European Respiratory Society
Annual Congress 2013

Abstract Number: 4439
Publication Number: 395

Abstract Group: 5.2. Monitoring Airway Disease
Keyword 1: COPD - exacerbations **Keyword 2:** Immunology **Keyword 3:** Inflammation

Title: Altered inflammasome activity in bacterial acute exacerbations of COPD (AECOPD)

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Body: Inflammasome and its products, as part of the innate immune system, can be triggered to assist in defence against invading pathogens. We have previously shown increased levels of IL-18 in induced sputum of stable COPD patients, which were decreased in acute exacerbations of COPD (AECOPD), implying a possible dysregulation of inflammasome in AECOPD. Aim of this study was to assess the inflammasome activity in AECOPD in the case of proven bacterial infections versus AECOPD where only bacterial colonization was proved. 30 patients hospitalized for an infectious AECOPD according to Anthonisen's criteria were included in the study. We examined the inflammatory properties of induced sputum and assessed bacterial infection using PCR. IL-18, caspase-1, TLR-2, and IL-1b were measured in induced sputum and serum by immunosorbent analysis (ELISA). Immunocytochemistry of IL-18 expression in sputum cells was performed using a mouse monoclonal IL-18 antibody. IL-18 levels in sputum were found significantly lower in AECOPD caused by a pathogen compared to colonized AECOPD (207 pg/ml (range 47-2301) vs 420 pg/ml (58-1201), p=0.05). Similarly, although non statistically significantly, decreased levels of caspase-1 and TLR2 were found in infectious versus colonized AECOPD (1.6 pg/ml (1.22-19) vs 3.6 pg/ml (1.27-117), p>0.05). IL-1b was statistically significantly increased in induced sputum of infectious AECOPD (539 pg/ml (1.5-973) vs 88 (2.9-989), p<0.05). Positive staining of IL-18 was observed in macrophages in immunocytochemistry. Our data show that in bacterial AECOPD there may be a dysregulated activation of inflammasome mediating IL-18 production.