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Original Article

Risk Factors of Prostate Cancer Among Patients Diagnosed at the University College Hospital, Ibadan, Nigeria: A Case-Control Study

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Abstract

Background and aims: Prostate cancer (PCa) is the second most commonly diagnosed cancer in men and its incidence is higher among black folks for reasons yet unclear. Although few risk factors have been linked to the development of PCa among Nigerian men, it remains unclear whether these risk factors modulate the odds of PCa.

Methods: The case-control study comprised forty-three cases and one hundred and twenty-nine age-matched controls (±5 years) without PCa by prostatic specific antigen (PSA) examination. Conditional logistic regression analysis was employed to identify risk factors associated with PCa at $P<0.05$ using SPSS 20.

Results: Increasing age (adjusted odds ratio [AOR]: 2.8; 95% CI: 1.7-4.5) was the strongest risk factor for PCa, followed by increasing age at first sex (AOR: 1.2; 95% CI: 1.1-1.4) and sexual activity (AOR: 0.2; 95% CI: 0.1-0.9) as a protective risk factor against PCa in this Nigerian Population.

Conclusion: The study confirms age as a recognized risk factor and backed evidence for other hypothesized risk factors. The study recommends findings with other confirmatory studies that can help to guide policies for better health care decisions among Nigerian men and interventions centered on routine screening for PSA with an emphasis on the elderly clinic is encouraged.

Keywords: Risk, Prostate, Cancer, Case-control, Nigerian

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Introduction

The prostate is an organ that covers the prostatic urethra and is located below the bladder[1]. There are ways to cause medical problems for the prostate and they include; enlarged prostate, prostatitis, and prostate cancer (PCa).[1]

PCa is the fourth major cancer globally, accounting for 1.3 million (7.1%) of the overall burden of cancer incidence.[2] It is the second most frequently diagnosed form of cancer in men, accounting for 13.5% of the overall incidence of cancer, and also ranks second in both developed and developing countries in incidence.[2] The number of new cases ranges by more than 50 times, with the highest rates occurring in North America, Australia, and North/Central Europe; in Southeast and South-Central Asia and Northern Africa, the lowest rates occur.[3] Studies have shown that the burden of PCa in developing communities in southwestern Nigeria is prevalent among ages between 46-99 years with a peak incidence in the ≥70 years age group and a prevalence of 1.05%.[4,5]

The risk factors of PCa can be broadly categorized into modifiable and non-modifiable. The non-modifiable factors include increasing age, black race, and family history of PCa.[6,7]. Some modifiable risk factors have been identified and they include lifetime alcohol consumption,[8] obesity,[9] smoking,[10] sedentary lifestyle,[11] prostatitis history,[12] and high level of cholesterol.[13] Earlier studies have shown that obese men with a body mass index (BMI) greater than 30 kg/m^2 are more likely than men with a BMI lower than 25 kg/m^2 to develop PCa.[9,14,15]

Obesity is associated with food eating patterns that tend to play a key role in the development of PCa.[14] Dietary factors such as fish, high vegetable intake, vitamins C and E are protective for PCa.[16] A decrease in the risk of PCa has been observed among men who had a high intake of tomatoes and garlic.[17,18] Conversely, age is a key risk factor in the development of PCa, and its incidence rate increases with age. The reason behind this could be increased oxidative stress which results in the onset of PCa as age increases.[19,20] Cigarette smoking is another potential risk factor for PCa development owing to changes in hormones. Smokers have increased levels of testosterone and androsterone and may be involved in cancer progression.[21]

Data collected from the GLOBOCAN program of the International Agency for Research on Cancer (IARC) show that PCa is also the leading cancer in sub-Saharan African men, accounting for 14% of all cancer diagnoses and 12% of all cancer-related deaths[22-24] Nigerian men

suffer the highest burden from PCa in West Africa with estimated yearly age-adjusted incidence and mortality rates of 23.3 and 19.2 per 100 000 respectively. This accounts for 18.2% and 17.7% of all cancer-related diagnoses and mortality respectively, in men in this region.[23-26] Nigeria being the most populous country in Africa, the rates and percentages above translate to a huge burden in absolute numbers of men affected by PCa.

A systematic review of PCa research in Nigeria showed that the earliest publication on PCa dates to 1973.[27] This study reported a low incidence at this time, but in 1980 the incidence rate reported in this Nigerian population was similar to other black men in Washington. However, further reports have shown an increasing prevalence of PCa. Aside from this, there is a global focus on PCa differences among black men, and this demands more efforts from Africa.[27] However, few risk factors have been linked to PCa development in this population. These include having a first-degree relative with PCa, decreasing height, decreasing weight, and decreasing waist circumference.[28] It remains unclear whether these risk factors modulate the odds of PCa. Thus, our study was set out to assess risk factors of PCa among patients diagnosed at the University College Hospital, Ibadan.

Materials and Methods

Study Design

A case-control study design was employed for this study.

Study Population

The cases comprised incident PCa patients histologically confirmed either by biopsy, immunochemistry markers or microscopic examination admitted and/or going through treatment at the University College Hospital, Nigeria during the study period from December 2019 to March 2020. The controls were men who did not have a prior history or symptoms related to PCa recruited from orthopedic, ophthalmologic, and hypertensive clinics at the same ascertainment period as the cases and were age-matched by a factor of three making the cases and controls similar in age. Study criteria included cases whose diagnosis of PCa was within two years period before study contact and must never have been interviewed as a control for the study. Controls underwent prostatic specific antigen (PSA) screening established by reading of < 3 ng/mL to reduce ascertainment bias,[29] was suitable to be matched to a case by age (± 5 years),[10,30] participants who were of Nigerian descent and were physically and mentally capable of performing the interview. The study excluded cases who were severely ill, unable to give informed consent, controls who had a personal history of cancer, and controls who had a history of radiation therapy or chemotherapy. To control for Berkson's bias, cases were patients whose primary diagnosis for hospitalization was PCa.

Sample Size

A total of 43 cases and 129 controls were estimated using the formula for case-control studies case-control studies with unequal group sizes.

Sampling Method

Consecutive sampling was employed: Every patient that met the inclusion criteria for the study was recruited until the required sample size was achieved

Data Collection Tool

The questionnaire collected information on socio-demographics, cancer history, lifestyle factors, anthropometry and occupational exposure. Medical history of other diseases such as hypertension and diabetes mellitus were collected by reviewing medical records. Face validation of the questionnaire was done by the study supervisors, translated into Yoruba, then pretested at the University College Hospital, Ibadan and preliminary analyses including reliability tests for each section of the questionnaire were carried out.

Data Collection Procedure

Medical records were reviewed weekly to identify eligible cases and controls and their appointment dates. A case was enrolled first during their urological visits, then moved to look for corresponding controls, explained the purpose of the study and interviewed them thereafter. All enrolled participants completed a pre-tested structured questionnaire with an interviewer to obtain the study's exposure variables. Data on dietary intake for food frequency was taken with the aid of a show-card and the physical activity level was assessed by adopting the International Physical Activity Questionnaire (IPAQ). Blood samples were collected under aseptic condition once by pricking from the finger among the controls for rapid PSA examination.

Statistical Analysis

Statistical Package for Social Sciences (SPSS 20) was employed for the statistical analyses. Descriptive statistics including mean, standard deviation, frequency, and proportion were used to summarize the characteristics of the cases and their controls. Chi-square and independent-sample t-tests were used to determine the difference between the cases and their controls for categorical and continuous variables as appropriate. Normality of continuous variables was assessed using histogram, Q-Q plots, and Shapiro-Wilk test. Missing data were eliminated using pairwise deletion and regression analysis. Analytically, conditional logistic regression was used to estimate the odds ratio for effect size at 90% and 95% confidence intervals (CIs) for conditional univariate and multivariable logistic regressions respectively. Data analyses were in two stages; the exploratory stage identified potential risk factors defined by variables associated with PCa at $P < 0.1$ in a univariate logistic regression. Adjusting for confounders, all potential risk factors retaining a significant association with PCa in a

multivariable logistic regression model at $P<0.05$ were identified as related factors. These related factors were selected using the ENTER method (i.e., all variables were introduced and variables whose coefficients were not significant were eliminated).

Results

The distribution of the socio-demographic characteristics and cancer history of the respondents shows that a proportion of 18(41.9%) of the cases reported being < 70 years while 71(55%) of the controls reported being < 70 years alike. The mean ages for the cases and controls were 70.3 ± 8.0 years and 67.4 ± 8.7 years respectively and there was no significant difference in the mean ages of cases and controls (t test = 1.881 and P value = 0.062) as shown in Table 1.

Lifestyle and Anthropometric Characteristics of the Respondents

The distribution of lifestyle and anthropometric characteristics of cases and controls shows that 14 (32.6%) had ever smoked tobacco among the cases, similarly 46 (35.7%) of the controls had ever smoked tobacco. The mean ± SD of cigarettes smoked per day by the cases was 2.8 ± 2.3 sticks/day and 5.2 ± 6.3 sticks/day by the controls. A proportion of 32 (72.6%) cases revealed they have ever consumed any alcoholic drink while a proportion of 86 (66.7%) of controls reported having ever consumed any alcoholic drink. The mean ± SD of bottles drunk/day for the cases was 3.5 ± 4.0 and 2.7 ± 2.8 for the controls as shown in Table 2.

Dietary, Physical Activity and Occupational Exposure of Respondents

Data on dietary intake reveal that among the controls, 16 (38.1%) reported having consumed fruits for two days/week. Similarly, in the control, the majority 49 (38%) reported having consumed fruits for two days/week. Among those cases who reported having consumed fruits, the majority 17 (43.6%) reported having consumed two servings per intake, while among the majority of the controls 57 (45.2%) reported having consumed two servings per intake alike. On vegetable intake, the majority 17 (40.5%) of the cases reported having eaten vegetables for ≥ 4 days/week, while the majority 46 (46.1%) of the cases reported having eaten vegetables for three days/week as shown in Table 3.

Risk Factors of Prostate Cancer

Univariate comparisons between cases and controls at $P<0.10$ identified potential risk factors for PCa including; age, employment status, family history of cancer, family history of PCa, first degree relative with PCa, intake of a local concoction made with alcohol, age at first sex, sexual inactivity, fruit intake, vegetable intake, serving per vegetable intake, additional salt intake, and aromatic-amine related occupations. Age had a significant positive

Table 1. Frequency Distribution of Socio-demographic Characteristics and Cancer History of the Respondents

Variables	Cases = 43 n (%)	Controls = 129 n (%)	χ^2	*P* Value
Age (y)			2.243	0.134
< 70	18 (41.9)	71 (55)		
≥ 70	25 (58.1)	58 (45)		
Income (Naira)			0.229	0.892
< 50000	11 (30.6)	17 (34)		
50000-99999	11 (30.6)	16 (32)		
≥ 100000	14 (38.9)	17 (34)		
Ethnicity			2.855	0.240
Yoruba	35 (81.4)	116 (89.9)		
Igbo	5 (11.6)	6 (4.7)		
Others	3 (7)	7 (5.4)		
Education			5.828	0.120
No formal education	4 (9.3)	5 (3.9)		
Primary	4 (9.3)	25 (19.5)		
Secondary	16 (37.2)	32 (25)		
Tertiary	19 (44.2)	66 (51.6)		
Occupation			2.204	0.531
Government employees	21 (48.8)	57 (45.2)		
Business men	17 (39.5)	61 (48.4)		
Army	3 (7)	6 (4.8)		
Farmer	2 (4.7)	2 (1.6)		
Marital status				
Single	3 (7.3)	4 (3.3)		
Married	36 (87.8)	109 (90.8)	1.865	0.601
Divorced	0 (0)	2 (1.7)		
Widowed	2 (4.9)	5 (4.2)		
Religion				
Christian	32 (78)	92 (72.4)		
Muslim	8 (19.5)	32 (25.2)	4.619	0.202
Traditional	0 (0)	3 (2.4)		
Others*	1 (2.4)	0 (0)		
Employment status				
Self employed	12 (27.9)	45 (35.2)		
Currently employed	5 (11.6)	25 (19.5)	3.158	0.206
Retired	26 (60.5)	58 (45.3)		
Family history of cancer			4.835	0.028[c]
Yes	10 (23.3)	13 (10.1)		
No	33 (76.7)	116 (89.9)		
Family history of PCa			5.080	0.024[c]
Yes	7(16.3)	7(5.4)		
No	36(83.7)	122(94.6)		
First degree relative with PCa[a]			5.080	0.024[c]
Yes	7(16.3)	7 (5.4)		
No	36(83.7)	122 (94.6)		
Second degree relative with PCa[b]			0.675	0.411
Yes	1 (2.3)	1 (0.8)		
No	42 (97.7)	128 (99.2)		
Family history of breast cancer			0.000	1.000
Yes	1 (2.3)	3 (2.3)		
No	42 (97.7)	126 (97.7)		

*Atheist; [a] Brother, father, son; [b] Uncle, cousin, nephew; [c] significant at $P<0.05$

Table 2. Frequency Distribution of Lifestyle and Anthropometric Characteristics of the Respondents

Variables	Cases (n=43) No. (%)	Controls (n=129) No. (%)	χ^2	*P* Value
Smoking of tobacco				
Ever	14 (32.6)	46 (35.7)	0.137	0.712
Never	29 (67.4)	83 (64.5)		
Current smokers				
Yes	0 (0)	7 (5.4)	2.432	0.119
No	43 (100)	122 (94.6)		
Regular contact with smokers				
Yes	6 (14.3)	22 (17.1)	0.177	0.674
No	36 (85.7)	107 (82.9)		
Alcohol consumption[a]				
Ever	32 (76.2)	86 (66.7)	1.344	0.246
Never	10 (23.8)	43 (33.3)		
Local concoction with alcohol[b]				
Yes	12 (28.6)	55 (42.6)	2.630	0.105
No	30 (71.4)	74 (57.4)		
Current alcohol consumers				
Yes	9 (21.4)	18 (14)	1.331	0.249
No	33 (78.6)	111 (86)		
Alcohol intake (last 30 days)				
Yes	4 (9.5)	15 (11.6)	0.142	0.706
No	38 (90.5)	114 (88.4)		
Currently sexually active				
Yes	13 (32.5)	86 (66.7)	14.690	<0.001[c]
No	27 (67.5)	43 (33.3)		
Duration of sexual inactivity				
<12 months	4 (15.4)	3 (13.6)	0.029	0.864
≥12 months	22 (84.6)	19 (86.4)		
BMI (kg/m^2)				
<25	11 (61.1)	7 (36.8)	2.179	0.14
≥25	7 (38.9)	12 (63.2)		

[a] Beer, spirit, palm wine, burukutu, etc.,; [b] Jedi-Jedi, opa-eyin, iba; [c] Significant at $P<0.05$.

association with case status. That for every unit increase in age, the cases were 3.2 times more likely to develop PCa than their controls (odds ratio: 3.2; 90% CI: 2.2-4.6). To control for residual age confounding and other potential confounding effects between the potential risk factors, multivariable conditional regression analysis was conducted. In this model after adjusting for age at first sex and sexual activity, it showed that for every unit increase in age there were 179.7% excess odds of developing PCa among the cases. After adjusting for age and sexual activity, for every unit increase in the age at first sex there were 17.4% excess odds of developing PCa among the cases and after adjusting for age and age at first sex, sexually active men had 79.5% lower odds of developing PCa than sexually inactive men as shown in Tables 4 and 5.

Table 3. Frequency Distribution of Dietary, Physical Activity and Occupational Exposure of Respondents

Variables	Cases =43 n (%)	Controls=129 n (%)	χ^2	p-value
Fruit Intake(days/week)				
Never	3(7.1)	3(2.3)	3.078	0.545
1	6(14.3)	28(21.7)		
2	16(38.1)	49(38)		
3	5(11.9)	13(10.1)		
≥4	12(28.6)	36(27.9)		
Serving per Intake				
1	15(38)	35(27.8)	2.118	
2	17(43.6)	57(45.2)		0.347
3	7(17.9)	34(27)		
Vegetable Intake(days/week)				
1	7(16.7)	9(7)	14.958	0.002[†]
2	8(19)	37(28.9)		
3	10(23.8)	59(46.1)		
≥4	17(40.5)	23(18)		
Serving per Intake				
1	24(60)	19(14.8)	32.989	<0.001[†]
2	11(27.5)	65(50.8)		
3	5(12.5	44(34.4)		
Additional Salt Intake				
Yes	10(25)	7(5.5)	12.635	<0.001[†]
No	30(75)	120(94.5)		
Vigorous Physical Activity (Met mins/wk.)				
≥ 600	10(23.3)	46(35.7)	2.259	0.133
<600	33(76.7)	83(64.3)		
Moderate Physical Activity (Met mins/wk.)				
≥ 600	18(42.9)	47(37.3)	0.175	0.676
<600	24(57.1)	79(62.7)		
[Φ] **Occupation A**				
Yes	8(20.5)	31(25)	0.328	0.567
No	31(79.5)	93(75)		
[a] **Occupation B**				
Yes	1(2.6)	71(57.3)	35.990	<0.001[†]
No	38(97.4)	53(42.7)		
[b] **Occupation C**				
Yes	2(5.1)	5(4)	0.087	0.768
No	37(94.9)	119(96)		
[c] **Occupation D**				
Yes	5(12.8)	23(18.5)	1.035	0.596
No	34(87.2)	100(81.5)		
[h] **Occupation E**				
Yes	5(12.8)	17(13.7)	0.020	0.887
No	37(94.9)	107(86.3)		
[|] **Occupation F**				
Yes	2(5.1)	11(9.2)	0.630	0.424
No	37(94.9)	109(90.8)		

[†] Significant at p<0.05 [Φ] Exposed to Asbestos and Asbestiforms [a] Exposed to Aromatic amines (e.g. 2-naphthyl amine 4-aminobiphenyl), [b] Exposed to Cadmium and Cadmium compounds, [c] Exposed to Benzene and Toluene, [h] Exposed to Poly Aromatic Hydrocarbons [|] Exposed to Radiation.

Discussion

Cancer is the largest cause of death worldwide and its burden is expected to rise by 70% in the next 14 years.[22] A world report on cancer has shown that by 2030 the most cancer population would be in low and middle-income countries to which Nigeria belongs.[22] The substantial gaps in our present awareness about the risk factors

Table 4. Associations Between Different Variables and Prostate Cancer in a Univariate Logistic Regression Model at $P<0.10$

Variables	OR	90%CI		*P* value
		Lower	Upper	
Age(years)				
<70	3.192	2.184	4.665	<0.001**
[1]≥70	0.065	0.011	0.385	0.012**
Education				
No Formal Education	2.494	0.797	7.801	0.183
Primary	0.565	0.208	1.536	0.348
Secondary	1.652	0.848	3.217	0.216
[1]Tertiary				
Income				
<50000	1.167	0.431	3.161	0.799
50000-99999	1.039	0.428	2.522	0.943
[1]≥100000				
Employment Status				
Self Employed	0.508	0.243	1.063	0.131
Currently Employed	0.323	0.110	0.942	0.082**
[1]Retired				
Family History of Cancer				
Yes	2.468	1.193	5.108	0.041**
[1]No				
Family History of PCa				
Yes	3.245	1.289	8.168	0.036**
[1]No				
First Degree Relative with PCa				
Yes	3.245	1.289	8.168	0.036**
[1]No				
Second Degree Relative with PCa				
Yes	3.000	0.293	30.716	0.437
[1]No				
Family History of Breast Cancer				
Yes	1.000	0.150	6.681	1.000
[1]No				
Smoking Tobacco				
Ever	0.861	0.454	1.634	0.700
[1]Never				
Current smokers				
Yes	0.026	0.000	14.472	0.343
[1]No				
No. of cigarette smoked /day	0.906	0.744	1.103	0.408
Regular Contact with Smokers				
Yes	0.844	0.380	1.874	0.123
[1]No				
Alcohol Consumption				
Ever	1.530	0.785	2.982	0.294
[1]Never				
Local Concoction with Alcohol				
Yes	0.500	0.259	0.966	0.083**
[1]No				
Current Alcohol Consumers				
Yes	1.723	0.818	3.630	0.230
[1]No				
No. of bottles taken/ day	1.028	0.993	1.064	0.192
Age at First Sex	1.132	1.065	1.203	0.001**
Sexual Activity				
Yes	0.219	0.108	0.444	<0.001**
[1]No				
Duration of Sexual Inactivity				
<12months	0.457	0.167	3.121	0.503
[1]≥12months				
[Φ] **Occupation A**				
Yes				
[1] No				
[A] **Occupation B**				
Yes				
[1] No				
[b] **Occupation C**	0.750	0.350	1.605	0.534
Yes	0.024	0.004	0.127	<0.001**
[1] No	1.061	0.267	4.224	0.943
[3] **Occupation D**	0.621	0.239	1.613	0.412
Yes	1.000	0.411	2.435	1.000
[1] No	0.531	0.146	1.924	0.418
[b] **Occupation E**				
Yes				
[1] No				
[I] **Occupation F**				
Yes				
[1] No				
Fruit Intake (days/ week)				
[1]Never	0.205	0.043	0.963	0.092**
1	0.344	0.086	1.382	0.207
2	0.481	0.103	2.241	0.434
3	0.338	0.079	1.440	0.218
≥4				
Serving of fruit/Intake				
[1]1	0.651	0.309	1.377	0.286
2	0.417	0.191	1.045	0.343
3				
Vegetable Intake(days/ week)				
[1]1	0.315	0.114	0.874	0.062**
2	0.264	0.094	0.741	0.034**
3	1.194	0.403	3.54	0.788
≥4				
Serving of vegetable / Intake				
[1]1	0.112	0.046	0.272	<0.001**
2	0.081	0.029	0.225	<0.001**
3				
Additional Salt Intake	6.247	2.322	16.808	0.002**
Yes				
[1]No				
Vigorous Physical Activity (Mets mins/ wk.)	0.526	0.263	1.052	0.128
≥ 600				
[1] <600				
Moderate Physical Activity (Mets mins/ wk.)	1.13	0.559	2.302	0.770
≥ 600				
[1] <600				

[1] Reference ** Significant at p<0.10, CI: Confidence Interval.

associated with these cancers need to be resolved. The goal of this study was to assess risk factors associated with PCa among patients diagnosed at the University College Hospital, Ibadan. First-degree relative with PCa was significantly associated with PCa risk by a 3.2-fold at 90% CI. Conversely, in the fully adjusted model at 95% CI, it was insignificantly associated with PCa. This finding is similar to reports in previous studies,[11,12] but a hospital study in Nigeria has reported a significant association at 95% CI without adjusting for confounders.[28] The reasons for these inconsistencies in results may be the lack of knowledge or forgetfulness of the diagnosis of PCa in a family member (measurement bias), insufficient sample size, and confounding bias. Age is taken to be the

Table 5. Associations Between Age and Sexual Activity and Prostate Cancer Risk in a Multivariable Logistic Regression Model at $P<0.05$

Variables	AOR	95%CI		*P* value
		Lower	Upper	
Age	2.797	1.730	4.523	<0.001*
Age at First Sex	1.174	1.031	1.338	0.016*
Sexual Activity				
Yes	0.205	0.046	0.926	0.039*
[1]No				

[1] Reference * Significant at $P<0.05$, CI: Confidence Interval, AOR: Adjusted odds ratio

strongest epidemiological risk factor PCa in our study and other studies in India and Pakistan.[31-33] Our study has shown that increasing age increases the odds of PCa among Nigerian men by 2.8 folds. It has shown that men that are less than 70 years have lower odds of PCa than older men. This report is similar to a previous study.[12] The explanation behind this may be elevated oxidative stress, contributing to the onset of the disease as age increases.[19,20] Markedly, the research design of age-matching did not reject the odds, with increasing age remaining a strong risk factor for PCa case status, even with the controls being within ± 5 years of their matched cases. The median age at first sex in six sub-Saharan African countries ranges from 17-20 years.[34] The mean age at first sex in our study was 25 years and 22 years for the cases and controls respectively. This shows relative lateness with age at first sex among Nigerian men diagnosed with PCa. After adjusting for age and sexual activity, for every unit increase in the age at first sex there were 17.4% excess odds of developing PCa among the cases. Overage reporting (recall bias) may explain the reason for this association. This finding is inconsistent with previous reports that older age at first sex decreases the risk of PCa.[35,36] This is likely due to the difference in study designs employed and as well as relying on self-reports from respondents on sexual activity in our study. Sexual activity as a risk factor has shown to be significantly associated with PCa in the fully adjusted model. Sexual activity lowered the odds of PCa by 79.5%. This result is consistent with a previous study.[13] The increase in the frequency of ejaculation may reduce the carcinogens present in the prostatic fluid and stabilize testosterone levels.[37,38] There may be some drawbacks to this study: The questionnaire captured self-reported information, largely depending on respondents providing the correct information. Information bias can occur, particularly when reporting on family and sexual background. However, these were minimized by ensuring that the participants were assured of a high degree of confidentiality. Misclassification bias was minimized by using a standard classification tool for all the participants. Ascertainment bias for cases and controls was minimized by using standard classification guidelines for cases and screening for PSA for controls.

Conclusion

Conclusively, increasing age, increasing age at first sex and sexual inactivity increase PCa risk in this population. However, larger-sized population studies are essential to strengthen the available evidence and reduce the inherent flaws. Finally, interventions centered on routine screening for prostatic-specific antigen with an emphasis on the elderly clinic are recommended.

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Authors' contributions

Conception and Design: BI, SB and IOA made substantial contributions to the conceptualization and design of this study. Administrative support: N/A. Provision of study materials or patients: BI provided all materials and participants necessary for this study. Collection and assembly of data: BI collected and assembled all datasets from this study. Data analysis and Interpretation: BI analyzed all data derived from this study and interpretation was done under the supervision of IOA. Manuscript Writing: BI, SB and IOA wrote and revised the manuscript for publication. Final approval of manuscript: BI, SB and IOA accented on the final manuscript for publication.

Conflict of Interest Disclosures

The authors have no conflict of interest to declare.

Ethical Approval

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted following the Declaration of Helsinki (as revised in 2013). Ethical approval and official permission to carry out the study were sought from UI/UCH Ethics Review Committee with identification number: UI/EC/19/0474.

References

1. Hoffman, M. WebMD LLC. 2020 Available from: https://www.webmd.com/men/picture-of-the-prostate
2. International Agency for Research on Cancer (IARC). GLOBOCAN: Latest global cancer data: Cancer burden rises to 18.1 million new cases and 9.6 million cancer deaths in 2018. Geneva: World Health Organization; 2018. https://www.iarc.who.int/wp-content/uploads/2018/09/pr263_E.pdf.
3. American Cancer Society (ACS). Cancer Facts & Figures. Atlanta: ACS; 2019.
4. Badmus TA, Adesunkanmi AR, Yusuf BM, Oseni GO, Eziyi AK, Bakare TI, et al. Burden of prostate cancer in southwestern Nigeria. Urology. 2010;76(2):412-6. doi: 10.1016/j.urology.2010.03.020.
5. Ikuerowo SO, Omisanjo OA, Bioku MJ, Ajala MO, Mordi VP, Esho JO. Prevalence and characteristics of prostate cancer among participants of a community-based screening in Nigeria using serum prostate specific antigen and digital rectal examination. Pan Afr Med J. 2013;15:129. doi: 10.11604/pamj.2013.15.129.2489.
6. Batty GD, Kivimäki M, Clarke R, Davey Smith G, Shipley MJ. Modifiable risk factors for prostate cancer mortality in London: forty years of follow-up in the Whitehall study. Cancer Causes Control. 2011;22(2):311-8. doi: 10.1007/s10552-010-9691-6.
7. Caple C, Schub T. Prostate Cancer: Risk Factors and Prevention. Glendale, CA: Cinahl Information Systems; 2013.
8. McGregor SE, Courneya KS, Kopciuk KA, Tosevski C, Friedenreich CM. Case-control study of lifetime alcohol intake and prostate cancer risk. Cancer Causes Control.

2013;24(3):451-61. doi: 10.1007/s10552-012-0131-7.
9. Gong Z, Neuhouser ML, Goodman PJ, Albanes D, Chi C, Hsing AW, et al. Obesity, diabetes, and risk of prostate cancer: results from the prostate cancer prevention trial. Cancer Epidemiol Biomarkers Prev. 2006;15(10):1977-83. doi: 10.1158/1055-9965.epi-06-0477.
10. Jiménez-Mendoza E, Vázquez-Salas RA, Barrientos-Gutierrez T, Reynales-Shigematsu LM, Labra-Salgado IR, Manzanilla-García HA, et al. Smoking and prostate cancer: a life course analysis. BMC Cancer. 2018;18(1):160. doi: 10.1186/s12885-018-4065-7.
11. Subahir MN, Shah SA, Zainuddin ZM. Risk factors for prostate cancer in Universiti Kebangsaan Malaysia Medical Centre: a case-control study. Asian Pac J Cancer Prev. 2009;10(6):1015-20.
12. Hosseini M, SeyedAlinaghi S, Mahmoudi M, McFarland W. A case-control study of risk factors for prostate cancer in Iran. Acta Med Iran. 2010;48(1):61-6.
13. Shafique K, McLoone P, Qureshi K, Leung H, Hart C, Morrison DS. Cholesterol and the risk of grade-specific prostate cancer incidence: evidence from two large prospective cohort studies with up to 37 years' follow up. BMC Cancer. 2012;12:25. doi: 10.1186/1471-2407-12-25.
14. Dagnelie PC, Schuurman AG, Goldbohm RA, Van den Brandt PA. Diet, anthropometric measures and prostate cancer risk: a review of prospective cohort and intervention studies. BJU Int. 2004;93(8):1139-50. doi: 10.1111/j.1464-410X.2004.04795.x.
15. Wolk A. Diet, lifestyle and risk of prostate cancer. Acta Oncol. 2005;44(3):277-81. doi: 10.1080/02841860510029572.
16. Ceber E, Çakır D. Dietary patterns affecting prostate cancer: medical education. Turk Klin J Med Sci. 2009;29(3):733-9.
17. Mazdak H, Mazdak M, Jamali L, Keshteli AH. Determination of prostate cancer risk factors in Isfahan, Iran: a case-control study. Med Arh. 2012;66(1):45-8. doi: 10.5455/medarh.2012.66.45-48.
18. Salem S, Salahi M, Mohseni M, Ahmadi H, Mehrsai A, Jahani Y, et al. Major dietary factors and prostate cancer risk: a prospective multicenter case-control study. Nutr Cancer. 2011;63(1):21-7. doi: 10.1080/01635581.2010.516875.
19. Crawford ED. Epidemiology of prostate cancer. Urology. 2003;62(6 Suppl 1):3-12. doi: 10.1016/j.urology.2003.10.013.
20. Khandrika L, Kumar B, Koul S, Maroni P, Koul HK. Oxidative stress in prostate cancer. Cancer Lett. 2009;282(2):125-36. doi: 10.1016/j.canlet.2008.12.011.
21. Masko EM, Allott EH, Freedland SJ. The relationship between nutrition and prostate cancer: is more always better? Eur Urol. 2013;63(5):810-20. doi: 10.1016/j.eururo.2012.11.012.
22. International Agency for Research on Cancer (IARC). World Cancer Report. 1st ed. Lyon, France: IARC; 2014.
23. Rebbeck TR, Devesa SS, Chang BL, Bunker CH, Cheng I, Cooney K, et al. Global patterns of prostate cancer incidence, aggressiveness, and mortality in men of African descent. Prostate Cancer. 2013;2013:560857. doi: 10.1155/2013/560857.
24. International Agency for Research on Cancer (IARC). GLOBOCAN: Estimated Cancer Incidence, Mortality and Prevalence Worldwide in. Geneva: World Health Organization; 2012.
25. Odedina FT, Ogunbiyi JO, Ukoli FA. Roots of prostate cancer in African-American men. J Natl Med Assoc. 2006;98(4):539-43.
26. Odedina FT, Akinremi TO, Chinegwundoh F, Roberts R, Yu D, Reams RR, et al. Prostate cancer disparities in Black men of African descent: a comparative literature review of prostate cancer burden among Black men in the United States, Caribbean, United Kingdom, and West Africa. Infect Agent Cancer. 2009;4(Suppl 1):S2. doi: 10.1186/1750-9378-4-s1-s2.
27. Akinremi TO, Ogo CN, Olutunde AO. Review of prostate cancer research in Nigeria. Infect Agent Cancer. 2011;6(Suppl 2):S8. doi: 10.1186/1750-9378-6-s2-s8.
28. Agalliu I, Adebiyi AO, Lounsbury DW, Popoola O, Jinadu K, Amodu O, et al. The feasibility of epidemiological research on prostate cancer in African men in Ibadan, Nigeria. BMC Public Health. 2015;15:425. doi: 10.1186/s12889-015-1754-x.
29. American Cancer Society (ACS). Prostate Cancer Early Detection, Diagnosis, and Staging. ACS; 2019.
30. Ambs S, Alexander R, Lee D. A Case-Control Study of Prostate Cancer in the Greater Baltimore Area. An Epidemiological Study of Genetic and Environmental Risk Factors for Prostate Cancer in African-American and Caucasian Males. 2012. https://ccr.cancer.gov/sites/default/files/nci_prostate_cancer_study_protocol8.pdf.
31. Ganesh B, Saoba SL, Sarade MN, Pinjari SV. Risk factors for prostate cancer: an hospital-based case-control study from Mumbai, India. Indian J Urol. 2011;27(3):345-50. doi: 10.4103/0970-1591.85438.
32. Bashir MN, Ahmad MR, Malik A. Risk factors of prostate cancer: a case-control study in Faisalabad, Pakistan. Asian Pac J Cancer Prev. 2014;15(23):10237-40. doi: 10.7314/apjcp.2014.15.23.10237.
33. American Cancer Society (ACS). Cancer Facts & Figures. Atlanta: ACS; 2015.
34. Marston M, Slaymaker E, Cremin I, Floyd S, McGrath N, Kasamba I, et al. Trends in marriage and time spent single in sub-Saharan Africa: a comparative analysis of six population-based cohort studies and nine Demographic and Health Surveys. Sex Transm Infect. 2009;85 Suppl 1:i64-71. doi: 10.1136/sti.2008.034249.
35. Hennis AJ, Wu SY, Nemesure B, Leske MC. Urologic characteristics and sexual behaviors associated with prostate cancer in an African-Caribbean population in Barbados, West Indies. Prostate Cancer. 2013;2013:682750. doi: 10.1155/2013/682750.
36. Jian Z, Ye D, Chen Y, Li H, Wang K. Sexual activity and risk of prostate cancer: a dose-response meta-analysis. J Sex Med. 2018;15(9):1300-9. doi: 10.1016/j.jsxm.2018.07.004.
37. Del Rosario AD, Bui HX, Abdulla M, Ross JS. Sulfur-rich prostatic intraluminal crystalloids: a surgical pathologic and electron probe x-ray microanalytic study. Hum Pathol. 1993;24(11):1159-67. doi: 10.1016/0046-8177(93)90210-8.
38. Svatek RS, Karam JA, Rogers TE, Shulman MJ, Margulis V, Benaim EA. Intraluminal crystalloids are highly associated with prostatic adenocarcinoma on concurrent biopsy specimens. Prostate Cancer Prostatic Dis. 2007;10(3):279-82. doi: 10.1038/sj.pcan.4500954.