Supplemental Figures

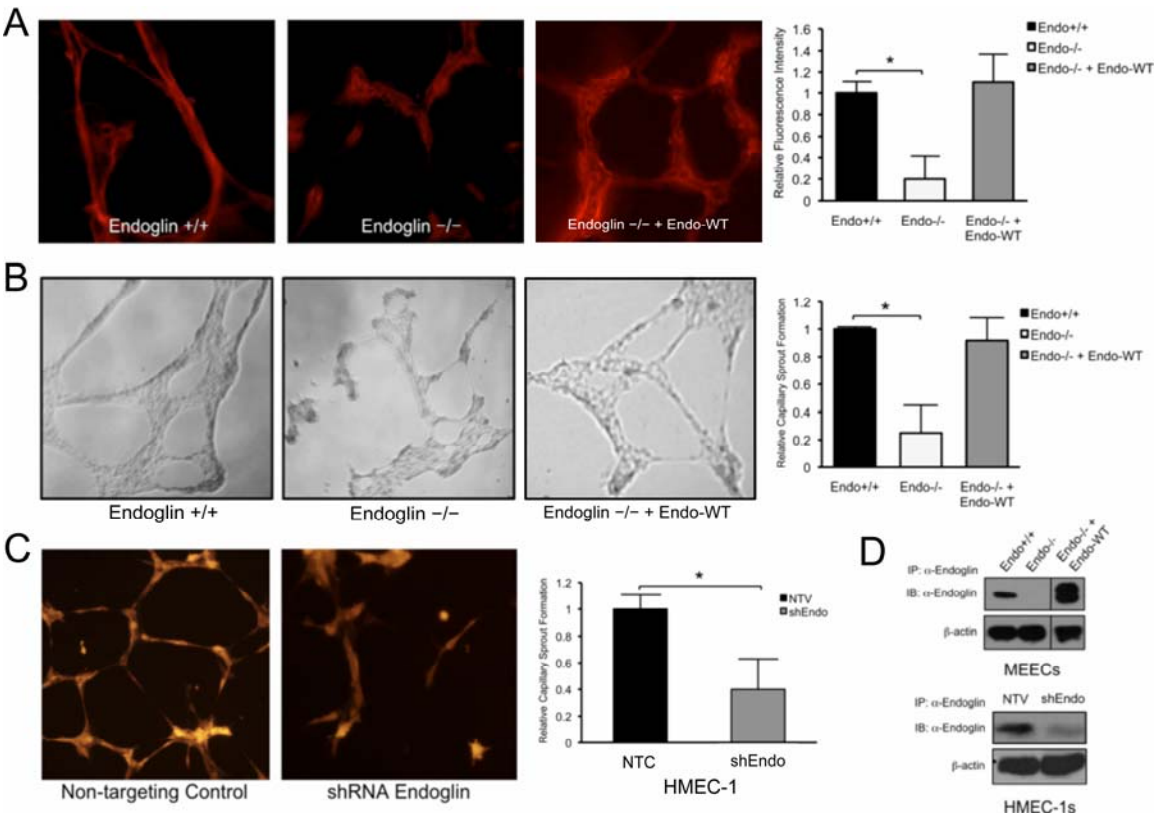

Fig. S1

(A) Immunofluorescence staining of VE-cadherin levels in Endo+/+, Endo-/- MEECs and Endo-/- + Endo-WT.

(B) 3-D collagen matrix assay assessing the effects of endoglin expression on tube formation (Endo+/+, Endo-/- MEECs and Endo-/- + Endo-WT MEECs).

(C) Fluorescence (red) of RFP-expressing HMEC-1s indicates capillary sprouting levels when HMEC-1s are infected with either non-targeting vector (NTC) or shRNA to endoglin.

(D) Biochemical analyses of endoglin expression in MEECs (upper panels) and HMEC-1s upon shRNA-mediated knockdown of endogenous endoglin (lower panels).

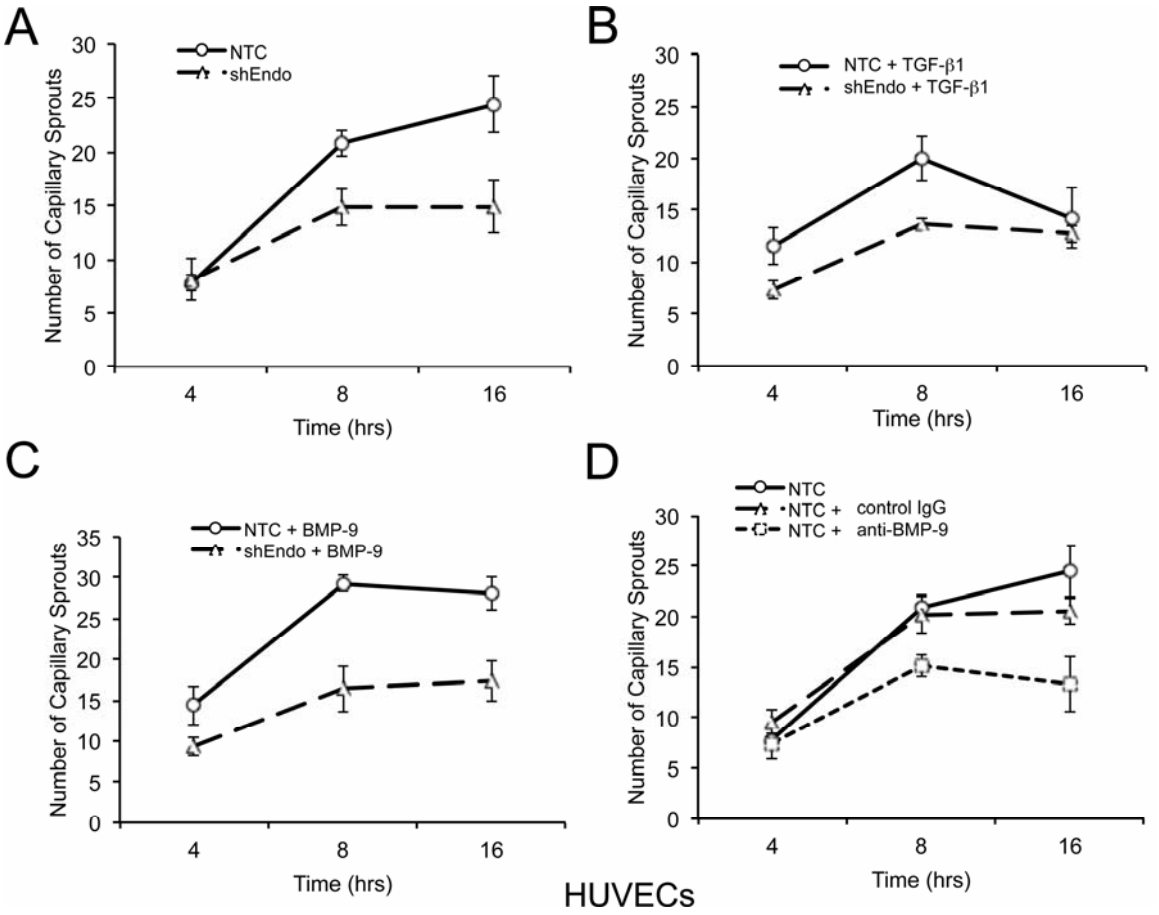

Fig. S2

(A) Primary HUVECs infected with adenovirus for non-targeting control (NTC) or shRNA to endoglin are plated for Matrigel-induced endothelial capillary sprouting assay.

(B-C) Primary HUVECs infected with adenovirus for NTC or shRNA to endoglin are plated for Matrigel-induced endothelial capillary sprouting treated with TGF-β1 (50 pM) or BMP-9 (16.5 nM) for 16 h.

(D) Primary HUVECs infected with adenovirus for NTC or shRNA to endoglin are plated for Matrigel-induced endothelial capillary sprouting no treatment, IgG control, or BMP-9 antibody (1 μg/mL).

Quantification of capillary sprouting for HUVECs represented in each graph is derived from at least 3 independent experiments. Each time point is statistically significant with $p<0.05$.

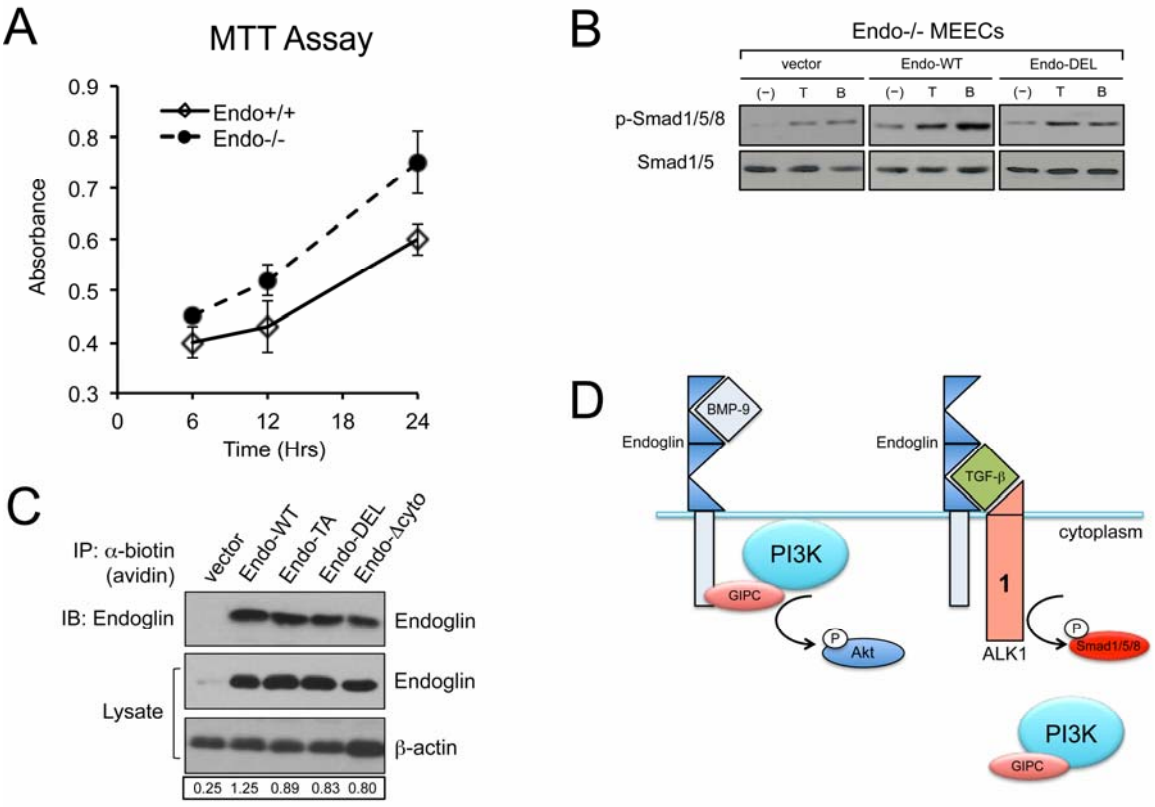

Fig. S3

(A) Endo+/+ and Endo-/- MEEC cell proliferation determined by MTT assay. Data reflects three independent experiments done in triplicates.

(B) Comparison of Smad1/5/8 activation in Endo-WT versus Endo-DEL expressing Endo-/- MEECs. Cells were treated with TGF-β1 (50 pM) or BMP-9 (16.5 nM) for 30 min.

(C) Comparison of cell surface endoglin expression by cell surface biotinylation. Cell surface expression of Endo-WT, Endo-TA, Endo-DEL, and Endo-Δcyto in Endo-/- MEECs were quantified via densitometric analysis based on band intensities of biotinylated endoglin (top panel) relative to endoglin expression in lysate (middle panel). β-actin was used as loading control (lower panel).

(D) Schematic model of endoglin regulation of PI3K/Akt signalling.