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Body: VEGF is both a growth and permeability factor implicated in the pathogenesis of idiopathic pulmonary fibrosis (IPF). The presence of hypoxia and its treatment with high flow oxygen have been proposed to contribute to lung injury. VEGF gene transcription is tightly regulated by a hypoxia response element. We hypothesised that: 1) VEGF and its receptors would be differentially expressed between normal (NF) and fibrotic fibroblasts (FF) and 2) Hypoxia and hyperoxia would alter fibroblast VEGF expression. Methods: NF(n=5) and FF(n=5)(from patients with proven usual interstitial pneumonia) were extracted from lung biopsies using the explant method. VEGF receptors levels were analysed at the mRNA and protein level (qPCR and western blotting (WB)). Pan VEGF isoforms were detected at the protein level by ELISA. Fibroblasts were grown in culture for 24 hours in normoxic, hyperoxic (90% O2) and hypoxic conditions (21% O2 with CoCl2). Results: Both NF and FF expressed VEGFR1, VEGFR2, NRP-1 and NRP-2. No significant difference was detected in receptor expression at mRNA or protein level. FF expressed significantly more total VEGF than NF by ELISA:(NF 180.5 pg/ml vs FF 332.0 pg/ml, p=0.01). Moreover, a significant increase in VEGF expression was observed in both NF and FF in response to hypoxic growth conditions (NF 205.7 pg/ml vs 1382.0 pg/ml, p = 0.05, FF 394.6 pg/ml vs 1113 pg/ml.0, p = 0.01). A trend towards increased VEGF expression was also seen in FF vs NF exposed to hyperoxic conditions. Conclusion:Differential expression of VEGF between NF and FF suggests a potential role in the development of IPF. Hypoxia and possibly hyperoxia may alter VEGF bioavailability with important implications in the use of oxygen therapy to manage this disease.