European Respiratory Society
Annual Congress 2013

Abstract Number: 7202

Publication Number: P4395

Abstract Group: 10.1. Respiratory Infections

Keyword 1: Immunology **Keyword 2:** Pneumonia **Keyword 3:** Animal models

Title: Pulmonary infections as a result of stroke induced immunodepression

Ms. Claudia 1419 Dames claudia.dames@charite.de [1], Mr. Levent 1420 Akyüz levent.akyuez@charite.de [1,2], Mr. Odilo 1421 Engel odilo.engel@charite.de [3], Ms. Katarzyna 1422 Winek katarzyna.winek@charite.de [3], Prof. Dr Hans-Dieter 1423 Volk hans-dieter.volk@charite.de [1,2], Prof. Dr Andreas 1426 Meisel andreas.meisel@charite.de [3] and Dr. Christian 1427 Meisel chr.meisel@charite.de [1]. [1] Institute for Medical Immunology, Charité Universitätsmedizin, Berlin, Germany ; [2] Berlin-Brandenburg Center for Regenerative Therapies, Charité Universitätsmedizin, Berlin, Germany and [3] Department of Experimental Neurology, Charité Universitätsmedizin, Berlin, Germany .

Body: Pulmonary infection is a highly relevant complication and leading cause of death in patients with acute CNS injury. The high incidence of pneumonia in these patients is likely to be the result of temporary stroke induced immunodepression. We here analysed changes in cellularity and function of lung immune and epithelial cells by FACS and ex vivo stimulation at different time points after experimental stroke in mice. In addition, we monitored pulmonary host responses in vivo during the course of pneumococcal pneumonia by analyzing cellular changes, cytokine/ chemokine levels and barrier function in lungs of stroke mice and sham controls. Analysis of cellularity at different time points after stroke showed a loss of interstitial macrophages within the first day after stroke as well as decreased numbers of dendritic cells and lymphocytes in lung. Functional analysis of immune cells indicates an impaired cytokine production in lung cells after ex vivo TLR stimulation. In a model of induced pneumonia we analysed the in vivo host response to Streptococcus pneumonia after stroke. Our data suggest that pulmonary phagocytes in stroke mice are not able to clear the bacteria. It seems that this results in a prolonged hyper-inflammatory response with increased cytokine levels in lungs followed by a disruption of the epithelial cell barrier function. The correlation between impaired activation of pulmonary antimicrobial immune responses and development of infectious complications is still incompletely understood. A detailed knowledge of leukocyte recruitment and defects in anti-bacterial defences may lead to new therapeutic strategies such as local immunostimulatory treatment to prevent infections and improve outcome.