Figure I

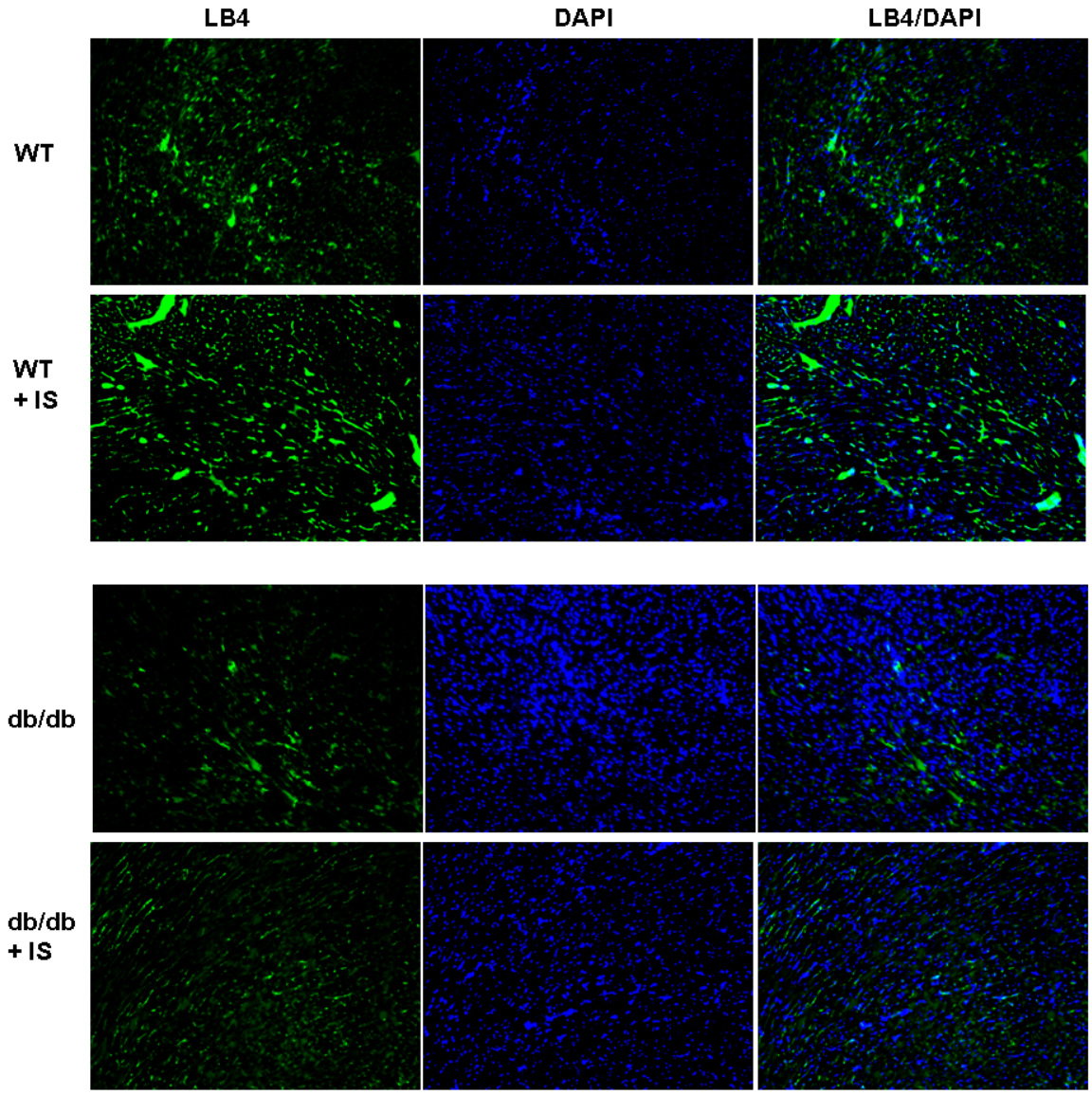

Figure I. Representative images showing that in WT mice, myocardial ischemia for 14 days significantly increased myocardial capillary density, whereas myocardial capillary density was markedly impaired in db/db mice. Endothelial cells in the remote zone of myocardial infarctions were stained with LB4 (green, 10X) and nuclei by DAPI counterstaining (blue, 10x).

Figure II

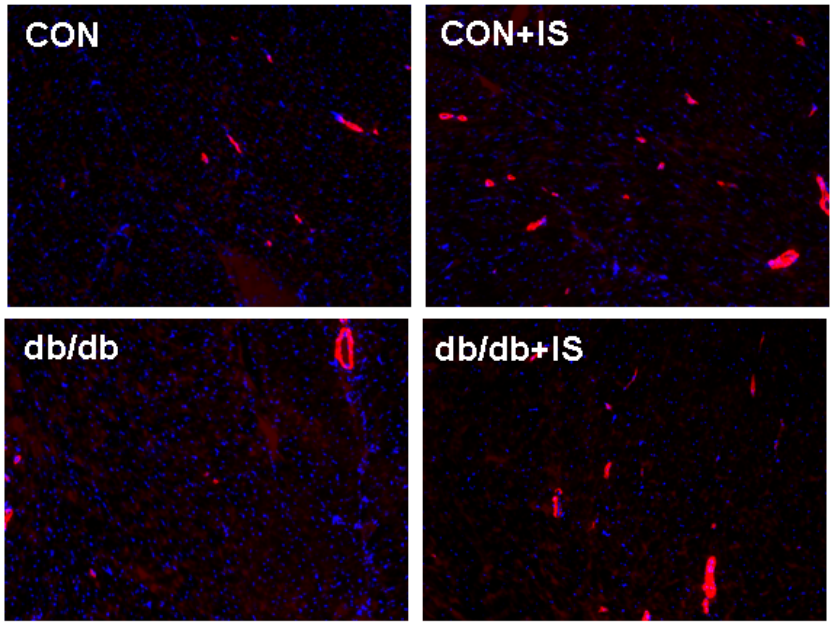

Figure II. Representative images showing that in WT mice, but not in diabetic db/db mice, myocardial ischemia significantly increased myocardial arteriole formation. SMC in the remote zone of myocardial infarctions was stained with smooth muscle actin (Red, 10X) and nuclei by DAPI counterstaining (blue, 10x).

Figure III

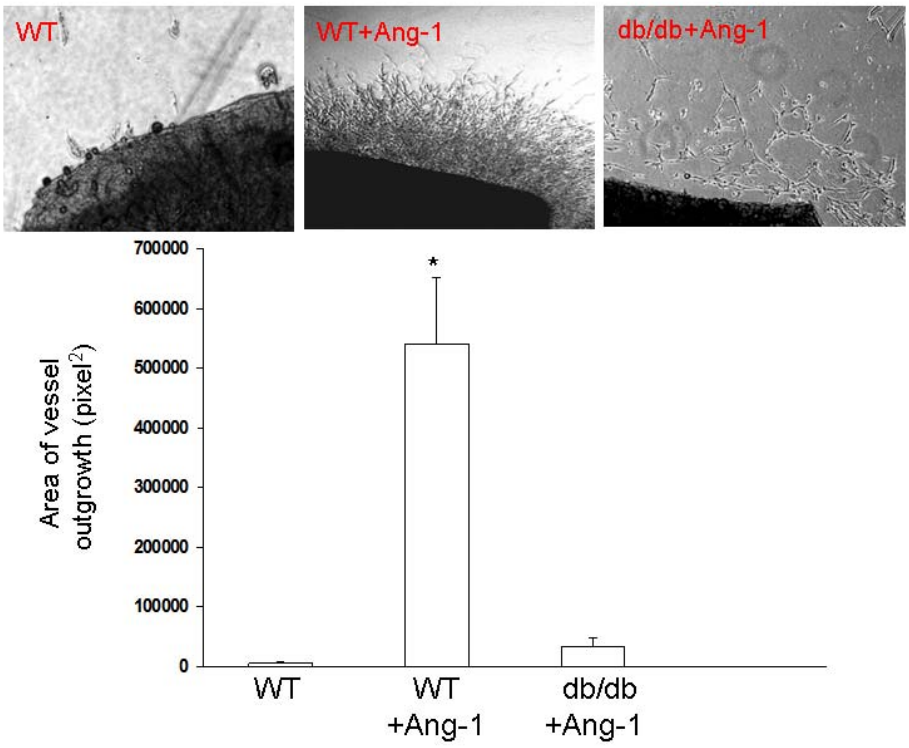

Figure III. Representative images of vessel outgrowth and quantitative analysis of vessel outgrowth area in WT or db/db mice after being stimulated with Ang-1 for 5 days. Quantitative analysis area of vessel outgrowth revealing that stimulation of aortic rings isolated from WT mice with Ang-1 (250ng/ml) for 5 days led to a significant increase in vessel outgrowth (4X), whereas Ang-1-induced vessel outgrowth was dramatically attenuated in aortic rings isolated from db/db mice. (n=8 mice, *p<0.05).

Figure IV

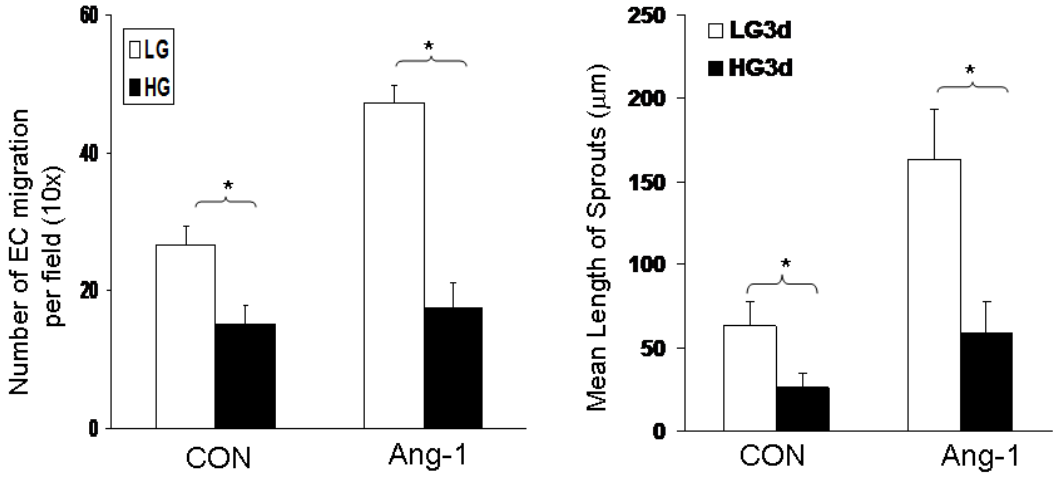

Figure IV. Left panel: Basal MHMEC migration capacity and Ang-1-induced endothelial cell migration were significantly decreased under HG conditions (n=4, * p< 0.05).

Right panel: Quantitative analysis of the length of sprouts from endothelial cell spheroids. Exposure of MHMEC to HG resulted in a significant decrease in Ang-1-stimulated endothelial cell sprout length (n=4, * p< 0.05).

Figure V

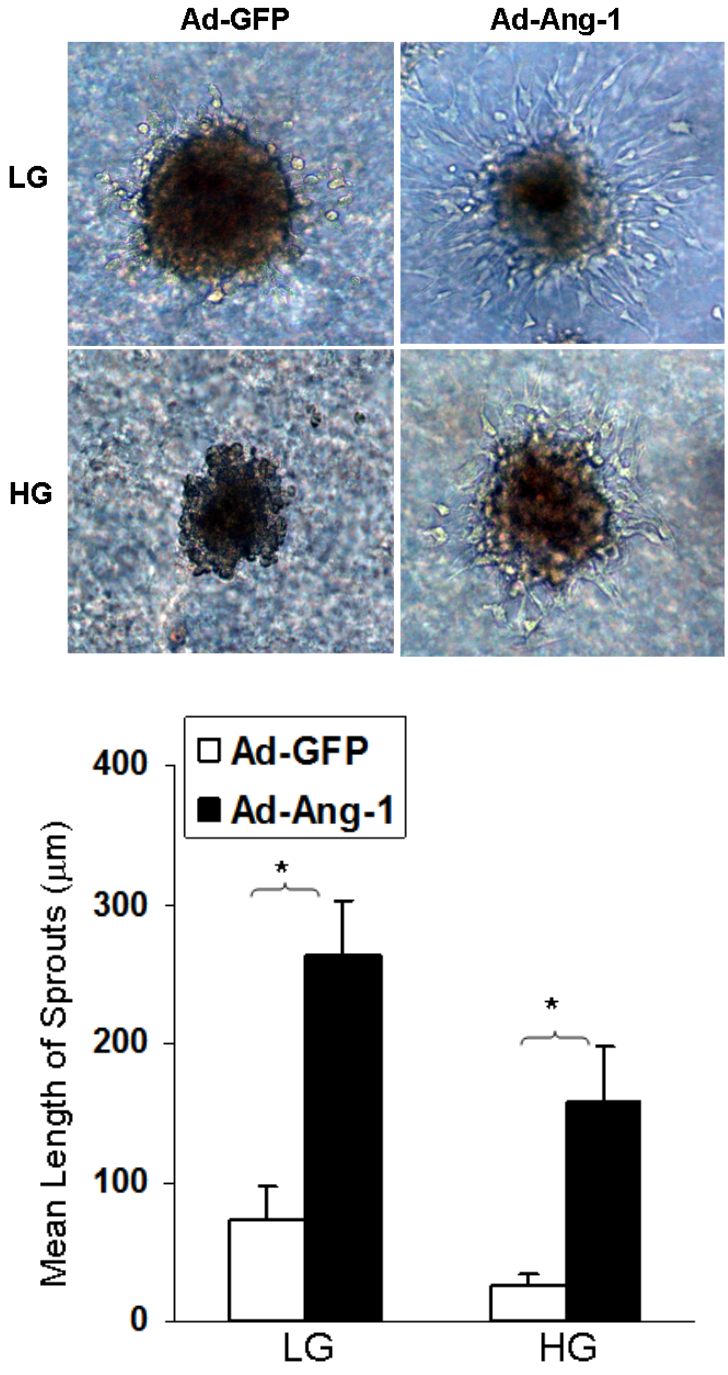

Figure V. Representative images and quantitative analysis of the length of sprouts from endothelial cell spheroids. Overexpression of Ang-1 in MHMEC significantly increased endothelial cell sprouting under both LG and HG conditions (n=4, * $p<0.05$).