



Research Article

Central Adiposity and Prostate Cancer in a Black Population

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Abstract

Background: The relationship between central adiposity and prostate cancer remains unclear.

Methods: This report includes 963 newly diagnosed cases of histologically confirmed prostate cancer and 941 randomly selected age-matched controls ascertained from the population-based *Prostate Cancer in a Black Population* study conducted between July 2002 and January 2011 in Barbados, West Indies. Trained nurse interviewers obtained data on height, weight, waist and hip circumferences, family and medical history, and lifestyle factors. ORs and 95% confidence intervals (CI) were used to assess associations between anthropometric measures and prostate cancer.

Results: A two-fold increased risk of prostate cancer was found among men in the highest quartile of waist-hip ratio compared with those in the lowest quartile (OR = 2.11, 95% CI, 1.54–2.88). Similarly, men with the largest waist circumferences had an OR of 1.84 (95% CI, 1.19–2.85) compared with those with the smallest waist sizes.

Conclusions: These results suggest that measures of central rather than global adiposity may be more predictive of prostate cancer, especially in westernized African populations, where patterns of visceral fat distribution are different than other groups.

Impact: The findings highlight the need to further elucidate the mechanisms underlying the relationship between central adiposity and prostate cancer in populations of predominantly African descent. *Cancer Epidemiol Biomarkers Prev*; 21(5); 851–8. ©2012 AACR.

Introduction

Obesity is a major public health issue, particularly among African-Americans who have a higher prevalence of global, as well as central, obesity than other groups (1, 2). African-Barbadians have similar characteristic patterns of body size and fat distribution as African-Americans, with lower visceral and more subcutaneous fat than populations of European descent (3, 4). While it has been postulated that obesity plays a role in the pathogenesis of prostate cancer, there is conflicting evidence on the relationship between prostate cancer and body size (5–7). This is likely due to the different outcome measures used to define obesity, population differences, and other influential factors.

Findings from numerous studies that have assessed a possible association of prostate cancer risk with global obesity [defined as body mass index (BMI) ≥ 30 kg/m²] have been inconsistent (8, 9). These discrepancies may be due, at least in part, to the limitations of BMI to differentiate between muscle and fat mass and to provide information relating to body fat distribution (10). As such, measurements of visceral fat, which may be more closely linked to metabolic changes, may serve as better predictors of prostate cancer development. Similarly, indicators of central fat distribution such as waist circumference, hip circumference, and waist-hip ratio (WHR) may be more appropriate than BMI for evaluating the potential relationship between body size and prostate cancer (11). Fewer investigations have included measures of central adiposity, however, and only a limited number have been conducted in populations of African origin. The findings from these studies have been inconsistent, with higher waist circumference implicated as increasing total prostate cancer risk in one study of African-American men in Michigan (12) but not another including men from Jamaica (11). The Jamaican study, however, reported that larger WHRs increased risk among high grade and all prostate cancer cases, whereas race-stratified data from a study in North Carolina did not corroborate these findings (13). The role of abdominal obesity on prostate cancer risk thus remains unclear, particularly among men of African descent.

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The Prostate Cancer in a Black Population (PCBP) study was designed to evaluate the contribution of epidemiologic and genetic factors to prostate cancer in Barbados, West Indies. One of the study's main aims was to evaluate the contribution of body size to prostate cancer risk, focusing particular attention to specific fat distribution patterns and the extent to which abdominal fat (as opposed to global obesity) may have different implications for risk. The purpose of this report is to describe the relationship between prostate cancer and body size variables for central obesity in the PCBP study.

Materials and Methods

The PCBP study was funded by the National Human Genome Research Institute and the Office of Minority Health, with subsequent funding provided by the National Cancer Institute of the NIH. The organizational structure of the study included a Coordinating Center (Stony Brook Medicine, Stony Brook, NY), a Clinical Center (Ministry of Health and University of the West Indies, Bridgetown, Barbados, WI), a Local Laboratory Center (University of the West Indies, Bridgetown, Barbados, WI), a center at the National Human Genome Research Institute (Bethesda, MD), and a Gene Discovery Center (Translational Genomics Research Institute, Phoenix, AZ).

Study population

Eligible participants for the PCBP study, a nationwide case-control study, included all male citizens of Barbados with newly diagnosed, histologically confirmed, primary prostate cancer. Cases were identified by the country's only Pathology Department, located at the Queen Elizabeth Hospital, Bridgetown, Barbados, WI, between July 2002 and January 2011. Controls were randomly selected from a national database of Barbados citizens and permanent residents (14) and were frequency age matched (by 5-year age groups) on the basis of the case's age at the date of diagnosis. Of the 1,260 eligible cases and 1,333 eligible controls, 1,007 (80%) of the cases and 1,005 (75%) of the controls participated. The primary reasons for nonparticipation in both groups were death, illness/disability, leaving the island, and refusal. No significant difference in age between participants and nonparticipants was found. All study participants provided informed consent and the study protocols conformed to the Declaration of Helsinki.

Data collection

Certified nurse interviewers, masked to case-control status, administered a comprehensive study questionnaire designed to ascertain information before the date of diagnosis for the cases (and a similarly assigned date for the matched controls). Standardized forms were used to collect the following data: (i) demographic (e.g., age at study visit, ethnicity, marital status, education, religious preference, and lifetime occupation); (ii) medical history

(e.g., physician diagnosed diabetes, hypertension, heart disease, high cholesterol, cancer, and other conditions); (iii) family history (e.g., history of any type of cancer among parents, siblings, and children) and; (iv) lifestyle factors (e.g., current weight, weight 5 years before the reference date, physical activity, history of smoking, and alcohol use). Blood samples were drawn to assess HbA1c, prostate-specific antigen (PSA), and the Duffy blood group, as well as to evaluate several genetic variants.

Anthropometric and other measurements were conducted by trained nurse interviewers, using strict standardized guidelines and reproducibility was regularly monitored within and between observers. Weight was measured to the nearest 0.1 pound with a balance beam scale after participants removed footwear and excess clothing. Height was measured to the nearest 0.1 cm with a metric rule attached to the wall and a right angled wood block. BMI was calculated as the weight (in kilograms) divided by the square of the height in meters. Waist and hip measurements were obtained using steel tapes and were defined as the maximum circumference between the lower ribs and the hip, usually around the area of the navel (waist circumference) and the level of greatest protrusion of the buttocks (hip circumference), respectively. WHR was calculated as waist circumference/hip circumference.

Statistical analyses

Differences in demographic factors between cases and controls were evaluated using *t* tests (for continuous variables) and χ^2 tests (for categorical variables). Age-adjusted logistic regression analyses were conducted for each of the body size factors and further multivariate logistic regression analyses were conducted for the measures of central adiposity, based on significant findings from the age-adjusted results. The abdominal measurement variables were divided into quartiles with values in the lowest (first) quartile serving as the reference group for comparisons. We also stratified prostate cancer cases into low-grade (Gleason score <7) and high-grade (Gleason score \geq 7) categories. The regression analyses were adjusted for age, BMI, family history of prostate cancer, and all demographic variables with *P* < 0.10. These included marital status, religion, occupation, and smoking. Results are presented as ORs and 95% confidence intervals (CI). The Statistical Analysis System (SAS; Institute, Inc.) was used to conduct the analyses.

Results

The PCBP study included 1,007 cases and 1,005 controls. Of those, 963 (96%) cases and 941 (94%) controls self-reported their race as African-Barbadian and represent the basis for this investigation.

Table 1 presents the demographic characteristics for the prostate cancer cases and controls in the study. The mean \pm SD ages of the cases and controls were similar

Table 1. Characteristics of African-Barbadian men from the PCBP study

Demographic factors	Cases (N = 963)	Controls (N = 941)
Age, mean \pm SD (median), y	67.2 \pm 9.0 (68.0)	67.0 \pm 9.2 (67.0)
Religion (%)		
Anglican	48.9	43.0
Pentecostal	11.1	13.4
None	8.2	8.1
Other	31.8	35.5
Marital status, ^a (%)		
Single and never married	17.0	22.3
Married or living together	65.1	58.0
Separated or divorced	11.0	12.8
Widowed	6.9	6.9
Years of education, mean \pm SD (median)	11.6 \pm 3.7 (11.0)	11.3 \pm 3.2 (10.0)
Occupation, ^a (%)		
Professional/administrative/managerial	22.7	19.0
Ever smoke cigarettes/cigars (%)	35.8	39.8
Ever drink alcohol (%)	70.3	68.7
Physical activity level (%)		
Inactive	1.0	1.0
Not very active	7.7	7.8
Somewhat active	23.5	20.7
Very active	67.6	70.6
Hypertension (%)	68.0	68.3
Diabetes history (%)	24.7	25.8
HbA1c	6.5 \pm 1.9 (6.1)	6.4 \pm 2.4 (6.1)

^a $P < 0.05$, χ^2 test.

67.2 \pm 9.0 and 67.0 \pm 9.2 years, respectively. Significant differences ($P < 0.05$) between the groups were found for marital status and lifetime occupation, and marginally significant differences were noted for the distributions of religion ($P = 0.06$) and smoking history ($P = 0.07$). Although cases and controls did not significantly differ with regard to the remaining variables presented in Table 1, it is interesting to note that in this population, more than 90% of men report being physically active (somewhat or very active). In addition, similar proportions of hypertension and reported diabetes were noted among cases and controls, with more than two-thirds of men having hypertension and approximately one-fourth with a history of diabetes. Furthermore, cases were twice as likely to have a family history of prostate

cancer (22.9% vs. 10.4%, $P < 0.0001$) and 47.5% had a Gleason score ≥ 7 .

The distribution of body size variables for study participants is presented in Table 2. Age-adjusted logistic regression analyses indicated that height, as a continuous variable, was not associated with prostate cancer in this population (OR, 1.00; 95% CI, 0.99–1.02). Additional multivariate-adjusted analyses further supported this finding (OR, 0.98; 95% CI, 0.75–1.28) for men in the fourth quartile compared with the reference group (those in the first quartile of height); the results were similarly maintained when low-grade and high-grade disease were considered separately.

Current weight and BMI were also not related to prostate cancer risk in this investigation (OR, 1.00; 95% CI, 1.00–1.00 and OR, 1.00; 95% CI, 0.98–1.02, respectively). Although cases tended to weigh more than controls 5 years before their diagnosis and weigh less during the study visit, the differences between the groups were not statistically significant and the reduced weight (postdiagnosis) in the men with prostate cancer is likely attributable (at least in part) to the disease itself. Cases had a marginally larger waist circumference (on average) than controls (92.8 vs. 91.8 cm, $P = 0.07$) and were significantly more likely to have a larger WHR.

To further evaluate the associations of prostate cancer with measures of central adiposity, the abdominal measurement variables were divided into quartiles and multivariate logistic regression analyses were conducted. As indicated in Table 3, when all cases were considered, a positive association with prostate cancer was found for each increasing quartile increment of waist circumference (P trend = 0.007). Men in the second to fourth quartiles were at significantly increased risk of disease (compared with men with the smallest waist size), such that those having the largest waist circumference (fourth quartile) were found to be at greatest risk (OR, 1.84; 95% CI, 1.19–2.85). A similar pattern was noted among the subset of cases with low-grade prostate cancer, yielding an OR (95% CI) of 1.77 (1.04–3.03) for men in the top quartile of waist circumference (P trend = 0.04). The pattern was less consistent among men with high-grade prostate cancer but the overall direction of the relationship was similar.

Similar to the waist circumference findings, WHR was also positively associated with prostate cancer, regardless of the severity of disease. When all men were considered, a 1.5- to 2-fold increased risk was found for those with larger WHR (compared with the reference group). Risk increased steadily with increasing WHR, yielding significant ORs of 1.41, 1.43, and 2.11 for the second, third, and fourth quartiles, respectively (P trend < 0.0001). Likewise, men with high-grade disease and larger WHR were at increased risk [OR (95% CI): 1.59 (1.11–2.27) and 2.11 (1.42–3.14) for the third and fourth quartiles, respectively; P trend = 0.0001]. When comparing men with low-grade prostate cancer versus controls, those with a WHR between 0.87 and 0.91 (second quartile) were found to be

Table 2. Body size distribution among African-Barbadian men from the PCBP study

Body size characteristics	Cases (N = 963) mean \pm SD (median)	Controls (N = 941) mean \pm SD (median)	Age-adjusted OR (95% CI)
Height, cm	171.5 \pm 6.8 (172.0)	171.4 \pm 6.9 (171.0)	1.00 (0.99–1.02)
Current weight, lbs	167.4 \pm 30.7 (165.0)	168.4 \pm 32.0 (167.0)	1.00 (1.00–1.00)
Weight 5 y prior, lbs	172.0 \pm 30.6 (169.0)	170.8 \pm 32.2 (165.0)	1.00 (1.00–1.00)
BMI, kg/m ²	25.8 \pm 4.3 (25.5)	26.0 \pm 4.5 (25.6)	1.00 (0.98–1.02)
Waist circumference, cm	92.8 \pm 11.5 (92.0)	91.8 \pm 12.2 (91.0)	1.01 (1.00–1.02) ^a
Hip circumference, cm	100.3 \pm 8.0 (100.0)	100.5 \pm 8.8 (100.0)	1.00 (0.99–1.01)
WHR	0.92 \pm 0.07 (0.92)	0.91 \pm 0.07 (0.91)	11.93 (3.05–46.66) ^b

^aP = 0.07.^bP < 0.001.

at increased risk (OR = 1.43; 95% CI, 1.02–2.01), with the strongest association noted for those with a WHR \geq 0.96 (fourth quartile; OR, 1.88; 95% CI, 1.28–2.77).

The multiple logistic regression analyses presented in Table 3 were adjusted for BMI. Since some studies have reported a positive association between tall stature and prostate cancer, we conducted additional analyses adjusting for height (in place of BMI) in these regression models. The results were similar (data not shown). Furthermore, no statistically significant differences between low- and high-grade cancer, with respect to body size, were found and additional analyses using cutoff points based on controls alone (instead of cases and controls

combined, as presented in Table 3) yielded similar findings.

Discussion

The PCBP study represents the largest nationwide case-control study of incident prostate cancer in a predominantly African-origin population to date. The current report investigated the relationship between central adiposity and prostate cancer risk in the Afro-Caribbean population of Barbados, West Indies, and the results indicated a 2-fold positive association between WHR and prostate cancer, regardless of disease grade status. Similar

Table 3. Association of prostate cancer and central adiposity

	All prostate cancer cases ^a (N = 963) OR (95% CI)	High-grade prostate cancer ^a (N = 434) R (95% CI)	Low-grade prostate cancer ^a (N = 480) OR (95% CI)
Waist circumference, cm			
Q1 (<84)	1 (reference)	1 (reference)	1 (reference)
Q2 (84–92)	1.36 (1.01–1.85) ^b	1.34 (0.91–1.97)	1.40 (0.96–2.04)
Q3 (92–99)	1.67 (1.14–2.44) ^b	1.64 (1.01–2.67) ^b	1.54 (0.97–2.46)
Q4 (\geq 99)	1.84 (1.19–2.85) ^b	1.53 (0.86–2.71)	1.77 (1.04–3.03) ^b
Hip circumference, cm			
Q1 (<95)	1 (reference)	1 (reference)	1 (reference)
Q2 (95–100)	1.16 (0.84–1.58)	1.11 (0.75–1.64)	1.33 (0.90–1.97)
Q3 (100–106)	1.18 (0.82–1.69)	1.07 (0.67–1.69)	1.43 (0.92–2.23)
Q4 (\geq 106)	0.90 (0.59–1.39)	0.77 (0.44–1.34)	1.05 (0.62–1.79)
WHR			
Q1 (<0.87)	1 (reference)	1 (reference)	1 (reference)
Q2 (0.87–0.91)	1.41 (1.07–1.85) ^b	1.27 (0.89–1.82)	1.43 (1.02–2.01) ^b
Q3 (0.91–0.96)	1.43 (1.08–1.89) ^b	1.59 (1.11–2.27) ^b	1.27 (0.89–1.82)
Q4 (\geq 0.96)	2.11 (1.54–2.88) ^b	2.11 (1.42–3.14) ^b	1.88 (1.28–2.77) ^b

NOTE: Logistic regression analyses adjusted for age, marital status, religion, occupation, smoking, family history of prostate cancer, and BMI.

^aN = 941 for controls; 49 with unknown Gleason score.

^bP < 0.05.

findings were obtained for waist circumference among all prostate cancer cases.

Waist circumference

There is conflicting evidence in the literature about the relationship between waist circumference and prostate cancer risk. Most studies report some positive association (11, 12, 15–18), whereas 2 investigations found no association (19, 20) and another identified an inverse relationship, with increasing waist circumference resulting in decreased risk (21). Although 2 of the studies reporting a positive association did so for all cases (12, 18), several others report such findings only among a subgroup of participants. For example, the Prostate Cancer Prevention Trial, which included 10,258 men (1,936 prostate cancer cases), indicated that the association was only apparent among men in the placebo arm who had a positive family history of prostate cancer (15). Those in the highest quartile of waist circumference (≥ 108 cm) with a family history had a 2-fold increased risk of disease compared with those with waist circumference < 0.95 (OR, 2.12; 95% CI, 1.10–4.11), whereas men without such history were not found to be at elevated risk. Two additional studies reported the association only among cases with aggressive or advanced disease (16, 17), whereas a third investigation reported that higher waist circumference was associated with increased risk only after adjustment for BMI (11).

A meta-analysis conducted by MacInnis and English (9), including 4 cohort studies and 5 case-control studies, found no association between waist circumference and prostate cancer risk [rate ratio (95% CI), 1.03 (0.97–1.09) for cohort studies; 1.03 (0.98–1.08) for case-control studies; and 1.03 (0.99–1.07) for all studies combined]. A subsequent pooled analysis conducted by Hsing and Sakoda (8) showed similar results. Among 4 cohort studies, 2 overlapping with the MacInnis and English analysis, the pooled relative risk (RR) and 95% CI was 0.94 (0.83–1.06). Likewise, for the 2 case-control studies considered in this report (1 of which overlapped the MacInnis and English analysis), the pooled RR (95% CI) was 1.31 (0.94–1.82). Interestingly, the second case-control study in the pooled analysis (which was not part of the original meta-analysis) included a predominant number of African-American men, representing more than one third of the pooled sample. Although the RR of 1.31 did not achieve statistical significance, the finding, in contrast to previous analyses that included only a limited number of men of African origin, suggests that perhaps waist circumference may play a more significant role in prostate cancer development in populations of African descent, known to have different patterns of visceral fat distribution than other groups.

Of the studies evaluating the relationship of waist circumference with prostate cancer, only 2 have involved men mainly of African origin. The first was included in the pooled analysis by Hsing and Sakoda described earlier. It was a community-based case-control study of African-American men in Michigan that included 139 prostate cancer cases and 359 controls (12) and found that men

with a waist circumference > 102 cm were more likely to have prostate cancer than those with smaller waist sizes (OR, 1.84; 95% CI, 1.17–2.91). In addition, this study also found no association between BMI and prostate cancer. A second hospital-based case-control study in a predominantly African population included 243 cases and 275 controls from Jamaica (11). This investigation did not find an association between waist circumference and prostate cancer among all cases or those with low-grade cancer. However, a 5-fold increased risk of disease was reported for men with high-grade prostate cancer and a waist circumference ≥ 102 cm, as compared with men with a waist circumference < 90 (OR, 5.57; 95% CI, 1.43–18.63), after controlling for BMI in the analysis. BMI was not associated with prostate cancer in this study and the relationship between waist circumference and prostate cancer risk was only statistically significant after adjustment for BMI in the model.

Results from the PCBP study, which included a substantially larger number of participants (963 cases and 941 controls) than previous studies in African populations, further support an association between waist circumference and prostate cancer risk in men of African descent. Among all cases, the risk of developing prostate cancer was 1.84 (95% CI, 1.19–2.85) for those with the highest waist circumferences (≥ 99 cm) compared with the reference group (waist circumference < 84 cm). This finding was consistent with the results from the Michigan study, which also found a 1.8-fold increased risk among men with the largest waist circumferences.

Waist to hip ratio

Findings relating to the relationship between WHR and prostate cancer have been inconsistent. Several studies have indicated that WHR does not influence the development of prostate cancer (8, 9, 15, 16, 19, 22, 23, 24), whereas others report a significant positive association (11, 13, 17, 21). Although some investigations support the relationship between higher WHR measurements and increased prostate cancer risk regardless of disease severity, the association has been shown to be stronger among those with advanced, high-grade disease (13, 17).

A large prospective study of more than 129,000 men from 8 European countries reported a significant association of WHR with advanced prostate cancer (RR, 1.21; 95% CI, 1.04–1.39) but not low-grade cancer (17). Similarly, the North Carolina-Louisiana Prostate Cancer Project, which included 1,049 African-American men and 1,083 Caucasian-American males, found that (overall) men with a WHR > 0.98 (compared with those with a WHR < 0.90) had an increased risk of highly aggressive disease (OR, 1.42; 95% CI, 1.00–2.00; ref. 13). However, when the data were stratified by race, the results showed that larger WHR (taken at the time of interview) was not related to prostate cancer risk in men of African origin (OR, 1.18; 95% CI, 0.76–1.83) but was significantly associated with aggressive cancer in men of European descent (OR, 2.03; 95% CI, 1.10–3.74).

Findings from the North Carolina–Louisiana Prostate Cancer Project highlight potential differences in the relationship between body size and prostate cancer among different racial groups. Although the majority of studies to date have been conducted in primarily European-derived populations, one additional study assessed the relationship between WHR and prostate cancer risk in a population of primarily African descent. The hospital-based case–control study conducted by Jackson and colleagues (11) found that Jamaican men with $\text{WHR} \geq 0.95$ had a 2-fold increased risk of high-grade disease compared with men in the reference range of WHR (OR, 2.02; 95% CI, 1.03–3.96) and a significant association between WHR and prostate cancer was reported among all cases, regardless of disease grade (OR, 1.72; 95% CI, 1.01–3.00).

The PCBP study provides further support for an association between WHR and prostate cancer risk, with more than a 2-fold increased risk for men in the fourth quartile of WHR compared with those in the first quartile. This result was significant for all cases combined (OR, 2.11; 95% CI, 1.54–2.88), as well as those in the high-grade subgroup alone (OR, 2.11; 95% CI, 1.42–3.14). Of note, most of the investigations identifying a positive association between WHR and prostate cancer risk reported the finding only among men with advanced or aggressive disease. Interestingly, while most of these studies were conducted in European-derived populations, the 2 studies including Afro-Caribbean men found a significant relationship among all cases, regardless of disease severity. It is unclear whether this discrepancy is the result of actual differences between Afro-Caribbean men and other groups or whether the finding is confounded by other factors.

To date, the mechanisms through which increased central obesity might be linked to prostate cancer risk are not well understood, though several theories have been proposed. One hypothesis suggests that androgen concentrations may be involved and that because larger abdominal adiposity tends to lower the levels of free and total testosterone, as well as sex hormone binding globulin (SHBG), these alterations may, in turn, stimulate disease development (25). However, the influence of androgens alone does not fully explain the relationship, as evidenced in a study by Platz and colleagues (26) who reported that an increase in total testosterone resulted in a decrease in the risk of high-grade prostate cancer while increasing the risk of low-grade cancer. This finding suggests that the relationship between the androgen pathway and prostate cancer is not straightforward and that disease pathogenesis is likely dependent on tumor type and grade, as well as other factors. A second explanation for the underlying mechanism(s) responsible for prostate cancer development is based on the resulting effects of larger body size on insulin resistance, regulation of leptin, lipid levels, release of free fatty acids, and other metabolic indicators (25, 27). It is likely that these factors, individually and in concert, may also influence risk. Furthermore, diet and lifestyle factors, endogenous and other hormones, and

genetic and environmental interactions are likely to play a role in the complex interrelationship between abdominal adiposity and prostate cancer. Further research is needed to disentangle the mechanisms contributing to the etiology and pathophysiology of prostate carcinogenesis.

Strengths and limitations

The PCBP study represents the largest nationwide sample of incident prostate cancer cases from an African-derived population to date. In addition to its significant sample size, other strengths of the study include its standardized protocols and high participation rates. Although anthropometric measurements were obtained by certified nurse interviewers, one limitation of this study was that these body size measures were taken after diagnosis for the cases, thereby potentially biasing the results. Weight, on average, decreased from 5 years prior (to the study visit) among the cases. This weight loss might also be reflected by smaller waist and hip circumferences, thus possibly influencing the findings related to central adiposity and prostate cancer risk. However, reduction of body mass may be more prominent in the waist than the hip (28), thereby resulting in a lower WHR. Therefore, if the described reductions in body mass were a consequence of the disease itself, the resulting effect would be an underestimate of the risk of disease. In such a case, the associations between WHR and waist circumference with prostate cancer would be even stronger than those reported in this investigation. Given that westernized African populations are known to have different distribution patterns of visceral and subcutaneous fat and that BMI does not reflect body fat distribution, the findings from the PCBP study suggest that measures of central adiposity such as WHR and waist circumference may serve as better predictors of prostate cancer risk than BMI among men of African descent.

Conclusion

Results from this study support a positive association between prostate cancer and both WHR and waist circumference, suggesting that higher central adiposity may influence the development of prostate cancer, regardless of disease severity, in this Afro-Caribbean population. The relationship between body size and prostate cancer is complex, perhaps resulting from influences of the insulin and androgen pathways, hormonal factors, and/or other genetic and environmental contributors. Studies designed to evaluate the relationship between body fat distribution and prostate cancer in populations of African origin have been limited to date; the present findings highlight the need for further investigations in African-derived populations to better elucidate the mechanism(s) by which increased central adiposity may influence disease.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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