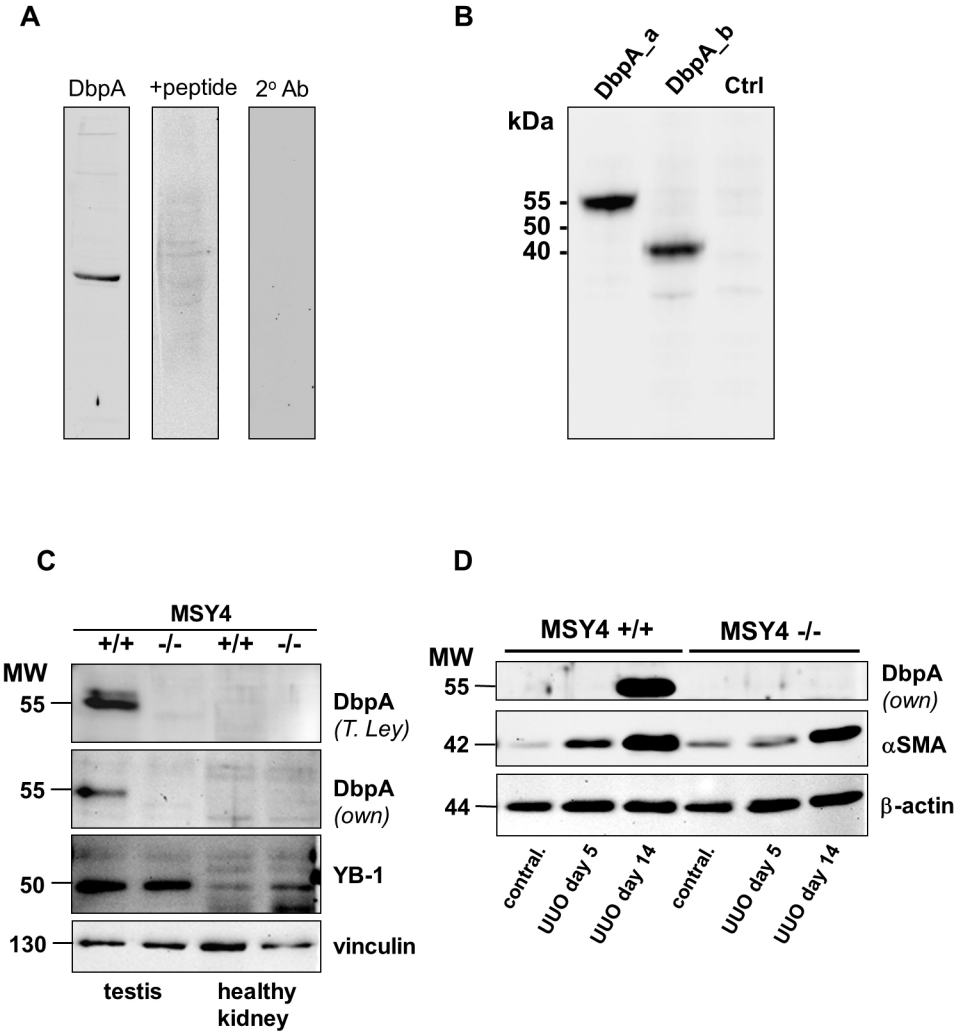
Supplementary Figure 1. Specificity of the anti-DbpA N-terminal sera. (**A**) Rat mesangial cell lysates were separated by SDS-PAGE, transferred onto membranes, and blotted using a rabbit anti-peptide DbpA sera that recognizes the conserved unique N-terminal epitope HVAGNPGGDAAPAA (aa 51-64) in human DbpA (left panel). Addition of the peptide antigen completely blocks staining (middle panel). As an additional control, the blots were incubated with secondary antibody alone, which gives no signal (right panel). (**B**) COS7 cells were transfected with a vector encoding either the DbpA_a or DbpA_b isoforms or the empty vector alone as a control. Transfected cell lysates were separated as described and blotted with the anti-DbpA N-terminal sera. (**C**) Whole-tissue lysates were prepared from the testis and kidneys of wild type and MSY4-deficient mice. The lysates were separated by SDS-PAGE, transferred onto nitrocellulose membranes, and blotted with the antibodies indicated. Our own DbpA serum shows comparable results to the serum provided by Dr. T. Ley, who also provided us with the MSY4-deficient mice. (**D**) Tissue lysates were prepared from kidneys of mice undergoing unilateral ureteral obstruction 5 or 14 days prior to sacrifice. In MSY4 wildtype animals a strong induction of DbpA is observed at 14 days following ureteral ligation, whereas a similar upregulation is absent in MSY4 knockout animals. αSMA staining as a marker of disease progression is upregulated in obstructed kidneys, β-actin served as loading control.

Supplementary Figure 2. RNA was prepared from the testis and kidneys of wildtype, heterozygous, and MSY4 knockout mice. DbpA transcripts were amplified using primers that overlapped with the N- and C-terminus of the coding sequence. Sequencing identified that the MSY4_1 transcript, which is abundantly expressed in testis, corresponds to

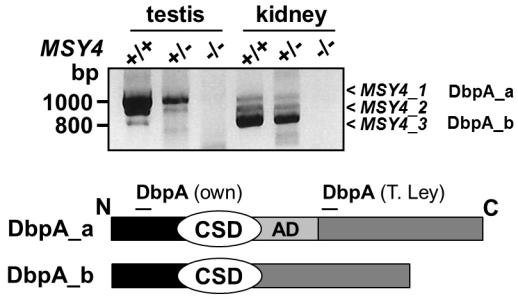
DbpA_a, whereas the MSY4_3 transcript, which is abundantly expressed in kidney, encodes DbpA_b. Although a third product MSY4_2 was observed, however all attempts at cloning have so far proven unsuccessful. Thus, observed differences in cDNA gel mobilities may be due to secondary structures.

Supplementary Figure 3. Western blot analysis of DbpA protein expression in rat mesangial cells stimulated with PDGF-BB (50 ng/ml) for the indicated time periods shows a time-dependent up-regulation, with a 2.2-fold induction of DbpA protein (44 kDa) after PDGF-BB stimulation for 24 hours (*p<0.05, **p<0.01; n=3).

Supplementary Figure 1



Supplementary Figure 2



Supplementary Figure 3