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HISTOLOGICAL CHANGES IN THE LUNGS OF ADULT WISTAR RATS FOLLOWING EXPOSURE TO PAINT FUMES

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ABSTRACT

Whether through employment, home remodeling, or through any contact, a large majority of us get exposed to hazardous paint fumes throughout our lifetime. The present work investigated the effect of paint fumes on the histology of the lungs. Sixteen adult male Wistar rats weighing about 130-160 g were used for the study. The rats were divided into four groups A-D of four rats per group. Groups A-C was the experimental animals while Group D acted as the control. Groups A, B and C were exposed to paint fumes for 8 hours daily for three weeks, four weeks and five weeks respectively. Group D animals were exposed to air for 8 hours for five weeks. The weight of the animals was recorded at the end of each week during the experiment. On the last day of exposure, the animals were sacrificed and the lungs were excised and fixed in 10% formal saline and processed. Tissue sections were stained with: Hematoxylin and Eosin for general histology and Periodic Acidic Schiff for type 2 alveolar cells. The alveolar cells of the treated groups undergo fibrosis compared to the control group and increase in the number of type 2 secretory alveolar cells. Exposure to paint fumes over time is dangerous to the lungs and can cause respiratory distress

Keywords: VOCs, Histology, Lungs, Alveoli, Type 2 alveolar cells

INTRODUCTION

Demand media (2011) wrote that whether through our jobs, home refurbishing or huffing to get high, a large majority of the population has been exposed to hazardous paint fumes throughout their lifetime. Both short and long term exposure to these fumes can and will most likely affect us in a variety of ways, which are more serious than we often expected.

Paints used in the home contain -potentially harmful chemicals such as -solvents and volatile organic compounds (VOCs). Ethylbenzene and toluene are two of the common biogenic VOCs found in paint (Kesselmeier and Staudt, 1999). Volatile organic compounds (VOCs) are organic chemicals that have a high vapour pressure at room temperature (Behr and Johnen, 2009).

Exposure to paint fumes that contain VOCs either during painting or in newly painted environment have been associated to nausea (Wang *et al.,* 2007), sensory irritation (Wolkoff *et al.,* 2006), respiratory distress, allergic and immune effects (Irigaray *et al.,* 2007*;* Mendell, 2007; Bernstein *et al.,* 2008). Even in Denmark, specialists have identified a neurological condition brought on by long-term exposure to paint solvents; they call 'PAINTER'S DEMENTIA' (Epstein, 2010). Song *et al.,* (2009) reported the death of some factory workers where nanoparticles were used in paint; they also reported serious lung problems in the other workers.

It is common knowledge that we get expose to paint fume on daily basis. There are however no well-documented cases of effect resulting from such exposure. The present work investigated the effect of paint fume exposure on the histology of the lungs using adult male Wistar rats.

METHODOLOGY

Animal handling: All animals used for this study have been treated in accordance with the ethics and guidelines of the Institutional Animal Care and Use Committee (IUCAC). Sixteen adult male Wistar rats of 9 weeks old weighing between 130-160 g were used for the study. They were obtained from the animal house of the Department of Anatomy, University of Ilorin, Nigeria. The rats were kept in well-ventilated conditions in the animal house of the Department of Anatomy and given normal rat feed and water *ad libitum.* The feeds were obtained from Bendel feeds Nigeria limited, Ilorin. They were left to acclimatize for 2 weeks.

Procurement of paint: Super lux gloss paint was purchased from a local Paint Store in Maraba Ilorin. The content of the paint was given as:

- Alkyd Resin
- Titanium Dioxide and Extender Pigments
- Not manufactured with lead or mercury containing materials

The VOC's content was given as 380 g/L (3.17 lbs/gal) maximum.

Animal grouping: After 2 weeks of acclimatization, the rats were randomly divided into 4 groups;

Group A animals were exposed to 20mL of paint fumes for 8 hours for three weeks

Group B animals were exposed to 20mL of paint fumes for 8 hours for four weeks

Group C animals were exposed to 20mL of paint fumes for 8 hours for five weeks

Group D animals were exposed to fresh air for 8 hours for five weeks

Mode of exposure: The paint fume was placed in an improvised fume chamber of diameter 75cm X 50cm X 30cm hole was bored on the cover of the chamber to allow little ventilation for breathing of animals. The animals were enclosed to paint fume in the chamber for 8 hours daily during the experimental period. After exposure the rats were returned to their cages.

Termination of treatment: On the last days of exposure to paint fumes in each experimental group, All rats were anaesthetized with ether (Sigma, MO), after which a thoraco-abdominal incision was made to exposed the chest and the lungs removed with forceps. Each lung was fixed in formol-saline.

Histological processing: Formol-saline-fixed lungs were dehydrated and embedded in paraffin wax. Eight micrometer-thick sections were cut on a rotary microtome; and sections were either stained by the Hematoxylin and Eosin (H&E) or periodic acid Schiff (PAS) as described by Bancroft and Stephens (1982). Images were captured with an MW1-HD2 digital microscope at magnification of ×400.

Statistical analysis; Results of the body weight was reported as Mean ± SEM. Statistical analysis was performed using ANOVA and $P<0.05$ considered statistically significant.

RESULTS

Body weight: The body weights of the animals were check at the end of each week during the experimental period. There was no statistical difference in the body weight of the paint fume exposed groups and the control group.

Histological findings: In the paint fume exposed rats, there was alveolar fibrosis which progresses as the week of exposure increases which was demonstrated by the H&E technique (Fig. 2). However, in the control rats, the lung showed well-stained alveoli with proper branching.

Similarly, PAS technique showed hyperplasia and necrotic cells in the paint fume exposed rats with highest effect seen in-group C (Fig. 3). Type 2 cells integrity was however preserved in the control group (Fig. 2).

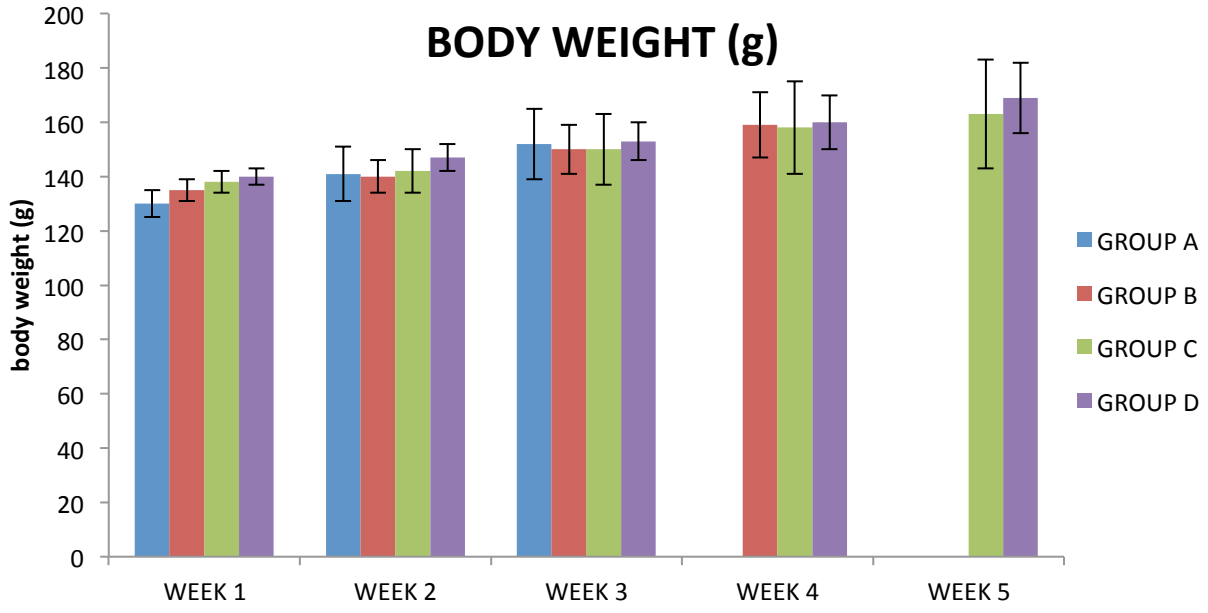
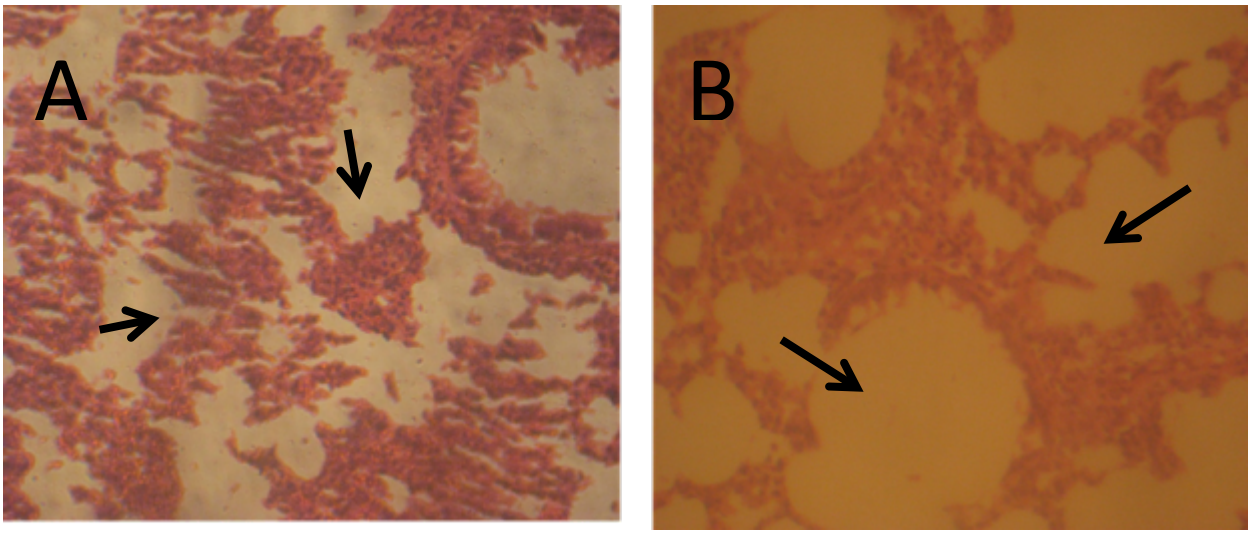


Fig. 1: Mean body weight of rats (in grams) at the end of each week. Values are Mean ± SEM.



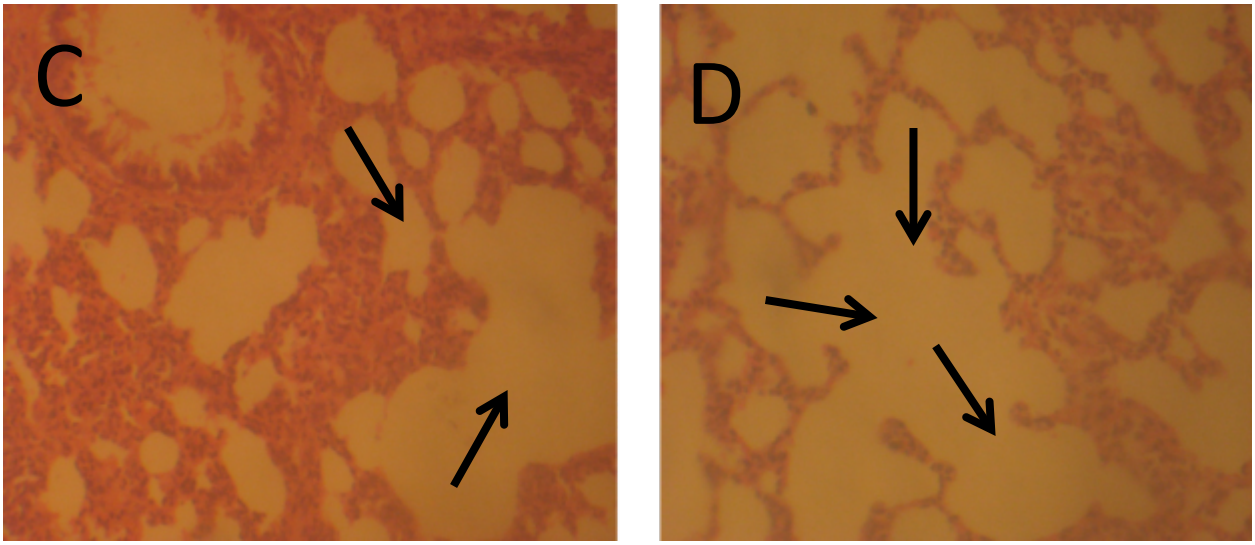
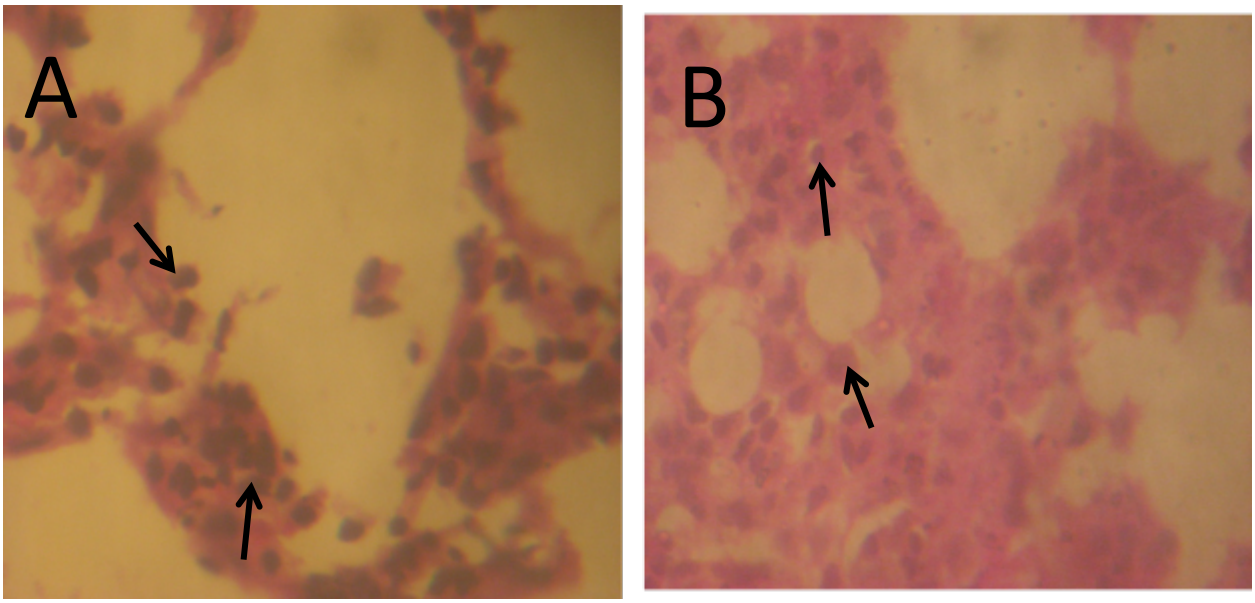


Fig. 2 Representative Photomicrographs of the lungs of the group A, B, C and D. alveolar fibrosis is observable in the A, B and C group (black thick arrow), in contrast to the well-stained branching alveolar in the group D. Haematoxylin and Eosin (Magnification, x100).



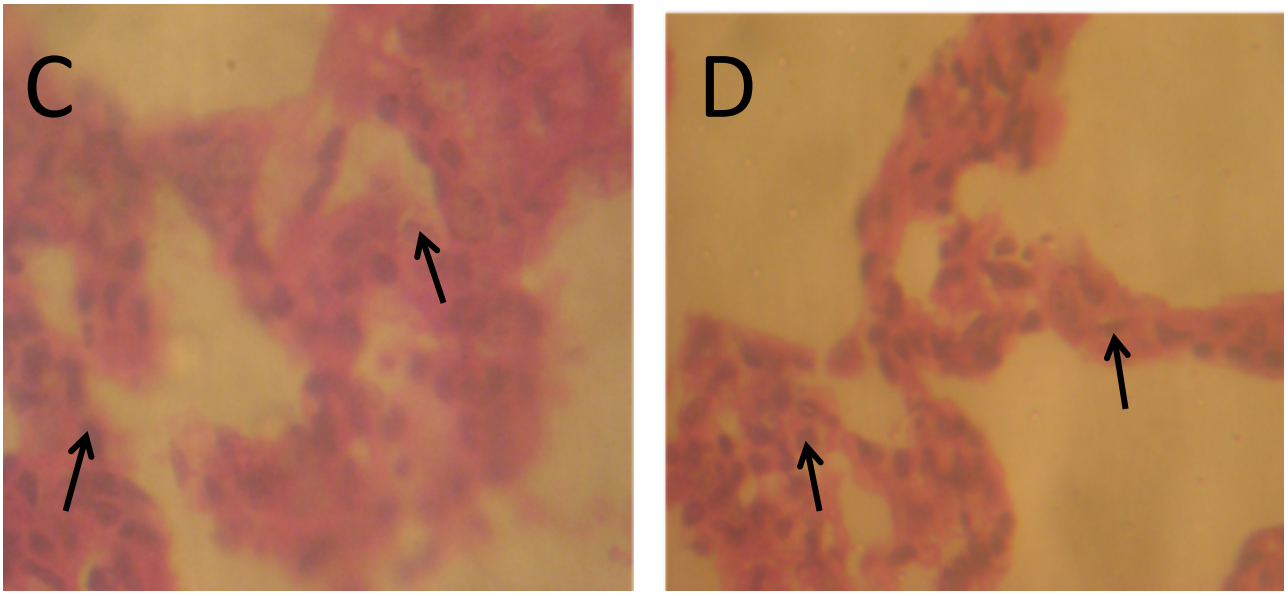


Fig. 3: Representative Photomicrographs of the lungs of the A, B, C and D groups. Hyperplasia and necrosis are observable in the type 2 cells of group A, B and C (black thin arrow); in contrast to the well stained type 2 cells in the group D. Periodic acid Schiff stain [PAS] (Magnification, x400).

DISCUSSION

Histologic evaluation of paint fume exposed rats over 3, 4 and 5 weeks demonstrated a temporal progression of pulmonary lesions that are characteristic of an acute toxic insult, these histologic lesions are similar to those described in other paint fume studies (Mauderly, 1977; Mendell 2007). Atkinson *et al.,* (1989) reported nausea, drowsiness and hepatotoxicity in a previously healthy man exposed to paint fume. Epstein (2010) wrote that Dr. Keith Prowse, of the British Lung Foundation explained that Inhaling paint fumes can exacerbate asthma and sinusitis. W.H.O (2010) reported that painters have 20 percent increased risk of cancers, particularly lung cancer.

In the PAS staining (plate 5-8), there is presence of degenerating type 2 alveolar cells, the number of these cells is highest in the group with highest level of exposure (plate 7). Abuelfadl *et al.,* (2010) reported similar finding when he shows degenerated Clara secretory cells in the lungs of car sprayer. There was a conspicuous lack of airway epithelial damage or infiltration of inflammatory cells. This would seem to indicate that the first pulmonary response to paint fume exposure is an unknown reaction centered on the pulmonary microvasculature.

The insignificant effect in the body weight implies that paint fumes have no effect on body weight although long term exposure will be needed to authenticate such.

The toxicologic effects of paint fumes seen in the present study maybe due to the presence of volatile organic compounds (VOC's) in the paint which are the chemicals that we humans are also allergic to (Demand media, 2011). Demand media (2011) also wrote that exposure to paint fume that are high in VOC's can damage organs like liver and the fragile lining of the lungs, this is similar what is seen in the paint fumes exposed rats using H&E staining (plate 1-3).

Duninho *et al.,* 2002 reported alveolar and interstitial flooding coupled with inflammatory cell infiltration and terminal airway epithelial degeneration in the lung of mice exposed to phosgene another known VOCs. The symptoms of VOC exposure have been reported to range from slight respiratory irritation to death (Ashley et al., 1996). Examples include; upper respiratory irritation found in workers exposed to styrene (NIOSH, 1983). Liver damage has been found among factory workers exposed to chloroform (Phoon *et al.,* 1983), and cases of carbon tetrachloride hepatotoxicity have been

previously reported in humans (Straus, 1954). Central nervous system depression has been associated with exposure to various VOC's like carbon tetrachloride (Cohen, 1957), tetrachloroethene (Rowe *et al.,* 1952), and toluene (Devathasan *et al.,* 1984).

Currently, our laboratory is working on the mechanism by which volatile organic compounds in paint fumes causes deleterious effect on the structure and function of the lungs.

Conclusion: These findings suggest that long term exposure to paint fumes should be avoided as they can cause loss of lungs function and serious pulmonary problems like lung cancers and sinusitis.

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