ApolipoproteinA1 Promote Resolution of Lung Inflammation and Fibrosis in Experimental Silicosis

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Background: In our previous study, ApoA1 is decreased in the lung of idiopathic pulmonary fibrosis and administration of ApoA1 into the lung shows a potentials to reduce the experimental bleomycin induced lung injury/fibrosis. It remains unknown whether ApoA1 exert the anti-inflammatory and anti-fibrotic effect on other types of lung injury/fibrosis.

Aims: To evaluate the anti-inflammatory and anti-fibrotic effect of ApoA1 in experimental lung silicosis and to reveal the mechanism behind the effects.

Methods: BALB/C mice were assigned into four groups. Silica suspension (SiO2; 20 mg/50 ml in saline) was intratracheally instilled at Day 0. ApoA1 was treated via intranasal route at Day 7, 8, 9 (group 1) to evaluate the prevention effect of ApoA1 and to evaluate the resolving effect at Day 16, 17, 18 (group 2). The Mice were sacrificed at day 30. Broncholaveolar lavage, histology, collagen, TUNEL assay and cytokine measurement including TGF-b1 were performed.

Results: Both group 1 and group 2 had a significant reduction in the number of inflammatory cells in BAL fluids as well as the number of silicotic nodules fractions and collagen deposition compared to those of silica-treated mice. TGF-b1 level in BALF was markedly decreased in both ApoA1-treated groups compare to silica treated group. TUNEL assay shows that silica induced increased apoptotic cell numbers, which was significantly reduced by ApoA1.

Conclusions: ApoA1 have preventive and resolving effects on the experimental lung silicosis via down regulation of fibrotic cytokine and apoptosis.