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Implications of Tobacco Smoke on the Lung Histopathology of Albino Rats in Relation to Sex

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Abstract: ***The purpose of this study was to explore the histopathological changes in lungs of both sexes of albino rats. Experimental rats (150-200 gram) were kept in standard laboratory conditions and grouped into 6 sets (5 rats each)-Control rats (I_M and I_F) without exposure, For 60 days, six filtered cigarettes were exposed to the fumes of cigarettes hourly on the experimental rats (IIM and IIF). For a period of 120 days, six filtered cigarettes were exposed to the smoke of experimental rats (IIIM and IIIF). When comparing the lung tissue of rats exposed to tobacco smoke to that of control rats, several histopathological alterations were noted, including pulmonary edema, debris, thick epithelium, and capillary permeability. Given that female rats are more sensitive than male rats, these changes are more noticeable in female rats after 120 days of tobacco smoke inhalation.***

Keywords: Tobacco smoke, Albino rats, Lung Histopathology

1. Introduction

Among the most important public health issues the world has ever confronted is tobacco recrudescence. Statistics from the World Health Organization indicate that tobacco use causes over 8 million deaths annually globally. According to this data, smoking directly causes almost 7 million deaths annually, while second-hand smoke exposure causes 1.2 million deaths among nonsmokers [1].Presently, over 37 million adults in the US smoke, and over 16 million have smoking-related illnesses annually. Additionally, smoking-related diseases claim the lives of over 480 000 people [2].With over 8 million fatalities from tobacco use each year, including over 1.2 million from secondhand smoke exposure, the tobacco epidemic is one of the worst risks to public health the world has ever faced [3].There is no acceptable level of tobacco exposure, and all tobacco products are dangerous. In addition to water pipe tobacco, other smokeless tobacco products, cigars, cigarillos, roll-your-own tobacco, pipe tobacco, bidis, and kreteks, cigarette smoking is the most popular type of tobacco use in the globe.

The most frequently recognized cause of chronic obstructive pulmonary disease (COPD) is tobacco use.It has a significant global impact on both medicine and the economy in the Western world. By 2030, COPD is expected to rank third globally in terms of disease-related mortality, according to predictions made by the World Health Organization (WHO). Cadmium is one such interesting molecule, which is a dangerous heavy metal that was linked to lung damage as early as 1950.Thousands of chemicals that are oxidative, inflammatory, and carcinogenic can be found in cigarette smoke [4].

A complex cocktail of chemicals, including several genotoxic lung carcinogens, are present in cigarettes.250 million women and 1 billion men are believed to smoke worldwide. In certain regions of Eastern Europe, the prevalence of male smoking is notably high, whilst the incidence of female smoking is highest in certain regions.

The lungs are a crucial component of breathing because they supply humans with oxygen, which enters the bloodstream and is quickly delivered to the tissues and organs that need it to function. Therefore, study aims to investigate the tobacco smoke-induced alterations in pulmonary tissue in both sexes of albino rats.

2. Materials and Methods

Throughout the trial, adult male Wistar albino rats weighing 150–200 g.b.wt were raised in polypropylene cages with conventional laboratory settings: 25±0.5ºC, 60±5% relative humidity, and 12 hours of photoperiod per day. Commercial food pellets (Golden Feed, New Delhi) and unlimited water was provided to the rats. Prior to the trial, the experimental albino rats underwent a month of acclimation. Animal experiments were designed and conducted following guidelines of Institutional Animal Ethical Committee.

Selection of Cigarette
For the exposure to tobacco smoke Capstan filtered cigarette, ITC Ltd., Kolkata was selected.6 cigarette/hr/day for 6 day are used for the exposure. The length of cigarette is 69mm.

Experimental Protocol
The rats were grouped into 6 sets. Each set contain 5 rats.

Control set IM and IF: Rats were kept in separate room without tobacco smoke exposure.

Experiment set II M and IIF: Rats were exposed to tobacco smoke for 60 days at a time, one hour every day. In one hour, six cigarettes with filters were consumed.

Experiment set III M and IIIF: Rats were exposed to tobacco smoke for 1hr/day for 120 days.

Exposure to Tobacco Smoke
'Mini exposure cabinet' (60×30×30 cm) manufactured by Precision Instruments, Varanasi was used for the exposure of tobacco smoke. For 120 days, the rats were exposed to six

filtered cigarettes of tobacco smoke every hour throughout the day.

3. Result and Discussion

Emphysema, pulmonary edema, debris, thick epithelium, and capillary permeability are among the histopathological alterations in the lung tissue of tobacco smoke-exposed rats compared to control rats after 60 days of exposure, but these alterations become more noticeable after 120 days.(**PLATE: I_M and I_F, PLATE: II_M and II_F,PLATE : III_M and III_F**).

Lungs are the target organ for oxidative stress. Tobacco smoke triggers the release of oxidants which is a leading cause of oxidative stress resulting in pulmonary injury in rats. More quickly, smoke particles are ingested into the lungs. This forceful intake of smoke aerosols widens the alveoli and speeds up the process of nicotine saturation. Capillary permeability, cell debris and Thick epithelium have seen in most of the places in both the sexes of albino rats. These changes are more pronounced in female albino rats. In addition, both sexes of albino rats exposed to cigarette smoke had lung edema, increased capillary permeability, and thicker epithelium[5] Emphysema in female rats brought on by cigarette smoke[6].Long-term exposure to cigarette smoke cause emphysema in mice [7] .

Emphysema and inflammatory illnesses are thought to be largely caused by the excessive amounts of free radicals that are released by alveolar macrophages in response to cigarette smoke [8].Enzymes leak out when cells die or become damaged from inflammation, which raises the amounts of these enzymes in the blood. It is commonly employed as a sign of tissue oxidative stress brought on by free radical damage to the cell membrane, which increases membrane permeability and causes cellular damage [9]. Additionally, cigarette smoke causes oxidative stress by increasing NADPH oxidase and lowering antioxidant levels [10]. One of the main factors influencing the respiratory system is tobacco smoke intake.[11]. Nicotine smoke (CS) aqueous extract contains reactive oxygen species (ROS, oxidants), which are the source of oxidative damage [1]. Inflammation and oxidative lung damage brought on by tobacco smoke may hasten anatomical and functional alterations while progressively reducing gaseous exchange [12] .Cigarette smoke weakens the antioxidant defenses in relation to the increased quantities of reactive oxygen species (ROS) [13]. The length of exposure to tobacco smoke and the quantity of cigarettes smoked are the main contributors to lung histological alterations [14]. Additionally lacking the defense found in the major bronchi are the alveolar area and the lung's secondary and tertiary branches. The muscles of the bronchial wall gradually deteriorate as a result of inflammation brought on by inhaling tobacco smoke. Cigarette smoke contains a variety of toxicants that are known to cause oxidative stress [15] and airway inflammation, which worsen the course of the disease. Oxidative stress and inflammatory mediators are typically released in large quantities by alveolar macrophages and broncho-epithelial cells exposed to cigarette smoke [16]. Tobacco smoke triggers the release of oxidants which is a leading cause of oxidative stress resulting in pulmonary injury in rats. More quickly, smoke particles are ingested into the lungs. The smoke aerosols' forced inhalation widens the alveoli and speeds up the process of nicotine saturation.

A rapid onset of emphysema and pulmonary edema after tobacco smoke inhalation alternates the membrane permeability of epithelial cells of alveoli. Destruction of the wall of alveoli which leads to massive rupture of capillary membrane and cause capillary permeability in alveoli, which show infiltration condition of cell in alveoli in both the sexes of albino rats. Thick alveolar septa in rats after cigarette smoke [17]. Extremely high levels of free radicals are known to be released by female mice's alveolar macrophages in response to cigarette smoke [8]. Long-term exposure to cigarette smoke has also been shown to cause emphysema in mice [7].

In the present study, cigarette smoke is correlated with the lung tissue injury in rats. The oxidant-antioxidant balance is impacted by cigarette smoking's increased oxidative stress and localized inflammation in the lungs [18]. Smoking frequency has a significant impact on the degree of oxidative damage and the antioxidant defense system, both of which lead to elevated oxidative stress [19]. Passive smoking damages the lungs by introducing toxic compounds and oxidants into the lungs and obstructing the lungs' natural repair process [20]. Injury to the lung parenchyma causes damage to the alveoli and emphysema [21] and [22]. Additionally, exposure to cigarette smoke seriously harmed the respiratory system of rats [23]. The results align with the research conducted [24], which indicated that using cigarettes and e-cigarettes can have adverse effects on lung biology even after a few days of exposure.

4. Conclusion

Due to the fact that females are more sensitive than males and that the toxicity of tobacco smoke increases with exposure duration in albino rats of both sexes, the current study demonstrates that histological changes in female rats are more noticeable 120 days after tobacco smoke exposure as opposed to 60 days.

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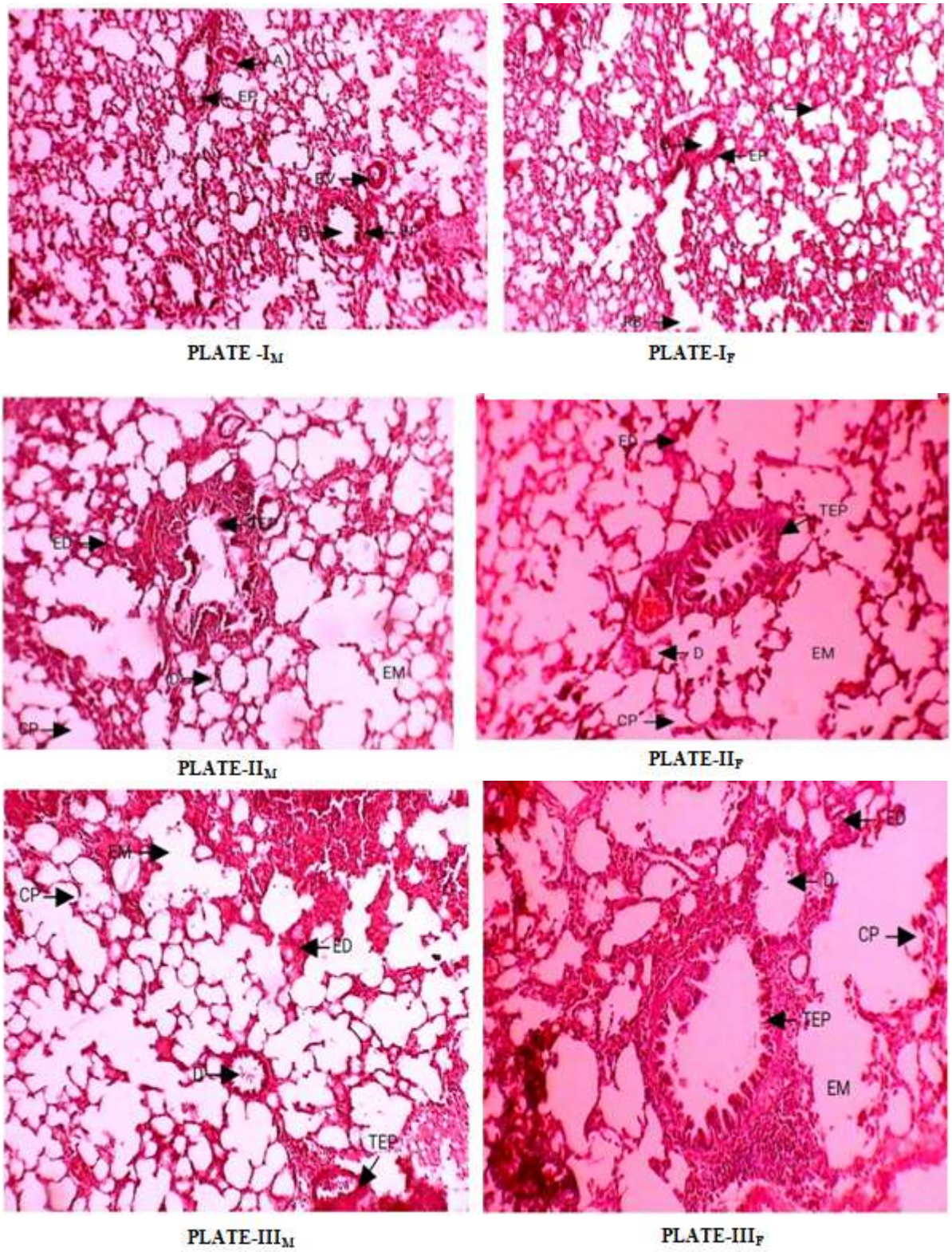

Plate- I_M and I_F showing section of lung of control rats, Plate-II_M and II_F Experimental rats after 60 days cigarette smoke exposure and Plate-III_M and III_F Experimental rats after 120 days cigarette Smoke (X 400(EP-Epithelium, A-Alveoli, B-Bronchiole, BV-Blood vessel, RB-Respiratory bronchiole, C-Capillary, TEP-Thick Epithelium, D- Debris, ED-Edema, EM-Emphysema, CP-Capillary permeability)