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Original article

Effects of Subacute Exposure to Diesel and Burnt Tire Fumes on the Liver of Wistar Rats

Josiah Soipiriala Hart[1], Oghenefego Michael Adheke[1], Doris Kasarachi Ogbuokiri[2], Osah Martins Onwuka[3], Datiyama Isah Samiala[4]

1. *Department of Anatomy, Faculty of Basic Medical Sciences, University of Port Harcourt, Port Harcourt, Rivers State, Nigeria*
2. *Department of Anatomy, Faculty of Basic Medical Sciences, Nnamdi Azikiwe University, Nnewi Campus, Nnewi, Nigeria*
3. *Department of Human Physiology, Faculty of Basic Medical Sciences, Gregory University, Uturu, Abia State, Nigeria*
4. *Department of Biomedical Technology, School of Science Laboratory Technology, University of Port Harcourt, Port Harcourt, Rivers State, Nigeria*

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Abstract

Background: Concerns associated with the uncontrolled combustion of petroleum-related products for local agricultural benefits have emerged in most parts of Nigeria, thereby posing health issues for humans living around affected areas. **Aim**: The present study examined the subacute effects of exposure to diesel and burnt tire fumes on the livers of Wistar rats. **Methods**: Thirty adult male Wistar rats were divided into three groups of ten each. The first group was the control group (exposed to clean air); the second group was exposed to Nigerian D6-diesel fumes and the final group was exposed to burnt tire fumes. The exposures were conducted in well-ventilated chambers for 4 hours daily for 14 days. After exposure, the rats were sacrificed, and blood samples were collected for analysis. **Results**: Compared with those in the control group, the concentrations of aspartate aminotransferase, alanine transaminase, total protein, albumin, and total bilirubin in the exposed groups significantly increased. There were substantial decreases in the levels of reduced glutathione, catalase, and superoxide dismutase; and increases in the malondialdehyde levels in both the diesel-exposed and burnt tire groups. Significant reductions in the packed cell volume, hemoglobin concentration, and red blood cell count, as well as increases in white blood cell and platelet counts, were detected in the exposed groups. Mild histopathological changes in the liver were observed in the exposed groups. **Conclusion**: Subacute exposure to both diesel and burnt tire fumes affected proper liver function by causing significant fluctuations in the expression levels of antioxidant enzymes, liver profile markers, and hematological profiles of Wistar rats.

I. Background

Air pollution caused by soot usually results from the incomplete combustion of petroleum-related products. People are exposed to soot through the deposition of these substances in water, air, or soil, which can result in several health problems (Akpoghelie et al., 2021; Kieta et al., 2022). The detrimental consequences of soot pollution for human health have made it a major environmental problem,

DOI:
*Corresponding author: **Oghenefego Michael Adheke**. Department of Anatomy, Faculty of Basic Medical Sciences, College of Health Sciences, University of Port Harcourt, Choba, Port Harcourt, Nigeria.
Email address: mikeadheke@gmail.com

especially in the southern parts of Nigeria, which are usually impacted by artisan refining of petroleum products. Over fifty years of exploration and production of oil and gas have resulted in significant environmental deterioration in Nigeria due to gas flaring and oil spills (Okeke et al., 2016; Usiobaifo et al., 2023; Olukaejire et al., 2024).

In Nigeria, the regular use of automobile waste for bush burning is a common practice that poses significant risks to human health and the environment (Ifeoluwa, 2019; Abaje et al., 2020; Okedere et al., 2021). This practice stems from the disposition of end-of-life vehicles (ELVs) and their components, such as tires, plastics, and other materials, through burning in open spaces (Babayemi et al., 2016; Ogbu et al., 2022). ELVs are a significant source of various pollutants, including heavy metals, polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds (VOCs), and particulate matter (Raboni et al., 2015). When these materials are incinerated in open fires, they release toxic substances into the air, soil, and water, leading to a range of adverse health effects, such as asthma, bronchitis, and chronic obstructive pulmonary disease (COPD). Long-term exposure to these pollutants has also been linked to cardiovascular diseases, lung cancer, and other serious health issues (Lee et al., 2014; Manisalidis et al., 2020; Konduracka and Rostoff, 2022).

The state known as oxidative stress is an imbalance between the body's antioxidant defenses and the generation of reactive oxygen species (ROS) (Pisoschi and Pop, 2015; Afzal et al., 2023). Soot pollution is a complex mixture of gases and other particulate matter that can cause oxidative stress in different body tissues and organs. ROS can be produced as a result of interactions between biological systems and soot pollutants, which initiates a series of events that aggravate inflammation and damage cells (Niranjan and Thakur, 2017; Aryal et al., 2021; Tang et al., 2024). Particulate matter, for example, can enter the bloodstream and reach deep into the lungs, causing oxidative stress in lung epithelial cells. These contaminants may cause oxidative damage and the release of mediators that promote inflammation in lung tissues (Valacchi et al., 2020; Albano et al., 2022). The unfavourable cardiovascular effects associated with air pollution are mostly mediated by oxidative stress-induced alterations in lipid metabolism, in addition to the impacts of inflammation that are experienced throughout bodily systems (Yadav, 2015; Rao et al., 2018; Gangwar et al., 2020).

Research has further indicated that short-term exposure to various forms of soot pollution may lead to aberrant increases in the concentrations of specific blood cellular components and may also stimulate oxidative stress-related inflammatory reactions in blood cells, which could ultimately result in reduced bone marrow function (Adeyemi and Isukuru, 2020; Obasi et al., 2023; Suku et al., 2023). Different forms of soot inhalation have been shown to lead to pathological conditions of the liver, such as necrosis, an increase in inflammatory cells, and steatosis (Shvedova et al., 2013; Niranjan and Thakur, 2017). The present study aimed to examine the effects of subacute exposure to diesel and burnt tire fumes on liver function in Wistar rats by analysing their serum biochemical and hematological parameters.

II. Materials and Methods

Experimental animal procedures and handling techniques were performed in accordance with the directives of the Animal Use and Care Committee of the National Veterinary Research Institute, Vom, Nigeria, and ethical approval was obtained from the Research Ethics Committee of the University of Port Harcourt (with number UPH/CEREMAD/REC/MM78/049).

II.1 Study design:

This research utilized a randomized controlled trial (RCT) study design. Thirty adult male Wistar rats (weighing 175-210 g) were chosen for this experiment. The rats were divided into three experimental groups of ten (10) each. In line with Festing (2006), the resource equation method, which is based on degrees of freedom (E), was used to determine a suitable rat sample size for the study on the basis of two criteria; the number of groups (k) and the number (n) of rats per group. Therefore, E was calculated as follows: $kn - k = (30 - 3) = 27$. Since the value of E was greater than 20, the use of ten (10) rats per group was considered sufficient to determine the sample size.

All the rats were acclimatized in the experimental inhalation exposure chambers for approximately 6 hours per day for 2

days before the day of exposure. The first group was regarded as the control group (not exposed to any soot source); the second group was exposed to Nigerian D6-diesel fumes; and the final group was exposed to burnt tire fumes. The control group of rats was housed in a separate well-ventilated room free from any form of diesel or burnt tire fumes to serve as a baseline for comparison. For the diesel-exposed group, a 1000 mL beaker was filled with 500 mL of D6-diesel solution in line with Owumi et al. (2021) and dropped inside the inhalation chamber, whereas for the burnt tire-exposed group, a cut piece of damaged tire weighing 26 g inside a steel container was burned daily and kept inside the chamber. All exposures were conducted in well-ventilated chambers for 4 hours daily over 14 days. The exposure chambers were designed to allow the volatile components of the petroleum products to evaporate and saturate the environment.

II. 2 Methods

Analysis of Serum Biochemical and Haematological Indices

After exposure, the rats were sacrificed, and blood samples were collected in EDTA bottles. Immediately after collection, blood centrifugation was performed via a refrigerated centrifuge at 4000 rpm for 10 min to obtain plasma, which was subsequently analysed in the laboratory to assess the levels of serum biochemical and hematological indices. The determination of both the serum alanine transaminase (ALT) and aspartate transaminase (AST) activities was conducted via the Reitman and Frankel (1957) method. Additionally, the serum total protein concentration was determined via the biuret reaction method (Gornall et al., 1949), while the serum ALB concentration was determined via a dye-binding technique that uses the ability of albumin to form a stable blue-colored complex with bromocresol green dye, and the serum bilirubin concentration was determined via the dimethylsulfoxide method (Tietz et al., 1994). To validate the biochemical assays for measuring serum alanine aminotransferase (ALT), a sensitivity of 0.5 U/L was demonstrated for ALT, with an average recovery of 95%. No significant cross-reactivity was observed with other serum enzymes. The intra-assay coefficient of variation for ALT was 5.5%, whereas the interassay coefficient of variation (CV) was 6.2%. The limit of detection (LOD) for ALT was 0.3 U/L, and the limit of quantification (LOQ) was 0.5 U/L.

Catalase (CAT) activity was analysed via the Sinha (1972) method, superoxide dismutase (SOD) activity was assayed according to Misra and Fridovich (1972), and reduced glutathione (GSH) activity was assayed according to Adams et al. (1983), while the lipid peroxidation marker, malondialdehyde (MDA) was assessed via the reactive constituents of malonylurea (Buege and Aust, 1978).

After the exposure, three rats from each group were sacrificed, and blood sample were collected. Blood was collected in EDTA bottles to prevent clotting and taken immediately to the laboratory to undergo centrifugation. The collected blood samples were centrifuged at 4000 rpm for ten minutes in a refrigerated centrifuge to obtain the plasma from the cellular components of the blood. With the aid of the BC-3200 Auto Hematology Analyser, blood samples were analysed for the following hematological indices: white blood cell (WBC) count; red blood cell (RBC) count; platelet (PLT) count; packed cell volume (PCV); and hemoglobin (Hb) concentration.

Histopathological analysis

Liver samples were removed from sacrificed rats, and tissue processing procedures were carried out in accordance with Ogunsola et al. (2019). With the aid of an Accu-Scope 3000 digital microscope, photomicrographs were produced to examine histopathology.

II.3 Data analysis:

The data were analysed via the Statistical Package for the Social Sciences (SPSS) IBM version 23.0 and Microsoft Excel 2019 edition. The values are expressed as the means ± standard deviations via descriptive statistics. One-way analysis of variance (ANOVA) was used to compare the differences in the biochemical and hematological parameters between the groups, followed by Fischer's least significant difference (LSD) post hoc test. The confidence interval was set at 95%; therefore, $p<0.05$ was considered significant.

III. Results

As shown in Table 1, the AST levels of the control group (43.60 U/L) were lower than those of the diesel-exposed groups (48.20 U/L) but were significantly lower than those of the burnt tire-exposed groups (50.48 U/L). ALT levels in the control group (12.12 U/L) were significantly lower than those in the diesel-exposed and burnt tire-exposed groups (15.14 U/L and 15.40 U/L, respectively). The total protein concentration in the control group (63.60 g/L) was significantly lower than that in the diesel-exposed and burnt tire-exposed groups (70.00 g/L and 76.20 g/L, respectively). The albumin concentration in the control group (38.80 g/L) was significantly lower than that in the diesel-exposed and burnt tire-exposed groups (45.40 g/L and 48.40 g/L, respectively). TB levels in the control group (43.60 mg/dl) were significantly lower than those in the diesel-exposed groups (48.20 mg/dl) and were lower than those in the burnt tire-exposed groups (50.48 mg/dl).

As shown in Table 2, there were significant decreases in the activities of GSH, CAT, and SOD in the diesel-exposed groups (1.42 nmol/g, 3.99 nmol/g, and 0.31 nmol/g respectively) compared with those in the control group. On the other hand, there were significant decreases in the levels of GSH; and CAT in the burnt tire-exposed groups (1.30 nmol/g; and 2.92 nmol/g), respectively. Additionally, the activities of MDA were significantly greater in the diesel-exposed and burnt tire-exposed groups (0.45 nmol/g and 0.37 nmol/g, respectively) than in the control group (0.35 nmol/g).

Finally, the results in Table 3 revealed significant reductions in PCV (35.60%; 33.60%), Hb concentrations (11.18 g/dL; 11.84 g/dL), and RBC counts in the diesel-exposed and istoburnt tire-exposed groups compared with those in the control group. Additionally, WBC counts were significantly greater in the diesel-exposed and burnt tire-exposed groups than in the control group, whereas PLT counts were significantly greater in the diesel-exposed and burnt tire-exposed groups than in the control group.

Histopathology:

In comparison with the control liver histoarchitecture, the liver parenchyma of rats exposed to diesel fumes appeared less intact while those that were exposed to burnt tire fumes depicts hepatocytes that were not well arranged, as well as mild signs of cellular degeneration.

Table 1: The mean values of serum liver profile markers

Group	AST (u/l)	ALT (u/l)	TP (g/l)	ALB (g/l)	TB (mg/dl)
Control	43.60±4.04	12.12±1.33	63.60±6.30	38.80±6.53	8.78±0.59
Diesel fumes	48.20±3.03	15.14±1.02*	70.00±8.12*	45.40±3.29*	9.26±0.40*
Burnt tire fumes	50.48±4.97*	15.40±0.74*	76.20±5.76*	48.40±4.88*	8.92±0.65

AST=aspartate aminotransferase, ALT=alanine transaminase, TP=total protein, ALB=albumin, TB=total bilirubin, *significant difference compared with the control group at $p<0.05$

Table 2: Mean values of levels of the serum oxidative stress markers

Group	GSH (nmol/g)	CAT (nmol/g)	SOD (nmol/g)	MDA (nmol/g)
Control	1.57±0.40	4.19±1.06	0.39±0.12	0.35±0.11
Diesel fumes	1.42±0.32*	3.99±0.82*	0.31±0.05*	0.45±0.02*
Burnt tire fumes	1.30±0.36*	2.92±1.36*	0.41±0.16	0.37±0.14*

GSH=Glutathione, CAT=catalase, SOD=Superoxide dismutase, MDA=Malondialdehyde, *significant difference compared with the control group at $p<0.05$

Table 3: The mean values of the hematological parameters

Parameters	Control	Diesel fumes	Burnt tire fumes
PCV (%)	38.80±4.60	35.60±2.55*	34.60±3.36*
Hb (g/dL)	12.96±1.53	11.18±0.16*	11.84±1.12*
RBC (x 10^6/µL)	5.88±0.75	4.84±0.11*	4.40±0.52*
WBC (x 10^3/µL)	8.70±1.76	10.08±2.01*	9.40±2.81*
PLT (x 10^9/L)	467.60±82.12	636.40±83.74*	506.80±91.20*

PCV=Packed cell volume, Hb=hemoglobin, RBC=Red blood cell, WBC=White blood cell, PLT=Platelet count, *significant difference compared with the control group at $p<0.05$

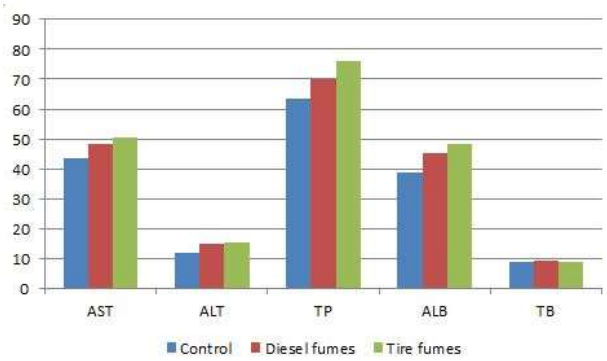

Figure 1. Serum liver biochemical profile markers

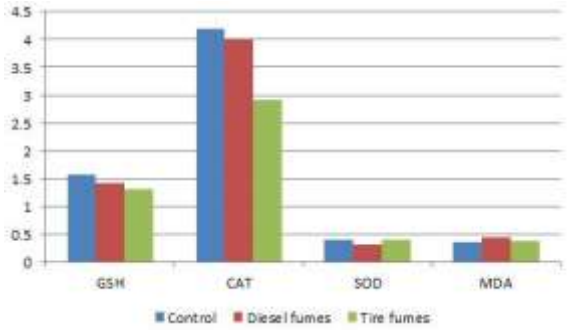

Figure 2 Serum oxidative stress markers

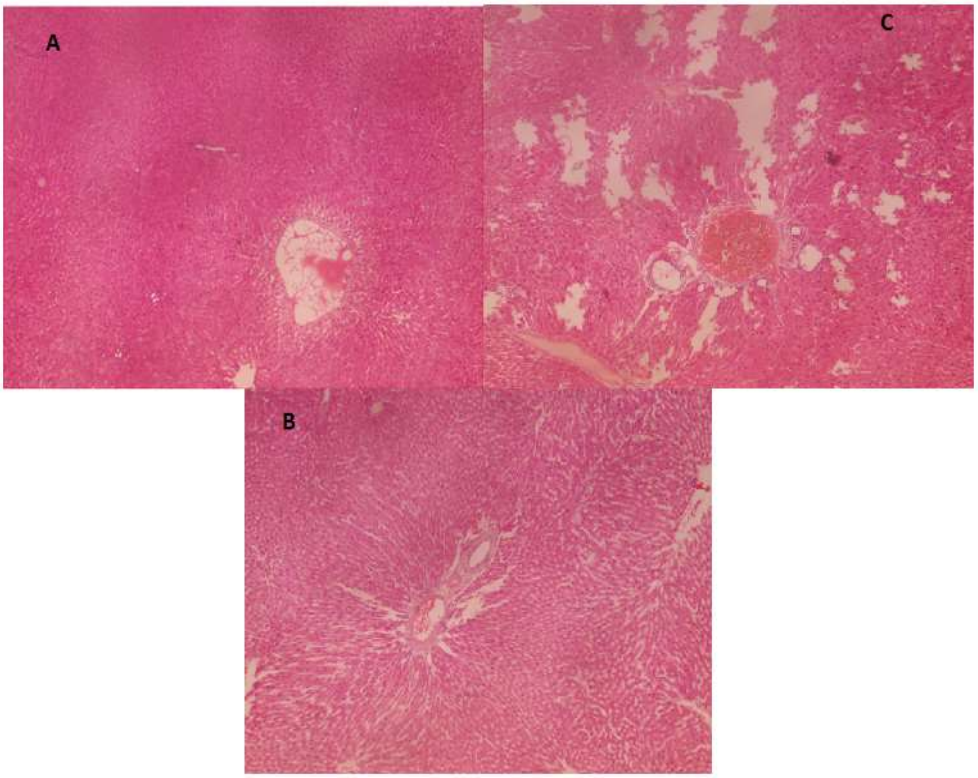

Figure 3. Photomicrograph of the liver. A (control) shows normal hepatoarchitecture with the presence of a central vein and hepatocytic cells. The liver parenchyma of B (diesel fumes) appears disranged, whereas that of C (burnt tire fumes) depicts a visible central vein, with hepatocytes not well arranged as well as mild signs of cellular degeneration.

IV. Discussion:

When a toxic gas is inhaled, the blood carries it to the heart, liver, and other related organs, where it can have negative consequences (Manisalidis et al., 2020; Sonwani et al., 2021). Reactive oxygen species and free radicals produced by harmful gases have the potential to upset an organism's hematological system and impair blood's capacity to maintain homeostasis (Aryal et al., 2021; Cortese-Krott, 2023). Tires contain a complex mixture of both natural and synthetic rubber compounds, including additives such as carbon black that are not typically found in petroleum products, and these toxic compounds can be introduced spontaneously into the atmosphere during combustion (Fazli and Rodrigue, 2020; Akbas and Yuhana, 2021). Furthermore, tires contain PAHs and VOCs from the breakdown of these rubber compounds and their additives, which could differ chemically from those of petroleum products such as petrol, diesel, and kerosene (Kazemi et al., 2023). The present study examined the comparative effects of subacute exposure to diesel and burnt tire fumes on liver function in Wistar rats through the analysis of their biochemical and hematological parameters. This is arguably the first study that has examined the influence of subacute inhalation of burnt tire fumes in Wistar rats.

Compared with the serum liver biochemical parameters of the control group, there were significant increases in the concentrations of aspartate aminotransferase (AST), alanine transaminase (ALT), total protein (TP), albumin (ALB), and total bilirubin (TB) among the exposed groups ($p < 0.05$). In line with a related study performed by Akinmoladun et al. (2021), similar findings were reported for the levels of AST, ALT, ALB, and TB, whereas Owumi et al. (2021) reported that AST levels were significantly increased in the rat groups that were exposed to subacute inhalation of the petroleum products that were studied. Since serum liver function biomarkers are frequently employed as indicators of liver health and function, abrupt elevations in these biomarkers following subacute toxic inhalation exposure may result in liver damage or future harm, depending on how long the exposure lasts (Tripathi and Tarrant, 2018). High blood levels of these enzymes result from inflammation or injury to the liver, which releases them into the circulation.

When inhaled harmful compounds such as petroleum products and other hydrocarbon-made gases are metabolized, free radicals and reactive oxygen species (ROS) can cause oxidative damage to cells. To prevent this, the antioxidant defense system is essential (Kehrer and Klotz, 2015; Leni et al., 2020). According to related studies, being around petroleum products can lower antioxidant levels, which increases oxidative damage. Disruption of the body's antioxidant defense system can make it more difficult for the body to repair oxidative damage and preserve cellular homeostasis, which increases the risk of illnesses linked to oxidative stress, including cancer, cardiovascular disease, and liver damage (Aryal et al., 2021; Martemucci et al., 2022; Jena et al., 2023; Tang et al., 2024). In the present study, as depicted in Table 2, there were significant decreases in the levels of reduced glutathione (GSH), catalase (CAT), and superoxide dismutase (SOD), whereas there were substantial increases in the levels of malondialdehyde (MDA) in both the diesel-exposed and burnt tire groups compared with those in the control group. Owumi et al. (2021) reported that the levels of SOD and GSH were reduced in rats ($p < 0.05$) exposed to petrol, kerosene, and diesel fumes, whereas Akinmoladun et al. (2021) reported that SOD levels decreased significantly in groups of rats exposed to liquefied natural gases. Additionally, Owagboriaye et al. (2018) previously reported similar findings to those of the present study, in which significant changes in the levels of antioxidants such as GSH, and CAT, as well as increased MDA levels, were observed upon exposure to gasoline fumes. There were significant reductions in the packed cell volume, hemoglobin concentration, and red blood cell count in the exposed groups compared with those in the control group, which is comparable to the findings of studies by Akpan et al. (2014), Isirima and Angalabiri-Owei (2014), Abubakar et al. (2015), Sani and Abdullahi (2019), Owumi et al. (2021), and Oriakpono and Enechukwu (2022). In this study, the PCV values in the diesel-exposed group were greater than those in burnt tire-exposed group, which could be explained by the differences in the toxicological constituents present in tires compared with those in diesel. Decreases in hemoglobin (Hb) and RBC levels were observed in this study for both exposed groups, with the burned tire-exposed group having

slightly lower values. These findings correlate with studies performed by Akpan et al. (2014), Abubakar et al. (2015), Akinmoladun et al. (2021) and Owumi et al. (2021). These findings indicate that alterations in the oxygen-carrying capacity of the blood could impart the overall health status of exposed rats. However, the present study revealed significant increases in white blood cell (WBC) and platelet (PLT) counts in the exposed groups compared with those in the control group. In correlation with studies performed by Akpan et al. (2014), Abubakar et al. (2015), Akinmoladun et al. (2021), and Owumi et al. (2021), the differences in WBC and PLT counts point to systemic changes in blood parameters that may have an impact on clotting mechanisms, oxygen transport, and immunological function. Finally, the normal architecture of the hepatic lobule was slightly disrupted in exposed rats, as were mild signs of cellular degeneration**.** The current study acknowledges that factors such as the small sample size of experimental animals, and the impact of fluctuating weather conditions during the research could limit the exposure rates of the animals to the fumes; however, the study emphasizes the potential health effects of short-term exposure to these fumes from an experimental point of view.

V. Conclusions:

On the basis of the findings of the present study, subacute exposure to both diesel and burnt tire fumes affected proper liver function by causing significant fluctuations in the expression levels of antioxidant enzymes, liver profile markers, and hematological profiles, as well as mild histopathological changes in the livers of Wistar rats.

Declarations

All authors declare that this research has not been published elsewhere and it is not under consideration for publication elsewhere.

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Ethical approval

Ethical approval was obtained from the Research Ethics Committee of the University of Port Harcourt (with number UPH/CEREMAD/REC/MM78/049).

Data availability statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Conflict of interest

There are no conflicts of interest among the authors.

References

Abaje, I. B., Bello, Y., & Ahmad, S. A. (2020). A review of air quality and concentrations of air pollutants in Nigeria. *Journal of Applied Sciences and Environmental Management, 24*(2), 373-379. https://doi.org/10.4314/jasem.v24i2.25

Abubakar, M. B., AbdullAh, W. Z., Sulaiman, S. A., & Ang, B. S. (2015). The effects of exposure to petrol vapours on growth, haematological parameters and oxidative markers in sprague-dawley male rats. *The Malaysian journal of medical sciences: MJMS*, *22*(1), 23.

Adams, J. D., Lauterburg, B. H., & Mitchell, J. R. (1983). Plasma glutathione and glutathione disulfide in the rat: regulation and response to oxidative stress. *Journal of Pharmacology and Experimental Therapeutics*, *227*(3), 749-754.

Adeyemi, O., & Isukuru, E. J. (2020). Toxicological Evaluation of Biodiesel Emission Particles on Growth and Haematological Properties of Albino Rat. *NISEB Journal*, *19*(2). https://doi.org/10.9734/ajrb/2020/v6i230114

Afzal, S., Abdul Manap, A. S., Attiq, A., Albokhadaim, I., Kandeel, M., & Alhojaily, S. M. (2023). From imbalance to impairment: the central role of reactive oxygen species in oxidative stress-induced disorders and therapeutic exploration. *Frontiers in Pharmacology*, *14*, 1269581. https://doi.org/10.3389/fphar.2023.1269581

Akbas, A., & Yuhana, N. Y. (2021). Recycling of rubber wastes as fuel and its additives. *Recycling*, *6*(4), 78. https://doi.org/10.3390/recycling6040078

Akinmoladun, A. C., Aladesanmi, O. O., Ojo, F. E., Bello, M., Taiwo, B. J., & Akindahunsi, A. A. (2021). Modifying influence of polyphenols on hematotoxicity,

cardiotoxicity, and hepatotoxicity induced by liquefied petroleum gas in rats. *Toxicology Research*, *10*(4), 751-760. https://doi.org/10.1093/toxres/tfab058

Akpan, K. V., Sogbanmu, T. O., & Otitoloju, A. A. (2014). Effects of volatile organic solvents inhalation on hematological and histological indices of *Mus musculus*. *Curr Advanc Environ Sci*, *2*(2), 46-51.

Akpoghelie, J. O., Ugbuku, U. A., & Esemedafe, U. J. (2021). A review of oil spill pollution and air quality in the niger delta: Causes, effects and control. *Journal of Chemical Society of Nigeria*, *46*(5). https://doi.org/10.46602/jcsn.v46i5.660

Albano, G. D., Montalbano, A. M., Gagliardo, R., Anzalone, G., & Profita, M. (2022). Impact of air pollution in airway diseases: role of the epithelial cells (cell models and biomarkers). *International Journal of Molecular Sciences*, *23*(5), 2799. https://doi.org/10.3390/ijms23052799

Aryal, A., Harmon, A. C., & Dugas, T. R. (2021). Particulate matter air pollutants and cardiovascular disease: Strategies for intervention. *Pharmacology & therapeutics*, *223*, 107890. https://doi.org/10.1016/j.pharmthera.2021.107890

Babayemi, J. O., Ogundiran, M. B., & Osibanjo, O. (2016). Overview of environmental hazards and health effects of pollution in developing countries: a case study of Nigeria. *Environmental Quality Management*, *26*(1), 51-71. https://doi.org/10.1002/tqem.21480

Buege, J.A., & Aust, S.D. (1978). Microsomal lipid peroxidation. *Methods Enzymology*. *52*, 302–310. https://doi.org/10.1016/s0076-6879(78)52032-6

Cortese-Krott, M. M. (2023). The reactive species interactome in red blood cells: oxidants, antioxidants, and molecular targets. *Antioxidants*, *12*(9), 1736. https://doi.org/10.3390/antiox12091736

Fazli, A., & Rodrigue, D. (2020). Recycling waste tires into ground tire rubber (GTR)/rubber compounds: A review. *Journal of Composites Science*, *4*(3), 103. https://doi.org/10.3390/jcs4030103

Festing, M. F. (2006). Design and statistical methods in studies using animal models of development. *Ilar Journal*, *47*(1), 5-14. https://doi.org/10.1093/ilar.47.1.5

Gangwar, R. S., Bevan, G. H., Palanivel, R., Das, L., & Rajagopalan, S. (2020). Oxidative stress pathways of air pollution mediated toxicity: Recent insights. *Redox biology*, *34*, 101545. https://doi.org/10.1016/j.redox.2020.101545

Gornall, A. G., Bardwall, C. J., David, M. M. (1949). Determination of serum proteins by means of the Biuret reaction. *Journal of Biology and Chemistry 177*(2), 751-766. https://doi.org/10.1016/s0021-9258(18)57021-6

Ifeoluwa, O. B. (2019). Harmful effects and management of indiscriminate solid waste disposal on human and its environment in Nigeria: a review. *Global journal of research and review*, *6*(1), 1-4.

Isirima, J. C., & Angalabiri-Owei, B. E. (2014). Haematologic and biochemical implications of inhalation of fumes of petroleum products. *African Journal of Cellular Pathology*, *2*(3), 40-47. https://doi.org/10.5897/ajcpath14.006

Jena, A. B., Samal, R. R., Bhol, N. K., & Duttaroy, A. K. (2023). Cellular Red-Ox system in health and disease: The latest update. *Biomedicine & Pharmacotherapy*, *162*, 114606. https://doi.org/10.1016/j.biopha.2023.114606

Kazemi, M., Parikhah Zarmehr, S., Yazdani, H., & Fini, E. (2023). Review and perspectives of end-of-life tires applications for fuel and products. *Energy & Fuels*, *37*(15), 10758-10774. https://doi.org/10.1021/acs.energyfuels.3c00459

Kehrer, J. P., & Klotz, L. O. (2015). Free radicals and related reactive species as mediators of tissue injury and disease: implications for health. *Critical reviews in toxicology*, *45*(9), 765-798. https://doi.org/10.3109/10408444.2015.1074159

Kieta, K. A., Owens, P. N., Petticrew, E. L., French, T. D., Koiter, A. J., & Rutherford, P. M. (2022). Polycyclic aromatic hydrocarbons in terrestrial and aquatic environments following wildfire: a

review. *Environmental Reviews*, *31*(1), 141-167. https://doi.org/10.1139/er-2022-0055

Konduracka, E., & Rostoff, P. (2022). Links between chronic exposure to outdoor air pollution and cardiovascular diseases: a review. *Environmental Chemistry Letters*, *20*(5), 2971-2988. https://doi.org/10.1007/s10311-022-01450-9

Lee, B. J., Kim, B., & Lee, K. (2014). Air pollution exposure and cardiovascular disease. *Toxicological Research*, 30(2), 71-75. https://doi.org/10.5487/tr.2014.30.2.071

Leni, Z., Künzi, L., & Geiser, M. (2020). Air pollution causing oxidative stress. *Current opinion in toxicology*, *20*, 1-8. https://doi.org/10.1016/j.cotox.2020.02.006

Manisalidis, I., Stavropoulou, E., Stavropoulos, A., & Bezirtzoglou, E. (2020). Environmental and health impacts of air pollution: a review. *Frontiers in public health*, *8*, 14. https://doi.org/10.3389/fpubh.2020.00014

Martemucci, G., Costagliola, C., Mariano, M., D'andrea, L., Napolitano, P., & D'Alessandro, A. G. (2022). Free radical properties, source and targets, antioxidant consumption and health. *Oxygen*, *2*(2), 48-78. https://doi.org/10.3390/oxygen2020006

Misra, H. P., & Fridovich, I. (1972). The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. *Journal of Biological chemistry*, *247*(10), 3170-3175. https://doi.org/10.1016/s0021-9258(19)45228-9

Niranjan, R., & Thakur, A. K. (2017). The toxicological mechanisms of environmental soot (black carbon) and carbon black: focus on oxidative stress and inflammatory pathways. *Frontiers in immunology*, *8*, 763. https://doi.org/10.3389/fimmu.2017.00763

Obasi, I. C., Ohaeri, O. C., Ijioma, S. N., Okoro, B. C., & Ugbogu, E. A. (2023). Inhalation of smoke from burning tire triggers oxidative stress and impairs liver and kidney functions in rats. *Comparative Clinical Pathology*, *32*(5), 837-846. https://doi.org/10.1007/s00580-023-03493-y

Ogbu, C. C., Twumasi, Y. A., Ning, Z. H., Attamah, G. N., Ezeaku, V. I., & Oladigbolu, O. I. (2022). Analysis of Forest Waste Management and Recycling Potential in Nigeria. *Natural Resources*, *13*(10), 191-205. https://doi.org/10.4236/nr.2022.1310013

Ogunsola, J. O., Oridupa, O. A., Awotunsin, K. O., & Saba, A. B. (2019). Chronic inhalation of 2,2-dichlorovinyl dimethyl phosphate (DDVP) induces organ pathology in the adult albino rats. *Eur. J. Anat*, *23*(3), 151-158.

Okedere, O. B., Elehinafe, F. B., Oyelami, S., & Ayeni, A. O. (2021). Drivers of anthropogenic air emissions in Nigeria-A review. *Heliyon*, *7*(3). https://doi.org/10.1016/j.heliyon.2021.e06398

Okeke, O. C., Emerue, U. C., Atama, E. O., & Ekere, J. T. (2016). Environmental problems of hydrocarbon exploration and production in Nigeria: An overview. *Inter. J. Environ. Res*, *2*(1), 29-42.

Olukaejire, S. J., Ifiora, C. C., Osaro, P. A., Osuji, L. C., & Hart, A. I. (2024). Petroleum Exploration in the Niger Delta Region and Implications for the Environment: A Review. *Journal of Energy Research and Reviews*, *16*(5), 19-29. https://doi.org/10.9734/jenrr/2024/v16i5350

Oriakpono, O. E., & Enechukwu, E. V. (2022). Biomarker response of albino rats (*Rattus norvegicus*) to generator fumes. *International Journal of Science and Research Archive*, *5*(2), 171-176. https://doi.org/10.30574/ijsra.2022.5.2.0071

Owagboriaye, F. O., Dedeke, G. A., Aladesida, A. A., Bamidele, J. A., & Olooto, W. E. (2018). Assessment of the effect of gasoline fume on stress hormones, antioxidant status and lipid peroxidation in albino rat. *Journal of king Saud University-science*, *30*(3), 393-399. https://doi.org/10.1016/j.jksus.2016.11.002

Owumi, S. E., Oladimeji, B. N., Elebiyo, T. C., & Arunsi, U. O. (2021). Combine effect of exposure to petrol, kerosene and diesel fumes: On hepatic oxidative stress and haematological function in rats. *Toxicology and industrial health*, *37*(6), 336-352. https://doi.org/10.1177/07482337211012498

Pisoschi, A. M., & Pop, A. (2015). The role of antioxidants in the chemistry of oxidative stress: A review. *European journal of medicinal chemistry*, *97*, 55-74. https://doi.org/10.1016/j.ejmech.2015.04.040

Raboni, M., Torretta, V., Urbini, G., & Viotti, P. (2015). Automotive shredder residue: a survey of the hazardous organic micro pollutants spectrum in landfill biogas. *Waste management & research*, *33*(1), 48-54. https://doi.org/10.1177/0734242x14559300

Rao, X., Zhong, J., Brook, R. D., & Rajagopalan, S. (2018). Effect of particulate matter air pollution on cardiovascular oxidative stress pathways. *Antioxidants & redox signalling*, *28*(9), 797-818. https://doi.org/10.1089/ars.2017.7394

Reitman, S., Frankel, S. (1957). A colorimetric method for determination of serum glutamate oxaloacetate and glutamic pyruvate transaminase. *American Journal of Clinical Pathology* *28*(1):56-58. https://doi.org/10.1093/ajcp/28.1.56

Sani, A., & Abdullahi, I. L. (2019). Effects of welding fumes on haematological parameters of male albino rats (*Rattus norvegicus*). *Biochemistry and Biophysics Reports*, *19*, 100651. https://doi.org/10.1016/j.bbrep.2019.100651

Shvedova, A. A., Yanamala, N., Murray, A. R., Kisin, E. R., Khaliullin, T., Hatfield, M. K., & Gavett, S. H. (2013). Oxidative stress, inflammatory biomarkers, and toxicity in mouse lung and liver after inhalation exposure to 100% biodiesel or petroleum diesel emissions. *Journal of Toxicology and Environmental Health, Part A*, *76*(15), 907-921. https://doi.org/10.1080/15287394.2013.825217

Sinha, A. K. (1972). Colorimetric assay of catalase. *Analytical biochemistry*, *47*(2), 389-394. https://doi.org/10.1016/0003-2697(72)90132-7

Sonwani, S., Madaan, S., Arora, J., Suryanarayan, S., Rangra, D., Mongia, N., & Saxena, P. (2021). Inhalation exposure to atmospheric nanoparticles and its associated impacts on human health: a review. *Frontiers in Sustainable Cities*, *3*, 690444. https://doi.org/10.3389/frsc.2021.690444

Suku, P. G., Ugwoha, E., Orikpete, O. F., & Ewim, D. R. E. (2023). Assessment of respiratory and reproductive impacts of artisanal refinery activities on male Albino Wistar rats: Implications for environmental health. *Bulletin of the National Research Centre*, *47*(1), 149. https://doi.org/10.1186/s42269-023-01121-x

Tang, R., Shang, J., Qiu, X., Gong, J., Xue, T., & Zhu, T. (2024). Origin, Structural Characteristics, and Health Effects of Atmospheric Soot Particles: A Review. *Current Pollution Reports*, 1-16. https://doi.org/10.1007/s40726-024-00307-9

Tietz, N. W., Prude, E. L., Sirgard-Anderson, O. (1994). In: Tietz Textbook of clinical chemistry. 2nd edition, W.B Saunders Company, London pp. 1354-1374. https://doi.org/10.1001/jama.1994.03520080086056

Tripathi, N. K., & Tarrant, J. M. (2018). Principles of clinical pathology. In *Toxicologic Pathology* (pp. 215-268). CRC Press. https://doi.org/10.1201/9780429504624-7

Usiobaifo, A. H., Chidiebere, A., Olusola, A. S., Dada, A. R., & Ukoje, N. L. (2023). Assessment of the Impact of Flared Gas and Oil Spilled on Human Health and Environmental Degradation: Evidence from the Niger Delta Region, Nigeria. *American Journal of Environmental Science and Engineering*, *7*(1), 5-16. https://doi.org/10.11648/j.ajese.20230701.12

Valacchi, G., Magnani, N., Woodby, B., Ferreira, S. M., & Evelson, P. (2020). Particulate matter induces tissue oxinflammation: from mechanism to damage. *Antioxidants & Redox Signalling*, *33*(4), 308-326. https://doi.org/10.1089/ars.2019.8015

Yadav, U. C. S. (2015). Oxidative stress-induced lipid peroxidation: Role in inflammation. *Free Radicals in Human Health and Disease*, 119-129. https://doi.org/10.1007/978-81-322-2035-0_9

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