

Supplementary Figure Legends

Figure S1. DMOG and hypoxia increase extracellular fibronectin with limited fibrillar assembly.

A. Immunolabeling of HK2 cells for extracellular fibronectin shows increased deposition but not assembly with hypoxia and DMOG compared with normoxia. Cells were labeled with fibronectin before cell permeabilization (magenta), and for actin filaments with rhodamine (green) after permeabilization. **B.** Immunolabeling of human lung alveolar A549 cells for extracellular fibronectin as indicated in (A) after maintaining cells for 7 days in the indicated conditions shows fibrillar deposited fibronectin with TGF- β that is markedly less with hypoxia. Scale bar 15 μ m

Figure S2. Hypoxia does not increase TGF- β secretion compared with normoxia in HK2 cells.

Relative secreted total and active TGF- β determined by luciferase assays using MLEC cells expressing a truncated plasminogen activator inhibitor-1 (PAI-1) promoter and treated with conditioned medium from normoxia or hypoxia HK2 cells. Data are expressed as luciferase units (RLU) and represent means \pm s.e.m. of six samples in two separate cell preparations.

Fig. S3. TGF- β and hypoxia increase total and cell surface expression of α 5-integrin.

A. Immunoblots of lysates from normoxia control HK2 cells and cells treated with TGF- β or 1% hypoxia for 48 h and probed with antibodies for α 5-integrin and for β -actin as a loading control. **B.** Quantified α 5-integrin signal normalized to β -actin and expressed relative to normoxia controls. **C.** FACS plots of cell surface labeled α 5-integrin including negative control of normoxia cells in the absence of α 5-integrin antibodies and percent cells labeled with α 5-integrin antibodies for the indicated conditions of normoxia, TGF- β and hypoxia.

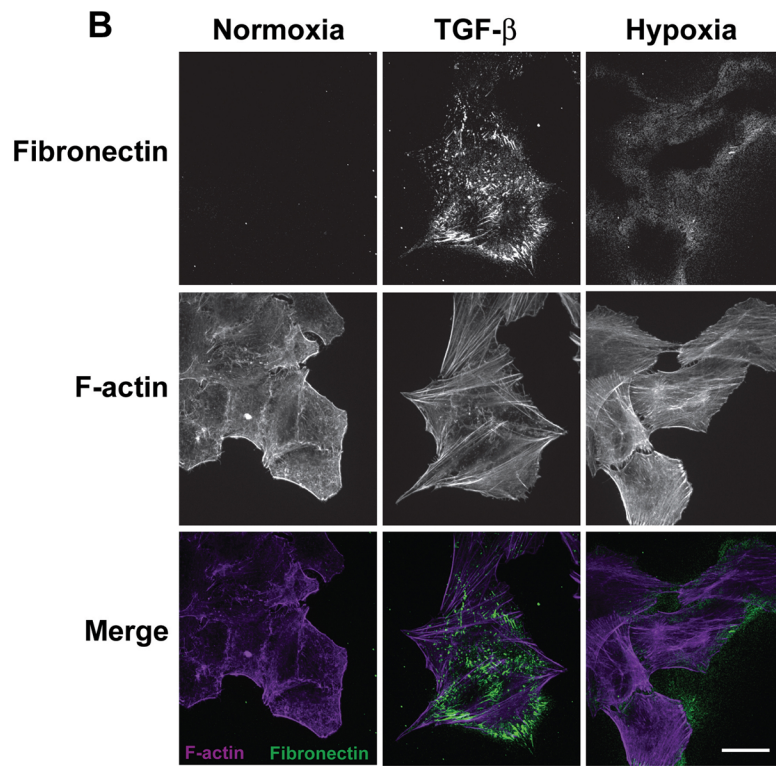
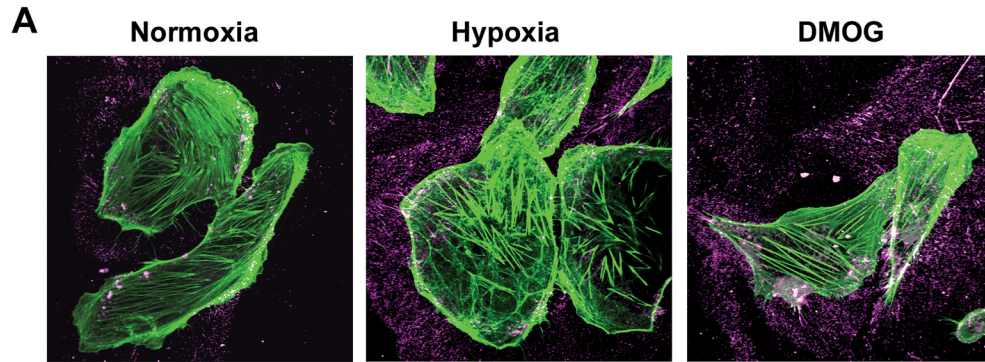


Fig. S1

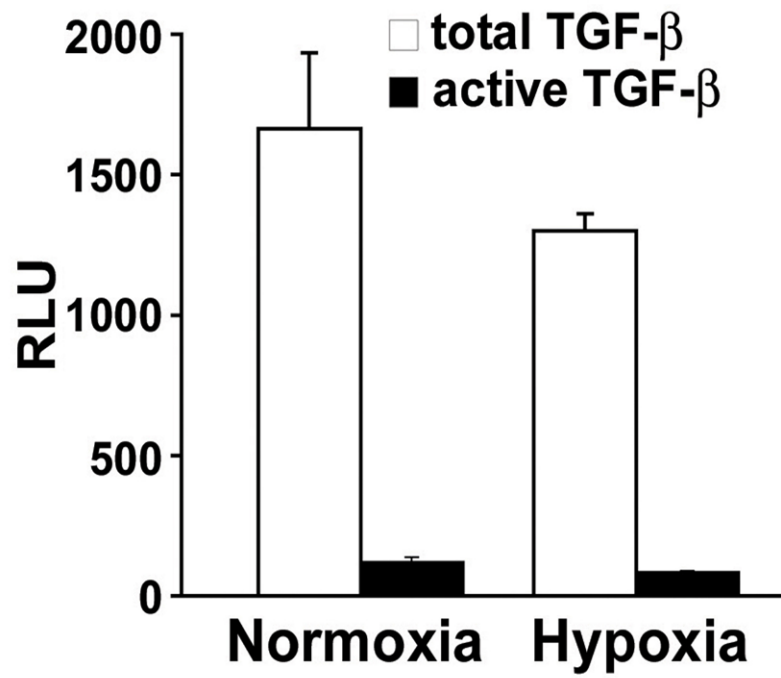


Fig. S2

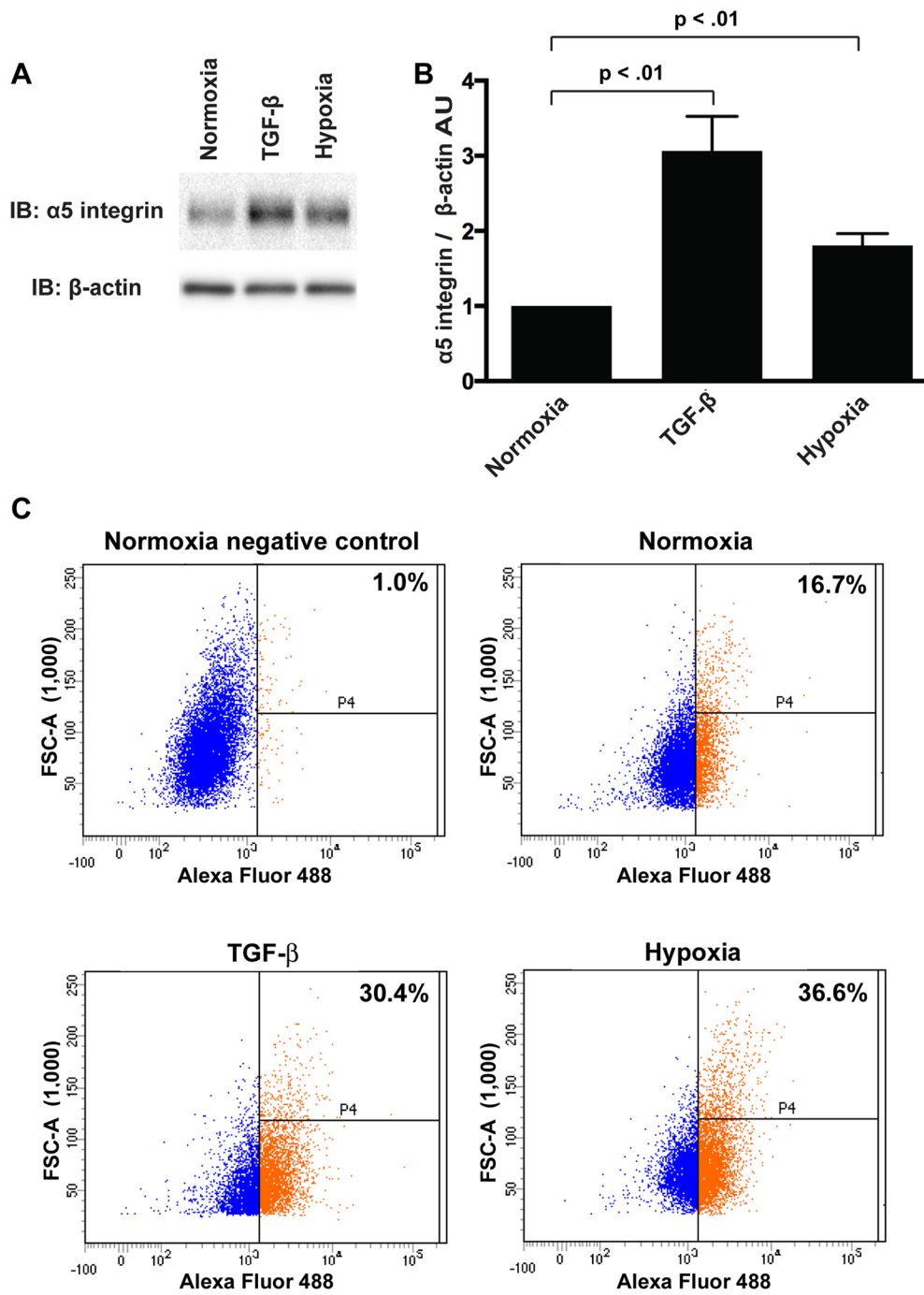


Fig. S3