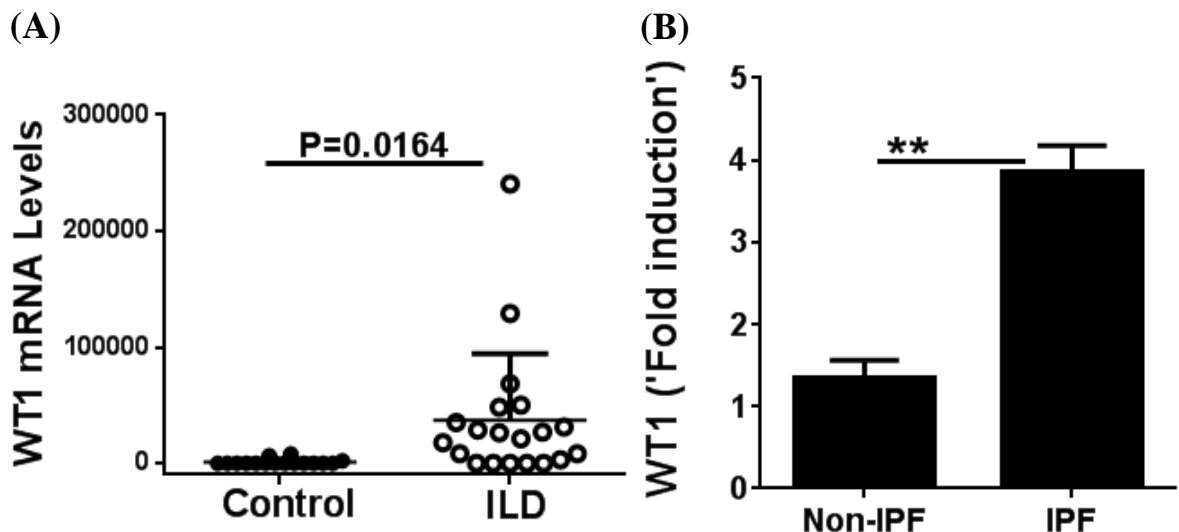
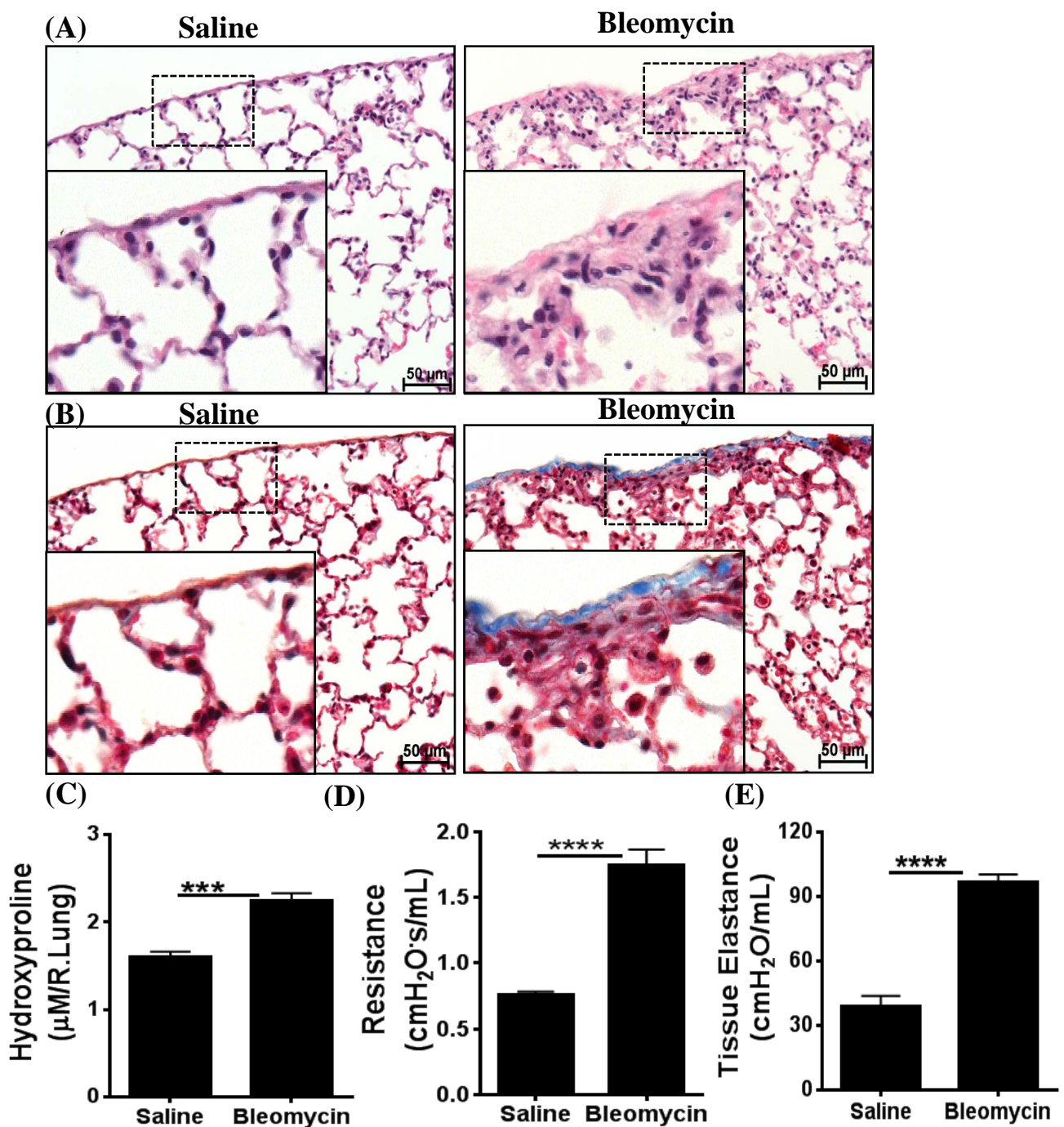


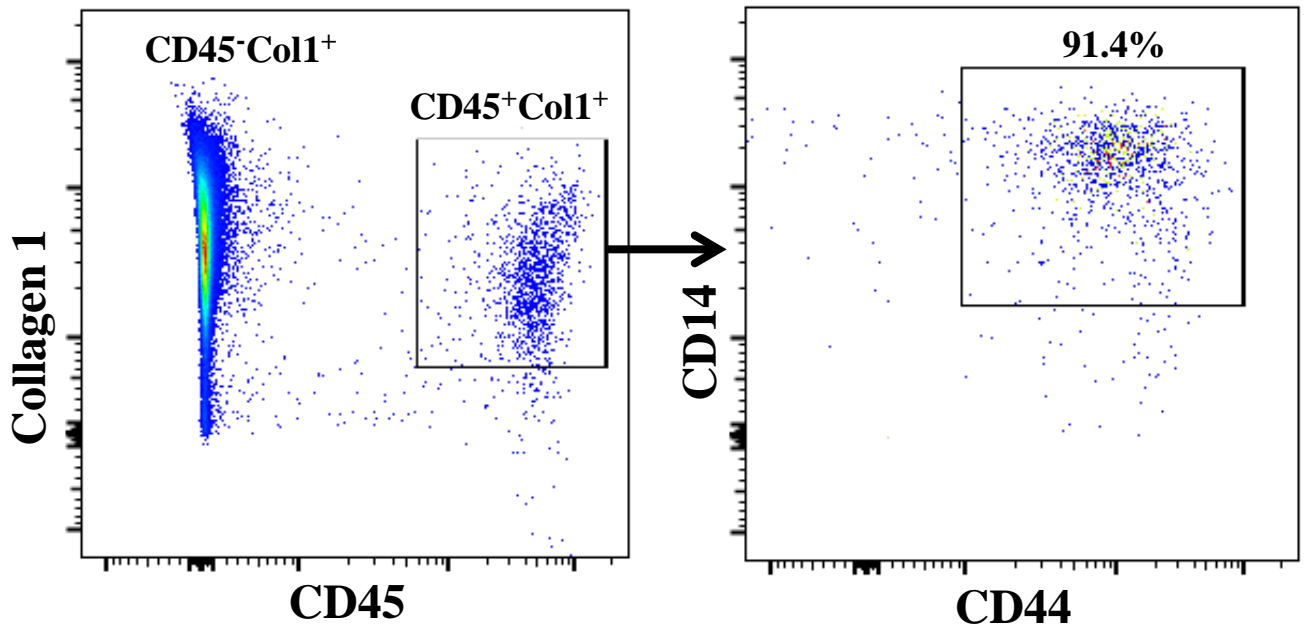
Supplementary Figure 1. Overexpression of TGF α alters the network of genes regulated by Wilms tumor 1 during TGF α -induced pulmonary fibrosis. (A) CCSP^{-/-} and CCSP/TGF α mice were placed on doxycycline (Dox)-treated food for 3 weeks, RNA was isolated, and RNA-seq analysis was performed using next-generation sequencing. Heat map shows two clusters of differentially expressed genes up or down regulated (indicated with color key) by two fold or more in TGF α -induced fibrotic lungs compared to CCSP^{-/-} control mice. (B) Ingenuity pathway analysis shows the network of genes regulated by Wilms tumor 1 during TGF α -induced pulmonary fibrosis. Shapes denote functional class of genes as indicated in the box with legends. Shapes filled with the colors red and green depict up and down regulated genes, respectively, in the lungs of TGF α mice compared to control mice. Solid and dashed lines depict direct and indirect interactions, respectively. (C) Transcripts of genes identified to be regulated through WT1 network were quantified using RT-PCR in the lungs of TGF α transgenic mice fed doxycycline-treated food for 0 and 28 days show increase in the transcript levels of IL-10, MYCN, TERT, and THBS4. Gene expression values were normalized using hypoxanthine guanine phosphoribosyltransferase as a control. Data are means + SEM, and the statistical significance between groups was measured using an unpaired Student's t-test. *P<0.05 (n=4/group).



Supplementary Figure 2. Up-regulation of WT1 in humans with fibrotic lung disease. (A) WT1 transcripts were elevated in individuals with interstitial lung disease (ILD) compared to non-ILD controls. WT1 expression data was extracted from the mRNA expression catalogue available in the database of the Lung Genomic Research Consortium (LGRC). Data are means + SEM, and statistical significance was calculated using Student's *t*-test between groups. (B) Primary lung resident mesenchymal cells from the mesenchymal-cell cultures of IPF or non-IPF were isolated by negative selection using anti-CD45 magnetic beads. RNA from cell lysates were analysed for WT1 transcript levels by RT-PCR. Gene expression values were normalized using β -actin as a control. Data are means \pm SEM (n=2). Statistical significance was calculated using an unpaired Student's *t*-test between groups. **P<0.005.



Supplementary Figure 3. Increased inflammation, collagen deposition and the lung function decline during bleomycin-induced pulmonary fibrosis Bleomycin was administered via intradermal injections (100 $\mu\text{g/day}$; 5 days/week) for 4 wks and lungs were assessed for fibrosis and lung function. **(A)** H&E staining indicate inflammatory cells in the sub-pleura and interstitium of bleomycin-treated mice. **(B)** Masson trichrome staining indicate collagen deposition (blue) in the pleura/subpleura of bleomycin-treated mice. **(C)** The total lung hydroxyproline levels were increased in bleomycin-treated mice compared to saline-treated control. **(D)** The resistance in airways was increased in bleomycin-treated mice compared to saline-treated control and **(E)** Tissue elastance was increased in bleomycin-treated mice compared to saline-treated control. Data shown are means + SEM. Statistical significance between groups was measured using an unpaired Student's *t*-test.



Supplementary Figure 4. The subpleural fibrotic lesions of IPF comprises of fibrocytes that express monocyte marker, CD14 and tissue invasion marker, CD44. The FACS gating was drawn to show the percentage of CD14⁺CD44⁺ cells in the total fibrocytes (CD45⁺Col1⁺).