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Semaphorin 7A Expression in Pulmonary Fibrosis**Abstract:**

We have recently identified a novel pathway in the pathogenesis of TGF- β 1 induced pulmonary fibrosis that may be applicable to human disease. Mouse modeling of inducible TGF- β 1 overexpression has shown that Semaphorin 7A (Sema-7A), a protein with important neurologic and immunomodulatory functions, plays a crucial role in the development of pulmonary fibrosis. Pilot translational studies by our group has found that peripheral blood mononuclear cells from patients with IPF (n=7) show a 10 fold increase in Sema-7A expression compared to patients with non-specific interstitial pneumonia (n=8, p<0.05) and normal controls (n=8, p<0.05). Flow cytometric studies are ongoing to determine the cell population(s) expressing this marker. We now plan to determine the lung expression of Sema-7A expression in human lung tissue. Using tissue from patients with pulmonary fibrosis and chronic obstructive lung disease obtained from the Lung Tissue Research Consortium, an NIH sanctioned anonymous tissue bank, we will use gene and protein expression analysis to test the hypothesis that expression of Sema 7A and its downstream mediators are elevated in the lungs of patients with pulmonary fibrosis compared to patients with other types of lung disease. If successful, these studies could lead to new areas of investigation and and novel therapies for this otherwise fatal disease.