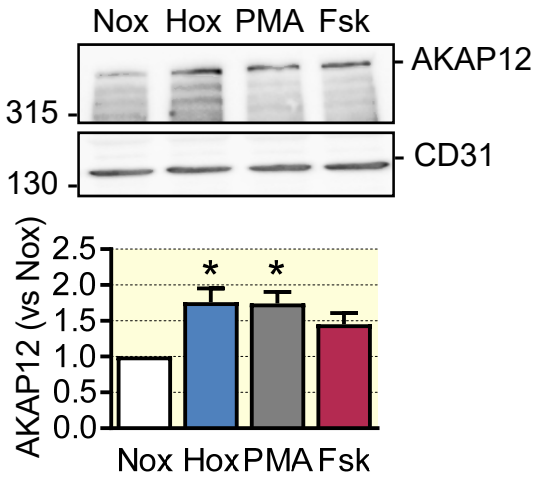

Figure S1. AKAP12 expression in sub-confluent human endothelial cells. Effect of normoxia (Nox), hypoxia (Hox, 1% O_2; 48 hours) on the expression of AKAP12 in sub-confluent cultures of human endothelial cells. PMA (0.1 µmol/L) and forskolin (Fsk, 1 µmol/L) were included as positive controls; n=5 experiments from 5 different batches of endothelial cells (one-way ANOVA with Newman-Keuls multiple comparison test). *P<0.05 versus Nox.

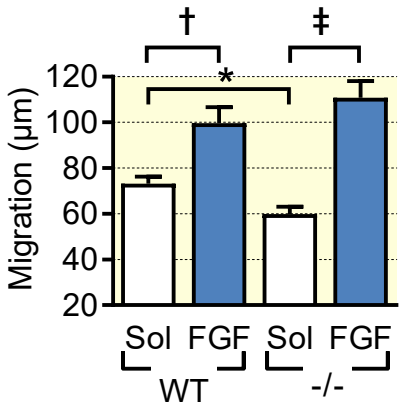

Figure S2. consequences of AKAP12 deletion on endothelial cell migration in vitro. Scratch wound assay with mouse lung endothelial cells from wild-type (WT) or AKAP12$^{-/-}$ (-/-) mice in the presence of solvent (Sol) or bFGF (10 ng/ml) n=3 different cell preparations per genotype, 6 experiments per preparation; (one-way ANOVA with Newman-Keuls). *P<0.05, †P<0.01, ‡P<0.001.

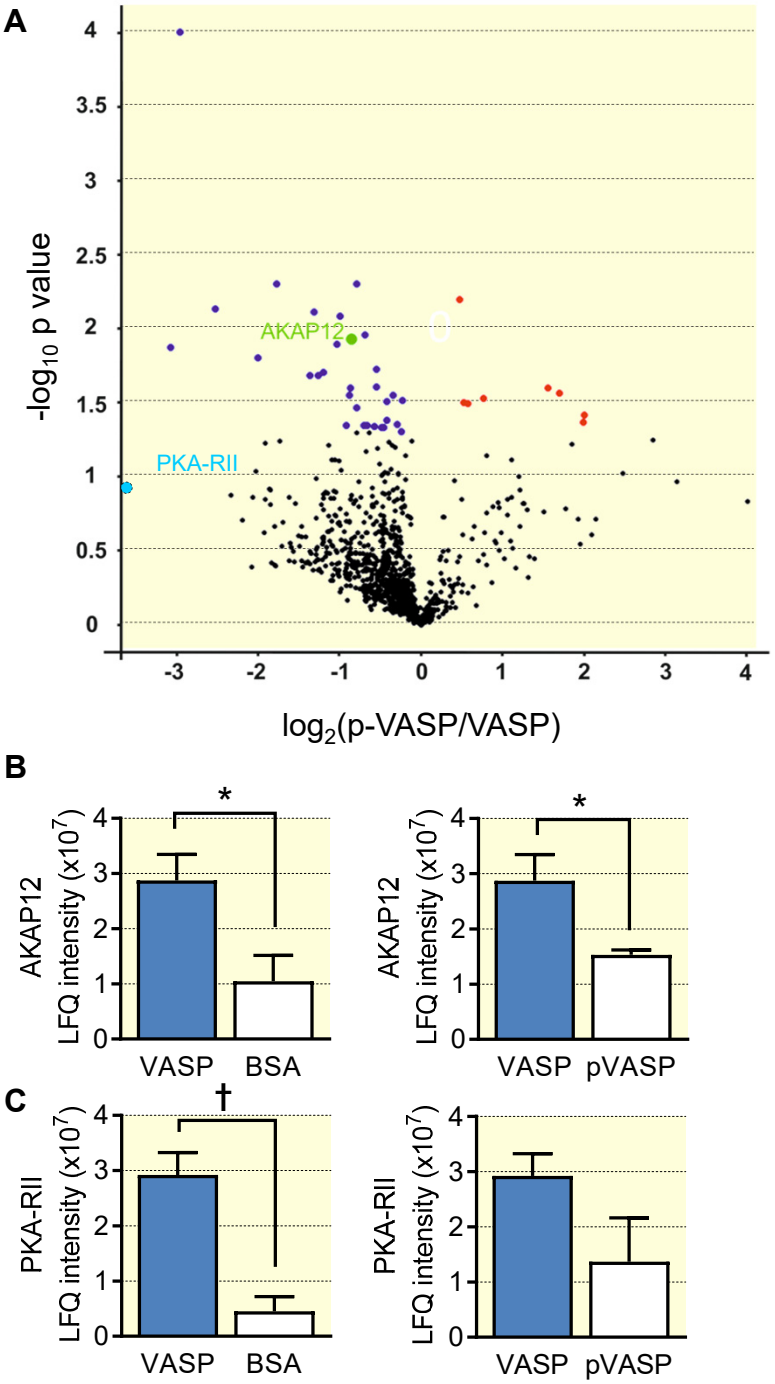

Figure S3. Pull-downs with human endothelial cells reveal VASP/AKAP12/PKA-RII complex formation. VASP, Tyr16/Tyr39-phosphorylated VASP (pVASP) or BSA (control) was used to pull-down proteins from human endothelial cells and precipitated proteins were subjected to mass spectrometry analysis. (**A**) Volcano plot showing proteins significantly enriched (purple) or decreased (red) in VASP pull-downs versus pVASP pull-downs. AKAP12 is highlighted in green (n = 4 independent experiments with 6 different cell batches; $P<0.05$), cAMP-dependent protein kinase type II-alpha regulatory subunit (PKA-RII) is shown in cyan. (**B&C**) Label free quantification (LFQ) intensities of AKAP12 (B) and PKA-RII (C) in pull-downs using VASP, BSA or pVASP as bait (Students's t test). *$P<0.05$, †$P<0.01$.

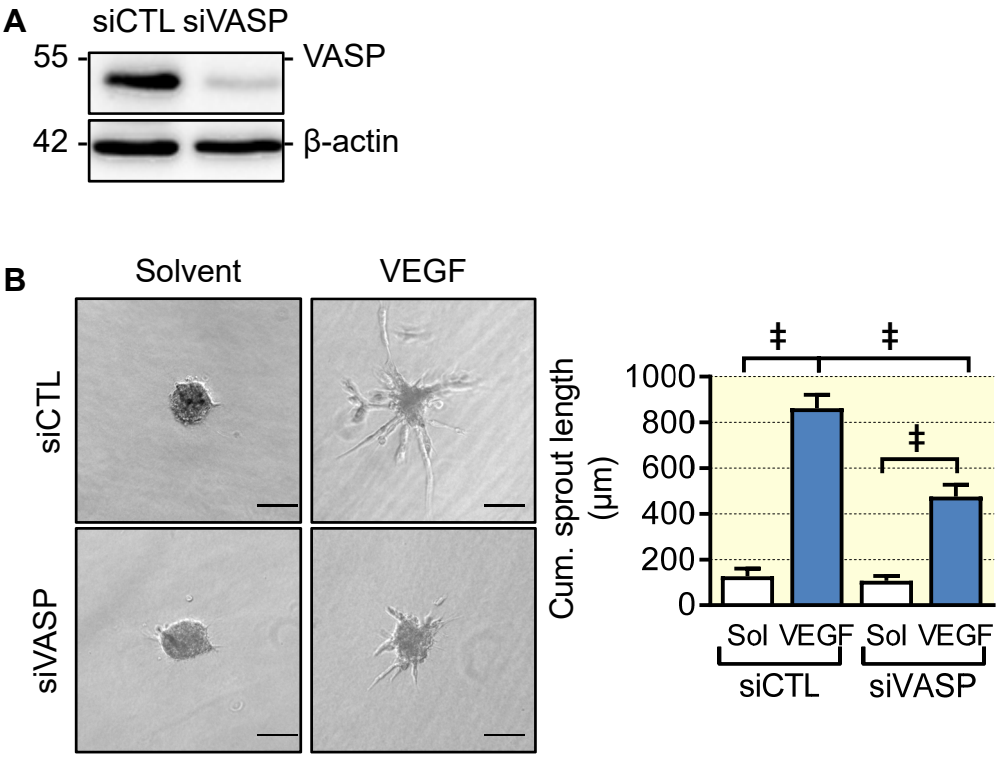

Figure S4. VASP down-regulation impairs VEGF-induced endothelial sprouting in vitro. (**A**) VASP protein expression in human endothelial cells treated with control oligonucleotides (siCTL) or siRNA directed against VASP (siVASP). Comparable results were obtained using 3 additional cell batches. (**B**) Endothelial cell sprouting in a modified spheroid assay with siCTL or siVASP treated primary cultures of human endothelial cells. Experiments were performed in the absence and presence of VEGF (30 ng/ml); bar=100 µm, n=13 different cell preparations (two-way ANOVA with Tukey's test). ‡P<0.001.

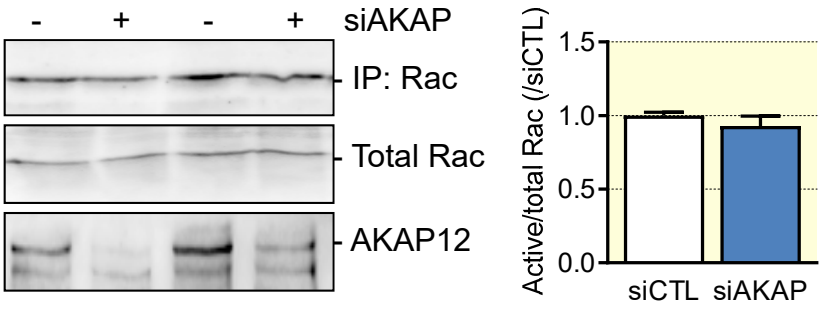

Figure S5. Consequence of AKAP12 downregulation on VEGF-induced Rac activation in subconfluent endothelial cells. Human endothelial cells were treated with siRNA against AKAP12 (siAKAP) or control. After 48 hours cells were stimulated with VEGF (50 ng/mL) for 30 minutes. Active RAC was pulled down using GST-tagged PAK-PBD protein on agarose beads, n=3 different cell batches (Students *t* test).