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Title: Role of macrophage activation in type II alveolar cells transplantation for the treatment of idiopathic pulmonary fibrosis

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Body: In previous experimental studies, we found that alveolar type II cells (ATII) are successful in reversing lung fibrosis. Recently it has been reported that alternatively activated macrophages (M2) play a critical role in idiopathic pulmonary fibrosis (IPF), and appear to be involved in lung tissue remodelling and extracellular matrix deposition. The purpose of this study is to investigate whether intratracheal transplantation of ATII can inhibit M2 polarized human alveolar macrophages or shifts them to a classic activation or M1 phenotype. This could play a significant role in the inhibition of pulmonary fibrosis progression. We included 15 patients with IPF diagnosed in the last 3 years, who underwent ATII trasplantation by fiberoptic bronchoscopy. Bronchoalveolar lavage cell isolation was performed 1 month before and 2 months after the ATII transplantation. The alveolar macrophages were isolated and purified in culture plates. We investigated the expression levels of M1 markers proteins TNF-α, IL-1β and iNOS, and M2 markers proteins IL-10, Arginase-I, FOLR2 and TGF-β using the real-time RT-PCR. Alveolar macrophages from transplanted patients released higher amounts of the three investigated M1 marker proteins TNF-α, IL-1β and iNOS, compared to cells from pre-transplanted patients. Moreover, a decrease in expression of M2 marker proteins TGF-β and Arginase-I was revealed, without any change in IL-10 or FOLR2 levels. In conclusion, we have demonstrated that ATII transplantation may produce a shift of alveolar macrophages activation to a M1 phenotype. This shift could thus modulate the fibrotic response and end up the progression of IPF in transplanted patients.