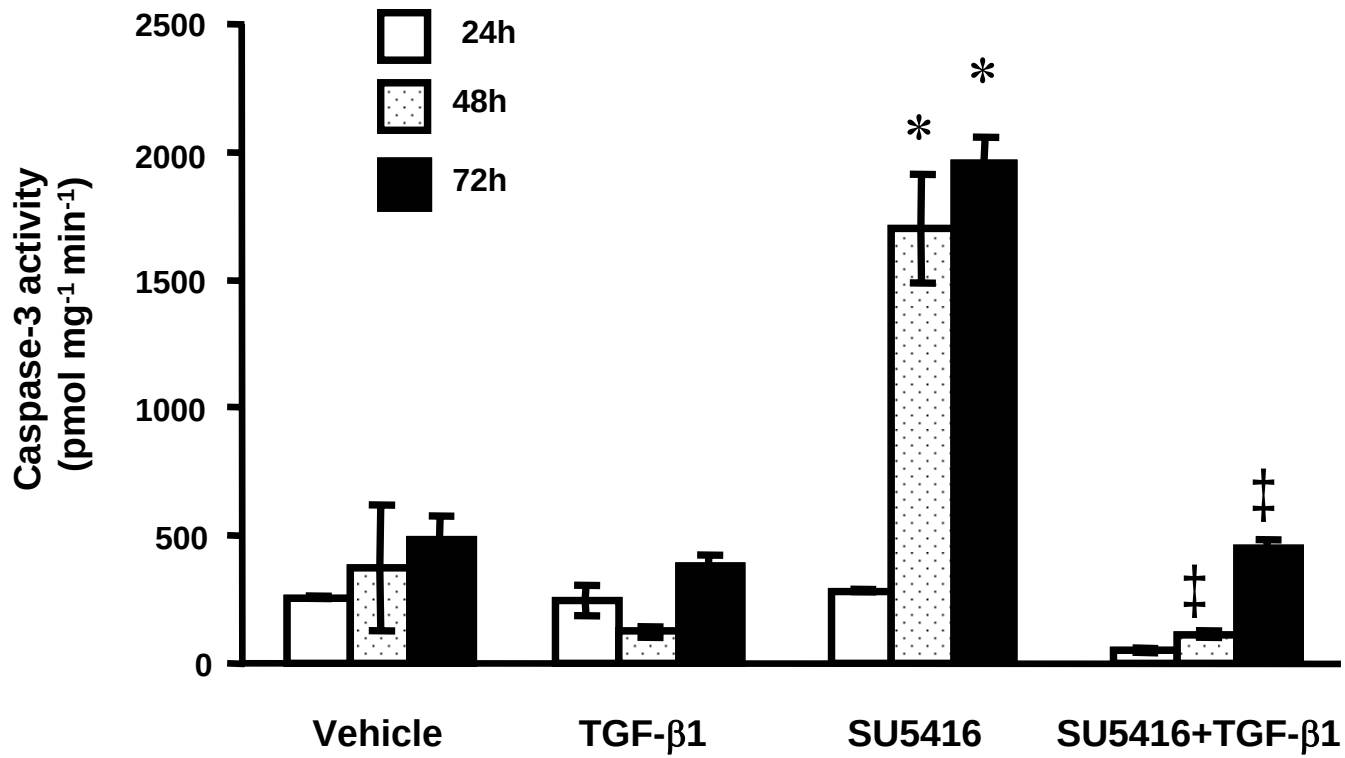
Supplemental Figure 1: ***The effects of TGF-β1 on bovine pulmonary artery endothelial cell apoptosis induced by blockade of VEGF receptors.*** Panel a: BPAEC were incubated in MEM plus 10% FBS with vehicle or SU5416 (5 μM) in the absence or presence of TGF-β1 (1 ng/ml) for 24, 48, and 72 h. Panel b: BPAEC were incubated in MEM plus 10% FBS with various concentrations (0, 0.1, 0.5, 5.0 and 10 μM) of SU5416 in the absence or presence of TGF-β1 (1 ng/ml) for 48h. Caspase-3 activities were assessed by enzyme activity assay. * $p<0.001$ vs BPAEC exposed to vehicle; ‡ $p<0.001$ vs BPAEC exposed to SU5416 in the absence of TGF-β1. Panel c: BPAEC were incubated with vehicle or SU5416 (5 μM) in the absence or presence of TGF-β (1 ng/ml) in MEM plus 10% FBS for 48h and caspase-3 cleavage was assessed by immunoblot analysis using an antibody recognizing both procaspase-3 and the cleaved caspase-3. The protein level of vinculin was used to control for protein loading. Panels a and b: all data are presented as the mean ± SE from three independent experiments. Panel c: a representative blot from three independent experiments is shown.

Supplemental Figure 2: ***The effect of TGF-β1 on apoptosis of human pulmonary artery endothelial cells.*** HPAEC were incubated with EBM2 medium in the absence or presence of varying concentrations of TGF-β1 for 24h and caspase-3 activation was assessed by immunoblot analysis using antibodies recognizing procaspase-3 and the cleaved caspase-3, respectively. The protein level of vinculin was used to control for protein loading. A representative blot from three independent experiments is shown.

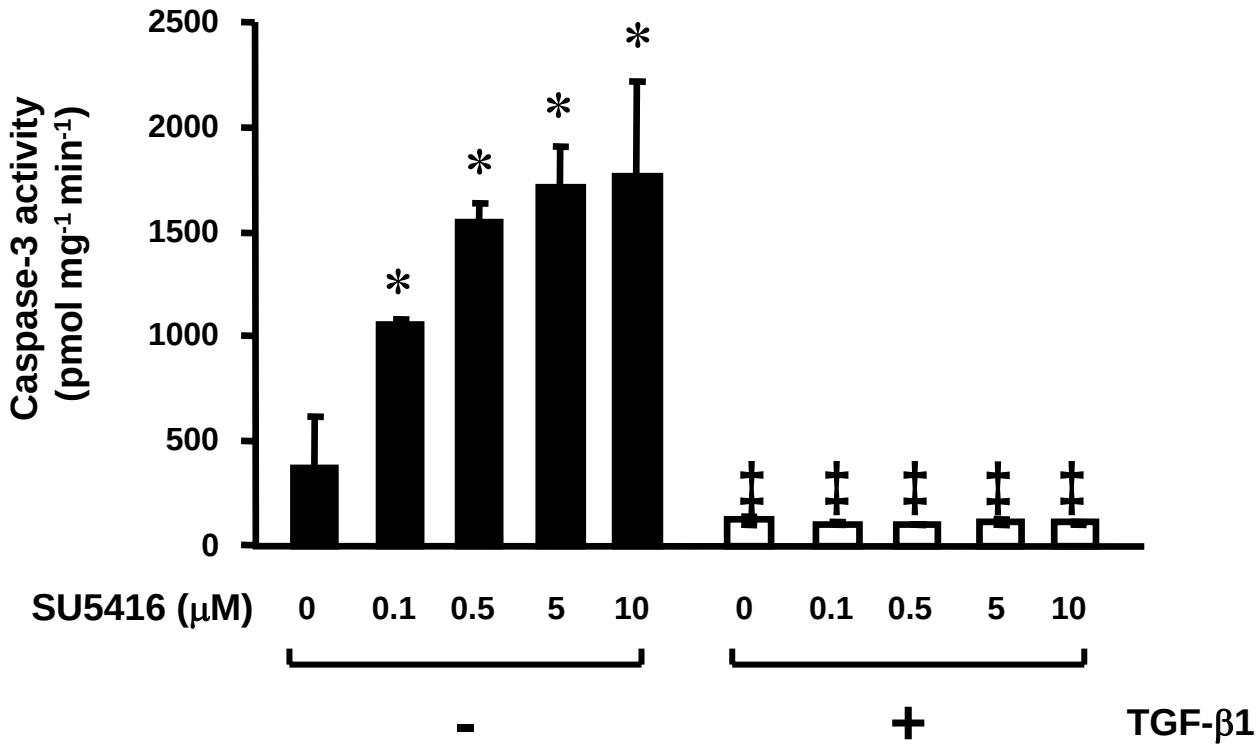
Supplemental Figure 1

a.



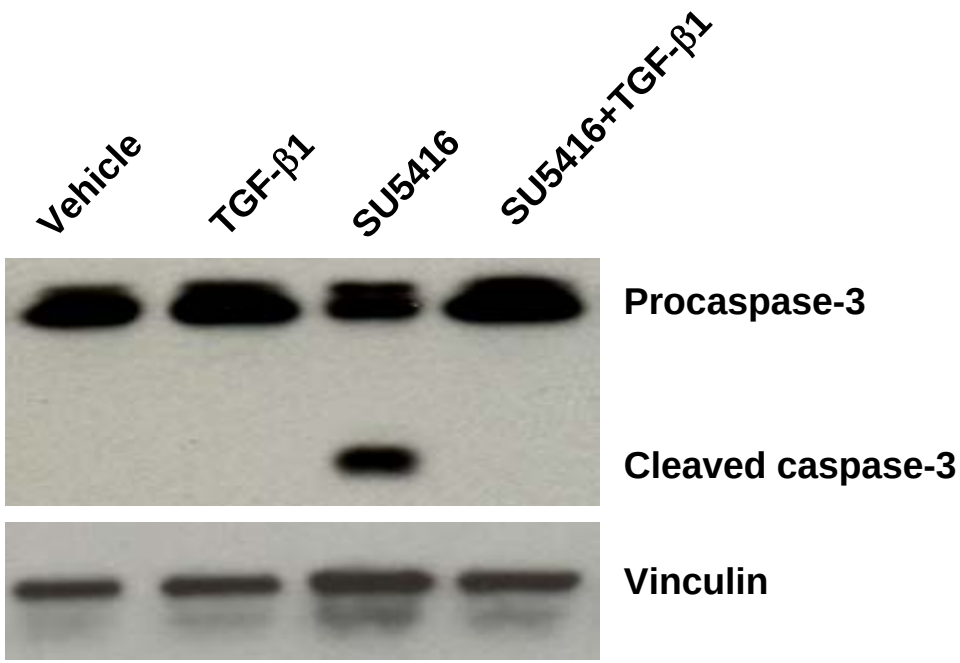
Supplemental Figure 1

b.



Supplemental Figure 1

c.



Supplemental Figure 2