Supplementary Figure Legends

Supplementary figure 1: Disruption of FGF signaling in cultured endothelial cells reduces junctional VE-cadherin localization without changing cell density

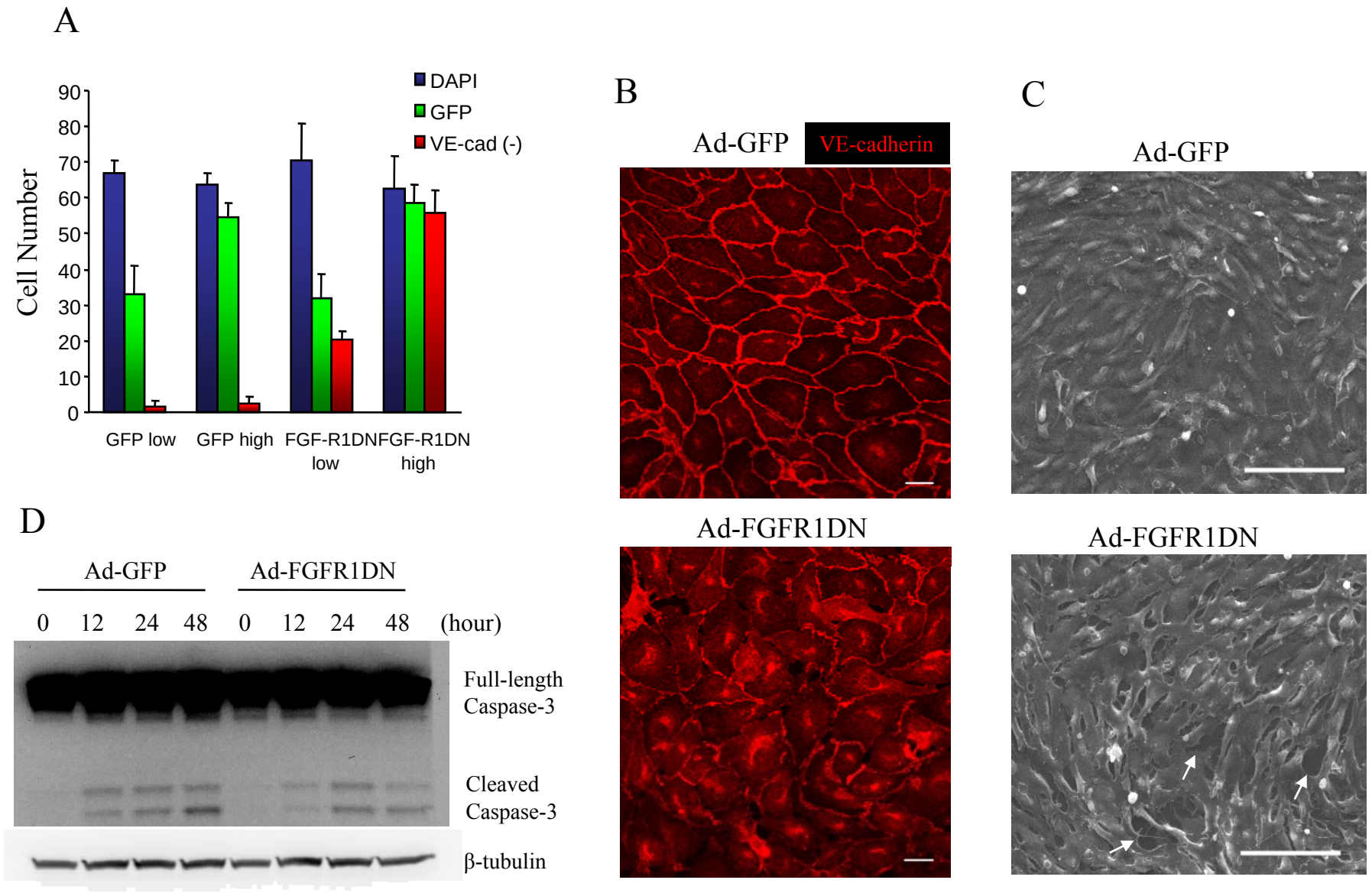
A. Quantitative analysis of VE-cadherin staining. Quiescent BAEC were transduced with Ad-GFP or Ad-FGFR1DN-GFP adenovirus using low and high MOI (25 and 50 pfu/cell for Ad-GFP and 50 and 100 for Ad-FGFR1-GFP, respectively). Number of DAPI-positive nucleai (blue), GFP-positive cell (green), and cells with discontinuous, interrupted VE-cadherin staining (red) were counted and shown in the graph. Junctional VE-cadherin staining is disturbed as transduction efficiency increases in Ad-FGFR1DN treated cells while overall cell number is unchanged between treatment groups. Data are shown as mean±SD for 4 different images in each group. **B**. Increased VE-cadherin cytoplasmic staining in FGFR1DN treated EC. Quiescent BAEC were treated with a high dose of Ad-GFP or Ad-FGFR1DN (MOI=100 pfu/cell) and VE-cadherin staining was performed 24 hours after transduction. In FGFR1DN cells, VE-cadherin has disappeared from junctions and redistributed in the cytoplasm. Bar, 20μm. **C.** Scanning electron microscopy (SEM) images of quiescent EC monolayer after adenovirus transduction. Quiescent BAEC were treated with Ad-GFP or Ad-FGFR1DN (MOI=100 pfu/ml) and scanning microscopy was performed 24 hours after transduction. Arrows indicate gaps between endothelial cells. Note overall cell density is not altered between the treatments. Bar, 100μm. **D.** FGF signaling inhibition does not enhance apoptosis in endothelial cells. Quiescent BAEC were treated with Ad-GFP or Ad-FGFR1DN (MOI=100 pfu/ml) and harvested at indicated time points. Total cell lysates were subjected to Western blot analysis using caspase-3 and β-tubulin

antibodies. The caspase-3 antibody detects full-length caspase-3 (35 kD, upper band) and activated fragments (cleaved caspase-3, 17/19 kD, lower bands).

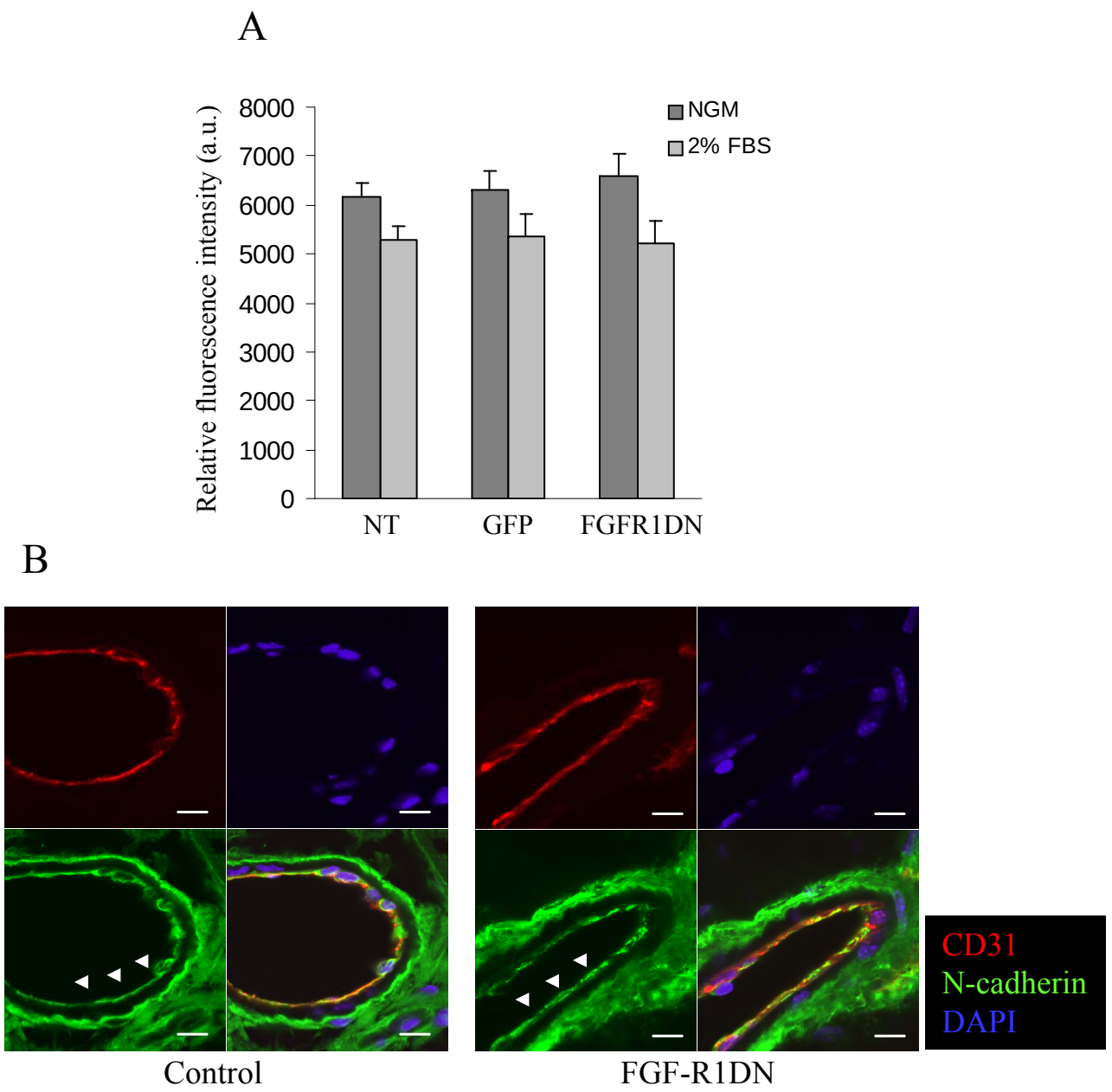
Supplementary Figure 2: N-cadherin function and distribution is not altered by disruption of FGF signaling

A. EC adhesion to SMC is not impaired by disrupting endothelial FGF signaling. BAEC was transduced with control or FGFR1DN adenovirus and stained with calsein AM (5 μg/ml). Thereafter ECs are detached with cell dissociation solution and plated on a 96 well-plate (10^4 cells) that had been seeded with BSMC two days prior and formed confluent SMC monolayers. After 30 min incubation with either normal growth medium (EGM-2 MV) or EBM-2, 2% FBS, unattached cells were washed with PBS and fluorescent measurement were performed.

B. N-cadherin staining is not altered in the vasculature of sFGFR1IIIc mouse. After 10 days of adenovirus injection, gastrocnemius muscle samples were immunostained with N-cadherin (green), CD31 (red) and DAPI (blue). N-cadherin staining that represents EC-SMC interface is indicated by arrowheads. Bar, 10μm.



Supplementary figure 1



Supplementary figure 2