IOSR Journal of Environmental Science, Toxicology and Food Technology (IOSR-JESTFT)
e-ISSN: 2319-2402,p- ISSN: 2319-2399.Volume 11, Issue 10 Ver. II (October. 2017), PP 26-38
www.iosrjournals.org

Exposure to Airborne Particles and Organic Solvents among Painting Workers

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Abstract: *Vehicle and furniture painting workers are artisan that could be occupationally at risk hazardous attributed to the chemical composition of paints. The present study was focusing on occupational air pollution found in spray painting workshops to evaluate the health hazards that might generate from paints. Hence, the prospective study was conducted to demonstrate the risk health effects to airborne particles and solvents of painting on lung functions of exposed painters in the car and furniture painting workshops, as well as compared with a non-exposed control. The results indicated all painters had significantly lower % predicted lung function (FVC, FEV1, and FEV1/FVC) compared with the non-exposed controls. Although, the workers in the car painting workshops had more significant decrease in pulmonary function than those in painting furniture workshops, there was no a significant difference in lung function parameters between the both of them. These findings observed also the rats that exposed to spray painting in the car and furniture painting workshops, induced changes in histopathology of trachea and lungs when compared with controls. These pathological changes were related to increasing duration of exposure. Therefore, the present study is to consider the health implications of exposure to paint. Hence great attention must be also paid to environmental protection in planning future social and economic development for exposure to airborne particles and organic solvent, especially spray painting exposure.*

Keywords: *Health risk, Pigment and solvents, Respiratory functions, Spray Painting*

Date of Submission: 26-09-2017 Date of acceptance: 10-10-2017

I. Introduction

Painting is the art of applying a liquid material release in mod of aerosol or vapour and directed into a surface to produce the required thickness film [1, 2]. Spray painting of automobile, furniture is one of the major activities of painting which leads to health hazards for exposure workers and highly toxic solvent [3, 4]. Paint is a simple mixture of ingredients including; pigments like acrylates, a binder that is hold the pigment and allows it to spread, solvent (isopropanol, toluene, isocyanates, xylene, and white spirits) and additives like metals [5]. Paints are manufactured from organic and inorganic materials that need to be strictly controlled to make these products environmentally safe [2].

Heavy metals like; lead, cadmium, antimony, copper, zinc, chromium, cobalt, arsenic and others, which are the most polluting metals, may also be encountered in paints as inorganic coloured pigments [6]. They are dangerous because they accumulate in our bodies causing several effects on the human systems including the pulmonary system [7, 8, 9].Solvents are widely used to keep paints in liquid form for easy application. They found as components of cleaning agents, in manufacturing of dyes and textiles, pharmaceuticals, pesticides, printing inks, adhesives, polishes, varnishes glues and spray paints [10].

In the past few decades, organic solvents like benzene, toluene and xylene have become increasing potential health hazards because of their widespread industrial use in the production of paints, glues and solvents [11].The millions of workers worldwide are at the risk of organic solvents in paints. Occupational solvents are atmospheric pollutants that absorbed into the human body either through inhalation of excessive amount of paint thinner fumes, ingestion or transdermal absorption which results in serious multi-organ toxicity, metabolic disturbances, pathological changes in various tissues and sudden deaths [12, 13].

These may cause asthma and bronchial hyper responsiveness, pulmonary impairment and high risk for development of dysfunction in other organs [14, 15].However, high exposure of acute poisoning can cause inflammation and hemorrhage in the lungs which may be lead to death. Emphysema and reduction in lung function have also been linked with exposure to paints [16, 17, 18].

Spray Painting is one of the greatest source and contributor of occupational exposure to isocyanate which is the causes of painter's lung worldwide [8, 19]. Xylene produces dose-related respiratory depression and respiratory tract irritation [17]. The acrylates and methylacrylates are associated with difficulty in breathing and irritation of throat, nose and lung passages [20].

There are few researches considering occupational air pollutants exposure in small scale workshops; like airborne and VOC among workers [21, 22, 23].Therefore, the purpose of a study conducted was to estimate the exposure risk related to specific processes. The risk of continued occupational exposure to spray painting on the lung functions of painters was determined in the car and furniture painting workshops under the conditions obtainable in workshops, and compared with non-exposed controls. The histopathological changes of the respiratory system; trachea and lung of rats encountered in the painting workshops with special reference to dust and organic solvents effects were also examined compared with controls.

II. Materials And Methods

Repair car and furniture painting is typically and aerosol a solvent-intensive process; the nature of solvent exposures among workers in this industry is VOC. The work processes are included polyurethane molding, spray painting, lacquering, and gluing in the painting workshops. During the process of spray-painting the painter is exposed to all the chemicals which are atomized. Though, components of the paint can be absorbed through other routes like skin and mucous membranes, inhalational route [13].

Environment and Painting Processes

The study was conducted in ten of car and 10 of furniture painting workshops in Al-Mansoura and Damietta city, Egypt, respectively. All spray painting workshops have small size and scattering within urban area of the city. The workshops are occupied the ground floor of the building. The areas of the selected workshops were limited; varies from 20 m^2 to about 96m^2. There is no exhaust ventilation in the most of the selected workshops. Spray painting can be done via an airless spraying, compressed air spraying or by electrostatic spraying. In these workshops, painters used the compressed air method.

Potential Health Impacts

2.2.1. Study of Population Characteristics

The study was performed on 146 individuals; 30 unexposed subjects (controls) and 116 spray painters exposed to painting for at least 8 hours a day. The painters were all nonsmoker males; 55 workers selected from 10 car painting workshops with age ranging from15- 60 years old with a mean 36.00 ± 2.45, and 61 workers selected from 10 furniture painting workers. A self-structured questionnaire was used to obtain information about, personal data (life style, respiratory and smoking history, headache, and anorexia, histology of liver, kidney and blood diseases, and skin and eye complaints), environmental chemical exposure, duration of exposure to paints, and the use of protective equipment. Painters in all workshops did not use a respirator protection and the poor maintenance of ventilation systems and dust control might have potential health hazards.

2.2.2. Pulmonary Function Test

The pulmonary ventilator functions were measured for all painters and control subjects in the standing position in the morning before starting the work to ensure a minimum recovery of a period of 24 hours. These functions were done by using a calibrated vitalograph spirometer two times per day. The best of three technically acceptable values was recorded [24].

The pulmonary ventilator functions were to determine Forced Vital Capacity (FVC) and Forced Expiratory Volume per second (FEV1) and FEV1/FVC were estimated automatically. FVC and FEV1 /FEV1% were used as measures of pulmonary function [25].

2.2.2.1. Statistical Analysis

The statistical analysis was performed using SPSS (version 13). This represents the descriptive statistics. Statistical significant differences were computed t-test and used for the comparison of means. A probability of 0.05 or less was considered as significant. Correlation coefficients as well as the derived equations from the relationships between the related parameters were also calculated.

2.2.3. Experiment Design

In the present study, forty five male albino rats (Rattus Norvergicus Wistar Strain), aged 50 days (6 to 7 weeks), weighing about 100-130 gm were used. They were housed and maintained in stainless cages with free access to tap water and food. They were divided into three groups (15 rats for each group); control group not exposed to inhalation paint fume. The rats in groups 2 and 3were exposed to inhalation of paint fumes for 14, 30 and 60 days in car repair painting group and furniture painting group. Rats were sacrificed at 14, 30 and 60 days post-exposure to painting inhalation (5 rats for each survival period). Trachea and lungs were excised, cleaned and small pieces. These tissues were immediately fixed in 10% neutral buffered formalin solution, dehydrated in

graded alcohol, cleared in xylene, then embedding in paraffin wax. The sections of 5um thickness were stained with haematoxylin and eosin. The slides were left to dry before histochemical investigations [26].

III. Results And Disscussion

3.1 Demonstration Spray Painting Exposure on Lung function of Painters at Studied Painting Workshops

Table 1 represents the comparison between physical parameters of Paint workers exposed in car and furniture paining workshops and as well as an unexposed group. It showed that all the painters were all nonsmoker males; 55 workers selected from 10 car painting workshops with age ranging from15- 60 years old with a mean 36.00 ± 2.45, and 61 furniture painting workers of nearly the same age (ranges from 19-59 years old with a mean 36.33 ± 1.96). The age of the 30 unexposed subjects (control group) ranged from 20-53 years, with mean of 34.95 ± 2.05 year. The duration of exposure was (3 to 51 years). The mean duration of exposure was (19.50±6.46 years)) for car painting and ranged from 10 to 51 with mean of (21.97±7.58 years) for furniture workshops. There was no significant difference in mean± S.E had been found among all studied groups in respect of age and duration of exposure.

Table (1): Demographic Characteristics of Controls and Paint Workers.

Parameters		Groups			
		Controls	**Car painting workshops**	**Furniture painting workshops**	p. value
Number		(n=30)	(n=55)	(n=61)	
Sex:	**Male%**	100	100	100	
Age (years)	**Mean± S.E**	34.95±2.05	36.00±2.45	36.33±1.96	*P>0.05*
Duration of exposure ((yrs)	**Mean± S.E**	----------	19.50±6.46	21.97±7.58	*P>0.05*

**S=Significant at P<0.05.* *NS=Not significant at P>0.05.*

Table 2 represents the comparison between pulmonary function parameters of Paint workers in car and furniture paining workshops of airborne particles and VOC exposed as well as an unexposed group. It showed that the comparing with that reported in controls, all workers exposures of spray painting workshops had significantly lower percentage predicted lung functions (p<0.0001)compared with the non-exposed controls. The findings of significantly low FVC, FEV1 and FEV1/FVC ratio suggested of an obstructive pattern. Obstructive airway disease was identified as a decrease in the FEV1/FVC ratio to less than 80% according to American Thoracic Society [27].

The adverse health effects of spray painting are well known, but there is a very little documentary evidence to support this knowledge. Present results have demonstrated the significant correlation between pulmonary functions of workers exposures indicate that spray painting show reduced on pulmonary function of painter's workers. This may be attributed to the high level of spray painting that release dust and irritating organic solvents chemicals in car and furniture painting workshops. Besides abstaining the workers from use of personal protective equipment might be the reason for deterioration of lung function. Similar observation by Metwally et al. [28]; Mandal and Majumdar [29] and Mølgaard et al. [30] observed the high solvent exposure workers resulting in further deterioration in lung function parameters due to longer duration of exposure. Siddanagoudra et al. [31] and Aribo and Antai [32] demonstrated again a significant reduction in FVC and FEV1 (P<0.001) of exposure workers to paint, suggestive of obstructive lung pattern comparing to workers had normal lung functions. They revealed that spray painters developed reduced lung functions impairment and respiratory symptoms correlated to paints concentration and with exposed duration. Also, safety attitude of workers in the industry was very poor and might have contributed to the decline in lung function.

As shown in Table 2, In spite of exposure to spray paints within two types of workshops, the obtained results showed that the effect of painters' exposures on predicted decrease in lung function parameters were more prominent among the car paining workshops activities compared with painters in furniture painting workers. Car paint fume exposures are considered at high risk of respiratory impairment due to the paints foam and volatility of the chemicals of the paint [15]. This similar with Abuelfadl et al. [33] and Arun and Shivakumar [34] provided evidence for, occupational exposure response, which induced Occupational Asthma. Decline in FVC, FEV1, FEV1/FVC ratio, values were significant P value <0.001 in spray painters when compared to non-painters. Such reduction is clinically diagnostic of obstructive pulmonary diseases like asthma and bronchitis. Gupta et al. [9] stated again that painters are at a risk of developing respiratory problems exposed to low molecular weight compounds (isocyanates) in paints at automobile workshops. The decrease of pulmonary function parameters of the workers were found to be significantly compared to that of control group. Duration of exposure had no statistically significant impact on pulmonary function tests.

Table (2): Mean Percentage Predicted of Ventilator Lung Function Indices (FVC, FEV1 and FEV%) between Paint workers and Controls.

Parameters		Groups		
		Control (n=30)	**Car spray painting workshops (n=55)**	**Furniture spray painting workshops (n=61)**
FVC %	**Mean± S.E**	94.7±6.64	83.1± 6.98	84.35±8.98
	p. value	<0.0001	<0.0001	<0.0001
FEV1%	**Mean± S.E**	93.65±7.3	78.98± 7.75	79.1± 9.63
	p. value	<0.0001	<0.0001	<0.0001
FEV1 / FVC%	**Mean± S.E**	98.08± 6.54	94.23± 5.05	96. 96±7.46
	p. value	<0.0001	<0.0001	<0.0001

**Significant at P<0.05. ***=P<0.0001*

Mean percentage predicted of lung function values = 100 x observed/ predicted lung function

FVC% = % of predicted Forced Vital Capacity

FEV $_1$%= % of predicted Forced Expiratory Volume in 1st second.

Table 3 represents the comparison between pulmonary function parameters of paint workers in car and furniture workshops of airborne particles and VOC exposed. It was established that there was no a significant difference in lung function parameters between spray car painters and those that in furniture painters workshops. This is may be due to, almost every shop is unique environment that different in premises and size of shop, work practices, workload, and in chemical structure and type and toxicity of the solvent used, and duration, amount and route of exposure, which is unfortunately not estimated due to technical limitations. These results corroborated with the study of Talini et al. [35] and Leffler and Milton [36] showed that workers in the furniture industry those were at higher risk of asthma than with other jobs. Although prevalence rates of shortness of breath with wheeze and dyspnea were higher in painting workers, no significant difference in pulmonary function FEV and FVC was found among paint groups with different job. The obtained results were nearly similar with results previously recorded by El-Gammal and Niazy [37] estimated an inverse correlation among percentage predicted FEV1 and inhalable spray paint exposure in furniture painting workshops at Damietta city. A decrease in percentage predicted FEV1 with increasing levels of spray paint compared with the non-exposed controls was demonstrated. The volatile organic compounds present in paints can cause a great effect on human health in many ways depending on chemical structure, the solvent, duration and amount of exposure, combination with other chemical exposures, health status and subject sensitivity as age and sex [23, 38].

Table (3): Comparison between the mean value of pulmonary function (FVC, FEV1 and FEV1%) of Spray Car Painters and Spray Furniture Painters

Parameters		**Car spray painting workshops**	**Furniture spray painting workshops**	**p. value**
		(n=55)	**(n=61)**	
FVC %	**Mean± S.E**	83.1± 6.98	84.35±8.98	NS
FEV1 %	**Mean± S.E**	78.98± 7.75	79.1± 9.63	NS
FEV1 / FVC%	**Mean± S.E**	94.23± 5.05	96. 96±7.46	NS

**S=Significant at P<0.05. NS=Not significant at P>0.05.*

Results from previous studies indicated that spray painting exposure, produce an increase of suffering from asthma symptoms and restrictive pulmonary function. They also confirmed that the implementation of appropriate measures and regular medical examinations should be applied to prevent adverse respiratory effects of paints exposure [15, 39, 40, 41]. Most paint workers had restrictive pulmonary function impairment, which can be checked by using high-quality protective equipment as also by reduction of VOC concentration in work environment and workers 'health education [28].

In contrast, Ernstgard et al. [42] demonstrated there were no effects of solvent exposure on the pulmonary functions. Revathi and Chandrasekhar [43] indicated that again FEV1/FVC % of painters had no significantly changed in pulmonary function parameters compared with that of controls this results may be due to the minimal duration of exposure to paints beside the usage of protective respirator.

Consequently, the present study has located the sources of spray paints in the car and furniture painting workshops. The obtained results observed prevalence rates of respiratory illness for workers exposed to paint fume inhalation in painting workshops.

3.2. Evaluation Effect of Spray Painting on Trachea and Lung of Exposed Rats at Studied Painting Workshops.

3.2.1. Histological Changes in Trachea of Exposed Rats.

The prospective study, found an inverse effects in trachea of rats exposed to spray painting in car workshops and in furniture workshops as well as compared with control rats.

A-Group I (unexposed rats): The tracheal tissues of control rats for two weeks, one month and four months revealed its epithelium formed of pseudostratified ciliated columnar epithelial cells in the mucosal layer. The submucosal layer contains few lymphatic cells with intact blood vessels. The layer of hyaline cartilage formed of chondrocytic cells surrounded with extracellular matrix (Fig. 1, 2 and 3). A photomicrograph of trans-section in the trachea of control rat after two weeks, showing normal pseudostratified ciliated columnar epithelial cells lining tracheal mucosa (arrow), normal submucosa containing few lymphatic cells (crossed arrow) and hyaline cartilage formed of chondrocytes (arrow head), (HE, x400), (Fig. 1).

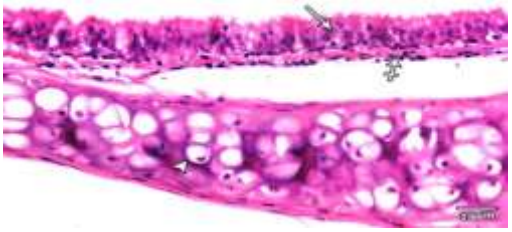

Fig. (1): A photomicrograph of trans-section in the trachea of control rat after two weeks, showing normal pseudostratified ciliated columnar epithelial cells lining tracheal mucosa (arrow), normal submucosa containing few lymphatic cells (crossed arrow) and hyaline cartilage formed of chondrocytes (arrow head). (HE, x 400).

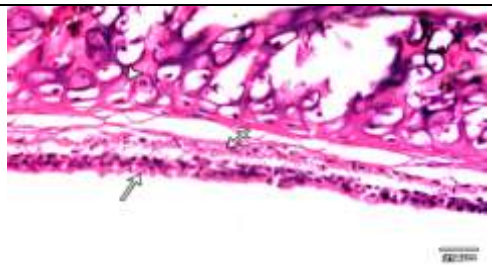

Fig. (2): A photomicrograph of trans-section in the trachea of control rat after one month, showing normal pseudostratified ciliated columnar epithelial cells lining tracheal mucosa (arrow), normal submucosa containing few lymphatic cells (crossed arrow) and hyaline cartilage formed of chondrocytes (arrow head). (HE, x 400).

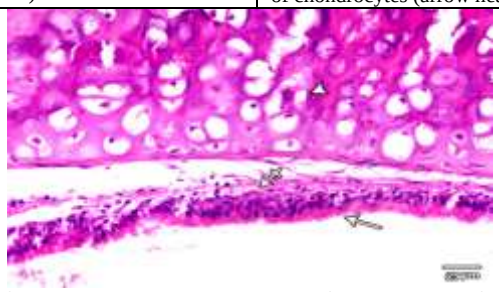

Fig. (3): A photomicrograph of trans-section in the trachea of control rat after two months, showing normal pseudostratified ciliated columnar epithelial cells lining tracheal mucosa (arrow), normal submucosa containing few lymphatic cells (crossed arrow) and hyaline cartilage formed of chondrocytes (arrow head) (HE, x 400).

B-Group II (Exposed rats): These rats that exposed to spray painting vapors inhalation in car painting workshops and in furniture workshops were divided into two sub groups (i) and (ii), respectively:

(i) Car workshops group:

Exposure to car painting inhalation for two weeks: The tracheal tissue of rats at this exposure interval revealed several histopathological changes, disappearance of cilia of tracheal epithelia at certain areas, increased lymphocytes in submucosa; degeneration in the pseudostratified columnar epithelial layer (Fig. 4).

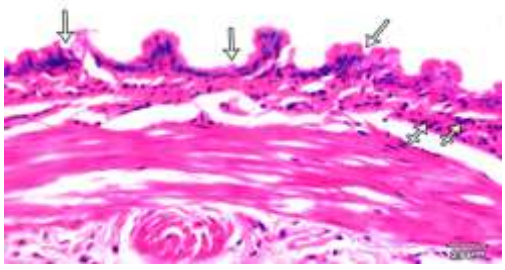

Fig. (4): A photomicrograph of trans-section in the trachea of rat, two weeks after exposure to car painting, showing areas for loss of the cilia in the lining epithelium (arrows). Notice increased lymphocytes in the submucosa (crossed arrows) (HE, x400).

At one month post-exposure: The tracheal tissue exhibited massive loss of cilia, lymphocytic infiltrations, thickening in the submucosa and degeneration in the pseudostratified columnar epithelial layer at certain area (Fig. 5).

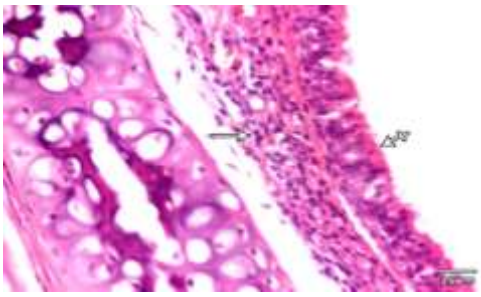

Fig. (5): A photomicrograph of trans-section in the trachea of rat, one month after exposure to car painting, showing lymphohistiocytic infiltrates and thickening in submucosa (arrow). Notice massive loss of cilia and degeneration in the pseudostratified columnar epithelial layer (crossed arrow) (HE, x 400).

At two months post-exposure: The tracheal tissue investigated at this interval exhibited a cushion of squamous metaplasia in the lining epithelium with fibroblastic proliferation in submucosa, complete loss for the submucosa together with necrosis and desquamation in the pseudostratified columnar epithelial layer, lymphocytes and lymphocytic infiltrations in submucosa (Fig. 6), degeneration in the pseudostratified columnar layer and intense lymphocytic (Fig. 7).

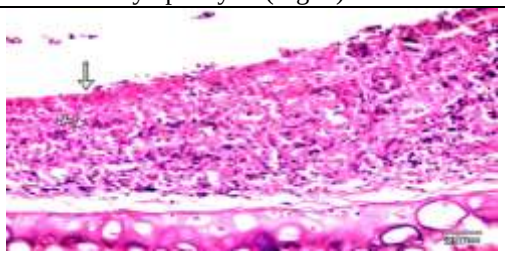

Fig. (6): A photomicrograph of trans-section in the trachea of rat, two months after exposure to car painting, necrosis and epithelial desquamation in the pseudostratified columnar layer (arrow), lymphocytes and lymphocytic infiltrations in submucosa (crossed arrow) (HE, x 400).

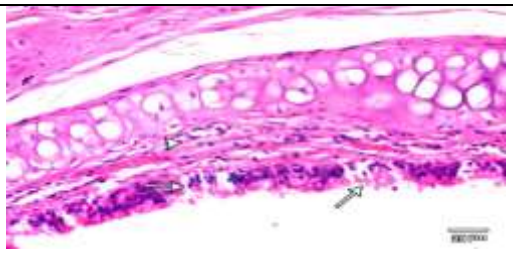

Fig. (7): A photomicrograph of trans-section in the trachea of rat, two months after exposure to car painting, showing areas of degeneration in the pseudostratified columnar epithelial layer (arrows) and intense lymphocytic infiltrations in submucosa (arrow head) (HE, x 400).

(ii) Furniture workshops group:

Exposure to furniture painting inhalation for two weeks: The light microscopic examination of the tracheal tissue at this interval post-exposure revealed degenerations in the pseudostratified columnar epithelial layer at certain areas, fibroelastic proliferation in submucosa and marked lymphocytic infiltrations in submucosa (Fig. 8).

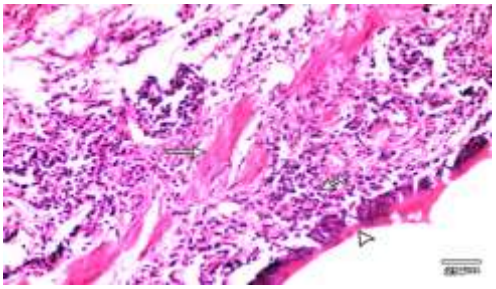

Fig. (8): A photomicrograph of trans-section in the trachea of rat, two weeks after exposure to furniture painting, showing areas of degenerations in the pseudostratified columnar epithelial layer (arrow head), proliferation in submucosa (arrow) and marked lymphocytic infiltrations in submucosa (crossed arrow) (HE, x 400).

At one month post-exposure: The tracheal tissue revealed loss of ciliated columnar epithelium, intense lymphocytic infiltrations in submucosa and mucosa, areas of degeneration in the pseudostratified columnar epithelial layer and congestion in submucosa (Fig. 9).

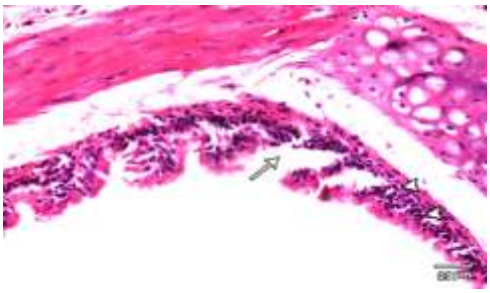

Fig. (9): A photomicrograph of trans-section in the trachea of rat, one month after exposure to furniture painting, showing areas of degeneration in the pseudostratified columnar epithelial layer with loss of cilia (arrow) and many lymphocytic infiltrations (arrow heads) (HE, x400).

At two months post-exposure: The tracheal tissue exhibited ulceration of lining epithelium with fibroelastic proliferation in submucosa, congestion in submucosa, intense lymphocytic infiltrations (Fig. 10), necrosis and desquamation in the pseudostratified columnar epithelial layer (Fig. 11).

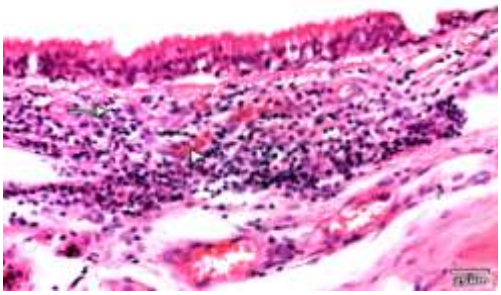

Fig. (10): A photomicrograph of trans-section in the trachea of rat, two months after exposure to furniture painting, congestion in submucosa (arrow head) and intense lymphocytic infiltrations (arrow). (HE, x 400).

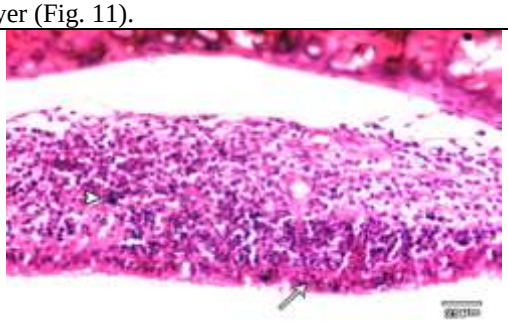

Fig. (11): A photomicrograph of trans-section in the trachea of rat, two months after exposure to furniture painting, showing necrosis and desquamation in the pseudostratified columnar epithelial layer (arrow) with lymphocytic infiltrations in submucosa (arrow head). (HE, x400).

3.2.1.1 Demonstration Effect of Spray Painting on Trachea of Exposed Rats at Studied Painting Workshops.

It was clear, that the tracheal slides of rats exposed to spray painting inhalation in car and furniture painting workshops gradual degenerative changes in the tracheal epithelium start from duration 14 days and increase of gradually till duration of two months exposure compared to in the control group. The changes showed disappearance of cilia of tracheal epithelia, increased lymphocytes in submucosa and degeneration in the pseudostratified columnar epithelial layer. It is noticed that distortion in the lining epithelium of respiratory bronchioles, loss cilia and increased lymphocytic infiltrations which markedly increased with exposed duration. Moreover, the tracheal tissue exhibited lymphocytic infiltrations inside the submucosal layer this obtained results agreement with Mohamed et al. [18]; Rusch et al. [44] and Bansal et al. [45] found that there were respiratory changes after exposure to formaldehyde vapour in paints, like cellular infiltrations, emphysema, hyperplasia of cells in the bronchiole and hemorrhages in the respiratory organs

Similar observation was investigators by Mehdi et al. [46] demonstrated there was squamous metaplasia in the lining epithelium with fibroblastic proliferation in submucosa, complete loss for the submucosa together with necrosis and desquamation in the pseudostratified columnar epithelial layer, lymphocytes and lymphocytic infiltrations in submucosa and degeneration in the pseudostratified columnar layer after two months exposure. Mehdi et al. [47] observed again that spray painting induced a lot of changes in respiratory system, particularly in the trachea and lung in mice exposed to xylene through painting, compared to in the control group, which were ranged from sloughing epithelium, induction of inflammatory responses to metaplastic change in trachea of exposed mice. They found focal mild infiltration of mononuclear cells and edema in the lamina propria.

3.2.2. Histological Changes in Lung of Exposed Rats.

The prospective, show an inverse changes between lungs of exposed rats to paints vapour inhalation in car and furniture painting workshops as well as compared with control rats.

A-Group (unexposed rats): the lung tissue for two weeks show normal histological, one month and four months in control rats appeared formed of pulmonary bronchioles, peribronchial blood vessels, alveolar ducts, alveolar sacs and alveoli with thin interalveolar septa (Fig. 12, 13 and 14).

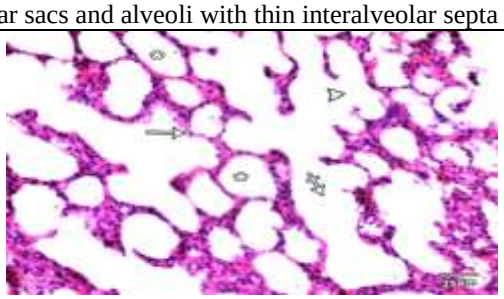

Fig. (12): A photomicrograph of lung of control rat after two weeks, showing normal alveoli (stars) (arrow), as well as alveolar ducts (arrow head) and alveolar sacs (crossed arrow) (HE, x 400).

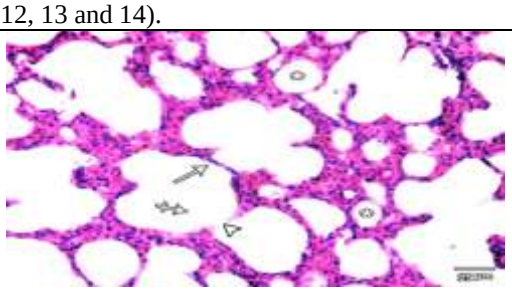

Fig. (13): A photomicrograph of lung of control rat after one month, showing normal alveoli (stars), **thin walls** (arrow), alveolar ducts (arrow head) and alveolar sacs (crossed arrow) (HE, x 400).

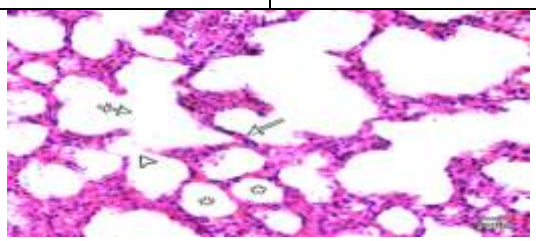

Fig. (14): A photomicrograph of lung of control rat after two months, showing normal alveoli (stars) with thin walls (arrow), as well as alveolar ducts (arrow head) and alveolar sacs (crossed arrow) (HE, x 400).

B-Group II (Exposed rats): These rats that exposed to painting vapors inhalation are divided into two sub groups:

(i) Car workshops group:

Exposure to car painting inhalation for two weeks: The light microscopic examination of lung tissue revealed langhanz giant cells surrounded with fibrous tissue, bronchiole filled with exudate as well as many lymphocytic infiltrations to lung tissue (Fig. 15), degeneration of alveoli, some exudates in alveoli and aggregations of many lymphocytes inside the lung (Fig. 16).

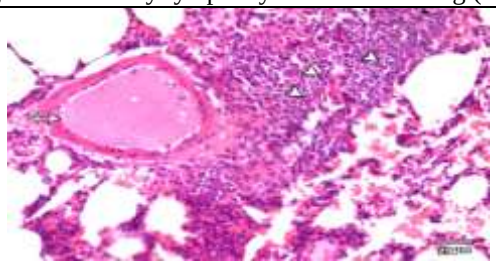

Fig. (15): A photomicrograph in lung of rat after two weeks exposure for car painting, showing bronchiole filled with exudate (arrow) as well as many lymphocytic infiltrations to lung tissue(arrow heads) (HE, x400).

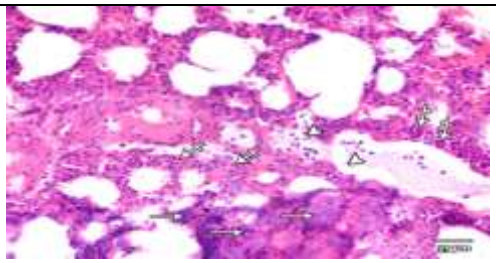

Fig. (16): A photomicrograph of lung of rat after two weeks from exposure to car painting, degeneration of alveoli (arrows), some exudates in alveoli (arrow head) and aggregations of many lymphocytes inside the lung (crossed arrows) (HE, x400).

At one month post-exposure: Lung sections showed alveoli filled with exudates and many lymphocytes (Fig. 17), consolidated lung alveoli, many lymphocytic infiltrates and thick interalveolar septa (Fig. 18).

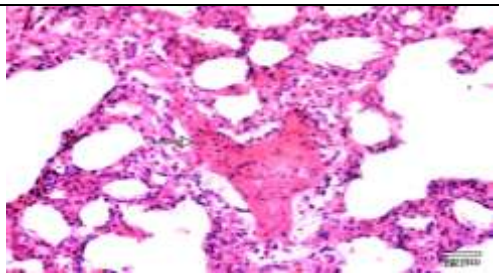

Fig. (17): A photomicrograph in lung of rat after one month from exposure for car painting, showing alveoli filled with exudates and many lymphocytes (arrow) (HE, x400).

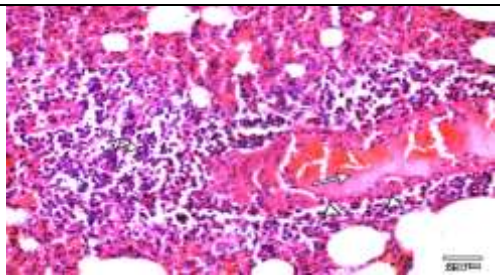

Fig. (18): A photomicrograph in the lung of rat after one month for exposure to car painting, showing fluid exudates in pulmonary alveoli (arrow), many lymphocytic infiltrates (crossed arrow) and thick interalveolar septa (head arrows) (HE, x400).

At two months post-exposure: The onset of histopathology of the lung tissue appeared much more complicated at this interval post-exposure period where; marvelous increased lymphocyte to lung tissue, serous exudates, sever lymphocytic infiltrations, increased interalveolar septa (Fig. 19), consolidated of alveoli by homogenous exudates (Fig. 20) and fibrocollagenous tissues in site of lung alveoli (Fig.21).

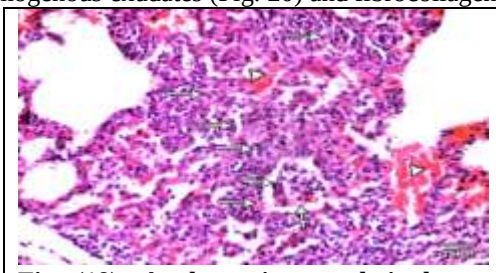

Fig. (19): A photomicrograph in lung of rat two months after exposure to car painting, showing sever lymphocytic infiltrations (arrows) with presence of exudates (arrow heads) and increased interalveolar septa (crossed arrows). (HE, x 400).

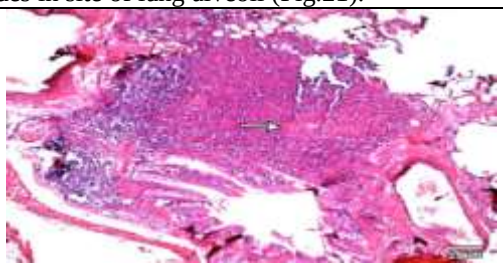

Fig. (20): A photomicrograph in the lung of rat two months after exposure to car painting, showing consolidated of alveoli by homogenous exudates (arrow). (HE, x 400).

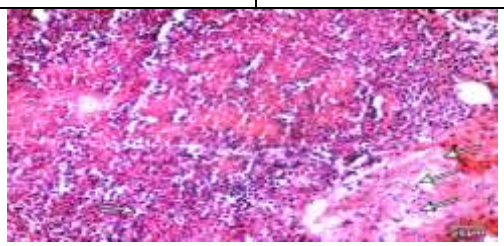

Fig. (21): A photomicrograph in the lung of rat two months after exposure to car painting, showing fibrocollagenous tissues in site of lung alveoli (arrows) (HE, x 400).

(ii) Furniture workshops group:

Exposure to furniture painting inhalation for two weeks: Lung tissue showed exudates in the alveoli, increased thickness of alveolar septum, and aggregation of lymphocytes in the pulmonary tissue and serofibrous exudates (Fig. 22).

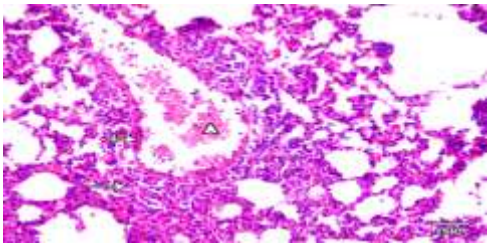

Fig. (22): A photomicrograph in the lung of rat, two weeks after exposure to furniture painting, showing exudates in the alveoli (arrow head) and increased thickness of alveolar septum (crossed arrow). Notice aggregation of lymphocytes in the pulmonary tissue (arrow) (HE, x400).

At one month post-exposure: The lung tissue of rats one month post-exposure to furniture spray painting showed pulmonary alveoli filled with exudates and many lymphocytes (Fig. 23), consolidated lung alveoli many lymphocytic infiltrates and thick interalveolar septa (Fig. 24).

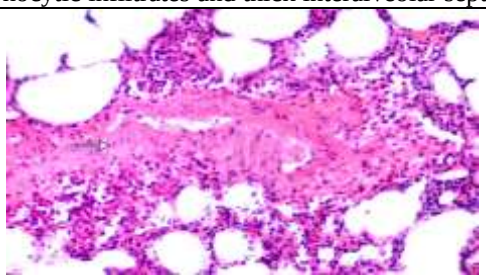

Fig. (23): A photomicrograph in the lung of rat, one month after exposure to furniture painting, showing alveoli filled with exudates and many lymphocytes (arrow) (HE, x400).

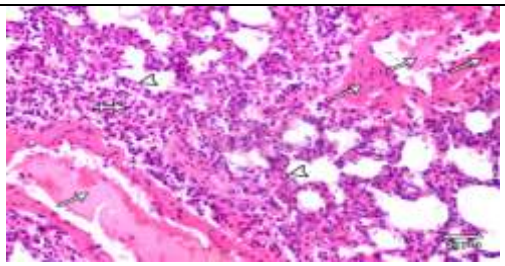

Fig. (24): A photomicrograph in the lung of rat, one month after exposure to furniture painting, showing fluid exudates in pulmonary alveoli (arrows), many lymphocytic infiltrates (crossed arrow) and thick interalveolar septa (head arrows) (HE, x 400).

At two months post-exposure: The lung environment at this interval post-exposure appeared consolidated lung alveoli with many lymphocytic cells, exudates in alveoli (Fig. 25), aggregation of macrophages, lymphocytes, plasma cells encircled by fibrous tissue replacing pulmonary tissue and sever lymphocytic infiltrations (Fig. 26).

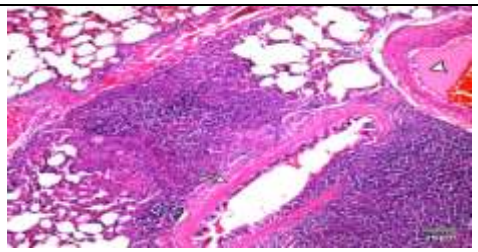

Fig. (25): A photomicrograph in the lung of rat, two months after exposure to furniture painting, showing consolidated lung alveoli with many lymphocytic cells (arrow) and exudates in alveoli (arrow head). (HE, x400)

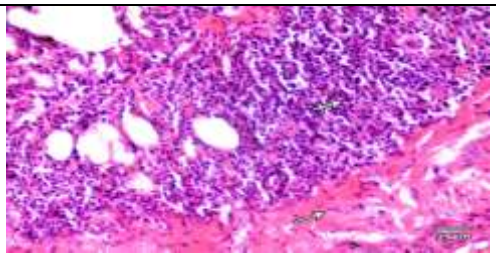

Fig. (26): A photomicrograph in the lung of rat, two months after exposure to furniture painting, showing alveoli filled with exudates and many lymphocytes (arrow) and sever lymphocytic infiltrations (crossed arrow).(HE, x400).

3.2.2.1. Demonstration Effect of Spray Painting on Lung of Exposed Rats at Studied Painting Workshops.

It is also clear evident, that the respiratory effects of exposure to painting vapors was gradual degenerative changes in the histological alterations in the lungs start from duration 12 weeks and increase pronounced in the exposed rats to inhalation paints fume more than two months as revealed from the histological slides compared with control rats. This may be due to vapour concentrations beside exposure duration that induce respiratory impairment. The changes lung tissue appeared much more complicated at this interval post-exposure period where; marvelous increased lymphocyte, sever lymphocytic infiltrations, increased interalveolar septa, and consolidated of alveoli by homogenous exudates and fibrocollagenous tissues in site of lung alveoli. These histologic lesions are similar to those described in other paint fume studies by Hanachi [48]; Mendell [49]; Njoya et al. [50] and Mohamed et al. [18] observed an alteration in the cytoarchitecture of the epithelium of the trachea and lung of rats exposure to solvent vapour in spray paints. The thickened alveolar septum, bronchiolar epithelial hyperplasia, lymphocytic aggregations, pulmonary fibrosis were found. Precancerous changes were also shown. There were increased aggregation of macrophages and interstitial pneumonia with fibrous tissue proliferation in peribronchial tissue. Balogun et al. [51] revealed that the alveolar cells of exposed rats to paints for eight hours per day for 21 days, 30 days and 45 days undergo fibrosis which progresses as the week of exposure increased comparing with the control group.

These results were in agreement with Akpan et al. [52] and Mehdi et al. [46] recorded also considerable inflammation of the lungs, mild edema and hemorrhage in a number of bronchial tissues in mice exposed to mixture of organic solvents when compared to mice in the control group, and resulted in various degrees of histological alterations in the lungs such as inflammation of alveoli, inflammatory cells in the interlobular septa, destruction of alveolar septa, fibrosis in the interstitial tissue, irregular dilation of the bronchioles and a large number of desquamated epithelium contains the bronchiolar lumen. Furthermore, Mehdi et al. [47] found again bronchiolitis as indicated by the presence of necrotic debris with hemorrhage in the lumen of bronchioles,

infiltrations cells in the peribronchiolar space with no signs of leukocytic alveolitis in mice exposed to xylene through painting.

Therefore, the present study has demonstrated also the exposed experimental animals (rats) to inhalation of paint fumes in the car and furniture painting workshops, for long time can cause respiratory distress and induced histopathological changes in their trachea and lungs when compared with controls. Histologic evaluation of paint fumes exposed rats over 2 weeks, 1 and 2 months, demonstrated a temporal progression of pulmonary lesions, loss of lungs function and serious pulmonary problems. The histopathological changes were increase proportionally to the exposed duration.

IV. Conclusion

Painting process has a great contribution to Mansoura and Damietta cities income due to different activities of workshops as car and furniture painting workshops, besides the poor urban planning, the establishment of workshops in urban areas has led to generation more hazardous pollutants.

Exposure to organic solvents and dust used for spraying might be of high risk of respiratory impairment and asthma; it can irritate the upper respiratory tract and lung. Since, this indicates high occupational health hazards and an alarming situation remains unchecked. However, the lack of safety regulations in small scale business should be taking more seriously.

In the present study, all painters had significantly lower % predicted lung function (FVC, FEV1, and FEV1/FVC %) compared with the non-exposed controls that the reduction in FVC may serve as a guide to identifying painting workers are at risk in lung function. The present findings revealed that restrictive pulmonary function in paint workers. These changes in pulmonary function may be attributed to spray painters using organic solvents-containing aerosol paints, the ignorance of workers to use of personal protective measures and awareness of potential health hazardous of spray paints exposure with lack of workplace dust control and ventilation systems.

The present study has demonstrated also histopathological changes in trachea and lungs of exposed rats to inhalation of paint fumes in the studied car and furniture painting workshops, in comparing with controls. Histologic evaluation of paint fumes suggest that long term exposure to paint fumes can cause loss of lungs function and serious pulmonary problems.

V. Recommendation

To avoid the risk effects of organic solvent and dust exposure on pulmonary function of painters and to prevent the translocation of paint fume outside the workshops, a management strategy should be implemented. Painting exposure can be improved by increasing the ventilation rate to avoid respiratory impairment. The control measures should be applied; education of paint workers, use of personal protective measures and regular examinations. Implementation should be introduced to prevent adverse respiratory effects and to ensure safe and healthy working condition of paint industry workers.

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