THE IMPACT OF VEHICULAR EMISSION ON ERYTHROCYTE LEVEL AND RED CELL INDICES OF OCCUPATIONALLY EXPOSED SUBJECTS IN A GIVEN CITY

Abstracts

Background

The association of combustive emission has been inter-related with haematologic parameters and a likelihood to pro-inflammatory state. This research is aimed at assessing the impact of vehicular emission on erythrocyte level and red cell indices of occupationally exposed subjects. **Method**: The level of haemoglobin concentration, haematocrit, red blood cells, mean cell volume, mean cell haemoglobin, mean cell haemoglobin concentration, and red cell distribution width were determined in four hundred subjects with the aid of Mythic 22 haematology autoanalyser. **Results**: The comparison of the haemoglobin and haematocrit shows that the value of mean ± standard error value of generator exposed and mechanics respectively were significantly higher than the other groups ($p< 0.00$). On the otherhand, the red blood cell count for generator exposed and mechanics ranked higher than the other groups ($p< 0.00$). The mean cell volume (MCV) of generator exposed and mechanics were 86.57±0.08 fl and 84.49±1.04 fl respectively while control and drivers had values of 93.24±1.13 fl and 93.22±1.13 fl respectively ($p< 0.00$). The mechanics recorded a mean cell haemoglobin (MCH) of 26.92±0.40 pg which was significantly lower ($p< 0.04$) than the control (30.37±0.47 pg), generator exposed (39.68±6.38 pg) and drivers (30.42±0.47 pg). **Conclusion**: The differences amongst the groups and none of the groups were within the medically acceptable ranges which is a pointer to the fact that there might be an underlying inflammatory condition which might be due to occupational exposure.

Keywords: Combustion emission, red blood cell, inflammatory, generator-exposed, drivers, control, mechanics, vehicular, haematocrit, haemoglobin, occupationally-exposed, red cell indices.

1.0 Introduction

Engine emissions are gases and particulates that are expelled by automobiles or other combustion-powered mechanical devices. The global economy relies on fossil fuel to function effectively [1]. Some of the emitted substances are particulate matter (PM), carbon monoxide (CO), carbon dioxide (CO_2), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone, polycyclic aromatic hydrocarbons (PAH) etc.

Emission from exhausts leads to poisoning of body cells thereby altering the pathways in the human body. The alteration might be connected to constriction of vessels and inflammatory pathophysiological response [2]. A selection of the health effects of these include mortality, disorders in the airways, cardiovascular diseases, formation of malignant cells, elevated stress of oxidative cells, mental challenge, dysfunction of endothelium and allergic reactions [3] [4] .

The association of combustive emission has been inter-related with haematologic parameters and a likelihood to pro-inflammatory state. It has been documented [5] that erythrocytes, haemoglobin, mean cell volume and mean cell haemoglobin concentration are decreased in fumes exposed subjects.

This decrease may be connected to aftermath of benzene oxide and xylene compounds [6]. Benzene is documented to initiate pancytopenia which could lead to bone marrow aplasia [7]. Previous studies documented increase in erythrocytes, haemoglobin, mean cell volume and mean cell haemoglobin concentration in gasoline exposed groups [8] [9].

Emission from exhausts leads to poisoning of body cells thereby altering the pathways in the human body. The alteration might be connected to constriction of vessels and inflammatory pathophysiological response. The deleterious constituents especially those in petrol, is linked with alteration in blood and decrease in haematocrit level through the induction of hypoplasia in animal models [7]. Combustion engine emissions (CEEs) are activated in the marrows of blood and the cytotoxic harms are seen through alteration in the functioning of deoxyribonucleic acid. Petroleum related fumes leads to depression of cells of the marrow leading to deficient generation of erythrocytes and some cellular components [10].

Materials and methods

Study population

A Comparative cross-sectional study was carried out on a random sample of exposed subjects in Benin City, South-South Nigeria. The groups includes commercial bus drivers, mechanics, generator/plant houses workers as well as controls (individuals not exposed occupationally).

Inclusion criteria of exposed group

Subjects who are working regularly for at least one years in CEE-related occupation (at risk of emission from generator and/or vehicular exhaust).

Exclusion criteria of exposed group

Subjects such as chronic diseases (e.g. cancer, hepatitis, HIV/AIDS, *Mycobacterium tuberculosis*, *Mycobacterium leprae* etc.), renal disease, hepatic disease, diabetes mellitus, blood donation in the past 6 months, clinical evidence of haemorrhage, cardiovascular disease, deficiencies (iron or folate or vitamin B12 deficiency etc.), chemotherapy, concomitant infections, individuals living or working near benzene industries/filling stations (fuel/diesel/kerosene), refinery workers, obesed and underweight individuals were excluded from the study. Individuals below 20 and above 60 years were not considered as well. Interestingly, sample collection and analysis were done before outbreak of coronavirus disease 19 (COVID 19) in Nigeria and most countries of the world and as such consideration was not given for this.

Inclusion criteria

Individuals that have continuous activities in any of these occupational exposures was enlisted after meeting the research selection criteria. Staff and student at University of Benin who were not occupationally exposed to vehicle exhaust/generator at their workplace/classes and; belonged to the same age group as the included subjects was enlisted.

Age range

All participants were adult (20-60 years). Informed consent was obtained from all the voluntary participants on random basis (complete randomized sampling).

Administration of questionnaire

Structured questionnaire was administered to everyone to gather demographic data. The details of the questionnaire includes personal data, occupational history, smoking habits, medical history; exposure to irradiation/chemotherapy/electronic waste and others.

Sample size determination

The sample size was calculated using the formula:

$$N = \frac{Z^2 P\,(1-P)}{d^2}$$

Where n = sample size, Z = z statistic for a level of confidence, P = expected prevalence or proportion, d = precision (Daniel, 1999).

According to Ali *et al*. (2015), the prevalence due to automobile exhaust emission was 8.80% in subjects.

P = 8.80 % (= 0.088)

Z = 1.96 for the level of confidence of 95 %, d = 5 % = (0.05).

$$N = \frac{Z^2 P\,(1-P)}{d^2} = \frac{1.96 \times 1.96 \times 0.088(1-0.088)}{0.05 \times 0.05} = 123.40 = 124.0$$ subjects (approximately).

For the purpose of this research, three hundred (300) occupationally exposed subjects (OESs) and one hundred (100) apparently healthy controls were used as sample size.

The sampling was carried in the order of generator/plant houses: 100 subjects; commercial bus drivers: 100 subjects; automobile mechanics: 100 subjects and apparently healthy controls: 100 subjects.

Collection of sample

Five (5) ml of blood was collected carefully from the cubital vein using a vacuutainer bottle, needle and holder while applying tourniquet lightly over the arm until blood flow was established under negative pressure. The K_3EDTA sample was used for full blood cell count.

Processing of sample

It will be carried out at the Haematology and Blood Transfusion unit, Medical Laboratory Science Department, University of Benin, Physiology Department, University of Benin, Medical Laboratory Services unit, University of Benin Teaching Hospital; as well as other research centers.

Analysis of data

This will be done with the aid of Graphpad Prism v7.03, California, USA. Data obtained from this research work was presented in table 1 and conclusion was drawn at $p < 0.05$.

Result

As seen in table 1, the comparison of the haemoglobin (HGB) and haematocrit (HCT shows that the value of mean ± standard error value (M±SEV) of generator exposed and mechanics respectively were significantly higher than the other groups ($p< 0.03$). On the otherhand, the red blood cell count (RBC) for generator exposed and mechanics ranked higher than the other groups ($p< 0.03$). The mean cell volume (MCV) of generator exposed and mechanics were 86.57±0.08 fl and 84.49±1.04 fl respectively while control and drivers had values of 93.24±1.13 fl and 93.22±1.13 fl as well ($p< 0.03$). The mechanics recorded a mean cell haemoglobin (MCH) of 26.92±0.40 pg which was significantly lower ($p< 0.04$) than the control (30.37±0.47 pg), generator exposed (39.68±6.38 pg) and drivers (30.42±0.47 pg). The mean cell haemoglobin concentration (g/dl) of generator exposed subjects were significantly higher ($p< 0.03$) than the other groups {controls (32.53±0.17), generator exposed (34.92±1.21), drivers (32.53±0.17) and mechanics (31.59±0.17)}. When the red cell distribution width of generator exposed groups were statistically tested against the other subjects, it was statistically higher than others ($p< 0.01$).

Table 1: The haemoglobin, haematocrit, red blood cell and red cell indices of subjects.

SUBJECT	PARAMETER							
	HGB (g/dl)	HCT (%)	RBC ($X10^6$ /ul)	MCV (fl)	MCH (pg)	MCHC (g/dl)	RDWC	RDWS

Controls n= 100	11.07± 0.22[a]	34.24±0.52[a]	3.83±0.08[a]	93.24±1.13[b]	30.37±0.47[a]	32.53±0.17[a]	14.82±0.23[a]	50.42±0.63[b]
Generator exposed n= 100	12.74±0.25[b]	39.93±2.20[b]	4.29±.20[b]	86.57±0.80[a]	39.68±6.38[b]	34.92±1.21[b]	26.16±5.47[b]	44.17±1.81[a]
Drivers n= 100	11.06±0.21[a]	33.91±0.53[a]	3.67±0.65[a]	93.22±1.13[b]	30.42±0.47[a]	32.53±0.17[a]	14.82±0.23[a]	50.52± 0.63[b]
Mechanics n= 100	11.50±0.21[a]	36.08±6.28[a]	4.32±.08[b]	84.49±1.04[a]	26.92±0.40[a]	31.59±0.17[a]	15.45±0.22[a]	48.26±0.80[b]
p-value	0.03	0.03	0.03	0.3	0.04	0.03	0.01	0.03

HGB: haemoglobin, HCT: haematocrit, RBC: red blood cell, MCV: mean cell volume, MCH: mean cell haemoglobin, MCHC: mean cell haemoglobin concentration, RDWC: RDWS red cell distribution width:

Key: Values with same superscript are not significantly different from each other.

Discussion

The result of this study showed that there was no significant difference (p> 0.05) between the control, driver, and mechanic groups in terms of haemoglobin (HGB) concentration, haematocrit, red blood cells (RBC), mean cell volume (MCV), mean cell haemoglobin (MCH), mean cell haemoglobin concentration (MCHC), and red cell

distribution width (RDWC). Diesel exhaust has been linked with adverse effects and underlying cellular mechanisms [1] and similar trend was obtained in this present work. Alongside, biochemical and physiological effects from exhaust emissions have been documented [2]. The literature that is currently available suggests that various vehicular pollution components are related to a relative decline in erythrocyte count. According to statistics, anaemia affects 1.62 billion people worldwide and is associated with a number of negative effects on health, including increased mortality and cognitive impairment. About 89% of all anemia-related problems are linked to underdeveloped countries [12]. The World Health Organization estimates that inflammatory anaemia accounts for 42% of cases, whereas iron deficiency anemia accounts for 50% of cases. As seen in third world nations, some subjects' inflammatory anemia or anemia of inflammation (IA/AI) is associated to infection [13]. Aside from infections, there are other known causes of inflammatory diseases, according to the literature, including chronic kidney disease, congestive heart failure, chronic pulmonary disease, and obesity [14]. The values from the generator-exposed groups, however, were noticeably greater than those from the other groups. Although there may not be a clear cause for this discrepancy, the fact that the groups being studied appear to be healthy and are exposed to occupational hazards is one item that comes to mind. In this situation, exposure to vehicle air pollution has also been implicated. The systemic inflammation caused by NO_2 and fine particulate matter (PM 2.5) with a diameter of 2.5 microns or smaller has a direct impact on the bone marrow [15, 16]. Acute exposure to this pollutant, which is quite hazardous in nature increases human mortality rates by 2.8%. Additionally, chronic obstructive lung illness and cardiovascular disease are linked to exposure to PM 2.5 and this could cause bronchiole inflammation that is dependent on oxidative stress [17] [18]. Red blood cell half-lives are shortened, which furthers the development of AI by reducing the erythropoietin response to anemia and the suppression of erythroid cell differentiation by inflammatory mediators [13]. The shortening of the erythrocyte half-life have both been linked to oxidative stress [19]. Air pollution may influence

haemoglobin (Hb), which is likely affect several age groups and result in anemia. The likelihood of developing anemia appears to be correlated with the use of biofuel. Children and different age groups have proven this effect. The results of this study demonstrate that in some groups, exposure to rising levels of vehicular emissions, including PM 2.5, nitric oxide, and others, was not directly associated with a drop in haemoglobin levels. Although the readings of the generator-exposed groups were greater than those of the other groups, there is evidence that these values are still modest when compared to those that are considered acceptable medically. The findings suggest that although anemia does not display an inflammatory process, vehicle pollution may be pointing to one in the human body [20].

The components of vehicle emissions have been associated to an increase in both chronic and systemic inflammation [21, 22]. This series of events may have played a role in the study's observed increase in anemia prevalence and decreased haemoglobin levels.

In order to illustrate a proportionate correlation with the inflammatory process and an inverse relationship with haemoglobin count, it has been shown that the levels of PM10 were connected with a decrease in haemoglobin concentration and erythrocyte counts but an increase in leucocyte level [23]. Furthermore, [18] shown that an increase in PM2.5 exposure was linked to a decrease in haemoglobin in elderly subjects. Other volatile pollutants have been connected to an increase in pro-inflammatory cytokines and a decrease in haemoglobin and other haematological indicators [24].

The inflammatory response caused by particulate matter and other pollutants has an impact on physiological parameters [26]. These effects are practically fleeting because they generate a surge in inflammatory markers and C-reactive protein levels [26]. It has been established that outdoor air pollution causes red cell haemolysis, which in turn causes anemia. Three mineral particles that were adsorbed following exposure to airborne pollution may be the origin of this incidence[27].

Conclusion

According to our findings, there were discrepancies across the groups and none of the groups fell within the boundaries of what is considered medically appropriate, which suggests that there may be an underlying inflammatory condition that may be brought on by work exposure. Due to increased demand, erythropoietic stimulation strengthens the precursor red cell's capacity to produce new cell lines. If red cell indices are noticeably high or low, they may indicate one type of pathological condition or another. Although some of the participants' peculiarities were depicted in this work, pathological abnormality was not specifically mentioned. VEE poses a grave danger to humanity and as a result there is prompt need to gradually adopt other forms of renewable energy to replace this current one. This study has provided insight to the general impact of vehicular emissions on erythrocyte level and red cell indices of occupationally exposed subjects. Although this research produced some reasonable results, more research is still required.

Ethical Approval and Consent

The ethical approval "number: HA-737/30" was obtained from the Health Planning, Research and Statistics unit of Edo State Ministry of Health, Benin City, Nigeria and informed consent was obtained from each participant. A letter introducing me as a student researcher "*introduction to subjects*/*to whom it may concern*" was provided by the Department of Medical Laboratory Science, University of Benin.

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