

European Respiratory Society Annual Congress 2012

Abstract Number: 2561

Publication Number: P3165

Abstract Group: 4.3. Pulmonary Circulation and Pulmonary Vascular Disease

Keyword 1: Pulmonary hypertension **Keyword 2:** Biomarkers **Keyword 3:** Proteomics

Title: Screening for biomarkers in pulmonary hypertension

Mrs. Svenja Lena 6742 Tiede Svenja.Tiede@mpi-bn.mpg.de^{1,2}, Dr. Soni Savai 9507 Pullamsetti Soni.Pullamsetti@mpi-bn.mpg.de¹, Dr. Akylbek 9509 Sydykov Akylbek.Sydykov@innere.med.uni-giessen.de², Prof. Dr Günter 9510 Lochnit Guenter.Lochnit@biochemie.med.uni-giessen.de³, Dr. Henning 9511 Tiede Henning.Tiede@innere.med.uni-giessen.de MD², Prof. Dr Hossein Ardeschir 9512 Ghofrani Ardeschir.Ghofrani@innere.med.uni-giessen.de MD², Prof. Dr Norbert 12803 Weissmann Norbert.Weissmann@innere.med.uni-giessen.de², Prof. Dr Friedrich 12810 Grimminger Friedrich.Grimminger@innere.med.uni-giessen.de MD², Prof. Dr Werner 12816 Seeger Werner.Seeger@innere.med.uni-giessen.de MD² and Prof. Dr Ralph Theo 12821 Schermuly Ralph.Schermuly@innere.med.uni-giessen.de². ¹ Lung Development and Remodelling, Max-Planck-Institute for Heart and Lung Research, Bad Nauheim, Hesse, Germany, 61231 ; ² University of Giessen Lung Center, Justus-Liebig University, Giessen, Hesse, Germany, 35392 and ³ Protein Analytik, Biochemisches Institut, Justus-Liebig University, Giessen, Hesse, Germany, 35392 .

Body: Introduction: Pulmonary hypertension (PH) is a progressive and fatal disease. The gold standard for diagnosing PH and estimating prognosis is the invasive method of right heart catheterization. To date no biomarker is available to prove or exclude the diagnosis of PH. Aims and objectives: The aim of this study is to identify and validate new biomarkers for PH. Methods: Plasma from the pulmonary artery banding (PAB) and the monocrotaline (MCT) rat model, and corresponding sham and control animals (n=9), was used for 2D-gel electrophoresis (2D-GE) and MALDI-TOF-MS analysis. Further, plasma changes of interesting candidates were confirmed by ELISA. Human study population consists of patients with idiopathic pulmonary arterial hypertension (n=40), PH associated with collagen vascular disease (n=45), pulmonary venous hypertension (n=44), chronic thromboembolic PH (n=45), and non-PH controls (n=34). Results: The spot density analysis of 2D-GE and identification by MALDI-TOF-MS revealed 7 proteins significantly changed in PAB vs. sham, and 15 proteins in MCT vs. control group. Complement component 4 (C4) and complement inhibitory factor H (CFH) were upregulated in PAB and MCT. ApoE was changed 15-fold in MCT plasma, but not in PAB. The analysis of the human samples revealed no significant difference in mean plasma ApoE between the patient groups (119.4±10.3, 147.6±11.6, 116.8±9.9, 110.2±8.3 µg/ml) and controls (135.3±14 µg/ml). Conclusions: Despite published data on the role of ApoE in PH and the significant changes in rats, ApoE seems not suitable as biomarker for PH in humans. Other candidates identified by mass spectrometry will be evaluated for their potential as biomarker.