Original Research Article

**Variation in hematological parameters among smokers and non-smokers in Benin City**

# ABSTRACT

**Background**: Cigarette smoking is a well-established risk factor for systemic inflammation and hematological alterations, even in early stages of exposure. Increasing prevalence of smoking, particularly among young adults and urban populations, understanding its effects on inflammatory and hematological markers is crucial for public health interventions.

**Aim:** This study aims to bridge the knowledge gap by examining the impact of smoking on CRP, albumin, and FBC among smokers in Benin City, thereby contributing to a better understanding of the systemic effects of tobacco use in this population.

**Methods:** This Cross-sectional study evaluated the hematological and inflammatory effects of cigarette smoking in healthy male adults by comparing the variations in C-reactive protein (CRP), albumin and haematological indices in smokers and non-smokers. A total of 166 male participants were enrolled and grouped into smokers (n = 83) and non-smokers (n = 83), with comparable age distributions. Fasting venous blood samples were collected and analyzed for serum C-reactive protein (CRP), albumin, full blood count, and derived indices such as platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR).

**Results:** The analysis revealed a significant increase in inflammatory markers among smokers, with CRP levels markedly elevated (22.48 ± 8.53 vs. 3.86 ± 1.08; p = 0.001), while albumin also increased (46.64 ± 3.79 vs. 36.22 ± 5.73; p = 0.001). Red blood cell indices such as haemoglobin (15.96 ± 3.88 vs. 13.34 ± 2.09; p = 0.005), haematocrit (53.67 ± 7.40 vs. 42.96 ± 4.61; p = 0.001), Mean corpuscular volume (MCV) (89.58 ± 5.13 vs. 79.00 ± 6.24; p = 0.008), and RDW-SD (Red cell distribution width-standard deviation) (54.72 ± 6.87 vs. 47.85 ± 5.79; p = 0.001) were significantly higher in smokers than in non-smokers. Conversely, PLT (platelet) (155.72 ± 17.76 vs. 264.20 ± 29.35; p = 0.001) and PLR (39.43 ± 8.64 vs. 81.55 ± 6.98; p = 0.001) were significantly lower. No significant differences were observed in total white blood cell count (WBC) red blood cell count (RBC), Mean corpuscular haemoglobin (MCH), and NLR (p > 0.05). Correlation analysis among smokers revealed strong positive relationships between RBC and HGB (r = 0.933; p = 0.001), and PLT and Plateletcrit (PCT) (r = 0.965; p = 0.001), while negative correlations were observed between CRP and PLR (r = –0.548; p = 0.005) and Mean corpuscular haemoglobin concentration (MCHC) and RDW-SD (r = –0.794; p = 0.001), indicating systemic dysregulation.

**Conclusion:** The results suggest that even in early exposure, cigarette smoking significantly alters hematological and inflammatory markers, with a potential predisposition to future cardiovascular and immune-related complications. Routine hematological profiling may serve as an early indicator of smoking-related stress and reinforce the need for early cessation and preventive interventions.

**Keywords**: Cigarette smoking, Inflammatory biomarkers, Full blood count, Albumin, C-reactive protein, Male smokers.

# INTRODUCTION

Smoking is a significant public health concern worldwide (WHO, 2025; Global Burden of Disease, 2023), with well-documented effects on various physiological and biochemical parameters (Ezeugwunne *et al*., 2019; Okwara *et al*., 2018; Analike *et al*., 2017). It has been identified as a major risk factor for cardiovascular diseases, respiratory disorders, and metabolic dysfunctions (Rahman *et al*., 2025; Zarghami *et al*., 2025; Fu *et al*., 2024). Despite widespread awareness regarding the health risks associated with cigarette smoking, its prevalence remains notably high particularly in developing nations, presenting an ongoing and substantial public health issue. Tobacco smoke, along with nicotine and other harmful substances, is initially absorbed into the lungs which can cause Chronic Obstructive Pulmonary Disease (COPD), subsequently enters the bloodstream, from where it is distributed across the body. This makes blood an ideal biological sample for investigating the systemic effects of tobacco smoke exposure. Nevertheless, these toxic components travel through the body by diverse means, leading to harm and numerous conditions, including cardiovascular diseases, anemia, altered blood viscosity, hypoxia (Victor and Ola, 2016; Hussein et al., 2024). Cigarette smoke contains more than 7,000 chemicals, many of which are known carcinogens and pro-inflammatory agents that contribute to systemic inflammation and oxidative stress (National Toxicology Program, 2016; U.S. Department of Health and Human Services, 2014; International Agency for Research on Cancer (IARC), 2004). Chronic exposure to these harmful compounds results in significant variations in C-reactive protein (CRP), albumin levels, and full blood count (FBC), making them key biomarkers in assessing the impact of smoking on health (Mohamed *et al*., 2023; Jiang *et al*., 2020; Adunmo *et al*., 2024). Understanding these variations is crucial in evaluating the potential risks associated with smoking, particularly among populations in Benin City, where smoking prevalence and its health consequences remain an area of concern though without adequate documented studies. Tobacco Smoking also affects red blood cells and hemoglobin. Hemoglobin and hematocrit are higher in smokers this Increase in hemoglobin concentration is believed to be mediated by exposure of carbon monoxide and some scientists suggested that an increase in hemoglobin level in the blood of smokers could be a compensatory mechanism (Malenica et al., 2017; Jindal et al., 2023).

C-reactive protein (CRP) is an acute-phase inflammatory marker synthesized by the liver in response to inflammation, infection, and tissue damage (Sproston and Ashworth, 2018; Wang and Shi, 2020; Cottin and Valenzuela, 2024). Elevated CRP levels have been strongly associated with increased risks of cardiovascular diseases, chronic obstructive pulmonary disease (COPD), and metabolic syndrome (Tonstad and Cowan, 2009; Barron *et al*., 2015; Keeratichananont *et al*., 2023; Banerjee *et al*., 2024). Studies have shown that smokers exhibit significantly higher CRP levels compared to non-smokers, indicating persistent systemic inflammation (Mustafa *et al*., 2025; Elisia *et al*., 2020). The mechanism behind this elevation is linked to the activation of inflammatory pathways due to the toxic effects of cigarette smoke on endothelial cells and immune responses (Ardiana *et al*., 2023; Koru and Atasever-Arslan, 2025; Khudhur *et al*., 2025). A study conducted in Nigeria found that smokers had higher serum CRP concentrations, suggesting that chronic smoking contributes to a sustained inflammatory state, predisposing individuals to cardiovascular complications (Ibeh *et al*., 2017; Eworo *et al*., 2019). Furthermore, CRP has been identified as an independent predictor of atherosclerosis and stroke, making its elevation among smokers a crucial area of concern (Van Der Meer *et al*., 2002; Molino-Lova *et al*., 2011).

Albumin, a major plasma protein synthesized in the liver, plays an essential role in maintaining oncotic pressure and serving as a transporter of various endogenous and exogenous substances (Moman *et al*., 2022; Mathew *et al*., 2023). Serum albumin levels are often used as a marker of nutritional status, hepatic function, and systemic inflammation (Keller, 2019; Gremese *et al*., 2023; Ehiaghe *et al*., 2025a; Ehiaghe *et al*., 2025a). Smoking has been linked to reduced serum albumin levels, which may result from increased oxidative stress and inflammatory responses leading to hepatic dysfunction (Conde de la Rosa *et al*., 2022). Reduced albumin levels have been associated with a higher risk of cardiovascular disease, chronic kidney disease, and poor immune function (Lang *et al*., 2018; Yoshioka *et al*., 2023; Sultan and Lesloom, 2024; Czinege *et al*., 2025).

The full blood count (FBC) is an essential hematological test used to assess the overall health of an individual by evaluating parameters such as red blood cells (RBC), white blood cells (WBC), and platelet count. Smoking has been shown to alter several hematological indices, reflecting its systemic impact on various physiological processes (Malenica *et al*., 2017; Ahmed *et al*., 2024). Studies indicate that smokers generally exhibit increased white blood cell counts (leukocytosis) (Pedersen *et al*., 2019), a marker of chronic inflammation and immune activation due to constant exposure to tobacco toxins (Dahdah *et al*., 2022). Additionally, smoking has been linked to polycythemia (elevated RBC count), which occurs as a compensatory response to chronic hypoxia induced by carbon monoxide in cigarette smoke (Thakur and Westover, 2011; Alkhedaide, 2020). Platelet aggregation and activation are also increased in smokers, contributing to a higher risk of thrombotic events, including heart attacks and strokes (Hung *et al*., 1995; Pamukcu *et al*., 2011). Beyond conventional FBC parameters, immune cell ratios such as the neutrophil-to-lymphocyte ratio (NLR**)** have emerged as powerful indicators of systemic inflammation and immune dysregulation in smokers. NLR reflects the balance between innate immune activation (neutrophils) and adaptive immune suppression (lymphocytes), and elevated values have been linked to cardiovascular disease, cancer, and poor prognosis in chronic inflammatory states (Buonacera *et al*., 2022; Kim *et al*., 2018). Elevated NLR in smokers has been documented in previous studies further supporting its role as a sensitive marker of chronic immune stress and systemic inflammation (Güden *et al*., 2022; Ibeh *et al*., 2017).

Despite extensive research on the impact of smoking on various health parameters, limited studies have specifically examined the variations in CRP, albumin, and FBC among smokers in Benin City. Given the increasing prevalence of smoking, particularly among young adults and urban populations, understanding its effects on inflammatory and hematological markers is crucial for public health interventions. Studies in other African populations have demonstrated significant alterations in these biomarkers among smokers, emphasizing the need for localized research to determine the specific risks and guide effective health policies. More so, environmental and genetic factors may influence the degree to which smoking alters these biomarkers, making it essential to conduct region-specific investigations. This study aims to bridge the knowledge gap by examining the impact of smoking on CRP, albumin, and FBC among smokers in Benin City, thereby contributing to a better understanding of the systemic effects of tobacco use in this population.

# MATERIALS AND METHODS

# Study Area

This study was carried out in the Haematology Laboratory, Benson Idahosa University, Benin, Edo State. Edo state lies between longitude 06o 04IE and 06o 43IE and latitude 05o 44IN and 07o 34IN with a land mass of 17, 450 sq.km located in the south-south geopolitical zone of Nigeria with a population of 3.1 million people.

# Study Design and population

This study adopted a cross-sectional approach in evaluating the variations in C-reactive protein (CRP), albumin levels, and full blood count (WBC: Total white blood cells Count; RBC: Red blood cell count; MCV: Mean corpuscular volume; MCH: Mean corpuscular haemoglobin; MCHC: Mean corpuscular haemoglobin concentration; PLT: Platelet count; PCT: Plateletcrit; HGB: Haemoglobin; HCT: Haematocrit; MPV: Mean Platelet Volume; LYM%: Lymphocyte count percentage; LYM#: Lymphocyte count absolute; GRAN%: Granulocyte count percentage; GRAN#: Granulocyte count absolute; MID%: Middle cell count percentage; MID#: Middle cell count absolute; RDW-CV: Red cell Distribution Width-Coefficient of Variation; RDW-SD: Red cell Distribution Width-Standard Deviation; NLR: Neutrophil-Lymphocyte ratio; PLR: Platelet-Lymphocyte ratio) among smokers and non-smokers in Benin City. The study population consisted of adult males aged 18 years and above, categorised into smokers and non-smokers based on self-reported smoking habits. A total of 166 participants, consisting of 83 smokers and 83 non-smokers, were recruited for this study using a simple random sampling method. A well-structured questionnaire was administered to collect information on biodata and some demographic characteristics. Participants were recruited from hospitals, public spaces, and smoking zones within Benin City. Smokers were individuals who had been actively smoking at least one cigarette per day for a minimum of one year, whereas non-smokers were individuals who had never smoked or were passive for at least five years.

# Sample Size Determination

The sample size was determined using the Cochran formula for cross-sectional studies:

n=Z2P(1−P)/d2​

where:

*n* = required sample size

*Z* = standard normal deviation at 95% confidence level (1.96)

*P* = estimated prevalence of smoking-related inflammation is not known but the prevalence of smokers in Nigeria is 5.7%, (Fawibe and Shittu, 2011).

*d* = margin of error (set at 5%)

Substituting the values in the formula:

 N = 1.96² × 0.057 × (1-0.057) / 0.05²

N = 3.8416 × 0.057× 0.943 / 0.0025 = 82.59

So, 166 participants were recruited for this study, 83 Smoker and 83 non-smokers.

# Inclusion Criteria

1. Adults aged 18 years and above.
2. Individuals categorized as smokers or non-smokers based on their smoking history.
3. Participants who provided informed consent.

**Exclusion Criteria**

1. Individuals with a history of chronic inflammatory diseases, liver dysfunction, or hematological disorders.
2. Participants on medications affecting CRP, albumin, or blood counts.

**Sample Collection and Preparation**

Samples for Full Blood Count (FBC) were collected in EDTA containers (2ml) and plain containers were used for the collection of samples for serum albumin and CRP (4ml). The samples collected in the plain tubes were centrifuged for 5minutes at 4000 rpm to separate the serum from the whole blood and the serum was transferred into another container. The levels of serum albumin, CRP and haematological parameters were determined using standard methods.

# Laboratory Analysis

**Measurement of Full Blood Count**

The White blood cells, Red blood cells and Platelet parameters, red cell indices etc were analyzed by a three part Biobase hematology analyzer (BK-6190, China), with neutrophil-lymphocyte ratio (NLR) and platelet-lymphocyte ratio (PLR) calculated.

**Determination of C-reactive protein Level (CRP)**

The sandwich enzyme-linked immunosorbent assay (sandwich-ELISA) method was used for the determination of CRP levels in the participants’ sera.

**Estimation of Serum Albumin**

This was determined using the Bromocresol green method (BCG), as described by Doumas and Watson (1971) and cited by Okpogba *et al*. (2021).

**Statistical Analysis**

Data were analyzed using IBM SPSS Statistics (version 25.0) and presented using mean $\pm $ standard deviation. Student t-test and Pearson correlation were used to determine statistical difference and the association between variables. The values were considered statistically significant at p value $<$0.05.

# RESULTS

The mean age (years) did not differ significantly in the smokers compared to the control group (p = 0.930), although the mean duration of smoking was significantly higher in the smokers than in control group (1.44±0.50 Vs 0.00±0.00; p = 0.001). Furthermore, there were statistically significantly higher mean serum albumin (46.64±3.79 Vs 36.22±5.73; p = 0.001) and CRP (22.48±8.53 Vs 3.86±1.08; p = 0.001) levels in the smokers compared to the control group respectively. See table 1.

The mean HGB (15.96±3.88 Vs 13.34±2.09; p = 0.005), HCT (53.67±7.40 Vs 42.96±4.61; p = 0.001), MCV (89.58±5.13 Vs 79.00±6.24; p = 0.008), RDW-SD (54.72±6.87 Vs 47.85±5.79; p = 0.001) and RDW-CV (16.56±1.30 Vs 15.34±2.21; p = 0.022) were all significantly higher in the smokers than in the control group respectively whereas, the mean PLT (155.72±17.76 Vs 264.20±29.35; p = 0.001) and PLR (39.43±8.64 Vs 81.55±6.98; p = 0.001) were significantly lower in the smokers compared to the observed values in the control group respectively. However, the mean WBC, LYM%, LYM#, MID%, MID#, GRAN%, GRAN#, RBC, MCH, MCHC, MPV and NLR values did not differ significantly (p>0.05) in the smokers compared to the control group respectively. See table 2.

There were statistically significant (p = 0.001) positive correlations found between the levels of WBC Vs LYM# (r = 0.987), WBC Vs GRAN# (r = 0.782), WBC Vs PLT (r = 0.711), WBC Vs PCT (r = 0.790), LYM# Vs MID# (r = 0.782), LYM# Vs GRAN# (r = 0.686), LYM# Vs PLT (r = 0.740), LYM# Vs PCT (r = 0.824), MID# Vs GRAN# (r = 0.867), GRAN% Vs NLR (r = 0.857), RBC Vs HGB (r = 0.933), RBC Vs HCT (r = 0.925), HCT Vs HGB (r = 0.976), MCV Vs MCH (r = 0.840), MCV Vs RDW-SD (r = 0.677), MCH Vs RDW-SD (r = 0.789), RDW-SD Vs RDW-CV (r = 0.794), PLT Vs PCT (r = 0.965), and MPV Vs MCH (r = 0.639) in the smokers respectively. on the other hand, statistically significant negative correlations were found between the levels of WBC Vs MID# (r = - 0.865; p = 0.001), LYM% Vs MID% (r = - 0.815; p = 0.001), LYM% Vs GRAN% (r = - 0.830; p = 0.001), LYM% Vs NLR (r = - 0.874; p = 0.001), MCV Vs MPV (r = - 0.612; p = 0.001), MCHC Vs MCV (r = - 0.648; p = 0.001), MCHC Vs RDW-SD (r = - 0.794; p = 0.001), CRP Vs PLR (r = - 0.548; p = 0.005), CRP Vs MCHC (r = - 0.589; p = 0.002), and PLR Vs MID# (r = - 0.547; p = 0.005), in the smokers respectively. See table 3.

There were statistically significant positive correlations found between the levels of WBC Vs LYM# (r = 0.929; p = 0.001), WBC Vs MID# (r = 0.678; p = 0.001), MID% Vs WBC (r = 0.678; p = 0.001), GRAN% Vs GRAN# (r = 0.788; p = 0.001), GRAN% Vs NLR (r = 0.794; p = 0.001), GRAN% Vs NLR (r = 0.971; p = 0.001), HGB Vs MCV (r = 0.963; p = 0.001), HCT Vs HGB (r = 0.963; p = 0.001), MCV Vs MCHC (r = 0.603; p = 0.001), RDW-SD Vs RDW-CV (r = 0.599; p = 0.002), RDW-CV Vs Age (r = 0.602; p = 0.001) and PLT Vs PCT (r = 0.988; p = 0.001), in the control subjects respectively. See table 4.

However, statistically significant negative correlations were found between the levels of WBC Vs PLR# (r = - 0.684; p = 0.001), LYM% Vs GRAN% (r = - 0.825; p = 0.001), LYM% Vs GRAN# (r = - 0.733; p = 0.001), LYM% Vs NLR (r = - 0.876; p = 0.001), MID% Vs LYM% (r = - 0.630; p = 0.001), MID% Vs MID# (r = - 0.750; p = 0.001), MID# Vs PLR (r = - 0.537; p = 0.006) and PLR Vs LYM# (r = - 0.630; p = 0.001), in the control subjects respectively. (See Table 4).

# DISCUSSION

## This study examined the impact of smoking on various hematological and biochemical parameters by comparing a group of smokers to a matched control group. The analysis revealed significant alterations in inflammatory markers, red blood cell indices, and platelet characteristics among smokers, which collectively reflect the systemic impact of tobacco exposure. The findings are discussed in detail, drawing comparisons with established literature, and highlighting the implications and divergences observed.

The elevation in C-reactive protein (CRP) among smokers was one of the most significant findings. As a key acute-phase reactant and marker of inflammation, the high CRP levels in smokers strongly suggest the presence of systemic inflammation triggered by smoke exposure. These results are consistent with those of Fathima and Kalyanikutty (2024), Adunmo *et al*. (2017), Ibeh *et al*. (2017) and Eworo *et al*. (2019), who noted a similar inflammatory response to cigarette smoke, which may be due to persistent oxidative stress as a result of antioxidant depletion and endothelial injury (Caliri *et al*., 2021; Ezeugwunne *et al*., 2019; Okwara *et al*., 2018). A review study by Strzelak *et al*. (2018) demonstrated how the imbalance between oxidants and antioxidants resulting from exposure to tobacco smoke leads to oxidative stress, increased mucosal inflammation, and increased expression of inflammatory cytokines (such as interleukin (IL)-8, IL-6 and tumor necrosis factor α ([TNF]-α). In addition, Addissouky *et al*. (2024) noted that combustible cigarettes release thousands of chemicals, including carcinogens and oxidants that can initiate inflammatory pathways and lead to respiratory, cardiovascular, and other diseases, with COPD, lung cancers, atherosclerosis, and stroke exemplifying smoking-related illnesses. Wannamethee *et al*. (2005) also supported this, linking smoking to increased levels of CRP and fibrinogen, both of which contribute to cardiovascular risk.

Contrary to predictions, smokers had higher serum albumin levels, which could be the result of nutritional variability or early adaptive liver responses. This finding is in line with Festus *et al*. (2024) who observed higher levels of total protein and albumin in smokers than in controls stating that the increased levels of albumin and total protein may be attributed to a compensatory increase in protein production by the liver, potentially in response to the dehydration commonly associated with smoking and that the heightened protein synthesis could reflect the body’s attempt to counteract the physiological stresses imposed by smoking. Nevertheless, while previous reports such as Nabila *et al*. (2017), Roohi *et al*. (2017) and Suriyaprom*e et al*. (2007) found reduced albumin in smokers due to hepatic stress or protein catabolism, the elevated levels in the current study may be reflective of early-phase adaptation or individual nutritional variability. The relatively short average duration of smoking (1.44 years) in this study population could account for this deviation, suggesting that albumin changes may only manifest with longer or more intense exposure.

Red blood cell indices were notably affected in smokers. Elevated hemoglobin and hematocrit levels suggest a compensatory response to chronic hypoxia caused by carbon monoxide in cigarette smoke, which reduces hemoglobin’s oxygen-carrying capacity. These findings are supported by Ahmed *et al*. (2024) and Hussain *et al*. (2022), who also observed increases in these parameters in smokers. The rise in mean corpuscular volume (MCV) and red cell distribution width (RDW) further indicates macrocytosis and anisocytosis, likely reflecting a combination of oxidative stress, nutrient deficiency, and toxic marrow effects. Several studies (Açık et al., 2020; Çiftçiler et al., 2019; Malenica et al., 2017) found an increase in MCV, which in direct agreement with our findings. RDW in particular is increasingly recognised as a sensitive marker of systemic stress and has been shown to correlate with smoking in studies like those by Ma *et al*. (2024) and Elisia *et al*. (2020).

Another significant observation in this study was the reduction in platelet count and platelet-to-lymphocyte ratio (PLR) in smokers. While many inflammatory and thrombotic conditions report elevated PLR, smokers in this study exhibited reduced PLT. One plausible explanation is the chronic activation and subsequent exhaustion of platelets due to endothelial injury and low-grade thrombotic activity induced by smoking. Elkhalifa *et al*. (2018) found that smokers showed altered platelet counts and coagulation markers, supporting the notion of progressive platelet consumption and dysfunction. Furthermore, Amalia *et al*. (2022) highlighted the predictive role of platelet-selectin in thrombotic risk, noting that smoking exacerbates platelet activation in autoimmune and vascular disorders. Adding to this, Cirillo *et al*. (2025) showed that e-cigarette exposure could induce the expression of tissue factor in endothelial cells, promoting a procoagulant state, further substantiating that smoking-related thrombogenicity may stem from both cellular and vascular mechanisms.

The correlation analysis further reveals the systemic nature of these hematologic disruptions. In the smoker group, there were strong positive correlations between WBC and LYM#, RBC and HGB, and PLT and plateletcrit, indicating that smoking may induce systemic, coordinated shifts in hematological profiles. Notably, negative correlations, such as between CRP and PLR or between LYM% and GRAN% hint at immunological dysregulation. These findings contrast with the more stable and predictable correlation patterns observed in non-smokers, suggesting that the act of smoking alters the internal regulatory relationships among inflammatory and hematological parameters.

Compared to existing literature, most findings from this studyespecially elevated CRP, hemoglobin, RDW, and macrocytosisare consistent with prior research, indicating a clear and reproducible pattern of systemic effects due to smoking. However, the higher albumin and lower PLR observed differ from many prior studies, suggesting that the timeline, intensity of exposure, and nutritional factors may play significant roles in modulating the hematologic response to smoking.

**Summary of key findings**

Results showed that smokers had significantly elevated C-reactive protein (CRP) levels, indicating systemic inflammation. Red blood cell indices *such* as hemoglobin, hematocrit, MCV, and RDW *were* also higher, suggesting a compensatory response to hypoxia and oxidative stress. Interestingly, serum albumin was elevated in smokers, possibly due to short smoking duration or individual nutritional factors. Platelet count and platelet-to-lymphocyte ratio (PLR) were significantly reduced, likely reflecting chronic platelet activation and exhaustion. Correlation analyses revealed disrupted hematological balance in smokers compared to stable patterns in non-smokers. Overall, the findings confirm that smoking induces early inflammatory and hematological changes, even before long-term exposure, emphasising the need for early screening and cessation strategies.

# ****Conclusion****

The findings of this study clearly demonstrate that smoking, even over a relatively short period, has significant physiological impacts on inflammatory and hematological parameters. The observed elevation in C-reactive protein (CRP) confirms the presence of systemic inflammation among smokers, while increases in hemoglobin, hematocrit, mean corpuscular volume (MCV), and red cell distribution width (RDW) suggest a smoking-induced alteration in erythropoiesis and red cell morphology. At the same time, reductions in platelet count and platelet-to-lymphocyte ratio (PLR) point toward potential disturbances in hemostatic balance, likely driven by endothelial dysfunction and chronic platelet activation. Moreover, the complex correlation patterns observed in smokers *as* opposed to the more stable hematological relationships in non-smokers *highlight* the disruptive influence of smoking on physiological homeostasis. These alterations, although subclinical, may represent early biomarkers of cardiovascular and hematologic disease risk.

#  Recommendation

This study highlights the need for routine hematological and inflammatory screening in smokers, even in early stages of exposure. Health practitioners should monitor markers such as CRP, RDW, and PLR as potential early indicators of systemic stress. Public health campaigns should emphasise the immediate effects of smoking on blood parameters to encourage early cessation. Future studies with larger populations and longer follow-up are recommended to further assess the progression and clinical implications of these changes. Interventions focusing on antioxidant support and lifestyle modification may also be explored as protective strategies.

Ethical approval

Ethical approval was obtained from the Edo State Ministry of Health, Benin City, Edo state,

Nigeria with Ref No: HA/737/25/D/0424068. It was also approved by the ethics committee of Benson Idahosa University to ensure compliance with ethical standards and guidelines for research involving human subjects.

Informed Consent

Informed consent was obtained from all participants prior to enrollment in the study, ensuring voluntary participation and protection of participants' rights and privacy.

Disclaimer (Artificial intelligence)

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, manuscript.

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Table.1: Age, Duration of smoking and serum levels of albumin and CRP in Smokers and Control group (Mean± SD, n = 50)

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Variables | Smokers(Mean± SD; n =25) | Control group (Mean± SD; n =25) | t-value | p-value |
| Age  | 28.16±8.34 | 27.96±7.64 | 0.088 | 0.930 |
| Duration of smoking | 1.44±0.50 | 0.00±0.00 | 14.212 | 0.001\* |
| Albumin  | 46.64±3.79 | 36.22±5.73 | 7.574 | 0.001\* |
| CRP | 22.48±8.53 | 3.86±1.08 | 10.821 | 0.001\* |

\*Statistically significant at p<0.05.

Table 2: Levels of Haematological parameters in Smokers and Control group (Mean±SD, n = 50)

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Variables | Smokers (Mean± SD, n = 25) | Control group (Mean± SD, n = 25) | t-value | p-value |
| WBC | 6.31±2.10 | 5.08±1.62 | 1.385 | 0.172 |
| LYM% | 70.93±6.28 | 68.42±9.54 | 1.099 | 0.277 |
| LYM# | 4.51±1.27 | 3.50±1.32 | 1.432 | 0.159 |
| MID% | 15.22±3.76 | 15.84±5.08 | - 0.493 | 0.624 |
| MID# | 0.98±0.61 | 0.81±0.38 | 1.180 | 0.244 |
| GRAN% | 13.40±4.16 | 15.61±5.96 | - 1.225 | 0.266 |
| GRAN# | 0.81±0.43 | 0.77±0.38 | 0.312 | 0.757 |
| RBC | 5.97±1.33 | 5.61±1.97 | 0.752 | 0.456 |
| HGB | 15.96±3.88 | 13.34±2.09 | 2.955 | 0.005\* |
| HCT | 53.67±7.40 | 42.96±4.61 | 3.380 | 0.001\* |
| MCV | 89.58±5.13 | 79.00±6.24 | 2.763 | 0.008\* |
| MCH | 26.63±2.49 | 25.40±1.94 | 1.941 | 0.058 |
| MCHC | 29.90±1.93 | 30.81±1.87 | - 1.683 | 0.099 |
| RDW-SD | 54.72±6.87 | 47.85±5.79 | 3.821 | 0.001\* |
| RDW-CV | 16.56±1.30 | 15.34±2.21 | 2.372 | 0.022\* |
| PLT | 155.72±17.76 | 264.20±29.35 | - 3.669 | 0.001\* |
| MPV | 10.96±0.87 | 11.39±2.08 | - 0.949 | 0.348 |
| PCT | 0.21±0.13 | 0.28±0.10 | - 1.048 | 0.300 |
| NLR | 0.20±0.08 | 0.25±0.16 | - 1.429 | 0.160 |
| PLR | 39.43±8.64 | 81.55±6.98 | - 6.422 | 0.001\* |

\*Statistically significant at p<0.05.

Note\*: KEYS: WBC: Total white blood cells Count; RBC: Red blood cell count; MCV: Mean corpuscular volume; MCH: Mean corpuscular haemoglobin; MCHC: Mean corpuscular haemoglobin concentration; PLT: Platelet count; PCT: Plateletcrit; HGB: Haemoglobin; HCT: Haematocrit; MPV: Mean Platelet Volume; LYM%: Lymphocyte count percentage; LYM#: Lymphocyte count absolute; GRAN%: Granulocyte count percentage; GRAN#: Granulocyte count absolute; MID%: Middle cell count percentage; MID#: Middle cell count absolute; RDW-CV: Red cell Distribution Width-Coefficient of Variation; RDW-SD: Red cell Distribution Width-Standard Deviation; NLR: Neutrophil-Lymphocyte ratio; PLR: Platelet-Lymphocyte ratio.

Table 3: Levels of Associations between parameters studied in the smokers (test group).

|  |  |  |
| --- | --- | --- |
| Variables | r-value | p-value |
| WBC Vs LYM# | 0.987 | 0.001 |
| WBC Vs MID# | - 0.865 | 0.001 |
| WBC Vs GRAN# | 0.782 | 0.001 |
| WBC Vs PLT | 0.711 | 0.001 |
| WBC Vs PCT | 0.790 | 0.001 |
| LYM% Vs MID% | - 0.815 | 0.001 |
| LYM% Vs GRAN% | - 0.830 | 0.001 |
| LYM% Vs NLR | - 0.874 | 0.001 |
| LYM# Vs MID# | 0.782 | 0.001 |
| LYM# Vs GRAN# | 0.686 | 0.001 |
| LYM# Vs PLT | 0.740 | 0.001 |
| LYM# Vs PCT | 0.824 | 0.001 |
| MID# Vs GRAN# | 0.867 | 0.001 |
| GRAN% Vs NLR | 0.857 | 0.001 |
| RBC Vs HGB | 0.933 | 0.001 |
| RBC Vs HCT | 0.925 | 0.001 |
| HCT Vs HGB | 0.976 | 0.001 |
| MCV Vs MCH | 0.840 | 0.001 |
| MCV Vs RDW-SD | 0.677 | 0.001 |
| MCV Vs MPV | - 0.612 | 0.001 |
| MCH Vs RDW-SD | 0.789 | 0.001 |
| MCHC Vs MCV | - 0.648 | 0.001 |
| MCHC Vs RDW-SD | - 0.794 | 0.001 |
| RDW-SD Vs RDW-CV | 0.794 | 0.001 |
| PLT Vs PCT | 0.965 | 0.001 |
| MPV Vs MCH | 0.639 | 0.001 |
| CRP Vs PLR | - 0.548 | 0.005 |
| CRP Vs MCHC | - 0.589 | 0.002 |
| PLR Vs MID# | - 0.547 | 0.005 |

Statistically significant at p<0.05.

Note\*: KEYS: WBC: Total white blood cells Count; RBC: Red blood cell count; MCV: Mean corpuscular volume; MCH: Mean corpuscular haemoglobin; MCHC: Mean corpuscular haemoglobin concentration; PLT: Platelet count; PCT: Plateletcrit; HGB: Haemoglobin; HCT: Haematocrit; MPV: Mean Platelet Volume; LYM%: Lymphocyte count percentage; LYM#: Lymphocyte count absolute; GRAN%: Granulocyte count percentage; GRAN#: Granulocyte count absolute; MID%: Middle cell count percentage; MID#: Middle cell count absolute; RDW-CV: Red cell Distribution Width-Coefficient of Variation; RDW-SD: Red cell Distribution Width-Standard Deviation; NLR: Neutrophil-Lymphocyte ratio; PLR: Platelet-Lymphocyte ratio.

Table 4: Levels of Associations Between the Parameters Studied in the Control group

|  |  |  |
| --- | --- | --- |
| Variables  | r-value | p-value |
| WBC Vs LYM#  | 0.929 | 0.001 |
| WBC Vs MID# | 0.678 | 0.001 |
| WBC Vs PLR | - 0.684  | 0.001 |
| LYM% Vs GRAN% | - 0.825 | 0.001 |
| LYM% Vs GRAN# | - 0.733 | 0.001 |
| LYM% Vs NLR | - 0.876 | 0.001 |
| MID% Vs LYM%  | - 0.630 | 0.001 |
| MID% Vs MID# | - 0.750 | 0.001 |
| MID% Vs WBC | 0.678 | 0.001 |
| MID# Vs PLR | -0.537 | 0.006 |
| GRAN% Vs GRAN# | 0.788 | 0.001 |
| GRAN% Vs NLR | 0.971 | 0.001 |
| GRAN# VS LYM% | -0.733 | 0.001 |
| GRAN# Vs NLR | 0.794 | 0.001 |
| HGB Vs MCV | 0.963 | 0.001 |
| HCT Vs HGB | 0.963 | 0.001 |
| MCV Vs MCHC | 0.603 | 0.001 |
| RDW-SD Vs RDW-CV | 0.599 | 0.002 |
| RDW-CV Vs Age | 0.602 | 0.001 |
| PLT Vs PCT | 0.988 | 0.001 |
| PLT Vs LYM# | - 0.630 | 0.001 |

\*Statistically significant at p<0.05

Note\*: KEYS: WBC: Total white blood cells Count; RBC: Red blood cell count; MCV: Mean corpuscular volume; MCH: Mean corpuscular haemoglobin; MCHC: Mean corpuscular haemoglobin concentration; PLT: Platelet count; PCT: Plateletcrit; HGB: Haemoglobin; HCT: Haematocrit; MPV: Mean Platelet Volume; LYM%: Lymphocyte count percentage; LYM#: Lymphocyte count absolute; GRAN%: Granulocyte count percentage; GRAN#: Granulocyte count absolute; MID%: Middle cell count percentage; MID#: Middle cell count absolute; RDW-CV: Red cell Distribution Width-Coefficient of Variation; RDW-SD: Red cell Distribution Width-Standard Deviation; NLR: Neutrophil-Lymphocyte ratio; PLR: Platelet-Lymphocyte ratio.