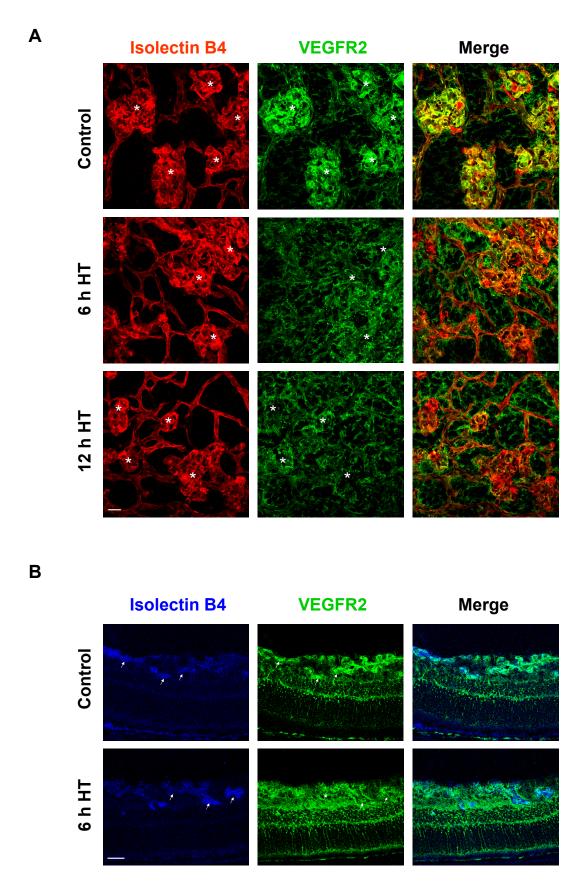
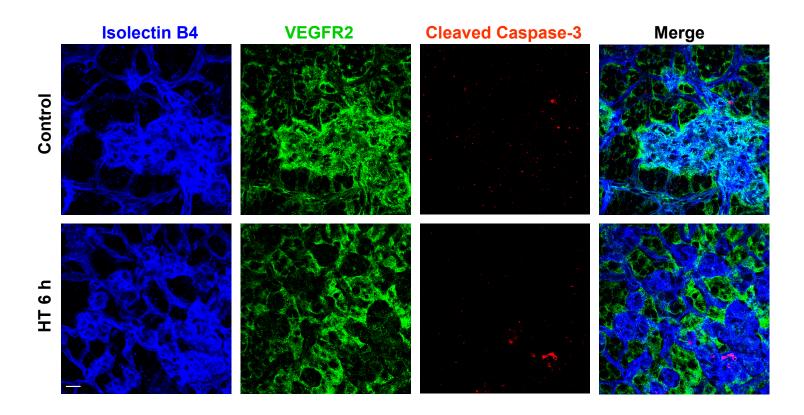


Supplemental Figure S1. Hyperoxia-induced regression of NV tufts is associated with the appearance of cleaved caspase-3 in pyknotic nuclei. OIR mice were treated with hyperoxia (75% oxygen, HT) for 12 hr at P17. Retinal flat-mounts were stained with isolectin B4 (red) to highlight the retinal vessels, anti-cleaved caspase-3 (green) for active caspase-3, and DAPI (blue) for nuclei. Representative confocal images of NV tufts are shown (630X). Bar, 20 µm.



Supplemental Figure S2. Hyperoxia treatment specifically down-regulates VEGFR2 in NV. (A) OIR mice were treated with hyperoxia (75% oxygen, HT) for 6 and 12 hr at P17. Retinal flat-mounts were stained with isolectin B4 (red) and anti-VEGFR2 (green). Representative confocal images of NV tufts (marked with white asterisk) in peripheral retina are shown (400X, n=4 mice). Bar, 20 μ m. (B) OIR mice were treated with hyperoxia (75% oxygen, HT) for 6 hr at P17. Retina frozen sections were stained with anti-VEGFR2 (green) and isolectin B4 (blue). Representative fluorescence microscopy images are shown (200X, n=6 mice). Arrows show examples of NV in retina sections. Bar, 50 μ m.



Supplemental Figure S3. Hyperoxia treatment induces VEGFR2 down-regulation prior to NV tuft apoptosis. OIR mice were treated with hyperoxia (75% oxygen, HT) for 6 hr at P17 and retinal flat-mounts were stained with isolectin B4 (blue), VEGFR2 (green) and cleaved caspase-3 (red). Representative confocal images of NV tufts are shown (400X, n=3 mice). Bar, 20 μ m.