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

Nibha Rathore¹, B. S. Sharma²

¹,²Department of Environmental Studies, School of Life Sciences, Dr. B. R. Ambedkar University, Khandari Campus, Agra-282002

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 (I₀ and I₉) 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 kretaks, 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.
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: IM and IF, PLATE: II M and II P, PLATE : III M and III P).

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. 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.

Acknowledgement

I express my gratitude to the Department of Environmental Studies in the School of Life Sciences at Dr. Bhimrao Ambedkar University, located in Agra.

References


Plate-Iₘ and Iₖ showing section of lung of control rats, Plate-IIₘ and IIₖ Experimental rats after 60 days cigarette smoke exposure and Plate-IIIₘ and IIIₖ 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)