**Characterization of a murine model of endothelial dysfunction induced by chronic intraperitoneal administration of angiotensin II**

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**Supplementary Figure S1. Chronic AGII administration increases the expression of eNOS mRNA but not iNOS mRNA.** Data are reported as mean ± SD and analyzed with a two-tailed, unpaired Student's t test (P < 0.05). \* indicate differences with respect to control.

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**Supplementary Figure S2. Evaluation of glomerular damage by PAS staining**. A-B) Renal capsule. C-D) Glomeruli. E-G) Analysis of glomerular area and percentage of vascular y mesangial glomerular areas. A decrease in glomerular vascular and a mesangial expansion were observed in AGII-treated mice (F-G). Arrows indicate vascular areas. M: macrophages; L: lymphocytes; P: plasmatic cells; F: fibroblasts, B: basophiles; N: neutrophils. Microphotographs taken at 100X. Data are reported as mean ± SD and analyzed with a two-tailed, unpaired Student's t test (P < 0.05). \* indicate differences with respect to control.

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**Supplementary Figure S3. Retinal neovascularization induced by AGII.** A-B) Vessels. C-D) Neovessels. A chronic AGII administration failed to alter the number of retinal vessels already formed; however, it induced the formation of new vessels, increasing its number 4-fold with respect to the control group (D). Data are reported as mean ± SD and analyzed with a two-tailed, unpaired Student's t test (P < 0.05). \* indicate differences with respect to control.