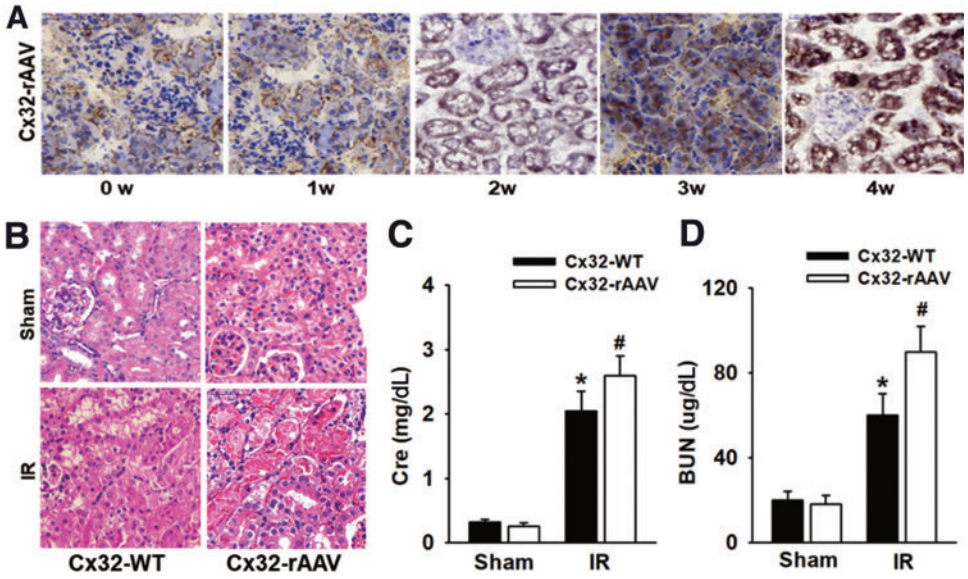

SUPPLEMENTARY FIG. S2. Cx32 overexpression aggravates IR-induced AKI. The kidney-specific Cx32 overexpression mice (Cx32-rAAV) were constructed by tail vein injection of recombinant adeno-associated virus (rAAV) vectors containing the genes for Cx32. **(A)** Cx32 expression alternation after 1×10^{12} vg rAAV 2/9-CMV-eGFP injection through tail vein at 4 weeks of life. Mice were sacrificed at 0, 1, 2, 3, and 4 weeks after injection (IHC; scale bar 50 μm). At 8 weeks of life, $Cx32^{-/-}$, $Cx32^{+/+}$, and Cx32-rAAV mice underwent renal IR were sacrificed at the time point of 24 h after reperfusion. **(B)** Renal damage of Cx32-WT and Cx32-rAAV mice after renal IR exposure (H&E; scale bar 50 μm). **(C, D)** Levels of Cr and BUN of Cx32-WT and Cx32-rAAV mice at 24 h after renal IR. Data are presented as mean ± SE ($n = 8$). $^{*}p < 0.05$ *versus* sham group of Cx32-WT mice; $^{\#}p < 0.05$ *versus* 24 h after reperfusion group of Cx32-WT mice in **(C, D)**. AKI, acute kidney injury; BUN, blood urea nitrogen; Cr, creatinine; Cx32, connexin32; $Cx32^{-/-}$, Cx32-gene knockdown; $Cx32^{+/+}$, wild-type; Cx32-rAAV, Cx32-gene overexpression; H&E, hematoxylin–eosin staining; IHC, immunohistochemistry; IR, ischemia reperfusion.