

Alterations in haematological and coagulation parameters among smokers: A case–control study in Enugu Metropolis, Nigeria

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Research Article

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Abstract

Smoking is a major public health concern associated with systemic inflammation, cardiovascular complications, and hematological alterations. This study assessed differences in haematological and coagulation parameters between smokers and non-smokers in Enugu Metropolis, Nigeria. A case-control design was employed, involving 44 smokers and 44 non-smokers aged 18–50 years. Full blood count indices and coagulation parameters were analyzed using automated analyzers and standard laboratory methods. Smokers demonstrated significantly higher median white blood cell counts ($6.0 \times 10^9/\text{L}$ vs. $4.0 \times 10^9/\text{L}$, $p < 0.001$), with elevated lymphocyte ($2.9 \times 10^9/\text{L}$ vs. $2.1 \times 10^9/\text{L}$, $p < 0.001$) and MID cell counts ($1.0 \times 10^9/\text{L}$ vs. $0.3 \times 10^9/\text{L}$, $p < 0.001$). Among red cell indices, smokers had higher median haemoglobin levels (13.4 g/dL vs. 13.2 g/dL , $p = 0.02$), mean corpuscular haemoglobin (28.0 pg vs. 27.2 pg , $p = 0.01$), and mean corpuscular haemoglobin concentration (32.4 g/L vs. 31.5 g/L , $p = 0.03$). Platelet counts ($198 \times 10^9/\text{L}$ vs. $178 \times 10^9/\text{L}$, $p = 0.004$) and plateletcrit (1.8% vs. 1.6% , $p = 0.03$) were also significantly higher among smokers. Coagulation analysis revealed prolonged prothrombin time (17.6 sec vs. 14.8 sec , $p = 0.001$), thrombin time (21.4 sec vs. 19.0 sec , $p = 0.02$), and international normalized ratio (1.5 vs. 1.1 , $p = 0.001$) in smokers, while activated partial thromboplastin time and fibrinogen levels showed no significant differences ($p > 0.05$). These findings indicate that smoking is associated with significant alterations in haematological and coagulation parameters, suggesting a disruption in haemostatic balance. These changes may have implications for clinical risk stratification and monitoring.

1. Background

Smoking remains a major global public health challenge, contributing substantially to morbidity and mortality across multiple organ systems, including the cardiovascular, reticuloendothelial, and respiratory systems (Zhang et al., 2019; Osman et al., 2020). According to the World Health Organization, approximately 2.4 billion people worldwide use tobacco in various forms, with smoking projected to contribute to over 8 million deaths annually in the coming years (WHO, 2024). Cigarette smoke contains numerous toxic substances, including oxidants and free radicals, which induce oxidative stress and are implicated in the development of cancer, cardiovascular diseases, and chronic respiratory conditions (Hikisz & Acenik, 2023).

Beyond these established effects, smoking has also been associated with alterations in haematological and coagulation parameters. Studies have reported increased red blood cell indices and haemoglobin levels among smokers, possibly reflecting physiological adaptation to chronic carbon monoxide exposure (Alqahtany, 2020). In addition, smoking-related oxidative stress has been linked to elevated white blood cell counts, which may indicate systemic biological responses to exposure (Strzelak, 2018). Haemostatic function, commonly assessed using parameters such as prothrombin time (PT), activated partial thromboplastin time (APTT), and platelet indices, plays a critical role in maintaining vascular integrity. Evidence suggests that smoking may influence these parameters through mechanisms

involving platelet activation and endothelial dysfunction; however, findings remain inconsistent across studies (Sandhya et al., 2015; Münzel et al., 2020).

Tobacco use continues to pose a significant public health concern in Nigeria, particularly in urban settings such as Enugu Metropolis, where smoking is prevalent among young and middle-aged individuals. While the adverse effects of smoking on respiratory and cardiovascular health are well documented, its impact on haematological and coagulation parameters remains underexplored in this context. Given the presence of reactive oxygen species in cigarette smoke and their potential effects on blood parameters, further investigation is warranted. This study therefore aimed to assess differences in haematological and coagulation parameters between smokers and non-smokers in Enugu Metropolis, Nigeria.

2. Materials and methods

2.1 Study area

This study was conducted in Enugu Metropolis, the capital of Enugu State, Nigeria. Enugu is located in the South-East geopolitical zone between latitudes 06°21'–06°36' N and longitudes 07°26'–07°57' E. As a major urban center with a diverse population and dynamic socioeconomic landscape, Enugu Metropolis provided a suitable setting for this research.

2.2 Study design and population

The study adopted a case–control design to compare haematological and coagulation parameters between smokers and non-smokers. The study population comprised apparently healthy male smokers aged 18–50 years, alongside non-smokers serving as controls. Eligible participants were residents of Enugu Metropolis within the specified age range and free from known chronic illnesses. Smokers were required to have actively smoked for at least one year, while non-smokers had no history of smoking. Individuals with known haematological disorders, those receiving anticoagulant therapy, or those with chronic diseases were excluded from the study. Additional exclusion criteria included substance abuse (other than smoking), recent history of surgery or trauma, and unwillingness to provide informed consent. To enhance recruitment, particularly among smokers who may be difficult to access, a snowball sampling technique was employed, allowing participants to refer others within their social networks.

2.3 Data collection and analysis

Socio-demographic data was collected using a structured questionnaire to obtain demographic and biosocial information, including age, smoking history, duration of smoking, and relevant medical history.

Venous blood samples were collected under aseptic conditions, and each participant's details were appropriately recorded. A total of 5.0 mL of blood was obtained from each participant, of which 2.7 mL was dispensed into a tube containing 0.3 mL of 3.2% trisodium citrate for coagulation analysis, while the remaining 2.3 mL was transferred into an ethylenediaminetetraacetic acid (EDTA) tube for full blood

count analysis. Haematological parameters were analyzed using a Mindray automated haematology analyzer (three-part differential), following the manufacturer's instructions. The EDTA samples were properly mixed, aspirated, and processed, and results were displayed and recorded accordingly. Coagulation parameters were assessed using a Mindray C2000-4 semi-automated coagulometer. For prothrombin time (PT) and international normalized ratio (INR), citrated blood samples were centrifuged at 3000 rpm for 10 minutes to obtain platelet-poor plasma. PT was determined by mixing 100 μ L of plasma with 200 μ L of PT reagent at 37°C, and clotting time was recorded, from which INR was calculated. Activated partial thromboplastin time (APTT) was measured by incubating plasma at 37°C with APTT reagent, followed by the addition of prewarmed calcium chloride, and the clotting time was recorded. Thrombin time (TT) was determined by incubating plasma and adding thrombin reagent, after which the clotting time was measured. Fibrinogen levels were assessed by diluting plasma in imidazole buffer, incubating at 37°C, and measuring clotting time following the addition of thrombin reagent, with concentrations determined using a calibration curve. ABO blood grouping was performed using standard agglutination techniques with anti-A and anti-B sera.

Statistical analysis was performed using IBM SPSS Statistics. Categorical variables were summarized as frequencies and percentages, while continuous variables were expressed as medians and interquartile ranges (25th–75th percentiles). Differences between smokers and non-smokers were assessed using the Mann–Whitney U test, with a p-value < 0.05 considered statistically significant.

3. Results

3.1 Demographic characteristics of respondents

The median age of smokers was 26 years, compared to 23 years among non-smokers. The median body mass index (BMI) was 20.1 kg/m² for smokers and 20.3 kg/m² for non-smokers. Most participants in both groups were childless, with 59.1% of smokers and 56.8% of non-smokers reporting no children. Regarding ABO blood group distribution, blood group O was the most common, accounting for 43.2% of smokers and 45.5% of non-smokers (Table 1).

Marital status was evenly distributed between the groups, with equal numbers of participants being married or single (Fig. 1). The majority of participants in both groups identified as Muslim, with 34 smokers and 33 non-smokers reporting this affiliation (Fig. 2). In terms of ethnicity, Hausa participants predominated, representing 12 smokers and 18 non-smokers (Fig. 3). Occupation was similarly distributed, with traders comprising the largest subgroup among smokers (15 participants) and non-smokers (12 participants) (Fig. 4).

Table 1
Demography of the study population

Characteristics	Smokers (n = 44)	Non-Smokers (n = 44)
Age (in years) Median(IQR)*	26 (20–34)	23 (22–26)
BMI (kg/m ²) Median(IQR)	20.1 (19.2–21.5)	20.3 (19.7–22.4)
Number of children		
0	26 (59.1%)	25 (56.8%)
1	3 (6.8%)	10 (22.7%)
2	5 (11.4%)	5 (11.4%)
3	4 (9.1%)	1 (2.3%)
≥ 4	6 (13.6%)	0 (0.0%)
ABO Blood group		
A	8 (18.2%)	7 (15.9%)
B	14 (31.8%)	17 (38.6%)
O	19 (43.2%)	20 (45.5%)
AB	3 (6.8%)	0 (0.0%)
*Interquartile range		

3.2 Haematological profile of respondents

The haematological profile of the study population is presented in Table 2. With respect to white blood cell (WBC) parameters, smokers demonstrated significantly higher median WBC counts ($6.0 \times 10^9/L$) compared to non-smokers ($4.0 \times 10^9/L$, $p < 0.001$). Similarly, median lymphocyte counts ($2.9 \times 10^9/L$ vs. $2.1 \times 10^9/L$) and MID cell counts ($1.0 \times 10^9/L$ vs. $0.3 \times 10^9/L$) were significantly elevated among smokers ($p < 0.001$). However, no statistically significant difference was observed in granulocyte counts between the two groups ($p > 0.05$). Regarding red blood cell (RBC) parameters, smokers exhibited significantly higher median haemoglobin levels (13.4 g/dL vs. 13.2 g/dL, $p = 0.02$), mean corpuscular haemoglobin (MCH) (28.0 pg vs. 27.2 pg, $p = 0.01$), mean corpuscular haemoglobin concentration (MCHC) (32.4 g/L vs. 31.5 g/L, $p = 0.03$), red cell distribution width–coefficient of variation (RDW-CV) (14.0% vs. 13.5%), and red cell distribution width–standard deviation (RDW-SD) (39.9 fL vs. 37.9 fL) compared to non-smokers. No significant differences were observed in RBC count and other red cell indices ($p > 0.05$). In terms of platelet parameters, median platelet count ($198 \times 10^9/L$ vs. $178 \times 10^9/L$, $p = 0.004$) and plateletcrit (PCT) (1.8% vs. 1.6%, $p = 0.03$) were significantly higher among smokers. Other platelet indices did not differ significantly between the groups ($p > 0.05$).

Table 2
Haematological profile of the study population

Parameter	Smokers (n = 44)	Non-smokers (n = 44)	P-value
WBC ($\times 10^9/L$)	6.0 (5.1–6.6)	4.0 (3.3–4.6)	< 0.001*
Lymphocyte ($\times 10^9/L$)	2.9 (2.4–3.2)	2.1 (1.6–2.5)	< 0.001*
Granulocyte ($\times 10^9/L$)	1.9 (0.6–2.4)	1.5 (1.3–1.8)	0.773
MID Cell ($\times 10^9/L$)	1.0 (0.6–2.2)	0.3 (0.2–0.4)	< 0.001*
RBC ($\times 10^{12}/L$)	4.9 (4.4–5.2)	4.8 (4.5–5.1)	0.907
HB (g/dL)	13.4 (12.9–14.0)	13.2 (11.9–13.6)	0.037*
PCV (%)	41.3 (40.0–43.8)	41.3 (39.0–42.1)	0.515
MCV (fl)	84.7 (81.3–88.8)	86.7 (78.6–89.4)	0.381
MCH (pg)	28.0 (25.7–29.2)	27.2 (24.2–28.8)	0.041*
MCHC (g/l)	32.4 (32.0–32.9)	31.5 (30.7–32.2)	< 0.001*
RDW-CV (%)	14.0 (13.5–14.7)	13.5 (13.2–14.5)	0.013*
RDW-SD (fl)	39.9 (38.0–41.0)	37.9 (35.5–39.3)	< 0.001*
PLT ($\times 10^9/L$)	198.0 (151.3–232.0)	178.0 (136.0–193.0)	0.031*
MPV (fl)	9.6 (8.9–10.0)	9.4 (9.1–10.1)	0.997
PDW (fl)	15.8 (15.4–16.0)	15.8 (15.6–16.1)	0.302
PCT (%)	1.8 (1.5–2.3)	1.6 (1.3–1.9)	0.026*
PLCR (fl)	0.3 (0.2–0.3)	0.3 (0.2–0.3)	0.499
*Statistically significant. Result reported as median (IQR-interquartile range), Median difference significant at $p \leq 0.05$, WBC – White blood cell, MID Cells – Other types of WBC not classified as lymphocytes and granulocytes, RBC – Red blood cell, PCV – Packed Cell Volume, RDW-CV – Red Cell Distribution Width–Coefficient of Variation, RDW-SD – Red Cell Distribution Width–Standard Deviation, MCV – Mean cell volume, MCH – Mean cell haemoglobin, MCHC – Mean cell haemoglobin concentration, PLT – Platelet, MPV – Mean platelet volume, PDW – Platelet distribution width, PCT – Plateletcrit, PLCR – Platelet large cell ratio			

3.3 Coagulation profile of respondents

The coagulation profile of the study population is presented in Table 3. There were no statistically significant differences in the median activated partial thromboplastin time (APTT) and fibrinogen levels between smokers and non-smokers ($p > 0.05$). However, smokers demonstrated significantly prolonged

median prothrombin time (17.6 seconds vs. 14.8 seconds), thrombin time (21.4 seconds vs. 19.0 seconds), and international normalized ratio (1.5 vs. 1.1) compared to non-smokers ($p < 0.05$).

Table 3
Coagulation profile of the study population

Parameter	Smokers (n = 44)	Non-smokers (n = 44)	P-value
PT (Sec)	17.6 (15.5–19.2)	14.8 (13.7–16.1)	< 0.001*
TT(Sec)	21.4 (17.7–24.9)	19.0 (18.0-20.2)	0.042*
APTT (Sec)	28.1 (19.7–63.6)	34.0 (31.4–42.0)	0.223
Fibrinogen (g/L)	2.4 (2.0–3.0)	2.5 (2.2–2.8)	0.509
INR	1.5 (1.2–1.7)	1.1 (1.0-1.3)	< 0.001*

*Statistically significant. Result reported as Median (IQR- interquartile range) Median difference significant at $p < 0.05$, PT - Prothrombin Time, TT - Thrombin Time, APTT – Activated Partial Thromboplastin Time, INR – International Normalized Ratio

4. Discussion

The socio-demographic characteristics of the study population provide important context for interpreting the observed haematological and coagulation findings. The slightly higher median age among smokers compared to non-smokers may suggest a tendency for smoking to be more common among relatively older individuals within the studied age range. This observation is consistent with previous reports indicating that smoking initiation often occurs in late adolescence or early adulthood, influenced by social and environmental factors (O'Loughlin et al., 2017). However, the relatively narrow age difference between the groups suggests that age is unlikely to have substantially influenced the observed laboratory outcomes. Body mass index (BMI) was comparable between smokers and non-smokers, with both groups falling within the normal range. This similarity indicates that nutritional status, as reflected by BMI, was unlikely to confound the relationship between smoking and the haematological parameters assessed. Although smoking has been associated with alterations in body composition and nutritional status in other populations (Algmami et al., 2024), the absence of a difference in this study may reflect the influence of other factors such as dietary patterns or physical activity levels, which were not assessed. The distribution of participants without children in both groups further reflects the relatively young age structure of the study population, consistent with findings from similar demographic settings (Sivagangailakshmi and Rajkumar, 2017). In addition, the predominance of blood group O among both smokers and non-smokers aligns with previous studies reporting its high prevalence in many populations (Anyiam et al., 2023; Apecu et al., 2016). While ABO blood group is not directly related to smoking status in this context, its inclusion provides useful background characterization of the study population. The distributions of marital status, religion, tribe, and occupation highlight the socio-cultural composition of the participants. The predominance of traders in both groups may reflect the occupational structure of the study setting. Although socio-economic factors have been identified as

potential determinants of smoking behaviour (Sandhya et al., 2015), the similarity in these characteristics between smokers and non-smokers in this study suggests a relatively comparable baseline profile, thereby strengthening the validity of subsequent comparisons.

The haematological findings of this study demonstrate significant differences in several blood parameters between smokers and non-smokers, particularly in white blood cell indices, selected red cell parameters, and platelet counts. Smokers exhibited significantly higher total white blood cell counts, as well as elevated lymphocyte and MID cell counts, compared to non-smokers. This observation is consistent with previous reports of increased leukocyte counts among smokers (Malenica et al., 2017). The elevation in white blood cell parameters may reflect a systemic biological response to exposure to cigarette smoke constituents, including oxidative stress. However, given that inflammatory markers were not directly assessed in this study, this interpretation should be considered with caution. The absence of a significant difference in granulocyte counts suggests that the observed changes may be more pronounced in specific leukocyte subpopulations. With respect to red blood cell parameters, smokers demonstrated higher haemoglobin, mean corpuscular haemoglobin (MCH), and mean corpuscular haemoglobin concentration (MCHC) compared to non-smokers, while no significant difference was observed in total red blood cell count. These findings are in agreement with earlier studies reporting smoking-related alterations in red cell indices (Sivagangailakshmi and Rajkumar, 2017). The elevated haemoglobin levels observed among smokers may represent a physiological adaptation to reduced oxygen availability associated with carbon monoxide exposure. The lack of a corresponding increase in red blood cell count suggests that these changes may occur at the level of haemoglobin concentration and red cell characteristics rather than overall erythrocyte production. In terms of platelet parameters, smokers had significantly higher platelet counts and plateletcrit compared to non-smokers. Similar findings have been reported in other studies (Algmami et al., 2024), suggesting that smoking may be associated with alterations in platelet-related indices. While increased platelet levels have been linked to smoking-related biological responses (Sandhya et al., 2015), the underlying mechanisms were not directly evaluated in this study and should therefore be interpreted cautiously. The present findings, however, differ from those reported by Elkhailifa (2018), who observed lower platelet counts among smokers. Such inconsistencies may reflect differences in study populations, sample sizes, or methodological approaches, highlighting the need for further research to better understand the effects of smoking on platelet dynamics.

The coagulation profile analysis revealed significant differences between smokers and non-smokers, particularly in prothrombin time (PT), thrombin time (TT), and international normalized ratio (INR). Median PT was significantly prolonged in smokers (17.6 seconds) compared to non-smokers (14.8 seconds), suggesting that smoking may influence the extrinsic coagulation pathway. This finding is consistent with Algmami et al. (2024), who reported longer PT among smokers, and indicates that smoking could contribute to alterations in coagulation dynamics. Similarly, thrombin time (21.4 seconds vs. 19.0 seconds) and INR (1.5 vs. 1.1) were elevated in smokers, supporting the notion that smoking affects multiple aspects of the coagulation cascade. These changes may reflect a disruption in hemostatic balance, which could increase susceptibility to thrombotic or bleeding complications,

although the clinical implications require further investigation. Unlike some previous studies, no significant differences were observed in activated partial thromboplastin time (APTT) or fibrinogen levels between smokers and non-smokers. While Sivagangailakshmi and Rajkumar (2017) and Elkhalifa (2018) reported prolonged APTT among smokers, the absence of such differences in this study may reflect population-specific factors, variations in smoking duration or intensity, or methodological differences in coagulation testing. Regarding platelet indices, this study observed significantly higher median platelet counts in smokers ($198 \times 10^9/L$) compared to non-smokers ($178 \times 10^9/L$). This contrasts with findings by Algmami et al. (2024), who reported no significant differences, and Sandhya et al. (2015), who observed lower platelet counts in smokers. Such discrepancies may be influenced by variations in study populations, smoking behaviors, or environmental and lifestyle factors, highlighting the complex and context-dependent effects of smoking on platelet physiology.

These findings have important implications for public health policy and clinical practice. The observed haematological and coagulation alterations among smokers highlight the potential for increased cardiovascular and thrombotic risk in this population, emphasizing the need for targeted interventions. Public health strategies in Enugu Metropolis and similar urban centers should prioritize smoking prevention and cessation programs, routine health screening for smokers, and educational campaigns on the hematological and cardiovascular consequences of tobacco use. Clinicians may also consider incorporating periodic haematological and coagulation assessments into the management of individuals with a history of smoking.

This study has some limitations that should be acknowledged. The relatively small sample size and restriction to male participants may limit the generalizability of the findings to other populations, including females and broader age groups. Additionally, the cross-sectional design precludes causal inference, and unmeasured factors such as diet, physical activity, or environmental exposures could have influenced haematological and coagulation parameters. Future studies with larger, more diverse populations and longitudinal designs are recommended to confirm these findings and to clarify the mechanisms underlying smoking-related haematological changes.

5. Conclusions

This study demonstrates that smoking is associated with significant alterations in haematological and coagulation parameters, including elevated white blood cell counts, selected red blood cell indices, platelet counts, prothrombin time, thrombin time, and INR. These changes suggest a disruption of haemostatic balance and a potential increased risk of inflammatory and thrombotic complications among smokers. The findings underscore the importance of public health interventions, including smoking cessation programs and routine monitoring of haematological and coagulation profiles, to mitigate these risks and promote cardiovascular and overall health in populations exposed to tobacco smoke.

Abbreviations

WHO
World Health Organization
EDTA
Ethylenediaminetetraacetic acid
BMI
Body Mass Index
WBC
White Blood Cell
MID Cells
Other types of WBC not classified as lymphocytes or granulocytes
RBC
Red Blood Cell
PCV
Packed Cell Volume
RDW
CV–Red Cell Distribution Width–Coefficient of Variation
RDW
SD–Red Cell Distribution Width–Standard Deviation
MCV
Mean Cell Volume
MCH
Mean Cell Haemoglobin
MCHC
Mean Cell Haemoglobin Concentration
PLT
Platelet
MPV
Mean Platelet Volume
PDW
Platelet Distribution Width
PCT
Plateletcrit
PLCR
Platelet Large Cell Ratio
PT
Prothrombin Time
TT
Thrombin Time
APTT
Activated Partial Thromboplastin Time

Declarations

Ethical consideration

The study adhered strictly to international ethical guidelines as outlined in the declaration of Helsinki. Ethical approval was obtained from the Enugu State Ministry of Health Research Ethics Committee (MH/MSD/REC21/633). Participants provided written and verbal informed consent after a thorough explanation of the study's objectives, risks, and benefits. Confidentiality was ensured through anonymized data collection and restricted access to research personnel. Blood samples were collected under sterile conditions by trained professionals, with post-procedure monitoring for adverse effects. Participants were treated with respect, and autonomy was upheld throughout the study.

Consent for publication

Not applicable

Availability of data and materials

The datasets supporting the conclusions of this article are included within the article (and its additional files).

Competing interests

The authors declare that they have no competing interests.

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Authors' contribution

EDE and ULN conceptualized the study, contributed to the study design, participated in data collection and analysis, and critically revised the manuscript. LOP was involved in the study design and also drafted and revised the manuscript. SAU contributed to the study design, data collection, analysis, and manuscript revision. CCO provided resources and assisted with manuscript revision, while ACUE also contributed resources and critically revised the manuscript. All authors read and approved the final version of the manuscript.

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Figures

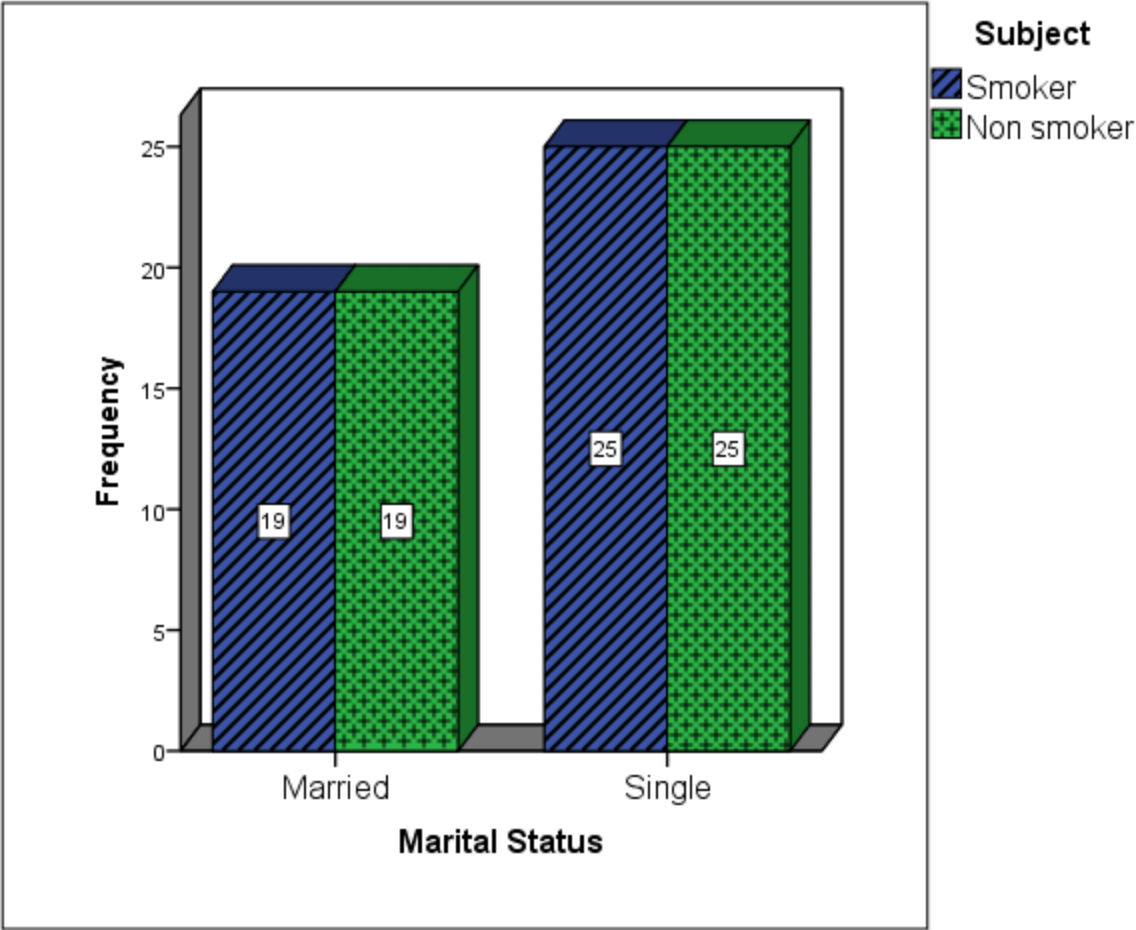


Figure 1

Distribution of marital status of respondents

Bar Chart

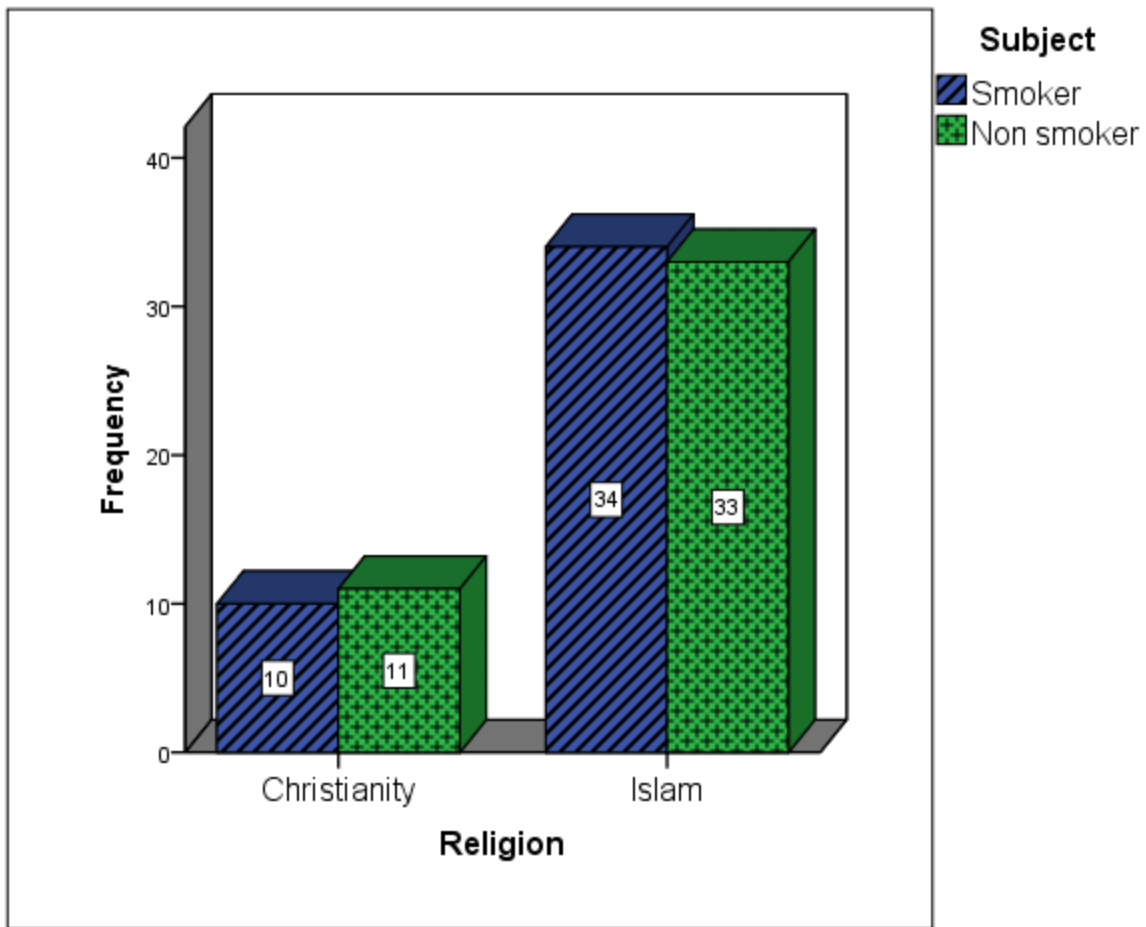


Figure 2

Distribution of religion of the study population

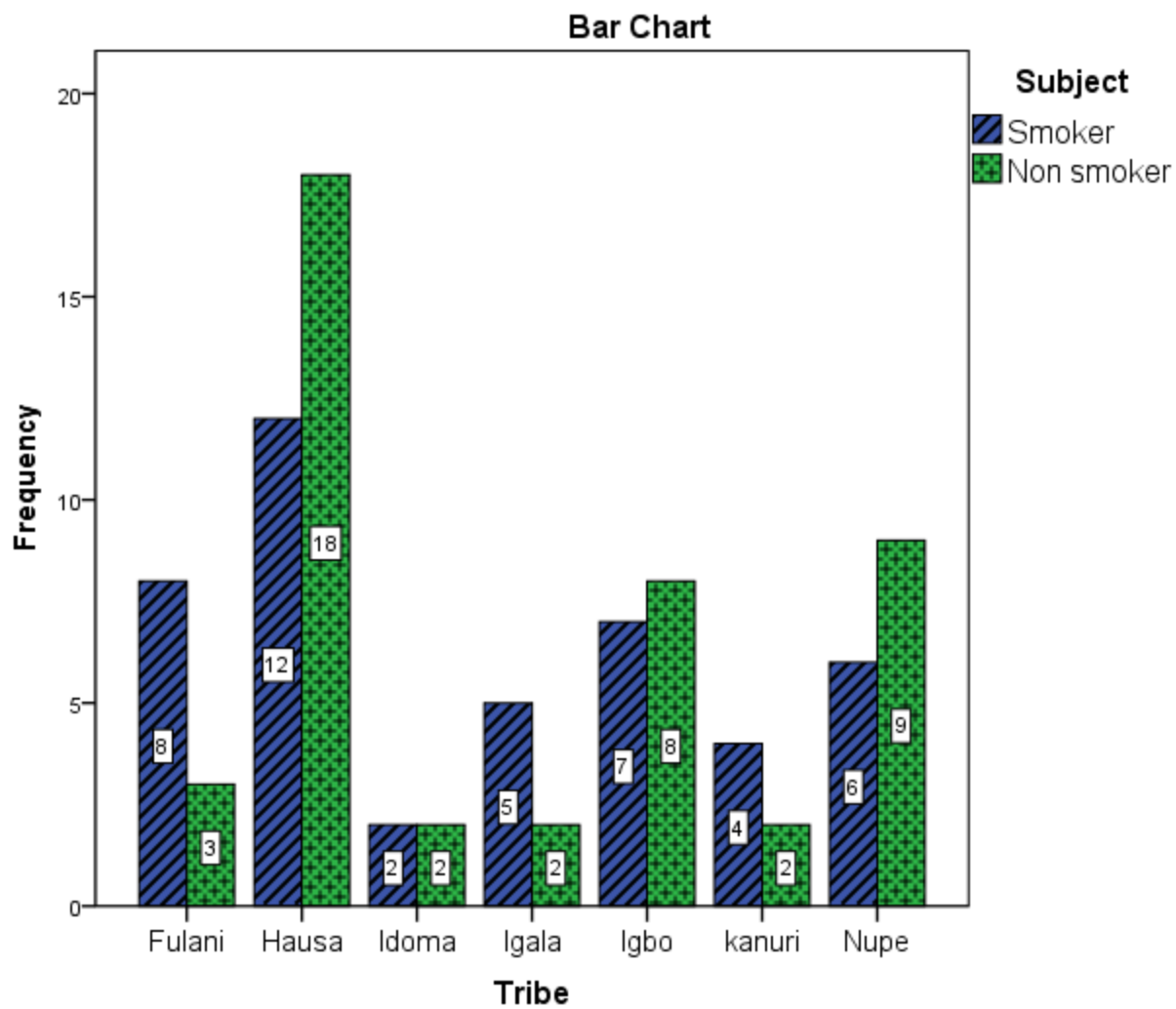


Figure 3

Distribution of tribe of the study population

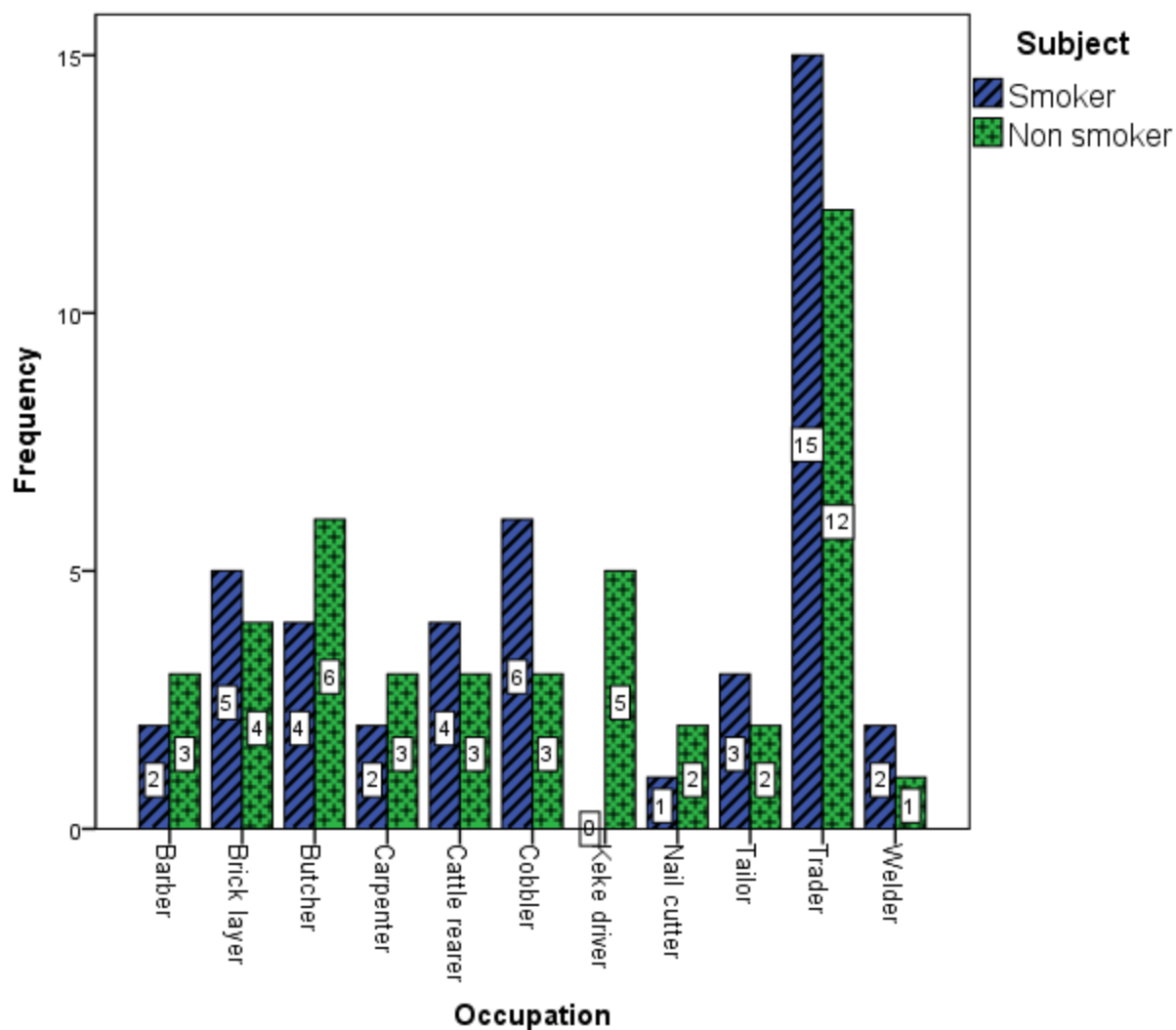


Figure 4

Distribution of occupation of the study population

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Dataset1smokers.xls](#)
- [Dataset2smokers.xlsx](#)