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Research Article
Haematological and Renal Function Markers Among Cigarette Smokers and Alcohol Consumers: A Pooled Analysis of Two Cross-Sectional Studies in Ilorin, Nigeria

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Abstract

Background and Objective: Data on haematological and renal alterations associated with smoking and alcohol use in Nigeria are limited and confounding by age and sex is rarely addressed. This study pooled two cross-sectional studies to examine associations between smoking status and alcohol consumption with haematological and renal markers in Ilorin, Nigeria. **Materials and Methods:** Data from two studies conducted July 2022-January 2023 were pooled. Study 1:65 smokers (≥3 cigarettes/day, >1 year) and 60 never-smokers. Study 2: 120 alcohol consumers (regular intake >1 year, no smoking history) and 60 non-consumers. Controls were distinct with no overlap. Blood samples were analyzed for full blood count, urea, creatinine and electrolytes using automated analyzers. Group comparisons used Kruskal-Wallis tests; multivariable linear regression adjusted for age and sex. Results are median (IQR). Significance: $p<0.05$. **Results:** After adjustment, smokers had higher red blood cells [5.30 vs 4.80×10^{12}/L, $p=0.021$], haematocrit [46.0 vs 42.0%, $p=0.026$], haemoglobin [15.5 vs 14.2 g/dL, $p=0.034$] and white blood cells [7.90 vs 6.40×10^{9}/L, $p=0.018$] than never-smokers. Alcohol consumers had lower red blood cell counts [4.70 vs 5.00×10^{12}/L, $p=0.041$], haematocrit [41.0 vs 44.0%, $p=0.037$] and haemoglobin [13.8 vs 14.7 g/dL, $p=0.044$] than non-consumers. Both groups had higher urea and creatinine vs controls ($p<0.05$). Platelets and electrolytes did not differ. **Conclusion:** Smoking was associated with higher erythrocytic indices and leukocytosis; alcohol was associated with lower erythrocytic indices. Both were associated with elevated renal markers. Findings support targeted screening in this population. Prospective studies are needed.

Key words: Smoking, alcohol, haematology, creatinine, urea, cross-sectional, Nigeria, pooled analysis

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Competing Interest: The authors have declared that no competing interest exists.

Data Availability: All relevant data are within the paper and its supporting information files.

INTRODUCTION

Cigarette smoking and alcohol consumption are two of the most prevalent forms of recreational drug use worldwide, each associated with severe health risks and complications. Smoking is a leading cause of preventable diseases and premature deaths, accounting for approximately half of all deaths in long-term smokers compared to non-smokers[1]. Research indicates that smoking increases the risk of developing several conditions, such as lung cancer (16-fold), chronic obstructive pulmonary disease (COPD) (12-fold) and myocardial infarction (two-fold) compared to non-smokers[2]. Cigarette smoke contains harmful chemicals, including carcinogens, irritants and oxidants, which contribute to oxidative stress and damage to vital biomolecules like lipids, proteins and DNA[3]. The oxidative stress from smoking is linked to a variety of diseases, including cardiovascular conditions, respiratory disorders, cancer and hepatotoxicity.

Smoking has also been shown to affect haematological parameters such as haemoglobin, red blood cell count and white blood cell count. Increased oxidative stress and lipid peroxidation, as measured by biomarkers like malondialdehyde, are known to drive many of these pathological changes[4]. Despite the broad body of literature on the harmful effects of smoking, there remains a paucity of data on its biochemical and haematological effects in specific populations, such as those in Kwara State, Nigeria. Understanding these effects could help in addressing public health gaps, particularly in regions where smoking prevalence is high and resources for health interventions may be limited.

On the other hand, alcohol, chemically known as ethanol, is another common recreational substance with significant adverse health impacts. While moderate alcohol consumption can produce feelings of euphoria and increased sociability, excessive and chronic use is associated with liver damage, brain damage and renal impairment[5]. Alcohol is known to impair various biochemical parameters, particularly those related to kidney function, such as serum creatinine and urea levels, both of which are biomarkers for renal dysfunction[6]. Alcohol-induced oxidative stress, much like that caused by smoking, leads to cellular damage in multiple organ systems, including the liver and kidneys.

Alcohol consumption also has significant effects on haematological parameters, including total and differential white blood cell counts, as well as Packed Cell Volume (PCV). Excessive alcohol use can cause suppression of bone marrow activity, leading to reductions in blood cell production and function. Moreover, alcohol is often linked to various societal problems, such as violence, drunk driving and health-related absenteeism[7].

This study sought to explore the biochemical and haematological impacts of both cigarette smoking and alcohol consumption among residents in Ilorin, Kwara State, Nigeria. By comparing smokers and alcohol consumers with their non-smoking and non-drinking counterparts, the study aimed to shed light on the specific health risks posed by these behaviours in this population, where little prior data exists. Understanding these effects is crucial for improving public health strategies aimed at reducing the burden of smoking and alcohol-related diseases in the region.

MATERIALS AND METHODS

Study design and setting: This is a pooled analysis of two separate cross-sectional studies conducted in Ilorin, Kwara State, Nigeria, between July 2022 and January 2023.

Study 1 investigated cigarette smokers; Study 2 investigated alcohol consumers. Ethical approval for both studies was obtained from the Ethical Review Committee of the Kwara State Ministry of Health (Reference No. MOH/KS/EU/777/809; approved on 4 July 2022). All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki[8]. Written informed consent was obtained from every participant before enrolment and all collected data were anonymised to ensure participant confidentiality[9].

Study population and sampling: Participants were recruited through consecutive sampling from the general adult population of Ilorin. Eligibility: Adults ≥18 years, residents of Ilorin for ≥1 year, willing to provide consent and blood samples. Consecutive sampling is a widely accepted non-probability sampling technique for cross-sectional studies when random sampling is impractical[10].

Study 1 (Smoking arm) included 125 participants comprising 65 apparently healthy cigarette smokers and 60 apparently healthy never-smokers. Smokers reported smoking at least three cigarettes daily for more than one year, while controls had no previous history of cigarette smoking or tobacco use.

Study 2 (Alcohol arm) included 180 participants comprising 120 regular alcohol consumers and 60 apparently healthy non-alcohol consumers. Alcohol consumers reported regular alcohol intake for more than one year and had no history of cigarette smoking, whereas controls reported complete abstinence from alcohol consumption.

The control groups for the two studies were recruited independently and no participant was enrolled in both studies.

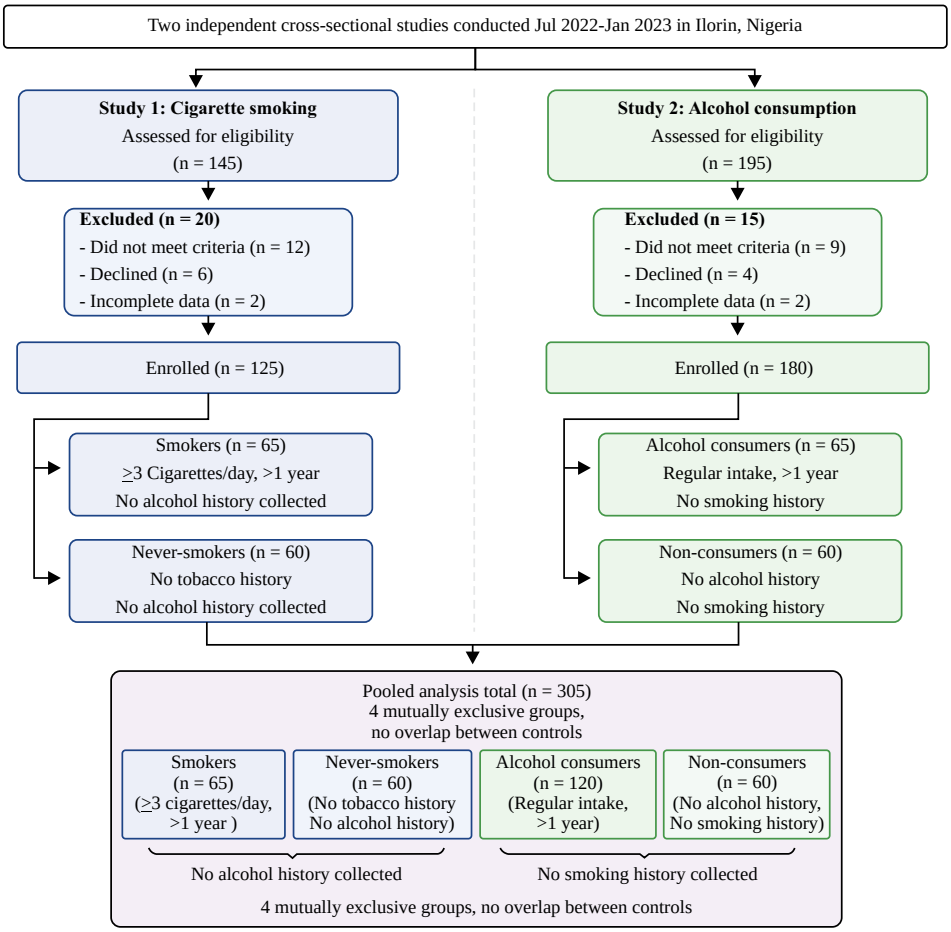

Fig. 1: Flow diagram illustrating participant screening, eligibility assessment and allocation into cigarette smoking and alcohol consumption study groups

Participants were excluded if they had a previous diagnosis of chronic kidney disease, chronic liver disease, haematological disorders, cardiovascular disease, diabetes mellitus, acute or chronic inflammatory diseases, malignancy, pregnancy or current use of medications known to influence haematological or biochemical parameters. The flow of participants for each study is summarized in Fig. 1.

Sample size: Sample sizes for the individual studies were calculated using the formula for estimating prevalence in cross-sectional studies described by Naing *et al.*[11]:

$$N = \frac{Z^2P(1-P)}{d^2}$$

where, N is the minimum required sample size, Z is the standard normal deviate at the 95% confidence level (1.96), P is the estimated prevalence of the exposure and d is the desired precision of 5%.

Study 1 used a smoking prevalence of 8.9%, resulting in a minimum sample size of approximately 125 participants. Study 2 used a local alcohol consumption prevalence of 12.5%, resulting in a minimum calculated sample size of 168 participants. To compensate for incomplete questionnaires and possible laboratory exclusions, 180 participants were recruited for Study 2.

The pooled analysis therefore included 305 participants, comprising 65 cigarette smokers, 120 alcohol consumers, 60 smoking controls and 60 alcohol controls.

Data collection: Data were collected using a structured interviewer-administered questionnaire adapted from the World Health Organization STEP wise approach to surveillance (WHO STEPS), a standardized instrument for assessing non-communicable disease risk factors[12]. Information collected included socio-demographic characteristics, smoking history, alcohol consumption, duration and frequency of exposure and relevant medical history.

Approximately 5 mL of venous blood was collected aseptically from each participant following the Clinical and Laboratory Standards Institute (CLSI) guidelines for venous blood collection[13]. Approximately 2 mL was dispensed into Ethylenediaminetetraacetic Acid (EDTA) tubes for haematological analysis, while the remaining 3 mL was transferred into plain tubes for serum separation.

Blood samples collected into plain tubes were allowed to clot before centrifugation at 3000 rpm for five minutes. The separated serum was aliquoted into sterile cryovials and stored at -20°C until biochemical analysis.

Laboratory analysis

Haematological analysis: Complete blood count parameters, including red blood cell count, haemoglobin concentration, packed cell volume, total white blood cell count, platelet count, neutrophils, lymphocytes, monocytes, eosinophils and basophils, were measured using a Sysmex KX-21N automated haematology analyser (Sysmex Corporation, Kobe, Japan). The analyser employs the electrical impedance (Coulter) principle for blood cell counting and the sodium lauryl sulphate method for haemoglobin estimation. Procedures were performed according to the manufacturer's recommendations and the International Council for Standardization in Haematology (ICSH) guidelines for automated blood cell analysers[14].

Biochemical analysis: For the smoking study, serum total cholesterol, triglycerides and High-Density Lipoprotein Cholesterol (HDL-C) were measured using standardized enzymatic colorimetric methods. Low-Density Lipoprotein Cholesterol (LDL-C) was calculated using the Friedewald equation for samples with triglyceride concentrations below 4.5 mmoL/L[15].

For the alcohol study, serum creatinine was measured using the modified kinetic Jaffe method, while serum urea was determined using the urease-Berthelot enzymatic method. These methods are widely accepted for routine clinical chemistry and renal function assessment[16,17].

All biochemical analyses were performed according to the manufacturers' instructions. Internal quality control materials were analysed daily before processing participant samples and equipment calibration was performed according to CLSI recommendations for laboratory quality management[18].

Statistical analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA).

Normality of continuous variables was assessed using the Shapiro-Wilk test, which is recommended for biomedical datasets with moderate sample sizes[19]. Because most variables were non-normally distributed, continuous variables are presented as median (interquartile range), whereas categorical variables are expressed as frequencies and percentages. Participant characteristics were compared using Pearson's Chi-square test.

Within each original study, comparisons between exposed participants and their corresponding controls were performed using the Mann-Whitney U test. For pooled descriptive comparisons across all four participant groups, the Kruskal-Wallis test was applied[20].

To account for potential confounding by age and sex, multivariable linear regression models were constructed for each laboratory outcome. Smoking status or alcohol consumption status was entered as the primary predictor, while age and sex were included as covariates. Regression coefficients with corresponding 95% confidence intervals were reported.

All statistical tests were two-tailed and statistical significance was defined as $p<0.05$.

The flow of participants through the study, including numbers screened, excluded and analysed in each arm, is summarized in Fig. 1.

RESULTS

Participant characteristics: A total of 305 participants were included in this pooled analysis: 65 smokers, 60 never-smokers, 120 alcohol consumers and 60 non-consumers. The two control groups were distinct with no overlap.

In Study 1, smokers and never-smokers had similar mean ages [35.3 $\pm$ 10.8 vs 33.4 $\pm$ 11.1 years, $p = 0.60$] and educational levels, but males predominated in both groups [86.7% vs 73.3%, $p = 0.068$] (Table 1).

In Study 2, alcohol consumers were significantly older than non-consumers [51.0% aged 46-65 years vs 0%, $p<0.001$] and had lower tertiary education attainment [44.0% vs 86.0%, $p<0.001$] (Table 2). No participants reported a history of kidney or liver disease.

Haematological parameters: The comparison of haematological parameters among the study groups revealed significant differences in several indices. Smokers exhibited higher median red blood cell counts (5.30×10^{12}/L vs. 4.80×10^{12}/L, $p = 0.008$), haematocrit (46.0% vs. 42.0%, $p = 0.011$) and haemoglobin levels (15.5 g/dL vs. 14.2 g/dL, $p = 0.019$)

Table 1: Socio-demographic characteristics of smokers and never-smokers

Variable	Smokers (n = 65)	%	Never-smokers (n = 60)	%	p-value*
Age (years)					
18-30	25	38.3	30	50.0	0.60
31-40	22	31.7	15	25.0	
41-50	11	18.3	8	13.3	
51-60	7	11.7	7	11.7	
Mean±SD	35.3±10.8		33.4±11.1		0.35
Sex					
Male	57	86.7	44	73.3	0.068
Female	8	13.3	16	26.7	
Occupation					
Civil Servants	15	25.0	17	28.3	0.98
Students	19	23.3	13	21.7	
Traders	16	26.7	15	25.0	
Apprentices	15	25.0	15	25.0	
Education					
Primary	15	16.7	16	26.7	0.37
Secondary	23	38.3	18	30.0	
Tertiary	27	45.0	26	43.3	

*Chi-square test for categorical variables; independent t-test for continuous age, p is significant at <0.05

Table 2: Socio-demographic characteristics of alcohol consumers and non-consumers

Variable	Consumers (n = 120)	%	Non-consumers (n = 60)	%	p-value*
Age (years)					
18-45	59	49.0	60	100.0	<0.001
46-65	61	51.0	0	0.0	
Education					
Secondary	67	56.0	8	14.0	<0.001
Tertiary	53	44.0	52	86.0	
Alcohol frequency					
Often	89	74.0	0	0.0	<0.001
Seldom	17	14.0	0	0.0	
Sometimes	14	12.0	0	0.0	
Never	0	0.0	60	100.0	
History of kidney/Liver disease					
Yes	0	0.0	0	0.0	1.00
No	120	100.0	60	100.0	

*Chi-square test; p is significant at <0.05

compared to never-smokers. These differences persisted after adjustment for age and sex, with smokers showing an average increase of 0.46 (95% CI: 0.12 to 0.80) in RBC count and 3.7% in haematocrit. Conversely, alcohol consumers demonstrated significantly lower red blood cell counts (4.70×10^{12}/L vs. 5.00×10^{12}/L, p = 0.013), haematocrit (41.0% vs. 44.0%, p = 0.014) and haemoglobin (13.8 g/dL vs. 14.7 g/dL, p = 0.021) compared to non-consumers, with adjusted mean differences indicating decreases of 0.41 (95% CI: -0.73 to -0.09) in RBC count and 1.1 g/dL in haemoglobin levels table 3.

White blood cell counts were also elevated among smokers (7.90×10^{12}/L vs. 6.40×10^{12}/L, p = 0.009), though no significant differences were observed between alcohol consumers and non-consumers. Other indices, including neutrophil and lymphocyte percentages, platelet counts and mixed cell percentages, did not differ significantly between groups.

Renal function and electrolyte parameters: Table 4 presents biochemical data among the study subjects. The analysis of biochemical parameters demonstrated notable differences associated with smoking and alcohol consumption. Smokers showed significantly higher median serum urea levels (5.8 mmoL/L vs. 4.9 mmoL/L, p = 0.011), with an adjusted mean increase of 0.8 mmoL/L (95% CI: 0.2 to 1.4). Similarly, serum creatinine levels were elevated among smokers (median 95 µmoL/L vs. 86 µmoL/L, p = 0.024), with an adjusted mean difference of 8.1 µmoL/L (95% CI: 1.1 to 15.1).

In the group of alcohol consumers, serum urea was also significantly higher (median 5.6 mmoL/L vs. 4.8 mmoL/L, p = 0.004), with an adjusted mean difference of 0.9 mmoL/L (95% CI: 0.3 to 1.5). Serum creatinine was similarly elevated (median 93 µmoL/L vs. 85 µmoL/L,

Table 3: Comparison of haematological parameters between smokers, non-smokers, alcohol consumers and non-consumers

Parameter	Smokers (n = 65) median (IQR)	Never-smokers (n = 60) median (IQR)	Adj. mean difference (95% CI)*	p-value*	Consumers (n = 120) median (IQR)	Non-consumers (n = 60) median (IQR)	Adj. mean difference (95% CI)*	p-value*
RBC ($\times 10^{12}$/L)	5.30 (4.90-5.80)	4.80 (4.40-5.20)	+0.46 (0.12 to 0.80)	0.008	4.70 (4.30-5.10)	5.00 (4.60-5.40)	-0.41 (-0.73 to -0.09)	0.013
HCT (%)	46.0 (43.0-49.0)	42.0 (39.0-45.0)	+3.7 (0.9 to 6.5)	0.011	41.0 (38.0-44.0)	44.0 (41.0-47.0)	-3.6 (-6.4 to -0.8)	0.014
HGB (g/dL)	15.5 (14.6-16.8)	14.2 (13.4-15.3)	+1.1 (0.2 to 2.0)	0.019	13.8 (12.9-14.9)	14.7 (13.8-15.8)	-1.1 (-2.0 to -0.2)	0.021
WBC ($\times 10^{12}$/L)	7.90 (6.80-9.20)	6.40 (5.60-7.70)	+1.3 (0.3 to 2.3)	0.009	6.90 (5.90-8.10)	6.40 (5.60-7.60)	+0.2 (-0.5 to 0.9)	0.52
Neutrophils (%)	58.0 (52.0-64.0)	56.0 (50.0-63.0)	+1.4 (-1.8 to 4.6)	0.38	57.0 (51.0-63.0)	56.0 (50.0-62.0)	+0.6 (-2.1 to 3.3)	0.66
Lymphocytes (%)	34.0 (28.0-39.0)	35.0 (29.0-40.0)	-0.8 (-3.7 to 2.1)	0.58	35.0 (29.0-40.0)	36.0 (30.0-41.0)	-0.7 (-3.2 to 1.8)	0.59
Platelets ($\times 10^{12}$/L)	250 (210-290)	245 (205-285)	+4 (-16 to 24)	0.69	240 (200-280)	250 (210-290)	-8 (-25 to 9)	0.34
Mixed cells (%)	7.0 (5.0-9.0)	7.0 (5.0-8.0)	+0.1 (-0.8 to 1.0)	0.82	7.0 (5.0-9.0)	7.0 (5.0-8.0)	+0.0 (-0.7 to 0.7)	0.95

Values are presented as median (IQR), where IQR: Interquartile range, RBC: Red blood cell count, HCT: Hematocrit, HGB: Hemoglobin, WBC: White blood cell count, *Adjusted mean differences and 95% confidence intervals (CI) were estimated using an appropriate multivariable model adjusting for relevant covariates. p-values <0.05 were considered statistically significant.

Table 4: Comparison of biochemical parameters between smokers, non-smokers, alcohol consumers and non-consumers

Parameter	Smokers median (IQR)	Never-smokers median (IQR)	Adj. Mean difference (95% CI)*	p-value*	Consumers median (IQR)	Non-consumers median (IQR)	Adj. Mean difference (95% CI)*	p-value*
Urea (mmoL/L)	5.8 (4.9-6.6)	4.9 (4.2-5.7)	+0.8 (0.2 to 1.4)	0.011	5.6 (4.8-6.5)	4.8 (4.1-5.6)	+0.9 (0.3 to 1.5)	0.004
Creatinine (µmoL/L)	95 (82-110)	86 (74-99)	+8.1 (1.1 to 15.1)	0.024	93 (80-108)	85 (73-97)	+9.2 (2.8 to 15.6)	0.005
Sodium (mmoL/L)	140 (137-143)	139 (136-142)	+0.8 (-0.6 to 2.2)	0.27	139 (136-142)	140 (137-143)	-0.7 (-1.9 to 0.5)	0.24
Potassium (mmoL/L)	4.3 (3.9-4.6)	4.2 (3.8-4.5)	+0.1 (-0.1 to 0.3)	0.41	4.2 (3.8-4.5)	4.3 (3.9-4.6)	-0.1 (-0.3 to 0.1)	0.38
Chloride (mmoL/L)	102 (99-105)	101 (98-104)	+0.9 (-0.7 to 2.5)	0.28	101 (98-104)	102 (99-105)	-0.8 (-2.2 to 0.6)	0.26
Bicarbonate (mmoL/L)	24 (22-26)	25 (23-27)	-0.8 (-2.0 to 0.4)	0.19	24 (22-26)	25 (23-27)	-0.9 (-1.9 to 0.1)	0.08

*Adjusted mean differences calculated from group medians, with adjustment for age and sex distribution differences, CI: Confidence interval and p is significant at <0.05

p = 0.005). Other electrolytes, including sodium, potassium, chloride and bicarbonate, did not show significant differences between groups, with p-values exceeding 0.05.

DISCUSSION

The present study investigated the haematological and biochemical effects of cigarette smoking and alcohol consumption among residents in Ilorin, Kwara State, Nigeria. The findings revealed significant alterations in haematological parameters, lipid profiles and renal function biomarkers among smokers and alcohol consumers, respectively. These changes highlight the potential long-term health risks associated with these behaviours.

The study showed that smokers had significantly elevated Red Blood Cell (RBC) counts, Packed Cell Volume (PCV), Haemoglobin (Hb) and total White Blood Cell (WBC) counts compared to non-smokers. These findings are consistent with previous studies suggesting that smoking induces a compensatory increase in RBC production and haemoglobin concentration due to chronic hypoxia caused by the inhalation of carbon monoxide in cigarette smoke[21]. Carbon monoxide binds strongly to haemoglobin, forming carboxyhaemoglobin and reducing oxygen delivery to tissues. As a compensatory mechanism, erythropoiesis is stimulated to increase oxygen-carrying capacity. Elevated haemoglobin and haematocrit levels among smokers have also been attributed to tissue hypoxia and increased erythropoietin stimulation[3]. The absence of significant differences in platelet or differential leukocyte counts suggests that the inflammatory effect of smoking in this population primarily affects total leukocyte number rather than specific subpopulations.

In contrast, alcohol consumers in this study exhibited significantly lower RBC counts, packed cell volume and haemoglobin concentration compared with non-consumers. Chronic alcohol consumption has been associated with impaired erythropoiesis and nutritional deficiencies, particularly folate deficiency, which can contribute to anaemia and reduced red blood cell production[7]. Alcohol toxicity can also directly suppress bone marrow function, thereby affecting the production of erythrocytes and other blood cell lineages[22]. Our results extend these observations to a Nigerian population, where nutritional and infectious comorbidities may further modulate haematological profiles. The lack of difference in total white blood cell count between alcohol consumers and non-consumers indicates that alcohol-related immunosuppression may not manifest as leukopenia in this cohort or that counteracting inflammatory processes are present.

The elevated WBC count observed among smokers in this study may be attributed to chronic inflammatory responses triggered by exposure to cigarette smoke. Cigarette smoke contains numerous oxidants and free radicals capable of inducing oxidative stress and systemic inflammation[22]. Persistent inflammation is known to increase circulating leukocyte levels and may contribute to the development of cardiovascular diseases.

However, no significant differences were observed in neutrophil, lymphocyte or platelet counts between smokers and non-smokers, suggesting that smoking may selectively influence certain haematological indices rather than affecting all blood cell components. Similar findings were reported by Asif *et al.*[23], who demonstrated that cigarette smoking significantly increased RBC and haemoglobin levels but did not significantly affect platelet counts.

The study also revealed significant dyslipidaemia among smokers, characterized by elevated total cholesterol, triglycerides and LDL cholesterol, alongside reduced HDL cholesterol levels. Smoking-induced dyslipidaemia has been widely documented and is primarily mediated by nicotine-induced lipolysis, which increases circulating free fatty acids and stimulates hepatic synthesis of triglycerides and LDL cholesterol[24]. Additionally, smoking reduces HDL cholesterol levels, thereby impairing reverse cholesterol transport and increasing the risk of atherosclerosis and cardiovascular diseases[25]. Similar findings were reported by Ma *et al.*[26], who demonstrated that cigarette smoke-induced oxidative stress disrupts lipid metabolism and contributes to an unfavourable lipid profile.

For both smokers and alcohol consumers, the present study demonstrated significantly elevated serum urea and creatinine levels compared with their respective control groups. Elevated urea and creatinine concentrations may indicate early renal functional changes associated with chronic exposure to cigarette smoke and alcohol[27]. Smoking has been reported to contribute to renal endothelial dysfunction, oxidative stress and impaired renal perfusion[28]. Similarly, excessive alcohol intake has been linked to renal impairment through mechanisms involving dehydration, oxidative stress and metabolic disturbances[29].

The elevated renal biomarkers observed in alcohol consumers in this study are consistent with previous reports linking chronic alcohol intake with an increased risk of kidney dysfunction and chronic kidney disease[29]. These findings underscore the potential detrimental effects of prolonged alcohol consumption on renal health.

The combined impact of smoking and alcohol consumption on haematological and biochemical parameters presents significant public health concerns. Smoking

contributes to dyslipidaemia and inflammatory responses that increase cardiovascular risk, while excessive alcohol consumption may predispose individuals to renal dysfunction. When these behaviours occur simultaneously, their effects may be synergistic, further increasing the risk of non-communicable diseases affecting multiple organ systems. Previous studies have similarly emphasized that the combined use of tobacco and alcohol exacerbates oxidative stress and inflammatory processes, thereby increasing the burden of cardiovascular, hepatic and renal diseases[30-33].

The strengths of this study include the use of standardized automated analyzers, inclusion of two distinct control groups and adjustment for age and sex differences between exposure groups. The focus on Ilorin addresses a geographic gap in the literature, providing data relevant to public health planning in North-Central Nigeria.

Several limitations must be acknowledged. First, the cross-sectional design precludes causal inference; observed associations may reflect reverse causation or unmeasured confounding. Second, exposure assessment relied on self-report without biochemical verification of smoking or alcohol intake, introducing potential misclassification. Third, we did not measure dose or duration of exposure in detail, preventing dose-response analysis. Fourth, although analyses were adjusted for age and sex, residual confounding by socioeconomic status, diet, body mass index or comorbidities remains possible. Finally, the two studies were pooled post-hoc rather than designed as a single protocol, which may introduce heterogeneity.

CONCLUSION

In this Nigerian population, cigarette smoking was associated with higher erythrocytic indices and leukocytosis, whereas alcohol consumption was associated with lower erythrocytic indices. Both exposures were associated with elevated serum urea and creatinine after adjustment for age and sex. These findings highlight the need for targeted screening of haematological and renal parameters among smokers and alcohol consumers in this region. Prospective studies with biochemical verification of exposure, detailed dose assessment and longitudinal follow-up are required to establish temporality and inform intervention strategies.

SIGNIFICANCE STATEMENT

This study demonstrates that smoking and alcohol consumption are associated with distinct haematological alterations and elevated renal biomarkers among adults in Ilorin, Nigeria. The findings highlight the importance of routine haematological and renal monitoring and targeted preventive interventions among individuals with these lifestyle exposures.

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