

**EFFECTS OF TOBACCO CIGARETTE SMOKING ON SOME
HEMATOLOGICAL PARAMETERS OF MALE CIGARETTE
SMOKERS IN SOUTHERN NIGERIA.**

Abstract

In Nigeria smoking is a common habit in both rural and urban areas. Cigarette smoking is associated with alterations in inflammatory markers among smokers and it affects various organs and tissues in the body including blood. Cigarette smoking is associated with development and progression of numerous chronic diseases worldwide. It is a known fact that smoking is one of the most important factors contributing to the evolution of atherosclerosis and chronic obstructive pulmonary disease. This study investigates the effects of smoking on haematological parameters among tobacco cigarette smokers in Southern Nigeria. A total of 150 smokers and 110 controls (non-smokers) within the age range of 20-50 years residing in Calabar metropolis of Cross River State, South-South, Nigeria were recruited. The smokers were apparently healthy males who are regular cigarette smokers who have at least smoked 100 cigarette sticks in their life time. Full blood count was done using sysmex, kx-210 automated hematology analyser (sysmex America, Illinois). The erythrocyte sedimentation rate was determined by westergren's method. The relative plasma viscosity was performed using the method of Bach et al. 1981. The tobacco cigarette smokers had significantly ($p<0.05$) higher hemoglobin, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, total white blood cell count, mixed cells, Erythrocyte sedimentation rate, relative plasma viscosity, mean platelet volume and platelet distribution width and significantly ($p<0.05$) lower neutrophil count as compared to controls. This study showed that smoking has negative effect on the haematopoietic system. The study has also shown that hematological parameters were altered in smokers and thus should be considered during diagnosis, interpretation of result and treatment of patients.

Key words: Tobacco, Cigarette, Smoking, Hematological

INTRODUCTION

Tobacco cigarette smoking is one of the leading cause of death and essential public health challenge in the world over². It is a major cause of death worldwide, resulting in millions of death annually more than malaria, tuberculosis and HIV/Aids². According to world health organization (WHO) report, there are about 2.4 billion people worldwide that have consumed tobacco in the form of estimated smoking, chewing, snuffing or dipping. WHO also estimated that tobacco related deaths will amount to 8.3 million in 2030 and one billion deaths during the 21st century³. There are more than 4000 chemicals present in cigarette smoke among

37 which includes nicotine, carbon monoxide, free radicals and other gaseous products which
38 are dangerous⁴. Cigarette smoking is **s** powerful risk factor for arteriosclerosis.

39 A variety of studies have shown an association of smoking with coronary heart disease
40 (CHD)⁵, peripheral artery disease,⁶ aortic aneurism,⁷ and stroke⁸.

41 More so, cigarette smoking accelerates pathogenesis in different type of cancers such as lung,
42 pancrease, breast, liver and kidney⁹. Similarly it changes the pH in stomach that results in
43 peptic ulcers and gastric diseases². Tobacco smoking rates have decreased in industrialized
44 countries since 1975, but there has been a corresponding 50% increases in **some** low-income
45 countries **in Asia**¹⁰. In respect to this, it becomes pertinent to adjust for these factors in
46 population based surveys when interpreting haematological values. The present study
47 investigates the effects of smoking on haematological parameters among tobacco cigarette
48 smokers.

49 **MATERIALS AND METHODS**

50 A total of 150 smokers and 110 controls (non-smokers) within the age range of 20-50
51 years residing in Calabar metropolis of Cross River State, South-South, Nigeria from October
52 2015 to November 2015 were selected. The smokers were apparently healthy males who are
53 regular cigarette smokers who have at least smoked 100 cigarette sticks in their life time. Ex-
54 smokers and those who also smoke marijuana or on drugs were excluded. The control group
55 were apparently healthy males who are not currently undergoing any clinical investigation.
56 The study was approved by Cross River State ministry of Health Research Ethics Committee,
57 Calabar, Nigeria.

58 Informed consent was obtained from the subjects before the commencement of the study.
59 Information of the subjects were obtained via a predesigned questioner. After taking

antiseptic precautions, 9ml venous blood was collected into EDTA vacutainers (Akureit, Eatern Medkit Limited), and 0.5ml 3.8% sodium citrate container. The EDTA blood samples were processed by using sysmex, kx-210 automated hematology analyser (sysmeic America, Illinois). The erythrocyte sedimentation rate was determined by westergren's method of ESR determination.¹¹ The relative plasma viscosity was performed using the disposable syringe method as described by Bach et al.¹². The barrel of a 20ml syringe with a hypodermic needle (21.6 x 0.40) was filled by drawing up undiluted plasma through the needle starting with the plunger of the 1ml mark taken in this fashion, the serum level was just over the 2ml mark at the moment the plunger is withdrawn completely from the barrel of the syringe. With the syringe hand clamped in a vertical position, the run-through time was started when the bottom of the meniscus passes the 2ml mark of the barrel and the timing was stopped when the meniscus reaches the zero mark of the barrel. The same procedure was performed for distilled water. The relative plasma viscosity (RPV) was derived by dividing the run-through time for the plasma against that of distilled water. **The alpha level of the study was set at 0.05.**

Statistical analysis: Mean and Standard deviation were used to access mean values while two sample t test was used to ascertain significant differences among the different hematological parameters. All statistical analysis were performed using SPSS version 20.0 (SPSS Inc., Chicago, USA).

Results

Table 1 shows the demographic data of the smokers and control subjects consisting of the age, height, weight, body mass index, systolic and diastolic blood pressures. The hematological parameters of both smokers and control subjects are given in table 2, the cigarette smokers had significantly ($p < 0.05$) higher mean values of hemoglobin, mean

84 corpuscular hemoglobin, mean corpuscular hemoglobin concentration, total white blood cell
85 count, mixed cells, erythrocyte sedimentation rate, relative plasma viscosity, mean platelet
86 volume and platelet distribution width and significantly ($p<0.05$) lower mean neutrophil
87 count as compared to controls.

88 Discussion

89 This study investigated the effect of smoking on the hematopoietic system via complete
90 blood count parameters, erythrocyte sedimentation rate and relative plasma viscosity. A
91 higher hemoglobin concentration was observed in the smokers than the control subjects. This
92 observation is in consonance with previous finding.¹³ This elevated Hb value is likely a
93 compensatory reaction to exposure to carbon monoxide. The inhaled carbon monoxide (CO)
94 has a high affinity for hemoglobin relative to oxygen. Hence, CO displaces oxygen from
95 hemoglobin in red blood cells to produce carboxyhemoglobin which reduce the release of
96 oxygen to tissues creating a chronic hypoxia¹⁴. An elevated total white cell count was
97 observed in this study. This observation is consistent with previous studies,^{15,16}. The exact
98 mechanism responsible for this smoking associated leucocytosis is unclear. However, it has
99 been hypothesized to be due to nicotine induced release of catecholamines as increase in
100 certain endogenous hormone levels such as epinephrine and cortisol have been reported to
101 increase leucocyte count¹⁷. More so, the resultant inflammatory response induced by
102 particulates of cigarette smoke may have been another mechanism for smoking induced
103 leucocytosis^{18, 19}. A significant elevation and reduction were observed in the mixed cell and
104 neutrophil counts, respectively. The result of the neutrophil count is consistent with report by
105 Tulger *et al.*, (2016)²⁰ with a contrasting result in the mixed cell counts. The mechanism
106 behind the differences in the mixed cell count and neutrophil count is unclear. An elevated
107 ESR was observed in the smokers when compared with the control. This finding is in
108 agreement with earlier report⁹. Cigarette smoke induces endothelial damage by producing

free radicals such as nitric oxide and hydrogen peroxide. This oxidative stress promotes systemic acute phase reaction, hence, increasing inflammatory cytokines and eventual rise in ESR value²¹. The association between inflammation and cardiovascular heart diseases has gained considerable interest²². Several systemic markers of inflammation including ESR have been found to be predictor of coronary heart disease²³ and heart failure²⁴. The observed elevated relative plasma viscosity in the smokers was consistent with earlier finding²⁵. Plasma viscosity has been indicated as a predictive risk factor for coronary heart disease²⁶. In other study, high plasma viscosity was shown to lead to an increased risk of acute myocardial infarction in patients with unstable angina pectoris²⁷. The observed elevated mean platelet volume is consistent with earlier report by Anandhalakshmi et al (2015)²⁸. However, it is in contrast with previous finding by Butkiewicz et al., (2006)²⁹. Mean platelet volume is an indicator of platelet activation. Increased mean platelet volume has been reported as an independent risk factor for cardiovascular events³⁰.

Conclusion

The findings of this study showed that smoking has negative effect on the hematopoietic system. These events have been reported to lead to vascular mortality and cardiovascular events, hence, death. More so, this study has shown that hematological parameters were altered in smokers and thus should be considered during diagnosis, interpretation of result and treatment of patients.

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262 **Table 1: Demographic parameters of smoking and control subjects**

Parameter	Smokers (N=150)	Control (110)
Age (Years)	35.90±8.20	34.7±7.6
Height (Meters)	1.60±0.04	1.63±.02
Weight (Kg)	57.40±3.20	59.1±4.3
Body Mass Index (BMI) (Kg/m ²)	24.20±1.30	24.1±2.1
Systolic Blood Pressure (mmHg)	121±7.90	106±5.1
Diastolic Blood Pressure (mmHg)	91±6.0	79±3.1

263 Note: Data are represented as Mean±SD

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276 **Table 1: Hematological parameters of smoking and control subjects**

Variables	Control (n=110) mean $\pm$ S.D	Smokers (n=150) means $\pm$ S.D
RBC X 106/110	5.09 $\pm$ 0.06	5.21 $\pm$ 0.07
Hb (g/dL)	14.19 $\pm$ 0.12	15.30 $\pm$ 0.18*
HCT (%)	44.454 $\pm$ 0.41	44.54 $\pm$ 0.60
MCV (fL)	88.54 $\pm$ 1.45	86.06 $\pm$ 1.37
MCH (Pg)	28.16 $\pm$ 0.38	29.53 $\pm$ -0.40*
MCHC (g/dL)	31.84 $\pm$ 0.23	33.91 $\pm$ 0.39*
RDW SD (fL)	43.98 $\pm$ 0.41	44.85 $\pm$ 0.52
RDW CV (%)	14.58 $\pm$ 0.17	14.81 $\pm$ 0.15
WB (x103/ μ)	4.56 $\pm$ 0.14	5.54 $\pm$ 0.17*
LYMPHOCYTE (%)	45.93 $\pm$ 1.52	48.25 $\pm$ 1.50
MIXED CELLS (%)	11.17 $\pm$ 0.58	13.20 $\pm$ 0.76*
NEUTROPHIL (%)	42.80 $\pm$ 1.72	37.72 $\pm$ 1.30*
ESR (mm/hr)	17.0 $\pm$ 4.52	24.0 $\pm$ 6.32*
RPV (MPA.S)	1.74 $\pm$ 0.12	1.84 $\pm$ 0.16
PLATELET X 103/ μ	209.64 $\pm$ 30.71	229.55 $\pm$ 23.43
MPV (FL)	9.50 $\pm$ 0.68	10.0 $\pm$ 0.97*
PDW (FL)	11.93 $\pm$ 2.02	13.20 $\pm$ 2.17*

- 277 • = P<0.05 compared to control; Hb = Hemoglobin
- 278 • HCT = Hematocrit, MCV = Mean corpuscular volume, MCH = Mean corpuslar
- 279 Hemoglobin, MCHC = Mean corpuscular Hemoglobin concentration, RDW = Red
- 280 Cell Distribution width, WBC = White Blood cells, ESR = Erythrocyte sedimentation
- 281 rate, MPR = Mean platelet volume, PDW = Platelet Distribution width