中文題目：環境汙染物誘導慢性呼吸道疾病之致病機制探討

英文題目：The Pathogenesis of Environmental Pollutants-Induced Chronic Airway Disease

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Introduction/Purpose: Chronic obstructive pulmonary disease (COPD), the fourth leading causes of death in the world, is a chronic inflammatory disease of the airway characterized by persistent airflow limitation progressively. COPD becomes a major public health problem worldwide. Long-term exposure to noxious particles or gases in the environment is considered as a major cause of COPD. Tobacco smoking, particulate matter 2.5 (PM2.5), polycyclic aromatic hydrocarbons (PAHs) are environmental hazards to induce inflammatory process leading to COPD or acute exacerbation of COPD (COPDAE). PAHs are produced due to incomplete combustion or pyrolysis processes and are major components of PM2.5. Increase concentrations of PM2.5 in air will elevate the rate of COPDAE. Seventy to eighty percentage of COPDAE is induced by respiratory infections. The aim of this study was to investigate the effect of PAHs on human bronchial epithelial cells and to explore its role in the pathogenesis of COPD.

Materials and Results: The air samples were collected in the urban area of Kaohsiung city in Taiwan, and analyzed the levels of PAH and endotoxin. To find the mechanisms of air pollution-induced lung injury, we choose benzo[a]pyrene (BaP), a dominant PAH in the air sample from Kaohsiung city, to treat human bronchial epithelial cells and evaluate its efficacy. We found that BaP alone did not stimulate ROS production, but pretreatment of BaP could augment LPS induced-ROS production. Similarly, BaP alone did not stimulate the phosphorylation of NFκB p65, whereas pretreatment of BaP could enhance the LPS-induced phosphorylation of NFκB p65 and expression of downstream inflammatory cytokines such as interleukin-1β (IL-1β) and interleukin-8 (IL-8) in human bronchial epithelial cells. Pretreatment of BaP also could augment LPS-reduced α1-antitrypsin activity. These results suggest the risk of COPD is increased by BaP. In addition, we found that increased oxidative stress in human bronchial epithelial cells could cause reduction in α1-antitrypsin activity.

Conclusion: BaP can enhance the LPS-induced ROS production and inflammation cytokines expression of human bronchial epithelial cells. That suggests long-term exposure of PAHs may cause chronic airway disease. A combined effect of BaP and endotoxin exits and that is worthy to be noticed and be further studied for exploring the pathogenesis of COPD.