

HISTOPATHOLOGICAL EFFECTS OF PAINT EFFLUENT ON SWISS ALBINO MICE (*Mus musculus*)

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Abstract

Industrial effluents are seen as the most potential and hazardous waste containing compounds like heavy metals and nutrients that are toxic. In this study, the compositions of effluent from a paint industry were assessed along with its histopathological effects on the Swiss albino mice. The effluent was taken from Golden Key Paints found along Lagos State University (LASU) Road, Akesan, Lagos. Twenty (20) laboratory mice were obtained from Nigerian Institute of Medical Research, Yaba, Lagos. There were four mice (a male and three females) in each group comprising of five groups. Group one was the control group. They were given distilled water, while groups 2 to 5 had 25, 50, 75 and 100 % v/v of the paint effluent, respectively as the single source of water. By the 4th week, the control mice had live birth of 12 young mice (babies) while the mice treated with paint effluent had no birth. It was observed that at least a mouse died in each cage of the treated group. These observations could be attributed to the treatment concentrations. Histopathological examination of the tissues of treated mice showed severe necrosis and fibromatosis colli of the kidney, hepatic necrosis of the liver, lung atrophy, tubular atrophy of the heart, loss of myocardial striation and tissue hyalination due to the concentrations of the effluents. These results express the harmful effects of paint effluent on the treated mice and are sensitive index to changes in ecological conditions. This can serve as a good diagnostic tool in toxicological studies. People are therefore advised to handle paints with care and keep them away from children.

Key words: Hepatic necrosis, Tissue hyalination, Atrophy, Anomaly

Introduction

Industrial effluents are seen as the most potential and hazardous waste containing compounds that are toxic, heavy metals and nutrients (Dhevagi and Oblisami 2002). These wastes or effluents from urban areas and industries are largely disposed of in rivers, streams and lakes which are sources of water for agriculture and domestic purposes (Manunatha, 2008). Textile industries, for instance, release effluents

that can be solid, liquid or gas in the form of organic or inorganic substances (Brown *et al.*, 1996). Bhalero and Adeeko (1981) reported that of all the three forms of pollutants discharged by paint manufacturing company, the liquid effluents are by far the most significant. Industrial effluents are usually discharged

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indiscriminately into streams and rivers, which are sources of water for most domestic use, especially in the rural areas. Several chemical surveys of effluents from industries have shown that effluents from industries may enter the aquatic and terrestrial environment via effluents discharges (Mary and Dolor, 2007). Industrial chemicals have been strongly implicated in metal pollution of water bodies. High levels of zinc (Zn), lead (Pb), cadmium (Cd), nickel (Ni), copper (Cu) and iron (Fe) were found in the leaves of vegetables grown in domestic gardens near a Copper smelter in Australia (Bearington, 1975). A study by the World Bank (1990) also reported that large volumes of arsenics found in the surroundings come from leather Industries (leather pigments), landfills, coal mines, pit heaps, smelters, and antifungal wood preservatives. Effluents may bio accumulate in plants, and when such plants are eaten by animals or humans, it may have adverse effects on both somatic and germ cells (Oyewo, 1998). Friskesjo (1990) reported that high concentration of some chemicals totally inhibited growth and induced different degrees of growth retardation on the root length of *Allium cepa*. According to Oladele *et al.*, (2013) some manufacturing companies release effluent such as paint effluent unto plants leading to bioaccumulation of toxins in the plants or high deposits of contaminants in the soil. Njoku *et al.* (2015) observed chromosomal aberrations in *Allium cepa* after treatment with paint effluents. A number of toxicological evaluation methods can be employed both *in vitro* and *in vivo* to determine the

toxicity of a waste. Histopathological parameters can serve as an important diagnostic tool in toxicological studies (Calstus *et al.*, 2002). Although it has been reported by Oladele *et al.* (2013), that paint effluents have significant hematotoxic effect on Swiss albino mice. Much work are yet to be done on the effect of paint effluents on the histopathology of animals. Therefore, this study was intended to investigate the effects of paint effluent on the histopathology (Internal organs) of mice (*Mus musculus*).

Materials and Methods

The histopathological study was done in the Cell Biology and Genetics Departmental section of the animal house, University of Lagos. The paint effluent used in this study was taken from the discharge point of Golden Key Paints, along Lagos State University (LASU) Road, Akesan, Lagos. Albino mice (*Mus musculus*) were the test organisms. These were collected from the National Institute of Medical Research (NIMR), Yaba, Lagos.

Instrument/Equipment/ Analysis

Atomic Absorption Spectro-photometer (AAS) Model 969, Weighing Balance, pH meter and Conductivity meter were used. The procedures for physico-chemical analysis followed those described by Ajao (1985). It includes pH, conductivity, alkalinity and total dissolved solids. Samples of neat effluent from the Paint Industry collected were analyzed at the Environmental Analytical

Laboratory of the Chemistry department, University of Lagos for analysis.

Histopathological Examinations

The method described by Taylor *et al.* (2003) was used in the preparation of the internal organs for Histopathology examinations.

Procedure:

About 3-5 mm thick sample were selected from various organs (Liver, Kidney, Heart and Lungs) and processed for Histological demonstration. The selected tissues were fixed in 10 % formal saline for 5-10 hrs to ensure proper fixation and to prevent the tissue from autolysis and putrefaction. The fixed tissues were placed in different grades of alcohol (100 %-95 %-70 % alcohol) in order to completely remove water from the fixed tissue. The dehydrated tissues were treated with xylene at different levels to eliminate any extra alcohol from the tissue as well as to remove the link between the tissue and paraffin wax. Embedded tissues were mounted on the wooden block and the sections/ribbons were floated out using 20 % alcohol. The sectioned tissues were floated out in warm water at about 35⁰C and prepared for proper adhesion of tissues to the microscopic slides. The haematoxylin and Eosin Technique were employed in staining the prepared slides.

Results

The physico-chemical characteristics of the effluent used in this study is shown on Table 1.

Table 1: Physico-Chemical Parameters of Golden Key Paint Effluent.

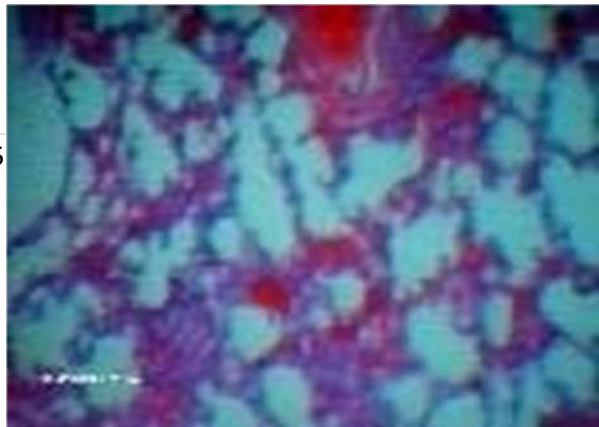
Parameter	Unit	Level Detected	FEPA'S Limit
pH		7.4	6-9
Colour		Blue	NI
Conductivity	µscm ⁻¹	780.0	NI
Alkalinity	mg/l	*150.0	20
Total Dissolved Solids	mg/l	390.0	2,000
Zinc	mg/l	35.6	< 1.0
Copper	mg/l	14.3	< 1.0

NOTE: NI - Not Indicated.* - Found with concentrations above legal limitsPermissible limits for drinking water (FEPA,1991). (www.epa.gov/safewater/mcl.html).

Histopathological Investigation

Histopathological examinations of the mice treated with paint effluent showed that paint effluent is very deadly to the animals. At the end of the treatment, the hearts of mice treated with 50 % concentration of the effluent showed loss of myocardial striation, loss of cellular outline, tubular atrophy and severe tissue hyalination (Figure 2). The lungs of treated mice showed small and large dark brown deposits of paint pigment leading to cellular changes (hyperplasia), interstitial oedema, broken bronchus and scanty bronchus terminal (Figure 4). Treated mice livers had partial hepatic necrosis, cellular

oedema and tissue hyalination (Figure 6). The



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kidneys of treated mice showed tubular atrophy, cellular necrosis and fibromatosis colli (Figure 8).

Figure 1: The Heart of untreated (control) Mouse. X 40. No tubular atrophy or loss of cellular outline.

On the other hand, none of these anomalies were detected in the untreated (control) mice (Figures. 1, 3, 5 and 7).

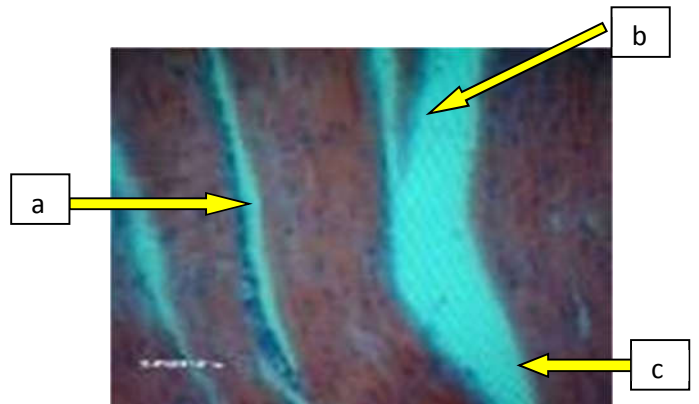


Figure 2: The Heart of mouse treated with 50 % effluent concentration showing (a) Myocardial striation, (b) Tubular atrophy and (c) loss of cellular outline. X 40.

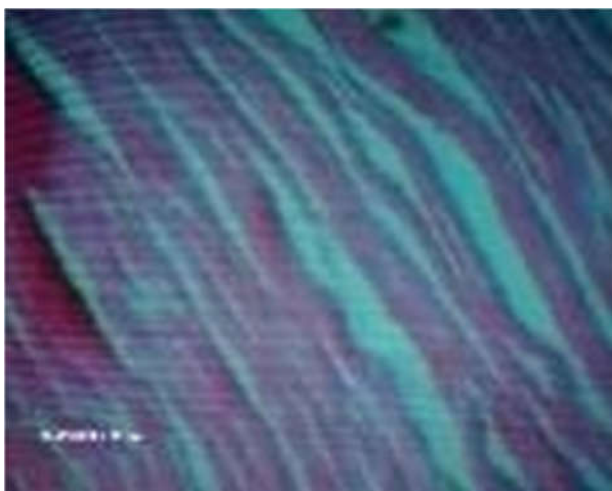


Figure 3: The Lung of untreated (control) mouse. X 40

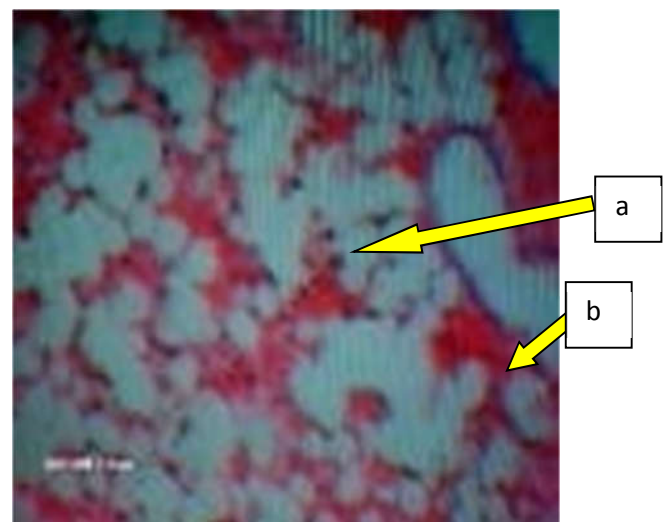


Figure 4: The Lung of mouse treated with neat effluent showing (a) dark brown deposits, leading to cellular changes (hyperplasia), (b) bronchus broken with scanty

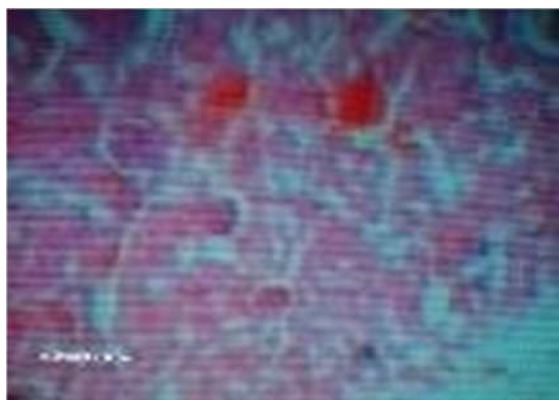


Figure 5: The Liver of untreated (control) mouse X40

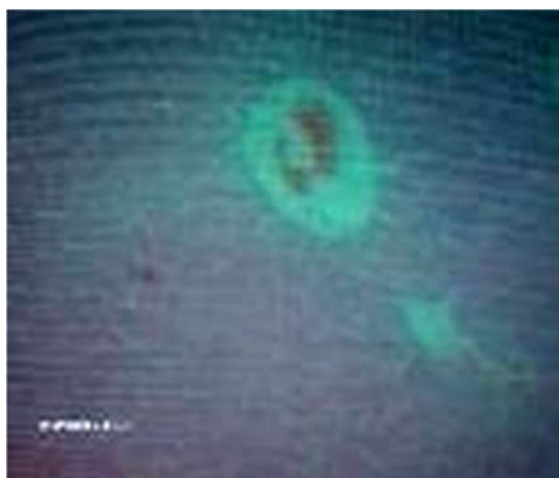


Figure 7: The Kidney of untreated (control) mouse. X 40

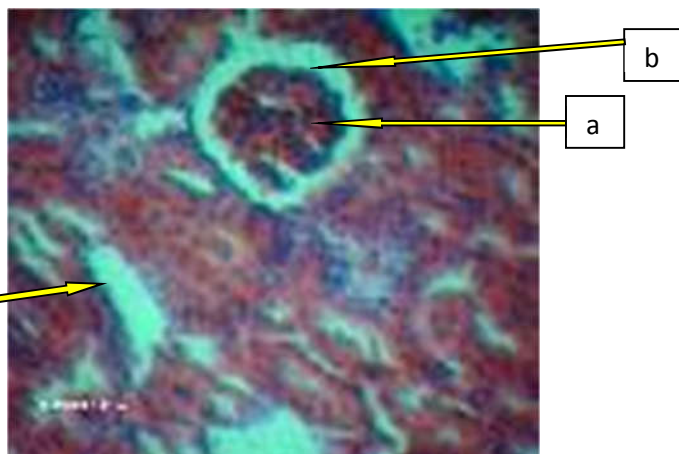


Figure 6: The Liver of mouse treated with 75 % effluent concentration showing (a) dark brown deposits, (b) partial hepatic necrosis and (c) tubular atrophy. X 40

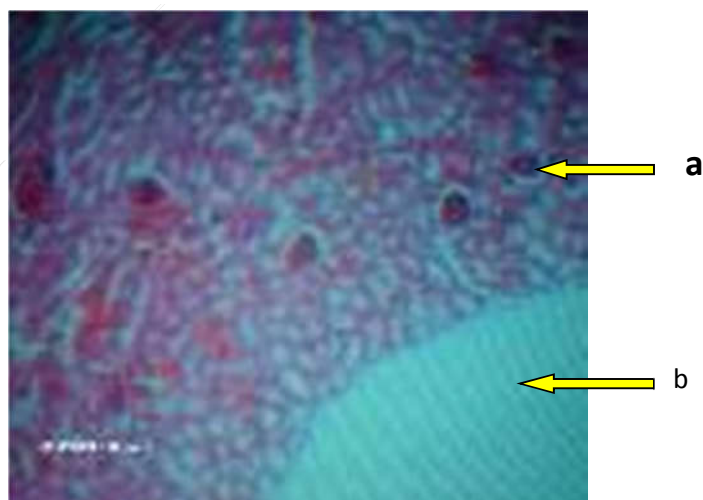


Figure 8: The Kidney of mouse treated with neat (undiluted). (a) dark brown deposits, (b) tubular atrophy. X 40

All the above observations showed that paint effluent is highly toxic. At the end of the 4th week of treatment, the hearts of mice treated with 25% to neat concentration of the effluent showed loss of myocardial striation, loss of cellular outline, tubular atrophy and severe tissue hyalination. Also, the mice used as control had live birth of 15

young mice (babies) while the mice treated with paint effluent had no birth. At least one mouse died in each cage of treated group (part of the results).

Discussion

Oladele *et al.* (2013) in the hematological examinations of mice have also observed that

paint effluent led to anaemic condition, infection and reduced growth rate. The Paint Effluent had significant effect on the treated mice. This can be attributed to the toxic substances such as heavy metals contained in the effluent. The fact that the internal organs of treated mice marked histopathological changes confirmed that paint effluent is pathogenic. The kidneys showed fibromatosis colli, severe necrosis and cellular oedema. Where there are: (a) Replacement of muscle fibres by hyalinized fibrous tissue and (b) All over the lesion there are scattered residual muscle fibres showing noticeable degenerative and reactive changes (atrophy, swelling, loss of cross striations). There is little cellular pleomorphism or mitotic activity. The lungs showed atrophies and interstitial oedema. The livers showed hyalination of tissue, hepatic necrosis and cellular oedema. The hearts also revealed some changes such as myocardial striation being loss along with the cellular outline and tissue hyalination. This also agrees with the work of Akinola *et al.*, 2008, where the histopathological investigation of internal organs of rats exposed to cement dust containing heavy metals such as the paint effluent under study showed interstitial oedema, hyalinization, loss of myocardial striations, progressive necrosis and atrophy of the cells of the heart. According to Serina (2017), oral administration of silk dye waste effluent led to severe histopathological

influence on the stomach and intestine of Swiss albino mice. He observed marked atrophy of musculature, disintegration of mucosal epithelial cells with cytoplasmic vacuolization, nuclear pyknosis and nuclear fragmentation. As a result, it can be said that histopathological parameters are delicate guide to changes in ecological conditions and can serve as a very good diagnostic tool in toxicological studies (Calstus *et al.*, 2002).

Conclusion

This study shows that exposure to industrial effluent is highly dangerous to human health (given this short period of mice exposure to paint effluent). The study indicated the possible hazards of paint effluent to the internal organs of animals. Results from this study will serve as a part of the preliminary toxicological studies. Following these observations, more work need to be done to evaluate the influence of paint effluents on the reproductive system and general body physiology on prolonged exposure.

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