

Figure S1. Low or no integration of USCs into the IR-injured kidney tissue.

(A) At 24 h after PKH26-positive USC treatment, immunofluorescence staining showed that PKH26-positive cells were mostly trapped in the lungs. A few positive cells were distributed in the liver, but no injected cells were found in the spleen or damaged kidney. Scale bars = 50 μ m. (B) Immunohistochemical staining of human nuclear antigen (hNA) in kidney sections on day 7 after injection of USCs via the renal artery (positive control, red arrows) or dorsal vein of the penis. Scale bars = 200 μ m in a,b; 100 μ m in c,d.

Figure S2. Expression levels of *miR-146a-5p* and *IRAK1* in H/R-induced oxidative stress model after treatment with different types of medium.

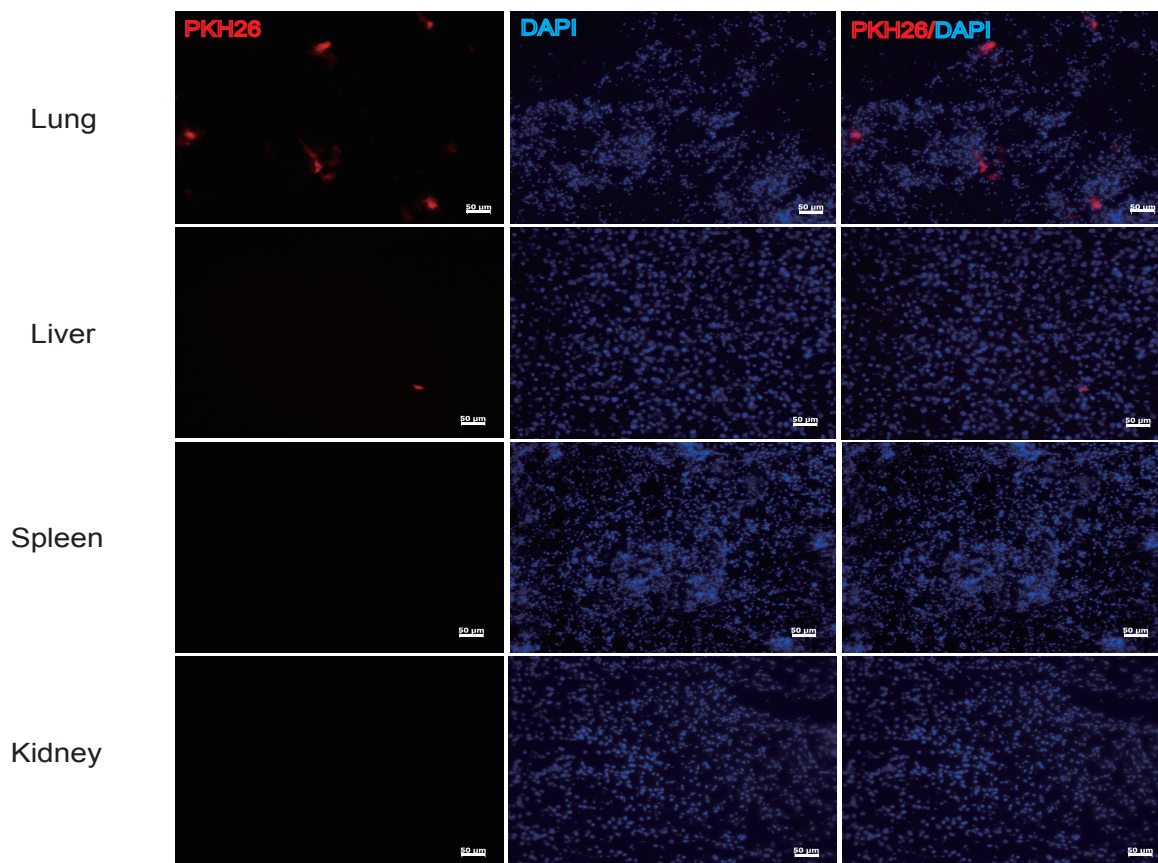
(A) Schematic diagram of H/R-induced injury in HK2 cells treated with HK2 medium, fresh USC medium, or USC conditioned medium (CM). (B) SOD and MDA analysis in H/R-stimulated HK2 cells treated with or without USC CM ($P=0.049$ for MDA, $P=0.0017$ for SOD). Each experiment was repeated three times. Data represent the mean \pm SEM.

* $P<0.05$, ** $P<0.01$, *** $P<0.001$.

Figure S3. Nuclear translocation of NF- κ B p65 after *IRAK1* knock down in HK2 cells subjected to H/R injury in vitro.

(A) Western blot analysis of the protein levels of IRAK1 after treatment with three different siRNAs. GAPDH was used as the loading control. (B) Western blot analysis of the protein levels of IRAK1 in cells treated with siRNA(2+3). GAPDH was used as the loading control. (C) Western blot analysis of the nuclear translocation of NF- κ B p65 in siIRAK1-treated and control cells. GAPDH and H3 were used as the loading controls, respectively.

A



B

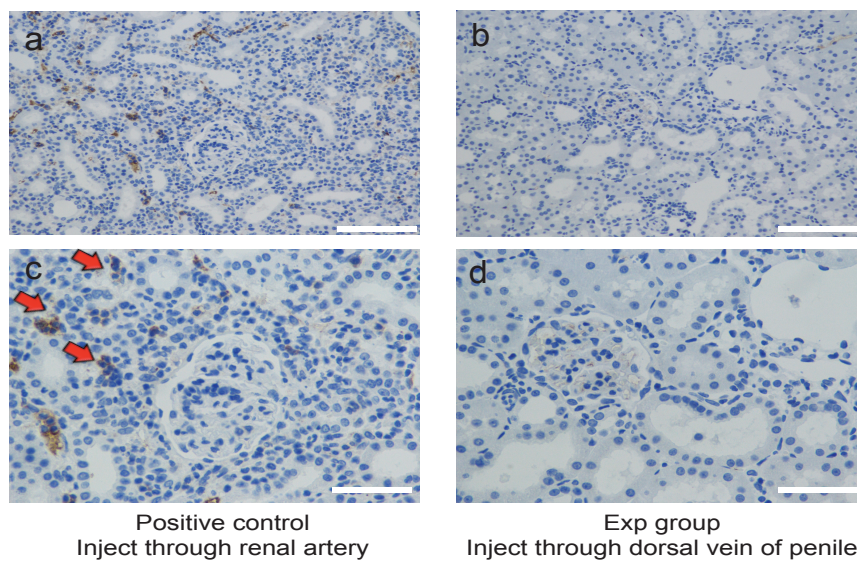


Figure S1

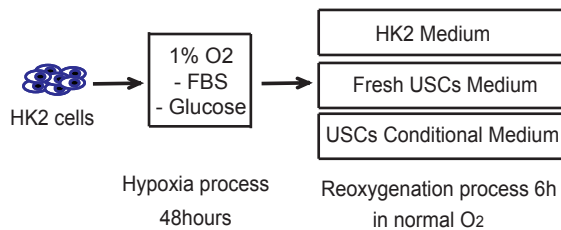
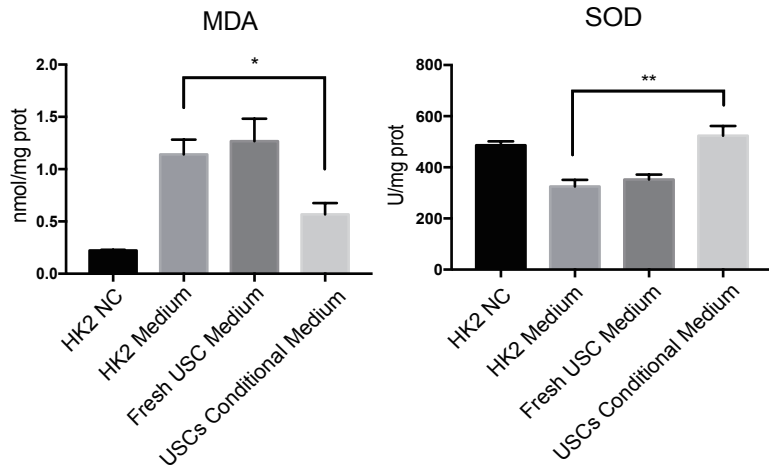
A**B**

Figure S2

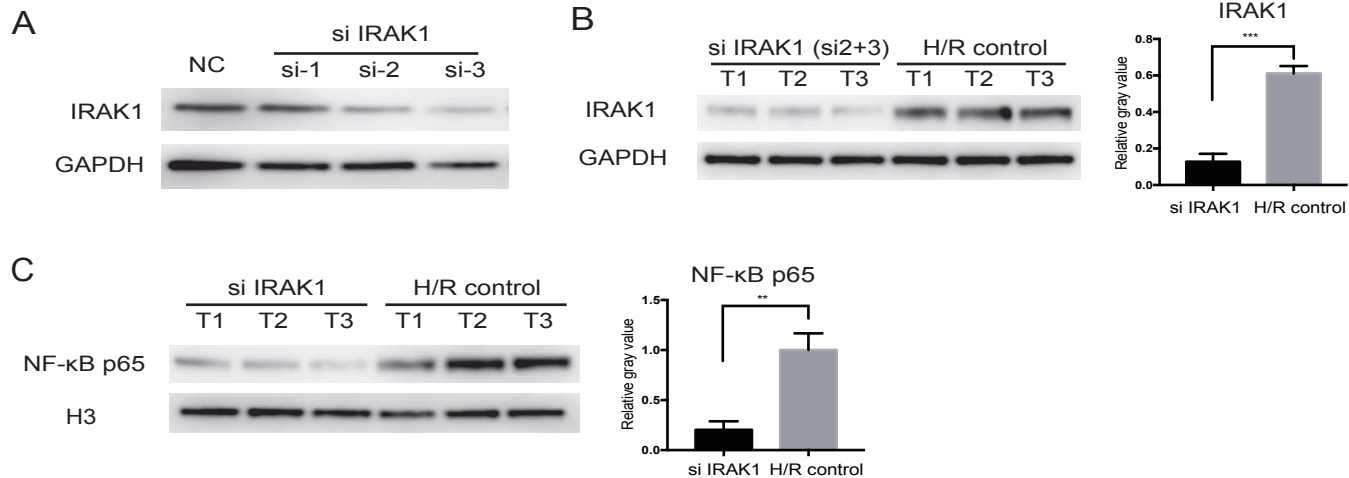


Figure S3