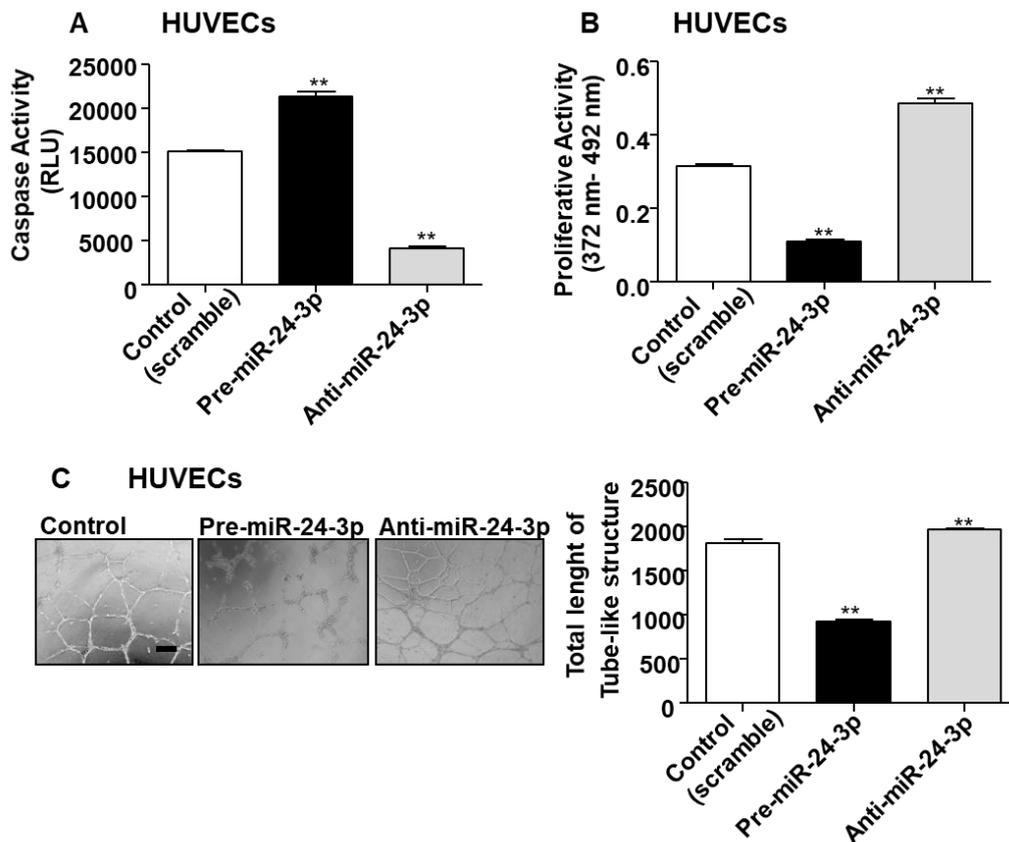
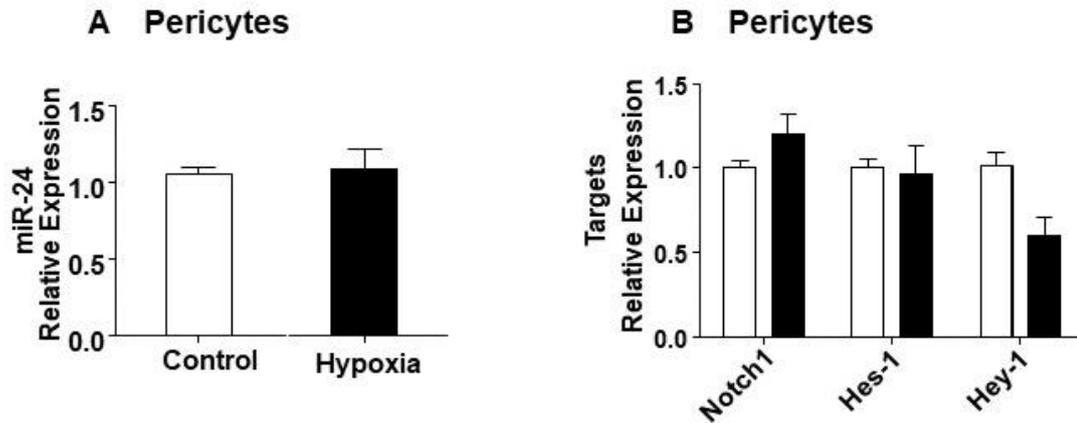


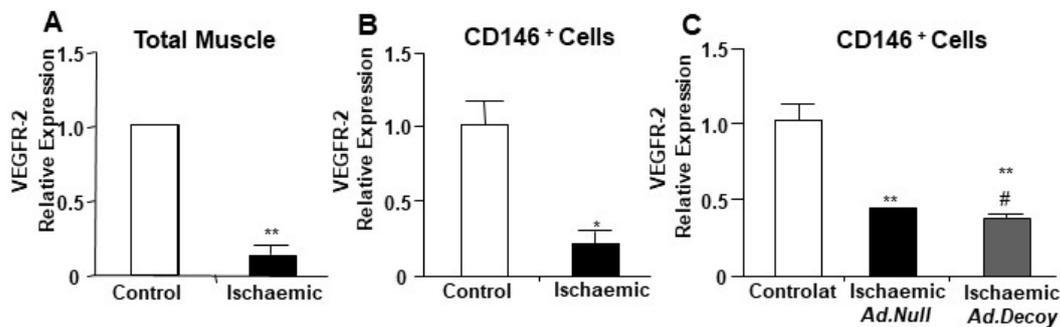
**Supplementary Figure S1:** In vitro miR-24-3p expressional modulation. miR-24-3p expression changes after 72 h post-transfection with either pre-miR-24-3p (miR-24-3p, black bar) or anti-miR-24-3p (miR-24-3p light grey column) compared to the control (scramble, white column). miR-24-3p expression was normalised to Snu6. Experiments were performed in triplicate and repeated three times. Values are means  $\pm$  SEM. \*\*  $p < 0.01$  vs. control.



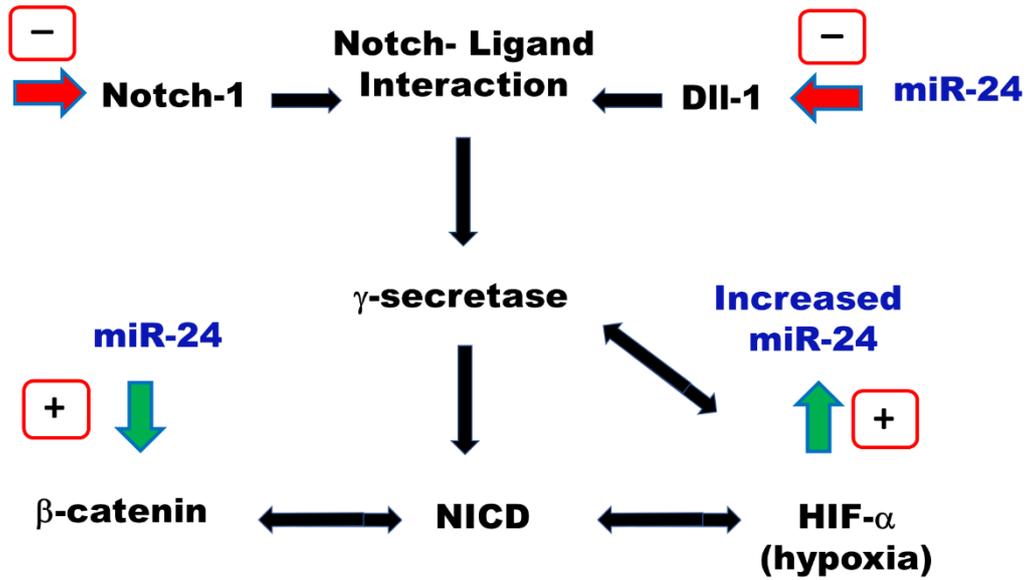
**Supplementary Figure S2.** Changes in the apoptosis, proliferation, and cord formation capacity induced by transfection of HUVECs with either pre-miR-24-3p (black bars) or anti-miR-24-3p (light grey bars) in comparison to a scramble control (white bars). (A) Cell apoptosis was evaluated by Caspase-GLO activity assay. RLU: relative luminescent units. (B) the proliferative capability of HUVECs evaluated by a BrdU incorporation assay (C) Photomicrographs of representative fields show the endothelial network capability formation on Matrigel (scale bar 40  $\mu$ m). Experiments were performed in triplicate and repeated three times. Values are means  $\pm$  SEM. \*\* $p < 0.01$  vs. scramble.



**Supplementary Figure S3:** Pericytes do not change miR-24-3p or Notch1 expression when exposed to hypoxia. Bar graph (A) shows miR-24-3p modulation at 48 h in hypoxia (black column) or normoxia (white column). Bar graph (B) shows Notch1 mRNA and Dll1 mRNA modulation at 48 h in hypoxia (black columns) or normoxia (white columns). miR-24-3p expression was corrected to Snu6, and data are compared to the matched time-point group by the  $2\Delta\text{Ct}$  method. The mRNA expression of Notch1, Hes-1, and Hey-1 was corrected to 18S. Experiments were performed in triplicate and repeated three times. Values are means  $\pm$  SEM.



**Supplementary Figure S4.** VEGFR-2 relative expression at 3 days of L.I. in a murine model. VEGFR-2 relative expression in total ischemic muscle (black bar) compared to the control non-ischemic muscle (white bar) (A). Modulation of VEGFR-2 in ECs sorted from ischemic muscle (black bar) compared to the ECs sorted cells from control non-ischemic muscle (white bar) (B). Modulation on VEGFR-2 in ECs after *Ad. Decoy-miR-24-3p* treatment (grey bar) compared to *Ad. Null* treatment (black bar) and contralateral muscle (control, white bar) (C). VEGFR-2 expression, corrected to 18S, is significantly reduced compared to the control. Experiments were performed in triplicate and repeated five times. Values are means  $\pm$  SEM. \*  $p < 0,05$ ; \*\*  $p < 0,01$  vs. control.



**Supplementary Figure S5.** Proposed model of the impact of miR-24-3p on the interaction between Notch and  $\beta$ -catenin in hypoxic endothelial cells. NICD interacts with  $\beta$ -catenin and HIF-1 $\alpha$ . Moreover, HIF-1 $\alpha$  increases the  $\gamma$ -secretase activity resulting in enhanced Notch signalling. miR-24-3p is upregulated under hypoxic conditions and can directly repress Notch-1 and DII-1, while it can indirectly increase  $\beta$ -catenin. Green indicates expressional promotion and red expressional suppression. Double arrows indicate physical interaction.