Supplemental information

FoxM1 drives proximal tubule proliferation during repair from acute ischemic kidney injury

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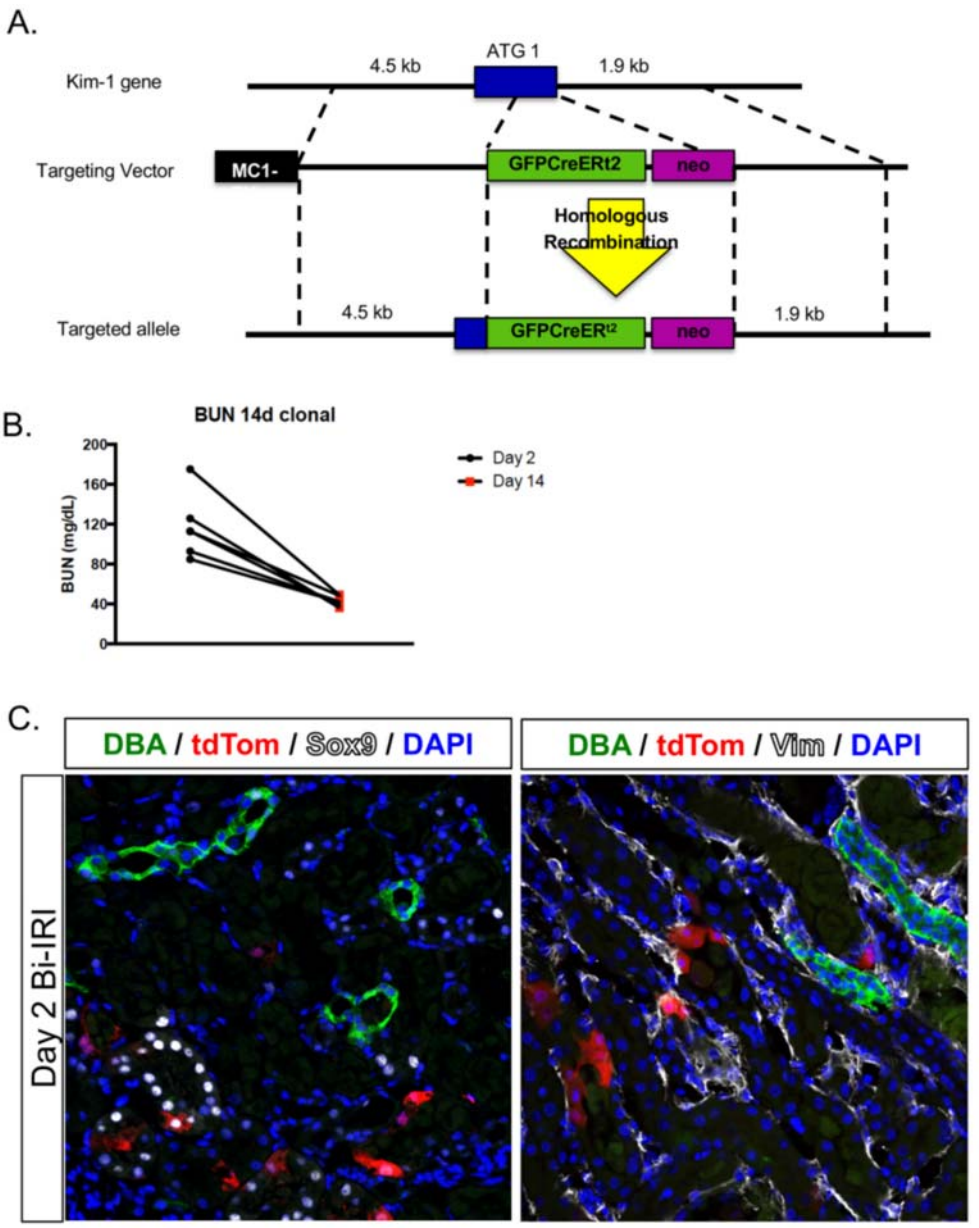
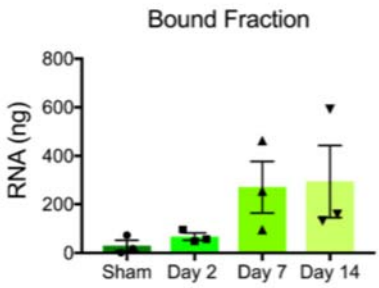


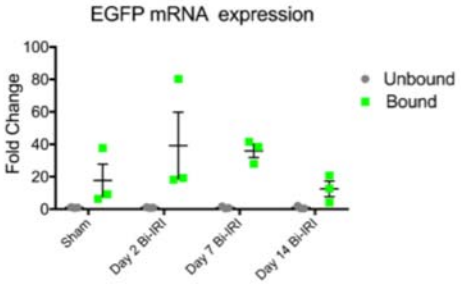
Supplemental Figure 1. A. Targeting strategy design for Kim1-GFPCreERt2 knock-in allele. A targeting vector was used to insert the EGFRPCreERt2 (GCE) transgene and a frt-flanked PGKneobpA selection cassette into the ATG codon of the Havcr1 gene. **B.** BUN increased between 80-150 mg/dL at 48 hours after injury and downtrended at day 14 as expected. **C.** Immunostaining with DBA, a distal tubular marker, and markers of dedifferentiation (Sox9 and Vimentin) in day 2 Bi-IRI kidney.

A.

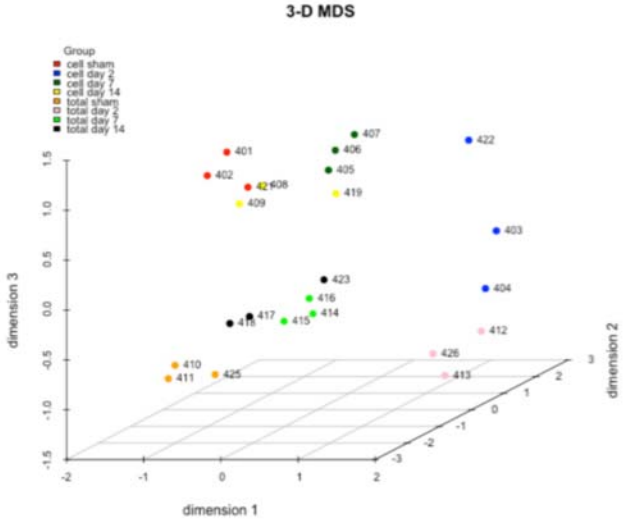
Fraction	Group	RNA mass (ng)	STDEV	RIN
Bound	Sham	30.64	37.37	9.3
	Day 2 IRI	67.31	25.75	9.0
	Day 7 IRI	271.20	184.64	9.4
	Day 14 IRI	293.92	258.22	9.6
Unbound	Sham	15224.59	11012.13	9.7
	Day 2 IRI	23399.00	5405.53	9.6
	Day 7 IRI	29291.90	4377.96	10
	Day 14 IRI	18779.92	5434.56	9.3



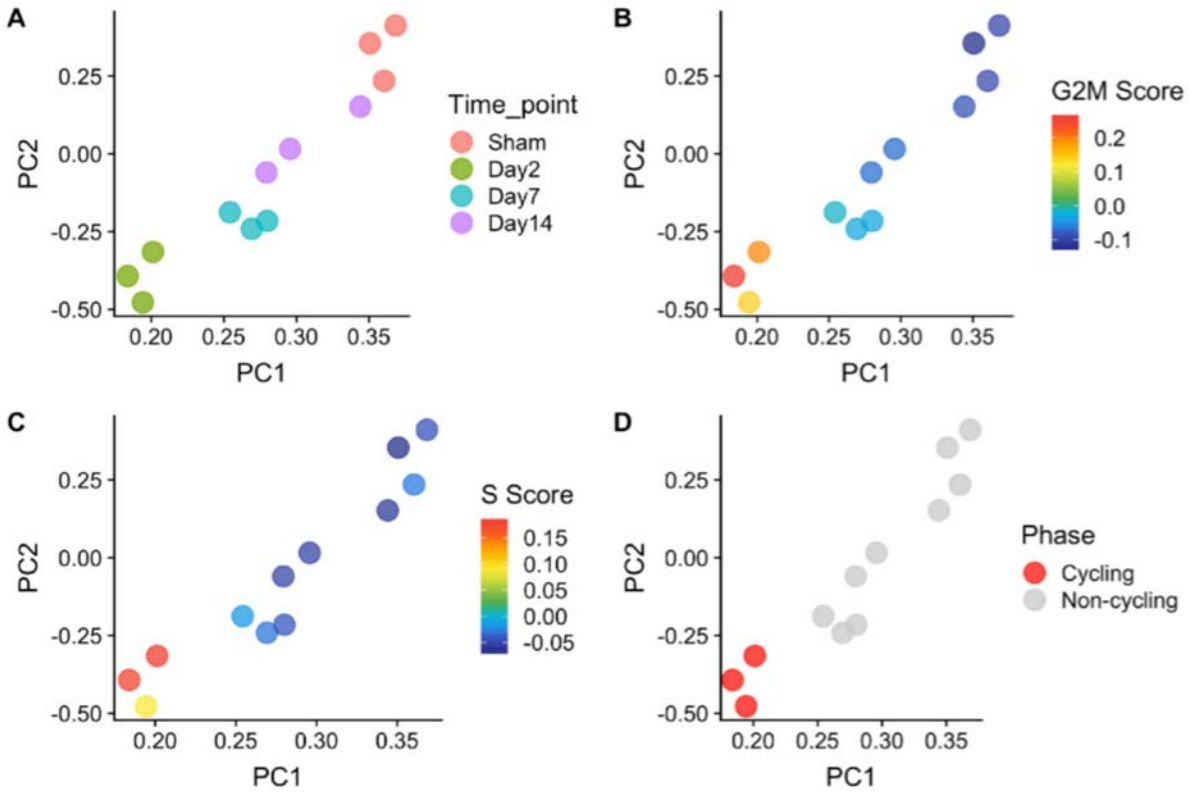
B.



C.



Supplemental Figure 2. A. Amount of isolated RNA from TRAP at the different points after injury. **B.** qPCR for EGFP in bound vs unbound fraction in sham vs. day 2 after injury. **C.** MDS plot showing good clustering of the samples in cell (bound) vs. total (unbound) at the different time points.



Supplemental Figure 3. Bioinformatic analysis of cell cycle gene expression in the Kim1-GCE TRAP samples across time. **A.** Principal component analysis (PCA) based on the expression of marker genes related to G2M and S phases. **B.** Visualization of the G2M phase score for each sample on the PCA plot. **C.**Visualization of the S phase score for each sample on the PCA plot. **D.** PCA plot illustrating the cell cycle state for each sample. Samples were assigned to “Cycling” if they are in G2M or S phase and “Non Cycling” otherwise.