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ORIGINAL RESEARCH ARTICLE

Comparative Pulmonary Toxicity of PMS and Kerosene Fumes in a Rat Model

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ABSTRACT

This study investigated the sub-chronic effects of inhaled Premium Motor Spirit (PMS) and kerosene fumes on pulmonary histology and physiology in albino rats. Fifteen adult male rats were randomly assigned to three groups: control (fresh air), PMS-exposed, and kerosene-exposed. Exposures were conducted for six hours daily over five weeks. Body and lung weights were recorded, and histopathological analysis of lung tissues was performed using hematoxylin and eosin staining. Compared to controls (body weight: 203 ± 0.96 g; lung weight: 5.72 ± 0.63 g), both exposed groups showed significant reductions in body and lung weights. The kerosene-exposed group had a mean body weight of 137 ± 2.08 g and lung weight of 4.53 ± 0.56 g ($p < 0.05$), while the PMS-exposed group had a mean body weight of 145 ± 2.09 g and lung weight of 4.83 ± 0.58 g ($p < 0.05$). Histologically, controls exhibited normal lung architecture. Kerosene exposure resulted in mild inflammation, whereas PMS exposure caused severe emphysematous changes characterized by alveolar wall destruction and cystic space formation. These findings demonstrate that sub-chronic inhalation of PMS and kerosene fumes induces significant systemic and pulmonary toxicity, with PMS exerting more pronounced structural damage to lung tissue. The results highlight the potential respiratory health risks associated with chronic exposure to petroleum fumes, particularly in occupational and domestic settings with poor ventilation.

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PMS, kerosene fumes, pulmonary histology, emphysema, sub-chronic inhalation

INTRODUCTION

Premium Motor Spirit (PMS) and kerosene are among the most commonly used petroleum products in Nigeria and other oil-producing economies. Both are complex mixtures of hydrocarbons; PMS contains volatile aromatic compounds such as benzene, toluene, ethylbenzene, and xylene (BTEX), along with aliphatic hydrocarbons (Kodidala et al., 2020). Kerosene consists predominantly of heavier aliphatic and aromatic hydrocarbons (Dantes et al., 2016). Although its fumes are less volatile than those of PMS, kerosene can generate substantial concentrations in poorly ventilated spaces, and incomplete combustion, common in older stoves and lamps, produces particulate matter and toxic by-products such as carbon monoxide. In many Nigerian households and student hostels, kerosene is used for cooking in enclosed areas without chimneys or smoke hoods. Additionally, it is employed as a mosquito larvicide, a solvent, and a cleaning agent by mechanics, who often use it alongside petrol (Igboh et al., 2001).

The respiratory system represents a major portal of entry for airborne toxicants, offering a vast surface area for gas exchange. Inhaled fumes can deposit along the respiratory tract and reach the alveolar region, where close contact with pulmonary tissues facilitates local damage. Inhalation exposure to petroleum products is known to irritate mucous membranes, eyes, and skin (Hathaway & Proctor, 2014). Such pollutants can activate immune responses, recruiting leukocytes and macrophages (Hiraiwa & van Eeden, 2013), and have been linked to serious health effects, including leukaemia, lung cancer, allergic pneumonitis, cardiovascular disease, immune suppression, and neurological disorders (Dantes et al., 2016; Vogelmeier et al., 2017).

The albino rat is widely used as a model in inhalation toxicology because its respiratory anatomy and physiology are well characterized and share important similarities with humans, allowing extrapolation of potential health effects (Smith et al., 2010). Histopathological evaluation of lung tissue remains a critical endpoint in such studies, providing direct visual evidence of cellular damage, inflammation, and structural alterations induced by inhaled toxicants (Jones & Williams, 2015).

In Nigeria, exposure to PMS and kerosene fumes is pervasive. These fuels are extensively used for transportation, power generation, and domestic purposes,

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often under poorly ventilated conditions. Consequently, a substantial segment of the population is at risk of chronic inhalation exposure. While acute toxicity following high-level exposure is relatively well documented, the sub-chronic effects of low-level fume inhalation on lung tissue architecture remain incompletely understood. This knowledge gap poses a public health concern, as unrecognized histopathological changes may contribute to the burden of respiratory diseases.

Given the widespread handling and use of these fuels in confined spaces, investigating the specific structural and cellular changes they induce in the lungs is essential. Respiratory diseases are a leading cause of global morbidity and mortality, and understanding the contribution of petroleum fume exposure to these conditions can inform risk assessments, awareness campaigns, and public health policies.

This study therefore aimed to evaluate the histopathological alterations in the lungs of albino rats following sub-chronic inhalation of PMS and kerosene fumes. By comparing the effects of these two commonly encountered petroleum products, the research seeks to provide scientific evidence on their differential pulmonary toxicity and to highlight the potential respiratory risks associated with long-term exposure in occupational and domestic settings.

MATERIALS AND METHODS

Animals and Chemicals

Healthy adult male albino rats (n=20, with extras to ensure a final sample size of 15), aged 8–10 weeks and weighing 150–200 g, were obtained from the University Animal House, Edo State University, Iyamho. The animals were acclimatized to laboratory conditions for one week prior to the experiment. They were housed in clean plastic cages (56 cm × 39 cm × 19 cm) with wooden chip bedding, under standard conditions (12-hour light/dark cycle, temperature 22 ± 2°C), and had free access to standard rodent chow and water *ad libitum*. Premium motor spirit and kerosene were purchased from a local filling station.

Ethical Approval

The ethical approval was obtained from Edo University Ethical committee with approval number: EDSUREC25/064

Exposure Protocol

This study utilized fifteen (15) adult male albino rats, randomly assigned to three (3) groups of five (5) animals each. Group A served as the control and was exposed to fresh air. Group B was exposed to premium motor spirit (PMS) vapor, and Group C was exposed to kerosene vapor. All exposures were conducted for six (6) hours per day over a period of five (5) weeks. All animal procedures were performed in accordance with the institutional guidelines for the care and use of laboratory animals.

The experiment was conducted in well-ventilated inhalation chambers to ensure uniform distribution of the test substances. For the exposure groups, PMS and kerosene were placed into individual nebulizers within their respective chambers to generate vapors. The control group was housed in a separate chamber and exposed to filtered fresh air for the same duration. The target vapor concentration, derived from existing literature on sub-acute inhalation toxicity of petroleum products, was chosen to induce histopathological changes without causing significant mortality. Vapor concentrations within the chambers were periodically monitored using appropriate gas detection techniques (e.g., gas chromatography-mass spectrometry).

Body and Organ Weight Analysis

Body weight was recorded for all animals at the beginning of the experiment (week 0) and at the end of the five-week exposure period, just prior to sacrifice, using a digital weighing balance. Following sacrifice, organ weights were also recorded. The percentage change in body weight was calculated for each group.

Anesthesia, Sacrifice, and Tissue Collection

Twenty-four hours after the final exposure, all rats were humanely euthanize by cervical dislocation. The thoracic cavity was opened, and the lungs were carefully excised. For histopathological examination, the lungs of each rat were inflated with 10% neutral buffered formalin via the trachea and then immersed in the same fixative for a minimum of 24 hours.

Histopathological Examination

Following fixation, the lung tissues were processed using a standard histology protocol. This involved dehydration through a graded series of ethanol (70%, 80%, 95%, and 100%), clearing in xylene, and embedding in paraffin wax. The embedded tissues were sectioned at 5 µm thickness using a microtome. Tissue sections were mounted on clean glass slides and stained with Hematoxylin and Eosin (H&E). The stained slides were examined under a light microscope at magnifications of ×100 and ×400. A systematic evaluation of lung structures (including airways, alveoli, and blood vessels) was performed to identify histopathological alterations such as inflammation (presence of inflammatory cells), congestion (accumulation of blood), and edema. The histological appearances of the lung tissues from the exposed groups were compared to those of the control group.

Representative photomicrographs were taken to document the findings.

Statistical Analysis

All data were expressed as mean ± standard deviation (SD). Statistical significance was determined using an appropriate test, with a p-value of less than 0.05 considered significant.

RESULTS

Body and Lung Weights

The effects of sub-chronic exposure to kerosene and Premium Motor Spirit (PMS) fumes on body and lung weights are presented in Tables 1 and 2, respectively. All values are expressed as mean ± standard deviation.

As shown in Table 1, exposure to kerosene fumes resulted in a significant reduction in mean body weight (137 ± 2.08 g) compared to the control group (203 ± 0.96 g; $p < 0.05$). Similarly, mean lung weight in the kerosene-exposed group (4.53 ± 0.56 g) was significantly lower than that of the control group (5.72 ± 0.63 g; $p < 0.05$).

Histopathological Findings

Histological examination of lung tissues revealed distinct morphological differences between the control and exposed groups (Plates 1–3).

Plate 1 shows a representative section of lung tissue from the control group (Group A). The architecture is well preserved, with normal bronchi (thick arrow) and alveolar sacs (thin arrow) separated by intact interstitium. These features are consistent with normal lung histology.

Table 1: Average Body and Lung Weights After Exposure to Kerosene (Corrected)

Parameter	Group A (Control)	Group B (Kerosene/PMS)	df	F-value	P-value
Body Weight (g)	203 ± 0.96	137 ± 2.08^{ab}	(1, 8)	4150.152	< 0.0001
Lung Weight (g)	5.72 ± 0.63	4.53 ± 0.56^{ab}	(1, 8)	9.966	0.013

^{ab} Superscripts indicate significant differences at $p < 0.05$. df = degrees of freedom.

Table 2: Average Body and Lung Weights After Exposure to PMS (Corrected)

Parameter	Group A (Control)	Group B (Kerosene/PMS)	df	F-value	P-value
Body Weight (g)	203 ± 0.96	145 ± 2.09^{ab}	(1, 8)	3179.764	< 0.0001
Lung Weight (g)	5.72 ± 0.63	4.83 ± 0.58^{ab}	(1, 8)	5.401	0.049

^{ab} Superscripts indicate significant differences at $p < 0.05$. df = degrees of freedom.

Note: F- and p-values were recalculated using one-way ANOVA from reported group means and standard deviations. n = 5 per group; df = (1, 8) for all comparisons.

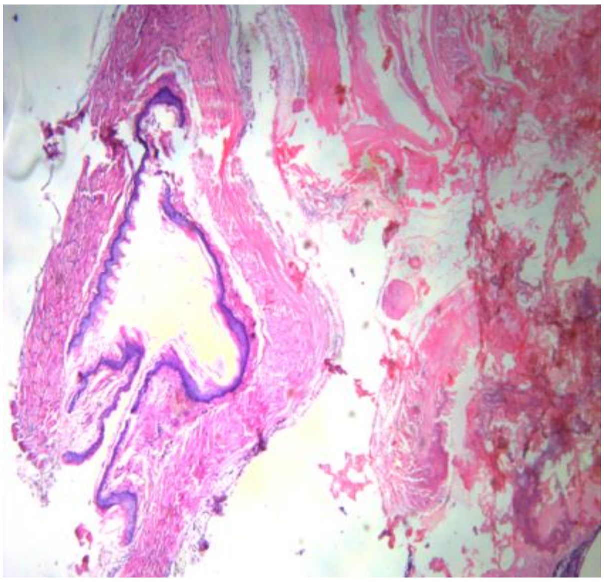

Plate 1: Control section of the lung from Group A, showing normal bronchi (thick arrow) and alveolar sacs (thin arrow) separated by intact interstitium. Features are in keeping with a normal lung. H&E ×400.

Plate 2 illustrates lung tissue from the kerosene-exposed group (Group B). The overall architecture remains largely normal, with identifiable bronchi (thick arrow) and alveoli (thin arrow) separated by the interstitium. No overt structural disruption is observed; however, mild inflammatory changes were noted in some sections.

Plate 3 depicts lung tissue from the PMS-exposed group (Group C). In contrast to the control and kerosene-exposed groups, PMS exposure induced marked structural alterations. Airways (thick arrow) and alveolar sacs (thin arrow) show evidence of septal destruction, with formation of cystic spaces. These features are characteristic of emphysematous change.

In summary, sub-chronic inhalation of kerosene and PMS fumes for five weeks resulted in significant reductions in both body and lung weights compared to the control group ($p < 0.05$). Histopathologically, kerosene exposure produced mild inflammation without major architectural disruption, whereas PMS exposure caused severe emphysematous changes characterized by alveolar wall destruction and cystic space formation. These findings indicate that PMS exerts more pronounced pulmonary toxicity than kerosene under the conditions of this study.

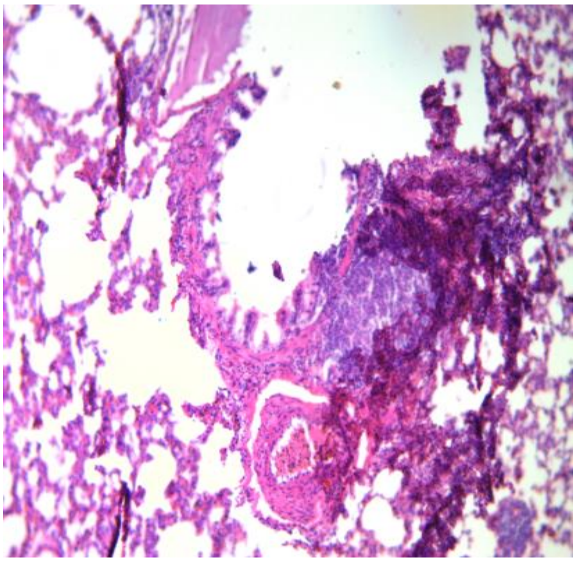

Plate 2: Section of the lung from Group B (kerosene exposure), showing a normal bronchi (thick arrow) and alveoli (thin arrow) separated by interstitium. Features are in keeping with normal lung. H&E ×400.

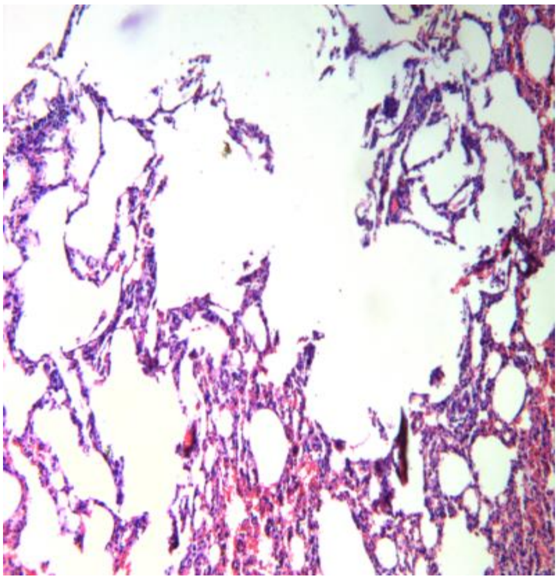

Plate 3: Section of the lung from Group C (PMS exposure), showing an airway (thick arrow) and alveolar sacs (thin arrow) with destroyed septa and formation of cystic spaces. Features are in keeping with emphysematous change. H&E ×400.

DISCUSSION

The current research paper examined the histopathological and physiological impacts of sub-chronic exposure of albino rats to the Premium Motor Spirit (PMS) and kerosene fumes, and specifically the body weight changes, the weight of lungs, and tissue changes. The results showed that there were great losses in body and lung weights among the exposed groups as compared to the controls, which were evidenced by histopathology results of inflammation and emphysematous damage, specifically in the PMS-exposed group. These findings are in line with the hypotheses that the petroleum product fumes have toxic effects on the lung tissue and health in general. Among the discoveries of this study was the loss of body mass of rats that were exposed to PMS and kerosene fumes. This observation is consistent with the reports in the past, which found that exposure to hydrocarbons can lead to systemic toxicity that can disrupt normal metabolism and appetite (Agbonlahor et al., 2018; Adeyemi et al., 2021). The drastic loss of weight in exposed groups could be an indication of increased oxidative stress and inflammatory load, which is known to cause derailment in energy metabolism and nutrient utilization. Moreover, the decreases in the weight of the lungs, observed in this research, have been linked with the destruction of the alveoli, as well as the loss of parenchymal integrity following exposure to noxious fumes (Ezekiel et al., 2019). The histopathological examinations of the tissues damaged by PMS and kerosene fumes were very instrumental in the determination of the nature of the tissue damage. Control and kerosene-exposed groups showed an overall normal architecture, whereas PMS exposure resulted in extensive emphysematous alterations with wall destruction of alveoli and cystic spaces. Such structural changes are characteristic of chronic obstructive lung disease pathology and support the previous results that PMS, which has high benzene levels and volatile organic compounds, is especially harmful to lung tissue (Udonwa et al., 2019; Olaniyan et al., 2022). Inflammation, though mild, is also found in groups exposed to kerosene, which is in line with previous research that blames the toxicity of kerosene on combustion by-products (soot and polycyclic aromatic hydrocarbons) (Ajayi et al., 2020). The fact that chemical composition is important in determining toxicological outcome is highlighted by the fact that lesions in both PMS and kerosene exposures differ in severity. Being more volatile and containing more benzene, PMS seems to be more harmful to the fragile alveolar structure than kerosene, which is less volatile and which causes irritant particulates during combustion (Adegoke et al., 2019; Adetona et al., 2021). Combined exposure has either a synergistic or additive effect, which is not explicitly evident in this study, but the latter is an area that warrants further investigation since in occupational and domestic environments, there can be a simultaneous exposure to a variety of petroleum products. In immunotoxicological terms, the histological inflammation observed is consistent with the published literature that shows that hydrocarbons cause neutrophil and macrophage recruitment with the release of pro-inflammatory cytokines, including TNF-alpha and IL-6 (Adeyemi et al., 2021). With such processes sustained over time, they can become fibrotic and irreversibly remodel structural features, as described in the case of such studies in animals (Ezekiel et al., 2019). The observed emphysematous changes in the PMS group also indicate the long-term consequences, such as decreased pulmonary compliance and gas exchange. The conclusions that this research will make have more widespread ramifications on the general health of society in places such as Nigeria, where people are exposed to PMS and kerosene fumes as a result of domestic and working conditions. Employees of fuel stations, mechanics, and those who cook using kerosene stoves in poorly ventilated places may be at high risk of respiratory maladies, just like those experienced in this laboratory model. The chronic exposure may lead to the population-level increase of chronic obstructive pulmonary disease (COPD), asthma, and lung cancer (Alqahtani et al., 2020; Etzel et al., 2019). Animal models are also found to be relevant in this study in terms of toxicological research. The histopathological results in rats are similar to those of pathology observed in humans that were exposed to petroleum hydrocarbons, thereby strengthening the translationalism of such experimental designs (Ezeokeke et al., 2018). Nevertheless, there are some constraints in the direct extrapolation results to human populations that are based on interspecies differences of physiology and exposure dynamics. Future researchers should thus use animal results to complement epidemiological research and sophisticated in vitro systems.

CONCLUSION

The paper measured the histopathological and physiological consequences of sub-chronic inhalation of Premium Motor Spirit (PMS) and kerosene fumes on the albino rats. The results showed that body and lung weight significantly reduced in the exposed groups as compared to controls, which shows systemic toxicity and lung dysfunction. Histopathological examination showed there were mild inflammatory changes in the kerosene group and emphysematous changes in the PMS group, indicating that PMS is more destructive to pulmonary architecture when compared to kerosene. The findings confirm the hypotheses of the research, which postulates that the use of petroleum product fume leads to physiological and histopathological changes in the lungs. Specifically, PMS exposure gave rise to the development of destructive alterations of the alveolar walls and cystic space development, which is in line with the emphysematous pathology. The inflammatory reaction that has been observed in the exposed groups is indicative of oxidative stress and immune stimulation caused by hydrocarbons. The implications of these findings to the health of the population are significant, particularly in the areas where PMS and kerosene are extensively used in transport, the generation of power, and household. People who are regularly exposed to such fumes, like fuel station workers, mechanics, and those households with kerosene stoves, stand a chance of developing respiratory diseases such as chronic obstructive pulmonary disease (COPD), pulmonary fibrosis, and emphysema. This experiment offers scientific proof that sub-chronic breathing of PMS and kerosene smokes lead to physiological and histopathological alterations in the lungs. This shows the immediate need to have better policies and regulations, better safety measures at work, and awareness campaigns among people to minimize the harmful exposures and protect respiratory health.

RECOMMENDATIONS

The awareness campaigns should also be increased to inform the general public about the risk of long-term inhaling Premium Motor Spirit (PMS) and kerosene fumes. This is particularly directed at households, fuel attendants, mechanics, and students who engage in using kerosene stoves in poorly ventilated facilities. Kerosene is still commonly in use as cooking and lighting fuel, and therefore, enhanced ventilation in homes and student hostels should be encouraged. With the provision of chimneys, smoke hoods and cross-ventilation systems, the level of toxic fume concentration inside will be greatly minimized. Individuals who were exposed to PMS and kerosene fumes in the course of their occupational or domestic activities over an extended period should be screened on a regular basis with a focus on lung tests and prompt identification of respiratory disorders.

CONTRIBUTIONS TO KNOWLEDGE

The study demonstrated that PMS fumes are more damaging to lung tissue than kerosene fumes, producing emphysematous changes characterized by alveolar wall destruction and cystic space formation. This adds to existing toxicological data by providing comparative evidence of differential pulmonary toxicity between two widely used petroleum products. While previous studies have often focused on acute exposure, this research highlights the sub-chronic effects of PMS and kerosene inhalation. It shows that even non-lethal, prolonged exposure leads to measurable histological alterations such as inflammation, congestion, and emphysematous lesions, thereby expanding the understanding of intermediate-duration inhalation risks.

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