Body Mass Index and Prostate Specific Antigen Levels in Jamaican Men

MK Tulloch-Reid[1], WD Aiken[2], BF Morrison[2], T Tulloch[2], R Mayhew[2], RL Wan[3], FI Bennett[4], KC Coard[4], MD Jackson[5]

ABSTRACT

Objective: *To investigate the relationship between body mass index (BMI) and prostate specific antigen (PSA) levels in Jamaican men.*

Methods: *Men, 40−79 years old, attending public and private urology clinics in Kingston, Jamaica were recruited to a case-control study on the role of dietary and lifestyle factors on prostate cancer. Trained interviewers administered questionnaires and measured weight and height using standardized techniques. Blood samples for PSA were measured at a central laboratory using a micro-particle enzyme immunoassay method. Prostate biopsy was used to confirm prostate cancer. Multivariable linear regression was used to examine the relationship between BMI and PSA separately in the cases and controls.*

Results: *Data from 501 men (233 cases and 263 controls) were assessed. Thirty-five per cent of subjects were overweight and 13% were obese. Among cases, the median PSA was 35.3 ng/dL in normal weight, 26.1 ng/dL in overweight and 14.5 ng/dL in obese men (*$p = 0.02$*). For controls, median PSA was 2.0 ng/dL in normal weight, 1.3 ng/dL in overweight and 1.1ng/dl in obese men (*$p = 0.01$*).*

Among cases, BMI was negatively associated with PSA (B(SE) per 5 kg/m^2 (BMI difference = -0.51 (0.13); $p < 0.01$*) and remained significant after adjustment for age, sexual activity, smoking, use of statins and tumour grade. For controls, the BMI was also inversely related to the PSA (B(SE) per 5 kg/m^2 difference -0.17(0.07)) but the effect became of borderline significance after adjusting for age.*

Conclusions: *Prostate specific antigen was inversely related to body mass index in Jamaican men with prostate cancer. Clinicians should consider this association when interpreting PSA results.*

Keywords: Black, Caribbean, prostate cancer, PSA, obesity

Índice de Masa Corporal y Niveles de Antígeno Prostático Específico en los Hombres Jamaicanos

MK Tulloch-Reid[1], WD Aiken[2], BF Morrison[2], T Tulloch[2], R Mayhew[2], RL Wan[3], FI Bennett[4], KC Coard[4], MD Jackson[5]

RESUMEN

Objetivo: *Investigar la relación entre el índice de masa corporal (IMC) y los niveles de antígeno prostático específico (PSA) en los hombres jamaicanos.*

Métodos: *Hombres en edades de 40–79 años, que asistían a clínicas de urología privadas en Kingston, Jamaica, fueron reclutados para un estudio de caso-control sobre el papel de los factores dietéticos y el estilo de vida en el cáncer de próstata. Entrevistadores especializados administraron las encuestas y midieron el peso y la altura usando técnicas estandarizadas. Las muestras de sangre para PSA fueron medidas en un laboratorio central usando como método de inmunoensayo enzimático de micropartícula. La biopsia de la próstata fue usada para confirmar el cáncer de la próstata. Se usó la regresión multivariable lineal para examinar por separado la relación entre IMC y PSA en los casos y los controles.*

Resultados: *Se evaluaron los datos de 501 hombres (233 casos y 263 controles). Treinta y cinco por ciento de los sujetos tenían sobrepeso y el 13% eran obesos. Entre los casos, el PSA promedio fue 35.3*

From: [1]Epidemiology Research Unit, TMRI, The University of the West Indies, [2]Department of Surgery, Radiology, Anaesthesia and Intensive Care, The University of the West Indies, [3]Department of Surgery, Kingston Public Hospital, [4]Department of Pathology, The University of the West Indies and [5]Department of Community Health and Psychiatry, The University of the West Indies, Kingston 7, Jamaica.

Correspondence: Dr M Tulloch-Reid, Epidemiology Research Unit, TMRI, The University of the West Indies, Kingston 7, Jamaica. E-mail: marshall.tullochreid@uwimona.edu.jm

*ng/dL en el peso normal, 26.1 ng/dL en el sobrepeso, y 14.5 ng/dL en los obesos (*p = *0.02). En los controles, el PSA promedio fue 2.0 ng/dL en el peso normal, 1.3 ng/dL en el sobrepeso y 1.1 ng/dl en los obesos (*p = *0.01).*

Entre los casos, el IMC estaba asociado negativamente con el PSA (B(SE) por 5 kg/m2 (diferencia de IMC = -0.51(0.13); p < *0.01) y permaneció significativo después del ajuste por edad, actividad sexual, hábito de fumar, uso de estatinas, y grado de tumor. En los controles, el IMC también estaba inversamente relacionado con el PSA (B(SE) por 5 kg/m2 de diferencia -0.17 (0.07)) pero el efecto alcanzó importancia significativa limítrofe tras el ajuste por edad.*

Conclusiones: *El antígeno prostático específico guarda una relación inversa con el índice de masa corporal en los hombres jamaicanos con cáncer de próstata. Los médicos deben considerar esta asociación al interpretar los resultados del PSA.*

Palabras claves: Hombre negro, caribeño, cáncer de próstata, PSA, obesidad

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INTRODUCTION

Prostate cancer is the most common cancer affecting Jamaican men with an estimated annual incidence of 78.1/100 000 men/year (1). It is also the most common cause of male cancer-related deaths in Jamaica (2). Since the introduction of prostate specific antigen (PSA) testing in Jamaica in 1991, an increasing number of prostate cancers are being diagnosed on the basis of evaluation of an abnormal PSA level rather than symptoms associated with the presence of locally advanced or metastatic disease.

A number of studies have reported an inverse relationship between PSA and body mass index (BMI), with obese men having lower PSA values compared to non-obese men (3−7), though this relationship has not been consistently demonstrated (8). The relationship between BMI and PSA may be modified by age and ethnicity. Kim *et al* (9) reported that in Korea the inverse relationship between PSA and BMI was significant only in men under 60 years old, while among US men, the inverse relationship between BMI and PSA was driven primarily by non-Hispanic white men (4, 10). One study has demonstrated an inverse association between PSA and BMI in a community-based sample of African-American men (11). All of these studies have been conducted in men without diagnosed prostate cancer.

The two most commonly proposed explanations for the inverse relationship between BMI and PSA are the hormone hypothesis (12, 13) and the dilution hypothesis (14) but it could also be the result of confounding. For instance, as BMI increases, patients are more likely to have hypercholesterolaemia requiring statin therapy – which has been demonstrated to lower serum PSA (15, 16). We are not aware of any studies which have explored the role of this potential confounder in the PSA-BMI relationship.

In Jamaica, where approximately 90% of men are of African descent, there is a high prevalence of overweight (25.8%) and obesity (12.4%), and 7.5% of adult men have an elevated total cholesterol (17). Given the local prevalence of prostate cancer, overweight/obesity and hypercholesterolaemia in this population, if an inverse relationship between PSA and BMI were present, it would have implications for the interpretation of PSA results in men. We therefore explored the relationship between PSA and BMI in Jamaican men with and without prostate cancer. We also examined whether the relationship between BMI and PSA was independent of potential confounders, particularly statin use.

SUBJECTS AND METHODS

Between March 2005 and July 2007, men 40−79 years old attending urology clinics at two tertiary referral centres or the private offices of urologists practising in Kingston were recruited into a case control study examining the effects of dietary and lifestyle factors on the risk of prostate cancer. Approval for the study was granted by the Faculty of Medical Sciences/The University of the West Indies and the Ministry of Health Ethics Committees. Informed consent was obtained from each patient prior to enrolment. Men who were on 5-alpha reductase inhibitors, prior or current hormone therapy or who had previous prostate surgery, advanced metastatic cancer and/or severe weight loss were excluded from the study.

Questionnaires were administered to study participants by trained interviewers prior to any knowledge of their disease status. Information was collected regarding demographics, sexual practices, medication history, smoking and family history of prostate cancer. Weight was measured using an electronic digital scale and height assessed using a portable height measurement rod after positioning the patient's head in the Frankfort Plane. The body mass index was calculated as the weight in kilograms divided by the square of the height in metres and subjects classified as normal weight, overweight or obese using the World Health Organization criteria (18). The waist circumference was measured at the widest circumference between the lowest rib and the anterior superior iliac crest using a non-stretchable tape. All measurements were performed with the patients wearing light clothing.

The serum PSA was measured using a micro-particle enzyme immunoassay method (Abbott IMX). Each participant was examined by a urologist and transrectal ultrasound-guided biopsy was performed on those men with an

abnormal digital rectal examination (DRE) and/or abnormal PSA. A case was defined as a man 40 to 79-years old with a newly diagnosed, histologically confirmed prostate cancer. Cases were graded according to the Gleason scoring system (19). Controls were initially defined as similarly aged men with a benign-feeling prostate on DRE and PSA less than 2 ng/ml. However, as there was difficulty recruiting sufficient numbers of controls from urology clinics and specialists' offices who met the stringent criteria, patients with PSA between 2 to 4 ng/ml and a free /total PSA ratio of ≥ 0.15 were also utilized as controls.

Subjects were grouped according to their disease status and their characteristics according to BMI category were determined. Means and Standard Deviations were calculated for continuous variables, except for the PSA where the median and 25th to 75th centiles are presented as it was not normally distributed. The percentage distribution for categorical variables was determined. The Kruskal-Wallis and the Fishers Exact Tests were used to compare differences across BMI categories.

The log transformed serum PSA was used as the dependent variable in linear regression analysis to explore the relationship between PSA and BMI. Separate models for participants according to their cancer status were utilized as there was evidence of interaction in the PSA-BMI relationship by cancer status. Variables considered to be related to serum PSA and BMI were included in the regression models. Patient age and Gleason score were entered as continuous variables, while sexual frequency (less than once per month, 1−3 times per month or 1 or more times per week), union status (married/cohabiting *versus* single/ widowed), smoking (never smoked, current smoker, ex-smoker) and statin use (yes, no) were entered as categorical terms in multiple regression analysis models. Results for the effect of BMI on the log transformed PSA are presented for a 5 kg/m^2 difference. Analysis was performed using Stata 8.0 (Stata Corporation, TX).

RESULTS

Of the 518 persons enrolled, 501 (233 cases and 263 controls) had information on all the characteristics of interest. There were no significant differences in age, central obesity, marital status or education between those included in this analysis and those with missing data. The characteristics of subjects according to their cancer and BMI status are presented in Tables 1A and 1B.

Among the cases, there were no significant differences in age or height according to the BMI category. As expected, mean weight and waist circumference increased with BMI ($p < 0.01$ for both). Obese participants were less likely to be current smokers than their non-obese counterparts. There was no difference in the use of apha-adrenergic blocker medications for prostate hypertrophy, reported sexual frequency, statin use or family history of prostate cancer by BMI category. The median PSA was lower for each increasing BMI category in men with prostate cancer, with a median PSA of 35.3 ng/dL in the normal weight, 26.1 ng/dL in the overweight and 14.5 ng/dL ($p = 0.02$) in the obese (Table 1A).

Table 1A: Characteristics of patients by body mass index (BMI) category

Patient Characteristic	Body Mass Index Category Normal	Overweight	Obese	*p* Value
Prostate Cancer – Cases	n = 129	n = 75	n = 29	
Age (years): mean ± SD	68.2 ± 7.7	67.2 ± 7.7	68.7 ± 8.6	0.42*
Proportion married or cohabiting	56	68	82	0.06†
Sexual frequency (%)†				
< 1 per month	46	47	52	0.98†
1 – 3 per month	31	29	28	
> 1 per week	23	24	20	
Smoking: %†				
Non-smokers	17	28	19	< 0.02†
Ex-smokers	63	60	81	
Current smokers	20	12	0	
Statin use (%)	2	6	4	0.22†
Family history of prostate cancer (%)	17	16	10	0.79†
Weight (kg): mean ± SD	63.6 ± 7.9	78.3 ± 7.7	96.1 ± 9.7	< 0.01*
Height (cm): mean ± SD	170 ± 7.0	169 ± 7.0	169 ± 5.0	0.45*
Waist (cm): mean ± SD	80.0 ± 7.0	94.0 ± 7.0	107.0 ± 7.0	< 0.01*
PSA (ng/dl) Median (25th –75th centile)	35.3 (14.0 – 214.0)	26.1 (13.8–67.9)	14.5 (7.7–37.1)	0.02*

*p value for Kruskal-Wallis Test, †Fishers Exact Test

Among the controls, similar relationships were seen between BMI and anthropometric measurements, medication use, reported sexual frequency and family history of prostate cancer (Table 1B). Smoking was also less common in the obese men. As was observed among the cases, the median PSA was lower in the more obese participants with values of 2.0 ng/dL in the normal weight, 1.3 ng/dL in the overweight and 1.1 ng/dL ($p = 0.01$) in the obese controls (Table 1B).

The regression analyses for models of PSA and BMI according to cancer status are presented in Table 2. For the cases, BMI was inversely related to PSA (B (SE) for 5 kg/m^2 difference = -0.51 (0.13); $p < 0.01$). This relationship remained unchanged after adjustment for age, smoking, statins or reported sexual frequency. Adjustments for Gleason score and union status attenuated the relationship between PSA and BMI; however, the relationship remained

Table 1B: Characteristics of patients by body mass index (BMI) category

Patient Characteristic	Body Mass Index Category Normal	Overweight	Obese	*p* Value
Prostate Cancer – Controls	n = 136	n = 97	n = 35	
Age (years): mean ± SD	62.6 ± 10.4	61.3 ± 11.1	59.7 ± 10.4	0.30*
Proportion married or cohabiting (%)	57	66	74	0.29†
Sexual frequency (%)				
< 1 per month	45	33	29	0.38†
1 – 3 per month	27	31	32	
> 1 per week	28	36	39	
Smoking (%)				
Non-smokers (%)	23	24	28	< 0.05†
Ex-smokers (%)	57	61	72	
Current smokers (%)	20	15	0	
Statin use (%)	2	6	10	0.41†
Family history of prostate cancer (%)	12	11	9	0.86†
Weight (kg): mean ± SD	64.7 ± 8.9	79.8 ± 7.6	98.2 ± 11.2	<0.01*
Height (cm) mean ± SD	172 ± 7.0	171 ± 6.0	172 ± 7.0	0.76*
Waist (cm) mean ± SD	79.0 ± 7.0	93.0 ± 7.0	107.0 ± 12.0	<0.01*
PSA (ng/dl): Median (25th – 75th centile)	2.0 (0.95 – 3.7)	1.3 (0.6-2.6)	1.1 (0.6-2.7)	0.01*

*** *p* value for Kruskal-Wallis Test, † Fishers Exact Test**

Table 2: Regression models examining the effect of BMI on log transformed PSA levels in prostate cancer cases and controls with adjustment for possible confounders.

	Cases Beta Coefficient (SE) for BMI*	*p* value	Controls Beta Coefficient (SE) for BMI*	*p* value
Model 1 – BMI	-0.51 (0.13)	< 0.01	-0.17 (0.07)	0.01
Model 2 – BMI + Age	-0.50 (0.13)	< 0.01	-0.11 (0.06)	0.08
Model 3 – BMI + Age + Union Status	-0.45 (0.12)	< 0.01	-0.11 (0.06)	0.06
Model 4 – BMI + Age + Sexual Frequency	-0.51 (0.13)	< 0.01	-0.07 (0.06)	0.26
Model 5 – BMI + Age + Smoking	-0.50 (0.14)	< 0.01	-0.12 (0.06)	0.05
Model 6 – BMI + Age + Statin	-0.50 (0.13)	< 0.01	-0.11 (0.06)	0.07
Model 7 - BMI + Age + Gleason Score	-0.41(0.11)	< 0.01		
Model 8 – BMI + Age + Gleason Score + Union Status	-0.35 (0.11)	< 0.01		

*** Coefficients and Standard Errors are presented for per 5 kg/m^2 difference in BMI**

Age, BMI and Gleason score entered as continuous variables, sexual frequency – less than once per month, 1 – 3 times per month or 1 or more times per week, union status – married/cohabiting *vs* single/visiting relationship/widowed, smoking – never smoked, current smoker, ex-smoker and statin use – yes or no entered as categorical variables

significant – B(SE) for a 5 kg/m^2 difference in BMI of – 0.35 (0.11).

Among controls, the regression coefficient for a 5 kg/m^2 difference in BMI was -0.17(0.07); however, this was no longer statistically significant after adjusting for age [B(SE) = -0.11(0.06)]. Further adjustment for the other potential confounders did not affect the regression coefficient which remained of borderline significance (Table 2).

Age did not modify the relationship between BMI and PSA in the cases or controls.

DISCUSSION

The relation of BMI to PSA has not received much attention in populations of African origin. This study demonstrates that in a population of predominantly African heritage, PSA declines with increasing BMI among men with prostate cancer. The adjustment for confounders that included age, sexual activity, smoking status and statin use did not alter this relationship, though the patient's union status and Gleason score attenuated the relationship. Among controls, a weaker inverse relationship was noted; however, the strength of the association was reduced with adjustment for age and other potential confounders and was no longer statistically significant.

Most studies of BMI and PSA have examined the relationship in persons without prostate cancer (3−5, 10). It has been shown that persons with higher BMIs are more likely to have advanced or metastatic cancer (20) and therefore, it would be expected that the obese patient would have a higher PSA value. While adjusting for tumour grade (Gleason score) attenuated the relationship between PSA and BMI, it did not remove or reverse it. Our finding of a strong independent, inverse relationship between BMI and PSA in men with newly diagnosed prostate cancer is consistent with a retrospective study of patients referred, for radical prostatectomy, to two large referral centres in the United States of America (21). Marital status also attenuated the BMI and PSA relationship and its effect appeared to be independent of reported frequency of sexual activity. That more obese participants were married or cohabiting may contribute to this association but the role of union status on PSA levels is not clear.

We also found a negative association between BMI and PSA among controls. This relationship lost significance after adjusting for age. Most studies that have demonstrated this relationship have done so in populations containing a significant number of non-Hispanic white men (4), though the relationship has also been demonstrated in middle aged Korean men (9). While two cross-sectional studies of BMI and PSA did not suggest that race significantly affected this relationship, neither presented the race-specific PSA values according to BMI status (4, 10). From analysis of NHANES data where race-specific BMI and PSA data are presented, the BMI-PSA relationship was only significant in the non-Hispanic white men but not the African-American or Hispanic men (10). In a cross-sectional study of 9000 Korean men, the BMI-PSA relationship was only significant in men under 60 years old (9). Only the study of African-American men has demonstrated an inverse relationship between BMI and PSA (11). We believe that the absence of an association among controls in our study may reflect the narrow range of PSA values used as the criteria for their selection and inadequate statistical power to demonstrate a significant relationship within this narrow range. Our findings are consistent with most studies which have examined the relationship between BMI and PSA among men of African origin without prostate cancer and suggest that the BMI-PSA relationship may not be as strong in men of African origin compared with men of other ethnic origins.

One limitation of this study is the absence of prostate volume measurements to determine whether differences in BMI are associated with prostate size. Joseph *et al* (12) suggested that prostate volume increases with BMI. Prostate volume has been shown to be positively associated with PSA levels (20) suggesting that PSA should increase with increasing BMI. The association that is generally demonstrated is in direct contrast to these findings. It should be noted that in other studies where prostate volume has been measured, increasing BMI was not associated with an increased prostate volume (20).

The two most commonly proposed explanations for the inverse relationship between BMI and PSA are the hormone hypothesis and the dilution hypothesis. In the first hypothesis, the peripheral aromatization of testosterone to oestrogens in adipose tissue is believed to result in lower testosterone, higher oestrogen concentrations and lower sex-hormone binding globulin which together result in diminished production of PSA (13, 22). With the dilution hypothesis, BMI is associated with a higher plasma volume which results in a lower PSA concentration (23). More recent studies, using plasma volume measurements based on anthropometric measurements, support the latter hypothesis (14, 21, 23). A dilutional effect with other tumour markers due to increased plasma volume with higher BMI has also been demonstrated (14).

Statin therapy is an important confounder in the BMI and PSA relationship, as statin therapy, more common in the obese patient, lowers the serum PSA (15, 16). None of these studies we reviewed examined the impact of this factor on the BMI-PSA relationship. There was a very low prevalence of statin use in our study population and so adjusting for statin therapy made no difference to the association in Jamaican men and could not be considered to be an important confounder in this study.

The findings of the present study support the importance of considering BMI when interpreting non-PSA readings in patients with and without prostate cancer. This association has important im-plications for Jamaica, a country with a high incidence of and mortality from prostate cancer and where approximately one-half of adult men are

obese or overweight. This inverse relationship between PSA and BMI may negatively impact the use of PSA for the early detection of prostate cancer. A lower serum PSA in obese men is likely to delay prostate cancer detection. Furthermore, obese men tend to have a more difficult DRE and poor diagnostic yield from biopsies (20).

Some authors have suggested the use of formulae to adjust PSA values for differences in plasma volume due to BMI, in order to prevent the under diagnosis of prostate cancer in the obese and the unnecessary evaluation of elevated PSA levels in the less obese men (23). The utility of this approach in our setting is not known. The per cent of free to total PSA may be less affected by anthropometric differences and could be an alternative measurement (11). Unfortunately, we did not have this measurement performed in enough participants to determine whether this would be a useful alternative in our population.

In conclusion, this study was able to demonstrate significantly lower PSA levels in men with a higher BMI, particularly in patients with prostate cancer. Clinicians should consider this relationship when interpreting PSA results for their patients. Further research on how to compensate for this relationship, when interpreting PSA results in Afro-Caribbean men, is required.

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