**Haematological Parameters Estimation in Cigarette and Non-Cigarette Smokers in Ibeju/Lekki Local Government Area of Lagos State**

# Abstract

Cigarette smoking is one of the major leading causes of death throughout theworld,it has both acute and chronic effects on haematological parameters. The aim of this study was to evaluate the haematological parameters in cigarette and non-cigarette smokers. About 150 subjects were recruited for this study, of which 100 were cigarette smokers while 50 were non-cigarette smokers. Questionnaire was administered prior to blood collection from each ofcigarette and non-cigarette smokers. The cigarette smokers were regularly consuming at least 5 sticks of cigarette daily for at least 5 years. Complete blood cell count was analyzed with the use of an automatic haematological analyzer. Hemoglobin (Hb) (p<0.001), hematocrit (HCT) (p<0.001) and mean corpuscular volume (MCV) (p=0.002) values were statistically significantly higher in cigarette smokers (test group) when compared with that ofnon-cigarette smokers(control group). Vice versa, RDW was lower incigarette smokers when compared withnon-cigarette smokers (p=0.001). Leukocyte (p<0.001), neutrophil (p=0.001) and lymphocyte counts (p=0.04) were statistically higher amongcigarette smokers when compared withnon-cigarettes smokers. There was no statistical significant difference in parameters of platelet indices between the 2groups. From this present study, it was observed that continuous cigarette smoking increases erythrocyte, hemoglobin, hematocrit, leukocyte count and mean corpuscular volume and these alterations might be associated with a greater risk for developing polycythemia vera, atherosclerosis and cardiovascular diseases.

**Keywords:** cigarette smokers, Hemoglobin, Haematocrit, Leukocyte.

## Introduction

Smoking cigarettes involves burning a chemical, then inhaling the ensuing smoke to taste it and absorb it into the bloodstream (Tobacco fact sheet, 2014). The substance used most frequently is made up of dried tobacco plant leaves that have been rolled into a small rectangle of rolling paper to form a tiny, spherical cylinder known as a cigarette. Because the combustion of the dried plant leaves vaporizes and transports active compounds into the lungs, where they are quickly absorbed into the bloodstream and reach physiological tissue.Smoking cigarettes is largely employed as a mode of administration for recreational drugs (Inal et al. 2014). The pharmacologically active alkaloid nicotine is one of the substances present in the mixture of aerosol particles and gases found in smoking cigarettes; vaporization transforms heated aerosol and gas into a form that permits inhalation and deep penetration into the lungs, where the active substances are absorbed into the bloodstream (Tobacco fact sheet, 2014).

The ill effects ofcigarette smoking comes from the many toxic chemicals in the natural tobacco leaf and those formed in smoke from burning tobacco (Patra et al. 2020). People are addicted to cigarettes smoking because the [nicotine](https://en.wikipedia.org/wiki/Nicotine) (Djordjevic and Doran, 2009), the primary psychoactive chemical in cigarettes, is highly addictive.The nicotine decreases vascular function, causes clot formation in the coronary arteries, and worsens endothelial dysfunction (Hajek et al. 2014). Cigarettes, like narcotics, have been described as "strategically addictive", with the addictive properties being a core component of the business strategy about half of cigarette smokers die from a cigarettes smoking -related cause (Djordjevic and Doran, 2009). Cigarettesmoking harms nearly every organ of the body. Cigarettes smoking leads most commonly to diseases affecting the heart (Neal, 2010) liver, and lungs, being a major risk factor for heart attack, [strokes](https://en.wikipedia.org/wiki/Stroke), [chronicobstructive pulmonary disease](https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease) (COPD) (including [emphysema](https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease) and [chronic bronchitis](https://en.wikipedia.org/wiki/Chronic_obstructive_pulmonary_disease)), andcancer(particularly [lung cancer](https://en.wikipedia.org/wiki/Lung_cancer), [cancers of the larynx and mouth](https://en.wikipedia.org/wiki/Laryngeal_cancer) and [pancreatic](https://en.wikipedia.org/wiki/Pancreatic_cancer)). Hypertension and peripheral vascular disease are also caused by it (Neal, 2010). Although the precise cause of these problems in cigarette smokers are unknown, it is assumed that they are brought on by changes in the antithrombotic and fibrinolysis systems, anomalies in blood rheology, infection and inflammation, and oxidative stress (Shah et al. 2010). Smoking cigarette earlier in life results in increased tar levels, which raises the chance of developing certain diseases. According to the World Health Organization, tobacco use results in 8 million annual fatalities as of 2019 and 100 million over the course of the 20th century (WHO, 2020).

No matter the substance, inhaling smoke into the lungs is bad for one's health (Asif et al. 2013). Burning plant material, such as tobacco or cannabis, results in incomplete combustion, which creates carbon monoxide, which when breathed into the lungs reduces the blood's capacity to carry oxygen (Asif et al. 2013). Other harmful substances included in tobacco provide substantial health risks to long-term smokers from a variety of reasons, including stenosis, lung cancer, heart attacks, strokes, impotence, and low birth weight in children born to mothers who smoke cigarette (Catherine et al. 2016).

More than 4000 chemicals found in tobacco smoke have some sort of negative impact on human health, although free radicals, nicotine, and carbon monoxide are thought to be the ones mainly responsible for pharmacological effects (Wigand, 2006). The nicotine reduces vascular activity, causes clot formation in the coronary arteries, and worsens endothelial dysfunction (Djordjevic and Doran, 2009). Hypoxia may result from an increase in carboxy-haemoglobin levels, which are also to blame for sub-endothelial oedema due to changes in vascular permeability and lipid buildup (Gossett et al., 2009). In addition to having a clear link to physiological processes like the production of prostaglandins and thromboxanes, free radicals and peroxides from tobacco smoke play a role in the pathogenesis of a number of illnesses like atherosclerosis, cancer, and inflammatory processes (Gossett et al. 2009).

Carbon monoxide has been determined to mostly come from cigarette smoking (Djordjevic and Doran, 2009). Some scientists hypothesized that the rise in hemoglobin levels in smokers' blood could be a form of compensation. Smoking cigarette offers a convenient source of carboxy hemoglobin (Inal et al. 2014). There are detectable quantities of carboxyhemoglobin in non-smokers due to endogenous factors and environmental exposure. Hemoglobin and carbon monoxide combine chemically to form carboxyheamoglobin (Djordjevic and Doran, 2009). In heavy smokers, polycythemia may be a symptom of carbon monoxide overdose (Inal et al. 2014). A state in which a large amount of oxygen is delivered to the tissue to reduce erythropoetin output.

## It has been suggested over the past ten years that smoking has an adverse effect on blood properties, which causes death (Catherine et al. 2016).Studies have revealed that smoking affects white blood cell count (Torres et al. 2009). Numerous findings have revealed that smokers' white blood cell counts are higher than non-smokers (Wannamethee et al.2005). Despite the fact that smoking and red blood cell levels were linked in several earlier research (Tarazi et al. 2008). Some researchers hypothesized that smokers' blood hemoglobin levels may have increased as a form of compensation (Tarazi et al. 2008). Some, however, believed that smoking did not boost hemoglobin levels in all smokers and that this had to do with a person's ability to tolerate various diseases. Additionally, the detrimental effects of smoking on human blood features may have changed due to episodic smoking duration and the individual's age. In light of these reports, a comparison of the haematological parameters of smokers and non-smokers was conducted in the Ibeju/Lekki local government area of Lagos State.

## Materials and Methods

**Study Area/Population**

This study was carried out at Ibeju/lekki local government area of Lagos State, among subjects that only smoke cigarette for at least 5 years and subjects who have never been involved in cigarette smoking or any other form of tobacco productsbefore. The study population consisted of 100cigaretteand 50non-cigarette smokers.

## Research Design

This is a cross-sectional descriptive study of estimation of haematological parameters in cigarette and non-cigarette smokers in Ibeju/Lekki Local Government Area of Lagos State.

**Ethical Consideration**

The participation of subjects in this study was voluntary, and the principle of patient confidentiality was strictly adhered to. Each participant was duly counseled and a prepared consent form was signed by each of the subjects.

The protocol for this study was sought and approved by the ethics and research committee of Lead City University, Ibadan, Oyo State, with the approval number LCU-REC/22/181 dated 31st January, 2022.

**Inclusion criteria for cigarette and non-cigarette smokers**

Subjects that fulfilled the following criteria were included in this study:-

1. Subject for this study included both males and females between the ages of 22years to 65years and above, who have been smoking cigarette alone for at least 5 years.
2. Apparently healthy age matched males and females who are non-tobacco smokers and users, were recruited as non-cigarette smokers.
3. Those that consented to participate in the study.

**Exclusion Criteria for Cigarette and non-cigarette smokers**

Subjects with the following criteria were exempted from this study:-

1. Subjects who have not been smoking cigarettes for a period up to five (5) years at the time of sample collection.
2. Subjects whose ages are below 22 years
3. Subjects who had smoked cigarette or any other type of tobacco products before were exempted to be recruited as control for this study.
4. Subjects that refused participation

**Sample Size Determination**

Sample size for this study was determined using

n = Z2P(1-P) (Naing  et al. 2006)

d2

n= Required sample size

Z= Confidence level at 95% (standard value of 1.96)

P= Estimated prevalence (11%)

d= Accepted error

n= Z2 x P(1-P)

d2

Z= 95%= 1.96, P= 11%, d= 0.05

n= 1.962 x 0.10(1-0.10)

0.052

= 3.84x0.09

0.0025

= 138

Factoring in non response and attrition rate (r) of 10% i.e 0.1 then we have 1-r =0.9

Final sample size is thus

=138/0.9

= 153

This would be rounded up to 150 samples

**Questionnaire**

A well-structured questionnaire bothering on bio-data (Age, Gender, Academic Qualification) and socio-demographic characteristics was administered to each participant.

## Sample Collection and processing (adopted from Ochei and Kolhatkar, 2005)

A tourniquet was applied to the upper arm to dampen the blood**,**the ante cubital fossa was cleaned with wet swab.A 5ml syringe was inserted into the visible vein and the blood was extracted to 3.5ml mark**.** The tourniquet was loosened and cotton wool was applied to the punctured site, the syringe was removed slowly.The blood was dispensed into the labeled EDTA bottle and well mixed to avoid clotting**.** The blood was taken to the automated haematologicalanalyser machine and the blood was aspirated and observed for reading.

**Statistical Analysis**

Statistical analysis for Social Sciences (SPSS) version 25 was the statistical package used in analyzing all data obtained. Frequency table, Bar chart and Pearson Chi-Square were used to compare the means of the different analytes at *p*≤ 0.05 statistical significance.

**Result**

**Table 1.0: frequency distribution according to gender**

|  |  |  |
| --- | --- | --- |
| Gender | Cigarette Smokers(%)  n= 100 | Non-cigarette Smokers (%)n= 50 |
| Male | 78 (78%) | 23 (46%) |
| Female | 22 (22%) | 27 (54%) |

About 150 subjects were recruited for this study, out of which 100 were cigarette smokers and 50 non-cigarette smokers. Out of the 100 cigarette smokers, 78(78%) were males while 22(22%) were females. Out of the 50 non-cigarette smokers, 23(46%) were males while 27(54%) were females (Table 1.0).

**Table 2.0: Distribution of cigarette and non-cigarette Smokers According to their Age Groups.**

| **Age**  **(Years old)** | **Cigarette Smokers (%) n=100** | **Non-Cigarette Smokers (%) n=50** |
| --- | --- | --- |
| 22-31 | 37(37) | 18(36) |
| 32-41 | 26(26) | 14(28) |
| 42-51 | 15(15) | 08(16) |
| 52-61 | 13(13) | 06(12) |
| 62 and above | 09(9) | 04(8) |

Cigarette and non-cigarette smokers within the age group 22-31 years recorded a prevalence of 37% and 36% respectively; followed by 32-41 years age group recorded 26% and 28% respectively; 42-51 years age group recorded 15% and 16%; 52-61 years age group revealed13% and 12%; while subjects that are 62 years and above age group recorded 9% and 8% respectively (Table 2.0).

Table 3.0: Social demographical characteristics of cigarettes smokers

|  |  |
| --- | --- |
| Parameters | Frequency  n=100 (%) |
| Duration of cigarette smoking(yrs) |  |
| 5-10 | 56(56) |
| 11-16 | 32(32) |
| 17years and above | 12(12) |
| No of Cigarettes smoked per day |  |
| ≤ 5 | 54 (54) |
| 6-10 sticks | 29 (29) |
| 11-15 sticks | 11 (11) |
| 16-20 sticks | 6(6) |
| Brands of cigarette |  |
| Benson & Hedges | 78 (78) |
| Philip Morris | 3 (3) |
| Saint Moritz | 17 (17) |
| White London | 2 (2) |

Subjects that have been smoking cigarette for a period of 5-10 years are 56(56%) being the highest, followed by 11-16 are 32(32%); and those that have been smoking cigarette for a period of 17 years and above are 12(12%) being the lowest among the subjects (Table 3.0).

Among the cigarette smokers recruited for this study, 54(54%) smoked **≤** 5 sticks of cigarettes daily; followed by 29(29%) smoked 6-10 sticks of cigarette daily; 11(11%) smoked 11-15 sticks of cigarette daily; and 6(6%) smoked 16-20 sticks of cigarette daily (Table 3.0).

Among the subjects recruited for this study, 78(78%) usually smoke Benson & Hedges; 3(3) usually smoke Philip Morris; 17(17%) usually smoke Saint Moritz; and 2(2%) usually smoke White London being the lowest(Table 3.0).

Table 4.0: Baseline characteristics and comparison of hematological parameters between cigarette and non-cigarette smokers

|  |  |  |  |
| --- | --- | --- | --- |
| **Parameters** | **Cigarettes smokers n=100** | **Non-cigarettes smokers n=50** | ***p*-value** |
| **Hb(gr/dL)** | 14.9±1.5 | 13.5±1.7 | <0.001\* |
| **HCT(%)** | 43.2±3.7 | 39.7±4.5 | <0.001\* |
| **MCV(fL)** | 87±4 | 83±7 | 0.002\* |
| **RDW(%)** | 15.4±0.8 | 14.3±1.6 | 0.001\* |
| **Leukocyte(103/mm3)** | 8.0±2.1 | 6.7±1.7 | <0.001\* |
| **Neutrophils(103/mm3)** | 5.0±1.8 | 4.1±1.6 | 0.001\* |
| **Lymphocytes(103/mm3)** | 2.2±0.6 | 2±0.5 | 0.04\* |
| **Thrombocyte(103/mm3)** | 253±59 | 245±53 | 0.42 |
| **PDW(fL)** | 16.9±0.5 | 16.9±0.5 | 0.46 |
| **MPV(fL)** | 8.6±0.9 | 8.9±0.8 | 0.06 |
| **PCT(%)** | 216±51 | 219±57 | 0.71 |

HCT: Hematocrit, MCV: Mean corpuscular volume, RDW: Red cell distribution width, PDW: Platelet distribution width, MPV: Mean platelet volume, PCT: Platelet count, Hb: Hemoglobin

Significant at 0.05 level (*p*≤0.05)

Hemoglobin (Hb) (p<0.001), hematocrit (HCT) (p<0.001), mean corpuscular volume (MCV) (p=0.002), RDW (*p*=0.001), leukocytes (p<0.001), neutrophils (*p*=0.001) and lymphocytes (*p*=0.04) values were statistically significantly higher incigarette smokers group than in non-cigarette smokers group. Vice versa, MPV is lower in the cigarette smokers group than in non-cigarette smokers group (p=0.001). There was no statistical significant difference in parameters of platelet indices between the two groups (Table 4.0).

Table 5.0:1Baseline characteristics and comparison of hematological parameters between male cigarette smokers and female cigarette smokers

|  |  |  |  |
| --- | --- | --- | --- |
| **Haematological parameters** | **Male-cigarette smokers (n=78)** | **Female-cigarette smokers (n=22)** | ***p*-value** |
| WBC(103/mm3) | 8.1± 0.40 | 7.67 ± 0.287 | 0.040\* |
| Lymphocyte(103/mm3) | 2.2 ±1.3 | 2.23±1.5 | 0.159 |
| Neutrophil(103/mm3) | 5.0±1.8 | 5.12±1.8 | 0.561 |
| RBC(x1012/L) | 5.12 ± 0.07 | 4.66 ± 0.06 | <0.001\* |
| HB(g/dL) | 16.78 ± 2.03 | 12.65 ± 2.44 | <0.001\* |
| HCT(%) | 43.84 ± 1.97 | 39.76 ± 0.76 | 0.047\* |
| MCV(fL) | 88.92 ± 1.34 | 85.38 ± 1.17 | 0.050\* |
| PCT(%) | 215±1.4 | 213±1.43 | 0.0534 |

Significant at 0.05 level (*p*≤0.05)

The analysis of gender differences in the group of Cigarette smokers showed statistical significant difference in the number of leukocytes. The values of leukocytes were statistically higher in male subjects compared to female respondents. The statistical significant difference between other parameters of white blood cells was not found. Male population of Cigarette smokers had significant higher values for the number of red blood cells and hemoglobin, hematocrit, mean corpuscular volume (MCV) and PCT values (Table 5.0).

Table 6.0: comparisons of hematological parameters among cigarette smokers based on the number of years of cigarette smoking

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Duration of cigarette smoking (yrs) | | | | |
| Parameters | 5-10 years  n= 56 | 11-16 years  n=32 | ≥17 years  n=12 | *p*-value |
| Hb(gr/dL) | 14.1±1.3 | 14.7±1.6 | 15.9±1.1 | 0.01\* |
| HCT(%) | 42.6±3.6 | 43.5±3.7 | 45.8±2.7 | 0.01\* |
| MCV(fL) | 86±4 | 88±6 | 89±3 | 0.18 |
| RDW(%) | 13.4±0.7 | 13.7±1 | 13.9±0.6 | 0.5 |
| Leukocyte(103/mm3) | 8.0±2.3 | 6.9±2.1 | 8.9±1.3 | 0.05\* |
| Neutrophils(103/mm3) | 5.1±2.0 | 5.2±1.5 | 5.5±1.5 | 0.21 |
| Lymphocytes(103/mm3) | 2.1±0.5 | 2.3±0.8 | 2.5±0.4 | 0.04\* |
| Thrombocyte(103/mm3) | 276±62 | 217±49 | 230±36 | 0.01\* |
| PDW(fL) | 16.8±0.5 | 17±0.5 | 17.8±0.5 | 0.26 |
| MPV(fL) | 8.5±0.9 | 8.7±0.7 | 8.9±1 | 0.63 |
| PCT(%) | 234±57 | 187±37 | 199±26 | 0.02\* |

Hb: Hemoglobin, HCT: Hematocrit, MCV: Mean corpuscular volume, RDW: Red cell distribution width, PDW: Platelet distribution width, MPV: Mean platelet volume, PCT: Platelet count,

Significant at 0.05 level (*p*≤0.05)

The hematological parameters compared between the number of years subjects have been engaged in cigarette smoking, revealed that the level of Hb (p=0.01), HCT (p=0.01), lymphocyte (p=0.04), MCV (p=0.18) and thrombocyte counts (p=0.01) were significantly increased as number of years of engaging in cigarettes smokingrose. However, the PCT levels (p=0.18), leukocyte (p=0.05) and neutrophil (p=0.21) counts did not change significantly with the rise in the number of years engaged in cigarette smoking(Table 6).

**Discussion**

Cigarette smoke have both acute and chronic effects on haematological markers. However, the severity of these effects on a smoker will vary depending on how many sticks of cigarette they smoke each day and how long they have been smoking consistently.

In this study, about 150 subjects were recruited, of which 100 werecigarette smokers and 50 non-cigarette smokers. There were 49 (32.7%) females and 101 (67.3%) males recruited for this study. This is in agreement with WHO, 2011 that reported the number of males involved in cigarette smoking is twice that of females in south-west Nigeria. The ages of subjects recruited for this study ranges from 22years to 62years and above.Cigarette and non-cigarette smokers within the age group 22-31years recorded a prevalence of 37% and 36%; followed by 32-41years age group recorded 26% and 28%; 42-51 years age group recorded 15% and 16%; 52-61years age group revealed 13% and 12%; while subjects that are 62years and above age group recorded 9% and 8% respectively. This revealed that a higher percentage of youths are keenly involved in cigarette smoking. This is in tandem with reports by Takure et al. 2015 and Ajileye et al. 2021

Number of years of involving incigarette smoking among the test group varies with 56(56%) smoking for a period of 5-10 years being the most prevalence among them; followed by 32 (32%) for 11-16 years; and 12(12%) for 17 years and above. Among the cigarette smokers recruited for this study, 54(54%) smoked ≤ 5 sticks of cigarettes daily; followed by 29(29%) smoked 6-10 sticks of cigarette daily; 11(11%) smoked 11-15 sticks of cigarette daily; and 6(6%) smoked 16-20 sticks of cigarette daily.

This study revealed Hemoglobin (Hb) (*p*<0.001), Hematocrit (HCT) (*p*<0.001), Mean Corpuscular Volume (MCV) (p=0.002), Red Cell Distribution Width (RDW) (*p*=0.001), Leukocyte (*p*<0.001),Neutrophils (*p*=0.001), Lymphocyte (*p*=0.04), Thrombocyte (*p*=0.42) and Mean Platelet Volume (MPV)values were significantly high,while Platelet Distribution Width (PDW) (*p*=0.46), Mean Platelet Volume (MPV) (*p*=0.05) and Platelet count (PCT) (*p*=0.71) were significantly low in cigarette smokers group than in non-cigarette smokers group. This is in agreement with Shatha, 2017who observed significant differences in hematological parameters of smokers and non-smokers where the WBC, RBC, Hb, HCT and MCV were significantly high, whereas MCHC was significantly low in smokers as compared to non-smokers.

This study showed remarkable increase in hemoglobin concentration in both male and female cigarette smokers when compared withnon-cigarettesmokers. This is in tandem with a similar study conducted by Shatha, 2017 who studied the effect of cigarette smoking on some blood parameters, blood pressure and renal function test.Also, in the study carried out by Maja et al. 2017 on the effects of cigarette smoking on haematological parameters in healthy population, revealed that cigarette smokers had a significant higher values of hemoglobin concentration than the non-cigarette smokers regardless of the sex. The significant increase in Haemoglobin in smoker group is also correlated with previous study carried out by Jena et al. 2013 on the effects of chronic cigarette smoking on haematological Parameters. According to some scientists, exposure to carbon monoxide is thought to be a mediator of the increase in hemoglobin concentration, and an increase in hemoglobin levels in smokers' blood has been proposed as a potential compensatory mechanism. Inactive carboxy-hemoglobin is created when carbon monoxide attaches to hemoglobin (Hb), which has no ability to deliver oxygen. Additionally, carboxy-hemoglobin causes a shift on the left side of the hemoglobin dissociation curve, which reduces hemoglobin's capacity to carry oxygen to tissues. Smokers maintain a higher hemoglobin level than non-smokers do in order to make up for the reduced ability to supply oxygen to the blood (Aseel, 2008).

Hematocrit (HCT) (p<0.001) values were statistically significantly higher in cigarette smokers than in non-cigarette smokers group.However, compared to female smokers, male smokers had significantly higher hematocrit values. In a study conducted by Lakshmi et al. 2014 smokers had significantly higher hemoglobin and hematocrit levels and as smoking intensity increases, smokers’ RBC counts also will increase dramatically. The increased production of carboxy hemoglobin causes tissue hypoxia, which increases erythropoietin secretion, enhancing erythropoiesis, which explains why smokers have higher erythrocyte counts and hematocrit values. Additionally, carbon monoxide from tobacco smoke increases capillary permeability, which reduces plasma volume and ultimately resembles polycythemia, which is marked by a greater proportion of erythrocytes in blood volume and is also demonstrated by higher hematocrit readings (Nadia et al. 2015; verma et al. 2015). As a result of reduced coronary blood flow and enhanced platelet adherence to the aortic subendothelium, an elevated hematocrit may hasten the development of atherosclerosis and thromboembolic illness (Elfrieke et al. 2002).

According to this study, MCV values were statistically significant among the cigarette smokers when compared with non cigarette smokers. This is in agreement with Mufarah et al. 2022 who observed a higher MCV values among healthy medical students. Anemia is indicated by red blood cells that are less or larger than normal. MCV measures red blood cell size, and the presence of smaller or larger-than-normal red blood cells suggests anemia. Elevated levels of MCV suggest that people may have megaloblastic, hemolytic, pernicious, or macrocytic anemia, which are typically brought on by iron and folic acid deficiency (Muhammad et al. 2013).

The inhaled carbon monoxide gas (CO), one of the inhaled components of cigarette smoke, may be to blame for the rise in hemoglobin, hematocrit, and MCV. More than 600 times the amount of CO that industrial plants consider to be safe is contained in cigarette smoke. The amount of CO in a smoker's blood is typically 4 to 15 times higher than in a nonsmoker's. The amount of CO that can reversibly couple with oxygen-carrying sites on hemoglobin is between 210 and 240 times larger than that of oxygen. This decrease in oxygen-carrying capacity of the blood is made up for by an increase in hemoglobin and hematocrit (Yousif, 2015).

This study revealedstatistical significant higher Leukocyte (p<0.001), neutrophil (p=0.001) and lymphocyte counts (p=0.04)among cigarette smokers of both sexes in relation to non-cigarette smokers. In addition, the values of leukocyte count were statistically significantly higher in male cigarette smokers. This is in tandem to a study conducted by Aula and Qadir, 2023 whoobserved significant increase in leukocytes, neutrophils, eosinophils, basophils, lymphocytes and monocytes in Cigarette smokers in relation to the control group of non-cigarette smokers.The nicotine-induced release of catecholamine and steroid hormones from the centre of the adrenal gland can lead to an increase in the number of leukocytes. It is understood that a rise in the concentration of several endogenic hormones, such as cortisol and adrenaline, causes an increase in the number of leukocytes (Deutsh et al. 2007). The condition known as leukocytosis is thought to be brought on by long-term cigarette smoking. It raises the number of white blood cells (WBCs) in the blood and increases the level of polymorphonuclear leukocytes (PMNLs) and band cells in circulation. RDW was lower in the Cigarette smokers group than in non-cigarette smokers group (p=0.001).

There was no statistically significant difference in parameters of platelet indices between the groups.In this study, PDW, MPV and PCT were lower among the cigarette smokers when compared with the non-cigarette smokers. This result is consistent with the previous results by Shatha, 2017 who reported there was no statistically significant difference in platelet indices among cigarette smokers when compared with non cigarette smokers. Farhang and Fikry, 2013 also observed no statistically significant difference in platelet indices between the control and cigarette smokers in both groups. According to Blann et al. (1998), smoking two sticks of cigarette a day by chronic smokers of both sexes do not affect the platelet count. Some of these findings showed that smokers had higher platelet turnover and lower platelet survival; greater platelet destruction, while, was insufficient to lower the amount of circulating platelets (Farhang and Fikry, 2013).

This study revealed significant increase in RBCs count, TLC, PCT, HCT, HB and MCV in male cigarette smokers in relation to female cigarette smokers. Female cigarette smokers showed significant increase in the levels of neutrophil and lymphocyte. This is in rustle with the study conducted by Mufarahet al. 2022 who investigated on the consequence of smoking on haematological parameters in apparently healthy medical students, who observed Red blood cells, white blood cells, hemoglobin, hematocrit, and mean corpuscular hemoglobin were all significantly higher (p=0,001) in male smokers when compared with female smokers after smoking cigarettes. The number of years of actively involved in cigarette smoking and the number of sticks of cigarette smoke per day determines the effects of the smoke on haematological parameters. This study observed that the various haematological parameters counts increased as the number of years of cigarette smoking also increased. This is in agreement with a study conducted by Anandha et al. 2014 who carried out a study on the Effect of Intensity of Cigarette Smoking on Haematological and Lipid Parameters.

**Conclusion**

In this study, we can conclude that continuous cigarette smoking increased erythrocyte, hemoglobin concentration, hematocrit, leukocyte count and mean corpuscular volume and these alterations might be associated with a greater risk for developing atherosclerosis and other associated diseases

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