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Title: Novel biomarkers in bleomycin-induced pulmonary fibrosis

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Body: Idiopathic Pulmonary Fibrosis is a chronic lung disease of unknown etiology, with fatal outcome. Bleomycin model is the most used and known model of lung fibrosis, however a comprehensive compartmental screening of new targets with clinical translational potential is lacking. Extrapolation of novel markers from cellular compartments, i.e. broncho-alveolar lavage (BAL) and peripheral blood, are required to improve the understanding of the pathophysiology and develop an exhaustive assessment of the disease. Methods Fibrosis was induced by intratracheal installation of Bleomycin or PBS as control in 8-12 weeks old mice. BAL cell counts; lung function (via FlexiVent) and histology were performed at day 7, 14, 21, 28 and 56 after treatment. ICAM, VCAM, MMP9, E-selectin and PAI-1 were measured in serum via Luminex and MMP7 and WISP-1 via ELISA. Gene screening was performed with RealTime Custom panel by qPCR. Results Histology, and lung function parameters confirmed fibrosis. Increase in ICAM-1 levels in serum strongly correlated with a fibrotic phenotype and tissue stiffness (compliance r2= 0.5941, elastance r2=0.7149), MMP9 and MMP7 were significantly elevated in bleomycin-injured animals at day 7 and 14. WISP-1 was significantly elevated in resolution phase of fibrosis(PBS n=20; Bleomycin n=14). Developmental pathway signaling are regulated in alveolar macrophages of bleomycin-treated mice. Conclusion We identified novel discovery of disease regulators in different compartments (e.g. alveolar macrophages, BAL, serum) from bleomycin model and further correlation with fibrotic patients are critical to broaden the understanding, ultimately leading to detection accuracy, modify outcomes, and predict prognosis in IPF patients.