

Down-Regulation of Caveolin-1 in a Murine Model of Acute Allergic Airway Disease

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Background: Airway remodeling refers to the structural changes in the airways of asthma. Caveolin-1 can reduce cell growth and negatively regulates smooth muscle cell proliferation. The aim of this study was to investigate lung caveolin-1 status in a murine model of acute allergic airway disease.

Methods: Six to eight week-old female BALB/c mice were sensitized by intraperitoneal injections of phosphate-buffered saline or ovalbumin (OVA) and aluminium hydroxide on days 0 and 14, challenged with aerosolized saline or OVA (1%) on days 21-25, 28-32, and 35. The mice were killed 1 day after the last OVA/saline challenge.

Results: Serum OVA-specific IgE levels were significantly elevated in OVA-challenged mice when compared to saline-challenged mice. Percentage of inflammatory cells in the bronchoalveolar lavage was significantly higher in the OVA-challenged animals. The animals' lungs that were sensitized and challenged with OVA contained large numbers of inflammatory cells concentrated near the airways and in the perivascular areas. The thickness of the bronchial epithelial layer and smooth muscle layer and the numbers of total inflammatory cells and eosinophils significantly increased in OVA-challenged mice. Caveolin-1 mRNA expression had significantly decreased and type I collagen mRNA expression increased significantly in the lung tissue of OVA-challenged mice.

Conclusion: These results suggest that caveolin-1 seems to be involved in the pathogenesis of airway remodeling of acute allergic airway disease.