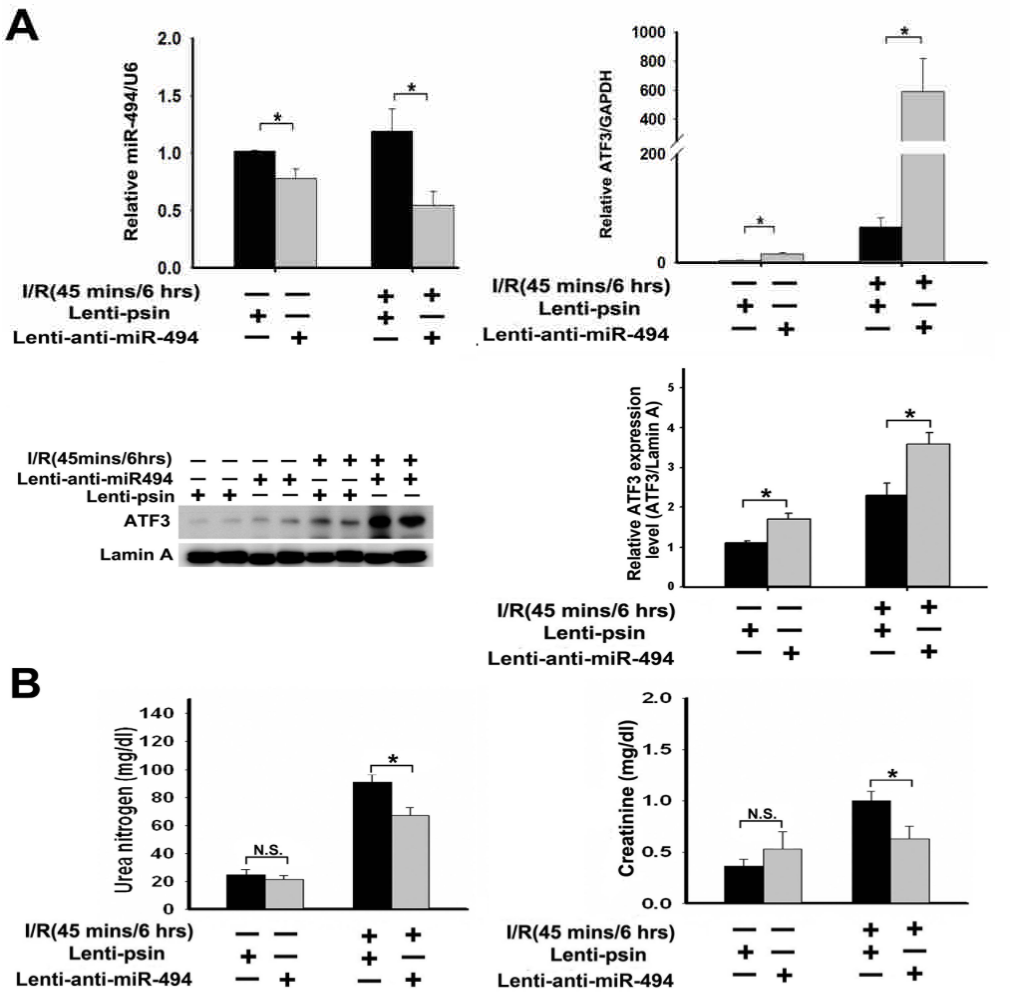
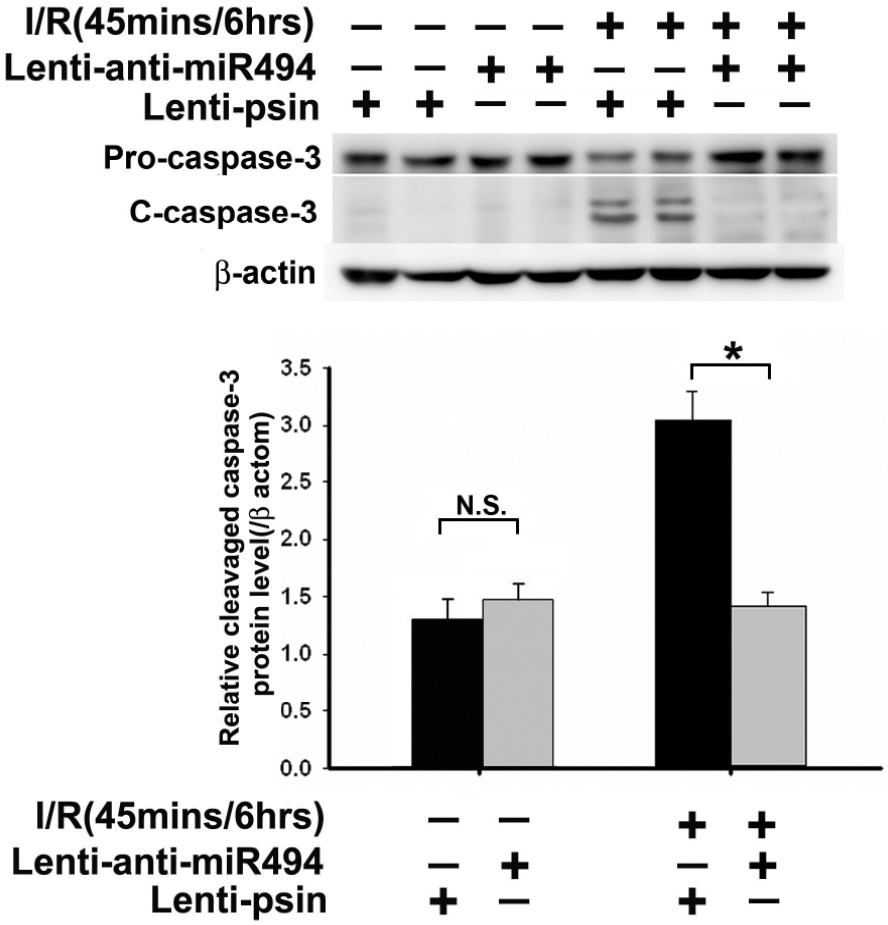
## Supplemental Figures and Figure Legends

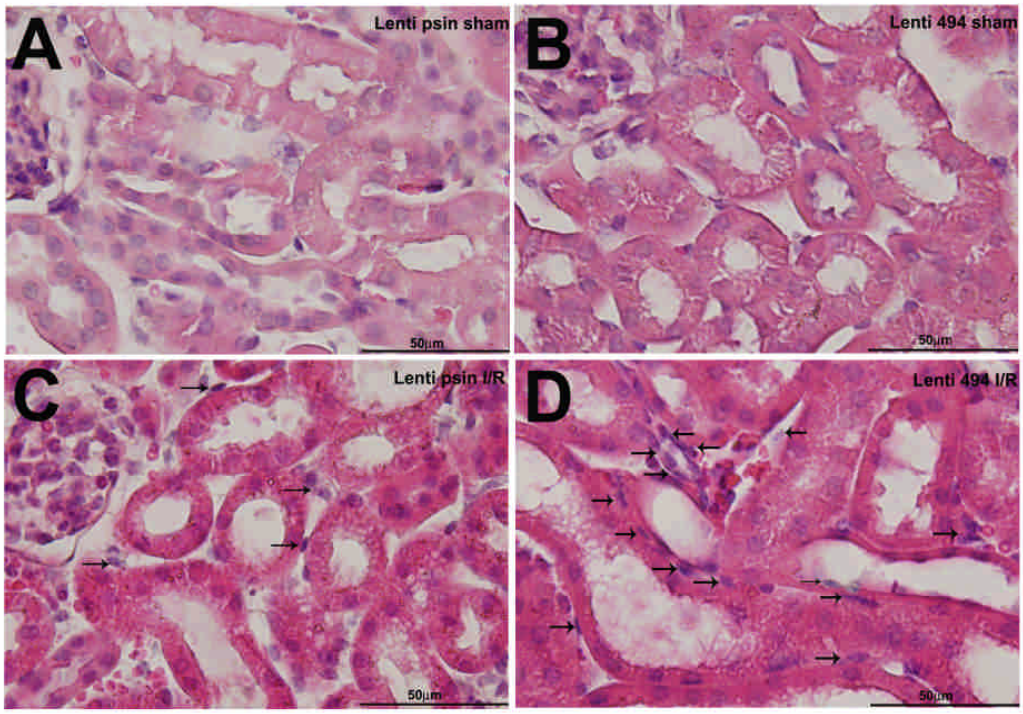
**Supplemental Figure S1.** Rescue of renal ischemia-reperfusion (I/R) injury in WT mice by lenti virus (Lenti)-mediated gene transfer of antisense-miR-494(anit-miR-494) (A) Lenti-anti-miR-494 mediated expression of ATF3 in the kidney. An amount of 25 μl Lenti-psin or Lenti-anti-miR-494 (1 x $10^{10}$ viral particles) was perfused into the kidney of WT mice through the renal artery as described in Methods. Two weeks after gene transfer, mice were subjected to shame or I/R treatment. Micro-RNAs were reverse transcribed utilizing miR-494 and U6 RNA-specific primers, and real time PCR was performed as described under "Methods". mRNA or protein level of ATF3 was assayed by real time PCR or western blotting separately. (B) Effect of Lenti-mediated gene transfer of antisense-miR-494 on I/R-induced renal function in mice. Two weeks after gene transfer, kidney function (blood urea nitrogen and creatinine levels) were monitored in mice 6 hours after sham operation or I/R injury. Data are means ± SEM (n=5 in each group). ** P < 0.05 Lenti-psin vs Lenti-anti-miR-494.



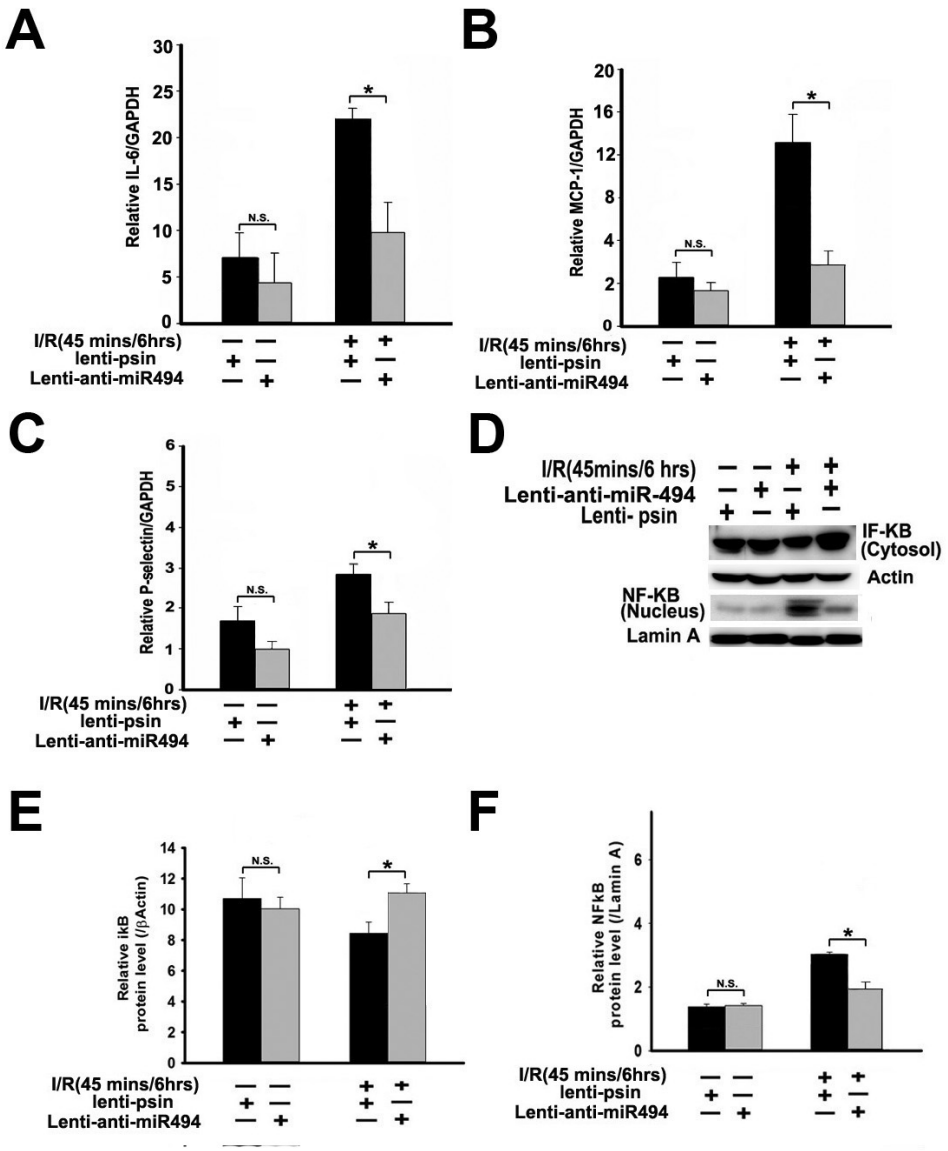
**Supplemental Figure S2.** Rescue of I/R-induced apoptosis by Lenti-anti-miR-494 transduction in the kidney. Activation of caspase-3. Kidney lysates from Lenti-psin or Lentianti-miR-494-tranduced animals subjected to sham operation or I/R injury, respectively, were probed with a caspase-3 antibody which can specific against the cleaved, active form of caspase-3 (C-caspase-3) and non-cleaved active form of caspase-3 (Pro-caspase-3).



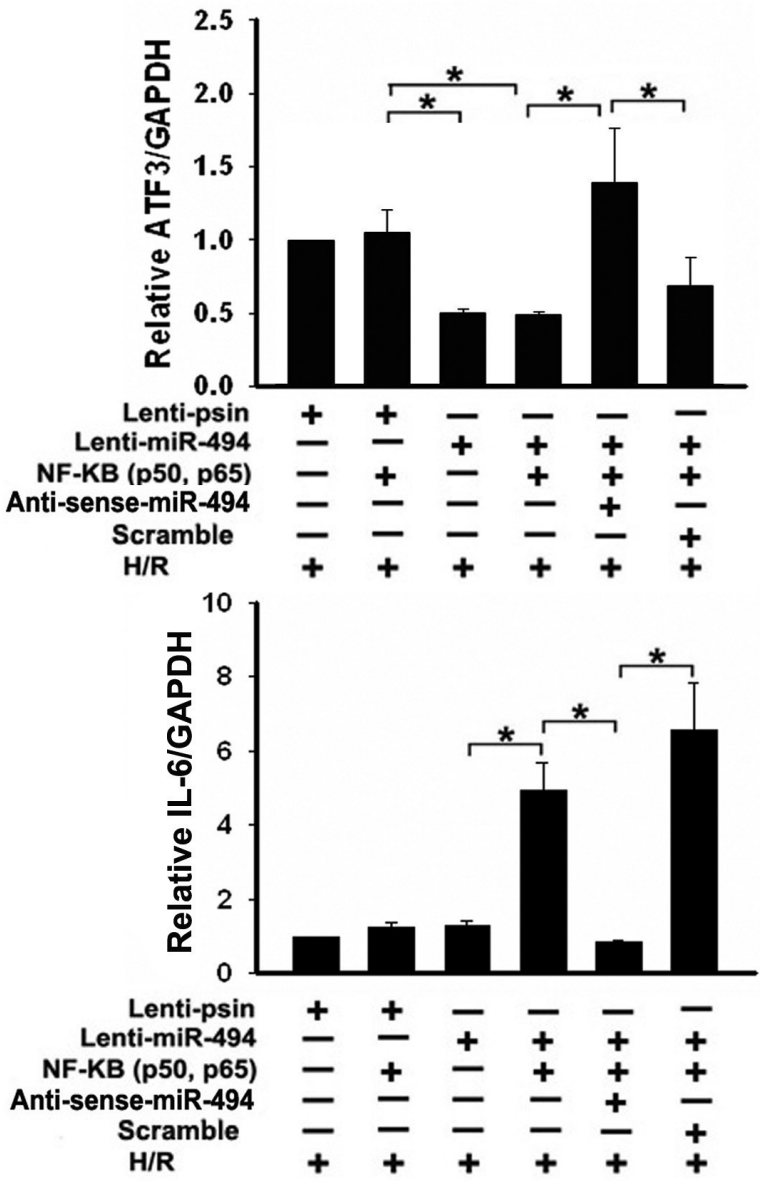
**Supplemental Figure S3.** Lenti-494 transduction increases polymorphonuclear leukocytes infiltration in kidney after I/R. Sham-operated (A and B) and I/R-injured (C and D) Lenti-psin (A and C) and Lenti-anti-miR-494 transduction (B and D) mice. A significant number of infiltrating polymorphonuclear leukocytes (black arrows) accumulated in the Lenti-494 transduction kidney (D), with fewer in the Lenti-psin (C) kidney.



**Supplemental Figure S4.** Reduction of I/R-induced inflammatory related gene transcription by Lenti-anti-miR-494 transduction in the kidney. Quantitative reverse transcriptase (RT)-PCR analysis of IL-6 (A), MCP-1 (B), and P-selectin (C) from renal cDNA derived from mice infused with or without anti-miR-494, and then subjected to sham or I/R experiments. RT-PCR was conducted 6 hrs after ischemia. Expression was normalized to that of GAPDH (glyceraldehyde 3-phosphate dehydrogenase). n = 5 mice in each group, *P < 0.05,**P < 0.01, N.S. denotes not significant statistically. (B) NF-κB activation, and IF-κB degradation. Nuclear or cytosolic extracts were probed with anti-NF-κB or anti-IF-κB antibody, respectively, to quantify protein levels in these subcellular compartments. Anti-β-actin served as a cytosolic loading control. Lamin-A served as a nucleic loading control. *P＜0.05.



**Supplemental Figure S5.** MiR-494 promoted NF-κB-induced IL-6 expression associated with ATF3 by q-PCR assay. The represent results were acquired from three independent experiments. *P < 0.05.



**Supplemental Figure S6.** Potential targets for miR494 by Ingenuity systems pathway analysis. ATF3: activating transcription factor 3. BCL2L11: BCL2-like 11 (apoptosis facilitator). EIF2AK2: eukaryotic translation initiation factor 2-alpha kinase 2. GFPT1: glutamine--fructose-6-phosphate transaminase 1. IGF1R: insulin-like growth factor 1 receptor. SERPINB3: serpin peptidase inhibitor, clade B (ovalbumin), member 3. SIRT3: sirtuin 3. TNFSF10: tumor necrosis factor (ligand) superfamily, member 10. XIAP: X-linked inhibitor of apoptosis. BIRC5: baculoviral IAP repeat containing 5. BIRC5: baculoviral IAP repeat containing 5. ABCC5: ATP-binding cassette, sub-family C (CFTR/MRP), member 5. ENC1: ectodermal-neural cortex 1. NCL: nucleolin. PTEN: phosphatase and tensin homolog.