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Original Research Article

A Histological Study on The Short-Term Effect of Kerosene and Diesel Exposure on the Olfactory Epithelium of Wistar Rats

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ABSRTACT

Exposure to kerosene and diesel, which are environmental contaminants, is mainly via inhalation. Adverse effects of kerosene and diesel on parts of the respiratory system have been reported. There is, however, limited data on their effects on the olfactory epithelium. This study aimed to determine and compare the effect of short-term inhalation of kerosene and diesel on the olfactory epithelium of Wistar rats. Fifteen (15) Wistar rats weighing 120-150g were divided into three (3) groups (n=5). Group 1 (control) was not exposed. Groups 2 and 3 were placed in different inhalation chambers with a beaker containing 500mls of kerosene (group 2) and 500 mls of diesel (group 3). Groups 2 and 3 were exposed for 1hour daily for 21 days. Histopathological examination of the olfactory epithelium was conducted and blood samples were analyzed to determine the levels of SOD, MDA, and CAT. Thinning of the olfactory epithelium in group 2 and loss of the olfactory epithelial lining in group 3 was observed after exposure. A decrease in SOD ($p>0.05$) was observed in group 2 (0.39±0.04) and group 3 (0.31±0.06), while an increase in MDA ($p>0.05$) was observed in group 2 (0.58±0.26) and group 3 (0.67±0.44). A decrease in CAT ($p>0.05$) was observed in group 2 (2.87±0.49) and group 3 (2.50±0.16) after exposure. Short-term exposure to kerosene and diesel adversely affects the structure of the olfactory epithelium in Wistar rats.

Keywords: Exposure, Kerosene, Diesel, Olfactory, Epithelium.

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Introduction

Petroleum hydrocarbons consist of hundreds of crude oil derived chemical compounds that are frequently described as the most prevalent environmental contaminants globally.[1] Diesel and kerosene are petroleum products whose fumes contain toxic organic components that are pervasive in the environment.[2] They are ubiquitous and exposure by inhalation is a potential route of neurotoxicity.[3] Occupational exposure which can occur in refineries, gas stations, transportation industries, by accidental spills and improper storage are also ways by individuals can be exposed to these petroleum hydrocarbons.[4]

Diesel fuel is widely used in various kinds of machines especially in vehicle engines which has been identified as an important source of pollution.[5] Kerosene is used as cooking and lighting fuel in homes.[6] Exposure to various forms of kerosene and diesel such as jet engine emissions and diesel exhaust emissions literally means exposure to volatile organic compounds and particulate matter consisting of inorganic carbon core with associated polycyclic aromatic hydrocarbons and metals.[7] Diesel is volatile and exposure to its vapour may be detrimental to the normal function of the systems in the body and the environment.[8,9]

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A study in 2023 revealed that exposure to crude oil vapour induced oxidative stress and altered the histopathological markers in the hippocampal region of rats leading to behavioral impairments.[10] A product of kerosene distillation: jet fuel was a toxicant found to promote epigenetic transgenerational inheritance of disease susceptibility in the offspring of pregnant rats.[11]

The fumes from diesel has been linked to adverse health effects in humans which include respiratory illnesses and asthma.[12] It has also been linked to atherosclerosis, hypertension, and cancer development.[13,14,15] Various studies have reported the adverse effects of kerosene on various systems which include the respiratory system and nervous system.[16,17,18,19] Other systems include gastrointestinal system, integumentary system and the Cardiovascular system.[20,21,22,23,24,17] Despite extensive studies on the various systems, studies on the effect of petroleum hydrocarbon exposure on the olfactory system have focused on mitochondria functions, olfactory mucosa cells and gestational exposure.[25,26,27] The studies on the effect of various petroleum hydrocarbons on the olfactory system is limited. This aim of this study was to determine the short-term effect of kerosene and diesel exposure on the olfactory epithelium of wistar rats.

Materials and Methods

Experimental animals

Fifteen Wistar rats with an average body weight of 120-150g were obtained from the animal house of the Anatomy department Madonna University Elele Campus Nigeria and used for this study. They were allowed to acclimatize for one week before the commencement of the experiment. The animals were housed in stainless steel cages and were allowed free access to food and water *ad libitum*. All animal experiments were carried out in accordance with the guidelines of the Institutional Animals Ethics Committee.

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Experimental design
Fifteen wistar rats (N=15) were randomly distributed into three (3) groups. Five (5) rats were placed in each of the groups (n=5). Group one served as the control and had no exposure to kerosene or diesel. Group two was exposed via inhalation to kerosene. Group three was exposed via inhalation to diesel. The three different groups were kept far apart from each other during the exposure period. Groups two and three were exposed to the petroleum products (kerosene and diesel) as stated above for the period of twenty - one (21) days. This was done according to the method of. [28]

Exposure to kerosene and diesel
Kerosene and diesel were purchased from a filling station in Elele Rivers state, Nigeria. The modified nose inhalation method was applied in which the animals in the test groups were placed in inhalation chambers (23.5 ×17.0×18.5 inches), with beakers of either kerosene or diesel, depending on the group. A beaker containing 500cm^3 of kerosene and diesel was placed in the respective chambers.[29] This was done in order to expose the animals to the vapour liberated from direct evaporation of liquid kerosene or diesel. The control group was kept in a kerosene and diesel-free section of the animal house. An exposure period of one hour daily, [18] was adopted for 21 days. [28,29]

Morphological Studies
Rats in groups 2 and 3 were observed for signs of kerosene and diesel toxicity at the initial stage of the study and after exposure. The appearance of the skin, fur, eyes, behavioral pattern, mobility and mortality were observed.

BIOCHEMICAL ANALYSIS
For tissues to be used for biochemical determinations. Blood was collected from the rats by cardiac puncture and blood samples from each animal was placed into a sample bottle for biochemical analysis of enzymes of oxidative stress (malondialdehyde, superoxide dismustase and catalyse).

Collection of Tissues
Chloroform sedation was utilized during the sacrifice of the animals at the termination of the study. The heads were removed, deskinned and the skull was split sagittally, thus exposing the nasal cavity. Respiratory and olfactory portions of the nasal epithelium were collected and placed in a sample bottle containing 10% formal saline. After a fixation period of 24 hours samples were rinsed with tap water, and embedded in paraffin wax. Slides for histopathology were stained with hematoxylin and eosin.

Results and Discussion

Effect of kerosene and Diesel exposure on Morphological observations
Results from the observation period carried out on all the rats in group one, two and three is shown in table 1. Observations such as sluggishness, reduced food intake, oligodipsia and diarrhea were made in group two. Reduced food intake, oligodipsia, sluggishness, clustering of rats, redness of eyes and diarrhea was observed in group two. There was an absence of reduced food intake, oligodipsia and sluggishness in the control group.

Table 1: Physical observations of the different groups of rats

GROUP	CLINICAL OBSERVATION
Control (group one)	
Food intake ,diarrhea, sluggishness,	–––––––
oligodipsia,clustering of rats	
redness of eyes	
‘Kerosene (group two)	
Food intake, diarrhea, sluggishness,	+ + + + ––
Oligodipsia, clustering of rats	
redness of eyes	
Diesel (group three)	
Food intake, diarrhea, sluggishness,	+ + + + + +
Oligodipsia, clustering of rats	
redness of eyes	

NOTE:++ = observation present ---= observation absent

Effect of kerosene and diesel inhalation on oxidative stress markers (SOD, MDA, CAT)
The effect of kerosene and diesel inhalation on SOD, MDA and CAT is shown in Fig 1. The level of SOD was lower (p>0.05) (0.31± 0.04) in group 3 compared to group 2 (0.31 ± 0.06). An increase in MDA was observed, which was higher (p>0.05) in group 3 (0.67 ± 0.44). Decrease in catalase level was increased (p>0.05) in group 3 (2.50 ± 0.16).

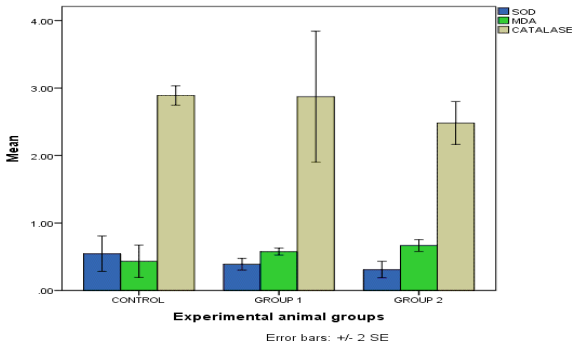

Fig 1: Effect of kerosene and diesel on MDA, SOD AND CAT

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Effect of kerosene and diesel inhalation on histology of the nasal epithelium

In the control group a normal epithelial lining was observed (fig 2). Thining of the epithelial lining was observed in group 2 (fig 3) while loss of epithelial lining was observed in group 3 (fig 4).

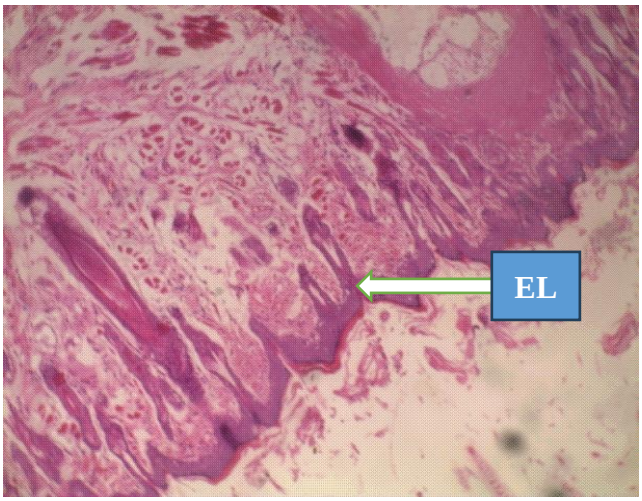

Fig 2: Photomicrograph of nasal epithelium (vestibule) of group one (control) showing normal epithelial lining (EL).

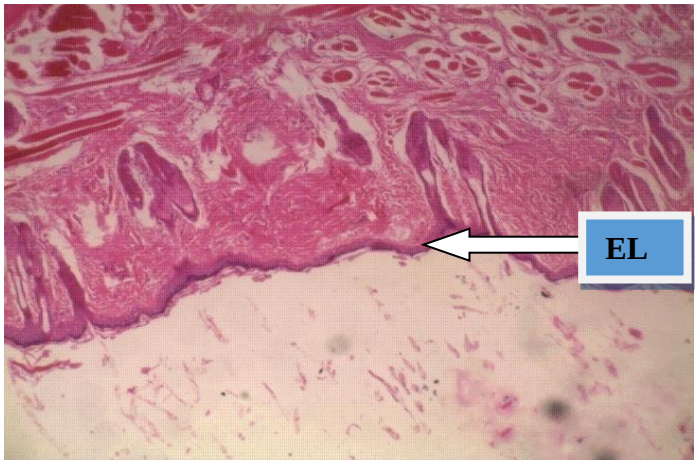

Fig 3: Photomicrograph of nasal epithelium in group two (kerosene) showing thinning of the epithelial lining (EL)

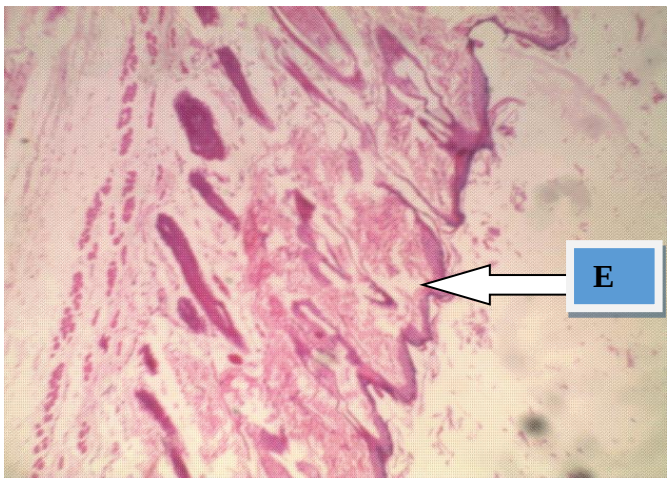

Fig 4: Photomicrograph of nasal epithelium in group 3 (diesel) showing loss of epithelial lining (EL).

Fumes from petroleum products are ubiquitous and have been stated to contain toxic organic components.[2] Exposure to these products can be due to contact with the skin and inhalation of vapours specifically hydrocarbons released during the process of refining. These products have an unstable nature which makes them promptly accessible in the air thereby making inhalation a means by which exposure to them occurs. [30]

In this study the aftermath of exposure to kerosene and diesel on the nasal epithelium and oxidative stress was reported. Morphological observation of the rats after exposure to kerosene and diesel indicated signs of kerosene and diesel toxicity. Redness of the eyes observed in groups 2 and 3 is similar to an observation reported in a case study, [31] in which an individual in contact with JP-5 vapor indicated a burning sensation in the eyes accompanied by itchy watery eyes. JP-5 is stated to be a kerosene based jet fuel. [32] Another study reported indications of irritation in the eyes of F-344 rats exposed to JP-8 vapor a kerosene based jet fuel.[33] Due to the revealing nature of the mucous membranes of the eyes it can be damaged after exposure to air pollutants resulting in detrimental effects on the corneal epithelial cells, inflammation and oxidative stress.[34] One of the substances used in this study kerosene is a petroleum product, that is released into the enviroment as a pollutant.[35,36] Exposure of the ocular surface to toxic irritants leads to the generation of immune and neuronal responses with increased levels of excitability and chronic pain.[37]

Sluggishness observed after exposure to kerosene and diesel could have occurred due to exposure to toluene. Incordination and muscle weakness was observed to have occurred in three volunteers after exposure to toluene.[32] Another report stated that toluene is a constituent of kerosene and diesel present in various fractions of pertroleum.[38] Diarrhea, reduced food intake and oligodipsia was noticed in this study. A study on a man exposed to diesel vapor for 10 days revealed abdominal cramps, diarrhea and vomiting.[39] Inhalation of volatile hydrocarbons compounds can result in irritation that presents as nausea, vomitting, diarrhea.[40]

The circumstance in which reactive oxygen species is in excess of the available antioxidant- buffering capacity is defined as oxidative stress. [41] Cellular proteins, lipids membranes and DNA can be destroyed by reactive oxygen species. [42, 43, 44,45] In this study increased levels of MDA indicated lipid peroxidation which has been reported to be one of the mechanisms of generation of free radicals. [46] Damage to the cell and structure of the tissue could occur due to the disintegration of the antioxidant defense system in the presence of an overabundance of free radicals. [47] The increase in MDA was higher in group 3 which was in consonance with another study in which rats exposed to petrol, kerosene and diesel recorded the highest MDA level in the diesel group. [48] Oxidative catabolism of polyunsaturated fatty acids referred to as lipid peroxidation is normally accepted as the major mechanism for cellular damage and death. This has been suggested in various pathological conditions. [49, 50] Superoxide dismutase and catalase termed endogenous antioxidant enzymes act as free-radical scavengers involved in the repair of the damage brought about by reactive oxygen species. [51,52] The decrease in the levels of SOD and CAT is also in consonance with another study, [53] in which it was reported that there was a decrease in CAT and SOD while there was an increase in MDA after exposure to petroleum fractions. The results in this study suggest that exposure to kerosene and diesel could induce oxidative stress by consuming the protective free radical scavengers.[54]

The nasal cavity is vulnerable to chemically stimulated injury due to the exposure to inhaled irritants. [55] Exposure of rats in this study to kerosene revealed thining of the epithelial lining, fibrosis while in those exposed to diesel there was loss of epithelial lining and loss of fibres. This is in consonance with another study, [56] in which exposure of the olfactory epithelium to gasoline revealed loss of the apical surface as well as cellular apoptosis. The main site of injury when the olfactory mucosa is liable to various volatile chemicals is the olfactory epithelial lining of the anterior dorsal medial meatus. [57] Xenobiotics may cause damage to the nasal epithelia which is activated by cytochrome P-450 pathway but it is the olfactory epithelium which appears to be the most susceptible to these toxicants. [55] The extent of olfactory damage may be connected to the timing, duration of exposure, concentration and intrinsic toxicity of the toxicant. [58]

Conclusion

The results from this study indicate that kerosene and diesel exposure is detrimental to Wistar rats as seen in the irritation of the eyes, levels of oxidative stress and the histopathologic changes in the olfactory epithelium.

Conflict of Interest

The authors declare no conflict of interest

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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