European Respiratory Society  
Annual Congress 2012  

Abstract Number: 4484  
Publication Number: P1432

Abstract Group: 3.2. Airway Cell Biology and Immunopathology  
Keyword 1: COPD - mechanism  Keyword 2: Cell biology  Keyword 3: Inflammation

Title: Enhanced cytotoxic function of NK and NKT-like cells associated with decreased CD94 (Kp43) in the airway in COPD

Prof. Greg 27195 Hodge greg.hodge@health.sa.gov.au 1,2, Dr. Violet 27196 Mukaro violet.mukaro@health.sa.gov.au 1,2, Prof. Dr Mark 27197 Holmes mark.holmes@health.sa.gov.au MD 1,2, Prof. Dr Paul 27198 Reynolds paul.reynolds@health.sa.gov.au MD 1,2 and Prof. Sandra 27199 Hodge sandra.hodge@health.sa.gov.au 1,2. 1 Thoracic Medicine, Royal Adelaide Hospital, Adelaide, Australia, 5001 and 2 Lung Research, Hanson Institute, Adelaide, Australia.

Body: NK and NKT-like cells represent a small but important proportion of effector lymphocytes that we have previously shown to be a major source of pro-inflammatory cytokines and granzymes1. We hypothesized that NK and NKT-like cells would be increased in the airway in COPD and that this would be accompanied by a reduction in expression of the inhibitory receptor CD94 (Kp43) and increased expression of the cytotoxic mediators granzyme B and perforin. We measured NK and NK-like T-cells and their expression of CD94 in the blood of patients with COPD (n=61), smokers (16) and healthy controls (25) and BAL from a cohort of subjects. We further assessed activation by expression of CD69 and cytotoxic potential by production of granzymes A and B and using a cytotoxicity assay. In blood from COPD subjects, there were no significant changes in NK or NKT-like cell numbers or expression of granzyme A or cytotoxic potential vs controls. There was however, increased expression of granzyme B and decreased expression of CD94 by both cell types vs controls. In the airway in COPD, NK and NKT-like numbers were increased, associated with increased NK cytotoxicity, increased expression of granzyme B and decreased expression of the inhibitory receptor CD94. Treatment strategies that target NK and NKT-like cells, their cytotoxicity and production of inflammatory mediators in the airway may improve COPD morbidity.