Abstract

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COPD Mice Model Optimization

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Abstract

Chronic obstructive pulmonary disease (COPD) is a common chronic respiratory disease of human beings characterized by not fully reversible airflow limitation, Animal model of cigarette smoke induced chronic obstructive pulmonary disease (COPD) is the primary testing methodology for drug therapies and studies on pathogenic mechanisms of disease. Animal models of disease provide a valuable, ethically and economically viable experimental platform to examine these mechanisms and identify biomarkers that may be therapeutic targets that would facilitate the development of improved standard of care ,The classic animal model of COPD is both time consuming and costly.. In the present study, a short time murine model of cigarette smoke extract induced COPD was used to investigate the time course of airway and pulmonary inflammatory response. Groups of BALB/C mice were intraperitoneally injected with cigarette smoke extract or to saline for 2 months, The pathological changes and the inflammation level was evaluated by hematoxylin eosin (H&E) staining. Results: Intraperitoneal injection of CSE induced in response to cigarette smoke extract , inflammatory cells (i.e. neutrophils, macrophages) progressively accumulated both in the airways and lung parenchyma of mice. Furthermore, a perivascular and peribronchial clear infiltration of lymphocytes was observed accompanied with a mild destruction of alveolar walls (emphysema) .Conclusions, the present study showed that short term (60 days) intraperitoneal exposure to CSE leads to chronic inflammation, alveolar septal degradation and emphysema accompanied in mice. All histopathological alterations in this model are shared with the COPD patients lung histopathology.