European Respiratory Society
Annual Congress 2012

Abstract Number: 1066
Publication Number: 365

Abstract Group: 5.3. Allergy and Immunology
Keyword 1: Asthma - mechanism **Keyword 2:** Animal models **Keyword 3:** Anti-inflammatory

Title: Apolipoprotein A1 regulate innate immune response and tight junction formation in the airway epithelium to promote the resolution of allergic airway inflammation

Dr. Sung Woo 7206 Park swpark@schmc.ac.kr MD [1], Ms. Eunhee 7207 Lee c0089@schmc.ac.kr [1], Dr. Ansoo 7208 Jang jas877@schmc.ac.kr [1], Dr. SooTaek 7209 Uh uhs@schmc.ac.kr [2], Dr. YongHoon 7210 Kim welkim@schmc.ac.kr [3], Dr. David 7211 Erle David.Erle@ucsf.edu [4] and Dr. Choonsik 7212 Park schalr@schmc.ac.kr [1]. [1] Internal Medicine, Soonchunhyang University Bucheon Hospital, Bucheon, Republic of Korea ; [2] Internal Medicine, Soonchunhyang University Seoul Hospital, Seoul, Republic of Korea ; [3] Internal Medicine, Soonchunhyang University Chonan Hospital, Chonan, Republic of Korea and [4] Lung Biology Center, University of California San Francisco, United States .

Body: Apolipoprotein A-1(ApoA1), main component of HDL have anti-inflammatory effect as well as reverse cholesterol transport. The objective of this study was to determine airway levels of ApoA1 in the asthmatics as well as its effect on innate immune response and resolution of inflammation on experimental asthma. Two-dimensional electrophoresis was performed for differential display proteomics in the bronchoalveolar lavage (BAL) fluids from asthmatics. Intra-nasal ApoA1 was treated (D24,25) after challenged ovalbumin(OVA, D22,23,24) in an established murine model of asthma. Mild to moderate asthmatics had significantly less ApoA1 in BAL fluid than healthy controls. In a murine model of asthma, ApoA1 suppressed the cardinal features of asthma when given after OVA challenge. ApoA1 significantly decreased lung IL-25, IL-33 levels as well as other Th2 cytokines.ApoA1 inhibits the production of IL-25, IL33, and CCL20 in the allergen treated cultured primary bronchial epithelial cells. ApoA1 also increased production of lipoxin A4(LXA4)in the OVA challnged lung and promote restore the allergen induced disrupted tight junctions proteins Zo-1 and occludin in the bronchial epithelium.Our data demonstrate that ApoA1 regulate both initiation and resolution of airway inflammation. The mechanism includes down regulation of IL-25, IL-33 and CCL20 expression in the epithelium which promote Th2 response. ApoA1 regulate pro-resolution mediator LXA4 and tight junction proteins production. Together, ApoA1 could be therapeutic strategy for chronic airway inflammation such as asthma.