Supplementary Data

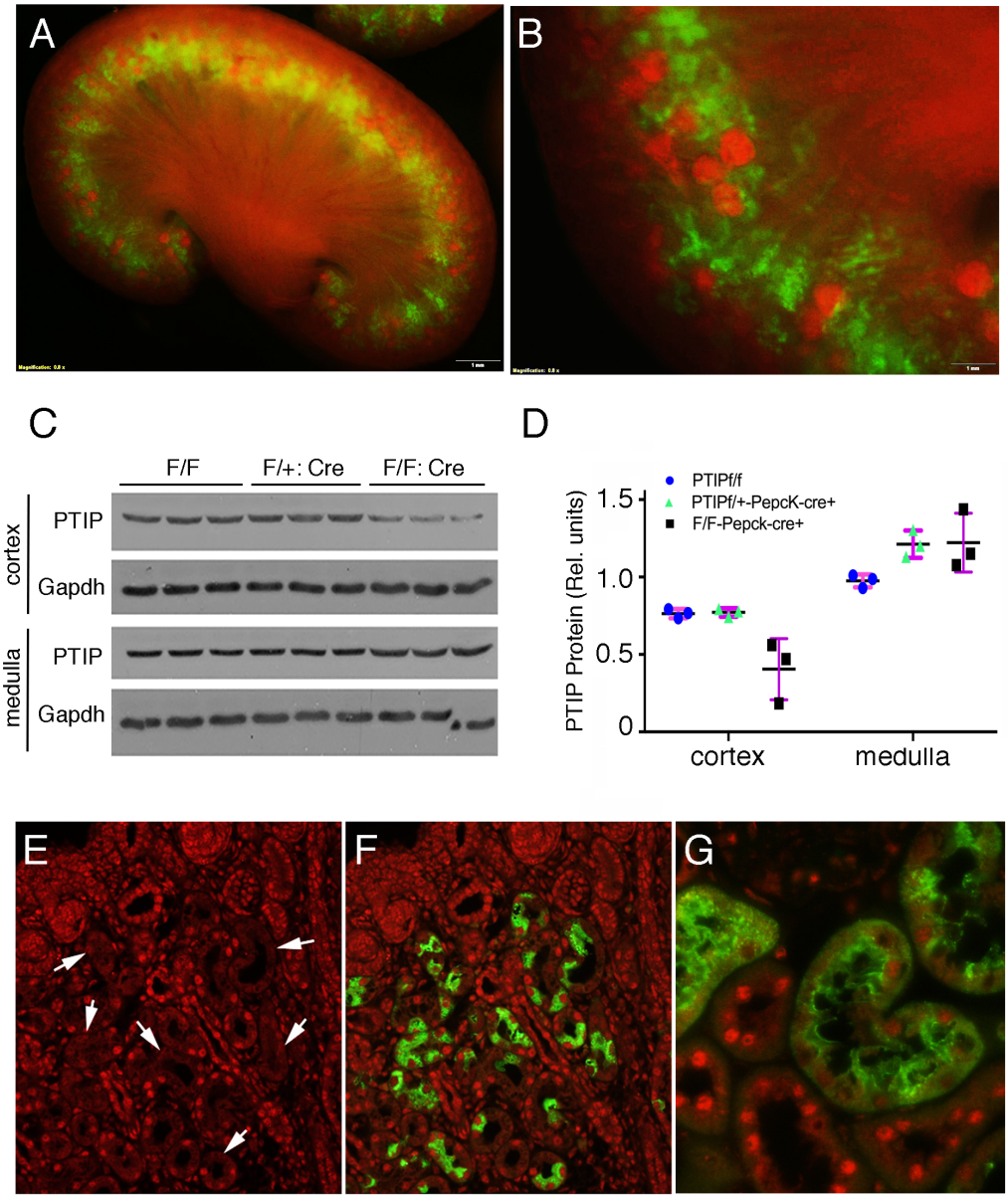

Figure S1. Generating Proximal Tubule Specific PTIP Deletions. A, B) A Pepck-Cre transgene was crossed to the Gt(Rosa)26Sor$^{tm4(ACTB-tdTomato,-EGFP)luo}$ reporter strain of mice to examine the tissue and cell type specificity of Cre expression. EGFP (green) marks cells with Cre activity, whereas tdTomato (red) marks unrecombined cell types. Note lack of Cre activity in the renal medulla, major collecting ducts, and glomeruli. C) Western blot of proteins lysates from three independent animals for each indicated genotype. PTIP protein is reduced in the cortex only in homozygous *Paxip*$^{Flox/Flox}$ mice carrying the Pepck-Cre transgene. D) Quantitation of the protein bands detected in C. E) Immunostaining for PTIP protein (red) in newborn kidneys of PTIP- mice; note absence of nuclear PTIP in presumptive proximal tubules that also are marked with the EGFP reporter (F). G) Higher magnification of proximal tubules (EGFP) that have deleted nuclear PTIP (red).

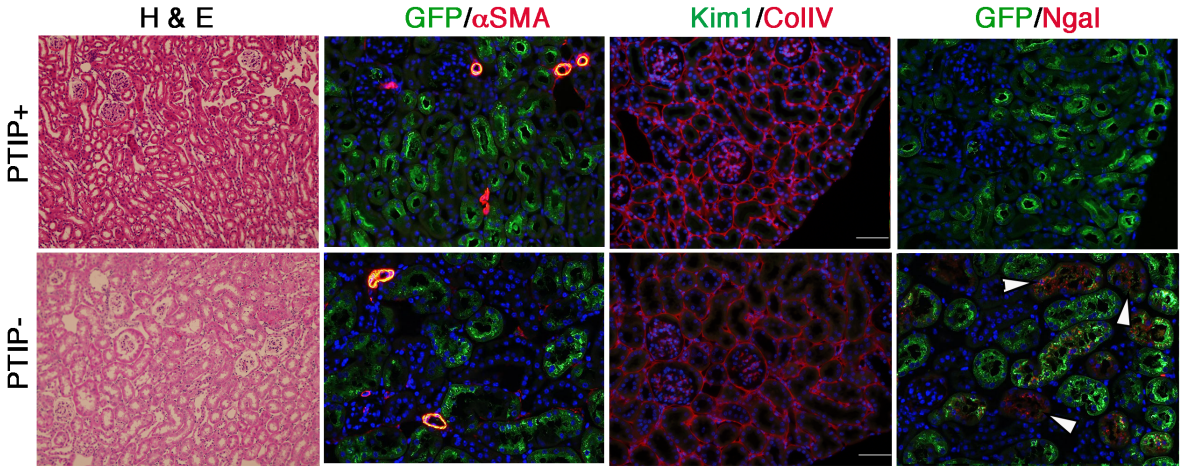

Figure S2. PTIP- Kidneys do not exhibit gross morphological defects. Hematoxylin and Eosin (H & E) stained histological sections and immunostaining for the indicated proteins are shown for wild-type (PTIP+) and PTIP- adult kidneys. No evidence for fibrosis or increased matrix deposition is observed in PTIP- kidneys, as determined by αSMA and ColIV staining. The GFP reporter marks proximal tubules with activated Cre. Also, Kidney Injury Molecule 1 (Kim1, green) is undetectable in PTIP- kidneys. Note faint NGAL staining is seen in some tubules (arrowheads), most of which are GFP negative, indicating a more distal tubule phenotype.