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IL17 Pathway involves moderating pulmonary hypertension, a common complication of COPD, in statins therapy in smoking rats

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Smoking and atmospheric pollution are major causes of COPD affecting public health. Lack of effective treatments leads to COPD and later on causes Pulmonary Hypertension. Statins, lipid lower drugs, are reported to be beneficial to COPD patients. However, the mechanism remains unknown. Here, we establish a smoking model with rats. Increase of mean linear intercept and decrease of mean alveolar number are seen in smoking rats ($P<0.01$). Neutrophils, lymphocytes, especially macrophage pullulate in bronchoalveolar lavage fluid. Infiltration of immune cells is substantially observed in the lungs of smoking rats. IL-17, RORγt and IL-21 positive cells manifold in bronchial epithelium and alveolar septa and walls of smoking rats in immunohistochemistry staining. The mRNA expression of IL-17, RORγt and IL-21 are increased in smoking group ($P<0.01$). The impair of lung function of smoking rats is demonstrated by significant subtraction of forced expiratory volume in 0.3 second vs. forced vital capacity, decrease of lung dynamic compliance and increase of resistance of inspiratory and expiratory. Pulmonary artery hypertension in smoking rats is determined by dramatically elevating pulmonary artery pressure. Statins therapy reverses increase of neutrophils, especially macrophage, but not lymphocytes. Expression of IL-17, RORγt and IL-21 in lung of smoking rats is significantly reduced by Statins at both protein and mRNA level ($P<0.05$). Surprisingly, although pathological changes and damage of lung function caused by smoking are slightly improved, the mPAP is largely reduced by Statins therapy in smoking rats. This study suggests that IL-17, IL21 and RORγt play an important role in the progression of COPD in smoking rats. Statins can relieve the PAH in smoking rats, probably through reducing the expression of IL17 and its mediators. Statins have a great potential in application for COPD patients complicating with pulmonary hypertension.

Biography

Xin Wang gained her MD at the age of 21 years from Hebei Medicine University in China. She specialized as neurologist to study cerebrovascular diseases. The project, stroke rehabilitation, awarded the First Prize of excellent paper in the Third Annal National Neurologic Rehabilitation meeting. After ten years clinical medicine practicing, including clinical research, she pursued PhD on Medical Science from Institute on Aging and Adaptation, Graduate School of Medicine, Shinshu University in Japan. During PhD course, she discovered an acute leukemia associated brain-specific gene and was the first one who studied its function at molecular & cellular biology level. The first postdoctoral training in NIH/NCI-Frederick in US, gave her an opportunity to complete a publication studying mouse model related to cardiovascular dysfunction that had been pointed as 'must reads' by Faculty1000. In Dental School & Medical School of University of Maryland, Baltimore, Xin studied head & neck cancer and prostate cancer with human specimen, cancer cell lines and animal models. Her summary of a project about tumor microenvironment as primary author gained Minority Scholar Award in AACR meeting. She is the CEO and CSO in biotechnology company, ACURE TECHNOLOGY, Inc. that she is the founder, to develop and discover stem cell therapeutic products based on her extensive medical research experiences. She has published more than 20 peer-reviewed papers in scientific journals.

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