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Dietary pattern and lipid profile of prostate cancer patients attending Abubakar Imam Urology Centre Kano Nigeria

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Abstract
Prostate cancer is one of the most prevalent malignancies affecting men globally, with accumulating evidence indicating that dietary habits and lifestyle factors play important roles in its development and progression. This study evaluated the dietary patterns and lipid profiles of prostate cancer patients attending Abubakar Imam Urology Centre, Kano, Nigeria. A cross-sectional study design was employed involving 60 male patients aged 41 years and above. Data were collected through anthropometric measurements, dietary assessments, biochemical analyses including lipid profile, prostate-specific antigen (PSA), and acid phosphatase, while statistical analysis was performed using Analysis of Variance (ANOVA). The findings showed that patients aged 41–60 years had the lowest mean PSA concentration (7.21 ± 1.21 ng/ml). Evaluation based on years of marriage revealed that participants married for 6–10 years, 11–20 years, and 21 years or more had mean body mass index (BMI) values of 19.60 ± 0.00, 20.65 ± 2.02, and 20.85 ± 3.92 kg/m², respectively, all falling within the normal BMI range. Assessment of occupational categories indicated that farmers recorded the highest mean acid phosphatase level (1.36 ± 0.021 U/L), possibly due to prolonged exposure to agrochemicals and pesticides when compared with civil servants, traders, and participants in other occupations. Furthermore, dietary assessment demonstrated that beverage consumption was associated with the highest mean serum PSA concentration (370 ng/ml), whereas dairy products were associated with the lowest mean PSA concentration (110 ng/ml). Overall, the findings suggest that dietary habits and lipid metabolism may influence prostate cancer progression. Consequently, encouraging the consumption of balanced diets rich in plant-based foods and antioxidant-containing foods may contribute to improved prognosis and enhanced quality of life among men living with prostate cancer.

Keywords: Prostate cancer, lipid profile, lifestyle, dietary pattern, anthropometry & prostate specific antigen

Introduction

Prostate cancer is the second most frequently diagnosed cancer among men worldwide and remains the fifth leading cause of cancer-related mortality in males (Bray *et al*., 2018; Matsushita, 2020). In 2018 [5, 10] alone, approximately 1.2 million new cases were reported globally, with an estimated 360,000 deaths attributed to the disease (Bray *et al*., 2018; Matsushita, 2020). The incidence of prostate cancer differs considerably across geographical regions, occurring approximately six times more frequently in Western countries than in many non-Western nations. Although numerous risk factors have been identified, including advancing age, ethnicity, and family history, the exact causes of prostate cancer remain incompletely understood. Evidence indicates that nearly 85% of diagnosed cases occur in men aged 65 years and above, emphasizing the influence of ageing on disease occurrence (Bostwick *et al*., 2004; Gronberg, 2003; Forsland, 2020) [10].

Among the modifiable risk factors, dietary intake has received considerable attention because it may explain the marked geographical differences in prostate cancer incidence and mortality. Nutritional epidemiology has consistently demonstrated a substantial rise in the global burden of cancer over recent decades, with reported increases in cancer cases from 6.7 million in 2002 to 8.2 million in 2012 [2], accompanied by approximately 1.5 million additional cancer-related deaths (Charoensiri *et al*., 2009; Shaharkar *et al*., 2017) [20, 30]. Current projections estimate that by 2030, global cancer incidence could reach 21 million new cases with approximately 13 million deaths annually (Maule, 2007; Zahra *et al*., 2021).

Within Africa, prostate cancer represents the most common malignancy among men and is a major contributor to cancer-related mortality. The disease accounts for approximately 28,000 deaths (11.3% of all male cancer deaths) and about 40,000 newly diagnosed cases (13% of all male cancers) across the continent (Ferlay *et al*., 2011; Paul, 2009) [29]. In East Africa, an estimated 9,000 new cases and 7,300 deaths have been reported, making prostate cancer the third most common male cancer in terms of both incidence and mortality (Ferlay *et al*., 2011; Paul, 2009) [29]. Although countries such as Saudi Arabia report comparatively fewer deaths than many African nations, regional variations remain evident (Belkahla, 2022) [3]. In developed countries, particularly the United States, widespread screening programmes have enabled early diagnosis, with over 90% of cases detected before advanced disease develops. Consequently, five-year relative survival has improved remarkably from approximately 68% to nearly 99% over the past two decades (Brenner, 2000; Compos, 2018; Mehdad, 2010) [5, 18].

In Nigeria, prostate cancer constitutes approximately 11–12% of all cancers affecting men, making it one of the country's most significant public health concerns (Osuchukwu *et al*., 2013) [28]. Unfortunately, routine screening programmes remain limited, and awareness regarding early detection strategies is generally poor among the male population. As a result, many patients present with advanced disease, reducing treatment success and survival outcomes. Prostate cancer develops following malignant transformation of cells within the prostate gland, with disease progression influenced by both genetic susceptibility and environmental exposures (Zorn and Gautum, 2010; Osuchukwu *et al*., 2013) [28].

Several epidemiological studies have associated high consumption of red meat, saturated fats, and animal-derived fats with an increased risk of prostate cancer, whereas diets rich in vegetables appear to confer a protective effect (WCRFI, 1997; Hodge *et al*., 2004) [13]. Nutrition also plays a crucial role in preventing chronic conditions such as cardiovascular disease, diabetes mellitus, and osteoporosis, all of which commonly coexist among prostate cancer patients, particularly those receiving androgen deprivation therapy (ADT). ADT has been associated with increased adiposity, insulin resistance, and metabolic dysfunction, thereby predisposing patients to additional health complications (Morote, 2007; Keat *et al*., 2012; Tsai *et al*., 2007; McLaughlin *et al*., 2019). Furthermore, ADT frequently results in increased fat mass and reduced lean muscle mass, changes that may adversely influence disease progression and increase overall mortality. Excess adiposity has also been linked to an elevated risk of type 2 diabetes and poorer prostate cancer outcomes (Wilson, 2020) [36].

Likewise, dietary patterns characterized by frequent consumption of meat and dairy products have been associated with greater prostate cancer incidence and more aggressive disease compared with diets emphasizing plant-derived proteins and healthy fats (Levy, 2016) [15]. Understanding the relationship between dietary habits, lipid metabolism, and prostate cancer is therefore essential for developing effective nutritional strategies aimed at improving patient outcomes. Consequently, this study investigated the dietary patterns and lipid profiles of prostate cancer patients attending Abubakar Imam Urology Centre, Kano, Nigeria.

Materials and Methods

Study Design

This investigation employed a cross-sectional research design incorporating both qualitative and quantitative approaches. The study was conducted among male patients aged 41 years and above who attended the Abubakar Imam Urology Centre, Kano, Nigeria. The design enabled the collection of demographics, dietary, anthropometric, and biochemical information required to assess the relationship between dietary patterns and lipid profiles in individuals diagnosed with prostate cancer.

Target Population

The study population consisted of male patients aged forty-one years and above who presented at the Abubakar Imam Urology Centre with urinary tract-related complaints and who met the study inclusion criteria. These participants constituted the accessible population from which the study data were obtained.

Sample Size

The sample size was calculated using the formula reported by (Singha, 1996) [31]

n= $= 60.2 \approx 60$

Where n = sample size

Z = confident interval (95%)

d = margin of error (5%)

P = prevalence (29.6% prevalence of prostate cancer in Nigeria (Bosland *et al.*, 2021) [38])

Data Collection Tools

Questionnaire Administration

Data were gathered using a semi-structured questionnaire designed to obtain relevant information from the respondents. Trained research assistants assisted participants in completing the questionnaire, thereby ensuring clarity of responses and minimizing errors during data collection. The instrument captured socio-demographic characteristics, dietary habits, and other pertinent clinical information.

Determination of Biochemical Indices

Fasting venous blood samples were collected from each participant by qualified medical personnel using sterile needles and syringes. Sample collection was carried out between 8:00 am. and 9:00 am. The blood specimens were immediately transferred into appropriately labeled plain sample bottles and allowed to clot for approximately 30 minutes. Thereafter, the samples were centrifuged at 500 rpm for 10 minutes to separate the serum, which was subsequently utilized for biochemical analyses.

Determination of Serum Total Protein

Serum total protein concentration was measured using the Biuret reagent method as described by Tietz (1995) [35].

Determination of Serum Albumin

Albumin was determined using Bromocresol green method (Coley-Grant *et al*., 2016) [6].

Determination of serum Triacylglycerol

Triacylglycerol was determined using the extraction method (Trinder, 1969) [34].

Determination of Serum High Density Lipoprotein (HDL)

The high-density lipoproteins were determined according to the method described by Artiss and Zak (1997) [1].

Acid Phosphatase

Test principle: acid phosphatase of serum acts on the monophosphate thymolphthalein substrate the addition of alkali inhibits enzymatic action and converts the released thymolphthalein in it blue form that is measured by calorimetry. The final product of reaction consists of a mixture of blue and substrate colour.

Determination of Serum Malondealdehyde Level (MDA) By Spectrophotometric

Serum (0.2mL) was put in a test tube containing 3 mL of 1% TBA in 2% NaOH, the mixture was put in boiling water bath for 15 minutes, allowed to cool and the absorbance of the pink-coloured product was read at 532 nm (Gulteridge and Wilkins, 1982) [12].

Prostate-Specific Antigen (PSA) Assay

Prostate-specific antigen was analyzed using the Anbio PSA Rapid Test Kit based on fluorescence immunoassay technology. The assay operates on a sandwich immunodetection principle in which fluorescence-labeled detector antibodies bind specifically to PSA antigens present in the specimen. As the sample migrates through the nitrocellulose membrane by capillary action, the antigen-antibody complexes are captured by immobilized anti-PSA antibodies on the test strip. The fluorescence signal generated is proportional to the quantity of PSA in the sample, and the AnbioFIA meter displays the corresponding PSA concentration.

Statistical Analysis

The collected data were analyzed using Analysis of Variance (ANOVA). Descriptive statistics were used to summarize the variables, while bar charts were employed to illustrate food consumption patterns and their relationship with serum PSA concentrations. Statistical interpretation focused on identifying variations among the study groups and evaluating the association between dietary practices and biochemical parameters.

Results

The socio-demographic characteristics of prostate cancer patients has influence on the Anthropometry (BMI [kg/m^2], MUAC [cm]), and lipid profile.

Table 1: The Age (years) of Prostate Cancer Patients, and the BMI (kg/m^2), MUAC (cm) and of the Study Subjects

AGE (YEARS)	BMI (kg/m^2)	NR/BMI (kg/m^2)	MUAC (cm)	N/R (CM)
41- 60 *n* = 18	20.12 3.28	18.50-24.9	24.64 3.00	≥ 25
61 – 70 *n* = 24	20.21 3.23	18.50-24.9	24.95 2.30	≥ 25
> 71 *n* = 18	19.70 3.11	18.50-24.9	23.99 2.85	≥ 25

Key: BMI (Body Mass Index), N/R (Normal Range), MUAC (Mid Upper Arm Circumference).

Table 1.1: The Age (Years) of Prostate Cancer Patients, and the Lipid Profile of the Study Subjects

AGE (YEARS)	Tchol (mmol/l)	TG (mmol/l)	HDL - chol (mmol/l)	LDL – chol (mmol/l)
41- 60 n =18	8.63 1.03	8.16 1.74	6.21 1.05	2.41 0.81
61 – 70 n =24	8.21 1.13	9.21 2.56	6.41 1.09	3.30 0.25
> 71 n = 18	9.11 2.17	11.51 1.22	4.44 1.06	5.20 0.72

Key: Tchol (Total cholesterol), TG (Triglycerides), HDL-chol (High Density Lipoprotein cholesterol), LDL-chol (Low Density Lipoprotein cholesterol).

Table 2: Years of Marriage of Prostate Cancer Patients BMI (kg/m^2) MUAC (cm) and of the Study Subjects

Years of Marriage (years)	BMI (kg/m^2)	MUAC (cm)
6- 10 *n* = 1	19.60 0.00	25.30 0.00
11 – 20 *n* = 24	20.65 2.02	25.43 1.47
> 21 *n* = 18	20.85 3.92	24.36 2.89

Key: BMI (Body Mass Index), MUAC (Mid Upper Arm Circumference).

Table 2.1: Years of Marriage of Prostate Cancer Patients, Lipid Profile of the Study Subjects

Years of marriage (years)	T-Chol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL-Chol (mmol/l)
6- 10 *n* = 1	5.79 0.00	6.44 0.00	6.12 0.00	2.36 0.00
11 – 20 *n* = 24	9.12 2.11	9.11 2.13	6.02 1.80	2.80 1.12
> 21 *n* = 18	9.14 1.61	9.61 2.22	4.30 1.41	3.41 1.22

Key: Tchol (Total cholesterol), TG(Triglycerides), HDL-chol (High Density Lipoprotein cholesterol), LDL-chol (Low Density Lipoprotein cholesterol).

Table 3: Number of Prostate Cancer Patients and the BMI (kg/m^2), MUAC (cm) of the Study Subjects

Number of Wives	BMI (kg/l)	MUAC (cm)
1 *n* = 33	20.22 3.35	23.15 3.22
2 *n* = 19	20.12 2.12	24.20 3.25
3 *n* = 4	19.25 1.37	22.31 2.19
4 *n* = 4	19.33 1.24	21.23 2.66

Key: BMI (Body Mass Index), MUAC (Mid Upper Arm Circumference).

Table 3.1: Number of Wives of Prostate Cancer Patients and the Lipid Profile of the Study Subjects

Number of Wives	T-Chol (mmol/l)	TG (mmol/l)	HDL-chol (mmol/l)	LDL-chol (mmol/l)
1 *n* = 33	7.45 1.67	8.21 1.93	9.33 2.45	2.06 0.31
2 *n* = 19	9.23 1.18	7.21 1.22	7.32 1.64	2.51 0.14
3 *n* = 4	9.24 2.49	8.12 1.06	7.46 1.24	2.68 0.31
4 *n* = 4	11.23 2.15	8.21 1.08	5.56 1.00	4.13 0.53

Key: Tchol (Total cholesterol), TG (Triglycerides), HDL-chol (High Density Lipoprotein cholesterol), LDL-chol (Low Density Lipoprotein cholesterol)

Table 4: The educational status of the prostate cancer patients, BMI (kg/m^2), MUAC (cm) and the of the study subjects

Educational Status	BMI (kg/m^2)	MUAC (cm)
Primary n = 20	20.54 2.61	23.35 2.61
Secondary *n* = 12	24.16 3.31	22.16 2.14
Tertiary *n* = 8	25.21 3.12	23.25 4.02
Others 20	20.24 2.90	20.22 2.21

Key: BMI (Body Mass Index), MUAC (Mid Upper Arm Circumference)

Table 4.1: Educational Status of the Prostate Cancer Patients and Lipid Profile of the Study Subjects

Educational Status	T-Chol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL-Chol (mmol/l)
Primary *n* = 20	8.21 1.21	6.11 1.00	**4.67^a 0.73**	3.21 0.21
Secondary *n* = 12	7.14 1.21	9.12 3.09	5.14 0.51	4.22 1.06
Tertiary *n* = 8	9.23 1.11	8.33 2.33	5.34 0.91	2.76 0.21
Others 20	9.82 1.49	8.21 1.22	8.25^a 2.11	2.51 0.64

Key: Tchol (Total cholesterol), TG(Triglycerides), HDL-chol (High Density Lipoprotein cholesterol), LDL-chol (Low Density Lipoprotein cholesterol).

Table 5: Occupation of the Prostate Cancer Patients, BMI (kg/m^2), MUAC (cm) and the Acid Phosphatase of the Study Subjects

Occupation	BMI (kg/m^2)	MUAC (cm)	Acids phosphatase (u/l)
Farmer *n* = 8	20.15 2.51	22.31 3.17	1.36^a 0.021
Trader *n* = 2	19.42 1.55	23.51 4.22	0.93$^{a, b}$ 0.04
Civil Servant *n* = 16	21.32 3.10	21.36 3.02	0.26^a 0.02
Others 34	20.61 3.11	22.51 4.21	1.02$^{a, b}$ 0.02

Key: BMI (Basal Metabolic Rate), MUAC (Mid Upper Arm Circumference).

Table 5.1: The Occupation of the Prostate Cancer Patients and Lipid Profile of the Study Subjects

Occupation	T-Chol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL-Chol (mmol/l)
Farmer *n* = 8	8.12 1.11	7.43 1.47	9.16 2.11	2.16 0.21
Trader *n* = 2	7.10 1.22	6.55 1.08	5.32 1.13	1.66 0.02
Civil Servant *n* = 16	9.21 1.36	4.32 1.02	7.12 1.16	2.83 0.27
Others 34	10.15 2.11	8.35 2.22	8.60 1.13	2.11 0.21

Key: Tchol (Total cholesterol), TG (Triglycerides), HDL-chol (High Density Lipoprotein cholesterol), LDL-chol (Low Density Lipoprotein cholesterol).

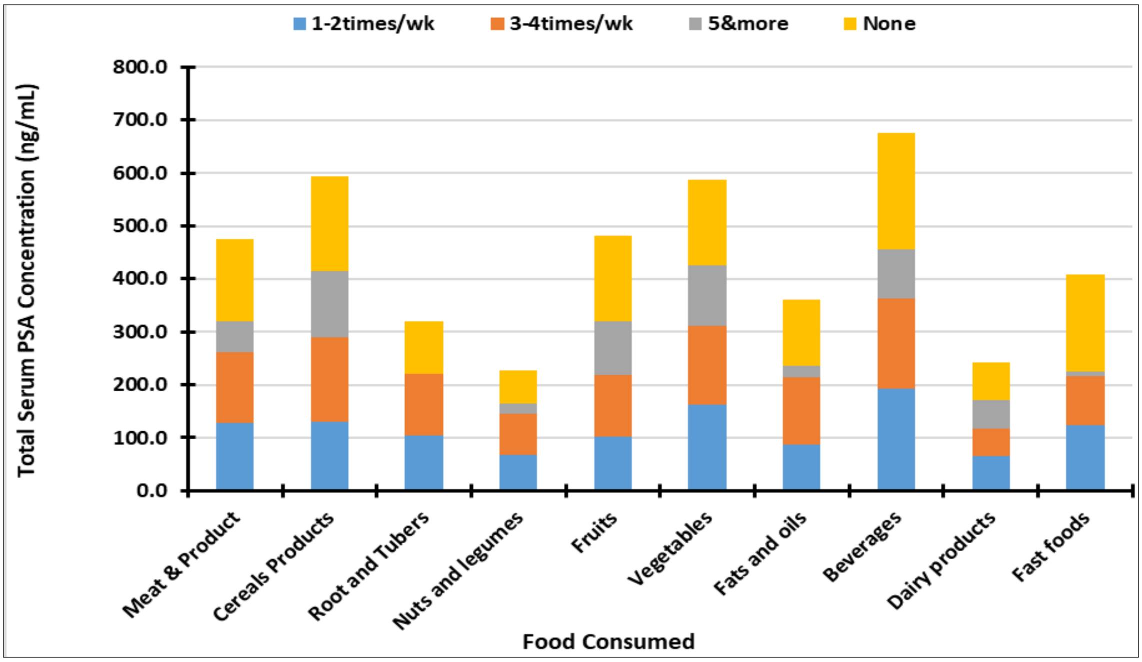

Fig 1: The food Consumption Pattern and the Total Serum PSA Concentration (ng/ml)

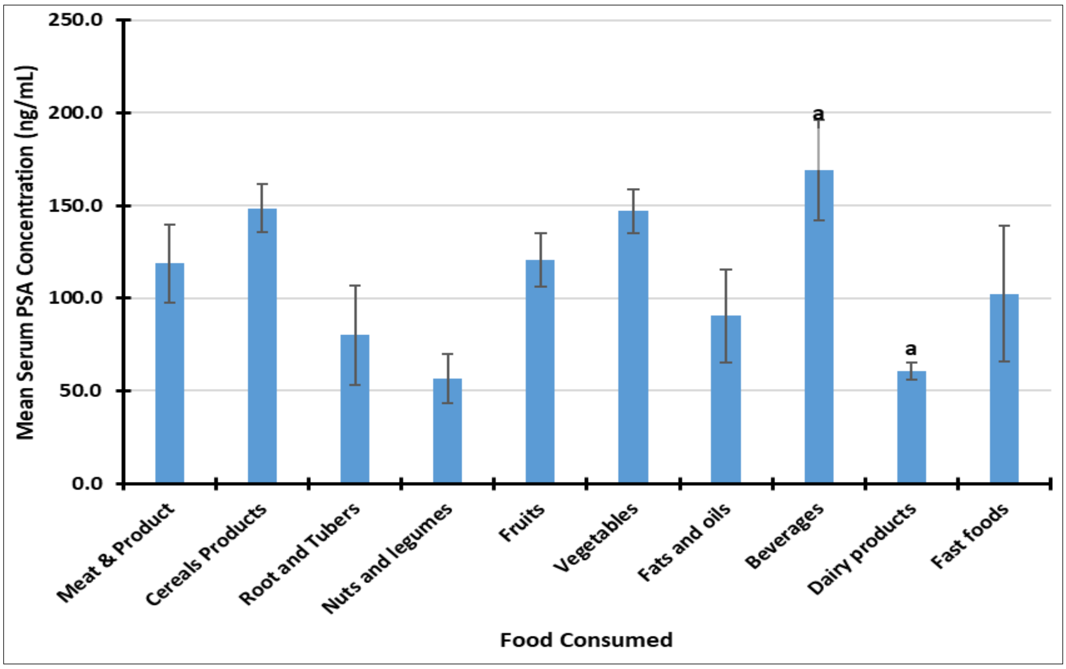

Fig 2: Result Indicating Food Consumption Pattern and Mean Serum PSA Concentration of Prostate Cancer Patients

Discussion

Age, Anthropometric Measurements, and Lipid Profile

The findings presented in Table 1 indicate that patients aged 61–70 years, who constituted the largest proportion of the study population, had a mean BMI of 20.21 ± 3.23 kg/m² and a mean mid-upper arm circumference (MUAC) of 24.95 ± 2.30 cm, values that fall within the normal nutritional range. Age has consistently been recognized as one of the strongest determinants of prostate cancer development and mortality. Previous studies have demonstrated that increasing age is associated with a greater likelihood of disease progression and poorer clinical outcomes. Furthermore, elevated BMI has been identified as an important risk factor for prostate cancer, particularly among younger men and individuals with a positive family history of the disease. These observations are consistent with the report of Dorel (2023) [7], who found that prostate cancer incidence increases with advancing age (72 ± 7.81 years) among men with normal BMI, whereas overweight and obese individuals tend to develop the disease at relatively younger ages.

Analysis of the lipid profile according to age showed a progressive decline in high-density lipoprotein (HDL) cholesterol as age increased from 41–60 years to above 70 years**.** Mean HDL values decreased from 6.21 ± 1.05 mmol/L to 4.44 ± 1.06 mmol/L, whereas low-density lipoprotein (LDL) cholesterol increased from 2.41 ± 0.81 mmol/L to 5.20 ± 0.72 mmol/L across the same age categories. These findings suggest that advancing age among prostate cancer patients is accompanied by worsening lipid metabolism, characterized by reduced protective HDL levels and elevated LDL concentrations. Such dyslipidaemia has been associated with increased PSA levels, disease progression, and poorer clinical outcomes. These results agree with Cavallo (2014) [4]**,** who reported that patients with serum triglyceride concentrations of 150 mg/dL or higher experienced a 35% greater risk of prostate cancer recurrence compared with individuals whose triglyceride concentrations remained within normal limits.

Among patients with abnormal lipid profiles, every 10 mg/dL increase in total serum cholesterol above 200 mg/dL was associated with an approximately 9% increase in the risk of prostate cancer recurrence**.** Conversely, for men with HDL concentrations below the recommended level of 40 mg/dL**,** each 10 mg/dL increase in HDL cholesterol corresponded to a 39% reduction in disease recurrence, highlighting the protective role of HDL cholesterol in prostate cancer prognosis.

The results presented in Table 2 demonstrate that marital duration influenced certain anthropometric and biochemical characteristics of the study participants. Patients who had been married for 6–10 years**,** 11–20 years**,** and more than 21 years recorded mean BMI values of 19.60 ± 0.00 kg/m²**,** 20.65 ± 2.02 kg/m²**,** and 20.85 ± 3.92 kg/m²**,** respectively. These values all fall within the normal BMI range. Likewise, the corresponding mean MUAC values of 25.30 ± 0.00 cm**,** 25.43 ± 1.47 cm**,** and 24.36 ± 2.80 cm indicate generally satisfactory nutritional status, although participants married for more than 21 years had the lowest MUAC values among the groups. These findings are comparable to those reported by Tang *et al.* (2024)**,** who observed that marriage significantly increased the likelihood of overweight (6.5%, $P < 0.01$) and obesity (2.8%, $P < 0.01$). Their study also showed that marriage tends to reduce time allocated to physical activity while having little influence on alcohol consumption, with more pronounced effects among younger adults than older individuals.

The lipid profile associated with marital duration further revealed that increasing years of marriage corresponded with higher concentrations of total cholesterol and serum triglycerides. Participants married for 6–10 years, 11–20 years**,** and more than 21 years had mean total cholesterol concentrations of 5.79 ± 0.00 mmol/L**,** 9.12 ± 2.11 mmol/L, and 9.14 ± 1.61 mmol/L, respectively. Similarly, triglyceride concentrations increased from 6.44 ± 0.00 mmol/L to 9.61 ± 2.22 mmol/L as marital duration increased. Elevated total cholesterol, triglycerides, and LDL cholesterol, together with reduced HDL cholesterol, are recognized components of metabolic syndrome, a condition that has been associated with increased prostate cancer risk and disease progression. These observations agree with the findings of Kehinde *et al*. (2023) [14], who reported significantly higher total cholesterol ($P < 0.001$) and HDL cholesterol ($P < 0.001$) among prostate cancer patients. Their study further demonstrated that triglyceride concentrations increased and HDL cholesterol decreased as disease stage advanced, although these changes did not reach statistical significance. Moreover, total cholesterol increased significantly with worsening tumour grade, whereas HDL cholesterol remained comparatively higher in patients with low-grade disease ($P < 0.001$).

Number of Wives, Anthropometric Indices and Lipid Profile

The findings presented in Table 3 indicate that the body mass index (BMI) of prostate cancer patients remained relatively consistent irrespective of the number of wives. Participants with one, two, three, and four wives recorded mean BMI values of 20.69 ± 4.45 kg/m², 21.02 ± 2.53 kg/m², 20.95 ± 0.77 kg/m², and 20.47 ± 1.87 kg/m², respectively. All these values fall within the recommended normal BMI range of 18.5–24.9 kg/m², suggesting that marital structure had no measurable influence on the nutritional status of the participants. Likewise, statistical analysis showed no significant variation in BMI across the different categories.

Similarly, the mean mid-upper arm circumference (MUAC) values for participants with one, two, three, and four wives were 24.25 ± 3.11 cm, 25.10 ± 2.21 cm, 24.78 ± 0.39 cm, and 24.53 ± 2.32 cm, respectively. Except for participants with two wives, whose MUAC was within the recommended reference value, the remaining groups recorded values that were slightly below the normal threshold of 25 cm. Nevertheless, no statistically significant differences in MUAC were observed among the groups. These findings are consistent with those of Dorel *et al*. (2023) [7], who reported that although prostate cancer incidence increases with advancing age, men with higher BMI values tend to be diagnosed at younger ages than individuals with normal BMI.

Evaluation of prostate-specific antigen (PSA) according to the number of wives revealed mean PSA concentrations of 14.25 ± 8.48 ng/ml, 22.77 ± 9.61 ng/ml, 11.92 ± 4.64 ng/ml, and 39.96 ± 2.66 ng/ml for participants with one, two, three, and four wives, respectively. Although PSA values varied numerically among the groups, the differences were not statistically significant. It is possible that individuals with multiple sexual partners may experience increased exposure to sexually transmitted infections and chronic prostatic inflammation, both of which can elevate PSA concentrations independently of prostate cancer severity. However, these observations contrast with the findings of Osaro *et al*. (2022) [26], who reported that men in monogamous relationships were approximately 2.5 times more likely to develop prostate cancer than those practicing polygamy (71.4% versus 28.6%).

Assessment of lipid parameters further demonstrated that increasing numbers of wives were associated with higher concentrations of total cholesterol and serum triglycerides. Mean total cholesterol increased from 7.45 ± 1.67 mmol/L among participants with one wife to 11.23 ± 2.15 mmol/L among those with four wives. Likewise, serum triglyceride concentrations remained relatively elevated across all groups. In contrast, HDL cholesterol progressively declined as the number of wives increased, decreasing from 9.33 ± 2.45 mmol/L among men with one wife to 5.56 ± 1.00 mmol/L among those with four wives. Elevated triglycerides and LDL cholesterol together with reduced HDL cholesterol have been widely associated with increased prostate cancer risk and disease progression. These observations support the findings of Emma *et al*. (2014) [9], who reported that elevated serum triglycerides significantly increased the risk of prostate cancer recurrence. Their study also indicated that among patients with dyslipidaemia, every 10 mg/dL increase in total cholesterol was associated with an increased likelihood of disease recurrence, although no significant associations were observed for LDL and HDL cholesterol in the overall study population.

Educational Status and Lipid Profile

The findings presented in Table 4 demonstrate that educational attainment influenced several anthropometric and biochemical variables among prostate cancer patients. Participants with primary, secondary, tertiary, and other educational qualifications recorded mean BMI values of 19.83 ± 2.58 kg/m², 25.26 ± 2.04 kg/m², 26.63 ± 7.52 kg/m², and 20.24 ± 3.05 kg/m², respectively. Individuals with primary education and those classified under "other" educational categories-maintained BMI values within the normal range, whereas participants with tertiary education were classified as overweight.

Comparable trends were observed for mid-upper arm circumference, with mean MUAC values of 24.33 ± 2.57 cm, 25.26 ± 2.04 cm, 25.95 ± 3.79 cm, and 23.85 ± 2.51 cm among participants with primary, secondary, tertiary, and other educational backgrounds, respectively. Only participants with secondary and tertiary education achieved MUAC values within the normal reference range, while those with lower educational attainment recorded comparatively lower values.

Analysis of lipid parameters indicated noticeable differences according to educational level. Mean serum triglyceride concentrations were 6.11 ± 1.00 mmol/L, 9.12 ± 3.09 mmol/L, 8.33 ± 2.33 mmol/L, and 8.21 ± 1.22 mmol/L among participants with primary, secondary, tertiary, and other educational qualifications, respectively. Participants with secondary education recorded the highest triglyceride concentrations.

The distribution of HDL cholesterol showed a gradual increase across educational categories, with mean values of 4.67 ± 0.73 mmol/L, 5.14 ± 0.51 mmol/L, 5.34 ± 0.91 mmol/L, and 8.25 ± 2.11 mmol/L, respectively. A statistically significant difference in HDL cholesterol was observed between participants with primary education and those classified under other educational qualifications.

Similarly, the mean LDL cholesterol concentrations were 3.21 ± 0.21 mmol/L, 4.22 ± 1.06 mmol/L, 2.76 ± 0.20 mmol/L, and 2.51 ± 0.64 mmol/L among the four educational categories. Overall, these findings suggest that educational attainment may influence lipid metabolism through differences in socioeconomic status, dietary practices, healthcare access, and lifestyle behaviours. These observations are consistent with the report of Lucaridra (2019) [16], who demonstrated that men with higher socioeconomic and educational status generally exhibited higher concentrations of total cholesterol, LDL cholesterol, and triglycerides, together with lower HDL cholesterol after adjustment for age, BMI, and other confounding variables.

Occupation, Acid Phosphatase and Lipid Profile

The results summarized in Table 5 demonstrate significant occupational differences in serum acid phosphatase among prostate cancer patients. Farmers recorded the highest mean acid phosphatase concentration (1.04 ± 0.37 U/L), followed by civil servants (0.59 ± 0.09 U/L), individuals in other occupations (0.40 ± 0.04 U/L), and traders (0.37 ± 0.02 U/L). The observed differences were statistically significant ($P < 0.005$). The elevated acid phosphatase levels among farmers may be attributable to prolonged exposure to agricultural chemicals, including pesticides and herbicides, whereas occupational and environmental exposures among civil servants and traders differ substantially. Lifestyle-related factors may also contribute to these variations. These findings agree with Marie-Elise *et al*. (2020), who reported approximately a two-fold increase in prostate cancer risk among farmers with substantial pesticide exposure after adjusting for age, ethnicity, educational level, and respondent status.

Occupational differences were also evident in the lipid profile of the participants. Civil servants recorded the highest mean total cholesterol concentration (9.21 ± 1.36 mmol/L), whereas traders had the lowest (7.10 ± 1.22 mmol/L). Regarding serum triglycerides, civil servants exhibited the lowest mean concentration (4.32 ± 1.02 mmol/L), while participants classified under other occupations recorded comparatively higher levels (8.35 ± 2.22 mmol/L).

The pattern of HDL cholesterol varied across occupations, with farmers exhibiting the highest mean HDL concentration (9.16 ± 2.11 mmol/L) and traders recording the lowest (5.32 ± 1.13 mmol/L). Elevated concentrations of total cholesterol, triglycerides, and LDL cholesterol have consistently been associated with aggressive prostate cancer, whereas higher HDL cholesterol appears to exert a protective effect. These observations are supported by Obunwo and Andy Nwokocha (2016) [27], who reported significantly higher PSA, total cholesterol, and LDL cholesterol concentrations among prostate cancer patients compared with healthy controls. Conversely, HDL cholesterol was significantly lower in affected individuals, suggesting that dyslipidaemia may contribute to the development and progression of prostate cancer.

Food Consumption Pattern and Serum PSA Concentration

The results presented in Figure 1 demonstrate the relationship between food consumption frequency and serum prostate-specific antigen (PSA) concentrations among prostate cancer patients. Participants who consumed beverages between 1–2 times per week, 3–4 times per week, and five or more times per week recorded mean serum PSA concentrations of approximately 190 ng/ml, 380 ng/ml, and 450 ng/ml, respectively. This trend suggests that increased consumption of beverages, particularly sugar-sweetened drinks, may be associated with elevated PSA concentrations. High intake of sugar-sweetened carbonated beverages has been implicated in increased prostate cancer risk through several biological mechanisms. Excessive sugar consumption stimulates insulin secretion and may promote the production of insulin-like growth factors, creating a metabolic environment that supports tumour cell proliferation and progression. These findings are consistent with those reported by Eloi *et al*. (2019), who demonstrated a significant association between sugary beverage consumption and overall cancer risk. Their study found that every additional 100 ml/day of sugar-sweetened beverages was associated with an increased risk of cancer (sub-distribution hazard ratio = 1.1; 95% confidence interval; $P < 0.0001$).

A similar trend was observed for dairy product consumption. Participants who consumed dairy products 1–2 times weekly, 3–4 times weekly, and five or more times weekly had mean serum PSA concentrations of 50 ng/ml, 110ng/ml, and 180 ng/ml, respectively. These findings suggest a positive relationship between frequent dairy consumption and increasing PSA levels. Previous studies have proposed that high intake of dairy products, particularly milk and cheese, may increase prostate cancer risk because these foods elevate circulating concentrations of insulin-like growth factor (IGF-1), a hormone known to stimulate both normal cellular proliferation and the growth of malignant cells.

The findings illustrated in Figure 2 further summarize the overall contribution of different food groups to serum PSA concentrations. Among the dietary categories evaluated, beverages contributed the highest mean PSA concentration (170 ng/ml), whereas dairy products contributed the lowest (70 ng/ml) when compared across all food groups. Despite this observation, beverages remained the dietary component most strongly associated with elevated PSA concentrations.

These findings further support previous evidence linking excessive intake of sugar-sweetened beverages with prostate cancer development and progression. Similar observations were reported by Fayth *et al*. (2018) [11], who found that men within the highest quartile of sugar consumption from sweetened beverages had a significantly increased risk of prostate cancer (HR = 1.21; 95% CI: 1.06–1.39) with a significant linear trend ($P < 0.01$). Collectively, these findings emphasize the importance of limiting the consumption of sugar-rich beverages as part of dietary strategies aimed at improving prostate health and reducing disease progression.

Conclusion

The present study demonstrates that dietary habits, nutritional status, and lipid metabolism are closely associated with clinical characteristics among prostate cancer patients attending Abubakar Imam Urology Centre, Kano, Nigeria. Patients aged 41–60 years exhibited the lowest mean serum PSA concentration (9.11 ± 6.51 ng/ml), while anthropometric assessment showed that participants, irrespective of the number of wives, generally maintained BMI values within the normal range. No statistically significant differences in BMI were observed among the marital categories.

Occupational analysis revealed that farmers had the highest mean serum acid phosphatase concentration (1.04 ± 0.84 U/L), a finding that may be associated with prolonged exposure to agricultural chemicals such as pesticides and other agrochemicals. In contrast, lower values were observed among civil servants, traders, and participants in other occupations, suggesting that occupational exposures may influence biochemical markers associated with prostate cancer.

Evaluation of dietary intake indicated that beverages, followed by vegetables, cereal products, roots and tubers, fruits, fast foods, and fats and oils, contributed substantially to total serum PSA concentrations, with corresponding mean values of 370 ng/ml, 310 ng/ml, 290 ng/ml, 220 ng/ml, 210 ng/ml, 210 ng/ml, respectively. These findings suggest that dietary composition may influence disease progression through its effects on metabolic and inflammatory pathways.

Overall, the study highlights the importance of adopting healthier dietary practices in the management of prostate cancer. Limiting the consumption of red and processed meats, foods rich in refined sugars, and sugar-sweetened beverages, while increasing the intake of vegetables, fruits, whole grains, and other antioxidant-rich foods, may contribute to improved prostate health, better treatment outcomes, and enhanced quality of life among affected individuals. Continued nutritional counseling should therefore be integrated into the routine management of prostate cancer patients to support long-term disease control and overall wellbeing.

References

1. Artiss JD, Zakk B. Measurement of Cholesterol Concentration. Handbook of Lipoprotein Testing, AACC Press, Washington, 1997, 99–114.
2. Bosland MC, Nettey OS, Phillips AA, Anunobi CC, Akinloye O, Ekanem IA, *et al*. Prevalence of Prostate Cancer at Autopsy in Nigeria – A Preliminary Report. Prostate,2012:81(9):553–559. DOI: 10.1002/pros.24133.
3. Belkahla S, Nahvi I, Biswas S, Amor NB. Advances and development of Prostate Cancer, treatment, and strategies: A systematic review, 2022. DOI: 10.3389/fcell.2022.991330. Pubmed.ncbi.nlm.gov/36.
4. Cavallo JO. High Level of Cholesterol and Triglycerides May increase the risk of Prostate Cancer Recurrence. Cancer Epidemiology, Biomarkers & Prevention, 2014.
5. Compos C, Sotomayor P, Jerez D, Gonzalez J, Schmidt CB, Schmidt K, *et al*. Exercise and Prostate Cancer: From Basic Sciences to Clinical Application, 2018. https://onlinelibrary.wiley.com/doi/10.1002/pros.2350
6. Coley-Grant D, Herbert M, Cornes MP, Barlow IM, Ford C, Gama R. Impact of Change in Albumin Assay on Reference Intervals, prevalence of hypoalbuminaemia and albumin prescriptions. Annals of Clinical Biochemistry,2016:53(1):112–116. DOI: 10.1177/0004563215599560.
7. Dorel P. Associations Between Body Mass Index and Prostate Cancer: The Impact on Progression-Free Survival. National Library of Medicine, 2023.
8. Eloit C, Srour B, Desmetz E, Kesse-Guyot E, Julia C, Deschamps V, *et al*. Sugary Drinks Consumption and Risk of Prostate Cancer: Result from NutriNet-Sante Prospective Cohort, 2019. DOI: 10.1136/bmj.l2408.
9. Emma AH, Howard LE, Cooperberg MR, Kane CJ, Aronson WJ, Terris MK, *et al*. Serum Lipid Profile and Risk of Prostate Cancer Recurrence. Cancer Epidemiology, Biomarkers & Prevention,2014:23(11):2349–2356. DOI: 10.1158//1055-9965
10. Forsland M. A Nutrition Intervention in Men with Prostate Cancer. Exploring Effects on Bowel Symptoms from Radiotherapy, Patient Experience, and Nutrient Intake. Digital Comprehensive Summaries of Uppsala Dissertations from the Faculty of Medicine 1666,83. Uppsala: Acta Universitatis Upsaliensis, 2020. ISBN 978-91-513-0945-3.
11. Fayth LM, Mariam LN, Zuo-Feng Z. Concentrated sugar and incidence of prostate cancer in a prospective cohort. Randomized controlled trial. British Journal of Nutrition,2018:120(6):703–710. DOI: 10.1017/s000711458001812
12. Gutteridge JMC, Wilkins S. Copper-dependent hydroxyl radical damage to ascorbic acid: Formation of a thiobarbituric acid-reactive product. FEBS Letters,1982:137(2):327–330.
13. Hodge AM, English DR, McCredie MRE, Severi G, Boyle P, Hopper JL. Food, Nutrient, and Prostate Cancer. Cancer Causes and Control,2004:15:11–20.
14. Kehinde OA. Association of Serum Lipid Profile with Prostate Cancer Risk and Aggressiveness in Nigeria Men. Cancer Epidemiology, Biomarkers, 2023, 32(12). DOI: 10.1158/1538-7755.DISP23-C087
15. Levy D. Manipulating Diet May Impact Prostate Cancer Risk, Progression. Functional Medicine, Prostate Cancer Urology, 2016. Https://Consultqd.Clevelandclinic.Org/Tag/Functional-Medicine/.
16. Lucaridra S. Socioeconomic Status and Educational Level Associated with Higher Cholesterol and Triglycerides. International Journal of Health Services,2019:14(4):346–355.
17. Mutsushita M. Influence of Diet and Nutrition on Prostate Cancer. Department of Urology, Osaka University Graduate School of Medicine, Suita, Osaka 565-0871, Japan. International Journal of Molecular Sciences,2020:21(4):1447.
18. Mehdad A, McBride E, Grillo MC, Ravasco P. Nutritional Status and Eating Pattern in Prostate Cancer Patients. Nutrition Hospitalaria,2010:25(3):422–427. PMID: 20593125.
19. McLaughlin K, Hidden Lindsey L, Pollock P, Higgano C, Murphy RA. Assessing the Nutritional Needs of Men with Prostate Cancer. Nutrition Journal,2019:18:81.
20. Merie-Elise P, Marie D, Jack S. Does Exposure to Agricultural Chemicals Increase the Risk of Prostate Cancer among Farmers? Systemic Review, 2009, 12(1). DOI: http://doi.org/10.26443/mjm.v12i1.363.
21. Mucci L, Giovannucci E. The Role of Nutrition and Diet in Prostate Cancer. Preventive Nutrition, 2009, 195–218.
22. Muhammad. Nutritional Status and its Determinants Among Adult Cancer Patients Undergoing Chemotherapy Treatment at Hawassa University Comprehensive Specialized Hospital, Hawassa, Southern Ethiopia, 2020.
23. National Cancer Control Plan (2018–2022). Federal Ministry of Health. Http://www.Cancer-Gov/About-Cancer/Causes-Prevention/Hp-Prevention-Overview-Pdq.
24. Nutrition Guide for Clinicians (NGC). Prostate Cancer. Risk Factors, Screening, Diagnosis, Treatment and Nutritional Consideration, 2020. https://nutritionguide.pcrm.org

25. Nwagwu SA, Ibebuike JE, Nwokike GI. Knowledge and Attitude of Men 50 Years and Above: About Prostate Cancer in Umugolo, Ehime Mbano L.G.A Imo State. Nursing and Care Open Access Journal, 2020, 7(2).
26. Osaro E, Hussaini AM, Abdullahi AA, Tosan E. Some Oxidative Stress Biomarkers among Patients with Prostate Cancer in Sokoto, North-western Nigeria. Open Journal of Blood Diseases, 2022, 12(3). DOI: 10.4236
27. Obunwo CC, Andy-Nwokocha. Evaluation of Levels of Prostate Specific Antigen and Lipid Levels in Prostate and Non-Prostate Cancer Patients in Port Harcourt, Nigeria. Academic Journals,2016:6(6):37–40.
28. Osuchukwu EC, James OE, Emmanuel AR, Ada RA, Otukwa AE. Assessment of Patients' Knowledge, Attitude and Perception Regarding Prostate Cancer Screening at the University of Calabar Teaching Hospital and Arubas Clinic, Calabar. Journal of Survey in Fisheries Sciences,2013:10(25):1521–1528.
29. Paul W. Prostate Cancer Awareness, Knowledge, Perception on Self-Vulnerability and Uptake of Screening Among Men in Nairobi County, Kenya, 2009.
30. Sakharkar P. Age and Racial/Ethnic Disparities and Burden of Prostate Cancer: A Cross-Sectional Population-Based Study. Journal of Basic and Clinical Pharmacy, 2017, 8.
31. Singha P. Introductory Biostatistics. Lagos: University of Lagos Press, 1996.
32. Sinha KA. Analytical Biochemistry,1972:47:389.
33. Song-Yi P, Christopher AH, Iona C, Sungshim LP, Laurence NK, Loic LM, *et al*. Racial/Ethnic Differences in Lifestyle-Related Factors and Prostate Cancer Risk: The Multiethnic Cohort Study, 2015. DOI: 10.1007/s10552-015-0644.
34. Trinder P. Enzymatic Colorimetric Determination of Triglycerides by GOP-PAP Method. Annals of Clinical Biochemistry,1969:6:24–27. https;//doi.org/10.1177/000563269006600108
35. Tiez W. Determination of Total Protein in Serum. Textbook of Clinical Chemistry, 1995.
36. Wilson RL. Exercise and Nutrition Induced Weight Loss for Prostate Cancer Patients. Edited Cowan University. Research Online, 2020. https//ro.ecu.edu.au/theses/2370
37. World Cancer Research International (WCRFI). Diet, Nutrition, Physical Activity and Prostate Cancer. Analyzing Research on Cancer Prevention and Survival, 2014. www.wcrf.org/sites/default/file/prostate-cancer-2014-report.
38. Ye RK, Chang KC, Young-Hoon L, Seong-Woo C, Hye-Yeon K, Min-Ho S, *et al*. Association between Albumin, Total Bilirubin and Uric Acid Serum Levels and Risk of Cancer: A Prospective Study in a Korean Population. Yonsei Medical Journal,2021:62(9):792–798. https://doi.org/10.3349/ymj.
39. Zhongyou X, Xueqin F, Xinzhu Y, Jinze L, Hao W, Jing S, *et al*. Serum Albumin to Globulin Ratio Prior to Treatment as a Potential Non-Invasive Prognostic Indicator for Urological Cancer. Frontiers in Nutrition, 2022. DOI: 103389/fnt.2022.1012181