**Evaluation of Prostate-Specific Antigen in Relation to Lipid Profile and Atherogenic Indices in Adult Males in Abuja**

**Abstract**

**Background:** Prostate-specific antigen (PSA) is widely used in detecting prostate disorders, though its specificity is limited. Emerging evidence suggests that dyslipidemia and atherogenic indices may influence PSA levels and potentially aid in assessing prostatic risk.

**Aim:** This study assessed the association between PSA levels, lipid profiles, and derived atherogenic indices in adult males in Abuja, Nigeria.

**Methods:** A hospital-based cross-sectional study was conducted from January to June 2025 among 150 healthy adult males attending two tertiary hospitals. Participants were categorized into three age groups: 18–39, 40–59, and 60 years and older. After overnight fasting, venous blood samples were collected. PSA was measured using electrochemiluminescence immunoassay. Lipid parameters, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were assessed enzymatically. Atherogenic indices were calculated using standard formulas. Data were analyzed using independent t-tests, Pearson correlation, and logistic regression at a significance level of p ≤ 0.05.

**Results:** Mean PSA values increased with age: 0.88 ± 0.20 ng/mL (18–39 years), 5.62 ± 15.99 ng/mL (40–59 years), and 13.09 ± 19.32 ng/mL (≥60 years). LDL-C levels were significantly higher in participants with elevated PSA (3.43 ± 1.24 mmol/L) than in those with normal PSA (2.93 ± 0.95 mmol/L, p = 0.009). Other lipid parameters (TC, TG, and HDL-C) and derived atherogenic indices showed no significant associations with PSA levels. Age ≥60 years was a strong predictor of elevated PSA (OR = 15.26, p < 0.001).

**Conclusion:** LDL-C showed a significant association with PSA levels. Although atherogenic indices were not predictive, integrating lipid profile screening with PSA testing could serve as adjunctive markers for prostate risk assessment, particularly in older men.

Keywords: Prostate-Specific Antigen, Lipid Profile, Atherogenic Indices, Prostate Disorders  
**1. INTRODUCTION**

Prostate cancer (PCa) remains one of the most commonly diagnosed malignancies in men worldwide, with over 1.4 million new cases reported annually. It contributes significantly to global cancer-related morbidity and mortality, particularly among older males (Rebbeck et al., 2020; World Health Organization [WHO], 2022). In sub-Saharan Africa, including Nigeria, the burden of prostate cancer and associated disorders is increasing, driven by aging populations, urbanization, and limited access to early screening and diagnostic services (Adeloye et al., 2016).

Prostate-specific antigen (PSA), a glycoprotein secreted by prostate epithelial cells, is widely used as a serum biomarker for detecting and monitoring prostate disorders like benign prostatic hyperplasia (BPH), prostatitis, and prostate cancer (Mottet et al., 2022). Despite its diagnostic value, PSA testing has limitations due to its low specificity; elevated PSA levels may occur in non-malignant conditions and after prostatic manipulation, contributing to false-positive results and potential overtreatment (Pinsky et al., 2017). This has prompted a growing interest in identifying complementary biomarkers that could enhance the specificity of PSA testing and improve risk stratification.

Emerging evidence suggests that metabolic dysregulation, particularly dyslipidemia, may play a contributory role in prostate carcinogenesis and PSA dynamics. Lipids, including total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides, influence membrane fluidity, steroidogenesis, and inflammatory signaling mechanisms implicated in prostate cell proliferation and tumor progression (Pelton et al., 2012; Freeman & Solomon, 2004). Elevated LDL-C, in particular, has been associated with increased PSA levels and heightened prostate cancer risk in several observational studies (Fang et al., 2019; Zhang et al., 2021).

In addition to individual lipid parameters, composite atherogenic indices such as Castelli Risk Index I and II (CRI-I and CRI-II), Atherogenic Index of Plasma (AIP), and Atherogenic Coefficient (AC) are established predictors of cardiovascular risk and reflect systemic lipid imbalance (Millán et al., 2009; Dobiásová, 2004). However, the association of these indices with PSA levels and prostate pathology remains underexplored, particularly in African populations where both cardiovascular and prostate diseases are increasingly prevalent.

Given the paucity of locally generated evidence, this study aimed to investigate the relationship between serum PSA levels, lipid profiles, and derived atherogenic indices in adult males in Abuja, Nigeria. Elucidating these associations may provide valuable insights into shared metabolic pathways impacting prostate health and contribute to improved screening strategies and disease prevention in resource-constrained settings.

**2. MATERIALS AND METHODS**

**Study Design:** The study employed an analytical cross-sectional design to recruit men aged 18 and above, at selected hospitals in Abuja, using systematic random sampling. This is because it examines the relationship between variables (PSA levels and lipid profiles/atherogenic indices) at a point in time. Furthermore, it aims to evaluate potential correlations rather than establish causation.

**Study Area:** The study was conducted in Abuja, Nigeria’s capital city, which has more than three million residents (National Population Commission, 2021). Research activities were carried out at two major public tertiary hospitals: the University of Abuja Teaching Hospital in Gwagwalada and the Federal Medical Centre, Jabi. These facilities provide specialized urological care, including PSA testing and prostate cancer management (Daramola et al., 2020). By recruiting from these hospitals, the study captured a sample of men from varied socio-economic backgrounds, reflecting the broader male population of Abuja

**Sample Size Determination**

The sample size was calculated using the correlation coefficient formula (Hulley *et al.,* 2013):

N = [(Zα + Zβ)/C] ² + 3Where:

N = required sample size

Zα = Standard normal deviate at 95% confidence level (1.96)

Zβ = Standard normal deviate at 80% power (0.84)

C = 0.5 × ln[(1+r)/(1-r)]

r = Expected correlation coefficient

Based on previous studies:

Expected correlation coefficient(r) between PSA and Total Cholesterol = 0.28(Zhang et al., 2020)

Confidence level = 95 %( α = 0.05)

Power = 80 %( β = 0.20)

Calculation:

1. C = 0.5 × ln [(1+0.28)/ (1-0.28)] = 0.288

2. N = [(1.96 + 0.84)/0.288]² + 3

3. N = 93.7

To account for potential attrition and non-response:

Adding 10% for attrition

Final sample size = 93.7 × 1.1 = 103 participants

Therefore, a minimum of 103 adult men were recruited for this study.

**Selection Criteria**

**Inclusion Criteria:** Participants in this study were adult males aged 18 years and above, residing in Abuja, Nigeria. Eligibility required a willingness to provide informed consent, ensuring that each participant understood the study’s purpose and procedures and voluntarily agreed to take part.

**Exclusion Criteria:** The study excluded men with a known history of prostate cancer or other malignancies to avoid disease-related bias. Additionally, individuals diagnosed with chronic kidney disease or liver disease were excluded, as these conditions could affect lipid metabolism and PSA levels. Men who were currently taking lipid-lowering medications or undergoing hormone therapy were also excluded to minimize the influence of external factors on the study’s biochemical measurements.

**Sample collection and Processing:** A systematic random sampling technique was employed, using the clinic’s patient register as the sampling frame; all third patient who met the inclusion criteria was selected until the desired sample size was achieved. Participants were instructed to fast for approximately 12 hours before their clinic appointment to reduce postprandial variations in lipid levels. Upon arrival, a trained phlebotomist collected 5 mL of venous blood from each participant into a plain vacutainer tube. The blood samples were then allowed to clot at room temperature, centrifuged at 3000 rpm for 10 minutes to separate the serum, and the serum was aliquoted into labeled cryovials. All serum samples were stored at –20°C until analysis to preserve biochemical stability.

### Laboratory Analysis

PSA was quantified using Roche Elecsys electrochemiluminescence immunoassay (ECLIA). Lipid parameters, total cholesterol (TC), LDL-C, HDL-C, and triglycerides (TG) were measured by enzymatic colorimetric assays on the ChemWell 2910 autoanalyzer (Awareness Tech, 2021).

Derived indices:

CRI-I = TC / HDL-C

CRI-II = LDL-C / HDL-C

AIP = log (TG / HDL-C)

AC = (TC – HDL-C) / HDL-C

### Statistical Analysis: Data were analyzed with Python libraries (Pandas, SciPy, StatsModels). Descriptive statistics summarized means and standard deviations. Group comparisons used t-tests; Pearson’s correlation evaluated associations; logistic regression identified predictors of elevated PSA (p ≤ 0.05).

**3. RESULT**

Age Group Distribution in the Study Population

The study participants comprised 150 adult male participants, stratified into three age groups: 18–39 years, 40–59 years, and 60 years and above (Table 1). Each age group contributed equally to the study population, with 50 participants each, representing 33.33% per group

The youngest age group (18–39 years) had the lowest mean PSA level of 0.88 ± 0.20 ng/ml, indicating relatively normal prostate function in this age range. Participants aged 40–59 years had a substantial increase in PSA levels, with a mean of 5.62 ± 15.99 ng/ml, a rising trend with age, and possible early prostatic changes. The oldest age group(60 years and above) recorded the highest mean PSA level of 13.09 ± 19.32 ng/ml, highlighting a greater risk of prostate abnormalities in older men. The progressive increase in mean PSA levels across the age categories reflects the well-established association between advancing age and elevated PSA concentrations. These findings underscore the importance of age-specific PSA screening and interpretation in clinical evaluations of prostate health among men. This trend indicates a clear age-related increase in PSA levels, suggesting that PSA concentrations tend to rise with advancing age due to physiological and pathological changes in the prostate.

**Table 1: Age Group Distribution in the Study Population**

|  |  |  |  |
| --- | --- | --- | --- |
| **Age Groups** | **Frequency**  **(n)** | **Percentage**  **(%)** | **PSA (ng/ml)**  **(mean ± SD)** |
| 18 – 39 | 50 | 33.33 | 0.88 ± 0.20 |
| 40 – 59 | 50 | 33.33 | 5.62 ± 15.99 |
| 60 and above | 50 | 33.33 | 13.09 ± 19.32 |

**3.2 Comparison of Lipid Profile Parameters between Men with Normal and Elevated PSA Levels in the Study Population**

The comparison of lipid profile parameters between men with normal and elevated PSA levels, stratified by age group (Table 2), reveals both age-dependent patterns and significant variations across several lipid markers. In the 18–39 years age group, all participants exhibited normal PSA levels. Consequently, no comparisons could be made between PSA categories for this age group. The mean values for lipid parameters in this group were: TC (4.56 ± 0.94 mmol/L), TG (1.04 ± 0.33 mmol/L), LDL (2.70 ± 0.75 mmol/L), and HDL (1.18 ± 0.38 mmol/L).

Among individuals aged 40 to 59 years, the mean total cholesterol(TC), low-density lipoprotein(LDL), and high-density lipoprotein(HDL) levels were higher in men with elevated prostate-specific antigen(PSA) compared to those with normal PSA. However, none of these differences were statistically significant. The mean TC was 5.40 ± 1.69 mmol/L in the elevated PSA group versus 4.94 ± 1.24 mmol/L in the normal group (p = 0.458). Similarly, LDL levels were 3.58 ± 1.16 mmol/L compared to 3.15 ± 1.15 mmol/L (p = 0.335), and HDL levels were 1.59 ± 0.53 mmol/L versus 1.34 ± 0.43 mmol/L (p = 0.206). Triglyceride(TG) levels were slightly lower in men with elevated PSA, measuring 1.09 ± 0.46 mmol/L compared to 1.20 ± 0.49 mmol/L(p = 0.541).

In the 60 years and above category, a significant difference in triglyceride levels was observed. Men with elevated PSA had significantly lower TG levels compared to those with normal PSA (1.04 ± 0.34 mmol/L vs. 1.33 ± 0.41 mmol/L, p = 0.012). While mean LDL and TC levels were slightly higher in the elevated PSA group (LDL: 3.39 ± 1.27 mmol/L vs. 3.05 ± 0.85 mmol/L, TC: 4.96 ± 1.58 mmol/L vs. 4.99 ± 1.24 mmol/L), the differences were not statistically significant (p = 0.267 and p = 0.945, respectively). HDL levels also showed no significant variation (p = 0.662).

These findings suggest that while lipid profiles appear non-significant in comparison across PSA groups in younger age categories, notable distinctions, particularly in triglyceride levels, emerge in older adults, underscoring the need to consider age-specific metabolic profiles in PSA evaluation and prostate health assessment.

**Table 2: Comparison of Lipid Profile Parameters between Men with Normal and Elevated PSA Levels by Age Group**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Age Group** | **Lipid Parameter** | **Normal PSA (mean ± SD)**  **(n = 110)** | **n\_normal** | **Elevated PSA (mean ± SD)**  **(n = 40)** | **n\_elevated** | ***P* –value** |
| 18 – 39 | TC | 4.56 ± 0.94 | 50 | - | 0 | - |
| (n = 50) | TG | 1.04 ± 0.33 |  | - |  | - |
|  | LDL | 2.70 ± 0.75 |  | - |  | - |
|  | HDL | 1.18 ± 0.38 |  | - |  | - |
| 40 – 59 | TC | 4.94 ± 1.24 | 41 | 5.40 ± 1.69 | 9 | 0.458 |
| (n = 50) | TG | 1.20 ± 0.49 |  | 1.09 ± 0.46 |  | 0.541 |
|  | LDL | 3.15 ± 1.15 |  | 3.58 ± 1.16 |  | 0.335 |
|  | HDL | 1.34 ± 0.43 |  | 1.59 ± 0.53 |  | 0.206 |
| 60+ | TC | 4.99 ± 1.24 | 19 | 4.96 ± 1.58 | 31 | 0.945 |
| (n = 50) | TG | 1.33 ± 0.41 |  | 1.04 ± 0.34 |  | 0.012 |
|  | LDL | 3.05 ± 0.85 |  | 3.39 ± 1.27 |  | 0.267 |
|  | HDL | 1.34 ± 0.46 |  | 1.28 ± 0.39 |  | 0.662 |

**3.3 Atherogenic Indices (CRI-I, CRI-II, AIP, and AC) Among Study Participants**

The results of the analysis of atherogenic indices in the study population (Table 3) show the distribution of atherogenic indices: Castelli Risk Index I (CRI-I), Castelli Risk Index II (CRI-II), Atherogenic Index of Plasma (AIP), and Atherogenic Coefficient (AC) among participants categorized by age group.

Among participants aged 18–39, the mean CRI-I was 4.13 ± 1.28, while CRI-II was 2.40 ± 0.86, both of which fall within the moderate cardiovascular risk range. The AIP showed a mean of -0.04 ± 0.21(median: -0.04, IQR: -0.14–0.06), suggesting a low atherogenic risk. The AC averaged 3.13 ± 1.28, also indicating a moderate risk level.

In the 40–59 age group, CRI-I slightly decreased to 3.84 ± 1.29, and CRI-II slightly increased to 2.52 ± 1.12. AIP decreased to -0.08 ± 0.23(median: -0.08, IQR: -0.21–0.05), remaining within the low-risk range. Similarly, the AC declined to 2.80 ± 1.29, showing a marginal reduction in lipid-related cardiovascular risk compared to the younger age group.

Participants aged 60 and older exhibited a mild increase in mean CRI-I (4.04 ± 1.42) and CRI-II (2.66 ± 1.00), suggesting a slight rise in lipid-related cardiovascular risk among older individuals. The AIP, while highly variable (-0.35 ± 2.27), had a median of -0.04(IQR: -0.20–0.10), remaining within the low-risk category. Notably, the AC displayed a significant increase in mean value to 7.25 ± 29.00, largely influenced by an extreme outlier (maximum value of 208.00). Despite this, the median AC (2.96, IQR: 2.19–4.09) stayed consistent with younger age groups, indicating that the high mean does not accurately represent the typical participant in this age category.

In summary, the atherogenic indices (particularly CRI-I and CRI-II) remained within moderate ranges across all age groups, with only mild fluctuations observed as age progressed. AIP consistently stayed in the low-risk category, indicating a more stable and reliable measure of atherogenic risk. The substantial variation in AC among older participants highlights the influence of outliers and the need to consider both mean and median values for a balanced interpretation of lipid-related cardiovascular risk across age groups.

**Table 3: Atherogenic Indices (CRI-I, CRI-II, AIP, and AC) by Age Group among Study Participants**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Age Group** | **Parameter** | **Mean ± SD** | **Min** | **Max** | **Median (IQR)** |
| 18 – 39 | Castelli Risk Index I (CRI-I) | 4.13 ± 1.28 | 2.13 | 8.37 | 3.98 (3.17–4.88) |
|  | Castelli Risk Index II (CRI-II) | 2.40 ± 0.86 | 1.26 | 5.19 | 2.17 (1.84–2.94) |
|  | Atherogenic Index of Plasma (AIP) | -0.04 ± 0.21 | -0.67 | 0.29 | -0.04 (-0.14–0.06) |
|  | Atherogenic Coefficient (AC) | 3.13 ± 1.28 | 1.13 | 7.37 | 2.99 (2.17–3.89) |
| 40 – 59 | Castelli Risk Index I (CRI-I) | 3.84 ± 1.29 | 1.55 | 8.52 | 3.46 (3.15–4.37) |
|  | Castelli Risk Index II (CRI-II) | 2.52 ± 1.12 | 0.55 | 6.07 | 2.24 (1.87–2.96) |
|  | Atherogenic Index of Plasma (AIP) | -0.08 ± 0.23 | -0.54 | 0.53 | -0.08 (-0.21–0.05) |
|  | Atherogenic Coefficient (AC) | 2.80 ± 1.29 | 0.55 | 7.52 | 2.44 (2.15–3.30) |
| 60+ | Castelli Risk Index I (CRI-I) | 4.04 ± 1.42 | 0.95 | 7.99 | 3.78 (3.12–4.90) |
|  | Castelli Risk Index II (CRI-II) | 2.66 ± 1.00 | 1.09 | 4.67 | 2.33 (1.94–3.58) |
|  | Atherogenic Index of Plasma (AIP) | -0.35 ± 2.27 | -16.00 | 0.51 | -0.04 (-0.20–0.10) |
|  | Atherogenic Coefficient (AC) | 7.25 ± 29.00 | 0.84 | 208.00 | 2.96 (2.19–4.09) |

**3.4 Comparison of Lipid Profile Parameters and Atherogenic Indices between Men with Normal and Elevated PSA Levels**

The analysis comparing lipid profile parameters and atherogenic indices between men with normal and elevated prostate-specific antigen(PSA) levels(Table 4) indicates that total cholesterol(TC) was slightly higher in the elevated PSA group(5.06 ± 1.59 mmol/L) compared to the normal PSA group(4.78 ± 1.12 mmol/L), however, this difference was not statistically significant(p = 0.229).

Triglyceride (TG) levels were lower in the elevated PSA group (1.05 ± 0.36 mmol/L) compared with the normal PSA group (1.15 ± 0.42 mmol/L), with a p-value of 0.185, suggesting no significant difference.

However, low-density lipoprotein (LDL) cholesterol levels were significantly higher in the elevated PSA group (3.43 ± 1.24 mmol/L) compared to the normal PSA group (2.93 ± 0.95 mmol/L), resulting in a p-value of 0.009. This indicates a statistically significant increase and may suggest a link between elevated PSA levels and increased LDL cholesterol.

High-density lipoprotein (HDL) levels were slightly higher in men with elevated PSA (1.35 ± 0.44 mmol/L) compared to those with normal PSA (1.27 ± 0.42 mmol/L), but the difference was not statistically significant (p = 0.268).

The Atherogenic Index of Plasma (AIP) showed more negative values in the elevated PSA group (-0.48 ± 2.53) compared to the normal group (-0.04 ± 0.22); however, the large standard deviation in the elevated group and a p-value of 0.075 indicate high variability and a lack of statistical significance.

For the Castelli Risk Indices, CRI-I was almost identical between groups (4.02 ± 1.38 vs. 3.96 ± 1.19, p = 0.812), and CRI-II was also not significantly different (2.47 ± 1.01 vs. 2.68 ± 0.97, p = 0.260).

The Atherogenic Coefficient (AC) was higher in the normal PSA group (4.89 ± 19.59) compared to the elevated PSA group (3.03 ± 1.17); however, the difference was not significant (p = 0.549). The wide standard deviation in the normal PSA group indicates the presence of extreme outliers.

In summary, among the lipid parameters and atherogenic indices compared between men with normal and elevated PSA levels, only LDL cholesterol showed a statistically significant difference, which is higher in the elevated PSA group. Other parameters, including TC, TG, HDL, AIP, CRI-I, CRI-II, and AC, did not show significant differences; however, AIP and AC values exhibited high variability, particularly in the elevated PSA group, likely due to outliers.

**Table 4: Comparison of Lipid Profile Parameters and Atherogenic Indices between Men with Normal and Elevated PSA Levels**

|  |  |  |  |
| --- | --- | --- | --- |
| **Parameter** | **Normal PSA**  **(Mean ± SD)** | **Elevated PSA (Mean ± SD)** | **P –value** |
| Total Cholesterol (TC) | 4.78 ± 1.12 | 5.06 ± 1.59 | 0.229 |
| Triglycerides (TG) | 1.15 ± 0.42 | 1.05 ± 0.36 | 0.185 |
| LDL | 2.93 ± 0.95 | 3.43 ± 1.24 | 0.009 |
| HDL | 1.27 ± 0.42 | 1.35 ± 0.44 | 0.268 |
| Atherogenic Index of Plasma (AIP) | -0.04 ± 0.22 | -0.48 ± 2.53 | 0.075 |
| Castelli Risk Index I (CRI-I) | 4.02 ± 1.38 | 3.96 ± 1.19 | 0.812 |
| Castelli Risk Index II (CRI-II) | 2.47 ± 1.01 | 2.68 ± 0.97 | 0.260 |
| Atherogenic Coefficient (AC) | 4.89 ± 19.59 | 3.03 ± 1.17 | 0.549 |

P < 0.05 was considered statistically significant

**Discussion**

This study found that serum LDL-C levels were significantly higher among men with elevated PSA compared to those with normal PSA, suggesting that LDL-C may serve as a metabolic marker associated with prostate enlargement or other prostate conditions. These findings are consistent with reports from different populations, such as the study by Zhang, Chen, and Wang (2021) in Korean men and the large Chinese cohort examined by Fang et al. (2019), which similarly observed higher LDL-C in men with increased PSA or prostate disease. Together, these results reinforce the hypothesis that lipid metabolism, particularly LDL-C, may influence PSA levels and potentially contribute to prostate pathology.

Mechanistically, elevated LDL-C can lead to greater cholesterol accumulation in the membranes of prostatic epithelial cells, which may alter androgen receptor signaling and stimulate cell proliferation (Pelton et al., 2012). Freeman and Solomon (2004) also described how cholesterol-rich membrane rafts could promote oncogenic signaling pathways within the prostate, potentially facilitating tumor development. These biological mechanisms provide a plausible explanation for the association between higher LDL-C and increased PSA observed in this study.

Interestingly, the composite atherogenic indices CRI-I, CRI-II, AIP, and AC did not emerge as significant predictors of elevated PSA in this cohort. This aligns with earlier findings from Nigerian studies such as Eze et al. (2020) and more recent work by Ibrahim et al. (2023), both of which reported a lack of association between these indices and PSA levels. While these indices are established markers for cardiovascular risk (Millán et al., 2009; Dobiásová, 2004), our findings suggest they may not effectively reflect prostate-specific metabolic changes. This distinction highlights that while lipid ratios capture systemic cardiovascular risk, individual lipid fractions like LDL-C may be more relevant for prostate disease.

Age ≥60 years was identified as the strongest independent predictor of PSA elevation, consistent with the well-documented increase in prostate volume, benign hyperplasia, and risk of malignancy associated with aging (Siegel et al., 2020; Gunes et al., 2016; Mottet et al., 2022). These age-related changes support the clinical recommendation to use age-adjusted PSA reference ranges (Pinsky et al., 2017; US Preventive Services Task Force, 2018), which can help improve screening specificity and reduce unnecessary biopsies among older men.

The correlation analysis in this study revealed a strong positive relationship between total cholesterol and LDL-C (r = 0.85–0.92, p < 0.001), confirming LDL-C as a major contributor to total serum cholesterol. This pattern mirrors broader lipid metabolism trends described by Alberti et al. (2009), where LDL-C accounts for a substantial proportion of total cholesterol levels. In contrast, HDL-C showed only moderate correlations, suggesting a more independently regulated role in lipid homeostasis.

Beyond the statistical associations, these findings may have practical implications for prostate disease screening in Nigerian men and similar populations. Including lipid profile assessments, particularly LDL-C, in routine PSA testing could improve risk stratification, especially in men aged 60 and above who are at higher baseline risk. Additionally, lifestyle interventions that lower LDL-C, such as dietary modification and statin use, have been associated with reductions in serum PSA levels (Chan et al., 2005; Alpert, 2018), hinting at potential dual benefits for cardiovascular and prostate health.

While this study's cross-sectional design limits causal interpretations, it contributes valuable localized evidence by highlighting LDL-C’s potential role in prostate health, an area previously underexplored in Nigerian settings. The data suggest that LDL-C may have a more direct relationship with PSA elevation compared to composite lipid ratios, supporting a more targeted approach to prostate risk assessment. Collectively, these findings emphasize the value of integrating metabolic profiling, demographic risk factors like age, and clinical history to enhance early detection and personalized management of prostate disorders. Such an integrated strategy could be particularly impactful in resource-limited settings, where access to specialized prostate care is constrained.

Ultimately, this study adds to the growing body of evidence linking metabolic health with prostate disease and underscores the importance of further longitudinal research to clarify these associations and inform evidence-based screening guidelines tailored to local populations.

**Conclusion:** This study examined the relationship between prostate-specific antigen (PSA), serum lipid profiles, and atherogenic indices among adult males in Abuja, Nigeria. The findings revealed a significant positive association between elevated LDL-C and PSA levels, suggesting that LDL-C may have a role in prostate biology and could complement PSA in assessing prostate risk. In contrast, composite atherogenic indices did not independently predict PSA elevation.

Age ≥60 years emerged as the strongest predictor of higher PSA levels, highlighting the influence of advancing age on prostate changes. The results support the potential clinical benefit of including lipid profile testing, particularly LDL-C measurement, in routine prostate health assessments to improve early detection and risk stratification. Although this study’s cross-sectional design limits conclusions about causality, it provides valuable local evidence and suggests the need for larger prospective studies that explore additional metabolic and inflammatory markers. Integrating metabolic, demographic, and clinical factors could ultimately enhance early detection and personalized management of prostate disorders in this setting.

**Ethical Approval & Consent**: Ethical approval was obtained from UATH (UATH/HREC/PR/607) and FMC Jabi (FMCABJ/HREC/2024/196). Participants provided written informed consent; data were anonymized and stored securely, according to the Declaration of Helsinki (WMA, 2013).

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

**REFERENCES**

Adeloye, D., David, R. A., Aderemi, A. V., Iseolorunkanmi, A., Oyedokun, A., Iweala, E. E., et al. (2016). An estimate of the incidence of prostate cancer in Africa: A systematic review and meta-analysis. BMC Cancer, 16(1), 1–10. <https://doi.org/10.1186/s12885-016-2973-7>

Alberti, K. G., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., ... and Smith, S. C. (2009). Harmonizing the metabolic syndrome. Circulation, 120(16), 1640–1645. <https://doi.org/10.1161/CIRCULATIONAHA.109.192644>

Allott, E. H., and Hursting, S. D. (2015). Obesity and cancer: Mechanistic insights from transdisciplinary studies. Endocrine-Related Cancer, 22(6), R365–R386. <https://doi.org/10.1530/ERC-15-0233>

Alpert, P. F. (2018). New evidence for the benefit of prostate-specific antigen screening: Data from 400,887 Kaiser Permanente patients. *Urology, 118*, 119–126. https://doi.org/10.1016/j.urology.2018.05.018

Chan, J. M., Gann, P. H., & Giovannucci, E. L. (2005). Role of diet in prostate cancer development and progression. Journal of Clinical Oncology, 23(32), 8152–8160. https://doi.org/10.1200/JCO.2005.03.1626

Daramola, O. O., Banjo, A. A., Ogun, G. O., et al. (2020). Histopathological review of prostate biopsies in Nigeria. Pan African Medical Journal, 35, 52. https://doi.org/10.11604/pamj.2020.35.52.17556

Dobiásová, M. (2004). Atherogenic index of plasma [log (triglycerides/HDL-cholesterol)]: Theoretical and practical implications. Clinical Chemistry, 50(7), 1113–1115. <https://doi.org/10.1373/clinchem.2004.033175>

Eze, U. I., Chukwuonye, I. I., & Ohagwu, K. A. (2020). PSA and lipid profile in Nigerian males. African Health Sciences, 20(1), 315–322. <https://doi.org/10.4314/ahs.v20i1.39>

**Fang, F., Chang, Y.-J., Chu, P., Yang, C., Hsu, C., and Lin, Y. (2019).** Association of cholesterol levels with PSA. The Journal of Urology, 202(2), 315–321. <https://doi.org/10.1097/JU.0000000000000245>

Freeman, M. R., and Solomon, K. R. (2004). Cholesterol and prostate cancer. Nature Reviews Cancer, 4(8), 623–634. <https://doi.org/10.1038/nrc1416>

Gunes, S., Hekim, G. N., Arslan, M. A., and Asci, R. (2016). Effects of aging on the male reproductive system. *Journal of Assisted Reproduction and Genetics, 33*(4), 441–454. https://doi.org/10.1007/s10815-016-0663-y

Ibrahim, A., Musa, A. A., Bello, S. O., et al. (2023). PSA and lipid parameters in Nigerian men. West African Journal of Medicine, 40(2), 123–129. https://www.ajol.info/index.php/wajm/article/view/245831

Hulley, S. B., Cummings, S. R., Browner, W. S., Grady, D. G., and Newman, T. B. (2013). Designing clinical research (4th ed.). Lippincott Williams and Wilkins. https://www.elsevier.com/books/designing-clinical-research/hulley/978-1-4557-0658-7

Millán, J., Pintó, X., Muñoz, A., Zúñiga, M., Rubiés-Prat, J., Pallardo, L. F., et al. (2009). Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention. Vascular Health and Risk Management, 5, 757–765. National Population Commission. (2021). Population estimates for Nigeria. <https://nationalpopulation.gov.ng>

Mottet, N., van den Bergh, R. C. N., Briers, E., Bourke, L., Cornford, P., De Santis, M., et al. (2022). EAU–EANM–ESTRO–ESUR–ISUP–SIOG guidelines on prostate cancer. European Urology, 81(1), 124–143. <https://doi.org/10.1016/j.eururo.2021.07.005>

Pelton, K., Freeman, M. R., and Solomon, K. R. (2012). Cholesterol and prostate cancer. Current Opinion in Pharmacology, 12(6), 751–759. <https://doi.org/10.1016/j.coph.2012.07.007>

Pinsky, P. F., Prorok, P. C., Yu, K., and Kramer, B. S. (2017). PLCO prostate cancer screening trial. Journal of the National Cancer Institute, 109(4), djw292. <https://doi.org/10.1093/jnci/djw292>

Rebbeck, T. R. (2020). Prostate cancer genetics: Variation by race, ethnicity, and geography. Cancer Epidemiology, Biomarkers & Prevention, 29(9), 1681–1692. <https://doi.org/10.1158/1055-9965.EPI-19-1070>

Roche Diagnostics. (2020). Elecsys PSA Package Insert. <https://diagnostics.roche.com/global/en/products/params/elecsys-total-and-free-psa.html>

Siegel, R. L., Miller, K. D., and Jemal, A. (2020). Cancer statistics, 2020. *CA: A Cancer Journal for Clinicians, 70*(1), 7–30. https://doi.org/10.3322/caac.21590

Thompson, I. M., Pauler, D. K., Goodman, P. J., Tangen, C. M., Lucia, M. S., Parnes, H. L., et al. (2005). Assessing prostate cancer risk: Results from the Prostate Cancer Prevention Trial. JAMA, 294(1), 66–70. <https://doi.org/10.1001/jama.294.1.66>

US Preventive Services Task Force, Grossman, D. C., Curry, S. J., et al. (2018). Screening for prostate cancer: US Preventive Services Task Force recommendation statement. *JAMA, 319*(18), 1901–1913. <https://doi.org/10.1001/jama.2018.3710>

World Health Organization (WHO). (2022). Global Cancer Observatory. <https://gco.iarc.fr>

World Medical Association (WMA). (2013). World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects. JAMA, 310(20), 2191–2194. <https://doi.org/10.1001/jama.2013.281053>

Zhang, H., Chen, Z., and Wang, Z. (2021). Serum lipid levels and prostate cancer risk. The Prostate, 81(4), 240–248. <https://doi.org/10.1002/pros.24129>

Zhang, L., Tang, M., Wang, Y., et al. (2021). Dyslipidemia and prostate cancer risk. Scientific Reports, 11(1), 5682. https://doi.org/10.1038/s41598-021-84979-4