

# Evaluation of Some Haematological Parameters and Some Heavy Metals Among Smokers and Individuals Exposed to Generator Fumes in Abia State

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## Abstract:

Cigarette smoking and exposure to toxic generator fumes deliver a number of heavy metals to the blood. Bioaccumulation of these toxic metals result in an increase in pathological consequences over time. The aim of this study is to assess some haematological parameters and some heavy metals among smokers and individuals exposed to generator fumes in Abia State. The study was a cross-sectional and analytical study of 210 participants including 70 smokers, 70 subjects exposed to toxic generator fumes, 35 non-smokers and 35 non exposed control subjects. A multistage sampling was used to select participants. Blood lead, cadmium levels and haematological parameters were analyzed using Atomic Absorption Spectrometer at 238.3nm wavelength and Mindray Auto Haematology Analyzer respectively. Obtained data was subjected to Statistical Package of Social Science (SPSS) and analyzed using One-way ANOVA. The study revealed that the mean age of smokers and non-smokers were  $34.20 \pm 0.83$  and  $35.11 \pm 1.25$  years respectively, while the mean ages of subjects exposed and non exposed to generator fumes were  $34.50 \pm 0.80$  and  $35.11 \pm 1.25$  years. The study showed the significant mean difference of haematological parameters of smokers versus non-smokers as follows: Lymphocytes % ( $43.21 \pm 13.93b$  versus  $30.95 \pm 14.91a$ ), HCT (PCV)% ( $40.85 \pm 5.55b$  versus  $37.82 \pm 4.18a$ ), Hb g/dL ( $13.52 \pm 1.89b$  versus  $12.28 \pm 1.37a$ ) and MCH pg ( $28.02 \pm 2.11b$  versus  $26.79 \pm 2.77a$ ), were significantly high ( $p < 0.05$ ). WBC  $\times 10^9/L$  ( $5.62 \pm 1.70a$  versus  $8.43 \pm 6.47b$ ), and Neutrophil % ( $48.32 \pm 15.16b$  versus  $60.79 \pm 15.92a$ ) were significantly low in smokers as compared to non-smokers ( $p < 0.05$ ). The changes in haematological parameters of generator fume exposure versus non exposed subjects: Lymphocytes% ( $41.58 \pm 13.18b$  versus  $30.95 \pm 14.91a$ ), MCV fl ( $85.07 \pm 4.80b$  versus  $37.82 \pm 4.18a$ ), were statistically significant; WBC  $\times 10^9$  ( $5.57 \pm 2.48a$  versus  $8.43 \pm 6.47b$ ), and Neutrophil % ( $49.58 \pm 14.22b$  versus  $60.79 \pm 15.92a$ ), were significantly low in subjects exposed to generator fumes as compared to non exposed subjects ( $p < 0.05$ ). The study also revealed the significant mean difference of blood Lead ( $\mu g/dL$ ) level among the controls, smokers and generator fume exposure as follows: Smokers versus Controls ( $7.66 \pm 3.71b$  versus  $5.98 \pm 2.81a$ ) was significantly high in smokers as compared to controls, and generator fume exposure versus controls ( $3.00 \pm 0.89c$  versus  $5.98 \pm 2.81a$ ) was significantly low in generator fume exposure compared to controls. The presence of Cadmium ( $\mu g/dL$ ) among the smokers, individual exposed to generator fumes and controls were not statistically significant. There was a significant positive and

negative correlation of haematological parameters among smokers and individuals exposed to generator fumes. The findings showed that continuous cigarette smoking and exposure to generator fumes have severe adverse effects on haematological parameters such as haemoglobin, haematocrit, WBC count, and platelet count, lymphocytes as well as significant increase in blood lead level among the groups, and these alterations could be associated with a greater risk for developing hypertension, atherosclerosis, chronic obstructive pulmonary disease and/or cardiovascular diseases in future.

**Key words:** haematological parameters; heavy metals; smokers generator fumes; Abia state

## Introduction:

Lead (Pb) and cadmium (Cd) are heavy metals that are harmful to human health. The commonest of these toxic heavy metals include: lead, cadmium, iron, and zinc .. When a harmful metal enters human body, it passes through the bloodstream to the vital organs like the kidney, the liver, the brain and then to the bones [1]. These harmful metals elements affect various organs in the body resulting in kidney failure, gastrointestinal disorders, anemia, nervous system disorders, congenital disorders and dermatological disorders [2]

Lead mainly affect the central nervous system and also cause anemia by interfering with the haemoglobin synthesis. Cadmium is a carcinogenic heavy metal and it mainly affect the kidney and also cause bone disorder [3].

Environmental exposure to lead (Pb) and cadmium (Cd) result from natural and industrial activities like volcanic eruptions and weathering of rocks. This is the reason these heavy metals are found on the earth surfaces like the soil, in the air and water and they can also accumulate in plants and animals . Moreso, Pb and Cd have been useful in several industries. Even though Pb has a special characteristics in industries, there has been a decline in its use due to environmental pollution. Lead is used in painting, plumbing, mining, pipes, manufacturing of batteries and metal recycling. Cd has declined in the United States of America since 2001 in response to environmental effect. Cadmium is useful in non-ferrous alloys, batteries, plastic stabilizers, pigment production, coatings, and plating [4].

The average concentration of blood lead in nonexposed healthy adults was 0.9 µg/dL and the acceptable blood lead reference levels are < 5 µg/dL in adults and < 3.5 µg/dL in children. The value of blood lead becomes critical when the levels are ≥ 70 µg/dL in adults and ≥ 20 µg/dL in children. The mean blood level of cadmium in a healthy nonexposed adults is 0.1–4.0 µg/dL and the reference range of blood cadmium is ≤ 4.0µg/dL. The blood cadmium level becomes toxic and of a clinical emergency when the value is > 5.0 µg/dL [5].

Lead (Pb) and cadmium (Cd) exposure is primarily through contaminated foods and vegetables such as tobacco leaves. Tobacco leaves have higher levels of cadmium as a result of biological build up from the soil . Meanwhile, people who do not smoke but eat meats and shellfish regularly have a higher risk of elevated blood cadmium . For the fact that these heavy metals are not of any biological importance in the human body, the tolerable monthly intake (PTMI) of Cd and Pb heavy metals of 25 µg/kg of body weight are permissible [6]

Tobacco smoking is the leading cause of avoidable illness and early death in smokers . There are different reasons why people especially the young ones smoke. Many smoke in order to reduce negative stress or anxiety and the need to establish their independence or for social validation. Others engage in smoking due to frustration or psychological reasons, or because their parents or guidians smoke. The pharmacological characteristics of nicotine have a significant impact on the habit's durability after it becomes regular [8].

Tobacco smoke contains over four thousand compounds, lead and cadmium are among these compounds. Lead and cadmium are found in tobacco and tobacco smoke . Lead is a poisonous heavy metal which can affect the neurons more especially when it is detected in children [9].

The blood level of lead and cadmium is a measure of the value of lead and cadmium in the blood. Individuals who engage in smoking have increased levels of lead and cadmium in their blood , although passive smokers (second hand smokers) also are at risk of increased lead and cadmium levels in their blood. Passive smoking (second hand tobacco smoke) is a predisposing factor for coronary heart disease [10].

Following the report of the World Health Organization (WHO), 1.1 billion people smoke globally. One-third of those smokers are within the ages of 15 and 20 years. Eight hundred million of these smokers are in developing nations, and seven hundred million of them are men. The use of tobacco in different field in Nigeria may be acceptable but it is a predisposing factor for hypertension, chronic obstructive pulmonary disease, coronary artery disease, cancer of the nasopharynx and bronchus . The main cause of untimely death globally, is smoking tobacco cigarettes and it is a major public health concern [11]

There are both short-term and long-term effects of smoking on haematological markers. Cigarette smoke contains about four thousand different chemicals and smoking exposes one to a host of dangerous substances such as free radicals, lead, cadmium,

nicotine, carbon monoxide, and other gaseous pollutants. It is well recognized that smokers are more likely to experience respiratory illnesses, coagulation disorders, inflammation, cardiovascular disease, and hypertension [12]

Most of the increased mortality linked to smoking cigarettes is caused by coronary heart disease, cancer, and other respiratory disorders. Smokers have higher risk of lung cancer, increased chances of developing chronic obstructive pulmonary disease (COPD), and also the chances of suffering a myocardial infarction in comparison with nonsmokers [13]

The results of tobacco smoking on the human body are mostly negative and can lead to reduction in bone marrow and blood cells which is known as haematological toxicity. Haematological toxicity can cause bleeding, anemia, or infection. The severity of the adverse effect is referred to as one of five classes for blood toxicity by the National Cancer Institute [14].

On the other hand, many people have installed private electrical generators randomly in nearly every neighborhood and this is as a result of epileptic power supply in Nigeria in which Abia State was not left out. The installation of these generators pose a dangerous and uncontrollable environment as it predisposes many users to the toxic generator fumes which contain lead, cadmium, carbon monoxide etc. This is because many generator users do not follow the manufacturer's manual or are ignorant of the toxic effect of exposure to generator fumes but are just concerned about the generators giving them light[15].

About sixty million people in Nigeria have power generating sets in their homes, offices, shops etc. These owners spend more than 1.5 trillion Naira to fuel them each year. It was stressed that poor public power supply has affected industrial growth in Nigeria and it is of immense concern [16].

The cost of fueling the generator is not only affecting the public sector but also private business owners who depend on electricity for their daily income generation. It is unfortunate that Nigerians have experienced worst electricity crisis among its contemporaries which has been extremely unpleasant and upsetting in electricity distribution and supply in the country. The most important infrastructural bottle neck in Nigeria is poor power supply and every establishment in Nigeria experience power outage. This explains the reason greater percentage of Nigerians have resorted to the use generators as alternative source of power generation [17]

Diesel exhaust consist of more than forty (40) toxic air contaminants such as benzene, formaldehyde and other toxic compounds such as carbon monoxide, nitrogen oxide etc. These substances are carcinogenic and about seventy percent (70%) cases of cancer have been characterized by inhalation of poisonous fumes from diesel exhaust and exposure are traceable to the cancer of the lung in work settings [18].

Evidence has shown that the danger of diesel exhaust in Nigeria has indirect link on lung cancer. This is as a result of increasing cases of nonsmokers within urban dwellers who are less than 60 years of age, that daily use power generating set on daily basis [19].

Electric generators is now the main source of powering people's homes and businesses in Nigeria. Increasing number of businesses and construction of houses had resulted in frequent use of power generating sets. The poor supply of electricity in Nigeria has caused every family to have a small generating set and this has created an environmental concern. In some houses, about 10 generating sets could be found within a radius of 5 meters. The fumes from generator contain carbon monoxide and when people are exposed to it, it becomes poisonous to human body and that is the reason many have been ignorantly dying in Nigeria. When generator fume is inhaled, carbon monoxide replaces oxygen in the tissue and it can lead to death. Sometimes, the death from exhaust fumes can come gradually in the form of dizziness, nausea, headache and even confusion [20].

It is pertinent to let the general public, especially the generator owners, repairers, users and all those within the areas where generators are powered, know that generator fumes kill, hence must not be in a confined space, rather, outside where people reside with its exhaust pipe facing the open air [11].

This study sought to draw attention to the harmful effects of smoking, exposure to toxic generator fumes on an individual's health, particularly as it relates to haematological parameters such as haemoglobin, packed cell volume, white blood cells, lymphocytes, neutrophils, eosinophils, basophils (mid), mean cell volume (MCV), mean cell haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), platelets (PLT) and heavy metals like lead, cadmium in smokers and subjects exposed to generator fumes in Umuahia, Abia State, Nigeria.

## **Material And Methods:**

### **Study Area**

The study was carried out Umuahia metropolis in Abia State, Nigeria.

### **Ethical Approval:**

The regulatory body on ethics in Federal Medical Centre Umuahia, Abia State gave approval for the study. Consent was gotten from the participants, and permission was also acquired from the appropriate authorities, who were informed about the purpose of the research. The subjects were assured that the data confidentiality, and all samples and questionnaires were maintained anonymously.

### Subjects:

The study included young adult males aged 25 to 45 years. Factors such as the period of smoking in years and the quantity of cigarettes taken daily were analyzed. A total of 210 individuals took part in this study, which was shared into two: the test subjects (smokers) and the control subjects (non-smokers and those not exposed). The test group consisted of 70 healthy male smokers and 70 individuals exposed to toxic generator fumes, all aged between 25 and 45 years, living in Umuahia metropolis, Abia State. The control group included 35 non-smokers and 35 individuals not exposed to generator fumes, also residing in Umuahia metropolis, Abia State, and aged between 25 and 45 years. All participants provided informed consent were enrolled in the study. Specimens taken from the subjects were evaluated for haematological parameters, including haemoglobin level, total leukocytes, lymphocytes, neutrophils, eosinophils, basophils (mid), and platelets. Additionally, blood levels of lead and cadmium were measured.

### Selection Criteria:

#### Inclusion Criteria:

The subjects were chosen based on these specific criteria.

Inclusion criteria for the test groups which include:

1. Subjects between the age range 25-45 years, sex (male), smoking duration (2 years or more), and smoking frequency (up to 5 cigarettes daily).
2. Also, they were subjects exposed to generator fumes, comprising 70 individuals (35 non-smokers and 35 non exposed).

#### Exclusion Criteria:

Participants were excluded based on sex (female), smoking less than 3 cigarettes daily, those with significant respiratory issues, individuals over the age of 45, pregnant women, and teenagers under 18 engaged in scavenging activities. Also, individuals with a medical history of diabetes mellitus, kidney disease, hypertension, heart conditions, or any other health disorders were also excluded.

### Sample Collection:

A volume of four (4) milliliters of venous blood was drawn from each participant using sterile syringes and needles. The obtained blood was transferred into bottles containing EDTA as an anticoagulant and mixed thoroughly through several gentle inversions before being sent to the laboratory for analysis of haemoglobin concentration, red blood cell, total white blood cell, platelet count, mean cell volume, mean corpuscular haemoglobin, and mean cell haemoglobin concentration utilizing a haematology auto-analyzer.

### Laboratory Procedures

#### Determination of Blood Lead and Cadmium Levels

Technique: Atomic Absorption Spectrophotometry (AAS)

The principle of operation for an atomic absorption spectrometer involves the uptake of sample into the flame, where it becomes atomized as the light from the atomic absorption Spectrophotometer goes through the flame to the monochromator, to the detector and then captures the light absorbed by the element that was atomized in the flame. The distinct absorption wavelengths with a specific lamp for the element is utilized, which helps minimize spectral or radiational interferences in the process. The energy absorbed in the flame its specific wavelength is directly proportional to the amount of the element in the sample.

### Full Blood Count Analysis

The full blood count haematological analyses were performed with the BC-10 Auto Hematology Analyzer.

A sample of blood taken was diluted, measured and then introduced into each transducer. The compartment of the transducer features a tiny opening known as the aperture. The cathodes and the anodes are positioned on either side of the aperture, where direct current flows. The cells of the diluted samples suspended passes through the aperture, changing the resistance in direct current between the cathode and the anode. When there is variation in the cell size, it registers as an electric pulse. This enables the cells to be counted by matching the pulses and analyzing the histogram. All procedures were executed in accordance with the standard operating procedures

### Statistical Analysis:

The analysis of data were done using the Statistical Package for Social Science (SPSS) version 27. The categorical variables were captured using frequencies and proportions, and continuous variables were expressed as mean, median and standard deviation. Variable comparisons were conducted using the Mann-Whitney U test and T-test. The relationship between blood lead, cadmium and haematological parameters were done using correlation analysis. The level of significance was placed at  $P < 0.05$ .

## Results:

Figure 4.1 shows demographic characteristics of smokers, generator fume exposure and controls. The mean age of smokers and non-smokers were  $34.20 \pm 0.83$  and  $35.11 \pm 1.25$  years, respectively. While the mean age of those exposed and non-exposed to generator fumes were  $34.50 \pm 0.80$  and  $35.11 \pm 1.25$  years. The findings showed that education level play a role in smoking habit and those exposed to generator fume, for primary level subjects, 37.14% weresmokers,30.00% were those exposed to generator fume and 15.71% were control subjects whereas for secondary level subjects, 48.57% were smokers, 50.00% were generator fume exposure, and 35.71% were control subjects. For those that attended higher institution, 14.28% were smokers, 18.57% were generator fume exposure, and 21.42% were control subjects.

Characteristics	Smokers (N= 70)	Generator fume exposure (N= 70)	Controls (N= 70)
Age (years)	$34.20 \pm 0.83$	$34.50 \pm 0.80$	$35.11 \pm 1.25$
15-25	4 (5.77 %)	7(10.00 %)	10 (14.28 %)
26-35	39 (55.71%)	36 (51.43 %)	40 (57.14 %)
36-45	27 (38.57%)	29 (41.42%)	20 (28.57 %)
Education Level			
Primary	26 (37.14 %)	21 (30.00 %)	11 (15.71%)
Secondary	34(48.57 %)	35(50.00 %)	25 (35.71 %)
Tertiary	10 (14.28 %)	13 (18.57 %)	34 (48.57 %)
Marital Status			
Married			
Unmarried	40 (57.14 %)	38 (54.28 %)	33 (47.14 %)
Married	30 (42.85 %)	32 (45.71 %)	37 (52.85 %)

*Table 4.1: Demographic characteristics of smokers, generator fume exposure and controls.*

Table 4.2 shows the significant mean difference in haematological parameters of smokers versus non-smokers: Lymphocytes % ( $43.21 \pm 13.93$  versus  $30.95 \pm 14.91$ ), PCV(%) ( $40.85 \pm 5.55$  versus  $37.82 \pm 4.18$ ), Hb(g/dL) ( $13.52 \pm 1.89$  versus  $12.28 \pm 1.37$ ), MCV (fL) ( $84.84 \pm 5.80$  versus  $82.49 \pm 7.72$ ), MCH(pg) ( $28.02 \pm 2.11$  versus  $26.79 \pm 2.77$ ), and PLT ( $\times 10^9/L$ ) ( $215.54 \pm 72.46$  versus  $195.68 \pm 63.50$ ) were significantly high; WBC ( $\times 10^9/L$ ) ( $5.62 \pm 1.70$  versus  $8.43 \pm 6.47$ ), and Neutrophils (%) ( $48.32 \pm 15.16$  versus  $60.79 \pm 15.92$ ) were significantly low in smokers as compared to non-smokers ( $p < 0.05$ ) and RBC, MID did not show any significant difference.

Hematological parameters	Smoker N = 70	Control N = 70	T- test value	p-value
WBC ( $\times 10^9/L$ )	$5.62 \pm 1.70$	$8.43 \pm 6.47$	31.898	0.001*
LYMPH (%)	$43.21 \pm 13.93$	$30.95 \pm 14.91$	0.863	0.001*
NEUT (%)	$48.32 \pm 15.16$	$60.79 \pm 15.92$	2.122	0.001*
MID (%)	$8.59 \pm 2.61$	$8.23 \pm 3.05$	0.024	0.462
PCV/HCT (%)	$40.85 \pm 5.55$	$37.82 \pm 4.18$	0.918	0.001*
HB (g/dL)	$13.52 \pm 1.89$	$12.28 \pm 1.37$	1.501	0.001*
RBC ( $\times 10^{12}/L$ )	$4.82 \pm 0.65$	$4.62 \pm 0.74$	2.7	0.084
MCV (fL)	$84.84 \pm 5.80$	$82.49 \pm 7.72$	8.955	0.044*
MCH (pg)	$28.02 \pm 2.11$	$26.79 \pm 2.77$	6.931	0.004*
MCHC (g/dL)	$32.95 \pm 1.17$	$32.32 \pm 0.75$	1.573	0.001*
PLT ( $\times 10^9/L$ )	$215.54 \pm 72.46$	$195.68 \pm 63.50$	0.866	0.001*

*Table 4.2 : Comparison of haematological parameters between Smoker and Control*

\* $P < 0.05$  was statistically significant.

Table 4.3 shows the significant mean difference in haematological parameters of Generator fume exposure versus non-exposed subjects: Lymphocytes(%) ( $38.21 \pm 13.93$  versus  $30.95 \pm 14.91$ ), PCV(%) ( $39.25 \pm 4.23$  versus  $37.82 \pm 4.18$ ), Hb(g/dL) ( $12.89 \pm 1.49$  versus  $12.28 \pm 1.37$ ), MCV(fL) ( $85.07 \pm 4.80$  versus  $82.49 \pm 7.72$ ), and MCH(pg) ( $27.01 \pm 2.11$  versus  $26.79 \pm 2.77$ ), were significantly



high; WBC( $\times 10^9/L$ ) ( $5.57 \pm 2.48$  versus  $8.43 \pm 6.47$ ), and Neutrophils (%) ( $41.58 \pm 13.18$  versus  $60.79 \pm 15.92$ ) were significantly low in smokers as compared to non-smokers ( $p < 0.05$ ) and RBC, PLT and MID did not show any significant difference.

Haematological parameters	Generator exposure N = 70	Control N = 70	T- test value	p-value
WBC ( $\times 10^9/L$ )	$5.57 \pm 2.48$	$8.43 \pm 6.47$	26.737	0.001*
LYMPH (%)	$38.21 \pm 13.93$	$30.95 \pm 14.91$	0.149	0.001*
NEUT (%)	$41.58 \pm 13.18$	$60.79 \pm 15.92$	1.097	0.001*
MID (%)	$8.82 \pm 2.76$	$8.23 \pm 3.05$	2.786	0.235
PCV/HCT (%)	$39.25 \pm 4.23$	$37.82 \pm 4.18$	0.188	0.047*
HB (g/dL)	$12.89 \pm 1.49$	$12.28 \pm 1.37$	0.394	0.013*
RBC ( $\times 10^{12}/L$ )	$4.63 \pm 0.58$	$4.62 \pm 0.74$	4.689	0.94
MCV (fL)	$85.07 \pm 4.80$	$82.49 \pm 7.72$	20.812	0.019*
MCH (pg)	$27.021 \pm 1.75$	$26.79 \pm 2.77$	15.632	0.002*
MCHC (g/dL)	$32.72 \pm 0.79$	$32.32 \pm 0.75$	0.986	0.003*
PLT ( $\times 10^9/L$ )	$231.52 \pm 67.75$	$215.54 \pm 72.46$	0.497	0.18

*Table 4.3: Comparison of haematological parameters between Generator fume exposure and Controls*

Table 4.4 shows the estimation of Lead ( $\mu g/dl$ ) between smokers and controls; smokers versus controls ( $7.66 \pm 0.44$  versus  $5.98 \pm 2.81$ ) was significantly higher in smokers compared to controls, and the estimation of cadmium ( $\mu g/dl$ ) between smokers and the control; smokers versus controls ( $4.18 \pm 1.65$  versus  $4.15 \pm 1.85$ ) was insignificant.

Heavy metals	Smokers N = 70	Controls N = 70	T- test value	p-value
Lead( $\mu g/dl$ )	$7.66 \pm 0.44$	$5.98 \pm 2.81$	5.573	0.02*
Cadmium( $\mu g/dl$ )	$4.18 \pm 1.65$	$4.15 \pm 1.85$	0.569	0.452

\* $P < 0.05$  was statistically significant

*Table 4.4 : Comparison of heavy metals between smokers and controls*

Table 4.5 shows the estimation of Lead ( $\mu g/dl$ ) between generator fume exposure and controls; generator fume exposure versus control ( $5.98 \pm 2.81$  versus  $3.00 \pm 0.89$ ) was significantly higher in smokers compared to controls, and the estimation of cadmium ( $\mu g/dl$ ) between generator fume exposure and the controls; generator fume exposure versus controls ( $4.15 \pm 1.85$  versus  $4.08 \pm 1.98$ ), was statistically significant ( $p < 0.05$ )

Heavy metals	Controls N = 70	Generator fume exposure N = 70	T- test value	p-value
Lead( $\mu g/dl$ )	$3.00 \pm 0.89$	$5.98 \pm 2.81$	26.737	0.001*
Cadmium( $\mu g/dl$ )	$4.08 \pm 1.98$	$4.15 \pm 1.85$	0.149	0.001*

*Table 4.5: Comparison of Heavy metals between those exposed to generator fume and non exposed to generator fumes*

Table 4.6 shows the Correlation of haematological parameters among the controls, subjects exposed to generator fumes and smokers. The white blood cells among the controls, generator fume exposure, and smokers. The correlation of white blood cells between the controls and generator fume exposure as well as the correlation of white blood cell between the smokers and generator fume exposure revealed a significant positive correlation among the groups while the correlation of white blood cells between the generator fume exposure and controls as well as the correlation of white blood cells between the generator fume exposure and smokers showed a significant negative correlation among the groups ( $p < 0.05$ ).

The correlation of lymphocyte between the controls and generator fume exposure as well as the correlation of lymphocytes between the smokers and non smokers revealed a significant positive correlation among the groups while the correlation of lymphocytes between the control and smokers showed a significant negative correlation among the groups ( $p < 0.05$ ).

The correlation of neutrophils between the controls and generator fume exposure as well as the correlation of neutrophils between the controls and the smokers revealed a significant positive correlation among the groups while the correlation of neutrophils between the generator fume exposure and controls as well as the correlation of neutrophils between the smokers

and controls showed a significant negative correlation among the groups ( $p < 0.05$ ).

The correlation of haemoglobin between the smokers and controls revealed a significant positive correlation while the correlation of haemoglobin between the controls and smokers showed a significant negative correlation among the groups ( $p < 0.05$ ). The correlation of red blood cell between the generator fume exposure and controls as well as generator fume exposure and smokers revealed a significant negative correlation among the groups ( $p < 0.05$ ).

The correlation of Mean Corpuscular Haemoglobin between the generator fume exposure and control revealed a significant positive correlation while the correlation of Mean Corpuscular Haemoglobin between the control and generator fume exposure showed a significant positive negative correlation among the groups ( $p < 0.05$ ). The correlation of Mean Corpuscular Haemoglobin Concentration between the controls and generator fume exposure as well as the correlation of Mean Corpuscular Haemoglobin Concentration between the controls and the smokers revealed a significant negative correlation among the groups while the correlation of Mean Corpuscular Haemoglobin Concentration between the generator fume exposure and controls as well as the correlation of Mean Corpuscular Haemoglobin Concentration between the smokers and control showed a significant positive correlation among the groups ( $p < 0.05$ ). The correlation of MID, MCV and platelet count showed insignificant correlation among the groups.

Hematological parameters	Comparison	Correlation	p-value
WBC ( $\times 10^9/L$ )	1 versus 2	2.86286*	0.041
	1 versus 3	-0.04286	0.999
	2 versus 1	-2.86286*	0.041
	2 versus 3	-2.90571*	0.038
	3 versus 1	0.04286	0.999
	3 versus 2	2.90571*	0.038
LYMPH (%)	1 versus 2	-10.63000*	0.001
	1 versus 3	-8.82286*	0.001
	2 versus 1	10.63000*	0.001
	2 versus 3	1.80714	0.734
	3 versus 1	8.82286*	0.001
	3 versus 2	-1.80714	0.734
NEUT (%)	1 versus 2	11.20571*	0.001
	1 versus 3	9.01429*	0.002
	2 versus 1	-11.20571*	0.001
	2 versus 3	-2.19143	0.671
	3 versus 1	-9.01429*	0.002
	3 versus 2	2.19143	0.671
MID (%)	1 versus 2	-0.58714	0.428
	1 versus 3	-0.25	0.856
	2 versus 3	0.58714	0.428
	2 versus 3	0.33714	0.755
	3 versus 1	0.25	0.856
	3 versus 2	-0.33714	0.755
PCV (%)	1 versus 2	-1.42857	0.162
	1 versus 3	-3.04286*	0.001
	2 versus 1	1.42857	0.162
	2 versus 3	-1.61429	0.099
	3 versus 1	3.04286*	0.001
	3 versus 2	1.61429	0.099

HB (g/dl)	1 versus 2	-0.60857	
	1 versus 3	-1.10000*	0.001
	2 versus 1	0.60857	0.065
	2 versus 3	-0.49143	0.166
	3 versus 1	1.10000*	0.001
	3 versus 2	0.49143	0.166
RBC (x10 <sup>12</sup> /L)	1 versus 2	-0.00857	0.997
	2 versus 3	-.29571*	0.029
	2 versus 1	0.00857	0.997
	2 versus 3	-.28714*	0.035
	3 versus 1	0.29571*	0.029
	3 versus 2	0.28714*	0.035
MCV (fL)	1 versus 2	-2.57714	0.053
	3 versus 2	-0.17714	0.986
	2 versus 1	2.57714	0.053
	2 versus 3	2.4	0.078
	3 versus 1	0.17714	0.986
	3 versus 2	-2.4	0.078
MCH (pg)	1 versus 2	-1.22857*	0.007
	1 versus 3	-0.42714	0.532
	2 versus 1	1.22857*	0.007
	2 versus 3	0.80143	0.112
	3 versus 1	0.42714	0.532
	3 versus 2	-0.80143	0.112
MCHC (g/dL)	1 versus 2	-.39143*	0.025
	1 versus 3	-.62429*	0.001
	2 versus 1	.39143*	0.025
	2 versus 3	-0.23286	0.263
	3 versus 1	.62429*	0.001
	3 versus 2	0.23286	0.263
PLT(x10 <sup>9</sup> /L)	1 versus 2	-15.98571	0.359
	1 versus 3	3.08571	0.962
	2 versus 1	15.98571	0.359
	2 versus 3	19.07143	0.234
	3 versus 1	-3.08571	0.962

**Table 4.6 : Correlation of haematological parameters among the controls, subjects exposed to generator fume and smokers.**

Key: 1=Control, 2= generator exposure, 3 = smokers; p<0.05 was statistically significant

Table 4.7 shows the Correlation of blood heavy metals among the controls, subjects exposed to generator fume, and smokers, the blood lead among the controls, generator fume exposure, and smokers. The correlation of blood lead between the smokers and controls; correlation between controls and generator fume exposure and control as well as the correlation of blood lead between the smokers and generator fume exposure revealed a significant positive correlation among the groups while the correlation of blood lead between the smokers and control as well as the correlation of blood lead between the smokers and generator fume exposure showed a significant negative correlation among the groups (p<0.05). The blood cadmium showed insignificant correlation among the groups (p<0.05)



Heavy metal	Comparison	Correlation	p-value
Lead( $\mu\text{g}/\text{dL}$ )	1 versus 2	2.97714*	0.001
	1 versus 3	-1.68571*	0.001
	2 versus 1	-2.97714*	0.001
	2 versus 3	-4.66286*	0.001
	3 versus 1	1.68571*	0.001
	3 versus 2	4.66286*	0.001
Cadmium( $\mu\text{g}/\text{dL}$ )	1 versus 2	0.06571	0.976
	2 versus 3	-0.03429	0.993
	2 versus 1	-0.06571	0.976
	2 versus 3	-0.1	0.944
	3 versus 1	0.03429	0.993
	3 versus 2	0.1	0.944

*Table 4.7: Correlation of haematological parameters among the controls, subjects exposed to generator fume, and smokers.*

Key: 1= Control, 2= generator exposure, 3= smokers

## Discussion:

The results showed that cigarette smoking and exposure to generator fumes has adverse effects on haematological parameters. Increase in haemoglobin concentration among smokers and subjects exposed to generator fumes is caused by inhalation of carbon monoxide. When carbon monoxide is inhaled, it results in formation of carboxyhaemoglobin and this is due to the binding of carbon monoxide with haemoglobin which results in inability to transport oxygen. Carboxyhaemoglobin alters haemoglobin dissociation curve shifting it to the left thereby reducing the oxygen transport to the tissue. To counter balance the hypoxic condition, smokers and subjects exposed to generator fumes maintain higher haemoglobin than non smokers and non exposed subjects [21]

Carbon monoxide inhaled from cigarette smoking and generator fumes cause the capillaries to become more permeable resulting in reduction of the plasma volume which then mimics polycythemia leading to an increase in red cell volume thereby reflecting increased values of haematocrit [10]. The effect of lead (Pb) and cadmium (Cd) on haematological parameters is dependent on the level of exposure to these heavy metals [22]. Exposure to lead and cadmium are considered normal and non exposed at less than  $10\mu\text{g}/\text{dL}$  and  $5\mu\text{g}/\text{dL}$  respectively, above which they become poisonous. Therefore, sources of exposure to lead and cadmium must be avoided [5]. The toxic levels of lead and cadmium are of clinical concern because they can result to a serious health challenge which might be difficult to reverse [7]. Presently, there is a serious concern not just on preventive measures but also on the effects of subclinical toxicities of lead and cadmium at lower doses [23].

There have been several studies in changes in haematological parameters due to Pb and Cd exposure but the effects on other blood cells are few in Nigeria in comparison with other countries. This study assessed some haematological parameters and their relationship with lead and cadmium levels. There is a significant difference in the mean blood Pb level in this study but there is no significant difference in the blood cadmium in smokers and individuals exposed to generator fumes. The effects of lead and cadmium exposure becomes abnormal at Pb and Cd levels of  $10\mu\text{g}/\text{dL}$ ,  $5\mu\text{g}/\text{dL}$  respectively. When the values are above  $10\mu\text{g}/\text{dL}$  and  $5\mu\text{g}/\text{dL}$  respectively, it can result in complications and then becomes a clinical emergency [24]. The effects on lymphocytes and the platelets are as a result of the direct toxic action of lead and cadmium on lymphoid organs and bone marrow which leads to lymphocytosis as well as platelet aggregation [25].

This study shows that some of the mean values of blood cells of the haematological parameters on smokers and individuals exposed to generator fumes were significantly increased. This study differs from the study carried out in Ghana, where a healthy population subjects were used and the mean values of the haematological parameters were normal [3]. This is because the majority of smokers and individuals exposed to generator fumes in Umuahia, Abia State Nigeria, have high blood lead levels in their blood. The high mean values of haematological parameters were as a result of the majority of the subjects, >70% in both smokers and exposed individuals with haematological parameters are higher in test subjects when compared to the control subjects.

These findings also varies from the studies in Egypt and Iran where the subjects were children and the results showed higher mean level and abnormal values of haematological parameters with other countries like Jordan, Iraq, and Taiwan where lead and cadmium levels were within an acceptable range [26]. The bone marrow in children are still developing and are immature in comparison with adults bone marrow. There is direct toxicity and increased destruction of red blood cells caused by exposure to lead and cadmium.

The lead and cadmium exposure on haematological parameters in this research have significant linear relationship and also in

correlation with smokers and individuals exposed to generator smoke. The correlation shows that there is a relationship between the variables and for the fact that they are correlated, there is tendency of toxic outcome. Therefore, there is need to avoid exposure to these toxic heavy metals and to constantly monitor blood lead and cadmium to maintain the normal levels [22]. This findings were consistent with the work of [27] who reported that cigarette smokers have high levels blood lead than non smokers but differs from the work of [28] who reported that smokers have higher white blood cell count. The findings further showed that uncontrolled lead and cadmium exposure with the effect on haematological parameters can cause anaemia, lymphocytosis, aggregation of platelets which is a risk factor for bleeding disorders.

### Conclusion:

The findings indicated that persistent cigarette smoking and exposure to generator emissions negatively impact hematological parameters, and these changes might be a predisposing factor for build up of fats, chloesterol and other substances on the walls of the arteries, chronic leukaemia, disorders of the lungs and heart disorder also known as cardiovascular disorders. In addition, haematological tests should form part of the routine investigations in Medical Laboratories as this will reveal the clinical effects of exposure to blood lead in addition to personal and environmental hygiene practices.

### References:

1. Akabs I. J., Jeremiah T. A., JeremiahZ. A (2023).Utility of World Health Organization Haematological Toxicity Scale in theAssessment of Smokers in Port Harcourt, Nigeria. *African Journal of Haematology and Transfusion Science*. 2(4):258-265.
2. Ganguly, K., Levänen, B., Palmberg, L., Åkesson, A and Lindén, A (2018). Cadmium in tobacco smokers: A neglected link to lung disease?. *European. Respiratory Review*. 27:170122.
3. Darwish, W. S (2020). Identification of cadmium-produced lipid hydroperoxides, transcriptomic changes in antioxidant enzymes, xenobiotic transporters, and pro-inflammatory markers in human breast cancer cells (MCF7) and protection with fat-soluble vitamins. *Environmental. Science. Pollution Reports*. 27:1978-1990.
4. Addai-Mensah O., Gyamfi D., Duneeh R. V., Danquah K. O., Annani-Akollor M. E., Boateng L (2019). Determination of Haematological Reference Ranges a Healthy Adult in Three Regions in Ghana. *Biomedical Resource International*. 2019:7467512.
5. Gil, F (2020). Biomonitorization of cadmium, chromium, manganese, nickel and lead in whole blood, urine, axillary hair and saliva in an occupationally exposed population. *Science Total Environment*. 409:1172-1180.
6. Hsieh N. H., Chung S. H., Chen S. C., Chen W. Y., Cheng Y. H., Lin Y.J (2017). Anaemia risk in relation to lead exposure in lead - related manufacturing. *Biomedical central Public Health*. 17(1):389.
7. Jeong, S. H., Sheridan, J., Newcombe, D and Tingle, M (2018). Plasma concentrations of cytisine, a commercially available plant-based alkaloid, in healthy adult smokers taking recommended doses for smoking cessation. *Xenobiotica*48: 1245-1248.
8. Ghasemi A., Nakhaei A. A., Ghamseri A. A., Salehi M., Kalani-Moghaddam F (2017). Anaemia, Iron Deficiency Anaemia, and Lead Poisoning in Children with Opiod Toxicity: A Study in North East of Iran. *Iranian Journal Pediatric Haematology Oncology* 7(2):90-97
9. Kim, J (2023). Smoking and passive smoking increases mortality through mediation effect of cadmium exposure in the United States. *Science Report*. 13: 3878.
10. Genchi, G., Sinicropi, M. S., Lauria, G., Carocci, A and Catalano, A (2020). The effects of cadmium toxicity. *International Journal Environmental Resource Public Health* 17: 3782.
11. Briffa J., Sinagra E., Blundell R (2020). Heavy metal pollution in the environment and their toxicological effects on humans. *Heliyon*. 1;6(9):e04691.
12. Lee, J. W., Kim, Y., Kim, Y., Yoo, H and Kang, H. T (2020) Cigarette smoking in men and women and electronic cigarette smoking in men are associated with higher risk of elevated cadmium level in the blood. *J. Korean Med. Sci*. 35, e15
13. Benowitz, N. L (2020) Biochemical verification of tobacco use and abstinence: 2019 update. *Nicotine Tobacco Resource*. 22:1086-1097.
14. Błażewicz, A (2022). Research into the association of cadmium and manganese excretion with thyroid function and behavioral areas in adolescents with autism spectrum disorders. *Journal Clinical Medicine*. 11(3), 579.
15. Taha G.E.M., Afify M.M ,Elmalah W.M., Gaber M.R (2018). Impacts of Blood Lead Level on Trace Elements Status and Haematological Parameters in Anemic Children from Beni - Suef, Egypt. *Journal Clinical Toxicology* 8(2):1000383.
16. Bovio, F., Melchiorretto, P., Forcella, M., Fusi, P and Urani, C (2021). Cadmium promotes glycolysis upregulation and glutamine dependency in human neuronal cells. *Neurochemical Interaction*. 149: 105-144.
17. Luo L., Wang B., Jiang J., Fitzgerald M., Huang Q., Yu Z., Li H., Zhang J., Wei J., Yang C., Zhang H (2021). Heavy metal contaminations in herbal medicines: determination, comprehensive risk assessments, and solutions. *Front Pharmacology*. 14:11:595335.
18. Minnesota Department of Health (2018). Occupational Health and Safety Data, Occupational Lead Exposure in Adults. Saint Paul, Minnesota, United States: Minnesota Department of Health.

19. Queiroz T. K., Naka K. S., Mendes L. D., Costa B. N., Jesus I. M., Câmara V. D., Lima M. D (2019). Human blood lead levels and the first evidence of environmental exposure to industrial pollutants in Amazon. *International Journal Environmental Resource Public Health*. 16(17) :3047
20. Gazwi H. S. S., Yassien E. E., Hassan H. M (2020). Mitigation of lead neurotoxicity by the ethanolic extract of Laurus leaf in rats. *Ecotoxicological Environmental Safety*. 1192:110297.
21. Courtney, R. J (2021). Effect of cytisine vs varenicline on smoking cessation: A randomized clinical trial. *JAMA* 326:56–64.
22. Saka A (2021). Environmental Effects of Generator fumes in Kwara State, Nigeria. A Survey- An Emperical Evidence. 9(5):59-88.
23. Zong, L., Xing, J., Liu, S., Liu, Z and Song, F (2018). Cell metabolomics reveals the neurotoxicity mechanism of cadmium in PC12 cells. *Ecotoxicol. Environmental Safety*. 147:26–33.
24. Shen X., Chi Y., Xiong K (2019). The effect of heavy metal contamination on humans and animals in the vicinity of a zinc smelting facility. *PLoS ONE*. 28(10):e0207423.
25. Muhammed, I. (2022). The Effect of Cigarette Smoking on HaematologicalParameters In Healthy College Students In The Ca Capital Baghdad. *European Journal of Molecular and Clinical Medicine*. 9(3):11013-11022.
26. Nargish S., Kabir M. H., Akhter K., Nahid K. A., Hossain M. M., Karmakar P., Ahmed M., Islam M. Z (2022) Effect of Smoking on the Red Blood Cell Count, Hemoglobin Concentration, Hematocrit and Red Cell Indices in Adult Male Smokers. *Eastern Medical College Journal*. 7 (1): 1-5.
27. Tsai M. T., Huang S. Y., Cheng S. Y (2017). Lead poisoning can be easily misdiagnosed as acute porphyria and nonspecific abdominal pain case reports in emergency medicine. *Case Report Emergency Medicine*.

## References:

1. Ekwochi, U., Osuorah, D. I. C., Ndu, I. K., Onah, K. S., & Ezenwosu, O. U. (2021). Challenges in neonatal resuscitation practices in Nigeria. *African Health Sciences*, 21(4), 1501-1510.
2. Ogunlesi, T. A., & Olawuyi, T. (2022). Neonatal resuscitation practices in sub-Saharan Africa: Challenges and prospects. *Journal of Neonatal Care*, 14(2), 101-112.
3. Sola, A., Salinas, C., & Schmölzer, G. M. (2020). Optimizing neonatal resuscitation training: Simulation and practical approaches. *Pediatric Research*, 88(2), 298-305.
4. World Health Organization (WHO). (2022). Newborn mortality and causes of death. Geneva: WHO.
5. Aliyu, I., Lawal, T., & Olatunde, O. (2021). Knowledge and practice of neonatal resuscitation among midwives in rural health facilities in Nigeria. *Journal of Neonatal Nursing*, 27(3), 45-51.
6. Oladapo, O. T., Sule-Odu, A., & Olagbuji, B. (2019). Challenges of neonatal resuscitation in Nigeria: Perspectives from healthcare workers. *International Journal of Child Health and Nutrition*, 8(1), 23-30.
7. Singhal, N., Lockyer, J., & Fiadjoe, J. (2020). Helping Babies Breathe: Lessons learned and future directions. *Global Health Science and Practice*, 8(2), 25-35.
8. Ogunlesi, T. A., Fetuga, M. B., & Olawale, O. (2020). Healthcare providers' knowledge and application of neonatal resuscitation guidelines in Nigeria. *BMC Pediatrics*, 20(1), 15-27.BS. (2023). Nigeria demographic and health survey 2023. Abuja: NBS.
9. Nwachukwu, C. E., Ezenwa, B., & Uche, C. (2022). Barriers to effective neonatal resuscitation in southeastern Nigeria: A mixed-methods study. *African Journal of Reproductive Health*, 26(4), 87-96.
10. Ojha N., Malla D. S., Thapa K., Shrestha B (2018). Knowledge and skills of neonatal resuscitation of health workers in a tertiary hospital in Nepal. *BMC Pediatrics* .;18(1):p. 62
11. O'Keefe-McCarthy S., McGovern G., Fitzpatrick J. J(2015).. Ensuring competency in neonatal resuscitation. *Nurse Education Today* 35(5):e45-e49.
12. Bell M., Toffol J., Cowell I. (2018). Neonatal resuscitation practices: Insights for midwifery education. *Journal of Obstetric, Gynecologic, and Neonatal Nursing* . 47(6):s27-s32.
13. Poggi S., Pennafina E., Chigi F., Cerone P(2016). Impact of simulation-based training in neonatal resuscitation. *Journal of Maternal-Fetal and Neonatal Medicine* .29(18):2996-3001.
14. Johnson M., Wilson T., Marsden C., Terbahn C., Van Riel E(2014). Assessment of hands-on training in neonatal resuscitation. *Journal of Nursing Education and Practice* ;4(5):21-27
15. Murila F, Obimbo MM, Musoke R(2012). Assessment of knowledge on neonatal resuscitation amongst health care providers in Kenya. *Pan African Medical Journal*. 11:78
16. Patel J, Posencheg M, Ades A(2012). Proficiency and retention of neonatal resuscitation skills by pediatric residents. *Pediatrics*. 130(3):515-521

17. Sankar MJ, Natarajan CK, Das RR, Agarwal R, Chandrasekaran A, Paul VK (2016). When do newborns die: a systematic review of timing of overall and cause-specific neonatal deaths in developing countries. *J Perinatol.* 36(Suppl 1Suppl 1):S1-S11.
18. Bassani DG, Kumar R, Awasthi S, Morris SK, Paul VK, (2010).. Causes of neonatal and child mortality in India: nationally representative mortality survey. *Lancet.*;376(9755):1853-1860.
19. Wall SN, Lee AC, Niermeyer S, English M, Keenan WJ, Carlo W, (2009). Neonatal resuscitation in low-resource settings: what, who and how to overcome challenges to scale up. *Int J Gynaecol Obs.* 107(Suppl 1Suppl 1):S47-62. S63-64.
20. Adebami O, Oyediji O, Joel-Medewase V, Oyediji G, Fadero F(2007).. Neonatal resuscitation in some Nigerian primary and secondary health institutions: an evaluation of ongoing practices. *Niger J Paediatr.*;34(1-2):8-13
21. Kim YM, Ansari N, Kols A, Tappis H, Currie S, Zainullah P, (2013). Assessing the capacity for newborn resuscitation and factors associated with providers' knowledge and skills: a cross-sectional study in Afghanistan. *BMC Pediatr.* 2;13:140
22. Eke GK, Nyengidiki T, Nte A(2012). Teaching of essential maternal and newborn interventions: how skilled are the trainers from health service provider training institutions in Port Harcourt, Rivers State. *Port Harcourt Med J.* 6:184-191.
23. Enweronu-Laryea C, Engmann C, Osafo A, Bose C(2009).. Evaluating the effectiveness of a strategy for teaching neonatal resuscitation in West Africa. *Resuscitation.*;80(11):1308-1311
24. Conroy N, Kaiwo J, Barr D, Mitchell L, Morrissey B, Lambert S(2015).. Skills retention 3 months after neonatal resuscitation training in a cohort of healthcare workers in Sierra Leone. *Acta Paediatr.* 104(12):1305-1307.

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