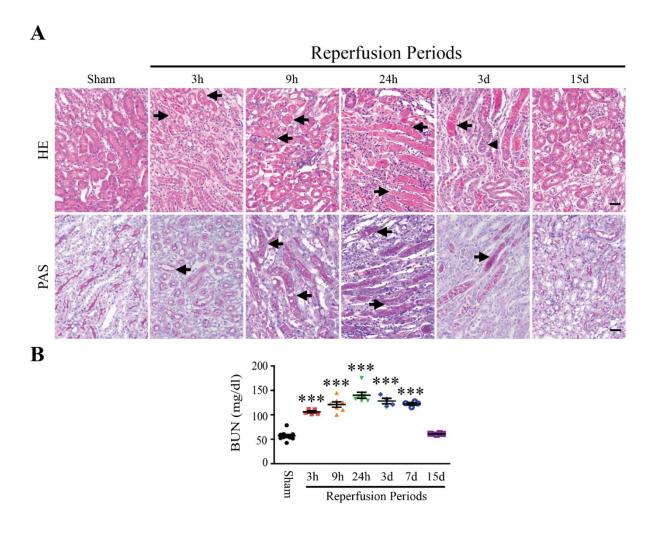
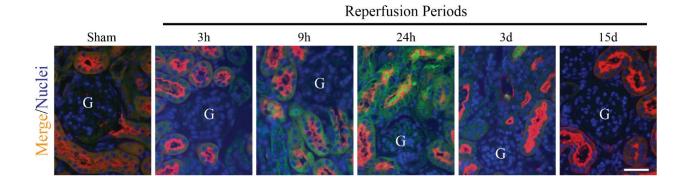
Supplemental Figure 1. Characterization of the AKI mouse model caused by 30 minutes of bilateral kidney ischemia. (A) H&E and PAS staining of paraffin sections from kidneys undergoing sham operation (n=10) or bilateral I/R insult after various reperfusion periods as indicated: 3 hours (n=5), 9 and 24 hours (n=15 at each time point), as well as 3 and 15 days (n=5 at each time point). Note loss of brush boarders and partial cytoplasmic degeneration (arrows) after 3 hours of reperfusion or acute tubular necrosis (arrows) after 9 or 24 hours or 3 days of reperfusion. Arrowhead indicates tubular regeneration. Scale bars: 40 μm. (B) BUN levels obtained from control mice (n=10) or mice with varying periods of reflow as shown: 3 hours (n=5), 9 and 24 hours (n=7 at each time point), as well as 3, 7 and 15 days (n=4 at each time point) (mean ± SEM). ***P<0.001 relative to sham-operated mice by ANOVA.



Supplemental Figure 2. Glomeruli after 30 minutes of bilateral renal ischemia are lack of CRELD2 expression. Dual IF staining for CRELD2 (green) and LTL (red) on Histochoice-fixed paraffin kidney sections from mice with sham operation or I/R injury at various reperfusion periods as indicated. Nuclei were counterstained with Hoechst 33342 (blue). G: glomerulus. Scale bars, $40 \mu m$.



Supplemental Figure 3. The same urine samples included in Figure 6, C-D were analyzed by SDS-PAGE (the urine volume was normalized to 4 μg of urine Cr excretion) and stained with Coomassie G-250. 1.2 μg of BSA was used as a reference for the band density equaling to UACR 300 $\mu g/mg$ in the urine containing 4 μg of Cr from ADTKD patients.

