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Cardiotoxic Effects of Car Paint Fumes Exposure on Cardiac Tissues of Male Wistar Rats

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Abstract

Background: *The majority of the people are exposed to hazardous paint fumes at some point in their lifetime either through their occupation, home, car refurbishing or through any contact. This current study examined the impact of automobile paint fumes on male abino wistar rats.* ***Method:*** *Forty matured male Wistar rats weighing 180-300g were used for the study. The rats were divided into five group A-E of eight rats per group. Group A served as control while Group B-E served as the experimental animals. Group A received only feed and clean water for 14 days. Group B-D were exposed to 40ml of Clear coat, Thinner and Hardener fume each via inhalation in addition to feed and water for 14 days. Group E: CTH (Clear + thinner + hardener)-exposed groups were exposed to 13.5ml of Clear coat + 13.5ml of thinner and 1 3.3 ml of Hardener fume via inhalation in addition to feeding and water for the 14 days. At the end of two-weeks experiment, animals were sacrificed by cervical dislocation. Blood and heart tissues were harvested and used for biochemical and histopathological study.* ***Result:*** *Exposure to car paint fumes caused cardiac damage evidenced by significant alteration in cardiac activity (Troponin-I, CK-MB, AST, LDH), lipid profile (HDL, LDL, CHOL, TAG) and antioxidant status (SOD, CAT, GSH, MDA, NO, XO) as compared to control rats.* ***Conclusion:*** *The results of these findings thus inferred that exposure to paint fume may be toxic to the heart.*

Keywords: *Paint fumes, Thinner, Hardener, Clear coat, Lipid profile.*

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1. Introduction

Cardiovascular disease (CVDs) is one of the leading causes of morbidity and mortality worldwide, accounting for 20 % of all deaths. World Health Organization (WHO) report augured that approximately 30 million people will die from CVDs by 2030 [1], [2]. More than 80% of deaths from CVDs occur in developing countries, with the majority being below 70 years of age [3]. CVDs majorly arise from vascular dysfunction, leading to organ damage. Major risk factors in vascular impairment include atherosclerosis, thrombosis, and high blood pressure (B.P.). In addition, fundamental risk factors for CVDs such as smoking, unhealthy diet, diabetes mellitus, hyperlipidemia, dyslipidemia, physically inactive, low-density lipoprotein cholesterol (LDL) high levels, suppressed levels of high-density lipoprotein cholesterol (HDL), and hypertension have identified as significant contributors to cardiovascular health risks [4], [5]. Apart from these fundamental risk factors, environmental toxic substances, including paint components, may affect novel risk pathways such as inflammation and oxidative stress [6]. Thus, ecological toxicants should be considered significant risk factors for cardiovascular disease [7].

Human beings have been exposed to hazardous paint fume in their entire lifetime in one form or the other, either through their jobs, home refurbishing, or use as a psychoactive substance. Acute or long-term exposure to paint fumes affects us in diverse ways that are more critical than ever imagined [8]. Paint is any mixture of liquid or mastic substance applied to a substrate in a thin layer to form a solid film [9]. It is used to beautify and protect objects on which it is applied [10]. It is virtually applied to most things in our surroundings (such as cars, houses, furniture, etc.); hence, we have been exposed to it constantly [10].

Paint consists of two parts, namely the solid part and the solvent part. Both are harmful to humans and animals and cause environmental hazards. The substantial amount contains pigments, extenders, fillers, resins, and minor additives/solvents, primarily organic and inorganic derivatives. Our household's paint comprises potentially harmful chemicals such as solvents and volatile organic compounds [11]. As most paint substances are toxic to humans, they constitute health hazard to the users. The paint is a mixture of solvents, resin, pigment, and other additives, and the spraying activities create paint mists that circulate the volatile matter to the surrounding air [12]. Also, the pressure and nebulizer of the spray gun influence the concentration of airborne contaminants produced in vapors. Therefore, it is perilous to human health when inhaled [13]. The solvent's hazards will be augmented due to the composition of paint additives such as diisocyanates, lead, and other heavy metals, which are known carcinogens and require a sully of clean air for the painter to dissipate their contact [13].

Automobile paint, mainly used nowadays, has been reported to consist of resin/latex/binders (in the form of clear coat/hardener). This content makes the car glossy and also protects the vehicle from damage/U.V. rays, pigments, and solvents (in the form of thinner) which reduces the thickness of oil-based paints [14]. Automobile paint fumes contain volatile organic compounds such as toluene, xylene, acetone, formaldehyde, benzene, and Isocyanates. Most volatile organic compounds (such as xylene, benzene, ethylbenzene, styrene, toluene, etc.) found in paints are anthropogenic. Because of its volatility, exposure to paint fumes causes hepatic damage [15] nausea [16], sensory irritation [17], dementia [18], respiratory disorder, allergic and immune effects, central nervous system (CNS) disorder or neurocognitive disorder which eventually lead to death [19], [20], [21], [22].

It is common knowledge that we get exposed to paint fume daily in our ambient environment or through our occupations. However, there are no well-documented effects of such exposure on cardiac tissue. Therefore, the effects of paint fume exposure on the cardiac tissue in adult male Wistar rats was investigated in this study.

2. Materials and Methods

2.1 Animals

Forty (40) male albino (Wistar) rats with an average body weight of 180-300g were obtained from the animal house of the Anatomy Department of my institution. The rats were housed in a well-ventilated plastic cage and fed with a standard pellet diet and *water ad libitum*. The rats were allowed to acclimate to the laboratory condition (temperature 25ºC and environment 12h light-dark cycle) for two weeks before exposure to paint fumes.

2.2 Treatment

The acclimatized animals were divided into five (5) groups as follows:

Group A: Control group; consisted of 8 rats each which received only feed and clean water for 28 days.

Group B: Clear exposed group; consists of 8 rats each which were exposed to 40ml of Clear coat fume via inhalation in addition to feeding and water for 14 days.

Group C: Thinner exposed group; consists of 8 rats each which were exposed to 40ml of Thinner fume via inhalation in addition to feeding and water for 14 days.

Group D: Hardener exposed group; consists of 8 rats, each exposed to 40ml of Hardener fume via inhalation in addition to feeding and water for 14 days.

Group E: CTH (Clear + thinner + hardener)-exposed groups; of 8 rats, each exposed to 13.5ml of Clear coat + 13.5ml of thinner and 13.3 ml of Hardener fume via inhalation in addition to feeding and water for the 14 days.

Animals were exposed to paint fumes (5ml/animal/5min) with a total of 40ml for 8 rats (23).

2.3 Chemicals

The car paint product (LATICO CLEAR, LATICO FAST HARDENER, FIDEA DIAMOND THINNER) were bought at a shop in Kuye, Ogbomoso, Oyo State. Different types of car paint components; Thinner, Hardener, and Clear Coat. The test material was stored and protected from direct sunlight. After administration, it was ensured that the cover was well tightened to avoid drying of the component.

2.4 Exposure to Paint Fumes

The route of administration was inhalation using a spraying machine (Model: GX-160) with a spray gun to spray the fume of the car paints daily for 14 days.

2.5 Blood Collection and Preparation of Tissue Homogenate

At the end of the experiment, rats were weighed and euthanized on the 15th day of their exposure to the fume. Animals were anesthetized with 1ml of ketamine through the intra-peritoneal region. The thoracic region was cut open to expose the heart. Blood samples were obtained from the animal by cardiac puncture into the well-labeled heparinized bottle and centrifuged at 3000rpm for 15 minutes to get serum for biochemical assay. The heart tissue was quickly removed

and rinsed in 1.15% KCl, dried, weighed, and homogenized in an equal volume of chilled 10mM Tris/HCl buffer pH 7.4 and 0.25M sucrose solution. The homogenate was centrifuged at 12,000 r.p.m for 60 minutes to obtain the supernatant used for biochemical assays.

3. Biochemical Analysis

3. 1 Estimation of Cardiac Biochemical Markers

The activity of Troponin I and creatinine kinase isoenzyme (CK-MB) was determined by the methods of Giuliani *et al*. [24] and Szasz *et al.* [25] respectively. In addition, lactate dehydrogenase (LDH) activity and aspartate aminotransferase (AST) activity in the heart tissues were evaluated by routine enzymatic methods using Randox Commercial Kits.

3.2 Estimation of Cardiac Lipid Profile

Total cholesterol, triglyceride, and HDL cholesterol were determined using Randox Commercial Kits. The chylomicrons' very-low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) serum are precipitated by phosphotungstic acid and magnesium ions. Therefore, LDL was calculated from the results of Total cholesterol, triglyceride, and High-density lipoprotein.

3.3 Estimation of Cardiac Antioxidant Markers

The activity of superoxide dismutase (SOD) in the heart was determined by the method of Misra and Fridovich [26] and modified by Kakkar *et al.* [27]. Catalase activity was estimated by the process of Aebi [28]. The level of lipid peroxidation as malondialdehyde (MDA) and reduced glutathione (GSH) concentration was determined based on the principle of Varshney and Kale [29] and Beutler et al. [30], respectively. Nitric oxide (NO) and xanthine oxidase (X.O.) activities were determined using the method of Tarpey et al. [31] and Shintani [32].

3.4 Histological Analysis

The hearts were removed, rinsed immediately with saline, and then fixed in 10% buffered formalin. The hearts stored in 10% buffered formalin were embedded in paraffin, section cut at 5µm, and stained with hematoxylin and eosin. These sections were examined under a light microscope for histological changes.

3.5 Statistical Analysis

Data obtained were analysed on GraphPad Prism for windows (Versions 5.01, GraphPad Software, Inc.). Statistical comparison was done using a one-way analysis of variance (ANOVA) followed by a Student t-test using. The data are expressed as mean ± standard error of the mean (Mean ± SEM). The p-value <0.05 was accepted as statistically significant.

4. Results

4.1 Estimation of Body Weight

There is significant decrease in body weight of rats exposed to clear coat, thinner, hardner and its mixture (CL+TN+HD) when compared to control rats at $p<0.05$ (Fig. 1).

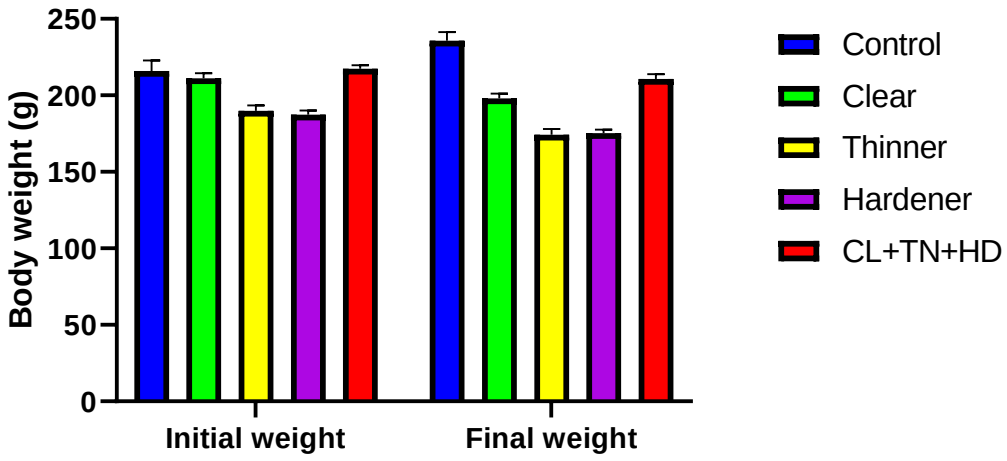
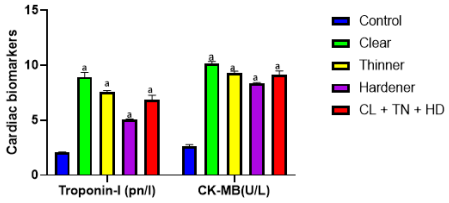


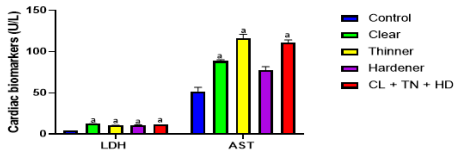
Fig. 1. Effects of car paint fumes on body weight of rats.

4.2 Estimation of Cardiac Biochemical Markers

The effects of exposure to different car paint fumes on the cardiac enzyme are summarized in Fig. 2. Exposure to a clear coat, thinner, hardener, and their mixture significantly increased troponin-I, creatinine kinase isoenzyme (CK-MB), and lactate dehydrogenase (LDH) activities when compared to control rats ($p<0.05$ in each case). Also, the clear coat, thinner, and the mixture of paint fumes significantly elevated Aspartate aminotransferase (AST) activity ($p<0.05$) whereas exposure to hardener paint fume caused a non-significant change in the level of AST activity ($p>0.05$) when compared to control rats.



(A)



(B)

Fig. 2. Effect of car paint fumes on troponin-I and creatinine kinase, CK-MB (A); aspartate transaminase, AST and Lactate dehydrogenase, LDH (B)

4.3 Estimation of Lipid Profile

The effects of exposure to different car paint fumes on lipid profile are depicted in Fig. 3. Exposure to various car paint fumes such as clear coat, thinner, hardener and their mixture produced significant increase in Triglyceride (TAG), Low-density lipoprotein (LDL), Total cholesterol (CHOL) with a corresponding decrease in High-density lipoprotein (HDL) when compared to control rats ($p< 0.05$ in each case).

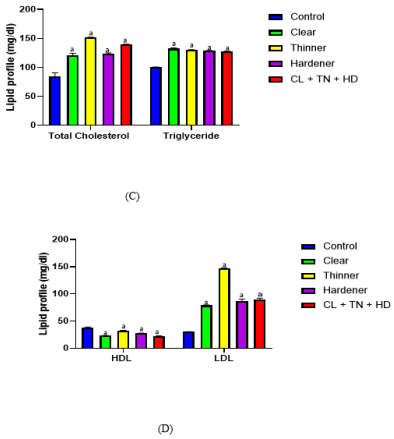


Fig. 3. Effect of car paint fumes on triglyceride, TAG, and total cholesterol, CHOL (C); high-density lipoprotein, HDL and low-density lipoprotein, LDL (D).

4.4 Estimation of Cardiac Antioxidant Markers

The effects of exposure to different car paint fumes on the cardiac antioxidant marker are depicted in Fig. 4. Exposure to various car paint fumes such as clear coat, thinner, hardener and their mixture caused a significant decrease in superoxide dismutase activities (SOD), catalase activities (CAT) and glutathione concentration (GSH) with a corresponding increase in malondialdehyde concentration (MDA), nitric oxide (NO) and xanthine oxidase (X.O.) activities when compared to control rats ($p< 0.05$ in each case).

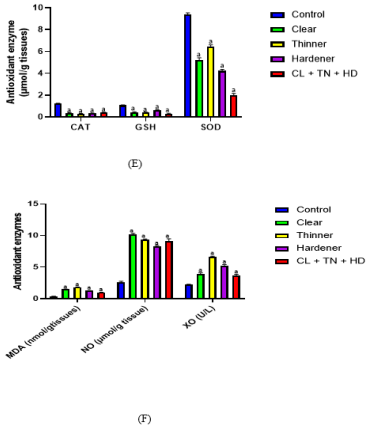


Fig. 4. Effect of car paint fumes on catalase, CAT, Glutathione, GSH and Superoxide dismutase, SOD (E) Malondialdehyde, MDA, Nitric oxide, NO and Xanthine oxidase, X.O. (F)

4.5 Histopathological Study

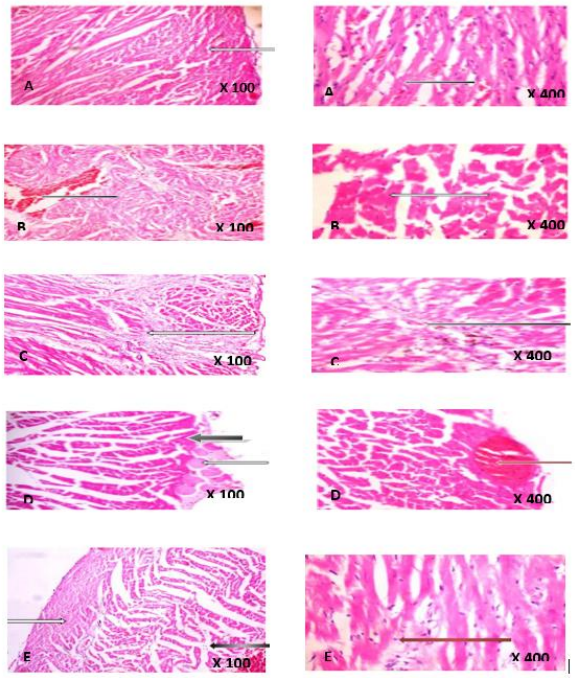


Fig. 5. Plate 1: Effect of paint fume exposure on the heart using adult male wistar rats.
A: Control B: Clear coat C: Thinner D: Hardner E: Mixture (CL+TN+HD)

Fig. 5 represent the histological micrographs of the diiferent experimental groups.

Histopathology examination of the myocardium of the control group animals showed typical architecture of the myocardium, normal epicardial (white arrow) and normal myocardial layer seen (black arrow), no hemorrhage nor any pathological lesion seen (Plate 1A). The photomicrograph of the heart section of Clear coat exposed, normal heart tissue, there is normal epicardial (white arrow) and myocardial layer seen with mild congestion (black arrow) (Plate 1B). However, the photomicrograph of a heart section of Thinner-exposed rats stained with Haematoxylin and Eosin revealed heart tissue with severe fibrosis within the epicardial (white arrow) and myocardium (black arrow) (Plate 1C). The heart section of Hardener-exposed rats revealed several thromboemboli (white arrow) and mild vascular congestion (black arrow) with area of moderate fibrosis (red arrow) (Plate 1D). The photomicrograph of the heart section of Mixture (Clear coat+ thinner + hardener)-exposed rats showed moderately normal epicardial (white arrow) but the myocardial layer seen (black arrow) show mild area of fibrosis (red arrow) as shown in Plate 1E, indicating the toxic effect of the mixture of paint component on the heart of rats .

5. Discussion

Humans are concomitantly exposed to paint fumes whose toxicity profiles have not been delineated in our household or occupation. Despite the abundance of knowledge regarding CVDs, its prevalence continues to rise. The present study demonstrates the cardiotoxic effect of car paint fumes such as clear coat, hardener, thinner, and their mixture in male

Wistar rats. This is evidenced by compromised cardiac markers, reduced antioxidant defense system, and increased lipid peroxidation and leakage of myocytes injury marker enzymes from the heart.

The myocardium contains many cardiac marker enzymes and proteins, such as creatine kinase (isoforms), lactate dehydrogenase (LDH), and troponins which move out of the cardiomyocytes and are released into the bloodstream owing to elevated lipid peroxidation. Thus, these enzymes/proteins diagnose myocardial injury [33]. Troponin-I has been considered a reliable, sensitive, and more specific biomarker of choice for detecting cardiac injury than CK-MB [34]. The cardiac troponin released in the bloodstream may not be from necrosis only but also reversible myocardial injury [34]. Serum creatine kinase activity is a more sensitive indicator in the preliminary stage of myocardial ischemia. At the same time, peak rises in lactate dehydrogenase are roughly proportional to the degree of damage to the myocardial tissue [35]. Elevations in the levels of these enzymes are connected with certain types of heart damage, such as myocardial infarction, myocarditis, and heart failure [36]. The increase in serum CK-MB, LDH, and AST activities in this study resulted from this phenomenon.

Alteration in the lipid profile in the car paint fume-exposed rats as shown in this finding is evidenced by a significant elevation in serum total cholesterol (T.C.), triglyceride (T.G.), and low-density lipoprotein (LDL) levels, coupled with a corresponding reduction in the high-density lipoprotein (HDL) level. These changes observed in the lipid profile of the car paint-exposed rats are an indication of abnormalities in lipoprotein metabolism. Lipoproteins are involved in various body metabolic processes such as immune reactions, coagulation, and tissue repair. Oxidative alteration of lipoproteins, particularly the LDL, followed by the suppression of lipoprotein antioxidants, especially Vitamin E, has been linked to causing accumulation of cholesterol and enhancing the susceptibility of atherosclerosis [37]. Generally, elevated levels of T.G., T.C., and LDL levels and a concomitant reduction in HDL indicate an increased risk of vulnerability to cardiovascular disease.

The present findings also revealed a significant elevation of malondialdehyde (MDA) level in rats exposed to paint fumes. Lipid peroxidation (LPO) products such as malondialdehyde (MDA) are highly reactive and exhibit prominent biological effects, depending upon their concentration. Hence, results in selective alteration in cell signaling mechanisms such as enhancing protein and DNA damage and promoting elevated lipid peroxides and aldehydes levels as observed in disease conditions like atherosclerosis, ischemia-reperfusion, heart failure, Alzheimer's disease, rheumatic arthritis, cancer, and other immunological disorders [38].

Oxidative stress is the major contributor to cardiac injury. Antioxidant enzymes are generally believed to be the first line of defense in response to oxidative challenges to protect cellular integrity and the pathogenesis of various degenerative diseases [39]. During oxidative stress, depending on the extent of alteration in the normal redox state within the cells, there is an overwhelming effect on the enzymatic antioxidant status, notably SOD, CAT, GST, and GPx, as well as the nonenzymatic antioxidant, GSH, due to excessive production of free radicals. Oxidative stress has played a significant role in the pathogenesis of diseases such as inflammatory diseases, alcoholism, smoking-related disorders, ischemic diseases, and many other [40]-[42]. The general reduction observed in the status of enzymatic and nonenzymatic antioxidants in the heart homogenates in car paint fume-exposed rats in this study indicated a net suppression of the total antioxidant capacity in the tissue. Car paint thinner may have caused more problems since the

entire base is made up of organic solvent that causes more oxidative stress. These results agree with previous findings of Künzli and Tager [43].

Nitric Oxide (NO) may play a significant role in myocardial injury and hypertrophy [44]. Xanthine oxidase (XO), on the other hand, catalyzes the oxidation of xanthine to uric acid, this process generates excessive reactive oxygen species (ROS) which play a significant role in atherogenesis [45]. This study demonstrated that serum NO and X.O. levels were significantly increased in car paint fume exposed rats. This result indicates that car paint fume components may cause tissue damage and inflammation of internal organs when exposed to them in high concentration for a long time.

Histopatological findings also showed that exposure to car paint fumes of clear coat, thinner, hardner and their mixture altered the histoarchitecture of the heart tissue evidenced by mild congestion, severe fibrosis and thromboembolic area of the epicardial and myocardial tissues as compared to that of the control rats.

Several epidemiological studies have demonstrated a statistical relationship between exposure to gaseous air contaminant as contained in paint fumes and increased rates of cardiovascular death [46], [47]. Exposure to heavy metals (a component of paint fumes) has been shown to be linked with sub-acute and long-term health risks such as hypertension and neurodegenerative disorder [48]. Existing literature shows that prolonged exposure to organic solvents embedded in paint fumes is significantly linked with a higher prevalence of high blood pressure [49]. Similarly, Uboh et al. [50] reported a significantly elevated atherogenic index of plasma (AIP) in animals exposed to organic solvent vapors. Thus, exposure to organic solvents present in paint components may be associated with the risk of atherosclerosis and hence cardiovascular disease [51].

Isocyanates are highly reactive compounds and may be in form of monoisocyanate, diisocyanate or polyisocyanate, both in vapour and particulate form, which directly or indirectly have effect on human exposed to it. Generally, polyisocyanates are trimers of hexamethylene diisocyanate (HDI). Human exposure to polyisocyanates can cause skin and eye irritation, respiratory sensitization, asthma and reduced lung function [52]. A dysfunction of the respiratory system may lead to cardiac abnormalities as the cardiovascular system complement the work of the respiratory system. Isocyanate, which have been reported to be present in Clear coat and hardner have been documented to cause short and long term adverse effects on health ranging from multi organ damage to central nervous system defect [53]-[55]. Other organic compound which are prominent in car paint solvent such as thinner include benzene, toluene, xylene, acetone, formaldehyde etc. Benzene have been reported to cause hematopoietic diseases/blood disorder such as leukemia, aplastic anemia, dysplatic bone marrow condition in automobile painters [56], [57]. A circulatory dysfunction such as blood disorder will affect the functionality of the heart tissue. Toluene on the other hand have been documented to causes toxic effects in any organ system, including the liver, heart, kidneys, and bone marrow, although the main toxic impact is on the nervous system [58]. Acute effects of xylene exposure have been revealed to cause central nervous depressant (CND), headache, nausea, dizziness, vomiting, and irritation of the eyes, nose, throat, and skin. Repeated exposure to liquid xylene may result in dermatitis [59]. All these above effect may lead to the loss of cardiac inegrity and cardiovascular function.

In summary, exposure to car paint fumes (40mls) of clear coat, thinner, hardner and their mixture through the use of spray gun caused cardiac injury/toxicity. This was associated with pronounced oxidative stress, compromised lipid profile and cardiac tissue damage in rats model with car paint thinner having the most prominent cardiotoxic damage [60]. This is in tanderm with the previous finding of Ogbuowelu et al. [50]. The results of these findings thus inferred that exposure to paint fume may be toxic to the heart tissue.

6. Recommendations

The following recommendations are made in light of the findings of this study:

- Further research should be conducted to ascertain the mechanism of toxicity of car paint fumes on the cardiovascular system.
- Further research should be conducted on possible ameliorative effect of car paint fume in cardiac tissue when treated with medicinal plants.

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