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Title: Alveolar type II cells transplantation in pulmonary fibrosis: Effect on the lung macrophage activation

Ms. Raquel 14017 Guillamat-Prats raquel.guillamat@iibb.csic.es [1,3], Dr. Sabrina 14018 Gea-Sorli sabrinagea7@hotmail.com [1], Dr. Gemma 14019 Gay-Jordi gemma.gayjordi@iibb.csic.es [1,3], Dr. Daniel 14020 Closa daniel.closa@iibb.cscic.es [1], Mr. Luis-Ignacio 14021 Sanchez-Lopez jisbam@iibb.csic.es [1], Ms. Valeria 14022 Sirenko valeria.sirenko@iibb.csic.es [1], Ms. Fernanda 14023 Hernandez-Gonzalez hernandez.gonzalez.fer@gmail.com MD [1,2,3], Dr. Oriol 14024 Bulbena obubam@iibb.csic.es [1], Dr. Antoni 14025 Xaubet axaubet@clinic.ub.es MD [2,3,4] and Dr. Anna 14052 Serrano-Mollar anna.serranomollar@iibb.csic.es [1,3]. [1] Experimental Pathology, Institut D'Investigacions Biomediques De Barcelona-CSIC, Barcelona, Spain, 08036 ; [2] Servicio De Neumologia, Hospital Clínic, Barcelona, Spain, 08036 ; [3] CIBERES, Centro De Investigaciones Biomédicas En Red De Enfermedades Respiratorias, Mallorca, Spain, 07110 and [4] UB, Universidad De Barcelona, Barcelona, Spain .

Body: The phenotype adopted by alveolar macrophages (AM) and interstitial macrophages (IM) could determine the progression of fibrosis. Macrophages activation is classified into M1 or M2. M1 is a proinflammatory phenotype and M2 is involved in tissue remodeling. Our group has shown that alveolar type II cells (ATII) transplantation is able to reverse pulmonary fibrosis. Therefore, the objective of the study was to evaluate the effect of ATII transplantation in macrophages activation in an experimental model of pulmonary fibrosis. The induction of fibrosis was performed in rats by the intratracheal instillation of bleomycin (3.5U/kg). The animals were transplanted with ATII ($2.5x10^6$ cells/animal) 15 days after bleomycin instillation and were sacrificed 21 days after the induction of fibrosis. AM were obtained by bronchoalveolar lavage and IM by digestion of the lung tissue. Macrophages activation was determined by RT-PCR of TNFα and IL1β specific M1 markers and arginase-I, mannose receptor (MR), IL10 and TGFβ specific M2 markers. In AM, the expression of M2 markers as arginase-I, IL10 and TGFβ significantly increased in the fibrotic group. ATII transplantation was able to decrease the expression of all these markers. In contrast, M1 markers were unchanged. In IM, expression of all M1 and M2 markers increased significantly in the fibrotic group. ATII transplantation was able to decreased the expression of IL1β, arginase-I, IL10 and TGFβ. During pulmonary fibrosis, all the macrophages adopted a M2 phenotype characteristic of tissue remodeling. ATII transplantation decreases M2 phenotype of both macrophage populations, which could be related to the reduction of fibrotic lesions.