Increasing TRPV4 expression restores flow-induced dilation impaired in mesenteric arteries with aging

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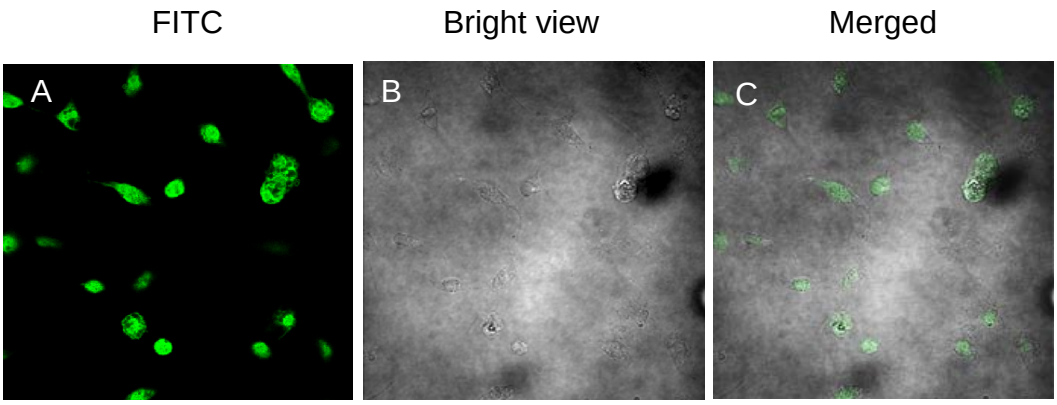
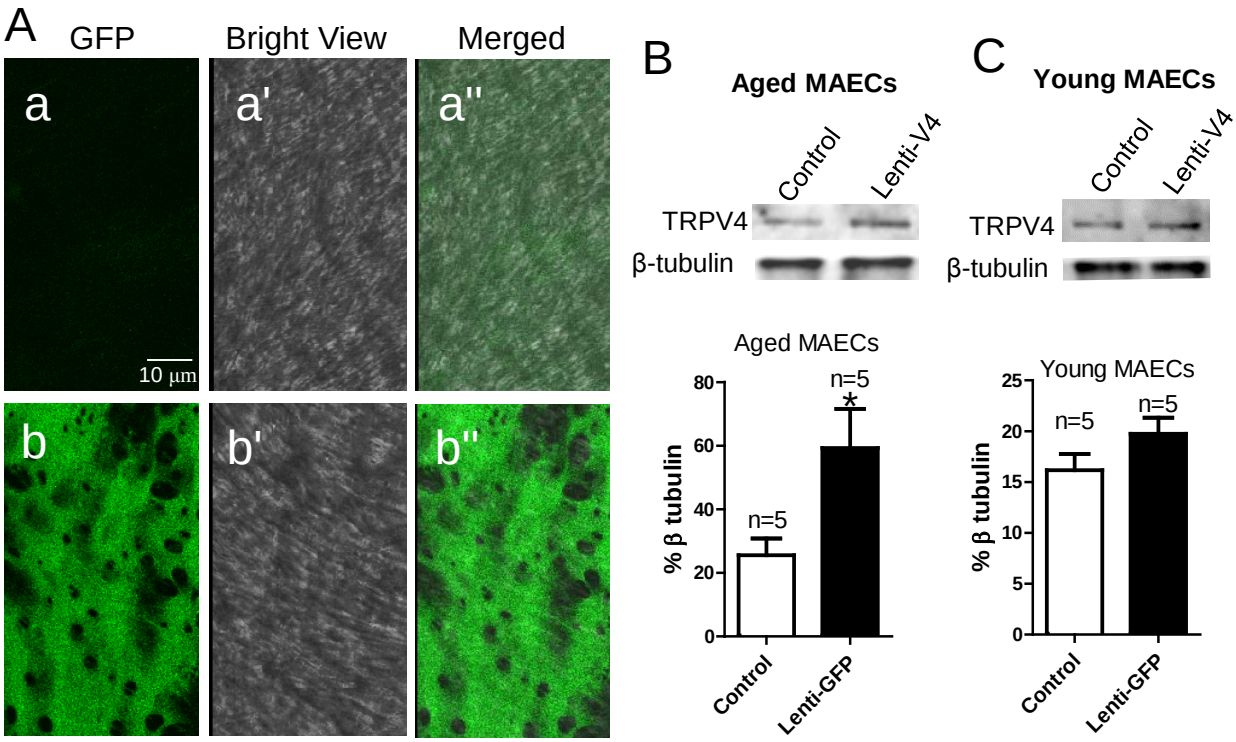
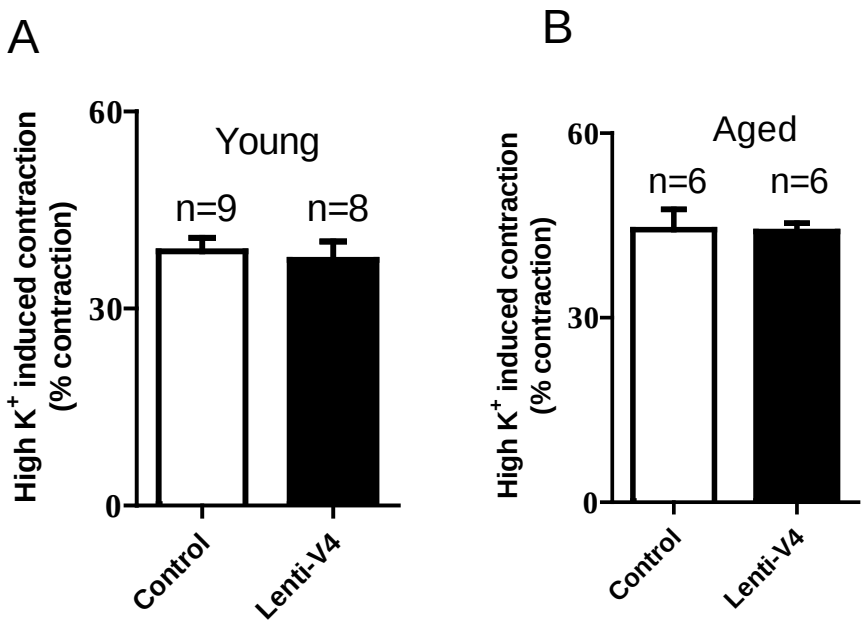


Supplementary Figure 1. Representative images showing immunostaining of cultured primary mesenteric artery endothelial cells using an antibody against von Willebrand factor.



Supplementary Figure 2. Effect of lenti-GFP and lenti-TRPV4 transfection in the expression of green fluorescent protein (GFP) in mesenteric artery endothelium and TRPV4 protein in primary cultured mesenteric artery endothelial cells (MAECs) from young and aged rats. A. Representative images showing the signal of green fluorescence indicating GFP in the endothelial layer of mesenteric artery from young rat. **a**-**a''**. Lenti-empty vector infection. **b**-**b''**. Lenti-GFP infection. **B** and **C.** Shown are representative images (upper) and data summary (lower) of immunoblotting experiments for TRPV4 in lenti-TRPV4 transfected young and aged MAECs. Proteins were extracted from primary cultured MAECs lysates after 72 h of virus infection. Immunoblots with anti-β-Tubulin antibody showed that equal amounts of protein were loaded onto each lane. Values are mean ± SE (n = 5). *P < 0.05, lenti-GFP (Control) vs. lenti-TRPV4 (Lenti-V4) viral vector-treated MAECs.



Supplementary Figure 3. High K^+ solution-induced mesenteric artery contraction. A and **B**. Summarized data showing the peak value of high K^+ solution-induced contraction. Values are mean ± SE (n = 6-9). *P < 0.05, lenti-GFP (Control) vs. lenti-TRPV4 (Lenti-V4) viral vector-treated vessels.