



Article Info

Received: 20th March 2025

Revised: 30th June 2025

Accepted: 3rd July 2025

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Cite this: *CaJoST*, 2025, 2, 264-271

Effects of Gasoline Fume Inhalation on Oxidative Stress Biomarkers and Pulmonary Histopathology in Wistar Rats

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Exposure to volatile gasoline fumes is prevalent in petrol stations and congested urban areas, and it has been associated with oxidative stress and respiratory damage. This study investigates the effects of gasoline fume inhalation on oxidative stress biomarkers and pulmonary histoarchitecture in adult female Wistar rats. Twenty Wistar rats (130–210 g) were randomly assigned to four groups (n=5): a control group and three exposure groups subjected to gasoline fume inhalation for 4, 6, and 8 hours daily for 14 days in a controlled exposure chamber. Oxidative stress markers were assessed spectrophotometrically, while lung tissue alterations were evaluated histopathologically. Results showed a significant increase in malondialdehyde (MDA) levels ($P<0.05$) across all exposure groups, indicating enhanced lipid peroxidation. Conversely, superoxide dismutase (SOD) and catalase (CAT) activities were markedly reduced ($P<0.05$), suggesting compromised antioxidant defense. Histological examination revealed dose-dependent pulmonary damage, including alveolar shrinkage, inflammatory infiltrates, and bronchiolar epithelial degeneration. These findings highlight the toxic effects of gasoline fume inhalation, emphasizing the need for environmental regulations and protective measures to mitigate respiratory hazards in exposed populations.

Keywords: Gasoline fume, Oxidative Stress, Pulmonary Toxicity, Antioxidant Enzymes, Inhalation Exposure.

1. Introduction

The increasing reliance on fossil fuels has led to significant environmental and occupational health concerns, particularly due to exposure to volatile organic compounds (VOCs) in gasoline fumes. Gasoline also known as premium motor spirit (PMS) consists of several toxic hydrocarbons, including benzene, toluene, and xylene, which can vaporize readily into the atmosphere (El-Hagrasy *et al.*, 2025). Among these, benzene is classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC), with no established safe exposure limit, making even minimal exposure a potential health risk (IARC, 2022). Globally, approximately 3.6 billion gallons of gasoline vapors are released into the atmosphere annually, with nearly 40% of these emissions occurring during vehicle refueling and fuel dispensing (NIOSH, 2024).

Inhalation is the primary route of exposure to gasoline fumes, particularly among individuals engaged in fuel production, transportation, and retail services. Petrol station attendants, tanker drivers, and urban

commuters in densely populated regions are among the most vulnerable groups (Al-Barnawi, 2025). Acute exposure to high concentrations of gasoline vapors can cause dizziness, respiratory distress, and neurological impairments, while chronic exposure has been linked to systemic oxidative stress, which disrupts the balance between reactive oxygen species (ROS) and antioxidant defense mechanisms. Biomarkers such as malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and glutathione (GSH) are commonly used to assess oxidative damage associated with long-term PMS inhalation (Owagboriaye *et al.*, 2016; Ochayi *et al.*, 2015). Studies in both human and animal models have demonstrated increased lipid peroxidation, reduced antioxidant enzyme activity, and significant alterations in pulmonary and hepatic function following prolonged gasoline fume exposure (Uboh *et al.*, 2013; Elsayed *et al.*, 2015; Abah *et al.*, 2024).

Despite the growing evidence of gasoline fume toxicity, there remains a paucity of standardized

clinical data, particularly in low-resource settings such as sub-Saharan Africa, where environmental regulations are poorly enforced. Rapid urbanization, coupled with inadequate occupational safety measures, exacerbates the health risks associated with PMS inhalation (Tiekwe *et al.*, 2024). Notably, fine and ultrafine particulate emissions from gasoline fumes have been implicated in triggering inflammatory responses, oxidative damage, and an increased predisposition to respiratory diseases among urban workers, including fuel attendants and motorbike drivers (Odewabi *et al.*, 2014). However, comprehensive studies assessing the cytological and biochemical impacts of gasoline exposure remain limited, necessitating further investigation.

This study aims to evaluate the extent of gasoline fume exposure and its impact on oxidative stress markers and pulmonary cytoarchitecture. The hypothesis is that chronic inhalation of gasoline fumes leads to increased oxidative stress and structural lung damage, which can be quantified through changes in specific biomarkers and histological analysis. Hence, we will analyze key oxidative stress biomarkers, such as MDA, SOD and CAT while assessing lung tissue alterations in exposed individuals. The findings of this study will contribute to a deeper understanding of gasoline-associated health risks, inform occupational safety policies, and provide evidence-based recommendations for environmental health regulations and preventive healthcare strategies.

2. Materials and Methods

2.1 Collection of Premium Motor Spirit (PMS):

Premium Motor Spirit (PMS) was obtained from a certified and routinely inspected commercial fuel station. To ensure consistency and reliability, the fuel source was verified through supplier certification and regulatory compliance documentation. The PMS was stored in tightly sealed, chemically resistant containers at room temperature in a well-ventilated area before use. The quality control procedures applied include visual inspection for phase separation of particulates, as well as random sampling for hydrocarbon profile confirmation using gas chromatography, as it ensures the absence of contaminants or adulterants before experimental application.

2.2 Experimental Animals: Twenty (20) adult female Wistar rats with a mean body weight of 150 ± 10 g were procured from the Animal House, Department of Anatomy, University of Port Harcourt, Rivers State, Nigeria. The animals were randomly assigned to four experimental groups ($n = 5$ per group). They were housed in well-ventilated polypropylene cages (dimensions: $45 \times 30 \times 25$ cm) with five rats per cage, under standard laboratory conditions: temperature $23 \pm 2^\circ\text{C}$, relative humidity 50–60%, and a 12-hour light/dark cycle. Animals were allowed free access to standard commercial rodent chow and water ad

libitum. A two-week acclimatization period was observed before the commencement of the experiment to minimize stress-related variations. All animal handling and experimental procedures were conducted following the ethical guidelines for the care and use of laboratory animals. Ethical approval for this study was obtained from the Institutional Animal Care and Use Committee (IACUC) of the University, with Approval Number: IACUC/2023/017.

2.3 Exposure Protocol for PMS Fumes A modified nose-only inhalation exposure method, adapted from Okuonghae *et al.* (2011), was used to expose rats to Premium Motor Spirit (PMS) fumes. In this study, the modification involved the use of whole-body exposure chambers instead of restraint-based nose-only tubes to reduce stress and allow free movement during exposure, while still ensuring effective inhalation of PMS vapors. The experimental groups were placed in custom-fabricated inhalation chambers (dimensions: $60 \times 40 \times 50$ cm) made of transparent acrylic. Each chamber housed five rats, with a 1000 mL open glass beaker containing 500 mL of PMS positioned at the center of the floor to allow passive evaporation. To maintain consistent airflow and ensure controlled vapor dispersion, each chamber was fitted with two 10 cm-diameter air vents on the lid, acting as passive inlet and outlet ports. Low-speed fans (mounted externally) ensured continuous airflow, simulating ambient air movement and promoting uniform distribution of vapors while preventing accumulation to toxic levels. The PMS was replenished every 72 hours to maintain steady-state evaporation and exposure levels. This volume and replenishment rate were based on previous studies (Okuonghae *et al.*, 2011; Atoyebi *et al.*, 2019), which demonstrated that 500 mL of PMS in a 1000 mL open beaker produces vapor concentrations reflective of occupational and environmental exposure in poorly ventilated Nigerian settings. The control group was housed in an identical exposure chamber without PMS (containing distilled water instead), under the same ventilation conditions. This method allowed for chronic low-dose vapor exposure over the experimental period, mimicking environmental and occupational scenarios relevant to human populations in areas with frequent PMS vapor exposure

2.4 Experimental Design: The rats were randomly divided into four groups ($n = 5$ per group):

Control Group: Not exposed to PMS fumes. Housed in a clean-air, fume-free chamber identical in size and design to the exposure chambers but with no PMS present. The chamber was located in a separate, well-ventilated room within the animal facility to eliminate any risk of cross-contamination from PMS-exposed environments.

Group A: Exposed to PMS fumes for 4 hours daily (09:00–13:00) for 14 consecutive days.

Group B: Exposed to PMS fumes for 6 hours daily (09:00–15:00) for 14 consecutive days.

Group C: Exposed to PMS fumes for 8 hours daily (09:00–17:00) for 14 consecutive days.

The rationale for selecting these exposure durations was to simulate common real-life occupational and environmental exposure patterns in Nigeria, where individuals such as petrol station attendants, mechanics, and residents of areas with high fuel use often experience varying durations of daily exposure to PMS fumes. Following the exposure period, all animals were relocated to a designated recovery room within the animal facility. This recovery section was located in a separate wing with no history of chemical use or contamination, and underwent prior decontamination and enhanced ventilation protocols. The animals were monitored under standard housing conditions ($23 \pm 2^\circ\text{C}$, 50–60% humidity, 12-hour light/dark cycle) during the recovery period to ensure the clearance of PMS fumes and to observe any delayed toxicological effects.

2.5 Collection and Preparation of Lung Tissue

Following the exposure period, animals were anesthetized in a chloroform-saturated chamber. Each rat was weighed and euthanized by cervical dislocation. Lung tissues were carefully excised, rinsed in phosphate-buffered saline (PBS, pH 7.4), blotted dry, and divided for biochemical and histological analyses. The tissues designated for histology were fixed in 10% neutral buffered formalin for 72 hours, while those for biochemical assays were stored at -80°C until analysis.

2.6 Antioxidant Enzyme Assays

Lung homogenates were prepared by homogenizing 100 mg of lung tissue in 1 mL of ice-cold 50 mM phosphate buffer (pH 7.4) and centrifuging at 12,000 rpm for 15 minutes at 4°C . The supernatants were collected for enzyme assays:

Superoxide Dismutase (SOD) Activity was measured using the xanthine-xanthine oxidase system, based on inhibition of cytochrome C reduction (Misra & Fridovich, 1972).

Catalase (CAT) Activity was assayed by monitoring the decomposition rate of hydrogen peroxide at 240 nm (Aebi, 1984).

Malondialdehyde (MDA) Levels were determined by the thiobarbituric acid-reactive substances (TBARS) assay, following Bird & Draper (1984).

2.7 Histological Examination: Lung tissues were dehydrated in ascending ethanol concentrations, cleared in xylene, and embedded in paraffin. Sections ($4 \mu\text{m}$ thick) were cut using a rotary microtome (Leica RM2245, Germany) and stained with Hematoxylin & Eosin (H&E) using an automated staining system

(Leica Autostainer XL). The stained sections were examined under a light microscope (Nikon Eclipse Boi), and images were captured using a digital microscope-mounted camera (OMX1200C). Periodic Acid-Schiff (PAS) Staining Lung sections ($4 \mu\text{m}$) were deparaffinized, rehydrated, and incubated in 0.5% periodic acid solution for 5 minutes. After washing, sections were treated with Schiff's reagent for 15 minutes, counterstained with hematoxylin, and differentiated in 0.5% acid alcohol. Stained sections were examined for glycogen deposits under light microscopy.

2.8 Statistical Analysis

Statistical analyses were performed using GraphPad Prism 9.0 (GraphPad Software, San Diego, CA, USA). Data were expressed as mean \pm standard error of the mean (SEM). Group comparisons were conducted using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test. Statistical significance was set at $P < 0.05$.

3. Results and Discussion

3.1 Results

3.1.1 Effect of PMS Exposure on Oxidative Stress Biomarkers

The levels of oxidative stress markers, malondialdehyde (MDA), superoxide dismutase (SOD), and catalase (CAT), in rats exposed to gasoline fumes are presented in Figures 1–3.

MDA levels were significantly elevated ($p < 0.05$) in all groups exposed to gasoline (A, B, and C) compared to the control. This was most marked in Group C (Figure 1).

A significant decrease ($p < 0.05$) in SOD activity was recorded in all exposed groups, specifying a compromised antioxidant defense mechanisms (Figure 2).

Similarly, CAT activity was significantly reduced ($p < 0.05$) in all exposed groups, with the most pronounced decrease in Group C (Figure 3).

These findings suggest that gasoline exposure induces oxidative stress in a dose-dependent manner

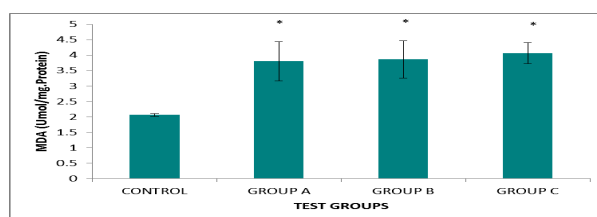


Fig 1: Effect of gasoline exposure on MDA levels in Wistar rats. Values are presented as mean \pm SEM. $p < 0.05$ and control.

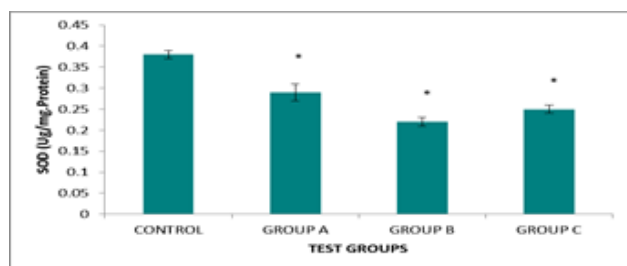


Fig 2: Effect of gasoline exposure on **SOD** activity. Values are presented as mean \pm SEM. $p < 0.05$ and control.

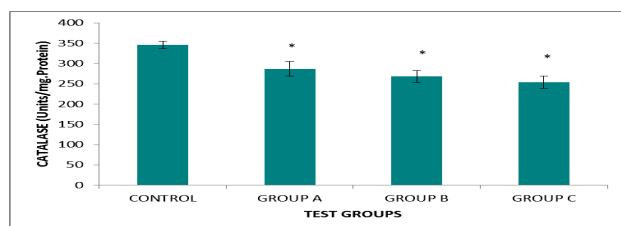


Fig 3: Effect of gasoline exposure on **CAT** activity. Values are presented as mean \pm SEM. $p < 0.05$ and control.

3.1.2 Effect of PMS Exposure on Lung Weight

The mean lung weights of rats in the control and gasoline-exposed groups are presented in Figure 4. There was a progressive increase in lung weight with longer gasoline exposure durations. A statistically significant increase ($p < 0.05$) was observed in Group C compared to the control.

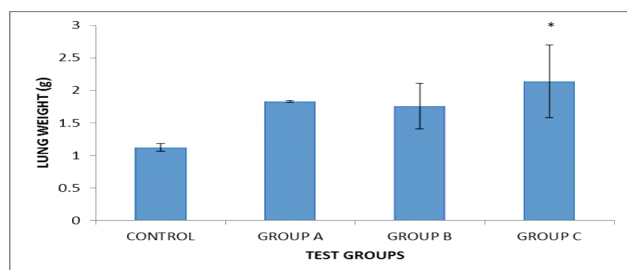


Fig 4: Effect of gasoline exposure on lung weight in Wistar rats. Values are expressed as mean \pm SEM. $p < 0.05$ and control

3.1.3 Histological Changes in Lung Tissue Following Gasoline Exposure

Histological examination of lung tissue was performed using Hematoxylin & Eosin (H&E) and Periodic Acid-Schiff (PAS) staining to assess structural and glycogen-related alterations, respectively.

Control Group (Plate 1 & Plate 5): Normal lung architecture, intact alveoli, and well-defined terminal bronchioles were observed.

Group A (Plate 2 & Plate 6): Moderate alveolar shrinkage and mild epithelial disorganization were noted. PAS staining revealed hyperplasia and glycogen accumulation in plate 6.

Group B (Plate 3 & Plate 7): The alveolar constriction was observed to be more severe while necrosis was also observed. Hyperplasia was present, but glycogen deposits were not detected.

Group C (Plate 4 & Plate 8): The most severe damage was noted, with collapsed alveoli, significantly dilated terminal bronchioles, and epithelial disruption. Hyperplasia was evident, but glycogen staining was negative.

These findings suggest that prolonged PMS exposure leads to progressive lung injury characterized by inflammation, necrosis, and loss of normal architecture

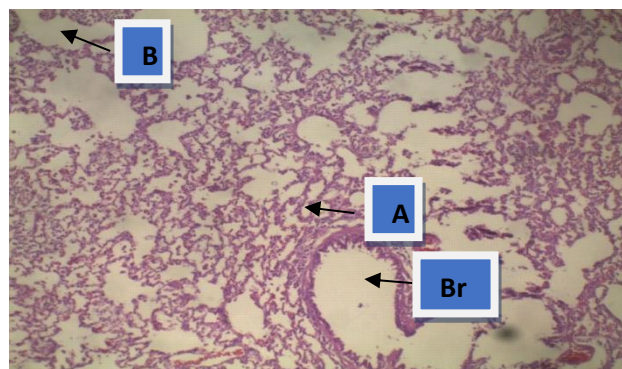


Plate 1 Lung section of the control group showing normal alveoli and bronchioles (H&E, x120). **B** (Bronchiole), **A** (Alveoli), **Br** (Bronchiole)

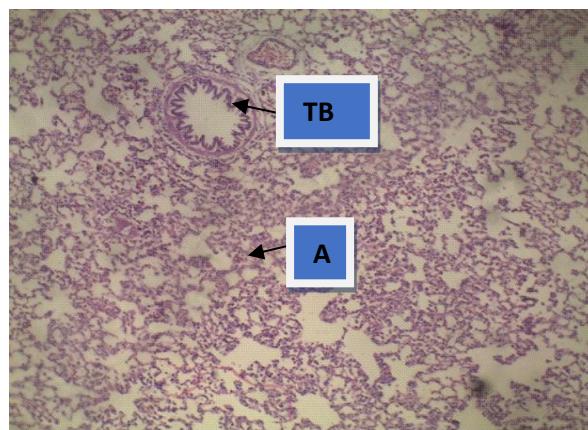


Plate 2 Lung section of Group A showing alveolar shrinkage (H&E, x120). **TB** (Terminal Bronchiole), **A** (Alveoli)

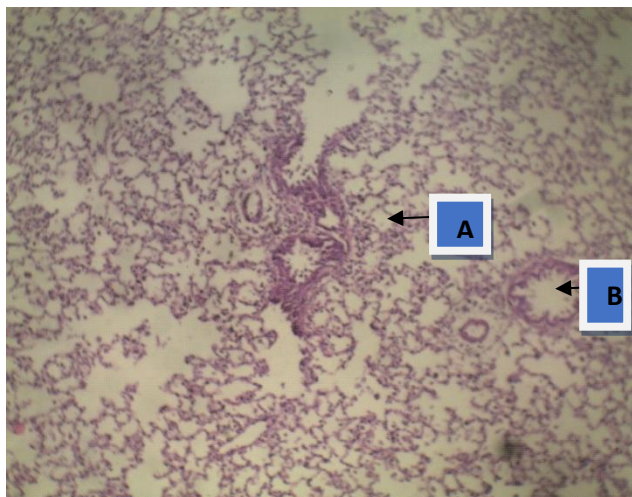


Plate 3 Lung section of Group B showing alveolar constriction and necrosis (H&E, x120). **A** (Alveoli), **B** (Bronchiole)

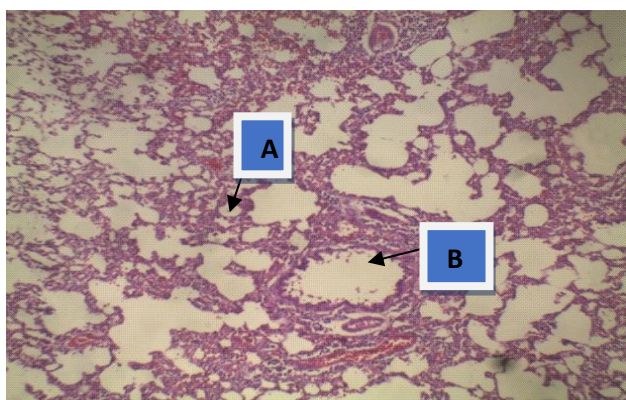


Plate 4: Lung section of **Group C** showing alveolar collapse and bronchiolar dilation (H&E, x120). **A** (Alveoli), **B** (Bronchiole)

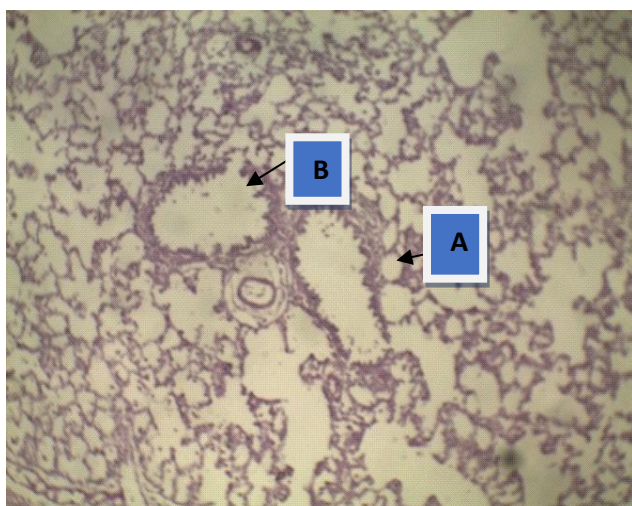


Plate 5: Lung section of the control group (PAS, x120) showing no glycogen accumulation. **B**(Bronchiole), **A** (Alveoli)

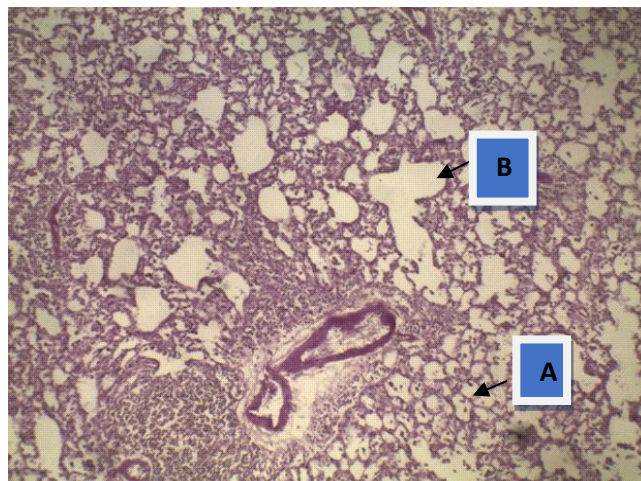


Plate 6: Lung section of **Group A** (PAS, x120) showing hyperplasia and glycogen accumulation. **B** (Bronchiole), **A** (Alveoli)

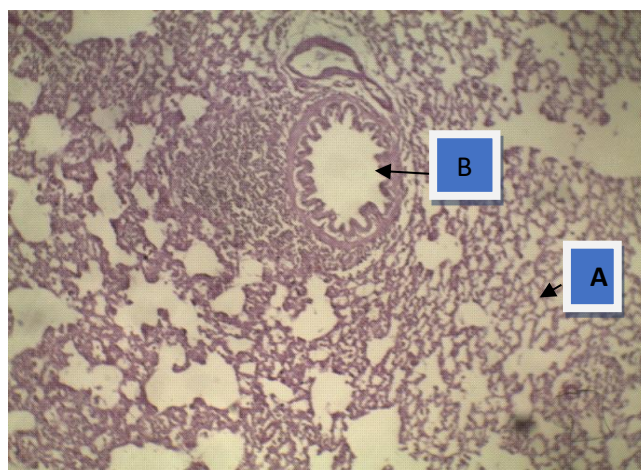


Plate 7: Lung section of **Group B** (PAS, x120) showing hyperplasia but no glycogen presence **B** (Bronchiole), **A** (Alveoli)

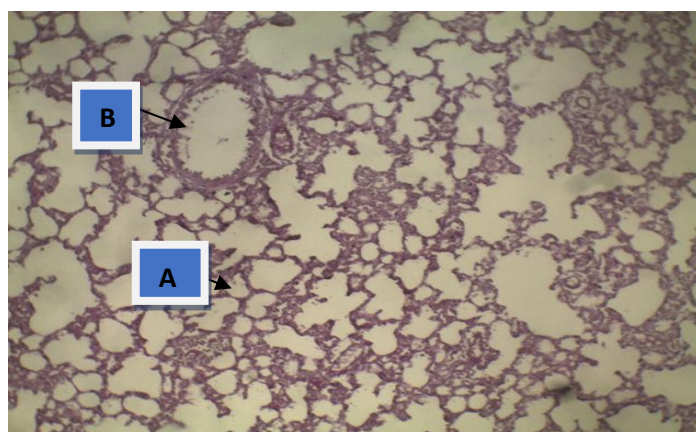


Plate 8: Lung section of **Group C** (PAS, x120) showing hyperplasia with the absence of glycogen.

3.2 Discussion

The present study investigated the impact of gasoline inhalation exposure on oxidative stress biomarkers, lung weight, and histopathological alterations in Wistar rats. The results in this study revealed that gasoline inhalation led to a significant increase in oxidative stress markers and lung weight. Alteration in the structure of lung tissues, which were related to the duration of exposure, was also observed.

In this study there was an elevation in malondialdehyde (MDA) levels and reduced activities of superoxide dismutase (SOD) and catalase (CAT) after gasoline exposure. MDA is a biomarker of lipid peroxidation, indicating increased oxidative damage to cellular membranes (Adly, 2010). The significant decrease in SOD and CAT activity suggests an impaired antioxidant defense system, rendering the lungs vulnerable to reactive oxygen species (ROS)-mediated damage. This aligns with previous studies that demonstrated increased oxidative stress in response to airborne pollutants, including hydrocarbons from fuel combustion (Xu *et al.*, 2018; Afsar *et al.*, 2019).

Gasoline has been documented to contain volatile organic compounds (VOCs) and polycyclic aromatic hydrocarbons (PAHs), which can undergo redox cycling, generating excessive ROS (Noori, 2012). These reactive species induce oxidative modifications in cellular components, leading to inflammatory responses, DNA damage, and apoptosis (Owagboriaye *et al.*, 2018). The dose-dependent increase in MDA and decline in antioxidant enzyme activity further indicate a progressive impairment in cellular defense mechanisms with prolonged exposure.

A significant increase in lung weight was observed across all gasoline-exposed groups, with the highest effect in Group C (8-hour exposure). Increased lung weight is often associated with pulmonary edema, congestion, or tissue remodeling in response to chronic inflammation (Brook *et al.*, 2010). The observed increase may result from inflammatory cell infiltration, increased vascular permeability, or extracellular matrix deposition, all of which contribute to tissue hypertrophy and altered lung mechanics.

Interestingly, previous studies have reported no significant difference in organ weights with short-term PMS exposure (Obidoa *et al.*, 2003). However, our findings suggest that extended exposure induces significant structural and functional changes, supporting the hypothesis that longer exposure durations exacerbate pulmonary toxicity.

Histopathological examination revealed severe structural damage to the lungs, including alveolar shrinkage, necrosis, and bronchiolar epithelial disorganization. These changes were more pronounced in Groups B and C, indicating a dose-dependent effect of gasoline exposure. The presence of peri-bronchiolar inflammatory cell infiltration and

vascular congestion suggests an inflammatory response, likely mediated by oxidative stress.

Previous research has demonstrated that prolonged exposure to airborne pollutants induces chronic bronchial inflammation, alveolar destruction, and fibrosis (Brook *et al.*, 2010; Afsar *et al.*, 2019). This report is supported by the findings in this study by showing progressive pulmonary injury with increased PMS exposure duration. The absence of glycogen deposits in PAS-stained sections of Groups B and C suggests possible metabolic alterations in lung cells due to oxidative stress.

The interplay between oxidative stress, inflammation, and lung tissue remodeling provides a plausible mechanism for gasoline-induced pulmonary toxicity. Prolonged exposure may lead to chronic obstructive pulmonary disease (COPD), fibrosis, or even carcinogenesis, highlighting the long-term risks associated with continuous PMS inhalation.

4. Conclusion

The results from this study highlights the fact that gasoline exposure induces oxidative stress, alters lung weight, and causes significant histopathological damage in Wistar rats. The findings indicate that oxidative stress is a key mechanism underlying gasoline-induced pulmonary toxicity, leading to inflammatory responses and structural lung damage. The severity of oxidative damage and histopathological changes increased with exposure duration, suggesting that prolonged PMS inhalation could contribute to chronic respiratory disorders. These findings emphasize the need for regulatory measures to minimize human exposure, particularly in occupational settings where workers are frequently exposed to fuel vapors.

Conflict of Interest

The authors declare no conflict of interest.

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