European Respiratory Society
Annual Congress 2013

Abstract Number: 1065
Publication Number: P3913

Abstract Group: 3.3. Mechanisms of Lung Injury and Repair
Keyword 1: Animal models **Keyword 2:** Cell biology **Keyword 3:** Epigenetics

Title: Cell adhesion molecules and cathelicidin in mice model of hypersensitivity pneumonitis

Marta 8852 Lemieszek martalemieszek@gmail.com [1], Matthias 8853 Wielscher matthias.wielscher.fl@ait.ac.at [2], Marcin 8854 Golec msgolec@yahoo.com MD [1], Klemens 8855 Vierlinger Klemens.Vierlinger@ait.ac.at [2], Czeslawa 8856 Skórska skorska@op.pl [1], Barbara 8857 Mackiewicz b.mack@wp.pl MD [3], Anna 8858 Góra angora@vp.pl MD [4], Rolf 8859 Ziesche rolf.ziesche@meduniwien.ac.at MD [5], Jacek 8860 Dutkiewicz dutkiewi@galen.imw.lublin.pl [1] and Janusz 8861 Milanowski janusz.milanowski@am.lublin.pl MD [1,3]. [1] Unit of Fibroproliferative Diseases, Institute of Rural Health, Lublin, Poland ; [2] Health & Environment Department Molecular Medicine, Austrian Institute of Technology, Vienna, Austria ; [3] Department of Pneumonology, Oncology and Allergology, Medical University of Lublin, Lublin, Poland ; [4] Department of Allergy and Environmental Hazards, Institute of Rural Health, Lublin, Poland and [5] Department of Internal Medicine II, Clinical Division of Pulmonary Medicine, Medical University of Vienna, Vienna, Austria .

Body: Hypersensitivity pneumonitis (HP) is a complex clinical condition involving a cascade of immune reactions triggered and sustained by lung injury due to repeated inhalations of fine particles inhaled to airways. Pathogenesis of this disease characterized by allergic, environmental and occupational background is still far from being completely unraveled. The aim of the work was to study pathomechanism of HP with the use of new mice model of HP predictive of the human response. 3- and 18-motnhs old C57BL/6J mice were exposed to cell extract of gram negative bacterium Pantoea agglomerans using the novel inhalation challenge set (2). Lung samples were taken before treatment and after 7 and 28 days of exposure. Gene expression profiling followed by qRT-PCR for the selected genes was performed on Fluidigms Biomark. Analysis of the microarray results unraveled activation of few groups of genes, including set of genes belonging to the cell adhesion molecules pathway. Conducted qRT-PCR confirmed significant up-regulation of expression of the following genes: claudin 5, intercellular adhesion molecule 2, platelet/endothelial cell adhesion molecule 1, integrin alpha 8 and cathelicidin antimicrobial peptide. Cell adhesion molecules ensure proper permeability of pulmonary epithelial barrier, including tight junctions. Cathelicidin peptide participates in epithelial wound healing and exerts antimicrobial activity. Significantly increased expression levels of genes coding these molecules in the presented above HP mice model indicate their role in response to lung injury and in the course of the disease. This work was supported by the EU FP7 Health Research Grant Number HEALTH-F4-2008-202047.