

European Respiratory Society Annual Congress 2013

Abstract Number: 3284

Publication Number: P612

Abstract Group: 3.2. Airway Cell Biology and Immunopathology

Keyword 1: Biomarkers **Keyword 2:** COPD - mechanism **Keyword 3:** No keyword

Title: Increased circulating endothelial microparticles in COPD

Jéssica 19646 García jssc5@hotmail.com¹, Dr. Roberto 19647 Del Pozo rdelpozo@clinic.ub.es MD¹, Nuria 19616 Coll ncoll85@gmail.com¹, Cristina 19617 Bonjoch bonjoch_14@hotmail.com¹, Dr. Isabel 19618 Crespo ICRESPO@clinic.ub.es¹, Dr. Melina 19619 Musri mmusri@clinic.ub.es¹, Dr. Olga 19620 Tura TURA@clinic.ub.es¹, Dr. Victor Ivo 19621 Peinado vpeinado@clinic.ub.es² and Dr. Joan Albert 19659 Barberà jbarbera@clinic.ub.es MD¹. ¹ Pulmonary Medicine, Hospital Clínic-IDIBAPS, Barcelona, Spain and ² Pulmonary Medicine, CIBER De Enfermedades Respiratorias, Barcelona, Spain .

Body: Rationale: COPD is associated with alterations of pulmonary circulation. Endothelial microparticles (EMPs) are <1 µm vesicles shed from the endothelium in response to cell activation, inflammation and/or apoptosis. The number of EMPs is considered a biologic marker of endothelial dysfunction, being increased in cardiovascular disorders. The number of EMPs could be increased in COPD. Aim: To assess the number of circulating EMPs in COPD patients, compared with control subjects, and assess if several indices of COPD severity are related with differences in circulating EMP counts. Methods: Circulating EMPs were determined in 31 control subjects and 57 COPD patients. EMPs were determined by flow cytometry in platelet-free plasma (PFP) according to the expression of membrane-specific antigens: CD31+CD42b-AnnexinV+ for the apoptotic phenotype and CD31+CD42b-CD62E+ for the activated phenotype. In COPD patients we assessed differences in EMP counts related to FEV1, DLCO, gas trapping, PaO2 and the presence of pulmonary hypertension (PH). Results: The number of circulating EMPs was increased in COPD patients compared with control subjects (median, 504 (IQR, 295-779) vs 340 (250-495) EMPs/µl PFP; p<0.05). EMPs with apoptotic phenotype were also higher in COPD (412 (221-654) vs 241 (198-353) EMPs/µl PFP; p<0.05), whereas no differences were observed in EMPs with activated phenotype. No associations were found between the number of EMPs and the values of FEV1, DLCO, gas trapping, PaO2 or the presence of (PH). Conclusion: COPD is associated with an increased number of circulating EMPs, presumably originated by an apoptotic mechanism. The severity of COPD does not seem to influence the number of circulating EMPs. Supported by FIS PS09/00536.