A mechanistic study of cigarette smoke-induced COPD in C57Bl/6 mice and the effects of cessation or switching to a pMRTP aerosol


1. **Materials and Methods**
   - **Animals**
     - C57Bl6 mice, 8–10 weeks old
     - Male mice
   - **Methods**
     - Random allocation
     - Exposure
     - Sham
     - Cigarette smoke
     - 3R4F
     - pMRTP

2. **Results**
   - **Transcriptomics**
     - Network Perturbation Amplitudes (NPA) and Biological Impact Factor (BIF)
     - Gene expression data
     - Affymetrix microarrays
     - Protein levels
     - MMP activity

3. **Conclusions**
   - We have established a model of smoke-induced emphysema in C57Bl6 mice that mimics many of the characteristics of human COPD.
   - Cigarette smoke results in increased infiltration of inflammatory cells and mediators into the lung, which were quantified in the bronchoalveolar lavage fluid.
   - Pulmonary function
   - Histopathology

**References**
- 1. **Introduction**
   - Chronic obstructive pulmonary disease (COPD) is defined by the WHO as a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is associated with narrowing of the small airways, chronic bronchitis, and the development of airway hyperresponsiveness.
   - In developed countries, cigarette smoking is the main etiological factor in the pathogenesis of COPD. However, the underlying pathogenesis of the disease is not fully understood. Identification of a murine model is a prerequisite for the mechanistic study of smoking-induced COPD. Among various mouse models for the study of experimental emphysema/COPD, it has been shown that the C57Bl/6 mouse is a useful model for cigarette smoke-induced COPD studies.

**Results and endpoints**

**Bronchoalveolar lavage fluid (BALF)**

**Free lung cells**

**Pulmonary function**

**Histopathology**

**Figure 1. Study design with groups, and total Animals per group**