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Title: CSE exposure abrogates regulatory effects of the bronchial epithelium on B cell survival and IgA production in a coculture system. A pivotal role of TACI

Dr. Maha Zohra 14864 Ladjemi maha.ladjemi@uclouvain.be [1], Mr. Bruno 14865 Detry bruno.detry@uclouvain.be [1], Ms. Marylène 14866 Lecocq marylene.lecocq@uclouvain.be [1,3], Prof. Dr Yves 14867 Sibille yves.sibille@uclouvain.be MD [1,2] and Prof. Dr Charles 14868 Pilette charles.pilette@uclouvain.be MD [1,3]. [1] Institut de Recherche Expérimentale & Clinique, Pole de Pneumologie, ORL & Dermatologie, Université Catholique de Louvain (UCL), Brussels, Belgium, 1200 ; [2] Service de Pneumologie, Cliniques Universitaires de Mont-Godinne, Yvoir, Belgium and [3] Service de Pneumologie & Centre de l'allergie, Cliniques UCL Saint-Luc, Brussels, Belgium, 1200 .

Body: Background: Chronic obstructive pulmonary disease (COPD) is associated with chronic airway inflammation and structural remodelling, in particular of the epithelium, which is impaired in its capacity to transport immunoglobulin (Ig) A. Although COPD is mostly related to cigarette smoking, only a minority of smokers develops this disease and factors of cigarette exposure susceptibility remain not clear. Even though peribronchial lymphoid follicles have been described in severe COPD, it remains unknown whether B-cell conditioning is altered in this disease, especially after CSE (Cigarette Smoke Extract) exposure. Objectives: In this study, we report on CSE exposure data using a coculture model of B cells with human primary bronchial epithelium (re)differentiated in vitro in air-liquid interface. Methods: IgA synthesis was studied following CSE exposure in CD19+ B cells (purified by immunomagnetic sorting from healthy blood donors) after co-culture for 13 days with a bronchial epithelium from severe COPD patients. B cells were also assessed by flow cytometry for cell activation and survival. Results: In four independent experiments, we observed that IgA production and cell survival were upregulated in B cells cocultured with the bronchial epithelium, as compared to B cells cultured alone. CSE exposure of the epithelium abrogated these effects, and this was associated with the suppression of TACI induction upon co-culture. Conclusion: These data suggest that a crosstalk exists between B cells and the epithelium with respect to COPD, which could be mediated at least in part through regulation of TACI.