European Respiratory Society
Annual Congress 2013

Abstract Number: 747

Publication Number: P659

Abstract Group: 3.3. Mechanisms of Lung Injury and Repair

Keyword 1: ALI (Acute Lung Injury) **Keyword 2:** ARDS (Acute Respiratory Distress Syndrome) **Keyword 3:** Lung injury

Title: Altered expression of claudin 5 isotype modulates tight junctions permeability in experimental LPS-induced acute lung injury in mice

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Body: INTRODUCTION: Acute Lung Injury and Acute Respiratory Distress Syndrome (ALI/ARDS) are characterized by lung injury, endothelial barrier disruption and changes in integrity and expression levels of Claudins (CLDN), a key component of endothelial tight junctions. CLDN5 is expressed and augments permeability in lung endothelial cells. This study aims to investigate CLDN5 expression levels in LPS-induced ALI in mice. METHODS: Lung injury was induced in adult male C57BL/6 mice, by intratracheal LPS administration. Animals sacrificed at 6h, 12h and 24h after LPS administration. Lung tissues were harvested, bronchoalveolar lavage fluid (BALF) was obtained, and the histological score of ALI was assessed according to the American Thoracic Society scoring system. Immunohistochemistry was used to assess CLDN5 cellular localization while its expression levels were verified by Western blotting. RESULTS: LPS administration induced ALI as was verified by increased BALF cellularity, protein content, and the histological score. CLDN5 immunoreactivity was distributed along the alveolar epithelium and vascular endothelium. The pulmonary protein expression of CLDN5 was increased as early as 6 hrs after LPS administration by 3.4 fold above the control group ($p= 0.002$). CONCLUSIONS: LPS administration in mice exhibited an early increase of CLDN5 protein expression levels in ALI which may have contributed to augmented paracellular permeability and inflammation. Observations accrued from this study not only elucidate the pathophysiology of this form of injury but also point to novel approaches in its treatment.