11] and examples from WHO.[10]

TABLE 3 Associations between anthropometric measures and low- and high-grade prostate cancer in the Ghana Prostate Study

			Gleason ≤7			Gleason ≥8		
	Controls (n = 964)	Cases (n = 443)	Age-adjusted OR (95% CI)	Multivariable-adjusted[a] OR (95% CI)	Cases (n = 199)	Age-adjusted OR (95% CI)	Multivariable-adjusted[a] OR (95% CI)	*P*-heterogeneity
BMI (kg/m^2)								
Per 5 kg/m^2 increase[b]	—	—	1.60 (1.36, 1.89)	1.43 (1.14, 1.79)	—	1.11 (0.90, 1.36)	1.01 (0.76, 1.33)	<.01
<25	571	155	1.00 (ref)	1.00 (ref)	117	1.00 (ref)	1.00 (ref)	<.01
25 to <30	292	136	1.80 (1.33, 2.44)	1.38 (0.92, 2.09)	46	0.82 (0.55, 1.22)	0.66 (0.40, 1.10)	
≥30	82	47	2.69 (1.72, 4.21)	2.36 (1.32, 4.21)	20	1.58 (0.89, 2.81)	1.51 (0.73, 3.12)	
Height (cm)								
Per 5 cm increase[b]	—	—	0.95 (0.86, 1.05)	0.94 (0.82, 1.08)	—	0.97 (0.86, 1.11)	0.97 (0.82, 1.14)	.57
<164[c]	259	107	1.00 (ref)	1.00 (ref)	58	1.00 (ref)	1.00 (ref)	.78
164 to <168.5[c]	239	96	1.25 (0.86, 1.79)	1.14 (0.70, 1.87)	49	1.19 (0.75, 1.88)	1.15 (0.64, 2.08)	
168.5 to <173[c]	223	84	1.24 (0.85, 1.81)	1.40 (0.84, 2.32)	45	1.24 (0.78, 1.99)	1.43 (0.78, 2.62)	
≥173[c]	224	51	0.92 (0.66, 1.84)	0.86 (0.50, 1.50)	31	1.10 (0.66, 1.84)	1.15 (0.60, 2.21)	
Waist circumference (cm)								
Per 5 cm increase[b]	—	—	1.19 (1.12, 1.26)	1.12 (1.03, 1.22)	—	1.11 (1.03, 1.20)	1.07 (0.96, 1.18)	.29
≤94[d]	750	207	1.00 (ref)	1.00 (ref)	122	1.00 (ref)	1.00 (ref)	.83
>94[d]	213	131	2.34 (1.73, 3.15)	1.95 (1.31, 2.91)	64	1.98 (1.36, 2.87)	1.87 (1.16, 3.01)	
Waist-hip ratio								
≤0.9[d]	282	57	1.00 (ref)	1.00 (ref)	32	1.00 (ref)	1.00 (ref)	.98
>0.9[d]	680	281	1.78 (1.26, 2.52)	1.77 (1.11, 2.82)	154	1.73 (1.11, 2.67)	1.76 (1.01, 3.07)	

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.

[a]Adjusted for age, ethnicity, education, insurance status, smoking status and occupation.

[b]Extreme outliers excluded (ie, observations more than 3*IQR above Q3 or below Q1).

[c]Cut points are based on the quartiles of the height distribution of controls.

[d]Cut points are based on the International Diabetes Federation's sex- and ethnicity-specific cut points for central obesity[11] and examples from WHO.[10]

4 | DISCUSSION

In our study population of Ghanaian men, overall obesity, as assessed by BMI, and abdominal obesity, as assessed by WC and WHR, were positively associated with prostate cancer risk. Effect modification by smoking status was not observed, and associations with WC and WHR were evident for both low- and high-grade disease.

In 2014, the Continuous Update Project (CUP) from the World Cancer Research Fund International concluded that greater body fatness is a probable cause of advanced prostate cancer, but not non-advanced or total prostate cancer.[3] This conclusion was based on meta-analytic findings that BMI (summary RR: 1.08, 95% CI: 1.04-1.12 per 5 kg/m^2), WC (summary RR: 1.12, 95% CI: 1.04-1.21 per 10 cm) and WHR (summary RR: 1.15, 95% CI: 1.03-1.28 per 0.1 unit) were associated with advanced prostate cancer, the definition of which varied across study as advanced stage, high grade, metastatic or fatal. However, the studies included in the CUP meta-analyses were conducted in populations with primarily European ancestry, with none conducted in Africa.

Our study is the first to examine risk factors for prostate cancer in West African men. Cancer risk factor associations may differ in this region due to potential differences in the underlying genetic drivers of cancer, as well as differences in social and cultural factors. Earlier reports from the Ghana Prostate Study have observed prostate cancers of these men to exhibit genetic and molecular differences from those of men with European ancestry. For example, only 10 of the 81 prostate cancer risk loci known at the time were replicated in our study population,[15] and only 18% of prostate cancers in the Ghana Prostate Study were found to be ERG-positive, compared to 49% of prostate cancers in men of European descent.[16] Studying the impact of obesity in Ghana is also particularly important and timely, given the high and rising prevalence of obesity in Ghana.[17] Among the controls in our study population, which represented a population-based sample of men ages 50 to 74 residing in the greater Accra region of Ghana between 2004 and 2006, more than a third of men were overweight or obese according to their BMI, and approximately one-fifth exhibited abdominal obesity. These estimates indicate that the burden of obesity in Ghana is substantial and that downstream health effects need to be examined.

Our study found that both overall and abdominal obesity were associated with an increased risk of prostate cancer diagnosis among men in Ghana. Obesity is thought to contribute to prostate carcinogenesis through several metabolic, hormonal and inflammatory pathways. Specifically, obesity is associated with higher levels of insulin and insulin-like growth factor, lower levels of androgens, and altered levels of adipokines and proinflammatory mediators.[18] Abdominal obesity is considered particularly detrimental as it is more strongly associated with visceral fat and metabolic dysfunction.[19,20]

Interestingly, in contrast to the CUP conclusions, abdominal obesity was associated with low-grade prostate cancer in addition to high-grade prostate cancer in our study population. This finding could potentially be attributed to the low background prostate cancer screening rates in this population.[9] Without screening, prostate cancers are primarily detected due to symptoms, and as a result, the low-grade prostate cancers in our study may have been more aggressive compared to those detected in highly screened populations. It is also possible that low-grade tumors of African-descent populations are inherently more aggressive, regardless of prostate cancer screening, as has been suggested by racial disparities in outcomes for low-grade prostate cancer observed in the United States.[21] Alternatively, this finding could suggest that obesity is a prostate cancer risk factor for men of West African descent regardless of tumor aggressiveness. Other studies have also observed obesity, and particularly abdominal obesity, to be associated with both low- and high-grade prostate cancer among African-American[5] and Caribbean men.[22] Although from distinct geographic regions, these study populations likely share a higher degree of genetic ancestry,[23] and the consistency in findings suggests that there may be a genetic component underlying the observed association between obesity and low-grade prostate cancer for these men.

The CUP also concluded that adult attained height is positively associated with prostate cancer,[3] a finding that was not observed in our study population. Adult height is thought to be an indicator of early-life hormonal and nutritional exposures that could potentially influence cancer risk. Our observed null association between height and prostate cancer is consistent with the few studies that have examined this association in African-American men[24] and suggests that these early developmental exposures may not be risk factors for prostate cancer in African-descent populations.

A unique strength of the study is that controls were probability samples screened for prostate cancer via both PSA testing and DRE, reducing the probability of controls having undiagnosed prostate cancer. Additionally, most cases were recruited from the hospital and were likely diagnosed with symptomatic disease, given the very low population prevalence of PSA testing. As a result, our study was able to make a relatively clean comparison of clinically relevant prostate cancer cases and cancer-free controls. However, cases recruited from the hospital and receiving treatment for their prostate cancer were likely of a higher socioeconomic status (SES) than the population-based controls, as reflected by the higher levels of educational attainment and health insurance of our case group. Though we adjusted for these factors in our regression models and conducted a sensitivity analysis restricted to cases and controls without health insurance, there may have still been residual confounding by SES or other factors affecting both obesity and the ability to seek medical care. Given that adjustment for SES-related factors attenuated our results toward the null, any residual confounding would likely lead us to overestimate our effect estimates, but such confounding would have to be strong to completely nullify our findings.

Another strength of our study is that weight, height, WC and hip circumference were measured for all participants using standardized procedures, thereby reducing opportunity for measurement error and recall bias. However, these measures were assessed at the time of cancer diagnosis for the cases. Although we restricted to cases diagnosed within 1 year of enrollment to limit potential for reverse causation, we cannot fully rule out the possibility that prostate cancer influenced the cases'

body size. Our study was also only able to examine body size at a single time point in late adulthood. Looking at body size at different time periods would have been more informative, given that associations between body size and prostate cancer vary across the life course[25] and that specific BMI trajectories appear most correlated with risk.[13,26,27] Future studies should also seek to distinguish lean mass from fat mass, and visceral fat from subcutaneous fat. Use of more advanced technologies such as dual-energy X-ray absorptiometry, computerized tomography and magnetic resonance imaging—though expensive—may eventually help improve understanding of how specific body fat compartments contribute to prostate cancer risk.

In conclusion, overall and abdominal obesity were associated with an increased risk of prostate cancer among West African men. These results support a role for excess body weight in the development of prostate cancer and suggest that interventions to target obesity may help reduce the burden of prostate cancer in this region. Future studies should continue to examine the impact of obesity on cancer risk across diverse populations to further tease apart the complexities of the obesity-prostate cancer relationship.

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CONFLICT OF INTEREST

The authors declare no potential conflict of interest.

DATA ACCESSIBILITY

The data that support the findings of our study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ORCID

Lauren M. Hurwitz https://orcid.org/0000-0001-8932-5028

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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