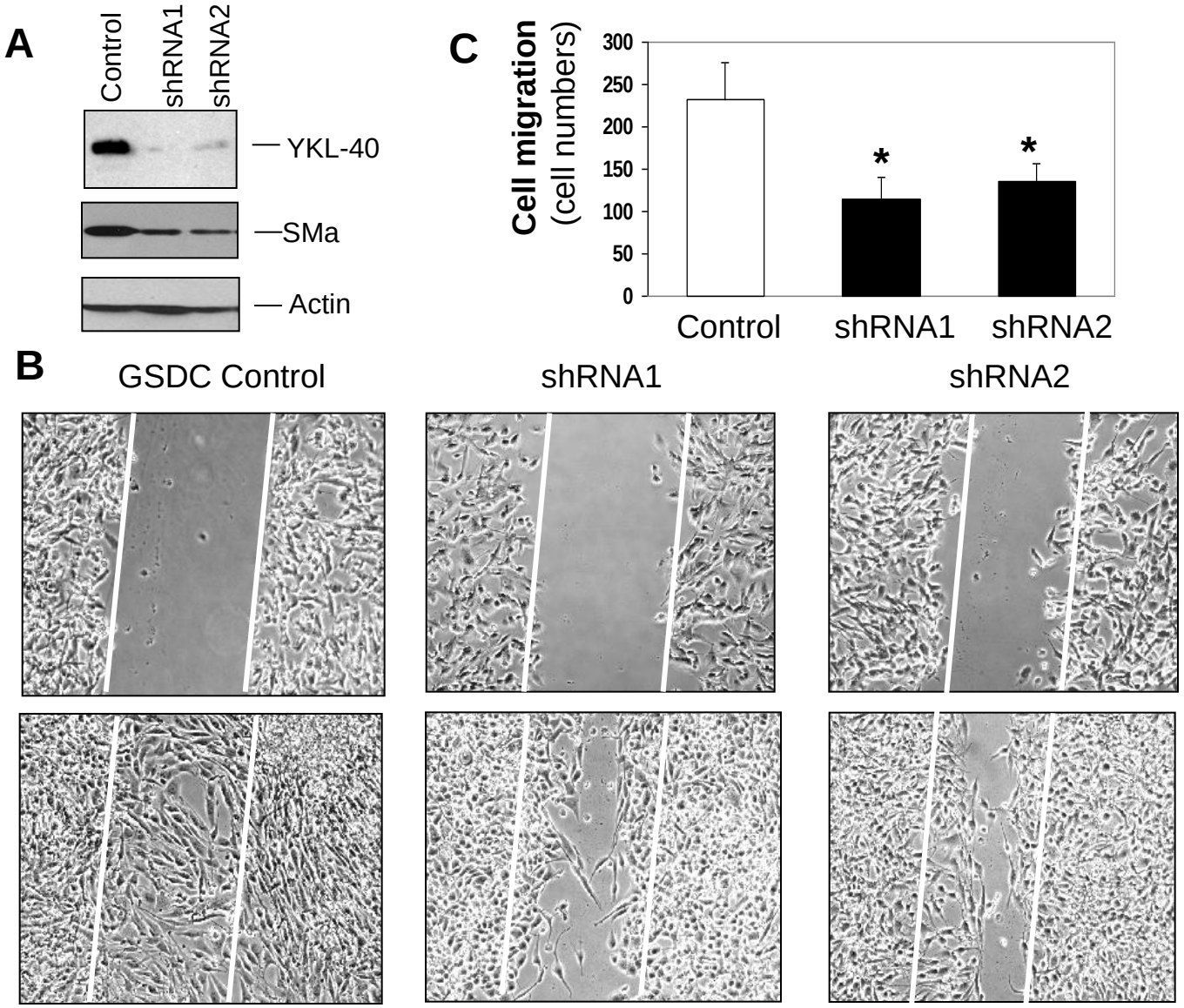
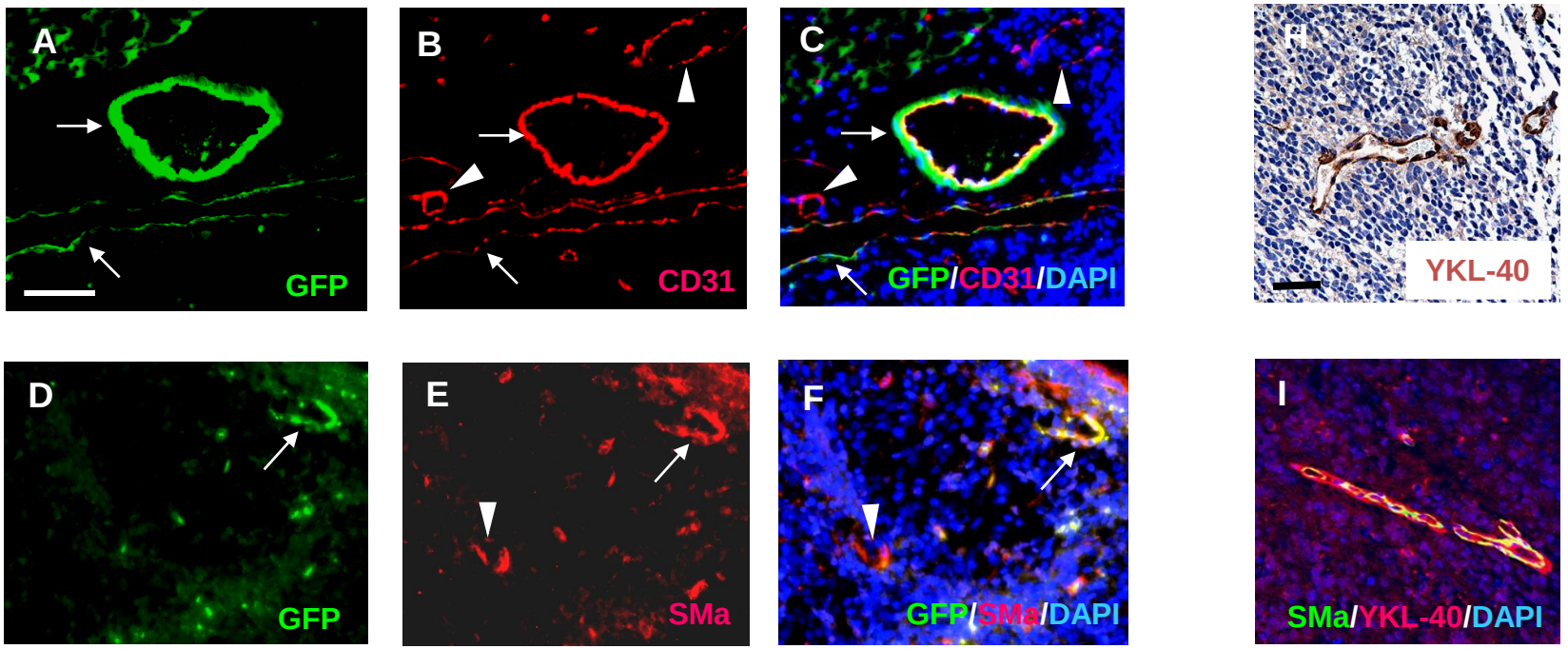
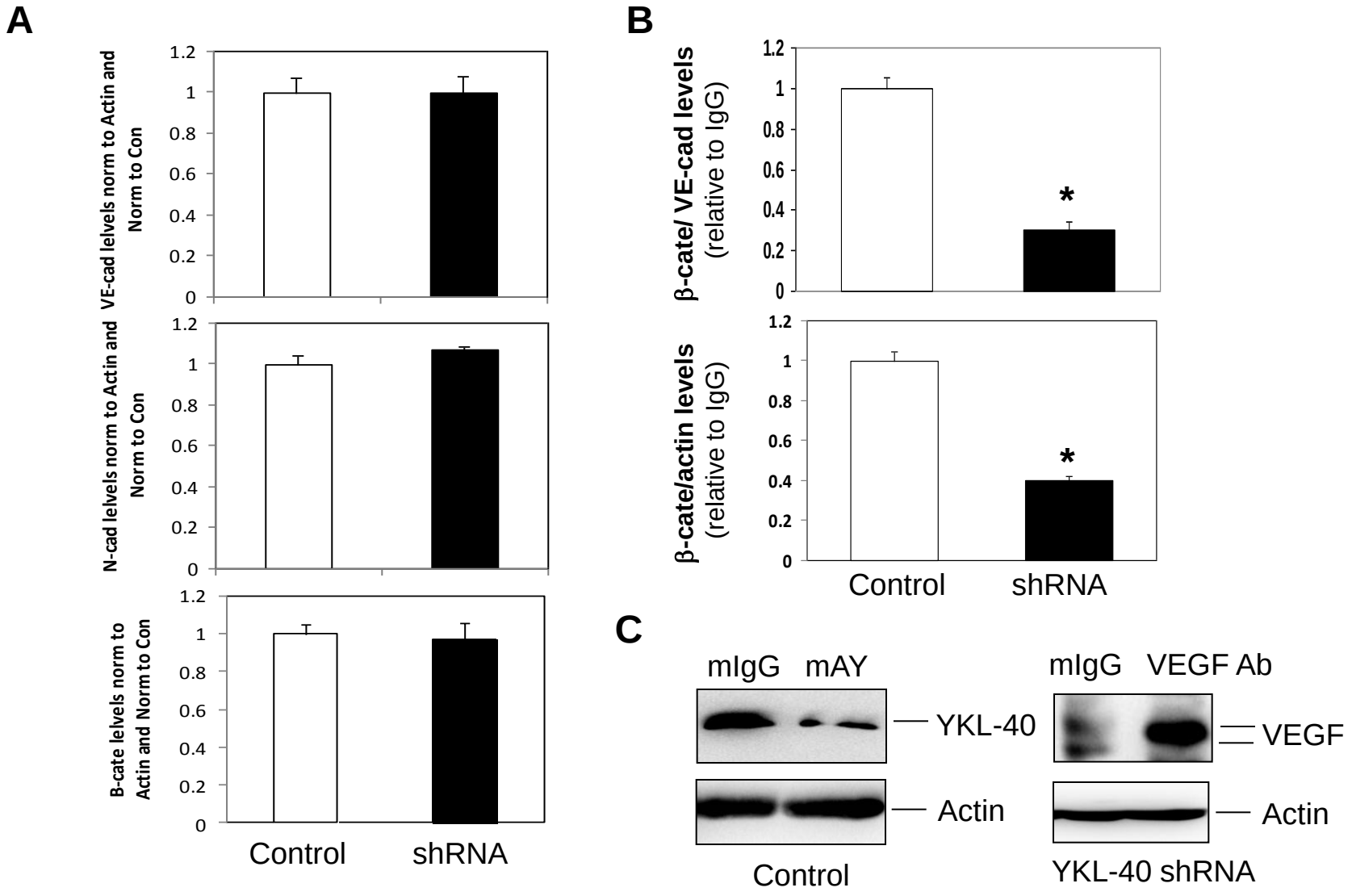


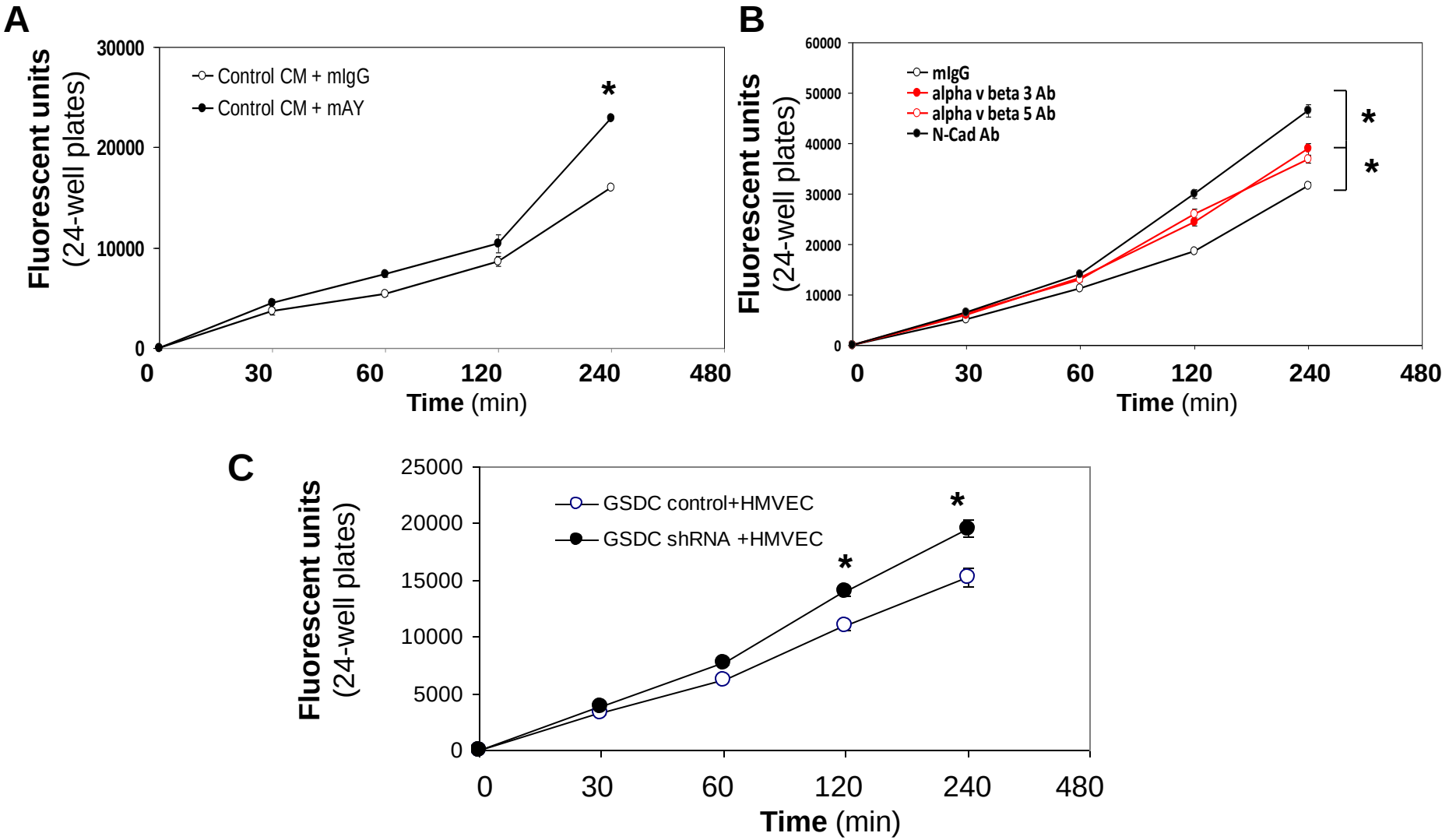
Supplemental Figure 1. *YKL-40 gene knockdown reduces SMa expression and the migratory capacity of GSDCs.* **A.** Western blot of the YKL-40 shRNA and the corresponding decrease in SMa expression. Actin is used as a loading control. **B.** Scratch wound migration assay at time point 0 h (Top panels) and 24 h later (Bottom panels) of the control and shRNA GSDCs. **C.** Quantification of the scratch wound migration assay portrayed in **(B.)**. N=3, * P≤0.05 compared to control.



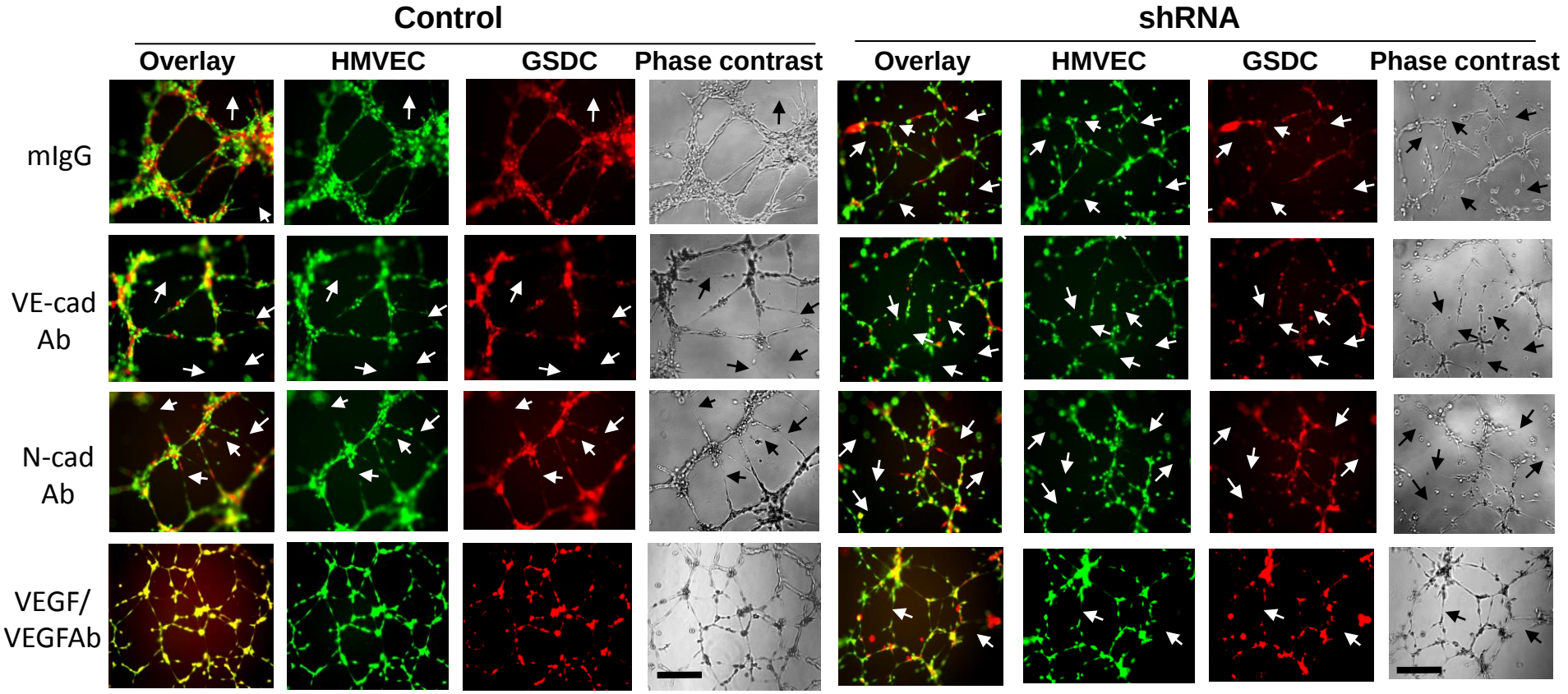
Supplemental Figure 2. *GSDCs cover a large portion of tumor vessels.* Brain tumor sections of SCID/Beige mice injected with GSDCs containing GFP were analyzed for GFP **(A. + D.)**, CD31 **(B)**, GFP + CD31 **(C)**, SMa **(E.)**, or GFP + SMa **(F)**. In **A-C**, most of vessels were lined by GSDCs indicated by arrows and a few vessels were devoid of GSDCs indicated by arrowheads. In **D-F**, co-expression of GFP and SMa surrounding vessel structures was shown by arrows. Mural cell-associated vessels expressing SMa but not GFP were indicated by arrowheads. Tumor tissue from GSDCs was stained for YKL-40 with IHC (H) and for SMa (green) and YKL-40 (yellow) with co-immunofluorescent assay (I). Bars: 100 μm.



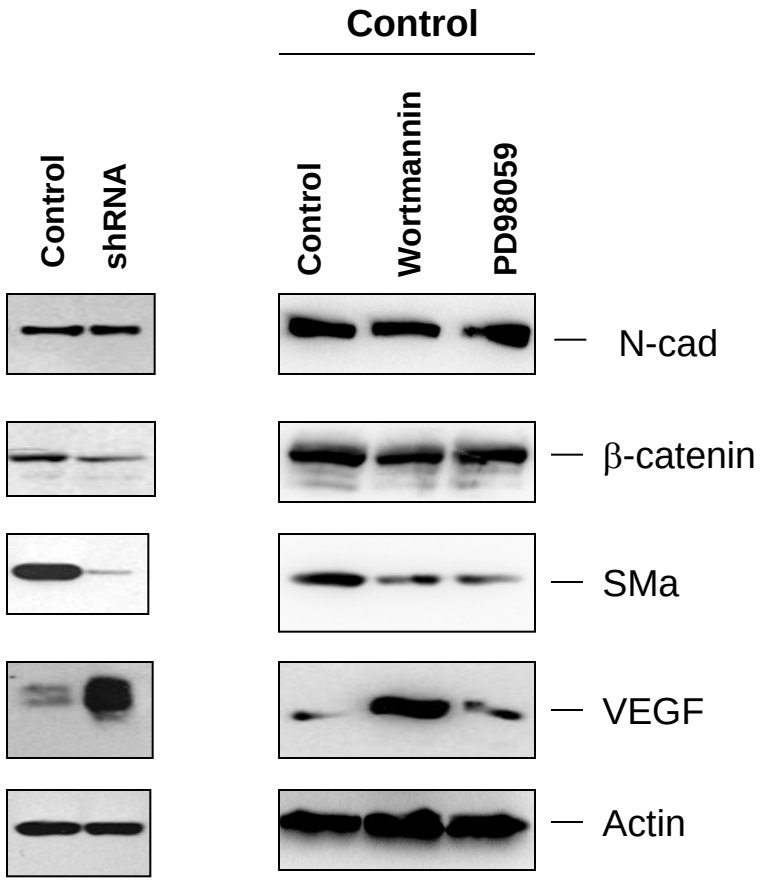
Supplemental Figure 3. *Data quantification and immunoblots*. **A.** Quantified data from immunoblotting of VE-cad, N-cad, and β-cate normalized with actin. **B.** Quantified data from immunoprecipitation and immunoblotting of VE-cad, β-cate, and actin normalized with IgG. N=2-4. *P<0.05 compared with controls. **C.** Control GSDCs and YKL-40 shRNA GSDCs were treated with mAY and a VEGF antibody, respectively, overnight. The conditioned media were used for immunoblotting against YKL-40 and VEGF.



Supplemental Figure 4. *Effects of mAY, YKL-40 shRNA, and integrin antibodies on cell permeability.* **A.** HMVECs were plated in inserts and treated with conditioned medium (CM) of control GSDCs in the presence of mIgG or mAY (10 μg/ml) overnight. Cell permeability was measured. **B.** Control GSDCs were plated in inserts in the presence of anti-N-cadherin (50 μg/ml), integrin αvβ3, or αvβ5 antibody (10 μg/ml). Then, cell permeability was determined. **C.** GSDC control or YKL-40 shRNA cells were plated for 2 hr followed by loading HMVECs on the top of the GSDCs. Cell permeability was determined on the next day. N=6, *P<0.05 compared with corresponding controls.



Supplemental Figure 5. *Inhibition of VE-cadherin or N-cadherin activity leads to decreases in vascular stability.* HMVECs and either control or YKL-40 shRNA GSDCs were pre-labeled with Calcein AM (green) and Calcein red, respectively, and plated together on Matrigel in the presence of an anti-VE-cadherin (20 μg/ml), anti-N-cadherin (50 μg/ml), or anti-VEGF antibody (100 ng/ml). 24 hr following incubation, tubule images with fluorescence were analyzed. White arrows demonstrated breaks in the tube networks, while black arrows on the phase contrast images depicted gaps in the corresponding networks. Note recombinant VEGF (10 ng/ml) in control and an anti-VEGF Ab in shRNA. Bars: 100 μm.



Supplemental Figure 6. ***PI3K and MAPK signaling pathways mediate expression of SMa and VEGF in GSDCs***. GSDCs were treated with PI3K inhibitor Wortmannin (10 nM) and MAPK inhibitor PD98059 (10 μM) overnight. Cell lysates and media were collected to test N-cad, β-catenin, SMa, and VEGF in immunoblotting. Actin was a control.