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Toxic Effect of Inhalation Polyurethane in Lungs, Liver, and Kidneys Fume in White Male Mice

Abstract - In this study, the toxic effect of exposure to inhalation of polyurethane resin fumes by White Male Mice was evaluated over 36 days. The study included the use of the inhalation method in an exposition room filled with the fume chemical to a period of 10, 20 and 40 minutes per day, and were then transferred to the fumigant section of the animal house. The organs of the mice was dissected, and the change of the weight determined. The lungs, liver, kidneys tissues of the exposed mice showed typical structural structures when compared with the control group. The liver cells of the mice group exposed to polyurethane were revealed to expose to some hydrolysis and led to an increase in their size. In terms of lung, tissue was characterized by the presence of interstitial infiltration and bloody congestion. It was observed that water degeneration of the lining cells of the parts of the urinary tracts of the kidney with the infiltration of inflammatory cells in the interstitial tissue. These results suggest that exposure of white mice to polyurethane coating fumes may cause observed harmful effects and cause serious health problems to their liver and lung.

Keywords- Volatile organic compounds; Histological study; body weight; paint thinner.

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1. Introduction

Volatile organic solvents (VOCs) are organic chemicals evaporated from heated equipment's parts, causing the emission of large numbers of particles or sublimation of fluid or rigid form of compound to the surrounding air. They are slowly removed from the paint and enter the air and have been found to misuse a variety of chemicals as crackers, which include cleaning fluid, spray paint, paint thinners, and nail polish remover [1]. Among them are a wood pigment used in wood paint, which is composed of solvent or coloring materials. The contents of the dye may not be well dissolved in the compound, but are suspended, so the compounds may not be real solvents. The compound may often be water, alcohol, petroleum distillation products or, finishing agents such as vapors, varnishes and, polyurethane. Colored pigments do not penetrate deep into the wood pores, and may disappear dramatically when the final layer is removed. The products used in wood coatings are polyurethane and nitrocellulose, where polyurethane products include isocyanate. The toxicity can occur from unintentional or intentional inhalation of excessive amounts of thinner veneers, ingestion, or absorption across the skin leading to serious multi-organ toxicity and death [2]. Exposure to paint thinners such as benzene, toluene, and styrene-butadiene was observed, with elevated

red blood cells, hemoglobin, platelets, and white blood cells [3]. For this reason, we chose the respiratory system to examine the effects of Varnish vapors components on these devices in laboratory mice as an animal model by assessing damage to the tissue of these organs, furthermore, because vapors from regular exposure to the user reduce lung function by about 25% less than those who do not use these vapors [4].

2. Materials and Methods

I. Test Animals

Experiments were performed on 130 healthy male white male rats, which are aged (5-6) weeks and have weights of their bodies (21-29) grams. Laboratory animals' dissection was performed at the Raze center laboratories / Ministry of Industry and Minerals. The mice were randomly separated into 12 groups. Each group contains 15 animals. The animals were kept in cages (diameter 30 cm x 15 cm x 15 cm) covered with a mesh cover and preserved at $25 \pm 2^\circ\text{C}$. They were washed, with hot water and alcohol orderly, then sprinkled with wood shavings to keep them dry and changed from time to time. Cages were under standard household conditions where moisture was $30 \pm 10\%$ and 12 ± 2 hours (light /day). The mice had access to feed and water.

II. Test Compounds

Polyurethane obtained from the local market and used without any special treatment.

III. Experimental Design

The animals were divided into 12 groups for each group 15 mouse. The groups divided according to time of exposure to sealer polyurethane B1 (10 min.), B2 (20min.), B3 (40min.), and control group. Camera from Canon, Japan was used with zoom power of $\times 100$, $\times 250$ [5].

IV. Histological Studies

The animals were dissected the next to the end of the exposition time. Their lungs of were injected with 0.5 ml (formalin 10%) using a 1 ml syringe to extend the lungs to approximately physiological size, thus facilitating histological examination as well as its usefulness in the rapid stabilization of thin tissue. The lungs, liver, and kidneys were dissected and washed with normal saline solution (0.9% NaCl) to clear the blood and maintain it in 10% formalin to study histological changes. The respiratory system (lungs), liver, and kidneys tissues of the mice control group and the vapors exposed mice groups, have been treated for the histological examination according to the standard methods. This method includes dehydration, clearing, infiltration, embedding, staining, and used respectively for the preparation of the slides. The dry tissue starts by increasing alcohol 60, 70, 80, 90, 100% Xylene is used for filtration and is guaranteed in Paraffin wax. The sections mixed with a feach were cut to 5 micrometers using a rotary microtome, then dyed with Hematoxylin and eosin and examined under the microscope.

4. Results and Discussion

I. Bodyweight

Results in Table 1 showed a decrease in the weight of exposed mice to polyurethane fumes during the exposure period 12 day. At the same conditions, the group exposed to polyurethane coating, there was an apparent decrease in the weight from (24.09 \pm 0.44) to (23.09 \pm 0.44) upon exposure period of 20, 40 minutes, respectively. The results presented in Table 2 showed significant differences of ($P < 0.05$) in the weights of mice according to the exposure period (24) days; there was a gradual decrease observed at a maximum of 40 minutes (22.55 \pm 0.45) in-group B3. The results in Table 3 showed that there were significant differences ($P < 0.05$) in the weights of mice according to the exposure period (36) days were showed a gradual decrease in all exposure

times with a maximum exposure period of 40 minutes (20.55 \pm 0.48).

Group	Exposure/ minute	Body weight (gm)		LSD value
		Weight of laboratory mice before exposure	Body weight of laboratory mice after 12 days	
B1	10	25.8 \pm 0.57	25.79 \pm 0.49	3.08 NS
B2	20	24.7 \pm 0.55	24.09 \pm 0.44	3.55 *
B3	40	23.7 \pm 0.38	23.09 \pm 0.44	4.17 *
Control group		25.2 \pm 0.46	28.6 \pm 0.50	

Table 1: The average body weight of laboratory mice exposed to polyurethane after 12 days

Table 2: The changes in body weight of laboratory mice exposed to polyurethane after 24 days

Group	Exposure / minute	Body weight (gm)		LSD value
		Weight of laboratory mice before exposure	Body weight of laboratory mice after 24 days	
B1	10	25.8 \pm 0.57	25.6 \pm 0.42	4.07 *
B2	20	24.7 \pm 0.55	23.55 \pm 0.61	3.38 *
B3	40	23.7 \pm 0.38	22.55 \pm 0.45	4.61 *
Control group		25.2 \pm 0.46	31.8 \pm 0.48	

Table 3: The average body weight of mice exposed to paint after 36 days

Group	Exposure / minute	Body weight (gm)		LSD value
		Weight of laboratory mice before exposure	Body weight of laboratory mice after 36 days	
B1	10	25.8 \pm 0.57	24.6 \pm 0.52	5.81 *
B2	20	24.7 \pm 0.55	22.05 \pm 0.51	6.33 *
B3	40	23.7 \pm 0.38	20.55 \pm 0.48	6.08 *
Control group		25.2 \pm 0.46	35.7 \pm 0.51	

The conclusion of this study is inconsistent with that obtained by Dahl et al. [6], they found that continuous exposure to polyurethane vapor for 12 weeks directly causes a decrease in body weight. Generally, it affects the whole body, leading to the cessation of many activities cellular tissue within the body tissue, causing anorexia. The present study confirms that direct exposure to different coatings causes anemia directly associated with weight loss due to oxidation of fat found in cellular tissues and blood cell tissues. The effect may be due to the self-oxidation of coatings and free oxygen-oxygen reaction with

cellular fat. The study also shows that the different histological changes led to functional changes in liver and lung tissues, which give a strong indication of the toxicity of different coatings that may be the result of the oxidative stress caused by the direct effect on the activity of the cells due to hemolytic and suppression of blood activity.

II. Histological Studies

Lung: tissue was examined during the dissection operation, and the following signs were observed: hypertrophy, necrosis, pulmonary edema, and congestion. The normal structure of bronchioles has no cartilage rings; the epithelium is reduced to cuboidal, and there are many lymphoid nodules at the base of the epithelial layer. The bronchioles lead to alveolar duct, which opened in the alveolar sac that consist of alveoli, the lung (parenchyma tissue) showed numerous alveoli, each one composed of a single layer of squamous epithelium between alveoli there is a slim layer of conjunctive tissue and numerous capillaries which padded with simple squamous epithelium. As shown in Figure 1.

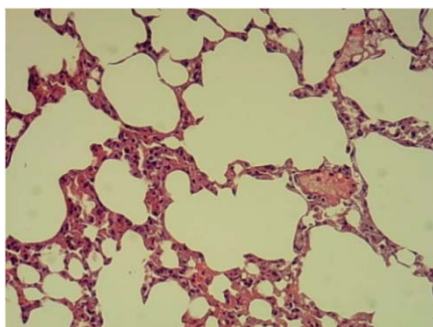


Figure 1: Histopathology of the Lung in the Control Mice Group Note the pulmonary hematopoietic and epithelial tissue of the lung (H & E x100).

While exposed to the polyurethane that show Infiltration of inflammatory cells in part of lung treated with polyurethane maybe take into consideration as the inflammatory response plays an important function in defense of biological systems against pathogens or invasive substances, clear cellular debris or releasing tissue repair mediators. However, after exposure to specific toxicants, inflammatory cells release mediators, causing tissue injury, and that mainly seen after treated with different concentrations of polyurethane, as in Figure 2.

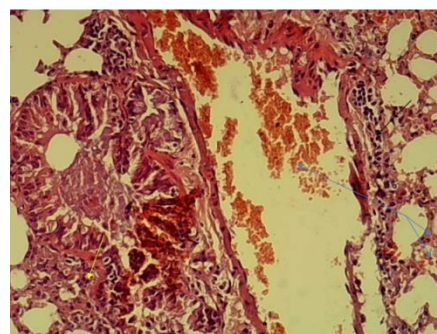


Figure 2: a histological section of the lung in a group of mice exposed directly to the polyurethane (B3) after 36 days show that single-core cells are encased around the blood vessel with cellular infiltration and bloody congestion (H&E Stain 200).

Liver:

Figure 3 offers the typical structure of the liver, which consist of several hepatic lobules. The center of the lobule is the central vein, each lobule discrete from other by thin connective tissue septa, which is consist portal area. The portal area includes the hepatic artery, portal vein, and bile duct. The hepatic lobule contains hepatic cells (hepatocytes) arranged as cords radially surround the central vein, and these cords discrete from each other by the vascular anal (sinusoids). The sinusoids house an essential part of the liver's defense system and populated by numerous types of fixed macrophage (kupffer cells).

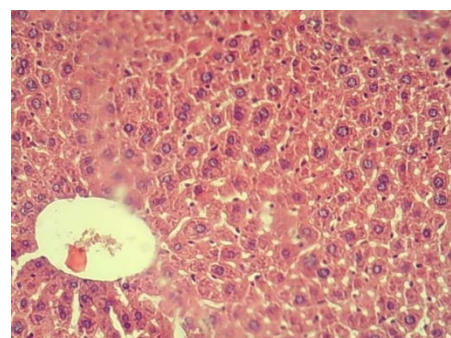


Figure 3: Histopathology of the liver in the control mice group The hepatic arrangement of the hepatic cells is observed with the hepatic pockets of the lobules and their distribution towards the hepatic vein (H & E 200).

While liver exposed to the polyurethane that shows Inhalation of polyurethane may produce pathological changes that lead to impaired liver function, which intervene with the secretion of plasma proteins and especially lead to lower blood osmotic pressure with subsequent lower drainage of tissue caused congestion [8,9]. In addition, the liver showed congestion of central vein, and fatty changes in the cytoplasm of the hepatocytes, depletion of glycoprotein and

increased inflammatory cell infiltration in the hepatocytes [10]. Cellular infiltration in the hepatic tissue was caused due to increased permeability of blood vessels. That occurs when the contraction of the endothelial cells of blood vessels in response to certain chemicals or because of the loss of particle desmosome lies between the endothelial cells allows the passage of blood vessels. Moreover, blood vessels were expanded lead the rush of inflammatory cells from the center to the periphery endothelial lining the blood to find way out of the vessel [11], as in Figure 4.

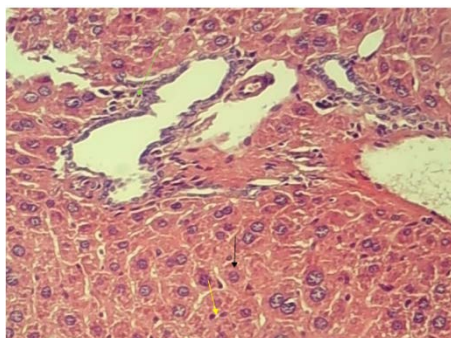


Figure 4: A histologic section of the liver in a group of mice exposed to polyurethane (B3) after 36 days, where the hydrolysis of the liver cells is observed, leading to large hepatic cells in the absence of gibbons and increasing the effectiveness of cofer cells with inflammation of inflammatory cells in the pylori region (H & E 200).

References

- [1] K.V. Akpan, T.O. Sogbanmu and A.A. Otitoloju, "Effects of Volatile Organic Solvents Inhalation on Hematological and Histological Indices of Mus Musculus," *Curr Advanc Environ Sci*, Vol. 2, No. 2, pp. 46-51, 2014.
- [2] K.C. Patrick-Iwuanyanwu, E.A. Okon, N.W. Areh, and M.O. Wegwu, "Toxicological Effect of Inhalation Exposure to Nitrocellulose Paint Thinner Fumes in Wistar Albino Rats," *Archives of Applied Science Research* Vol. 5, No. 1, pp. 264-269, 2013.
- [3] F.U. Igwe, H.W. Wosu, and C.U. Ogunka-Nnoka, "Hepatic Effects of the Dermal Absorption of Emulsion Paint Solution by Albino Rats," *International Journal of Scientific and Engineering Research*, Vol. 8, No. 2, pp. 425, 2017.
- [4] H.M. Aslam, S. Saleem, S. German, and W.A. Qureshi, "Harmful Effects of Shisha: Literature Review," *International Archives of Medicine*, Vol. 7, No. 1, pp. 16, 2014.
- [5] J.D. Bancroft and M. Gamble, "Theory and practice in histological techniques," Churchill-Livingstone, 6th ed, Edinburgh, 2008.
- [6] A.R. Dahl, I.M. Grossi, D.P. Houchens, L.J. Scovell, M.E. Placke, A.R. Imondi, G.D. Stoner, L.M. De Luca, D. Wang, and J.L. Mulshine, "Inhaled Isotretinoin (13-Cis Retinoic Acid) is an Effective Lung Cancer Chemopreventive Agent in A/J Mice at Low Doses, A pilot study," *Clinical Cancer Research*, Vol. 6, No. 8, pp. 3015-3024, 2000.
- [7] F.E. Uboh and S. Ufot, "Withdrawal from Exposure Reverses Hematotoxicity and Hepatotoxicity Caused by Oral Exposure to Nitrocellulose Thinner in Male Rats," *J. Clin. Toxicol*, Vol. 3, No. 5, pp.2-6, 2013.
- [8] I. Lacroix, M. Lapeyre-Mestre, H. Bagheri, A. Pathak, J.L. Montastruc, and Club de Reflexion des cabinets de Groupe de Gastro-Enterologie (CREGG) and General Practitioner Networks, "Nonsteroidal Anti-inflammatory Drug-induced Liver Injury: A Case-control Study in Primary Care," *Fundamental & Clinical Pharmacology*, Vol. 18, No. 2, pp. 201-206. 2004.
- [9] B. Young, W. Stewart, D. Odowd and P. R. Wheater, "Wheater's Basic Pathology a Text, Atlas and Review of Histopathology," Churchill Livingstone/Elsevier 5th ed., Edinburgh, 2011.
- [10] A.H. Silva, C. Locatelli, F.B. Filippin-Monteiro, P. Martin, N.J. Liptrott, B.G. Zanetti-Ramos, and A. Owen, "Toxicity and Inflammatory Response in Swiss Albino Mice after Intraperitoneal and Oral Administration of Polyurethane Nanoparticles," *Toxicology letters*, Vol. 246, pp. 17-27, 2016.
- [11] L.M. Majeed and A.K. Abass, "Biological Effects of Different Doses of Piroxicam in Albino Males Rats," *Iraqi Journal of Science*; Vol. 58, No.2C, pp.1009-1019, 2017.