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**Exploring the effects of low-dose** PM2.5 **exposure on pulmonary health**

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**Keywords:** Air pollution. PM2.5. Immunohistochemistry. COPD. Novel markers.

**Career Stage:** MPhil Graduate

**Introduction**: Chronic obstructive pulmonary disease (COPD) is a global health concern associated with significant morbidity and mortality, and air pollution, particularly fine particulate

matter with a diameter less than 2.5 μm (PM2.5), is a critical environmental risk. Annually, over 7 million individuals die prematurely due to air pollutants, with PM2.5 implicated in 62% of air pollution-related deaths. Despite extensive research on PM2.5 and COPD, knowledge gaps persist, especially regarding the impact of low-dose PM2.5 exposure on COPD development in non-smokers and the underlying mechanisms.

**Objective**: This study aims to investigate the morphological and molecular effects of low-dose PM2.5 exposure in a mouse model, focusing on epithelial thickness in small airways and alveolar destruction.

**Methods and Results:** In a previous study, mice were exposed to 10 μg/m³ of PM2.5 daily for 12 weeks, and lung samples were collected at 4, 8, and 12-week intervals. In this study, histological analysis was performed using Nanozoomer and image J software, with a focus on epithelial thickness in small airways and alveoli. Novel markers associated with liver fibrosis were explored, and their presence in human and mice lungs was investigated using bioinformatics. Immunohistochemistry targeted Sun2 and Ephb2 as potential fibrosis markers in the mice model. QuPath software facilitated data analysis.

Cross-sectional imaging revealed significant increases in epithelial thickness and the ratio of epithelial area to squared diameter of small airways, supporting the hypothesis of PM2.5-induced morphological changes in COPD. Increased mean linear intercept (MLI) indicated alveolar wall damage, particularly at the 12-week timepoint. Immunohistochemistry results showed increased Sun2 and decreased Ephb2 expression after 12 weeks of PM2.5 exposure, suggesting a potential role in fibrosis development. Changes were more pronounced at 12 weeks. A p-value with P ≤ 0.05 indicated statistical significance.

**Conclusion**: In conclusion, this study sheds light on morphological and molecular changes induced by low-dose PM2.5 exposure in a mouse model. Alterations in epithelial thickness, alveolar destruction, and novel fibrosis markers suggest a link between low-dose PM2.5 exposure and COPD development, contributing to our understanding of air pollution-induced respiratory diseases and informing future therapeutic strategies.