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Effects of chronic exposure to paint fumes among artisans in Lagos State, Nigeria

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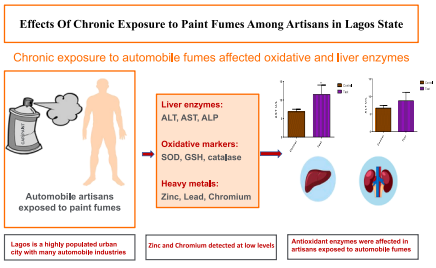
ABSTRACT

The chronic effects of automobile paint fumes and their volatile organic constituents form detrimental air pollution with hazardous consequences especially to paint industrial workers and the population within the vicinity. This study investigated the chronic effects of exposure to paint fumes in Mushin area of Lagos, Nigeria. Fifty artisans employed in automobile painting industries were compared with 50 control group whose work does not expose them to paint fumes. Five milliliters blood was collected and used for assessment of hematological and biochemical parameters. This was compared in artisan and unexposed control group and p value of < 0.05 indicates significant difference. In artisans, kidney function analysis showed a significant decrease in potassium (3.63 ± 0.1012 mEq/L) compared to healthy control (4.26 ± 0.1699 mEq/L, $p = 0.0049$), as well as bicarbonate ion concentration (23.89 ± 0.3795 vs 26.40 ± 0.3578 mmol/L respectively, $p = 0.0011$), however, a significant increase in creatinine level was recorded in artisans than control group (1.140 ± 0.1075 vs 0.76 ± 0.03578 mg/dL, $p = 0.03$); which is an indicator of renal function impairment. AST and ALT levels were significantly higher in artisans (11.44 ± 0.8190 and 8.78 ± 0.7558 U/L) compared to control group (6.83 ± 0.3086 and 6.67 ± 0.3354 U/L), respectively ($p < 0.05$), while ALP levels were similar. For oxidative stress parameters - CAT, MDA and protein, there was a significant increase in artisans while the corresponding GSH and SOD activities decreased significantly ($p < 0.05$). The results showed similar Zinc and Chromium levels in both groups but Lead was not detected in any participant. The findings of this study indicate that chronic exposure to paint fumes among automobile painting artisans may impair renal function, liver function and induce oxidative damages. Creating awareness of potential dangers and recommending use of personal protective equipment among automobile painting artisans can further decrease their exposure.



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1. Introduction

Nigeria is a rapidly industrializing country and automobile industry is one of the major emerging economic sectors [1]. Both small- and large-scale automobile service workshops make up the automobile industry in Nigeria. The automobile paints utilized by the automobile technicians and artisans, is composed of substances including volatile organic compounds (VOCs), hydrocarbons, etc., with notable toxicological concern to the body. Paint used in interior and exterior decoration are dissolved in potentially harmful solvents can generate free radicals that can lead to oxidative stress [2]. Exposure to these toxicants has been reported to cause minor or severe health conditions including headaches, birth defects, cancers, organ damage, central nervous system effects like loss of memory, confusion, seizures, allergic, neuropsychological, respiratory and other effects [3–14]; these harmful effects manifest when the paint fume is inhaled in poor ventilated areas. Go to:

In industrial areas, persons within these vicinities are exposed, on a long-term to these hazardous paint fumes and other air pollutants either directly through jobs, home refurbishing, or through indirect contact within the environment. Short-term and long-term exposure to the paint fumes can affect human health in a myriad of ways ranging from moderate to severe. In Denmark, a neurological condition induced by paint exposure is referred to as '*painter's dementia*' [15,16], lung problems and fatal cases in factory workers who used nanoparticles in paint have also been documented [16]. To reduce exposure to paint fumes and automobile pollutants, personal protective equipment (PPE) is recommended to be worn by the artisans, however, there are reports of low utilization of personal protective equipment among the auto-technicians at their service workshops in Nigeria, and this has been attributed to poor enforcement of the legislation by regulatory authorities [17]. Small-scale automobile service workshops constitute a larger portion of automobile industry especially in south-west Nigeria, where many roadside automobile technicians work in makeshift outdoor workshops along major roads within the urban cities [18]; this increases environmental pollution and risk of exposure to automobile fumes in these areas. Hence this study evaluated chronic exposure to heavy metal levels through paint fumes, and the effects on oxidative stress, and hematological parameters in artisan workers in Lagos, Nigeria.

2. Materials and methods

2.1. Study site and subject recruitment

This study was carried out at Mushin LGA of Lagos, Nigeria, in artisans employed in automobile painting workshops, across different age groups and varying employment durations. The study followed ethical guidelines for carrying out human research and the study protocol was approved by the College of Medicine, University of Lagos Health Research Ethics Committee (CMULHREC) with approval number CM/HREC/08/16/049. Age-matched individuals who are not automobile artisans were recruited as control. A total of 189 artisans were visited but 50 artisans from 33 workshops who agreed to participate and gave written informed consent were enrolled for the study. Fifty (50) non-artisan were recruited as control, the control participants were randomly selected from students in the Department of Biochemistry, University of Lagos, Nigeria, who gave written informed consent. Artisans were randomly recruited from different small-scale automobile workshops within the locality, they gave written informed consent, only participants who consented to the study were recruited. Any subject who declined was not coerced or induced to participate. A total of 100; 50 artisans and 50 consenting non-artisans were randomly selected for all the assays. General questions on diet, health of the participants, drug use, or knowledge of underlying disease condition were asked.

2.2. Exclusion criteria

Subjects were excluded from participating in the study if they are not automobile painters or artisans working in automobile painting workshop within the study area. Other artisans in workshops that did not engage in painting activities were excluded from the study. Participants who are below the age of 18 or older than 40 years were excluded from the study. Persons with known chronic medical conditions were not recruited.

2.3. Inclusion criteria

Subjects were included in the study if they consented to participate in the study, and if they are automobile painters and artisans working in automobile painting industry within the locality. Participants who are within ages 18 – 40 years were included in the study, only active and healthy individuals were recruited.

2.4. Sample Size

This was calculated using the online sample size calculator (available at http://www.raosoft.com/samplesize.html). Five percent error margin and 95% confidence interval (95% CI) was used. The sample size obtained was greater than population size, then finite population correction was used and the recommended sample size obtained was 50 subjects each (case and control group).

2.5. Sample collection and processing

A total of 100 samples were collected for the study (50 from artisans and 50 from control group). Five milliliters of blood were obtained by venous blood collection by a phlebotomist for all laboratory investigations so as to minimize any possible risk. The samples were kept in EDTA bottles for hematological parameters and was centrifuged at 2500 rpm for 10 min to obtain plasma, biochemical parameters were estimated using standardized test kits.

2.6. Determination of electrolytes, urea and creatinine levels

2.6.1. Bicarbonate ion (HCO_3^-)

1 ml of prepared bicarbonate reagent was pipetted into three arrays of test tubes labeled as blank, standard, and sample, then 10 μL of distilled water was pipetted into the blank labeled test-tube, 10 μL of bicarbonate standard was pipetted into the standard labeled test-tube, and 10 μL of plasma was pipetted into the sample labeled test tube. The setup was mixed gently and allowed to stand for 10 min at 37 °C. This was read on a spectrophotometer at 340 nm wavelength against water blank.

2.6.2. Chloride ion (Cl^-)

1 ml of chloride reagent was pipetted into three arrays of test tubes (blank, standard, and sample), 10 μL of chloride standard was pipetted into the standard labeled test tube, 10 μL of plasma was pipetted into the sample labeled test tube. The setup was mixed gently and allowed to stand for 3 min at 37 °C. This was then read on a spectrophotometer at 480 nm wavelength against reagent blank. Chloride concentration was calculated as:

$$\text{Chloride concentration (mEq/L)} = \frac{\text{Abs(sample)}}{\text{Abs(standard)}} \times \text{Standard conc·, (100mEq/L)}$$

2.6.3. Potassium ion (K^+)

1 ml of potassium reagent is pipetted into three arrays of test tubes (blank, standard, and sample), 10 μL of potassium standard is pipette into the standard labeled test tube, 10 μL of plasma was pipetted into the

sample labeled test tube. The setup was mixed gently and allowed to stand for 3 min at 37 °C. This was then read on a spectrophotometer at 500 nm wavelength against reagent blank. Potassium concentration was calculated as:

$$\text{Potassium concentration (mEq/L)} = \frac{\text{Abs(sample)}}{\text{Abs(standard)}} \times \text{Standard conc·, (4mEq/L)}$$

2.6.4. Sodium ion (Na^+)

For filtrate preparation, 1.0 ml of filtrate reagent was pipetted into three test tubes labeled blank, standard, and sample, 50 μL of standard was added into tubes labeled standard, 50 μL of samples was added into tubes labeled plasma, and 50 μL distilled water to the blank. The test tubes were shaking vigorously and mix continuously for 3 min. This was then centrifuged at high speed (1500× g) for 10 min and the supernatant fluids were collected into separately labeled test tubes (blank, standard, and plasma). For color development, 1.0 ml acid reagent was added to each tube, 50 μL supernatants was added in each respective tubes, then 50 μL of the color reagent was added to all tubes and mixed. The mixture was read using a spectrophotometer at absorbance of 550 nm, distilled water was used to blank. Sodium ion concentration was calculated as: $\text{Sodium concentration (mEq/L)} = \frac{\text{Abs of Blank} - \text{Abs of sample}}{\text{Abs of Blank} - \text{Abs of standard}} \times \text{Standard conc., (150mEq/L).}$

2.6.5. Urea

The assay was determined by using the Berthelot urease colorimetric method, 1 ml of prepared urease reagent was pipetted into three arrays of test tubes labeled as blank, standard, and sample, then 10 μL of distilled water was pipetted into the blank labeled test-tube, 10 μL of standard was pipetted into the standard labeled test-tube, and 10 μL of plasma was pipetted into the sample labeled test tube. The setup was mixed gently and allowed to stand for 10 min at 37 °C. This was read on a spectrophotometer at 580 nm wavelength against water blank.

2.6.6. Creatinine

For plasma creatinine concentration, 1 ml of plasma and 1 ml of creatinine standard were deproteonized using 1.0 ml of trichloroacetic acid respectively. The reaction was centrifuged at 4000 rpm for 10 min. The supernatant was then reacted with picric acid in an alkaline medium to form creatinine picrate. The reaction is allowed to stand for 2 min and the absorbance of the samples was read against reagent blanks at 492 nm wavelength.

2.7. Liver function tests

Plasma aspartate aminotransferase (AST): determination of AST in plasma, followed the method of Reitman & Frankel [19].

Alanine aminotransferase (ALT): determination of ALT in plasma followed the method of Reitman & Frankel [19].

Alkaline phosphatase (ALP): determination of ALP in plasma followed the established method of Roy, 1970 [20].

2.8. Antioxidant enzymes assay

Superoxide dismutase (SOD) activity: Superoxide dismutase activity was determined [21] by adding mixture (3 ml) containing 2.95 ml of 0.05 M sodium carbonate buffer, pH 10.2, 0.02 ml of plasma and 0.03 ml of epinephrine in 0.005 N HCl was used to initiate the reaction. The reference cuvette contained 2.95 ml buffer, 0.03 ml of substrate (epinephrine) and 0.02 ml of water. Enzyme activity was calculated by measuring the change in absorbance at 480 nm for 5 min $\sum = 4020\ M^{-1}cm^{-1}$.

Catalase activity determination (CAT): Catalase activity was determined colorimetrically at 620 nm and expressed as μmoles of H_2O_2 consumed/min/mg protein at 25 °C. The reaction mixture (1.5 ml) contained 1.0 ml of 0.01 M phosphate buffer (pH 7.0), 0.1 ml of tissue homogenate and 0.4 ml of 2 M H_2O_2. The reaction was stopped by the addition of 2.0 ml of dichromate-acetic acid reagent (5% potassium dichromate and glacial acetic acid were mixed in 1:3 ratio).

$\sum = 40\ M^{-1}cm^{-1}$.

Reduced glutathione determination (GSH): The reduced glutathione (GSH) content was estimated using standard method [22]. To the plasma, 10% TCA was added and centrifuged, 1.0 ml of supernatant was treated with 0.5 ml of Elman's reagent (19.8 mg of 5,5-dithiobisnitro benzoic acid (DTNB) in 100 ml of 0.1% sodium nitrate) and 3.0 ml of phosphate buffer (0.2 M, pH 8.0). The absorbance was read at 412 nm. $\sum = 1.34 \times 10^4\ M^{-1}cm^{-1}$.

Malondialdehyde (MDA) activities (Lipid peroxidation): 1.0 ml of the plasma was added to 2 ml of (1:1:1 ratio) TCA-TBA-HCl reagent (thiobarbituric acid 0.37%, 0.24 N HCl and 15% TCA) tricarboxylic acid- thiobarbituric acid-hydrochloric acid reagent boiled at 100 °C for 15 min, and allowed to cool. Flocculent materials were removed by centrifuging at 3000 rpm for 10 min. The supernatant was removed and the absorbance read at 532 nm against a blank. MDA was calculated using the molar extinction coefficient for MDATBA- complex of $1.56 \times 10^5\ M^{-1}cm^{-1}$.

Protein concentration: This was determined using biuret method [23] and Bovine Serum Albumin (BSA) as standard.

2.9. Determination of heavy metals levels

The plasma was first digested with nitric acid and perchloric acid, aliquots from the digest were used for Zinc, Lead and Chromium content determination, and their concentration were measured by Atomic absorption spectrophotometer [24].

2.10. Acid digest

For minerals analysis, 1 ml of plasma was placed into a 100 ml digestion flask and 10 ml nitric acid (HNO_3) was added, the flask was placed in a dark place overnight. After the overnight digest, 5 ml perchloric acid ($HClO_4$) was added. The mixture was heated at 50 °C for 15 min and gradually increased to 200 °C, this was done until white dense fumes of $HClO_4$ disappeared. The content was allowed to cool and filtered using Wattmann filter paper (# 2), thereafter it was diluted with distilled water.

2.11. Atomic absorption spectrophotometer

For the analysis of Zinc, Lead and Chromium, a Beckmann atomic absorption spectrophotometer model 1233 (USA) with hollow cathode lamp was used following the manufacturer's instructions. The hollow cathode lamps wavelengths for Zinc 0.7 nm, and Lead 0.7 nm, were used as a light source with lamp currents set at 10 mA for Lead and, 20 mA for Zinc. The gas used was acetylene with 20 Pa pressure and 45 Pa air Pressure. The instrument was calibrated with standard solutions and the samples were introduced using capillary tube.

2.12. Statistical analysis

Data was expressed as mean ± sem, and $p < 0.05$ denoted a significant difference in all comparisons and T-test were carried out to determine the significant effect that paint fumes imposed on the blood parameters.

3. Results

3.1. Socio-demographic data and baseline characteristics of study participants

The participants were from Igbo and Yoruba tribes, with age range

between 18 and 40 years and were all males. The artisans were composed of 50 different artisans including painters, panel beaters and rewire recruited from 33 automobile painting workshops. The weight and height of the participants was mean ± sem; 74.1 ± 1.9 (range: 50 −109 kg) and 169 ± 0.95 cm (range: 150 – 181 cm), respectively. The artisans indicated they were uneducated unlike the control group that were educated students, they worked for 8 h or more everyday and have been employed for at least 1 year in the industry. The questionnaire enquired about the level of protection during work, lifestyle habits of the artisans such as fruit or alcohol consumption. The level of protection adopted by the artisans was mostly wearing of overalls however, face shields, painting masks which reduces inhalation of the paint fumes was rarely worn by the artisans. Most of the artisans (>90%) indicated they do not smoke, except vert few who smoke and consume moderate alcohol. Majority of the artisans consume fruits but do not do regular medical check-ups.

3.2. Electrolyte levels in study participants

Table 1 shows the minerals and ions in the artisan and healthy control groups. Chronic effects of paint fumes on the kidney function showed significantly lower potassium in artisans (3.63 ± 0.1012 mEq/L) compared to healthy control with value of 4.26 ± 0.1699 mEq/L, $p = 0.0049$. Similarly, bicarbonate level of 23.89 ± 0.3795 mmol/L in artisans was significantly lower compared to the control group with 26.40 ± 0.3578 mmol/L ($p = 0.0011$). Creatinine was significantly higher in artisans than control group (1.140 ± 0.1075 vs 0.76 ± 0.03578 mg/dL, $p = 0.03$). However, Sodium, Chloride and Urea levels were similar between artisan versus control group.

3.3. Liver function tests

Table 2 shows the liver function enzyme levels in the study participants, ALP level was similar in artisan and control group but, AST and ALT levels were significantly higher in artisans (11.44 ± 0.8190 and 8.78 ± 0.7558 U/L) compared to control group with 6.83 ± 0.3086 and 6.67 ± 0.3354 U/L, respectively ($p < 0.05$).

3.4. Antioxidant parameters

Table 3 shows the antioxidant parameters measured in the study participants, Catalase (CAT), MDA and protein levels was significantly higher in the artisan versus control group ($p < 0.05$) while SOD (70.52 ± 10.54) and GSH levels (4.69 ± 0.957 μmol/min/ml/mgpro) were five to six times higher in control group ($p < 0.05$).

3.5. Heavy metals

Heavy blood metal concentrations for Lead (Pb), Zinc (Zn), and Chromium (Cr) are shown in Table 4. Lead was absent in both groups whereas Zn was slightly higher but not significantly different in artisans compared to control group. Chromium levels were also similar in the two groups ($p > 0.05$).

Table 1
Effects of paint fumes on the kidney function in artisans and healthy control.

Parameter	Healthy control (mean ± sem)	Artisan (mean ± sem)	P value
Potassium [K^+] (mEq/L)	4.26 ± 0.1699	3.63 ± 0.1012	0.0049*
Sodium [Na^+] (mEq/L)	142.60 ± 0.6708	140.70 ± 0.9898	0.2199
Bicarbonate (mmol/L)	26.40 ± 0.3578	23.89 ± 0.3795	0.0011*
Chloride [Cl^-] (mEq/L)	100.60 ± 0.7245	102.40 ± 0.6514	0.1062
Urea (mg/dl)	23.60 ± 1.002	31.44 ± 2.596	0.0598
Creatinine (mg/dl)	0.76 ± 0.03578	1.14 ± 0.1075	0.0307*

Values are expressed as mean ± sem (standard error of mean), *values are statistically significant at $p < 0.05$.

Table 2
Liver function test in artisans and healthy control.

Parameter	Healthy control (mean ± sem)	Artisan (mean ± sem)	P value
ALP (U/L)	24.17 ± 0.8721	27.22 ± 1.632	0.2306
AST (U/L)	6.830 ± 0.3086	11.44 ± 0.8190	0.002 *
ALT (U/L)	6.670 ± 0.3354	8.780 ± 0.7558	0.001 *

Values are expressed as mean ± sem (standard error of mean), *values are statistically significant at $p < 0.05$.

Table 3
Antioxidant parameters in artisans and healthy control.

Parameter	Healthy control (mean ± sem)	Artisan (mean ± sem)	P value
CAT (μmol/min/ml/mgpro)	12.03 ± 0.581	27.18 ± 2.919	0.0033*
SOD (μmol/min/ml/mgpro)	4.69 ± 0.957	0.82 ± 0.1613	• 0.0001*
MDA (μmol/min/ml/mgpro)	7.73 ± 1.118	20.11 ± 0.8095	• 0.0001*
GSH (μmol/min/ml/mgpro)	70.52 ± 10.240	11.02 ± 1.3950	• 0.0001*
PROTEIN	23.10 ± 3.265	54.05 ± 0.9835	• 0.0001*

Values are expressed as mean ± sem (standard error of mean), *values are statistically significant at $p < 0.05$ * .

Table 4
Blood heavy metal concentration in artisans and healthy control.

Parameter	Healthy control (mean ± sem)	Artisan (mean ± sem)	P value
Pb (ppm)	0.00	0.00	–
Zn (ppm)	1.790 ± 0.3309	3.290 ± 0.3854	0.0675
Cr (ppm)	0.0300 ± 0.01342	0.0600 ± 0.01897	0.3169

Values are expressed in mean ± sem (standard error of mean), values are not statistically significant at $p < 0.05$.

4. Discussion

The most common source of toxin exposure associated with paints is mostly through occupational exposure [25]. Occupational hazard has many health consequences due to exposure to chemicals such as heavy metals, biological agents, and prolonged ergonomic conditions. Persons working in the painting industry are often exposed to this occupational hazard however, since it is expedient to work to receive dividends which is used to provide basic amenities, reducing exposure to heavy metals is of great importance. The occupation and work environment can determine the quality of life, job satisfaction, or have detrimental effects on one's health, hence the need for improving occupational health and safety [26–28]. Exposure to heavy metals is an underlying cause of some diseases today but reducing exposure to these toxicants is often overlooked at some workplace which is responsible for many health consequences [29]. One of the prominent occupational hazards in most developed and developing countries is the insidious exposure to chemicals [30], these have mainly been associated with the automobile painting industries, enormous production of automobiles and the diverse chemical composition of modern automobile aerosol paints. The automobile industry workers are a group of people that are exposed to heavy metals daily, directly or indirectly through painting, welding, grinding or cutting of auto body parts. Other organic chemicals

contained in the paints such as primers, thinners, cleaners used to dissolve the paints can also be harmful [31].

Our study showed no difference in the plasma levels of sodium, chloride and urea detected in artisans compared non-artisans. This shows that exposure to the automobile paint fumes does not interfere with the plasma levels of sodium and chloride in the artisans. These two electrolytes maintain fluid and normal electrolyte balance in the body and plays roles in nerves and muscle functioning [32]. However, a significant reduction in potassium and bicarbonate levels was detected in the artisans. This suggest there is interference of inhaled heavy metals particles with these intracellular electrolytes which are important in the functioning of the body, whereas elevated urea and creatinine; which are byproducts indicates impairment of renal function [33,34]. Higher levels of creatinine observed in the artisans suggests that the exposure to the paint fumes had a significant effect on the kidney and its functionality in the artisans. This could be as a result of the regular exposure to these organic solvents for which artisans reported exposure of at least 3 times weekly. Numerous evidences have shown that one of the major organs liable to paint toxicity is the liver [35]. The liver plays important role in metabolic processes for the normal operation of cellular activities. Damages to certain tissues and organs lead to the increase in plasma levels of specific biochemical parameters, as a result of secretion from the damaged tissues and organs [36].

We determined liver injury using hepato-cellular injury markers - alanine amino transferase (ALT), alkaline phosphatase (ALP), and aspartate amino transferase (AST) [37], and found significant increase in plasma AST, ALT, but not ALP activities in the artisans relative to the control group [Table 2]. Significant elevation of the plasma ALT and AST and slight elevation of ALP activities in the artisans may indicate initiation of hepatotoxic effect from exposure to the toxic paint fumes reaching the liver which induced slightly moderate damage as at the time of sampling for the study. Previous studies have reported similar findings showing increase in the activity of AST as a consequence from paint exposure [38–40]. This significant increase shows that the paint fumes impose hepatic damages in the artisans. The result from the present study is in agreement with reports that a rise in AST activity is often accompanied by increased ALT level [41]; ALT plays important roles in the bioconversion of amino acids to ketoacids while increased ALP in the artisans suggests liver injury [42]. Alkaline-phosphatase (ALP) functions to transports metabolites across cell membranes, it is also involved in protein synthesis and other cell functioning. Increased ALP may be indicative of altered transport of metabolites in hepato-toxic conditions [43]. Although, there are controversial data regarding the toxicity of painting products, this might be explained on the basis of differences in exposure pattern, acute or chronic exposure, or other factors that contribute to changes in liver function tests [44].

Studies have shown that exposure to heavy metals and toxicants can induce oxidative damage via lipid peroxidation, reduced antioxidant enzymes such as GSH, SOD and GSH-Px [45–50]. Our study shows similarly that there was a significant increase in MDA but reduction of SOD and GSH enzymes in artisans, unlike in the non-artisans, which is a widely known biomarker of cell membrane damage [51–53]. This indicates lipid peroxidation due to buildup of reactive oxygen species as a consequence of ROS attack on the unsaturated fatty acids or suppression of antioxidant system during the course of paint exposure [54]. Reduced glutathione (GSH) is a copious thiol that has an important function of protecting intracellular components from toxic compounds [55] while SOD and CAT functions as cellular antioxidants.

SOD converts superoxide anion to hydrogen peroxide which is eventually removed by catalase [56]. Low activity of superoxide dismutase may be as a result of the over-consumption of these antioxidant enzymes due to continuous use by toxicants [57]. In artisans, there was a 2-fold increase in protein levels in artisans compared to the control. Free radicals such as superoxide, peroxyl radical, hydroxyl radical, nitrogen oxide, induce oxidative stress by degradation of proteins, nucleic acids, etc [58–61]. They interact with cysteine containing compounds like albumin in plasma to form complexes producing thiols, hence the albumin interaction with these radicals results in proinflammatory activation leading to ischemia modified albumin oxidative stress, these can also result in renal damage from proximal tubule cell cytotoxicity [58–61]. Several chemicals are used globally, some of which are present in paints and petroleum products handled by painters in their various work places, and some are reported to contain heavy metals like Zinc, Lead and Chromium, which have deleterious effects on biological system [62,63].

Chronic exposure of automobile artisans to these metals through inhalation can result in respiratory, nervous and other chronic diseases [62,63]. In the current study, Lead was absent in the blood samples of the artisans, this might be attributed to the prohibited use of Lead as a component in recently manufactured automobile paints, as it was banned in 1978 [64]. An insignificant increase in the level of zinc and chromium was recorded in artisans compared to control group who exposure to these metals in their work field. The findings on heavy metals are in contrast with some studies which reported significant increases in Lead, Chromium and other metals [64,65]. The level of protection adopted by the artisans was mostly wearing of workshop overalls however, face shields or painting masks which reduces inhalation of the paint fumes was rarely worn by the artisans.

Limitations for the study includes that the control group which are student biochemists, may have been previously engaged in practical activities which could pose limited level of chemical hazard from exposure to bench reagents used for practical exercise by the students. The nature and components of the paints were not analysed. Urine levels of metabolites of organic solvents such as: Hippuric acid, Phenol, Trichloroacetic acid, Hexanedione, etc were not determined, which can be used to confirm the bio-indicators of exposure to paint fumes. In conclusion, the chronic exposure to paint fumes among automobile painting artisans may impair renal function, liver function and induce oxidative stress, and toxicity might have been due to hazardous components of paints such as heavy metals and poly-aromatic hydrocarbon used as solvents in the paints. The results of this study suggest moderate toxicity due to heavy metal exposure hence the need for implementing use of protective equipment among automobile painters and artisans, to reduce their exposure and creating awareness of potential dangers.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests that could have appeared to influence the work reported in this paper.

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