Probing molecular mechanisms of tobacco smoke-induced lung inflammation via cell-specific multi-omic analysis in a murine smoke exposure model

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I. INTRODUCTION

- Tobacco consumption has been conclusively linked to lung cancer.
- Evidence suggests that tobacco smoke stimulate oncogenesis through the initiation of inflammation.
- Research has shown that oncogenesis is preceded by epigenetic changes.

II. EXPERIMENTAL METHODS

- Mice were kept in plastic holding cages where they were subjected to cigarette exposure for 4 days a week, 2 days for extended periods.
- Samples were collected following 3 weeks of exposure, 10 weeks of exposure, and 10 weeks of exposure with a four-week recovery.
- As a positive control for lung inflammation, mice were dosed intravenously with lipopolysaccharide (LPS) for 3 hours before being analyzed.
- Tissues were harvested, and RNA was isolated from each sample.

III. EXPOSURE OF LUNGS TO STIMULI RESULTS IN ALTERED PROTEIN ABUNDANCES

- A proteomic analysis was performed to investigate changes in protein expression.
- Following 10 weeks of smoke exposure, mice were sacrificed, and lung tissues were harvested.
- Samples were analyzed using mass spectrometry and protein expression analysis.

IV. PROTEIN ABUNDANCE AND RNA EXPRESSION ANALYSES

- A hierarchical clustering analysis was performed to identify protein expression changes.
- RNA expression analysis was conducted to identify changes in gene expression.

V. PROTEIN ABUNDANCE AND EPIGENOMIC CHANGE ANALYSIS

- A genomic analysis was performed to identify changes in DNA methylation and histone modifications.
- Correlations between protein abundance and DNA methylation were identified.

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