Supplemental Data

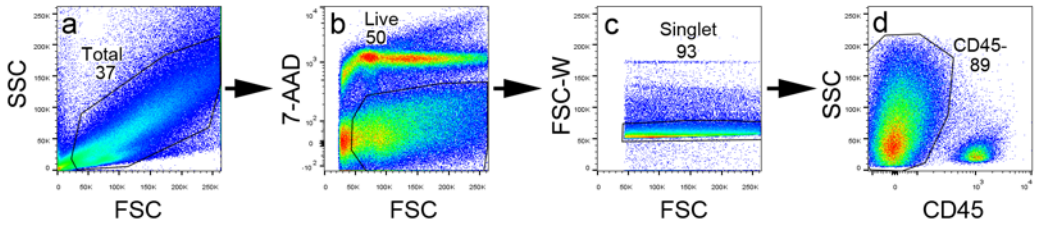

Figure S1. Gating strategy for flow cytometry. Total stained cells (single cell suspensions of whole kidney digests or purified glomeruli) were gated to exclude small particles and calibration beads (a). The live 7-AAD- cells (b) were further gated on singlets (c). The $CD45^+$ leukocytes and the $CD45^-$ non-hematopoietic cells were gated separately (d) for analysis as shown in the manuscript proper. Numbers in the plots indicate gated cells as percent of total in plot. FSC, forward scatter; FSC-W, FSC-width; SSC, side scatter.

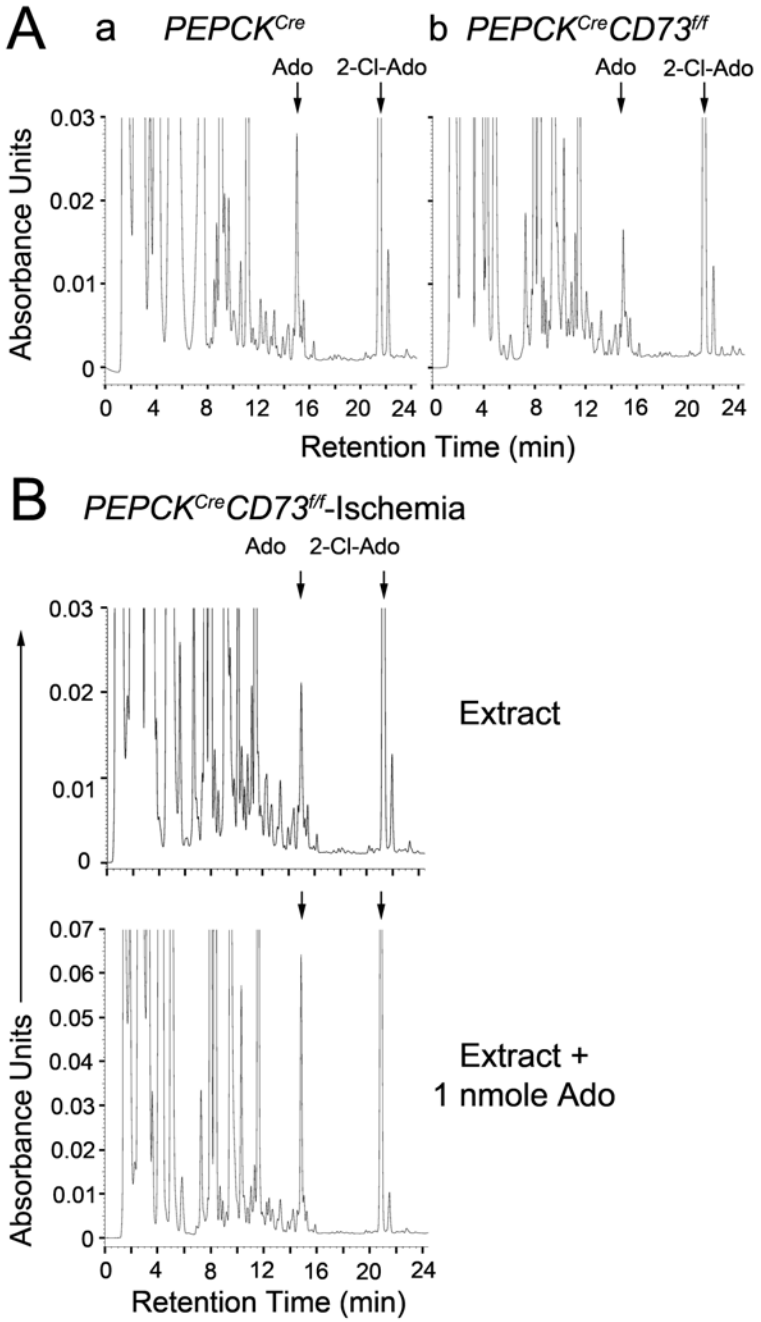

Figure S2. HPLC quantitation of renal tissue adenosine in sham and ischemia-reperfusion-treated WT and CD73-deficient mice. Perchloric acid extracts were analyzed by HPLC as described in Methods. Adenosine (ado) and the internal standard 2-Cl-adenosine (2-CL-Ado) are shown by arrows. (A) Measurements in kidneys from mice subjected to sham surgery. (B) Measurements in kidneys from mice after IRI. Extracts shown in the top panel of B (Extract alone) were spiked with 1 nmole of adenosine (Ado) and rerun (lower panel; Extract + 1 nmol Ado) to confirm the identity of the adenosine peaks. Note that the scale has been expanded to accommodate higher levels in the spiked sample.

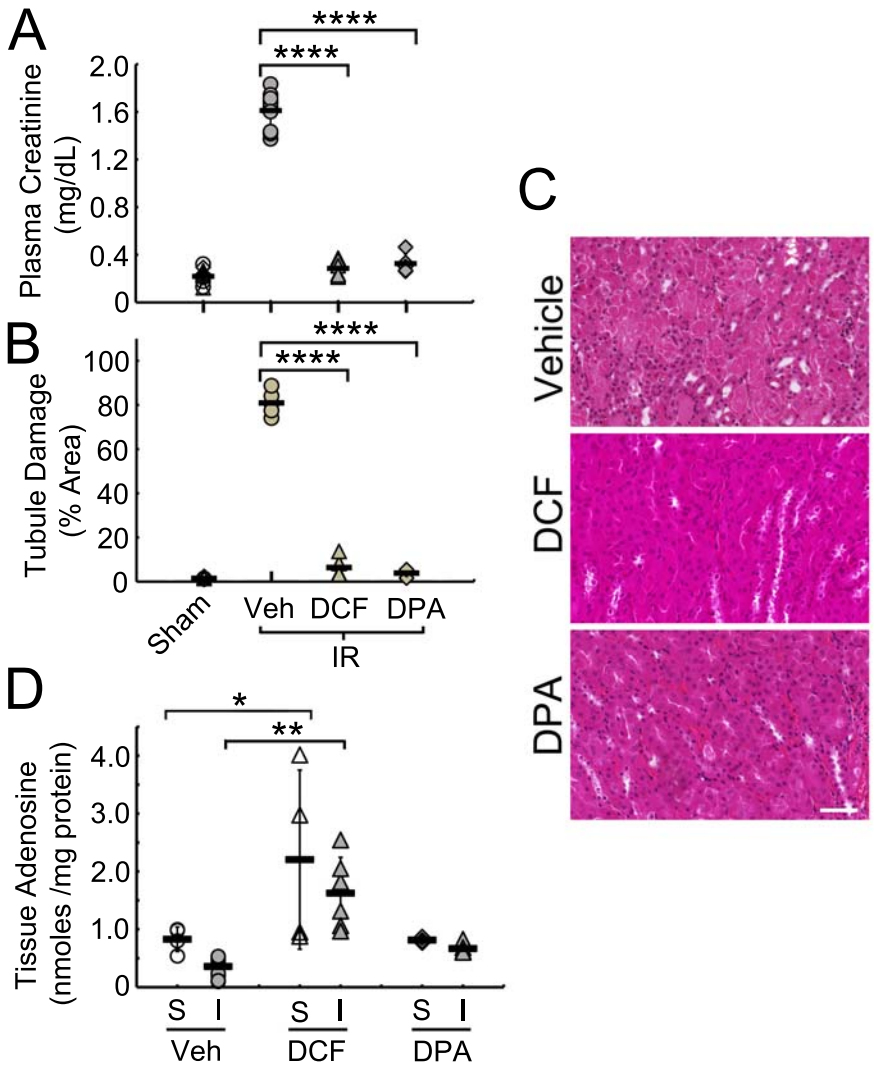

Figure S3. The inhibition of adenosine deaminase and equilibrative nucleoside transporter (ENT) protects WT mice from IRI. WT mice were treated with vehicle (Veh), the adenosine deaminase inhibitor deoxycoformycin (DCF; 4 mg/kg; PBS vehicle), or the ENT inhibitor dipyridamole (DPA; 20 mg/kg; 50% propylene glycol vehicle) as described in Figure 7A and subjected to sham surgery (S) or 26 min of ischemia followed by 24 h of reperfusion (I or IR). Values for PBS- and propylene glycol-treated mice were similar, therefore all vehicle mice were pooled in one group. (A-C) Plasma creatinine levels, tubular damage and H and E staining of kidney sections. Scale bar: 50 µm. (D) Total renal tissue adenosine was measured after sham surgery (S) or ischemia and reperfusion. Experiments were performed 3 times in A-B and twice in C. DCF and DPA protected mice completely from IR. Groups were analyzed for sham versus IRI and drug treatment in 2-way ANOVA. *, $p < 0.05$; **, $p < 0.01$; ****, $p < 0.0001$. Values are given for individual mice and mean ± SD.

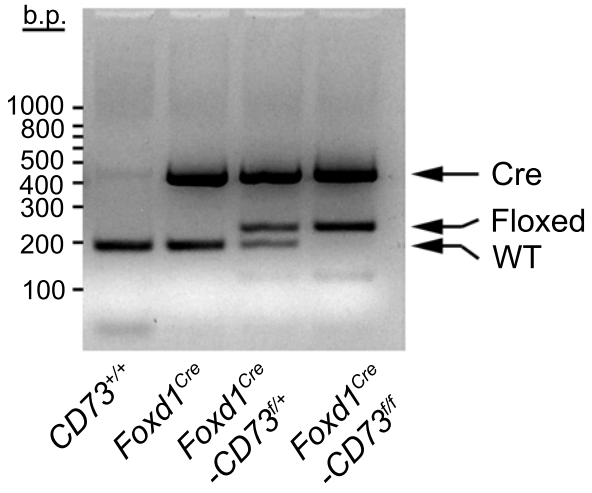

Figure S4: Genotyping of Foxd1^{Cre}CD73$^{f/f}$ mice. The typing of WT and floxed alleles was performed as described.[1] Typing using Cre primers has been described.[2] The gel shows mice with and without Foxd1^{Cre} and mice with heterozygous and homozygous floxed CD73 alleles.

References for Supplemental Data

1. Koszalka P, Ozuyaman B, Huo Y, Zernecke A, Flogel U, Braun N, Buchheiser A, Decking UK, Smith ML, Sevigny J, et al.: Targeted disruption of CD73/ecto-5'-nucleotidase alters thromboregulation and augments vascular inflammatory response. *Circulation Res* 95: 814-821, 2004.
2. Li L, Huang L, Ye H, Song SP, Bajwa A, Lee SJ, Moser EK, Jaworska K, Kinsey GR, Day YJ, et al. Dendritic cells tolerized with adenosine A(2)AR agonist attenuate acute kidney injury. *J Clin Invest* 122: 3931-3942, 2012.