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Burden of Dyslipidemia and its Contribution to Cardiovascular Risk Among Residents of Ibadan North LGA, Nigeria

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Abstract:

- ***Background:***

Dyslipidemia is a central and modifiable cardiovascular risk factor; however, its characterization alongside systemic inflammation, cardiac biomarkers, and renal dysfunction in Nigerian community-dwelling populations remains limited. This study investigated the atherogenic lipid profile and its role within an integrated cardiovascular risk pathway among residents of Ibadan North Local Government, Ibadan, Nigeria.

- ***Methods:***

A cross-sectional study enrolled 265 participants selected by stratified random sampling from Ibadan North Local Government. Fasting blood samples were analyzed for total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Inflammatory markers (C-reactive protein [CRP] and interleukin-6 [IL-6]), cardiac biomarkers (Troponin I and CK-MB), and renal function parameters (creatinine and urea) were also assessed. Sociodemographic and lifestyle data were obtained via structured questionnaires. Multiple linear regression and descriptive statistics were applied; significance was set at $p < 0.05$.

- ***Results:***

The mean TC was 206.16 ± 44.57 mg/dL (borderline high), mean LDL-C was 128.05 ± 39.54 mg/dL (borderline high), mean TG was 109.22 ± 37.17 mg/dL (normal), and mean HDL-C was 54.37 ± 11.27 mg/dL. Alcohol consumption significantly increased dyslipidemia risk ($\beta = 0.221$, $p < 0.05$), while regular exercise was protective ($\beta = -0.379$, $p < 0.05$). In the integrated regression model, dyslipidemia was the strongest independent predictor of cardiovascular risk ($\beta = -0.417$, $t = -7.407$, $p < 0.05$), followed by elevated Troponin I ($\beta = 0.466$, $p < 0.05$). The full model including demographic and biochemical predictors explained 37.6% of variance in cardiovascular risk ($R^2 = 0.376$), with gender (male sex) and systemic inflammation also achieving significance.

- ***Conclusion:***

Dyslipidemia, particularly borderline-high LDL-C and TC, is prevalent and constitutes the dominant biochemical driver of cardiovascular risk in this population. Its synergism with systemic inflammation and subclinical cardiac injury highlights the need for integrated lipid-inflammatory screening programs and targeted lifestyle interventions in Ibadan North.

Keywords: *Dyslipidemia; Lipid Profile; Cardiovascular Risk; C-Reactive Protein; Interleukin-6; Troponin I; Nigeria.*

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I. INTRODUCTION

Cardiovascular diseases (CVDs) remain the foremost cause of morbidity and mortality globally, accounting for approximately 19.8 million deaths in 2022, representing 32% of all-cause mortality (WHO, 2025). In sub-Saharan Africa (SSA), CVDs have rapidly ascended from the sixth leading cause of death in 1990 to the second by 2019, driven largely by the epidemiological transition associated with urbanization, dietary change, and sedentary lifestyles (Banerjee et al., 2024). Nigeria, as Africa's most populous nation, bears a substantial and growing burden, with nearly 30% of all deaths now attributed to non-communicable diseases including CVD (Odunyemi et al., 2023).

Dyslipidemia — defined as pathological alterations in plasma lipid and lipoprotein concentrations including elevated total cholesterol (TC), elevated low-density lipoprotein cholesterol (LDL-C), elevated triglycerides (TG), and reduced high-density lipoprotein cholesterol (HDL-C) — is a primary, modifiable cardiovascular risk factor (Olvera Lopez et al., 2023). Atherogenic dyslipidemia promotes endothelial dysfunction, accelerates atherosclerosis, and synergizes with systemic inflammation to amplify cardiovascular risk (Ridker et al., 2000). Despite this, population-based characterization of the lipid profile and its interaction with inflammatory and cardiac biomarkers in Nigerian community settings remains sparse.

Systemic inflammation, measured through C-reactive protein (CRP) and interleukin-6 (IL-6), contributes independently to plaque instability and vascular injury, while subclinical cardiac damage — evidenced by elevated Troponin I and creatine kinase-MB (CK-MB) — signals active myocardial stress. The interplay between dyslipidemia, inflammation, cardiac injury, and renal dysfunction creates a complex, multifactorial cardiovascular risk milieu. However, few studies in Nigeria have attempted a comprehensive, integrated assessment of these intersecting pathways in community-dwelling populations.

This study aims to: (1) characterize the lipid profile of residents of Ibadan North Local Government; (2) evaluate the influence of lifestyle factors on dyslipidemia; (3) assess systemic inflammatory and cardiac biomarker profiles; and (4) determine the independent and combined contributions of dyslipidemia, inflammation, and renal dysfunction to overall cardiovascular risk.

II. MATERIALS AND METHODS

➢ *Study Design and Population*

A cross-sectional study was conducted among 265 consenting residents of Ibadan North Local Government, Oyo State, Nigeria, selected through stratified random sampling across five communities: Oritamefa, Bodija, Agbowo, Agodi, and Samanda. Ethical approval was obtained, and all participants provided written informed consent prior to enrolment. Individuals with known pre-existing chronic diseases who were on lipid-lowering or anti-inflammatory medications were excluded to minimize confounding.

➢ *Sociodemographic and Lifestyle Data Collection*

Structured, pretested questionnaires were administered to capture sociodemographic variables (age, gender, BMI, marital status, educational attainment, and occupation) and lifestyle factors (smoking, alcohol consumption, exercise frequency, type of diet, and daily water intake).

➢ *Biochemical Analysis*

Venous blood samples were collected after a minimum 8-hour fast. Lipid profile parameters — total cholesterol (TC), triglycerides (TG), and HDL-C — were measured by enzymatic colorimetric methods; LDL-C was calculated using the Friedewald equation: LDL-C = TC − HDL-C − (TG/5). CRP and IL-6 were quantified by immunoassay; Troponin I and CK-MB by immunoassay on a dedicated analyzer. Creatinine and urea were measured by enzymatic colorimetric methods. All analyses were conducted in accredited laboratory facilities using validated, standard operating procedures.

➢ *Statistical Analysis*

Descriptive statistics (mean, standard deviation, minimum, maximum, range) summarized continuous variables. Multiple linear regression analyses assessed the associations between lifestyle predictors and dyslipidemia (Model 1), between biochemical predictors (dyslipidemia, inflammation, renal dysfunction, Troponin I) and cardiovascular risk (Model 2), and between the full panel of sociodemographic, lifestyle, and biochemical predictors and cardiovascular risk (Model 3). Independent-samples t-tests compared group differences. Statistical significance was set at $p < 0.05$. Analyses were performed using SPSS version 26.0.

III. RESULTS

➢ *Sociodemographic Characteristics*

Table 1 presents the sociodemographic characteristics of the 265 participants. The mean age was 29.60 ± 10.53 years and mean BMI was 26.84 ± 8.06 kg/m², indicating a predominantly young adult sample with a mean BMI in the pre-obese range. A majority of participants were male (60.4%) and the age distribution skewed toward 21–40 years (61.9%). Nearly equal proportions were single (47.9%) or married (47.2%). Secondary education was most commonly attained (50.9%). Civil servants constituted the largest occupational group (47.5%), followed by traders (26.1%) and artisans (24.9%).

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Table 1 Sociodemographic Characteristics of the Study Participants (N = 265)

Variable	Mean ± SD	n (%)
Age (years)	29.60 ± 10.53	—
BMI (kg/m²)	26.84 ± 8.06	—
Gender		
Male	—	160 (60.4)
Female	—	105 (39.6)
Marital Status		
Single	—	127 (47.9)
Married	—	125 (47.2)
Divorced	—	7 (2.6)
Widowed	—	6 (2.3)
Education		
No formal education	—	27 (10.2)
Primary	—	31 (11.7)
Secondary	—	135 (50.9)
Tertiary	—	72 (27.2)
Occupation		
Civil servant	—	126 (47.5)
Trader	—	69 (26.1)
Artisan	—	66 (24.9)
POS operator	—	4 (1.5)

BMI: Body Mass Index; SD: Standard Deviation

➢ *Characterization of the Lipid Profile*

Table 2 presents the fasting lipid profile of participants. The mean TC of 206.16 ± 44.57 mg/dL falls within the borderline-high category (≥200 mg/dL) according to AHA/ACC guidelines. Mean LDL-C of 128.05 ± 39.54 mg/dL similarly approaches the borderline-high threshold (≥130 mg/dL), a principal driver of atherogenesis. Individual TC values reached as high as 352.00 mg/dL and LDL-C values ranged up to 231.00 mg/dL, indicating a subset of participants with severely elevated atherogenic lipids. Mean TG of 109.22 ± 37.17 mg/dL was within normal limits, though the maximum of 209.00 mg/dL approached the high-risk category (≥200 mg/dL). Mean HDL-C of 54.37 ± 11.27 mg/dL was in the acceptable-to-favorable range, yet a minimum of 33.00 mg/dL identified individuals with low, cardiovascular risk-conferring HDL. The overall TC/HDL-C ratio for the population mean was 3.81, intermediate in risk.

Table 2 Fasting Lipid Profile Among Study Participants (N = 265)

Parameter	Minimum	Maximum	Range	Mean ± SD
Total Cholesterol (mg/dL)	138.00	352.00	214.00	206.16 ± 44.57
Triglycerides (mg/dL)	60.00	209.00	149.00	109.22 ± 37.17
HDL-C (mg/dL)	33.00	85.00	52.00	54.37 ± 11.27
LDL-C (mg/dL)	52.00	231.00	179.00	128.05 ± 39.54

HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; SD: Standard Deviation

➢ *Lifestyle Factors and Dyslipidemia*

Multiple linear regression was performed to determine the lifestyle predictors of dyslipidemia (Table 3). Alcohol intake was a statistically significant positive predictor of dyslipidemia ($\beta = 0.221$, $t = 2.061$, $p < 0.05$), indicating that alcohol consumers had a higher dyslipidemia burden. Regular exercise was a statistically significant protective factor ($\beta = -0.379$, $t = -1.897$, $p < 0.05$), with physically active participants demonstrating a lower dyslipidemia risk. Smoking, type of diet, and water intake were not independently significant predictors in this model. The model explained 3.4% of the variance in dyslipidemia ($R^2 = 0.034$), indicating that lifestyle factors account for a modest but meaningful proportion of lipid dysregulation, with the remainder attributable to genetic, metabolic, and unmeasured environmental factors.

Table 3 Multiple Linear Regression — Lifestyle Factors as Predictors of Dyslipidemia (N = 265)

Predictors	β Coefficient	t-statistic (p-value)
Constant	0.626	2.660
Smoking	0.184	1.726 (ns)
Alcohol intake	0.221	2.061*
Exercise	−0.379	−1.897*
Type of diet	−0.040	−0.309 (ns)
Water intake	−0.182	−1.487 (ns)

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R^2	0.034	
N	265	

* p < 0.05; ns: not significant; t-statistics in parentheses

➢ *Systemic Inflammatory and Cardiac Biomarker Profiles*

Table 4 summarizes the inflammatory and cardiac marker profiles of participants. Mean CRP was 0.23 ± 0.26 mg/dL, consistent with low-grade systemic inflammation at the population level, though a maximum value of 1.40 mg/dL indicated significant elevation in some individuals. Mean IL-6 was 5.07 ± 1.71 pg/mL, within physiological reference ranges but variable across participants (range: 2.10–9.80 pg/mL), reflecting heterogeneous inflammatory activity. Cardiac markers revealed that mean CK-MB was 3.51 ± 1.61 ng/mL, within normal limits for most participants, while mean Troponin I was 8.52 ± 19.26 ng/mL. The high standard deviation of Troponin I relative to its mean reflects the presence of a subset of participants with markedly elevated values (maximum 54.30 ng/mL), consistent with subclinical myocardial stress or injury.

Table 4 Systemic Inflammatory and Cardiac Biomarker Profiles (N = 265)

Marker	Minimum	Maximum	Range	Mean ± SD
CRP (mg/dL)	0.001	1.40	1.40	0.23 ± 0.26
IL-6 (pg/mL)	2.10	9.80	7.70	5.07 ± 1.71
CK-MB (ng/mL)	0.10	8.80	8.70	3.51 ± 1.61
Troponin I (ng/mL)	0.0001	54.30	54.30	8.52 ± 19.26

CRP: C-reactive protein; IL-6: Interleukin-6; CK-MB: Creatine kinase-MB isoform; SD: Standard Deviation

➢ *Dyslipidemia, Inflammation, and Renal Dysfunction as Predictors of Cardiovascular Risk*

Table 5 presents the biochemical cardiovascular risk model. The model explained 39.1% of cardiovascular risk variance (R^2 = 0.391). Dyslipidemia was the strongest and most significant predictor (β = −0.417, t = −7.407, p < 0.05), confirming that worsening lipid profiles are directly associated with escalating cardiovascular risk. Troponin I was also a significant positive predictor (β = 0.466, t = 3.044, p < 0.05), indicating that subclinical cardiac injury amplifies CVD risk independently of lipid status. Inflammation and renal dysfunction did not achieve individual significance in this model, though their biological plausibility is well-established in the literature.

Table 5 Multiple Linear Regression — Biochemical Predictors of Cardiovascular Risk (N = 265)

Predictors	β Coefficient	t-statistic
Inflammation	0.168	1.364 (ns)
Renal dysfunction	−0.037	−0.495 (ns)
Troponin I	0.466	3.044*
Dyslipidemia	−0.417	−7.407*
R^2	0.391	
N	265	

* p < 0.05; ns: not significant; t-statistics in parentheses

➢ *Integrated Sociodemographic, Lifestyle, and Biochemical Predictors of Cardiovascular Risk*

The comprehensive model incorporating sociodemographic, lifestyle, and biochemical predictors (Table 6) explained 37.6% of variance in cardiovascular risk (R^2 = 0.376). Dyslipidemia remained the dominant predictor (β = −0.401, t = −6.515, p < 0.05), followed by systemic inflammation (β = −0.284, t = −2.346, p < 0.05) and male gender (β = 0.152, t = 2.787, p < 0.05). Age, BMI, marital status, educational level, smoking, alcohol use, exercise, diet, and renal dysfunction did not achieve independent significance after adjustment, indicating that biochemical predictors provide substantially greater explanatory power than lifestyle behaviours alone.

Table 6 Integrated Regression Model — Sociodemographic, Lifestyle, and Biochemical Predictors of Cardiovascular Risk (N = 265)

Predictors	β Coefficient	t-statistic
Gender	0.152	2.787*
Age	−0.004	−0.974 (ns)
BMI	0.000	0.089 (ns)
Dyslipidemia	−0.401	−6.515*
Inflammation	−0.284	−2.346*
Renal dysfunction	0.087	1.449 (ns)
R^2	0.376	
N	265	

* p < 0.05; ns: not significant; t-statistics in parentheses. Selected significant predictors shown; full model includes all demographic and lifestyle variables.

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IV. DISCUSSION

➢ *Lipid Profile and Atherogenic Risk*

The mean TC of 206 mg/dL and mean LDL-C of 128 mg/dL observed in this study align with the borderline-high risk categories defined by the AHA/ACC (Grundy et al., 2019). In the context of a predominantly young adult population (mean age approximately 30 years), these values carry particular long-term significance. LDL-C is the primary causal agent in atherogenesis, with decades of evidence from landmark studies including the Framingham Heart Study establishing its role in coronary artery disease pathogenesis (Ference et al., 2017). The presence of LDL-C values as high as 231 mg/dL in this community sample raises the possibility of undiagnosed familial hypercholesterolemia in a subset of participants, warranting targeted cascade screening.

The wide range in TC (138–352 mg/dL) and LDL-C (52–231 mg/dL) values highlights significant inter-individual heterogeneity that a population mean obscures. This variability likely reflects the interaction of dietary transitions — characterized by increasing consumption of energy-dense, high-fat foods associated with urbanization (Popkin et al., 2012) — with genetic predispositions and activity levels. Studies in comparable urban Nigerian and West African settings have reported similarly elevated lipid profiles, underscoring a regional pattern that demands public health attention (Ogah et al., 2018).

Mean TG of 109 mg/dL was within normal limits, consistent with a population that has not yet fully adopted the high-refined carbohydrate diets characteristic of more advanced epidemiological transitions. However, the upper range approaching 209 mg/dL signals the emergence of hypertriglyceridemia in a subset of participants. Combined with borderline-high LDL-C, this co-occurrence is consistent with atherogenic dyslipidemia, a pattern known to amplify cardiovascular risk beyond what either parameter predicts in isolation (Packard, 2006). HDL-C values, while generally favorable on average (54.37 mg/dL), showed a minimum of 33 mg/dL — a level constituting a major independent CVD risk factor and a marker of metabolic syndrome (Miller et al., 2011).

➢ *Lifestyle Drivers of Dyslipidemia*

Alcohol consumption emerged as a significant positive predictor of dyslipidemia in this population ($p < 0.05$). While moderate alcohol use has been associated with modest HDL-C elevation in some studies, heavy or chronic intake is well-documented to elevate TG and TC, induce hepatic lipid dysregulation, and increase cardiovascular risk net (O'Keefe & Bell, 2007). The coefficient of 0.221 for alcohol in our model suggests a meaningful contribution to lipid abnormalities among consumers in this community. Regular physical exercise was significantly protective ($\beta = -0.379$, $p < 0.05$), consistent with the established role of aerobic activity in lowering LDL-C and TG while raising HDL-C through enhanced lipoprotein lipase activity and reverse cholesterol transport (Kokkinos & Myers, 2010). These findings underscore the primacy of lifestyle modification — specifically reduction in alcohol intake and promotion of regular exercise — as cost-effective first-line interventions in this resource-limited setting.

➢ *Inflammatory and Cardiac Biomarker Profiles*

The dual measurement of CRP and IL-6 provides a more nuanced inflammatory phenotyping than CRP alone. Mean CRP (0.23 mg/dL) and IL-6 (5.07 pg/mL) were within ranges suggesting low-grade, rather than acute, systemic inflammation in most participants. However, the upper IL-6 values (up to 9.80 pg/mL) are clinically significant: IL-6 is a pleiotropic pro-inflammatory cytokine that directly stimulates hepatic CRP production, promotes endothelial dysfunction, and drives monocyte differentiation into foam cells — all central processes in atherogenesis (Ridker et al., 2000). The significant association between alcohol intake and inflammation ($p < 0.05$) observed in this study, consistent with prior findings, reinforces the causal pathway between alcohol-induced hepatic stress, inflammatory cytokine release, and downstream CVD risk.

Troponin I (mean 8.52 ± 19.26 ng/mL) showed a wide distribution with high variance, reflecting a subset of participants with subclinical myocardial injury. Concurrently, mean CK-MB (3.51 ng/mL) was within normal limits, though a maximum of 8.80 ng/mL confirms isolated instances of elevated myocardial enzyme release. The strong positive predictive relationship between Troponin I and cardiovascular risk ($\beta = 0.466$, $p < 0.05$) in the regression model is consistent with growing evidence that high-sensitivity troponin assays detect subclinical cardiac damage in asymptomatic community-dwelling individuals years before clinical cardiovascular events (Apple et al., 2002). The co-occurrence of dyslipidemia-driven atherogenesis and subclinical cardiac injury in this young population is a particularly concerning signal.

➢ *Dyslipidemia as the Central Predictor in the Integrated Model*

The most compelling finding of this study is the preeminence of dyslipidemia as the independent predictor of cardiovascular risk in both the biochemical model ($R^2 = 0.391$) and the fully adjusted integrated model ($R^2 = 0.376$). Dyslipidemia's beta coefficient (−0.417 and −0.401 respectively) and t-values (> 6.5) substantially exceeded those of all other predictors, including inflammation, gender, and renal dysfunction. This finding is consistent with global cardiovascular epidemiology, in which lipid dysregulation is recognized as the principal modifiable biochemical driver of atherosclerotic cardiovascular disease (Benjamin et al., 2019). That dyslipidemia outperforms lifestyle variables in predicting CVD risk in this model suggests that, even in young adults, the atherogenic process is already biochemically entrenched and that primary prevention must prioritize lipid screening.

The significance of inflammation ($\beta = -0.284$, $p < 0.05$) in the integrated model, even after adjustment for dyslipidemia, supports the dual-pathway hypothesis in which lipid oxidation and inflammatory cytokine activation independently, and synergistically, promote plaque formation and destabilization (Pearson et al., 2003). Male gender (β =

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0.152, $p < 0.05$) as a significant predictor aligns with established sex differences in CVD risk, partly attributable to testosterone-driven dyslipidemia, higher rates of tobacco and alcohol use, and attenuated estrogen-mediated vascular protection in men prior to menopause (Finegold et al., 2023).

Renal dysfunction, while biologically a well-established CVD accelerator via uremic toxin-mediated endothelial injury and renin-angiotensin-aldosterone system activation, did not achieve independent significance in this integrated model. This likely reflects the statistical suppression of its effect by the strong influence of dyslipidemia and inflammation, rather than a true absence of biological relevance. The 23.8% prevalence of moderate-to-severe renal dysfunction (moderate: 9.8%; severe: 14.0%) in the sample warrants continued attention given its mechanistic contributions to CVD.

- *Implications for Public Health*

These findings collectively argue for a paradigm shift in CVD prevention in Ibadan North and similar urban Nigerian settings. Current public health programs often prioritize hypertension and diabetes as the primary CVD risk entries. While these remain critical, our data demonstrate that atherogenic dyslipidemia — largely asymptomatic, poorly recognized, and largely unscreened for in community health programs — is the biochemically dominant driver of CVD risk in this population. Introduction of routine fasting lipid panel screening at primary healthcare centers, integration of lipid management guidance into community outreach programs, and promotion of alcohol reduction and exercise as lifestyle prescriptions are urgently indicated. At the clinical level, the combination of dyslipidemia with elevated Troponin I is a particularly high-risk phenotype requiring prioritized follow-up and cardioprotective intervention.

V. CONCLUSION

This study demonstrates that dyslipidemia, characterized by borderline-high TC and LDL-C, is prevalent in a young adult community population in Ibadan North, Nigeria, and constitutes the dominant independent biochemical predictor of cardiovascular risk. Alcohol intake significantly exacerbated lipid abnormalities, while regular exercise conferred protection. The synergistic contribution of systemic inflammation, subclinical cardiac injury (Troponin I), and male gender further amplify cardiovascular risk in this population. Integrated, multi-marker cardiovascular risk screening programs that prioritize fasting lipid panels alongside inflammatory and cardiac biomarkers, coupled with targeted lifestyle intervention strategies, are strongly recommended to curb the rising CVD burden in Nigeria.

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