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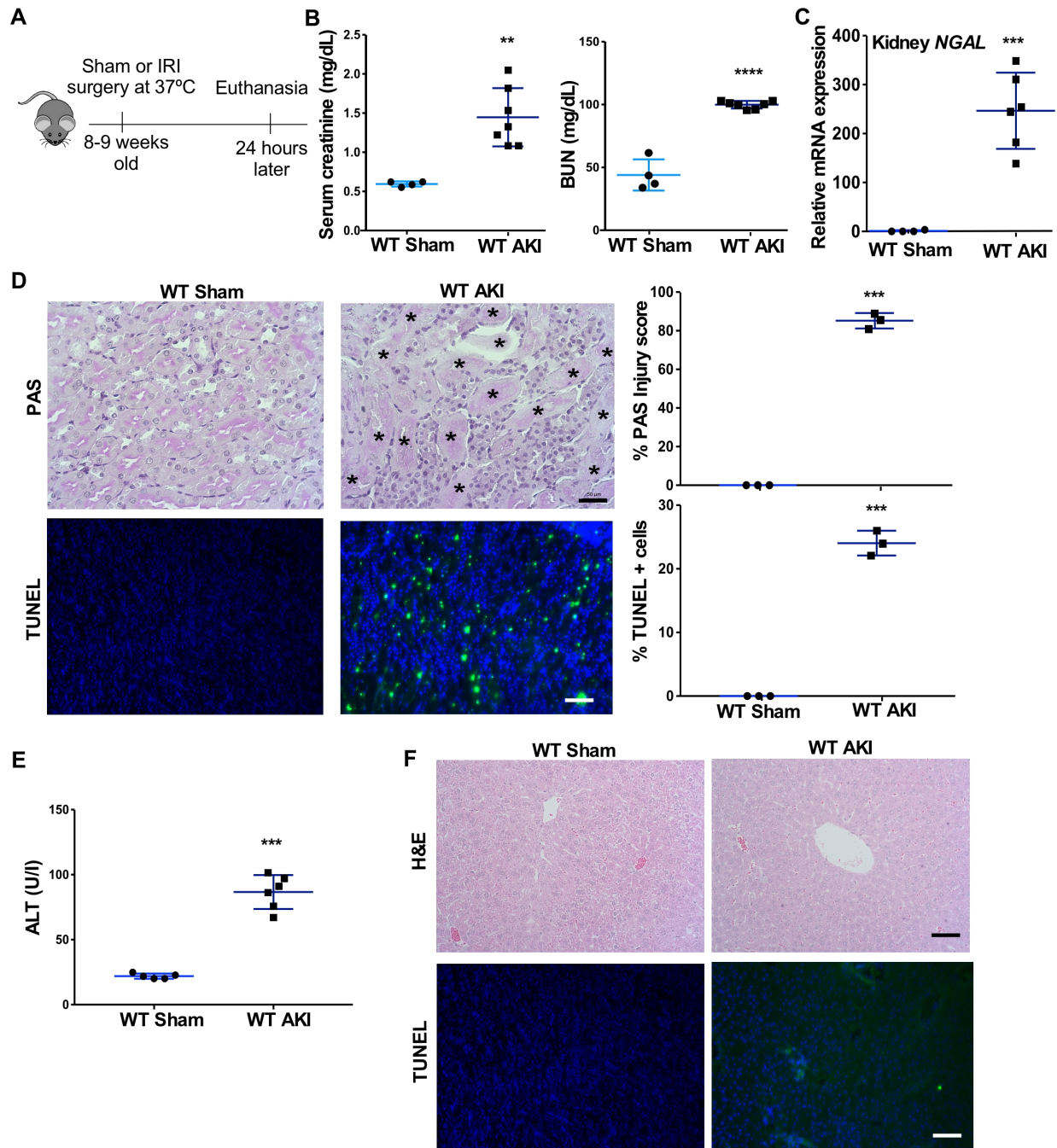
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Supplemental Table 1: Sequences of qRT-PCR primers.

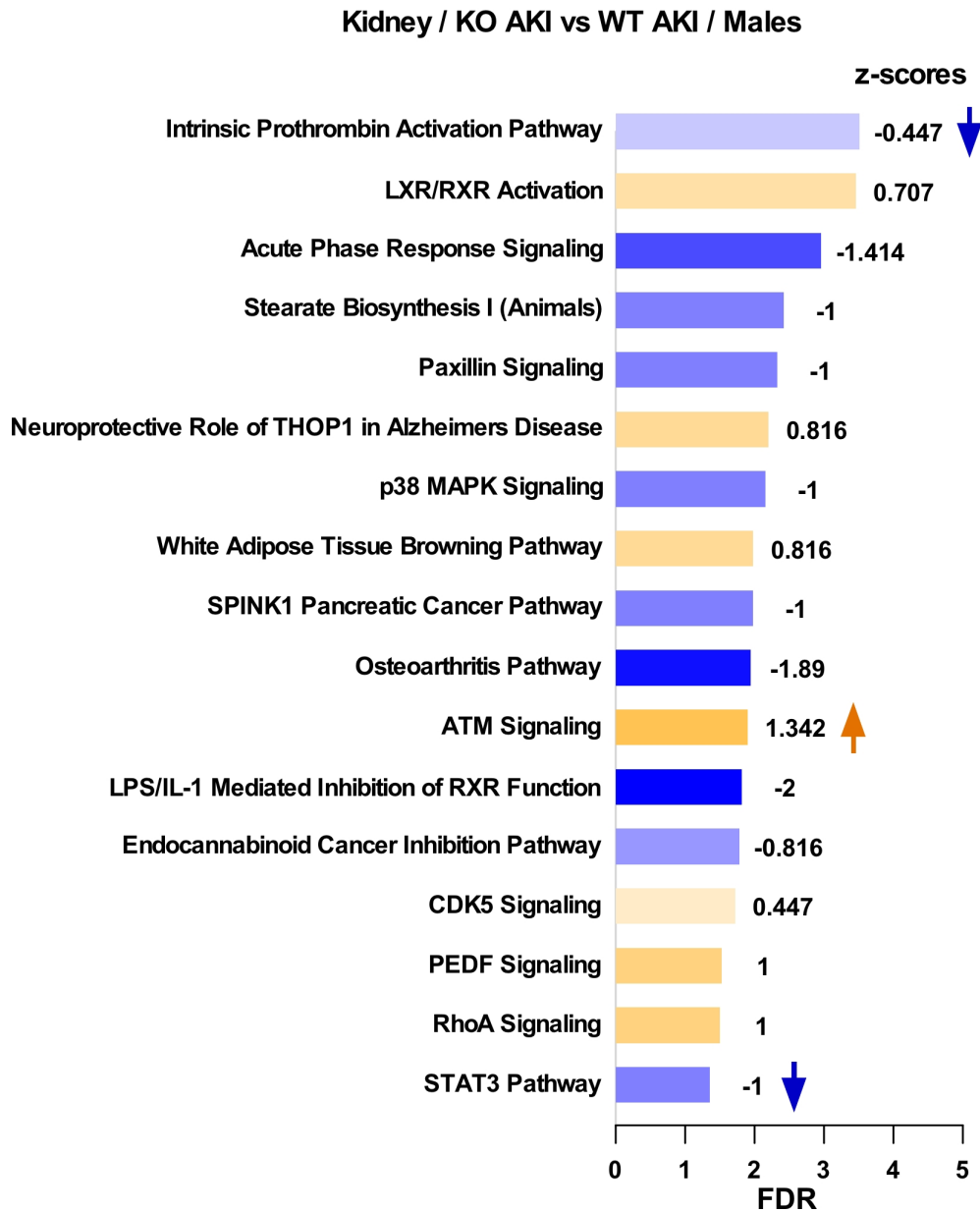
| Mouse genes | Forward | Reverse |
|-------------------------------|---------------------------|-------------------------|
| <i>Car</i> | GGAGGACCAGATCTCCCTTC | GTGGAGGATCGACTCCAAAA |
| <i>Cyp24a1</i> | GAGGAAGAAGCCCTGACCTT | TGCAGGGCTTGACTGATTTG |
| <i>Fgg</i> | GTGCTGGCTGTAAAGAGCTG | TGGGCAGAACTACCGAATCT |
| <i>Il-6</i> | TCCTCTCTGCAAGAGACTTCCATCC | GGGAAGGCCGTGGTTGTCACC |
| <i>Lxrα</i> | GCCTCAATGCCTGATGTTTC | CTGCATCTTGAGGTTCTGTCTTC |
| <i>Ngal</i> | AATGTCACCTCCATCCTGGT | ATTTCCAGAGTGA ACTGGC |
| <i>Stat5a</i> | GCTCAGCGCCCACTTCA | GACTCTGCACCACGCCTGT |
| <i>Sult1e1</i> | GCCAAAGATGTCGCCGTTTC | AACCATACGGA ACTTGCCCT |

Supplemental Figure 1



Supplemental Figure 1: Establishment of the bilateral renal ischemia reperfusion model of AKI. (A) Schematic representation of the ischemic AKI model. (B-F) WT male mice were subject to the 30-min ischemic AKI, and the mice were sacrificed 24 h after the surgery. Shown are serum levels of creatinine and BUN (B), renal mRNA expression of *NGAL* (C), kidney histology (D, with asterisks indicating tubular damage), serum ALT level (E), and liver histology (F). n=7 for each group. Scale bars are 50 μ m. Results are presented as the mean \pm SD. ***, P < 0.001; ****, P < 0.0001, compared with the sham group.

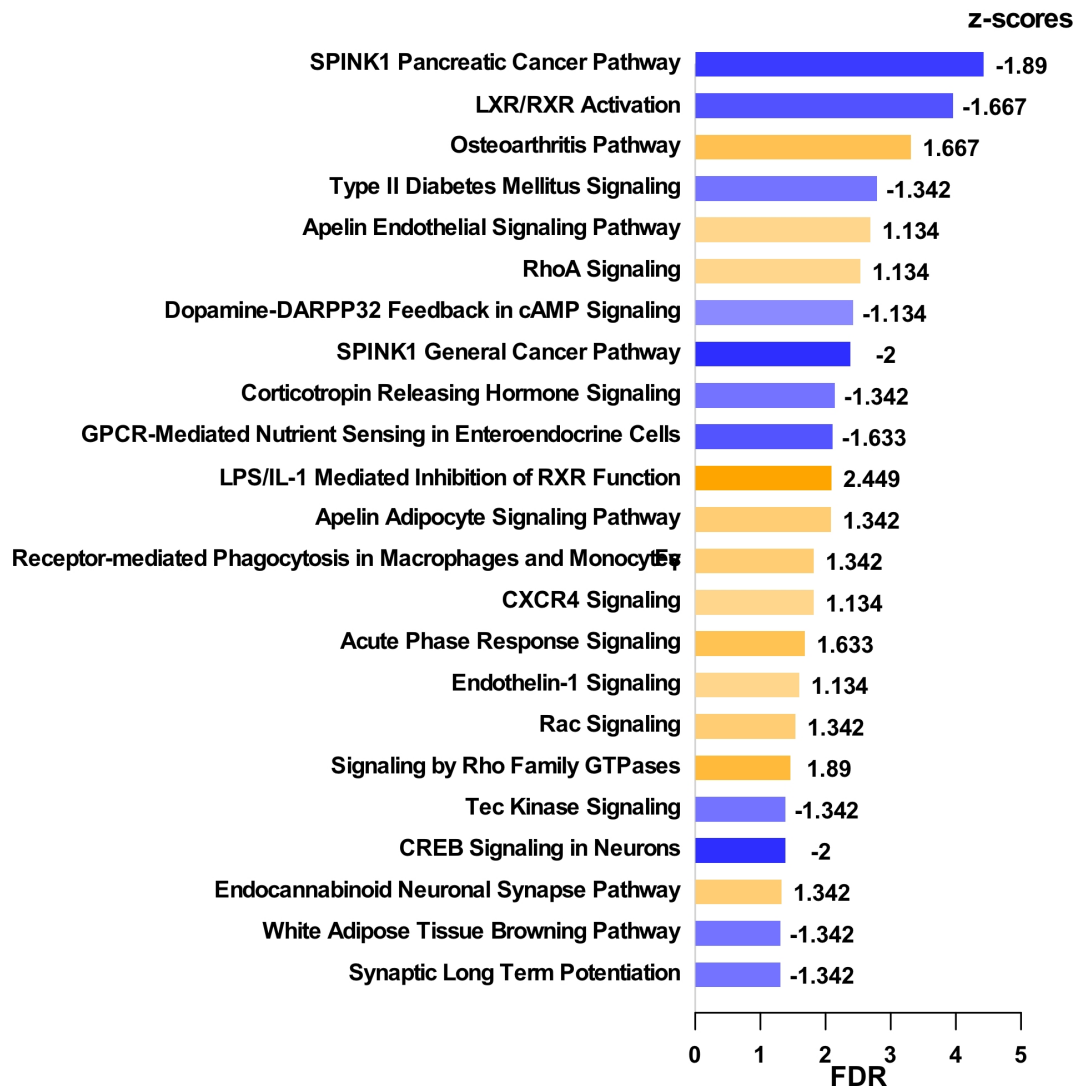
Supplemental Fig. 2A



Supplemental Figure 2: Ingenuity pathway analysis (IPA) of microarray results. (A-C) Shown are z-scores and false discovery rate (FDR) of male kidney (KO AKI vs WT AKI) (A), male liver (WT AKI vs WT Sham) (B) and female liver (WT AKI vs WT Sham) (C). Several up-regulated (orange arrows) and down-regulated (blue arrows) pathways are highlighted.

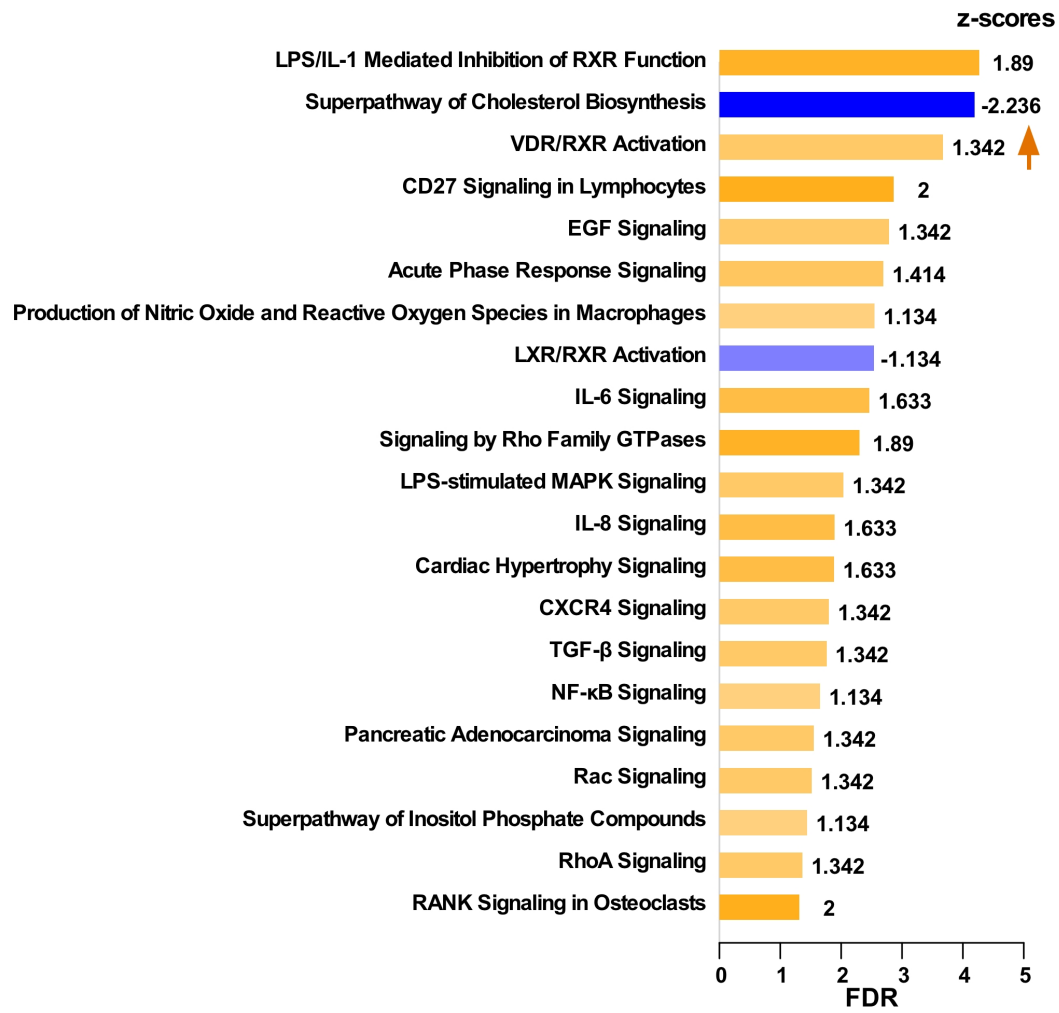
Supplemental Fig. 2B

Liver / WT AKI vs WT Sham / Males

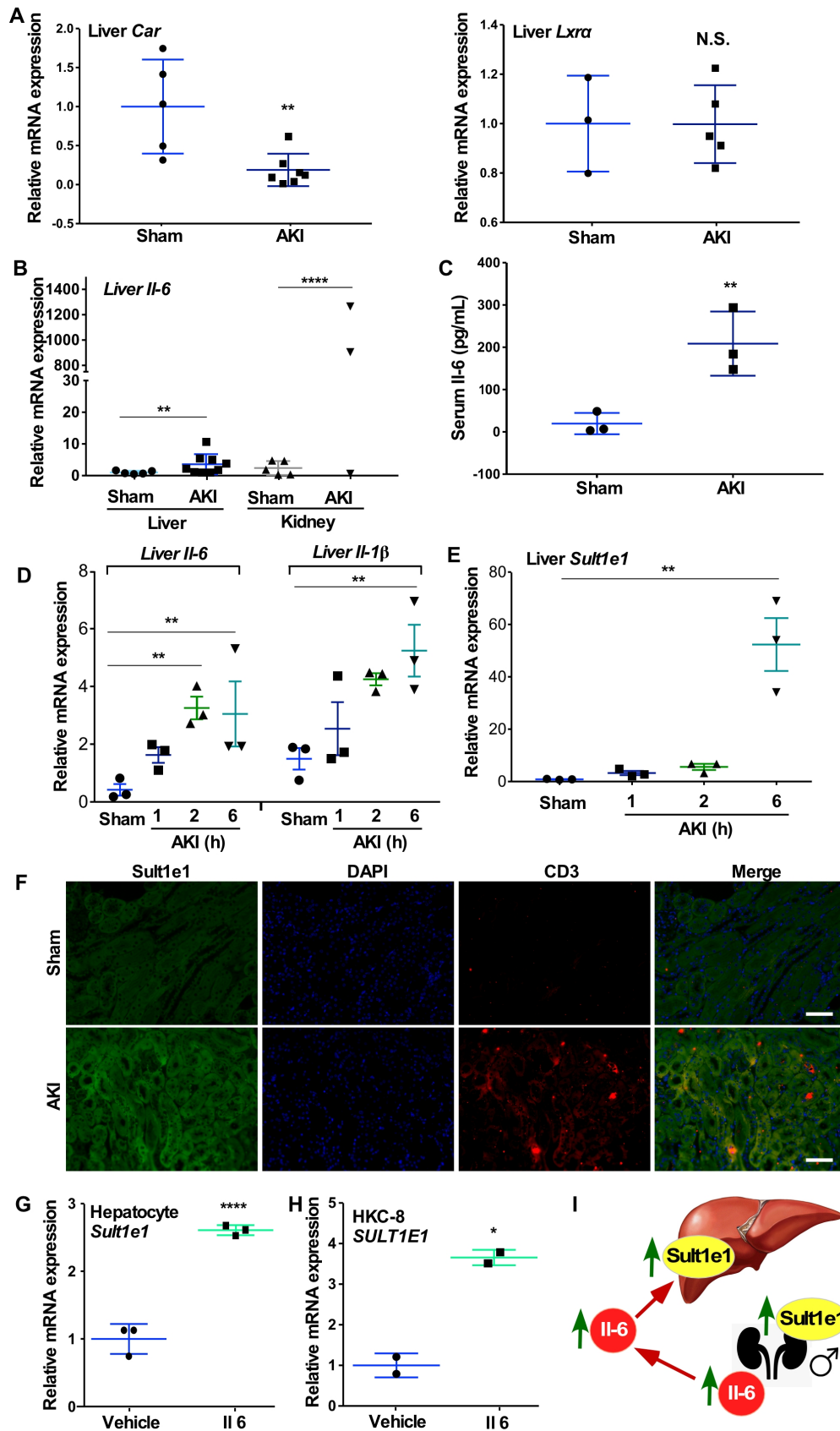


Supplemental Fig. 2C

Liver / WT AKI vs WT Sham / Females

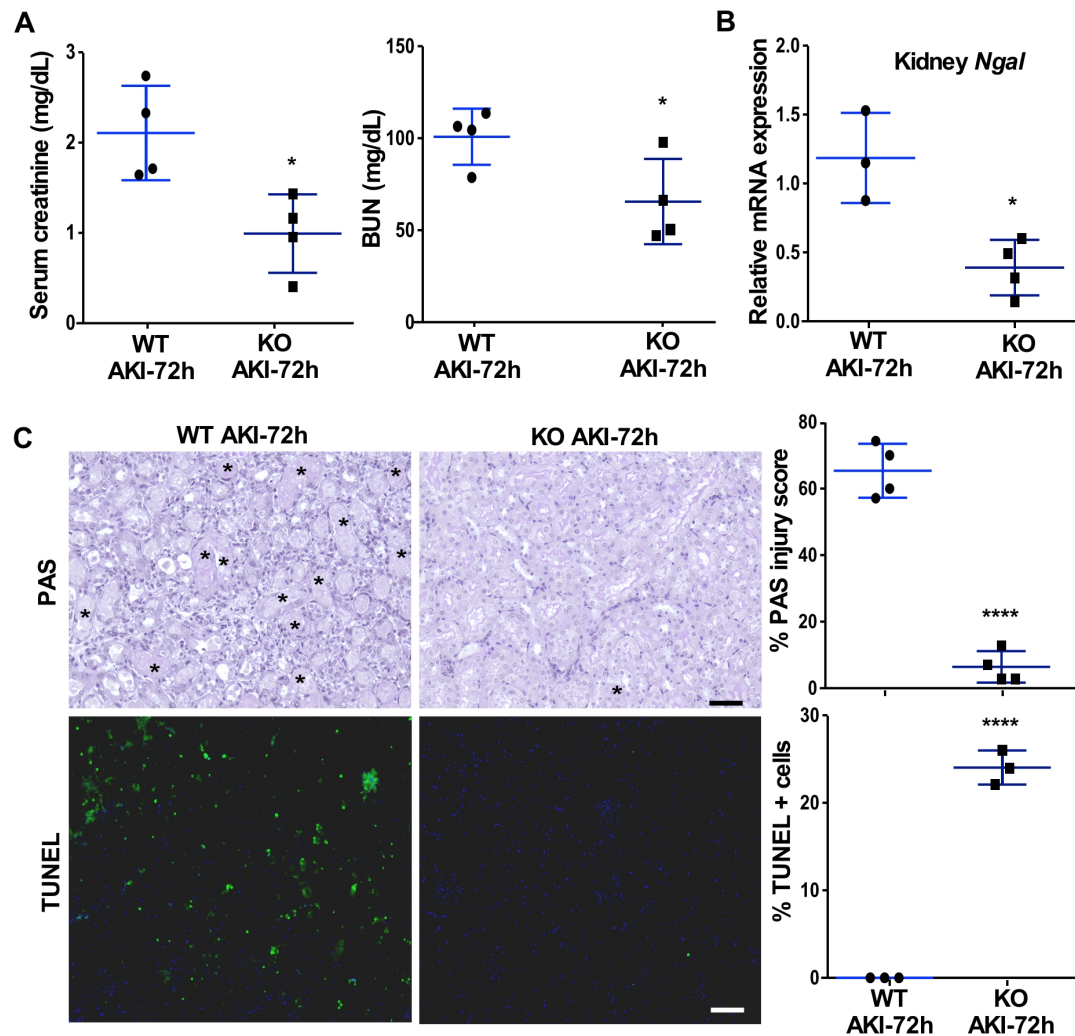


Supplemental Figure 3



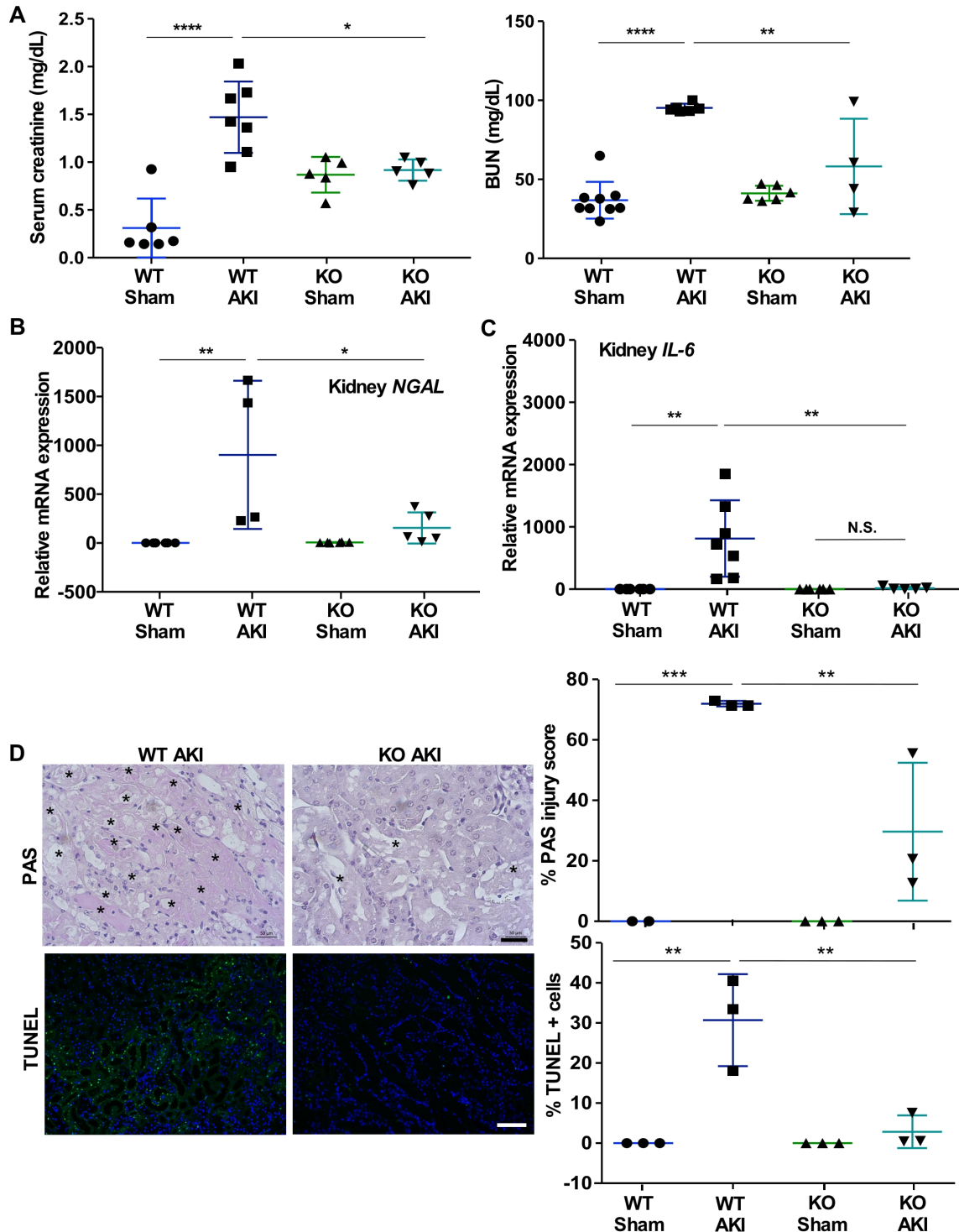
Supplemental Figure 3: Inflammation is a potential mechanism for AKI responsive induction of Sult1e1 in the liver. (A-F) Mice are the same as described in Figure 1. Shown are hepatic mRNA expression of *Car* and *Lxra* (A), hepatic and renal mRNA expression of *Il-6* (B), serum level of Il-6 measured by ELISA (C), time course of hepatic expression of *Il-6* and *Il-1 β* (D) and *Sult1e1* (E), and immunofluorescence of Sult1e1 and CD3 (F). **(G and H)** The expression of *Sult1e1* in primary hepatocytes (G) and HKC-8 cells (H) treated with vehicle or Il-6. **(I)** Proposed model of Il-6-mediated distal regulation of hepatic Sult1e1 by AKI. Scale bars are 50 μ m. Results are presented as the mean \pm SD. *, P < 0.05; **, P < 0.01; ****, P < 0.0001, N.S., statistically not significant, compared with the sham groups, or the comparisons are labeled.

Supplemental Figure 4



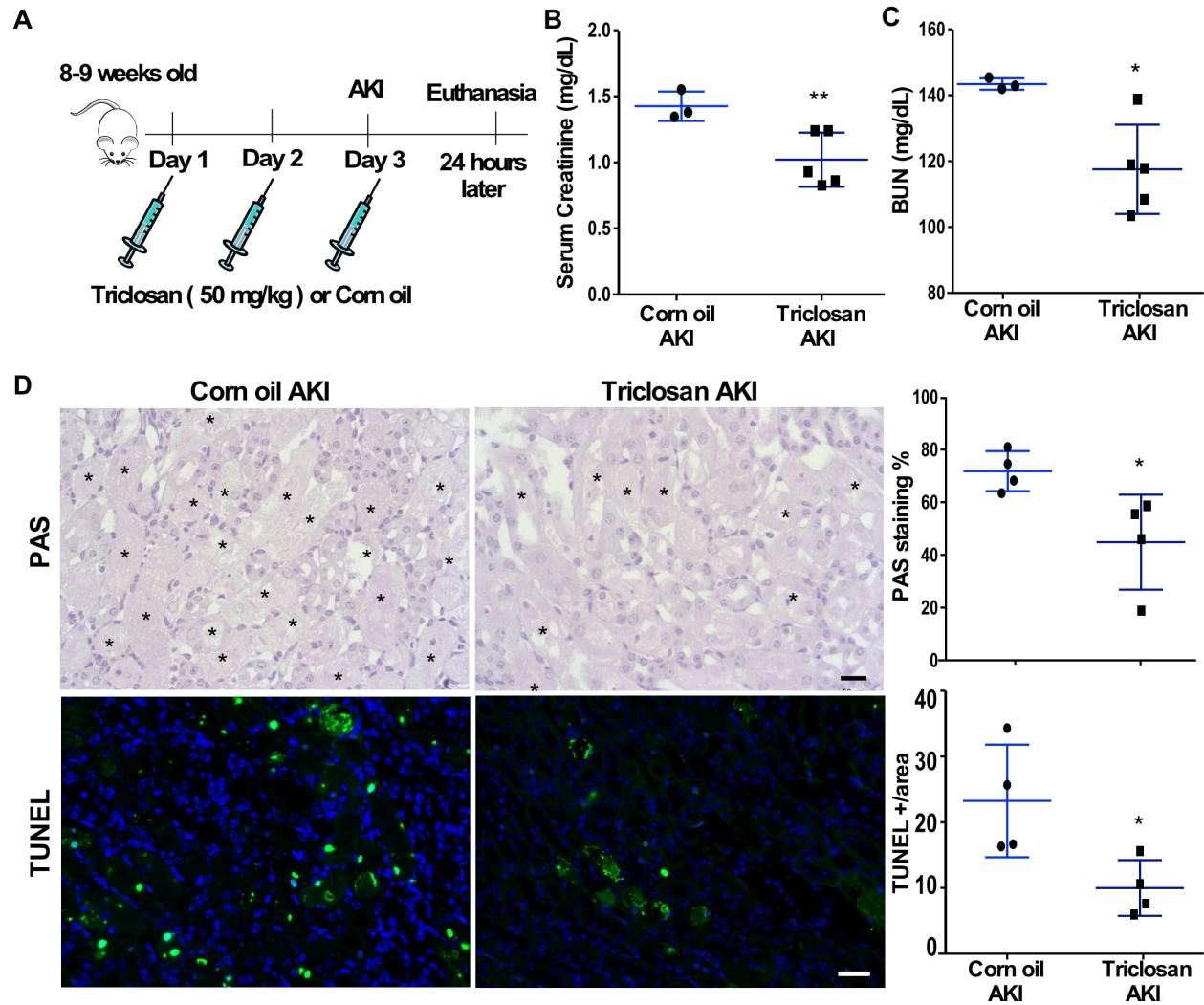
Supplemental Figure 4: Renal protective effect of Sult1e1 ablation 72-hours post AKI. (A-C) WT male mice were subjected to the 30-min ischemic AKI, and the mice were sacrificed 72 h after the surgery. Shown are serum levels of creatinine and BUN (A), renal mRNA expression of *NGAL* (B), and kidney histology (C, with asterisks indicating tubular damage). n=4 for each group. Scale bars are 50 μ m. Results are presented as the mean \pm SD. *P < 0.05, compared with the WT AKI-72h groups.

Supplemental Figure 5



Supplemental Figure 5: Knockout of Sult1e1 protects female mice from AKI. (A-D) WT and Sult1e1 KO female mice were subjected to the 30-min ischemic AKI, and the mice were sacrificed 24 h after the surgery. Shown are serum levels of creatinine and BUN (A), renal mRNA expression of *NGAL* (B) and *Il-6* (C), and kidney histology (D, with asterisks indicating tubular damage). n=4 for each group. Scale bars are 50 μ m. Results are presented as the mean \pm SD. *P < 0.05; **, P < 0.01; ***, P < 0.001; the comparisons are labeled.

Supplemental Figure 6



Supplemental Figure 6: Treatment with triclosan protects WT female mice from AKI. (A) Schematic representation of the triclosan (50 mg/kg) regimen. (B-D) Female mice were treated with three daily i.p. doses of triclosan or the vehicle corn oil before being subjected to the AKI surgery. Shown are serum levels of creatinine (B) and BUN (C), and the kidney histology (D, with asterisks indicating tubular damage). n=4 for each group. Scale bars are 50 μ m. Results are presented as the mean \pm SD. *P < 0.05; **, P < 0.01, compare to the corn oil AKI groups.