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**Title:** Innate immunity but not NLRP3 inflammasome activation correlates with severity of stable COPD

Dr. Antonino 844 Di Stefano antonino.distefano@fsm.it [1], Gaetano 845 Caramori crm@unife.it [2], Adam 846 Barczyk abarczyk@sum.edu.pl [3], Chiara 847 Vicari vicari.chiara@gmail.com [1], Paola 848 Brun paola.brun@unipd.it [4], Andrea 849 Zanini andrea.zanini@fsm.it [1], Francesco 850 Cappello francapp@hotmail.com [5], Elvira 851 Garofano elvira.garofano@unife.it [2], Anna 852 Padovani anna.padovani@unife.it [2], Marco 853 Contoli ctm@unife.it [2], Andrew 854 Durham a.durham@imperial.ac.uk [6], Peter 855 Barnes p.j.barnes@imperial.ac.uk [6], Kian Fan 856 Chung f.chung@imperial.ac.uk [6], Alberto 857 Papi ppa@unipd.it [2], Ian 858 Adcock ian.adcock@imperial.ac.uk [6] and Bruno 859 Balbi bruno.balbi@fsm.it [1]. [1] Divisione Di Pneumologia e Citoimmunopatologia, Fondazione S. Maugeri, IRCCS, Veruno, Tradate, Italy ; [2] Centro Interdipartimentale Per Lo Studio Delle Malattie Infiammatorie Delle Vie Aeree, Università Di Ferrara, Ferrara, Italy ; [3] Katedra I Klinika Pneumologii, Universytetu Medycznego W Katowicach, Slaskiego, Poland ; [4] Department of Biomedical Sciences, Università Di Padova, Padova, Italy ; [5] Dipartimento Di Biomedicina Sperimentale e Neuroscienze Cliniche, Università Di Palermo, Palermo, Italy and [6] Airway Disease Section, National Heart and Lung Institute, London, United Kingdom .

**Body:** Background: In models of COPD, environmental stressors induce innate immune responses, inflammasome activation and inflammation. However, the interaction between these responses and their role in driving pulmonary inflammation in stable COPD is unknown. Objectives: To investigate the activation of innate immunity and inflammasome pathways in the bronchial mucosa and bronchoalveolar lavage (BAL) of stable COPD patients of different severity and control healthy smokers and non-smokers. Methods: Innate immune mediators (IL-6, IL-7, IL-10, IL-27, IL-37, TSLP, IFNγ and their receptors, STAT1 and pSTAT1) and inflammasome components (NLRP3, NALP7, caspase 1, IL-1β and its receptors, IL-18, IL-33, ST2) were measured in the bronchial mucosa using immunohistochemistry. IL-6, soluble IL-6R, sgp130, IL-7, IL-27, HMGB1, IL-33, IL-37 and soluble ST2 were measured in BAL using ELISA. Results: In bronchial biopsies IL-27+ and pSTAT1+ cells are increased in severe COPD compared to control healthy smokers. IL-7+ cells are increased in both COPD and control smokers compared to control non-smokers. In severe stable COPD IL-7R+, IL-27R+ and TSLPR+ cells are increased in comparison with both control groups. The NALP3 inflammasome is not activated in stable COPD patients compared with control subjects. The inflammasome inhibitory molecule NALP7 and IL-37 are both increased in COPD compared to control smokers. In BAL of stable COPD patients, IL-6 levels are increased and soluble gp130 and ST2 levels decreased, compared to control smokers. Conclusion: Increased expression of IL-27, IL-37 and NALP7 in the bronchial mucosa may be involved in progression of stable COPD.