Long-Term Angiotensin II Receptor Blockade Limits Hypertension, Aortic Dysfunction and Structural Remodelling in a Rat Model of Chronic Kidney Disease

Supplementary Figures

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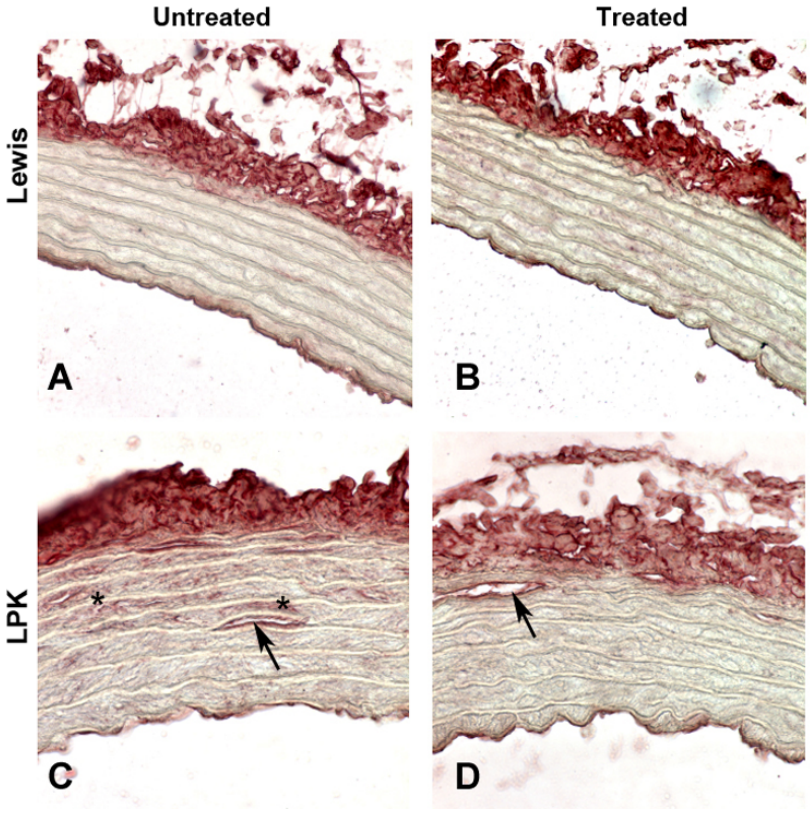
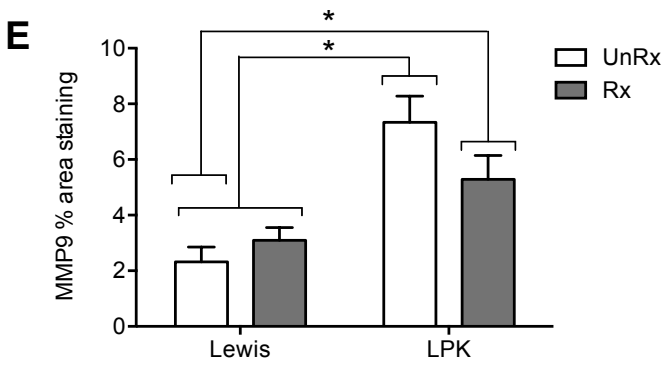
Supplementary Figures

Supplementary Figure 1: MMP9

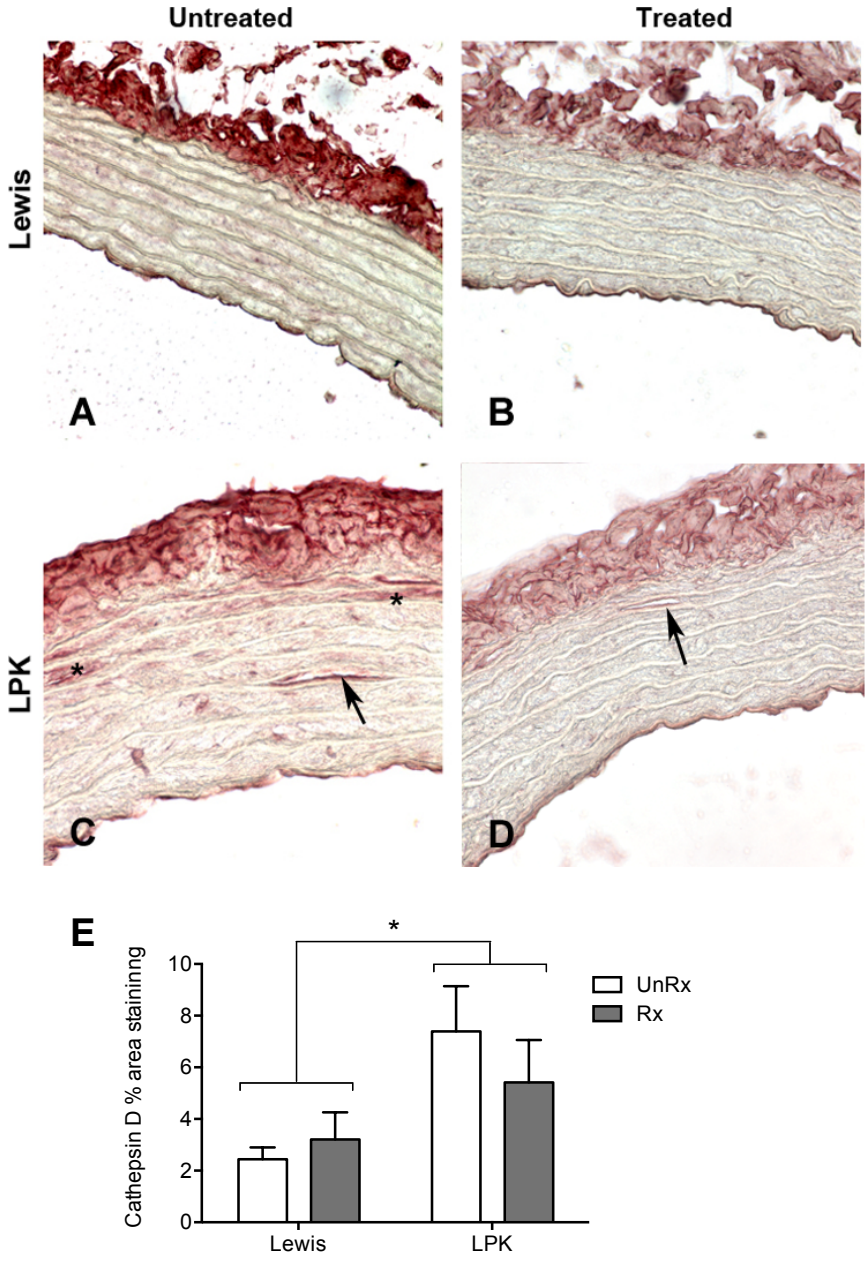
Immunohistochemical staining for matrix metalloproteinase 9 (MMP9) in the aortic wall of Lewis (A, B) and Lewis polycystic kidney (LPK; C, D) rats. Images are from untreated (A, C) and treated animals (valsartan; panels B, D). Media cysts are evident in the vessel wall (arrows). MMP9 expression was evident in the adventitia, in the media (*) and in association with cysts. When assessed as % area of media staining, there was a significant strain effect (P<0.001) with MMP9 staining greater in the LPK compared to Lewis (Lewis: 2.7±1.3 vs. LPK 6.2±2.3 % area, P<0.001, n=15 & 11 animals, respectively). There was also a significant treatment*strain interaction (P=0.033) which was due to a decrease in MMP9 staining in the LPK group after valsartan treatment, such that it was no longer significantly different to the Lewis valsartan treated group (p=0.158). Panel E: *P<0.05 between LPK and Lewis groups as indicated.





Supplementary Figure 2: Cathepsin D

Immunohistochemical staining for Cathepsin D in the aortic wall of Lewis (A, B) and Lewis polycystic kidney (LPK; C, D) rats. Images are from untreated (A, C) and treated animals (valsartan; panels B, D). Cathepsin D expression was also evident in the adventitia, in the media (*) and in association with cysts (arrows). When assessed as % area of media staining, there was a significant strain effect, being greater overall in the LPK (Lewis 2.8±1.9 % area vs. LPK: 6.3±3.9; P=0.027, n= 13 & 11 animals, respectively). There was no significant treatment effect or treatment*strain interaction. Panel E: *P<0.05 between LPK and Lewis groups as indicated.



Supplementary Figure 3: Nuclear Factor Kappa B

Immunohistochemical staining for nuclear factor kappa B (NFκB) in the aortic wall of Lewis (A, B) and Lewis polycystic kidney (LPK; C, D) rats. Images are from untreated (A, C) and treated animals (valsartan; panels B, D). NFκB staining was found in the cell deposits on the intima, colocalizing with the inflammatory cells on the endothelium (*) and was also present in the adventitia in both strains. Expression was noted around cysts in the vessel wall (arrows). There was a significant difference between Lewis and LPK when assessed as % area of the media immunoreactive for NFκB (Lewis 2.3 ± 1.4% area vs. LPK: 6.7 ± 2.9, n = 13 & 10 animals respectively $P< 0.001$. There was a trend towards a significant treatment*strain interaction but this was not significant (P=0.064). Panel E: *P<0.05 between LPK and Lewis groups as indicated.