



# Alcohol Consumption and Cardiovascular Remodeling in a Nigerian Population: Insights from Echocardiography, Electrocardiography, Carotid Imaging and Lipid Profile

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## ABSTRACT

## Original Research Article

**Introduction:** Alcohol consumption exerts a profound influence on cardiovascular health through intricate mechanisms. Chronic heavy drinking is strongly associated with conditions such as hypertension, arrhythmias, alcoholic cardiomyopathy, and atherosclerosis. While acute alcohol intake can lead to temporary effects like tachycardia, long-term excessive consumption causes irreversible cardiovascular damage. This study investigates alcohol's impact on cardiovascular health by examining parameters such as echocardiographic findings, lipid profiles, electrocardiograms (ECG), and carotid artery assessments, aiming to provide a comprehensive understanding of its effects on the cardiovascular system.

**Methods:** This cross-sectional study analyzed cardiovascular data, including echocardiographic metrics, lipid profiles, electrocardiographic markers and Carotid cIMT and Diameter. Participants were categorized by hypertension status (hypertensive, normotensive, prehypertensive), and smoking habits (smokers vs. non-smokers). Mean values with standard errors were compared across groups to explore differences, with particular attention to the interplay of these factors on cardiovascular health.

**Results:** A total of 53 participants were included in the study. Hypertension and smoking status were associated with measurable cardiovascular alterations, while advancing age demonstrated significant relationships with several cardiovascular parameters, including blood pressure levels and structural cardiac indices. Carotid imaging revealed variations in vascular parameters consistent with early vascular remodeling. Echocardiographic and electrocardiographic findings provided additional evidence of subclinical cardiovascular changes within the cohort.

**Conclusion:** This study provides preliminary evidence of structural and vascular cardiovascular changes among alcohol-consuming individuals in a Nigerian community. The findings highlight the importance of comprehensive cardiovascular assessment in populations with prevalent alcohol use and underscore the need for larger studies to further clarify the relationship between alcohol consumption and cardiovascular remodeling in African populations.

**Keywords:** Alcohol Consumption, Cardiovascular Remodeling, Echocardiography, Electrocardiography, Carotid Intima–Media Thickness.

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## Introduction

Cardiovascular diseases (CVD) remain the leading cause of

mortality worldwide, with their prevalence steadily increasing over the past two decades.<sup>1</sup>The interplay between

modifiable and non-modifiable risk factors has been established, with alcohol consumption emerging as a critical area of focus.<sup>2</sup> There have been controversial views on the impact of alcohol on cardiovascular health.<sup>3</sup> The view that had been held over the years and even validated by studies is that moderate consumption has been linked to potential protective effects, whereas excessive and chronic intake poses significant risks.<sup>4</sup> The World Heart Federation,<sup>5</sup> through its 2022 policy brief, debunked the widely held notion that moderate alcohol consumption reduces the risk of heart disease, asserting that such claims are myths. Consequently, calling for stricter control measures, particularly as alcohol has been implicated in the rising prevalence of cardiovascular diseases globally<sup>6</sup>.

The “French Paradox” brought the cardioprotective nature of alcohol into scientific discussions<sup>7</sup>. This phenomenon observed a lower prevalence of cardiovascular diseases among the French population, despite high alcohol consumption rates, compared to the American population<sup>8</sup>. Subsequent investigations<sup>9</sup> have sought to distinguish the protective components of alcohol, highlighting antioxidants like resveratrol over ethanol itself. Resveratrol has been noted to mitigate the prothrombotic effects of cholesterol, reduce LDL oxidation, and inhibit platelet aggregation, thereby influencing the pathogenesis of atherosclerosis.<sup>10</sup>

Despite these discussions, alcohol consumption has demonstrable adverse effects on various cardiovascular parameters.<sup>11</sup> Acute ingestion is associated with transient tachycardia and an elevation in blood pressure.<sup>12</sup> Chronic intake, however, has more severe implications, including arrhythmias, alcoholic cardiomyopathy, and hypertension.<sup>13</sup> Arrhythmias, particularly, stand out as one of the most prevalent complications in individuals with prolonged alcohol use<sup>14</sup>. Similarly, alcoholic cardiomyopathy, a mixed etiology disease with both genetic and acquired origins, represents another critical impact of alcohol on heart health.<sup>15</sup>

Hypertension, often classified as a modifiable risk factor, is heavily influenced by chronic alcohol consumption.<sup>16</sup> Studies have shown that alcohol elevates vascular intima-media thickness and impacts vessel distensibility, leading to arterial stiffness.<sup>17</sup> This contributes to increased blood pressure and, over time, hypertension<sup>18</sup>. Genetic predispositions also amplify the effect of alcohol on blood pressure.<sup>19</sup> For example, variations in the aldehyde dehydrogenase 2 (ALDH2) gene have shown differing responses to alcohol intake, influencing hypertension development.<sup>20</sup>

Beyond its effects on the heart and blood vessels, alcohol's role in infective endocarditis and endomyocardial fibrosis (EMF) has garnered attention.<sup>21</sup> Though research in humans is limited, studies in mice have identified a potential link between alcohol and EMF.<sup>22</sup> The contribution of alcohol to the inflammatory pathways, alongside its ability to affect vessel walls and endocardium, underscores the need for further exploration.<sup>23</sup>

Lipid metabolism is another area where alcohol exerts a significant influence.<sup>24</sup> Moderate alcohol consumption has been linked to increased HDL levels and reduced LDL levels, attributed to mechanisms such as CETP inhibition.<sup>25</sup> This interplay with cholesterol subtypes has been considered one possible explanation for its perceived cardioprotective effects, although excessive intake negates such benefits, contributing instead to adverse cardiovascular outcomes<sup>26</sup>.

Despite numerous studies evaluating the cardiovascular effects of alcohol, gaps remain. Few investigations have comprehensively analyzed the combined impact of alcohol on echocardiographic parameters, lipid profiles, carotid intima-media thickness, and electrocardiographic findings in a single population cohort.<sup>27</sup> Furthermore, cultural, ethnic, and genetic variations further complicate interpretations, as differences in alcohol metabolism and disease presentation vary widely across populations.<sup>28</sup>

This study, the Comprehensive Cardiovascular Impact of Alcohol Consumption Study (CCIACS), seeks to bridge these gaps by exploring the multifaceted effects of alcohol on cardiovascular health. By evaluating echocardiograms, lipid profiles, carotid intima-media thickness, and ECG results within the same study population, it aims to provide a holistic understanding of alcohol's impact. Findings from this study could contribute to nuanced public health strategies and further inform global discussions on the role of alcohol in cardiovascular health, especially in diverse populations where genetic and cultural factors interplay.<sup>29</sup>

## Method

### Study Design

This was a prospective, cross-sectional, community-based study.

### Study Site

The study was conducted in an ad-hoc laboratory created in the town hall of Rumuekini, a suburban community in Rivers State, Nigeria. The study took place over a period of two months.

### Ethical Consideration

Ethical clearance was obtained from the Community Development Committee of Rumuekini and the Royal Highness of the Rumuekini Community. Each study subject provided informed consent after a detailed explanation of the procedure and purpose of the study. Participants were assured of confidentiality and non-judgmental treatment.

### Study Population

Participants included consenting males from the Rumuekini community who consumed alcohol. All respondents were counseled on the study protocol, and consent was obtained from each subject. Diabetics were excluded based on blood sugar assessments.

## Sample Size

The study aimed to recruit at least 30 participants due to social stigma associated with alcohol consumption, achieving sample size of 30 was considered the minimum threshold for reliable data in statistical analysis based on the Central Limit Theorem (CLT). It recruited 53 participants all males as females refused to participate in the study for fear of being stigmatized.

## Data Collection

A questionnaire was used to collect details on biodata, occupation, symptoms, type of alcohol, estimated daily alcohol consumption, and smoking history. This was merged with a proforma for clinical findings, drug history, and recordings of electrocardiographic, echocardiographic, lipid profile, and carotid Doppler findings. Diabetics and those with known congenital heart disease were excluded from the study.

## Study Arms

### 1. Echocardiogram Arm recorded:

- Left atrial diameter (LAD)
- Left ventricular internal diameter in diastole (LVIDd)
- Interventricular septal thickness in diastole (IVSd)
- Left ventricular posterior wall thickness in diastole (LVPDd)
- Relative wall thickness (RWT)
- Left ventricular mass indexed to body surface area (LVM/BSA)
- Ejection fraction (EF)
- Fractional shortening
- E/A ratio

### 2. Electrocardiogram (ECG) Arm:

- P-wave amplitude and duration
- PR interval
- QRS duration
- ST segment duration
- QT interval

### 3. Lipid Profile Arm:

- Total cholesterol (TC)
- Low-density lipoprotein (LDL)
- High-density lipoprotein (HDL)
- Triglycerides (TG)

### 4. Carotid Vascular Scan Arm:

- Carotid intima-media thickness (cIMT) for right and left vessels.
- Average carotid artery diameter for right and left vessels

## Study Objectives

This study aims to evaluate the effects of alcohol consumption on cardiovascular health by utilizing

echocardiographic, electrocardiographic, lipid profile, and carotid Doppler findings. It also seeks to assess the impact of alcohol consumption on various subgroups, including smokers and non-smokers, hypertensives and non-hypertensives, as well as married and single individuals, focusing on different cardiovascular parameters. Furthermore, the study aims to identify independent predictors of adverse cardiovascular outcomes through multiple regression models and to explore the relationships between the quantity of alcohol consumption and cardiovascular parameters.

## Statistical Analysis

All statistical analyses were performed using SPSS 26 statistical software. Continuous variables were summarized as mean  $\pm$  standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between clinical subgroups, including hypertensive, prehypertensive, and normotensive participants, as well as smokers and non-smokers, were conducted using appropriate statistical tests depending on data distribution.

Correlation analysis was performed to evaluate relationships between cardiovascular parameters and selected clinical variables, including age, body mass index (BMI), blood pressure, smoking status, and alcohol consumption. Pearson correlation coefficients were calculated for normally distributed continuous variables. For variables that did not meet normality assumptions, non-parametric correlation analysis was applied.

Multiple linear regression models were subsequently used to explore the independent associations between selected cardiovascular parameters and clinical predictors. In these models, cardiovascular structural and functional indices—such as left ventricular diameter, left atrial diameter, carotid artery diameter, and carotid intima-media thickness—were treated as dependent variables. Independent variables included age, BMI, systolic blood pressure, diastolic blood pressure, smoking status, and alcohol consumption.

Regression analysis was performed to determine the relative contribution of these clinical variables to variations in cardiovascular measurements while controlling for potential confounding factors. The strength and direction of associations were expressed using regression coefficients and correlation coefficients where applicable.

A two-tailed p-value of less than 0.05 was considered statistically significant for all analyses.

## Results

The index study assessed diverse cardiovascular and physiological characteristics. The study participants had a mean age of  $32.44 \pm 13.66$  years. For electrocardiographic parameters, the P-axis was  $57.83 \pm 22.46^\circ$ , QRS-axis was  $52.77 \pm 45.56^\circ$ , and T-axis was  $35.66 \pm 40.30^\circ$ . The PR

interval averaged  $153.77 \pm 33.39$  ms, while the Pinterval was  $102.11 \pm 13.74$  ms, and the QRS interval was  $82.49 \pm 23.93$  ms. The T interval and corrected QT interval (QTc) measured  $156.86 \pm 46.30$  ms and  $375.82 \pm 55.70$  ms, respectively.

Lipid profile analysis revealed mean total cholesterol levels at  $182.21 \pm 44.58$  mg/dL, triglycerides (TG) at  $143.00 \pm 42.86$  mg/dL, low-density lipoprotein (LDL) at  $73.72 \pm 23.54$  mg/dL, and high-density lipoprotein (HDL) at  $64.72 \pm 12.27$  mg/dL. For cardiac dimensions, the right diameter (RTDIAM) averaged  $8.18 \pm 5.36$  cm, while the left diameter (LT DIAM0) was  $10.26 \pm 11.67$  cm.

Body mass index (BMI) was  $23.82 \pm 3.47$  kg/m<sup>2</sup>. Blood pressure showed mean systolic blood pressure (SBP) at  $130.13 \pm 21.37$  mmHg and diastolic blood pressure (DBP) at

$77.69 \pm 14.05$  mmHg. Pulse rate (PR) averaged  $74.04 \pm 10.93$  beats per minute. Alcohol consumption quantity was  $75.67 \pm 45.96$  units. Echocardiographic parameters included left atrial diameter (LAD) at  $3.49 \pm 0.45$  cm, aortic cusp separation (ACS) at  $2.31 \pm 0.43$  cm, interventricular septal diameter (IVSD) at  $1.28 \pm 1.23$  cm, left ventricular posterior wall diameter (LVPWD) at  $1.66 \pm 1.71$  cm, and left ventricular internal diastolic diameter (LVIDD) at  $4.71 \pm 0.79$  cm. Ejection fraction (EF) was  $61.95 \pm 14.86\%$ , fractional shortening (FS) was  $33.47 \pm 12.59\%$ , and the E/A ratio was  $1.53 \pm 0.46$ .

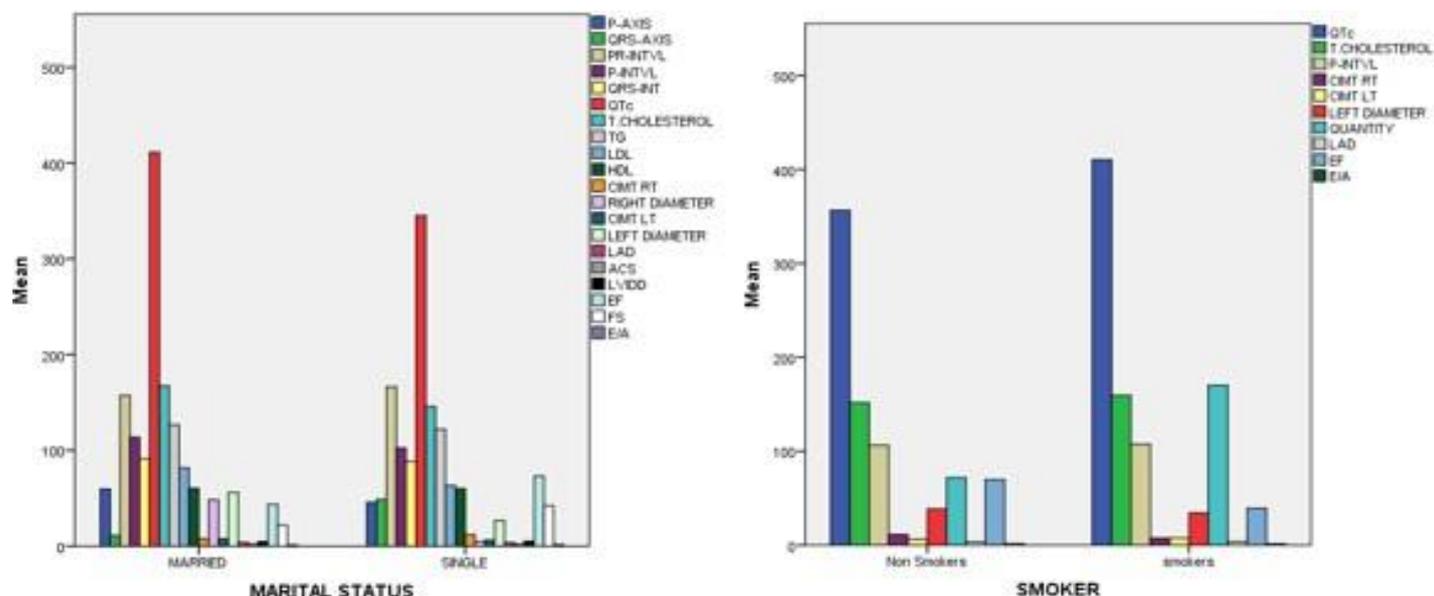
Comparison between the different independent variables, Hypertensives, non-hypertensives, Smokers, non-smokers, married and single participants types is as seen in table 1 and 2.

**Table 1:** Means of Dependent Variable son Smoking status and Blood Pressure

Dependent Variable	Grand Mean	Hypertensive Mean $\pm$ SE	Normotensives Mean $\pm$ SE	Prehypertension Mean $\pm$ SE	Smokers Mean $\pm$ SE(X)	Non-Smokers Mean $\pm$ SE (0)
Quantity (g)	96.83 $\pm$ 20.25	38.00 $\pm$ 42.95	125.25 $\pm$ 37.20	96.25 $\pm$ 30.37	150.25 $\pm$ 37.20	70.13 $\pm$ 24.01
AGE (years)	49.01 $\pm$ 12.74	42.75 $\pm$ 9.32	45.59 $\pm$ 10.46	52.78 $\pm$ 11.32	47.30 $\pm$ 13.45	50.61 $\pm$ 14.04
BMI (kg/m <sup>2</sup> )	24.45 $\pm$ 2.98	23.50 $\pm$ 2.51	24.18 $\pm$ 2.60	25.04 $\pm$ 3.09	24.12 $\pm$ 3.04	24.74 $\pm$ 2.89
LAD (cm)	3.71 $\pm$ 0.18	3.87 $\pm$ 0.19	3.63 $\pm$ 0.15	3.66 $\pm$ 0.19	3.78 $\pm$ 0.21	3.68 $\pm$ 0.15
LVIDD (cm)	4.85 $\pm$ 0.25	5.17 $\pm$ 0.30	4.67 $\pm$ 0.22	4.89 $\pm$ 0.29	4.73 $\pm$ 0.29	4.97 $\pm$ 0.27
EF (%)	62.40 $\pm$ 6.72	68.27 $\pm$ 9.05	59.77 $\pm$ 6.45	61.23 $\pm$ 7.85	58.98 $\pm$ 8.42	63.67 $\pm$ 7.03
FS (%)	32.10 $\pm$ 4.21	33.98 $\pm$ 3.62	31.83 $\pm$ 4.54	31.22 $\pm$ 4.03	30.75 $\pm$ 4.38	32.90 $\pm$ 4.42
ACS (cm <sup>2</sup> )	1.20 $\pm$ 0.52	1.45 $\pm$ 0.60	1.13 $\pm$ 0.50	1.19 $\pm$ 0.52	1.14 $\pm$ 0.57	1.27 $\pm$ 0.49
E/A Ratio	0.95 $\pm$ 0.19	1.11 $\pm$ 0.22	0.89 $\pm$ 0.17	0.94 $\pm$ 0.19	0.92 $\pm$ 0.21	0.97 $\pm$ 0.18
P-AXIS (°)	47.62 $\pm$ 13.03	45.36 $\pm$ 12.02	46.48 $\pm$ 11.58	48.92 $\pm$ 13.13	46.26 $\pm$ 11.35	47.85 $\pm$ 12.59
QRS-AXIS (°)	90.13 $\pm$ 20.48	88.33 $\pm$ 18.76	91.05 $\pm$ 19.24	92.07 $\pm$ 20.14	89.92 $\pm$ 19.65	90.32 $\pm$ 19.79
T-AXIS (°)	75.25 $\pm$ 15.82	74.18 $\pm$ 14.37	75.89 $\pm$ 15.13	76.29 $\pm$ 16.15	74.85 $\pm$ 15.36	75.64 $\pm$ 15.59
PR-INTVL (ms)	184.31 $\pm$ 42.88	180.94 $\pm$ 41.02	183.74 $\pm$ 42.21	185.25 $\pm$ 43.14	182.66 $\pm$ 42.36	184.96 $\pm$ 42.72
P-INTVL (ms)	115.48 $\pm$ 27.94	112.57 $\pm$ 26.73	114.26 $\pm$ 27.51	115.95 $\pm$ 28.13	114.42 $\pm$ 27.39	115.85 $\pm$ 27.72
QRS-INT (ms)	110.26 $\pm$ 15.43	109.42 $\pm$ 14.72	110.01 $\pm$ 15.12	111.03 $\pm$ 15.68	110.03 $\pm$ 15.34	110.48 $\pm$ 15.58
T-INTVL (ms)	250.91 $\pm$ 35.50	247.76 $\pm$ 33.91	249.89 $\pm$ 34.78	251.75 $\pm$ 36.13	249.51 $\pm$ 34.65	251.05 $\pm$ 35.15
QTc (ms)	397.45 $\pm$ 50.14	392.81 $\pm$ 48.98	395.26 $\pm$ 49.74	398.14 $\pm$ 50.74	395.67 $\pm$ 49.15	396.52 $\pm$ 50.38
Lt cIMT (cm)	112.53 $\pm$ 20.52	108.75 $\pm$ 18.90	110.58 $\pm$ 19.48	114.96 $\pm$ 20.83	112.11 $\pm$ 19.15	113.02 $\pm$ 20.05
Lt carotid Diam (cm)	3.49 $\pm$ 0.71	3.42 $\pm$ 0.69	3.46 $\pm$ 0.70	3.50 $\pm$ 0.73	3.46 $\pm$ 0.69	3.50 $\pm$ 0.71
Rt cIMT (mm)	8.77 $\pm$ 2.02	8.49 $\pm$ 1.90	8.66 $\pm$ 1.97	8.88 $\pm$ 2.06	8.73 $\pm$ 1.92	8.81 $\pm$ 2.04
Rt Carotid Diam (cm)	12.75 $\pm$ 3.32	12.58 $\pm$ 3.22	12.69 $\pm$ 3.27	12.82 $\pm$ 3.36	12.71 $\pm$ 3.25	12.78 $\pm$ 3.32
T. Cholesterol (mg/dL)	180.24 $\pm$ 30.53	176.86 $\pm$ 28.96	178.91 $\pm$ 29.45	181.37 $\pm$ 30.84	179.79 $\pm$ 29.33	180.69 $\pm$ 30.50
TG (mg/dL)	125.35 $\pm$ 15.97	122.97 $\pm$ 14.98	124.12 $\pm$ 15.48	125.89 $\pm$ 15.99	125.04 $\pm$ 15.73	125.56 $\pm$ 15.91
LDL (mg/dL)	90.57 $\pm$ 12.03	88.94 $\pm$ 11.58	89.76 $\pm$ 11.80	90.85 $\pm$ 12.09	90.40 $\pm$ 11.82	90.74 $\pm$ 11.99
HDL (mg/dL)	50.13 $\pm$ 4.78	49.72 $\pm$ 4.60	49.95 $\pm$ 4.69	50.27 $\pm$ 4.80	50.06 $\pm$ 4.63	50.20 $\pm$ 4.77

**Table 2:** Group Comparisons of Cardiovascular and Biochemical Parameters by Hypertension and Smoking Status

Parameter	Grand Mean	Hypertension SEM	Smoking SEM	t (HTN)	t (S)	P-value (HTN)	P-value (S)
Alcohol Intake (g)	96.83	42.95	24.01	-0.88	2.92	0.381	<b>0.004</b>
Triglycerides (mg/dL)	126.67	9.55	5.34	<b>11.93</b>	<b>23.88</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
LDL (mg/dL)	72.25	23.93	13.38	<b>2.65</b>	<b>5.53</b>	<b>0.008</b>	<b>&lt;0.001</b>
HDL (mg/dL)	59.50	4.03	2.25	<b>17.24</b>	<b>26.58</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
CIMT Left (mm)	5.40	6.31	3.53	0.64	1.75	0.526	0.085
Left Carotid Diameter (cm)	0.33	31.40	17.55	1.59	<b>2.30</b>	0.113	<b>0.022</b>
CIMT Right (mm)	6.97	15.56	8.70	0.26	0.98	0.798	0.330
Right Carotid Diameter (cm)	14.19	25.85	14.45	1.48	0.87	0.140	0.387
Left Atrial Diameter (cm)	3.75	0.21	0.12	<b>19.56</b>	<b>31.43</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
LV Internal Diameter (cm)	4.86	0.28	0.16	<b>18.82</b>	<b>33.38</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
LV Posterior Wall (cm)	1.11	0.28	0.16	<b>4.66</b>	<b>6.41</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>
Ejection Fraction (%)	62.62	9.16	5.12	<b>7.45</b>	<b>14.41</b>	<b>&lt;0.001</b>	<b>&lt;0.001</b>



**Figure 1:** Smoking Status and Cardiovascular parameters

**Table 4:** Significant Correlations Between Clinical Variables and Cardiovascular Parameters

Independent Variable	Dependent Variable	Correlation (r)	P-Value
Alcohol (g)	—	—	—
Age (Years)	Total Cholesterol (mg/dL)	0.53	0.006
	Triglycerides (mg/dL)	0.51	0.010
	Diastolic BP (mmHg)	0.47	<0.001
	Right Diameter (cm)	0.589	<0.001
	Left Diameter (cm)	0.524	0.001
P-Axis (°)	Diastolic BP (mmHg)	0.37	0.028
QRS-Axis (°)	QRS Interval (ms)	-0.62	<0.001
	T-Interval (ms)	-0.55	0.001
T-Axis (°)	QRS Interval (ms)	0.41	0.014
PR-Interval (ms)	QRS Interval (ms)	0.52	0.001
BMI (kg/m <sup>2</sup> )	Diastolic BP (mmHg)	0.323	0.021
	Systolic BP (mmHg)	0.323	0.021
Right Carotid Diameter (cm)	CIMT Right (mm)	0.409	0.022
	Left Carotid Diameter (cm)	0.543	0.002
Left Carotid Diameter (cm)	BMI	-0.355	0.043
SBP (mmHg)	Age (Years)	0.343	0.011
LAD (cm)	SBP (mmHg)	0.337	0.021
	Age (Years)	0.295	0.037

## Discussion

Rumuekini Community is a suburban community in Rivers State, very few people responded because of the social stigmata associated with alcohol and the fear of knowing the complication, women did not avail themselves of the opportunity. Women are known to have better health seeking behaviours in Nigeria from local studies, but the social stigmata associated with alcohol ingestion affected the turnout the married population accounted for 24.07%.

From table 1 hypertension, notable differences emerge between hypertensive, normotensive, and prehypertensive individuals. For example, hypertensive individuals show higher values for parameters such as left ventricular internal diameter in diastole (LVIDD) and systolic function markers like ejection fraction (EF), reflecting structural and functional changes associated with high blood pressure. Prehypertension demonstrates intermediate values, suggesting progressive changes compared to normotensives. Normotensive individuals generally exhibit lower values, highlighting healthier cardiovascular profiles. These distinctions underscore the escalating impact of blood pressure on cardiovascular health across different hypertension stages.

Similarly, smokers displayed distinct patterns compared to non-smokers in various parameters. Non-smokers often show better outcomes for cardiovascular markers like cholesterol levels, intima-media thickness, and diameters of key cardiac structures, indicating less exposure to the harmful effects of smoking. Meanwhile, smokers tend to exhibit higher T-axis and PR-interval values, potentially reflecting electrophysiological changes induced by smoking. These findings emphasize the dual influence of hypertension and smoking on cardiovascular health, showcasing specific trends that could guide interventions to mitigate risk in these populations. Smoking has significant detrimental effects on the cardiovascular system, contributing to conditions such as atherosclerosis, elevated blood pressure, reduced oxygen supply, increased blood clot formation, and damage to blood vessel walls. Nicotine raises blood pressure and heart rate, while carbon monoxide in smoke reduces the oxygen-carrying capacity of blood, both of which strain the heart. Smoking also promotes clotting, reduces "good" HDL cholesterol, and damages arterial linings, increasing the risk of coronary artery disease and peripheral artery disease. These effects align closely with the data from the study above, where smokers exhibited higher carotid intima-media thickness (CIMT), larger left atrial diameter, and a depressed left ventricular ejection fraction (EF). These findings reflect structural and functional cardiovascular damage associated with smoking. In contrast, the lower alcohol intake among hypertensives and non-smokers might indicate an attempt at healthier lifestyle changes. The elevated triglyceride and LDL cholesterol levels in hypertensives are consistent with the well-documented links between hypertension, lipid abnormalities, and cardiovascular risks. This study

underscores the compounded cardiovascular impact of smoking, alcohol consumption, and hypertension, highlighting the importance of lifestyle modifications.

In this cross-sectional analysis, we assessed the relationships between selected clinical variables and cardiovascular parameters among the study population. There was no statistically significant correlation between alcohol consumption (measured in grams) and any of the cardiovascular indices examined, suggesting that alcohol intake did not independently influence the measured cardiovascular outcomes in this cohort.

In contrast, age was consistently and positively correlated with multiple cardiovascular parameters. Specifically, increasing age was significantly associated with higher total cholesterol levels ( $r = 0.53$ ,  $P = 0.006$ ), elevated triglyceride levels ( $r = 0.51$ ,  $P = 0.010$ ), and increased diastolic blood pressure (DBP) ( $r = 0.47$ ,  $P < 0.001$ ). Systolic blood pressure (SBP) also rose with age ( $r = 0.343$ ,  $P = 0.011$ ). Moreover, age was significantly correlated with anatomical cardiac measurements, including right ventricular diameter ( $r = 0.589$ ,  $P < 0.001$ ), left ventricular diameter ( $r = 0.524$ ,  $P = 0.001$ ), and left atrial diameter (LAD) ( $r = 0.295$ ,  $P = 0.037$ ), indicating progressive chamber enlargement with advancing age. LAD was additionally associated with elevated SBP ( $r = 0.337$ ,  $P = 0.021$ ), in addition the older participants had larger left atrial size ( $r = 0.30$ ,  $P = 0.04$ )

Concerning electrical activity, the P-axis demonstrated a modest positive correlation with DBP ( $r = 0.37$ ,  $P = 0.028$ ), while a more negative QRS-axis was inversely correlated with QRS duration ( $r = -0.62$ ,  $P < 0.001$ ) and T-interval ( $r = -0.55$ ,  $P = 0.001$ ), indicating conduction slowing associated with axial shift. A positive correlation was observed between T-axis and QRS duration ( $r = 0.41$ ,  $P = 0.014$ ). Likewise, prolongation of the PR interval correlated significantly with longer QRS intervals ( $r = 0.52$ ,  $P = 0.001$ ).

Higher BMI was significantly associated with increased diastolic and systolic blood pressure ( $r = 0.323$ ,  $P = 0.021$  for both), suggesting a hypertensive phenotype in individuals with greater body mass. An inverse association was observed between BMI and left carotid diameter ( $r = -0.355$ ,  $P = 0.043$ ). Additionally, larger right carotid diameters were associated with greater carotid intima-media thickness (CIMT) ( $r = 0.409$ ,  $P = 0.022$ ), and there was a strong bilateral correlation between right and left carotid diameters ( $r = 0.543$ ,  $P = 0.002$ ), indicating symmetrical arterial remodeling.

Taken together, these findings suggest that age is a central determinant of structural and functional cardiovascular changes, influencing lipid profiles, hemodynamic parameters, cardiac chamber size, and electrical conduction. The role of BMI as a modifiable risk factor is also supported by its association with blood pressure and carotid remodeling. These results highlight the need for age-specific cardiovascular surveillance and early lifestyle interventions.

The interaction between various cardiac structures was also notable. A strong positive correlation was observed between ejection fraction (EF) and fractional shortening (FS), highlighting their interdependence as measures of cardiac function. Additionally, right and left diameters showed a positive association, suggesting concurrent structural changes in cardiac chambers. These findings collectively highlight how cardiovascular parameters interrelate, providing insights into potential mechanisms behind cardiovascular remodeling and functional changes.

In conclusion, alcohol consumption exhibits a dual and dose-dependent influence on cardiovascular health, with moderate intake offering potential cardioprotective benefits, such as improved lipid profiles, while chronic and excessive consumption leads to hypertension, arrhythmias, atherosclerosis, and structural changes like increased arterial stiffness. These adverse effects are amplified in the presence of hypertension and smoking, which together accelerate cardiovascular damage through mechanisms such as oxidative stress, vascular inflammation, and electrophysiological disruptions. Smokers exhibit higher T-axis and PR-interval values, while hypertensive individuals display structural cardiac remodeling and progressive deterioration, emphasizing the compounded risks.

Interestingly, the study highlights the added nuance of marital status in the interplay of alcohol consumption and cardiovascular health. Married individuals, while consuming more alcohol on average, appear to be partly protected from adverse outcomes through other mitigating factors. These include enhanced social support systems, healthier lifestyle choices, greater access to healthcare, and the psychosocial benefits of reduced stress in stable relationships. This dynamic underscores the complex and multifactorial nature of alcohol's impact, where demographic and lifestyle factors significantly influence outcomes.

Overall, this investigation reinforces the importance of targeted public health strategies aimed at modifiable risks. Interventions should encourage moderation in alcohol consumption, address the risks associated with smoking and hypertension, and consider the protective social and lifestyle factors associated with marriage. Future research should explore these interactions further to inform policies and interventions that promote cardiovascular health across diverse populations.

## Limitations of Study

The relatively small sample size limits the statistical power of the analysis and the generalizability of the findings. Secondly alcohol intake was assessed through self-reported questionnaires, which may introduce recall or reporting bias. Finally, the study population consisted predominantly of male participants due to sociocultural factors that limited female participation. Future studies involving larger, more diverse populations and including both drinkers and non-

drinkers will be important for clarifying the cardiovascular implications of alcohol consumption in African communities.

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