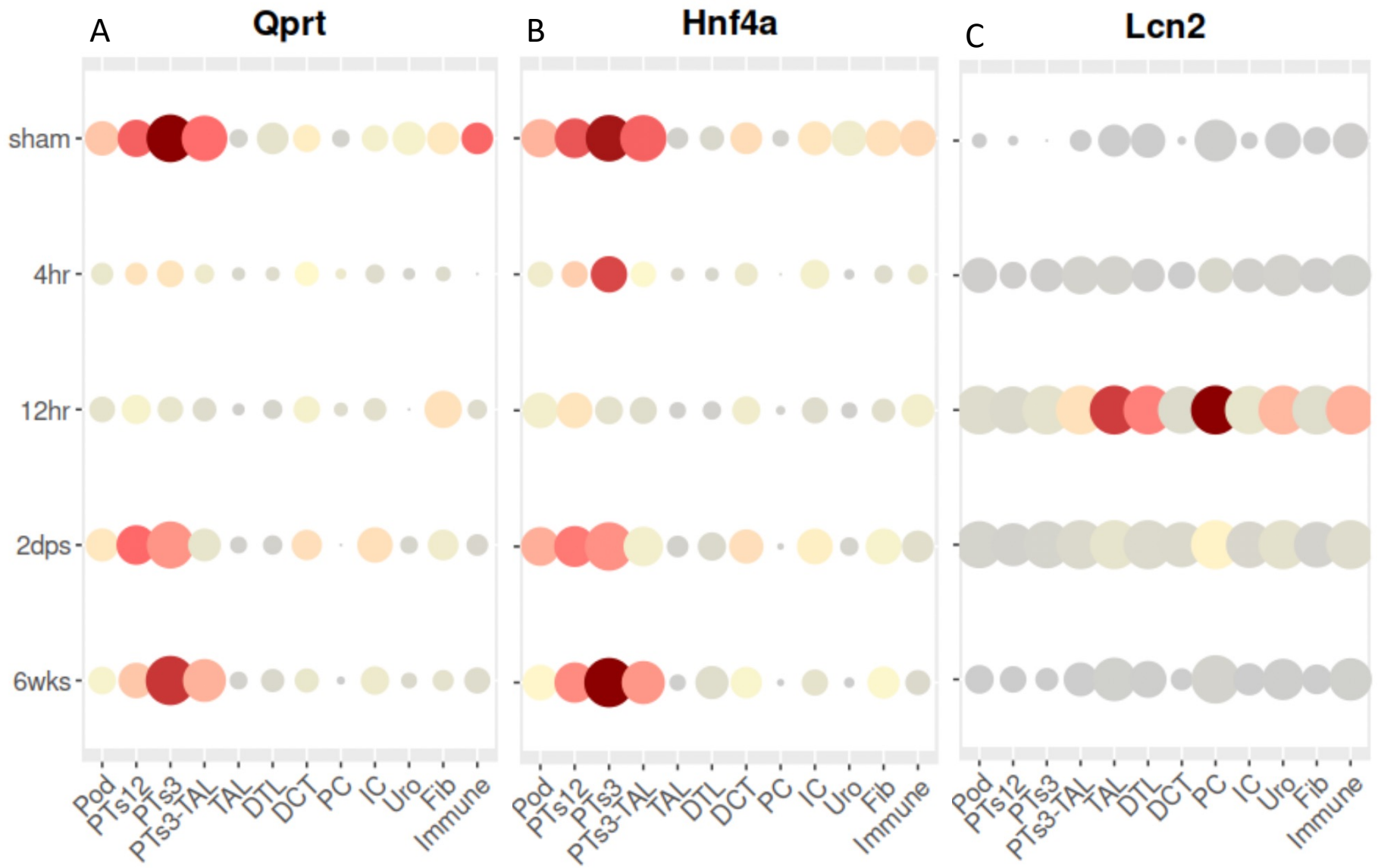

Figure S1: Published data (Dixon et al. JASN 2022) assessing spatially resolved kidney transcriptomics in female mice undergoing ischemia reperfusion injury demonstrate that both QPRT (a) and HNF4a (b) are suppressed during injury, marked by Lcn2 expression (c), in females and recover as injury subsides.

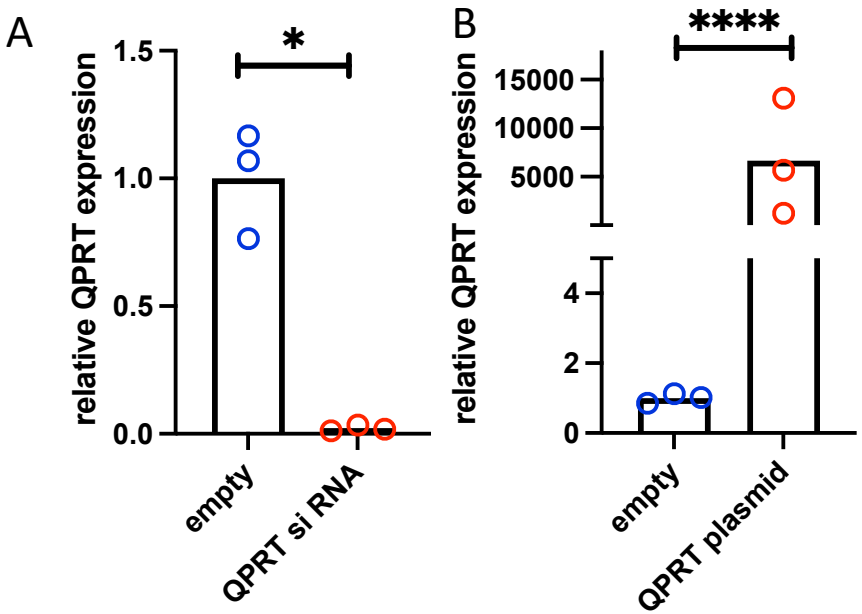

Figure S2: Validation of QPRT siRNA (a) and QPRT overexpression plasmid (b) in HK2 cells.

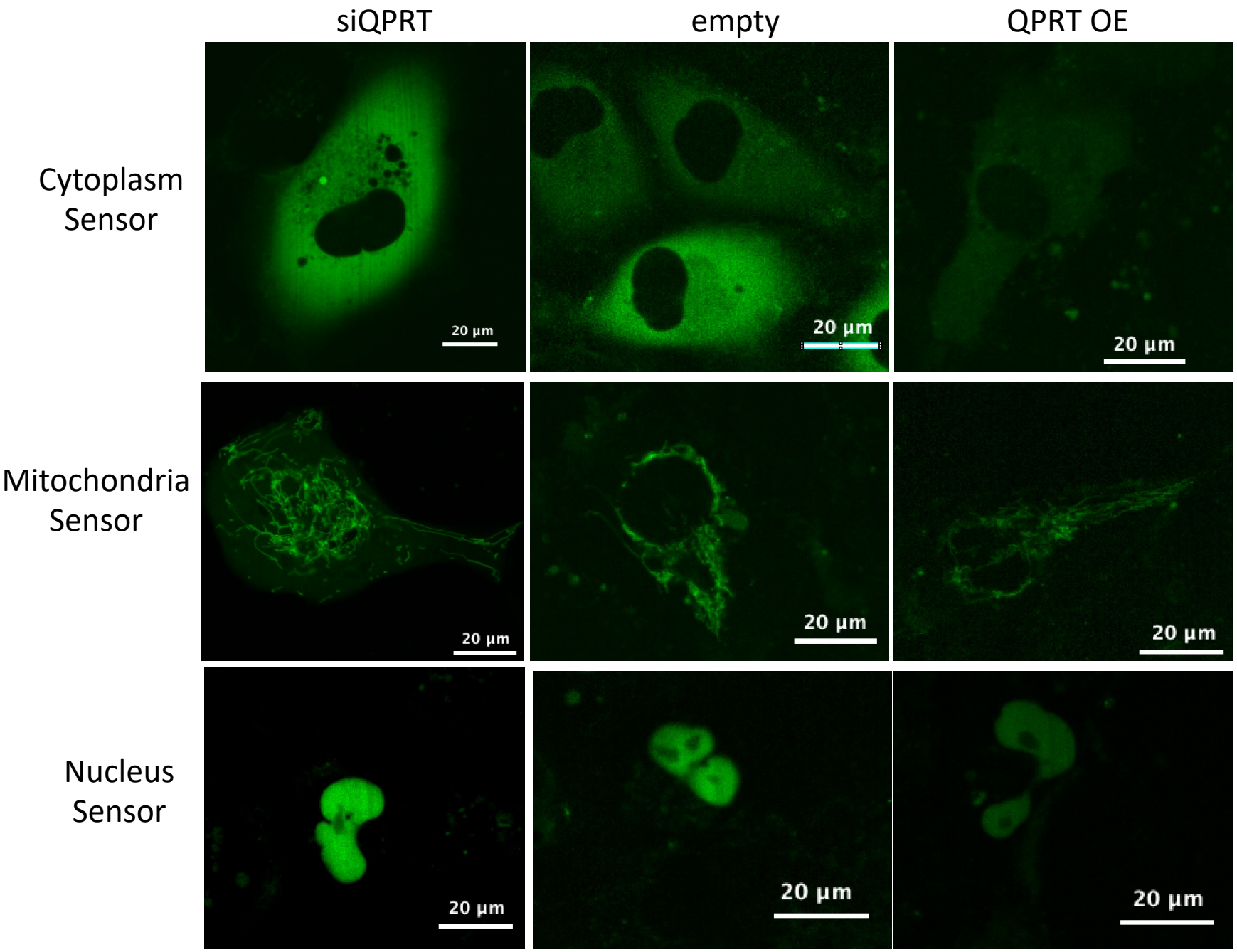

Fig S3: Representative images of the 488 wavelength in NAD+ biosensor images of QPRT overexpression and knock down. Emission at the 488 wavelength is NAD+ specific. Emission at a 405 wavelength (not pictured) is not NAD+ dependent and allows for correction of variations in plasmid expression, microscope focus, etc. The intensity of the 488/405 emission ratio is inversely related to NAD+. Lower NAD+ leads to brighter emission. After image gating in ImageJ and fluorescent intensity measurement of 488/405, the inverse is calculated to reflect relative NAD+ levels. (See Fig2E-G, Fig6 D-F, Fig S4 B-D).

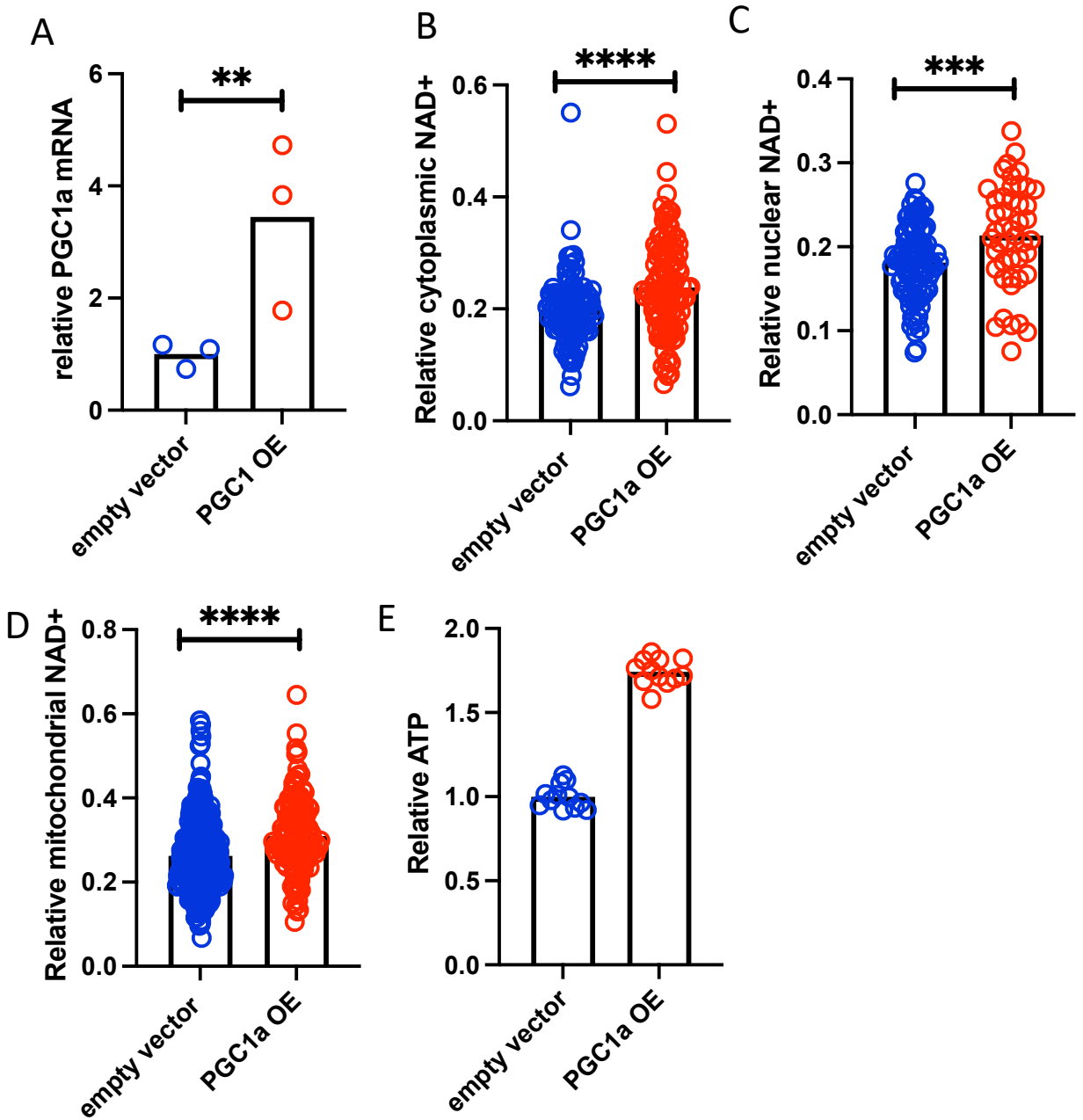

Figure S4: a. Validation of PGC1α overexpression in an LLC-PK1 cell line after transduction of Lentivirus carrying PGC1α OE plasmid. PGC1α OE increases NAD+ in the cytoplasm (b), nucleus (c), and mitochondria (d). PGC1α overexpression increases cellular ATP (e).

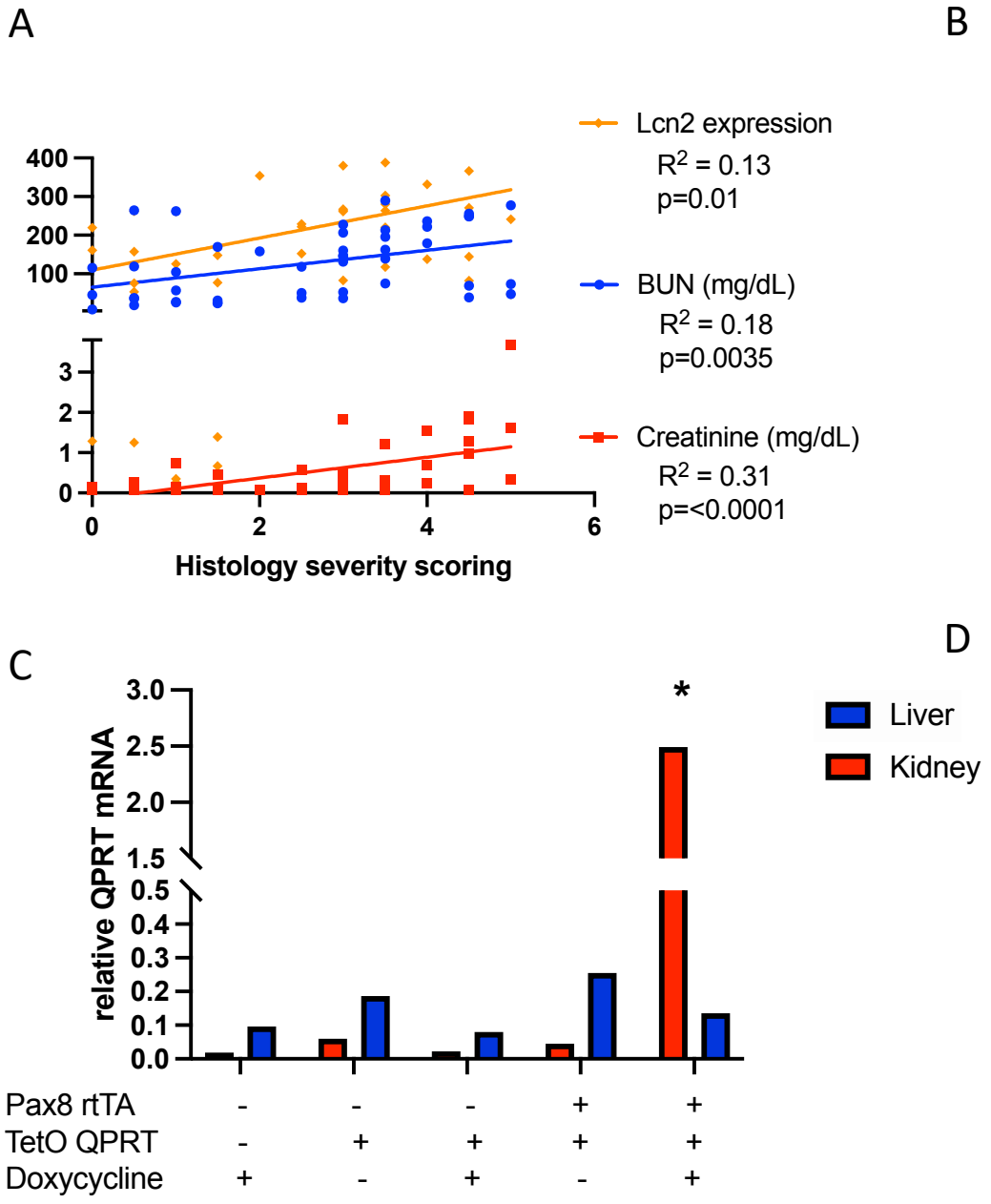

Figure S5: a. In the cisplatin AKI model, injury severity as assessed by kidney lcn2 expression, BUN, and serum creatinine all correlate to injury scoring on histology. b. Folic acid induced AKI leads to QPRT suppression in kidney. c. The iNephQPRT mouse exhibits non-leaky overexpression of QPRT in the kidney only when both transgenes are present with the addition of doxycycline. d. There is no difference in histological scoring in iNephQPRT mice compared to controls after cisplatin.

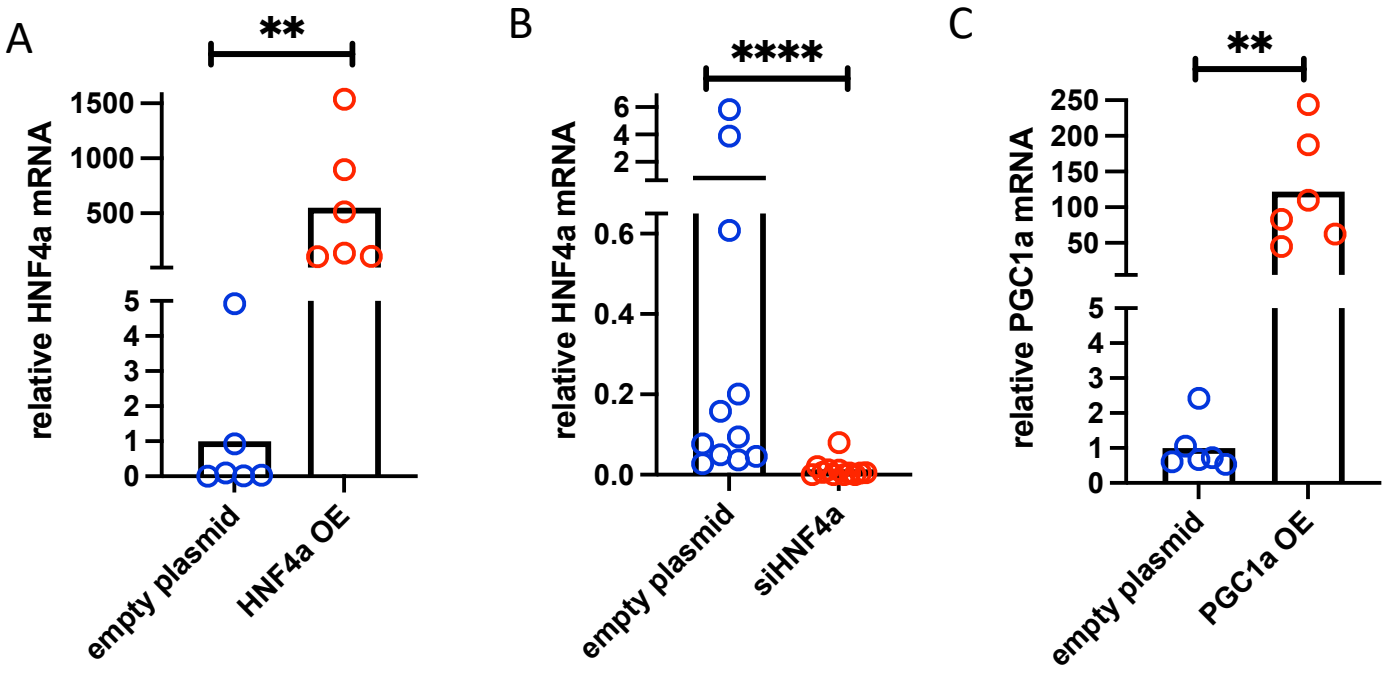

Figure S6: a-b. Validation of an HNF4α overexpression plasmid and siHNF4α in HK2 cells. c. Validation of PGC1α overexpression plasmid.