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Title: Lung dendritic cells from chronic obstructive pulmonary disease patients induce type 1 T regulatory cells

Dr. Maria 14212 Tsoumakidou tsoumak@yahoo.gr MD [1,2], Dr. Sofia 14213 Tsousa sofia.tousa@gmail.com MD [2], Dr. Maria 14214 Semitekolou msemi@bioacademy.gr [2], Dr. Panagiota 14215 Panagiotou nagiapan@hotmail.com MD [1], Dr. Anna 14216 Panagiotou nagiapan@hotmail.com MD [1], Dr. Eleni 14226 Litsiou elitsiou@yahoo.gr [1], Dr. Ioannis 14236 Morianos jmor@bioacademy.gr [2], Mrs. Maria 14259 Konstantinou tsoumak@yahoo.gr [3], Dr. Konstantinos 14261 Potaris tsoumak@yahoo.gr MD [3], Prof. Dr Spyros 14271 Zakynthinos szakynthinos@yahoo.com MD [1] and Dr. Georgina 14283 Xanthou gxanthou@bioacademy.gr [2]. [1] Critical Care Medicine and Pulmonary Services, Evangelismos General Hospital, Athens, Greece ; [2] Cellular Immunology, Biomedical Research Foundation of the Academy of Athens, Greece and [3] Thoracic Surgery, Sotiria Chest Hospital, Athens, Greece .

Body: The high mortality rate and health care costs associated with Chronic Obstructive Pulmonary Disease (COPD) are due to a great extend to recurrent infectious exacerbations. Impaired T cell immunity might explain this susceptibility to infections. Mature dendritic cells (DCs) are crucial players in the induction of T cell responses against infectious agents. By contrast, immature DCs induce tolerance by promoting the differentiation of regulatory T cells (Tregs). We have previously shown that lung DCs of COPD patients express low levels of co-stimulatory molecules, respond poorly to stimulation and display low ability to prime autologous lung T cells and allogeneic naive T cells. Importantly, naïve T cells primed with lung DCs from patients with COPD inhibit T cell proliferation. Here, we have characterized the gene and protein expression profile of these regulatory cells and investigated the mechanism of their suppressive function. Naïve CD4+ T cells primed with lung DCs from patients with COPD showed increased gene expression for Foxp3, Ahr and GATA3 (assessed by qRT-PCR) compared to T cells primed with lung DCs from smokers without COPD. Accordingly, flow cytometry analysis showed higher IL-10 and Foxp3 intracellular protein expression. These findings suggest that the induced regulatory cells are Tregs type 1. Type 1 Tregs suppress immune responses primarily through IL-10. Indeed, naïve T cells that had been primed with COPD lung DCs failed to inhibit T cell proliferation in the presence of blocking IL-10 receptor antibody. Our findings show that lung DCs from patients with COPD induce type 1 Tregs.