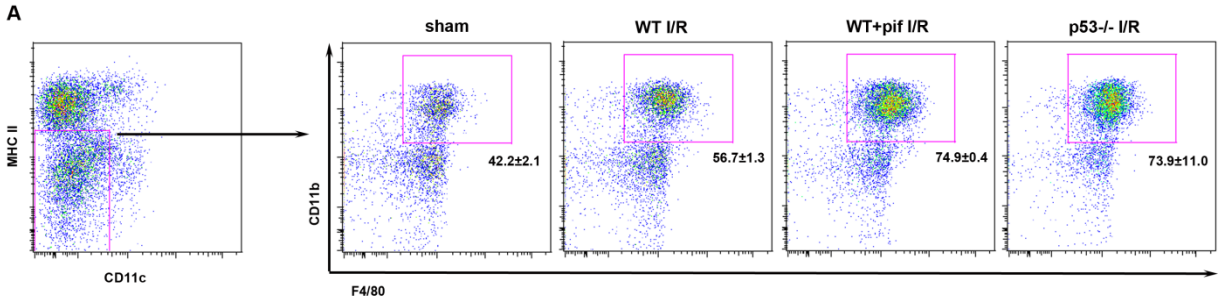
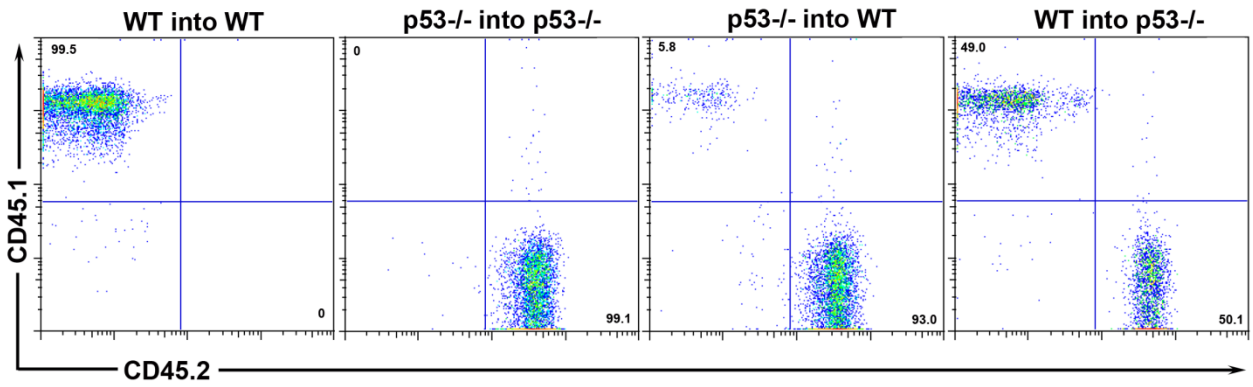


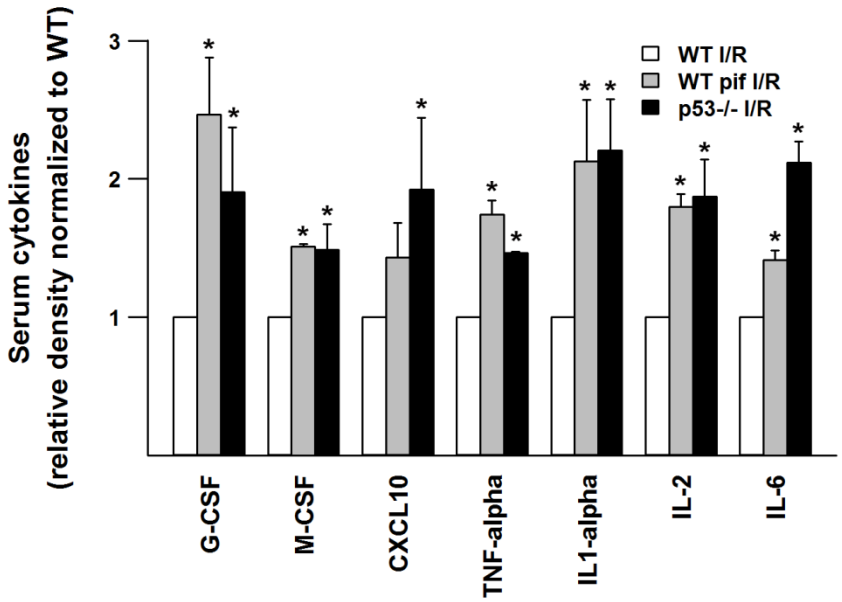
Supplemental Figure 1

Flow cytometric analysis of kidney leukocytes gated on $CD45^+$ cells for the identification of macrophages ($F4/80^+CD11b^+MHCII^-CD11c^-$). Representative dot plots demonstrate increased percentage of macrophages in pifithrin-α-treated WT mice as well as *p53*$^{-/-}$ mice as compared to WT I/R one week after ischemia-reperfusion injury (I/R). n=3 per group, $p<0.05$ for WT I/R versus WT+pifithrin I/R or *p53*$^{-/-}$ I/R.



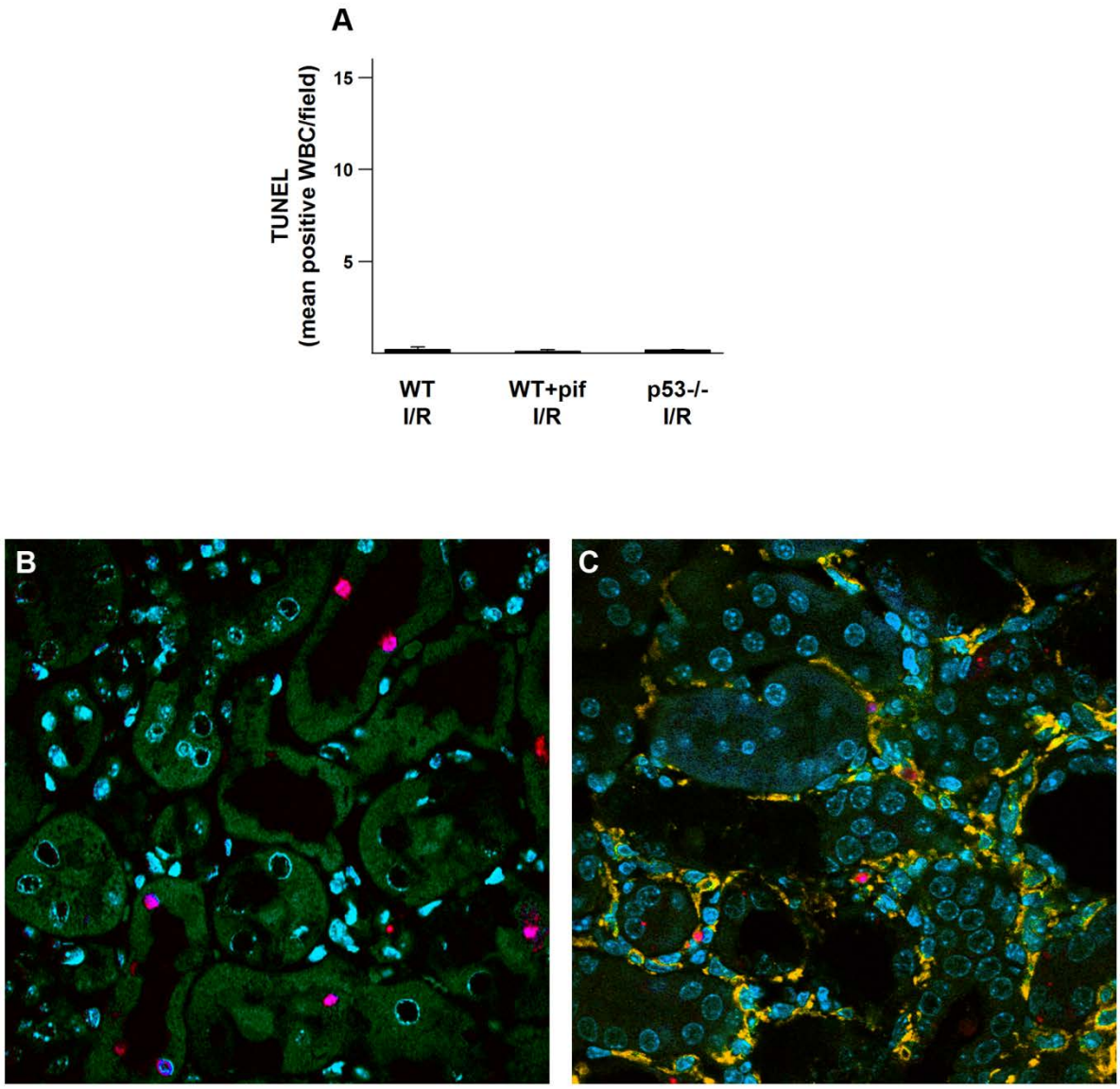
Supplemental Figure 2

Chimeric mice were generated as described in Methods. The degree of chimerism was assessed by flow cytometry using CD45.1 (BoyJ) and CD45.2 (*p53*$^{-/-}$) antibodies. Note that ablation of *p53*$^{-/-}$ bone marrow cells was not successful (WT into *p53*$^{-/-}$).



Supplemental Figure 3

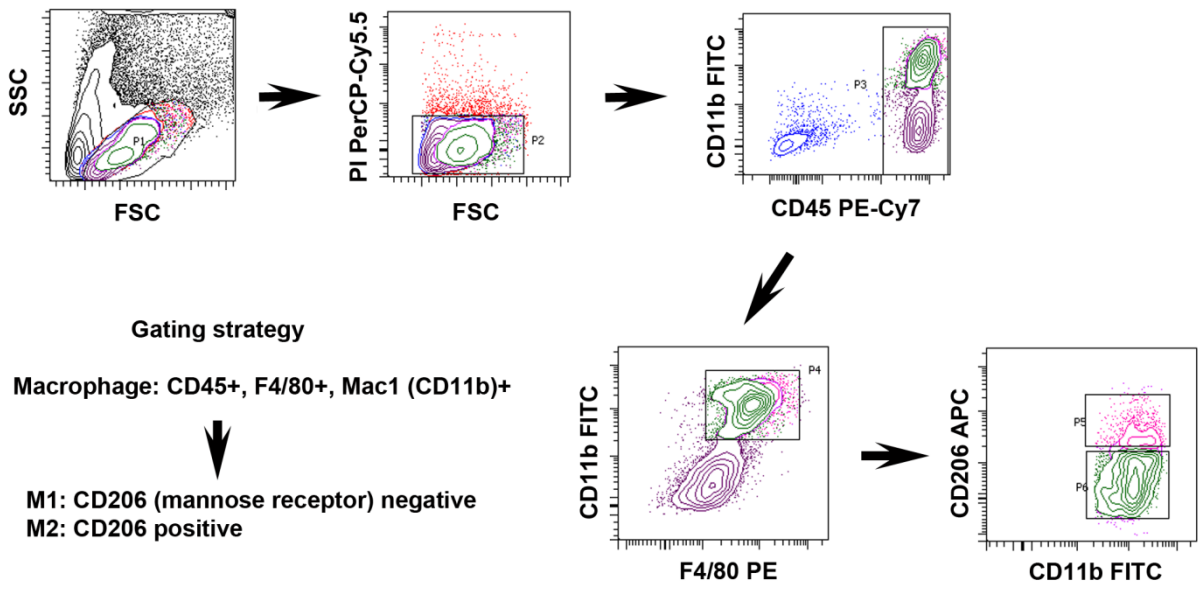
Serum cytokines are shown for WT mice treated with vehicle control or pifithrin α, as well as *p53*$^{-/-}$ mice 24 hours after ischemia-reperfusion injury (I/R). The relative densities were normalized to WT I/R. n=2 per group. *p<0.05 versus WT I/R.



Supplemental Figure 4

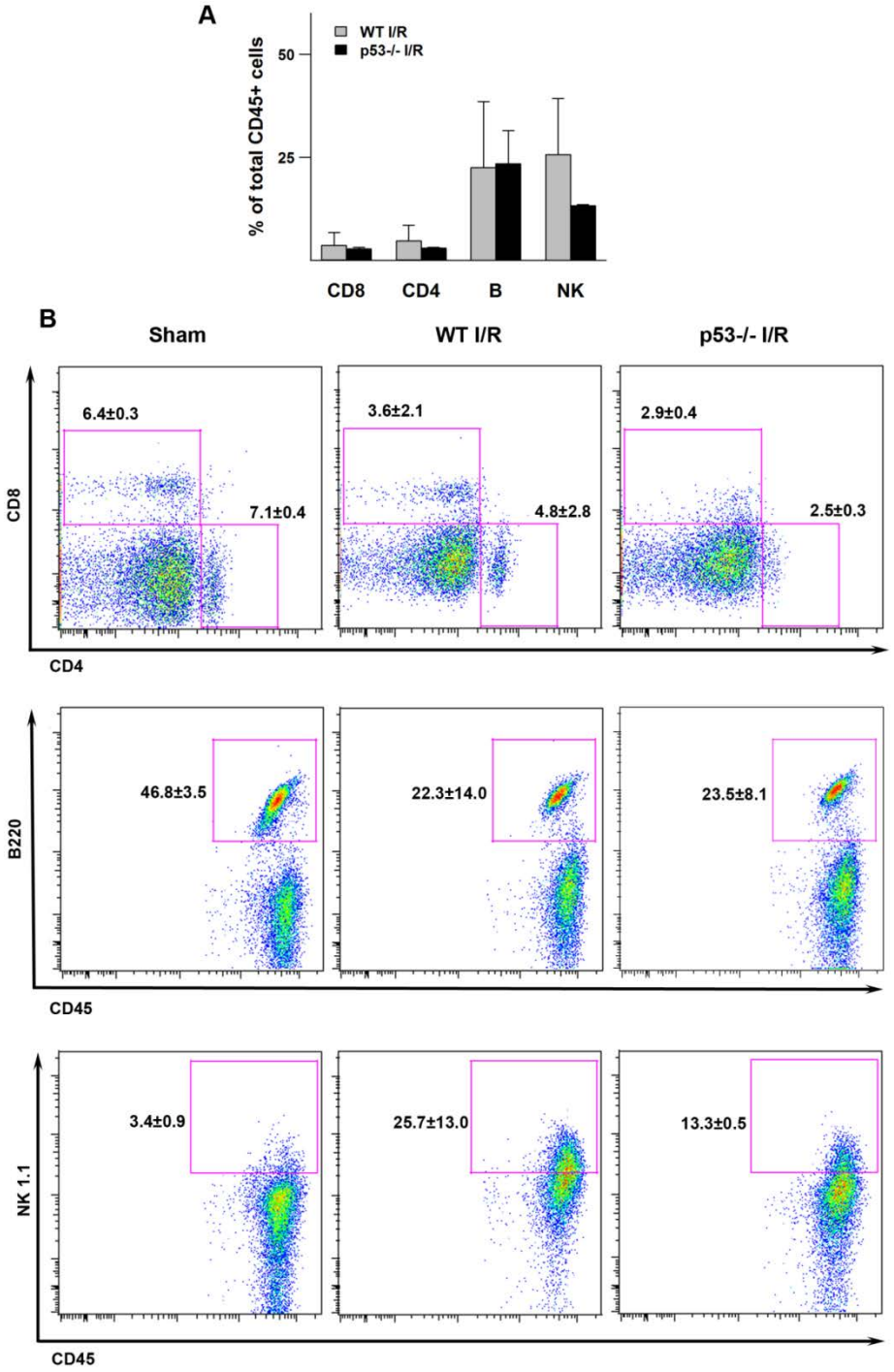
Panel A shows quantitation of TUNEL positive leukocytes 24 hours post IRI. At this time point, leukocyte apoptosis was minimal in all groups.

Panels B and C show representative high resolution TUNEL staining figures to illustrate our ability to differentiate tubular versus leukocyte apoptosis. Twenty-four hours after ischemia-reperfusion injury, TUNEL staining (red) localized predominantly to tubular cells with minimal or no staining in interstitial cells (B). One week after IRI, TUNEL positive staining (red) was readily detected in interstitial cells that were also F4/80 positive (yellow) (C).



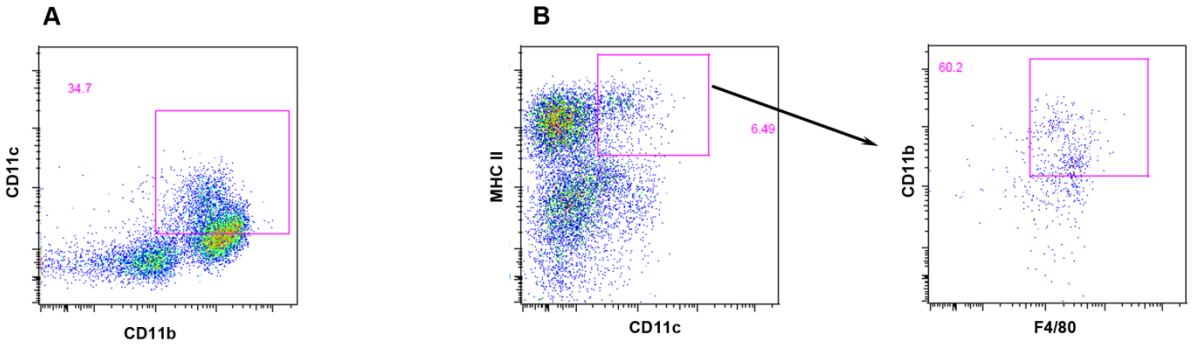
Supplemental Figure 5

Gating strategy used to analyze macrophage phenotype is shown. $CD45^{+}F4/80^{+}$ cells isolated from kidneys were first enriched using MACS (Miltenyi Biotec) as described in Methods.



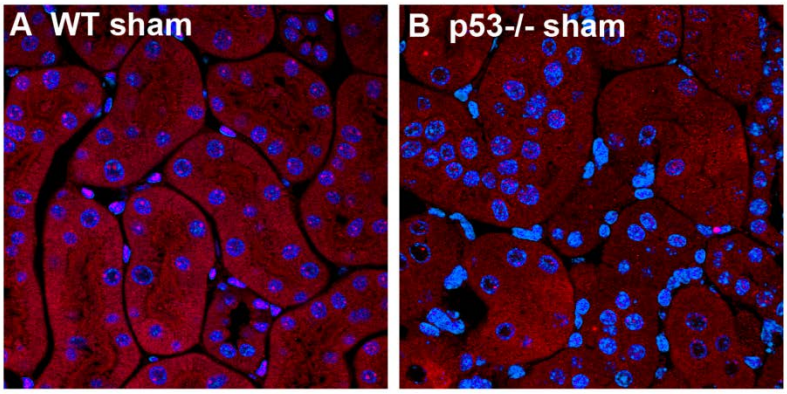
Supplemental Figure 6

Flowcytometric analysis of kidney leukocytes isolated from sham mice or 6 days after ischemia-reperfusion injury. $CD45^+$ kidney cells were first enriched using MACS (Miltenyi). Panel A shows the population of leukocyte subsets between WT and *p53*$^{-/-}$ after ischemia-reperfusion injury gated on $CD45^+$ cells. Panel B shows representative dot plots of $CD4^+$ T and $CD8^+$ T cells, B cells, and NK cells ($CD3^-NK1.1^+$) respectively.



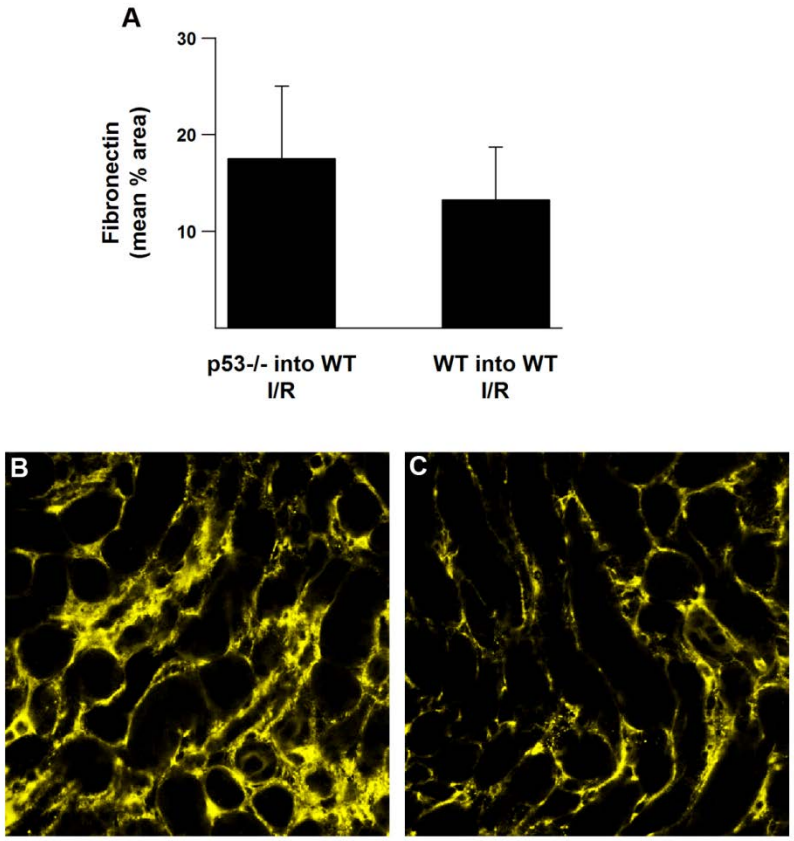
Supplemental Figure 7

Representative dot plots of kidney leukocytes gating on $CD45^+$ cells. Panels A and B show the significant overlap among conventional macrophage markers (CD11b and F4/80) and dendritic cell markers (MHC II and CD11c). MHC II^+CD11c^- cells in panel B left are primarily $B220^+$ B cells. Dot plots for the expression of CD11b and F4/80 gated on $CD45^+$MHC II^-CD11c^- are shown in supplemental figure 1.



Supplemental Figure 8

Representative images of KLF4 staining (red; DAPI, bule) at baseline are shown for WT and *p53*$^{-/-}$ mice.



Supplemental Figure 9

Representative images and quantitation of fibronectin staining in *p53*$^{-/-}$ into WT and WT into WT bone marrow chimeric mice 8 weeks after ischemia-reperfusion injury (I/R). *p53*$^{-/-}$ into WT chimeras (B) trended to have more fibronectin deposition as compared to WT into WT chimeras (C, p=0.3). Note the degree of fibronectin deposition in chimeras is higher than that of non-chimeric mice (Figure 10 Panel J).