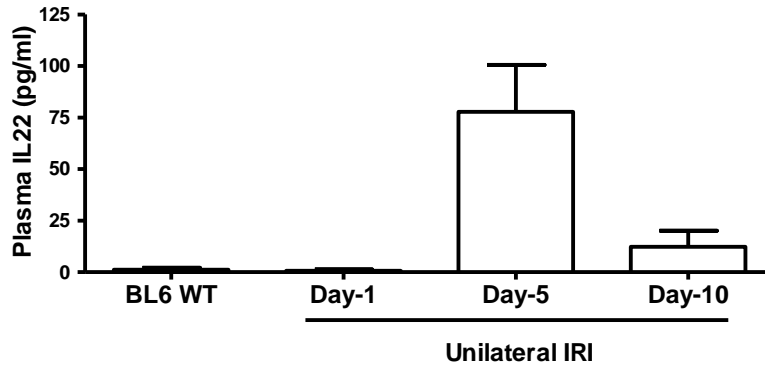


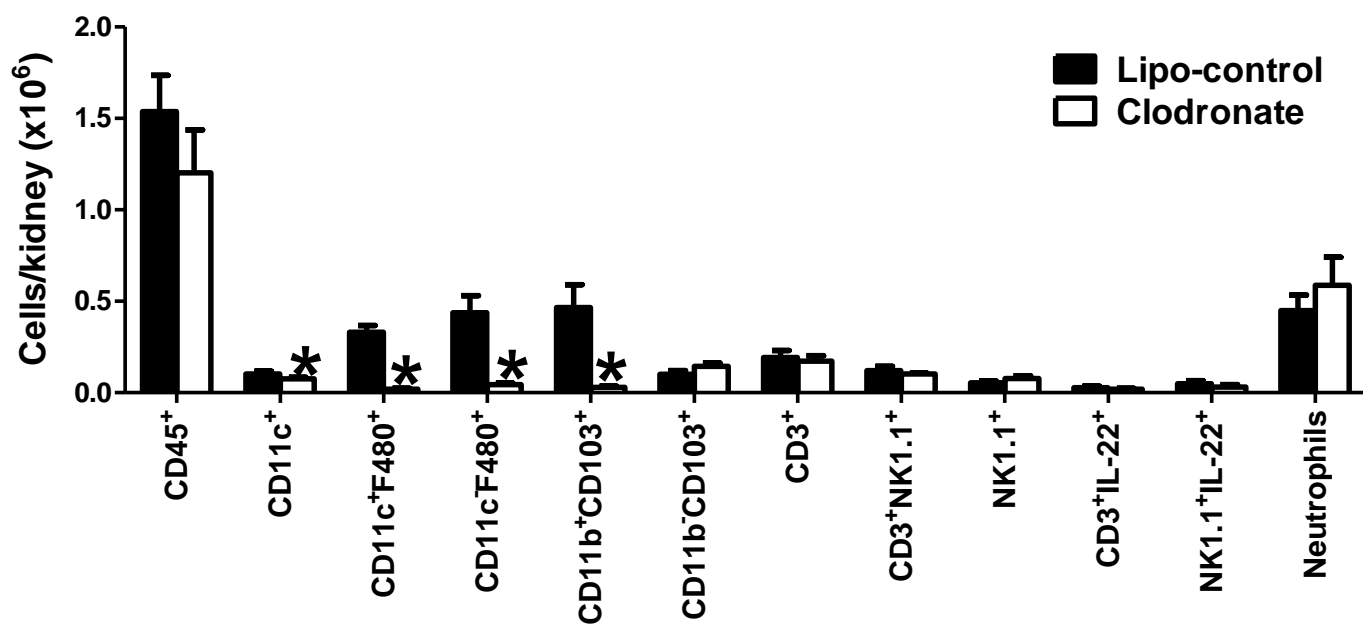
Supplementary Figure 1

A



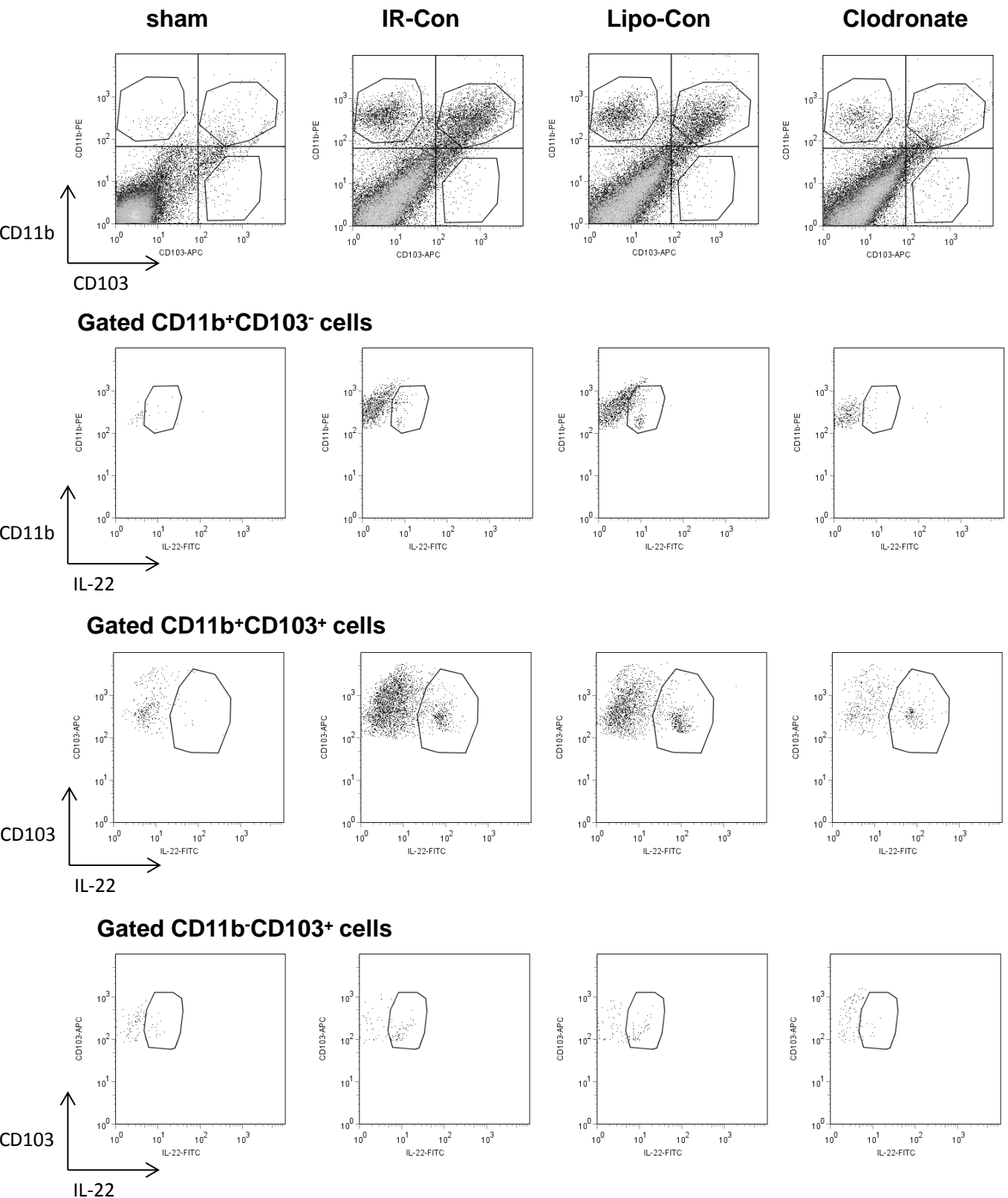
Supplementary figure 1. IL-22 expression in plasma and intrarenal AhR expression. (A) IL-22 is expressed systemically in acute ischemic renal injury. We observed transiently increased, with highest levels on day5, expression of plasma IL-22 measured by ELISA. Data are means \pm SEM. from five mice in each group. ** $P < 0.01$ vs. Control and *** $P < 0.001$ vs. Control

Supplementary Figure 2



Supplementary figure 2. Clodronate-liposome treatment do not affect number of T cells, NK cells and neutrophils in the ischemic kidney. We analysed effect of clodronate-liposome treatment on number of T cells, NK cells and neutrophils along with mononuclear phagocytes in ischemic kidney five days after induction of injury. Clodronate-liposome reduced CD11c⁺, CD11b⁺, CD103⁺ and F4/80⁺ cells but had no effect on CD3⁺, NK1.1⁺ and 7/4+Ly6G⁺ cells. Data are means ± SEM. from five mice in each group. *P< 0.05 vs. Lipo-control.

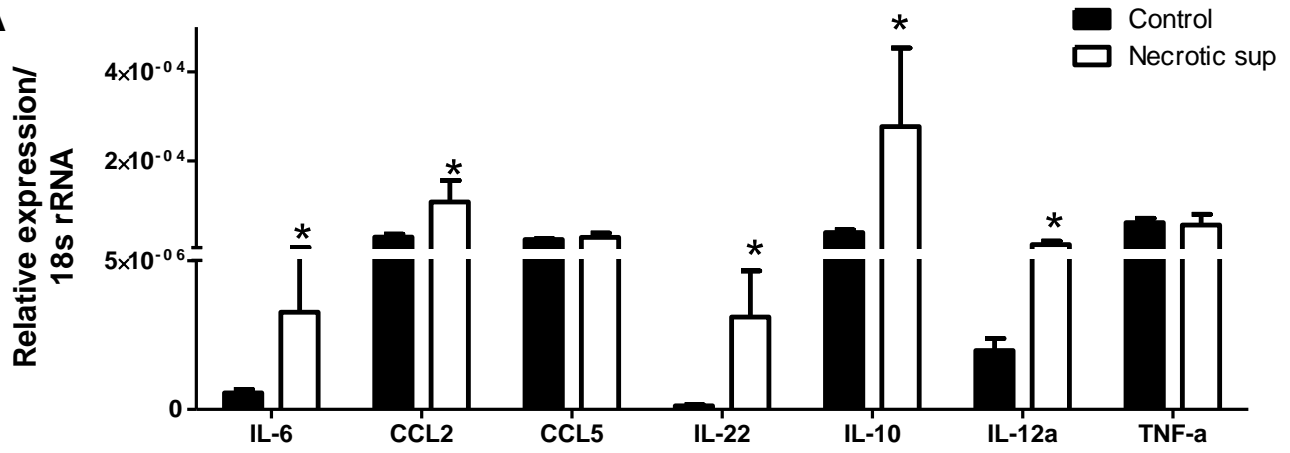
Supplementary Figure 3



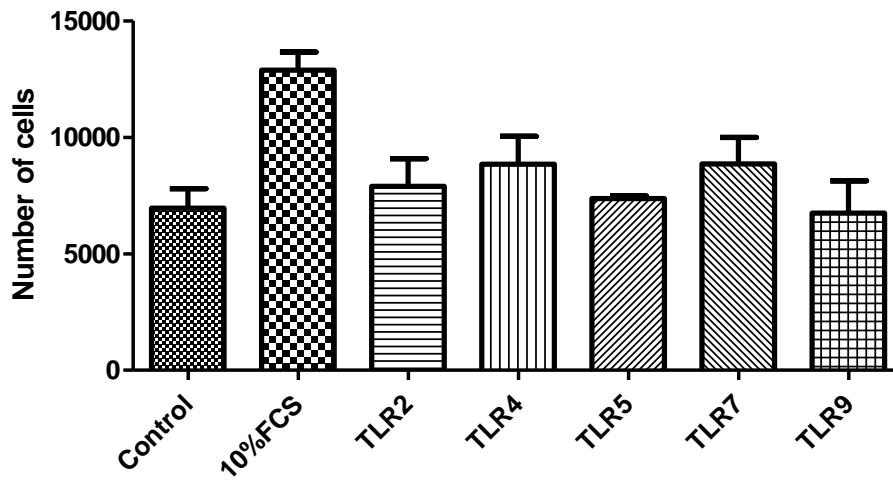
Supplementary figure 3. Clodronate-liposome treatment reduced IL-22 producing renal phagocytes. We analysed effect of clodronate-liposome treatment on IL-22 expressing mononuclear phagocytes in ischemic kidney five days after induction of injury. Clodronate-liposome significantly reduced CD11c⁺, CD11b⁺, CD103⁺ and F4/80⁺ cells expressing IL-22.

Supplementary Figure 4

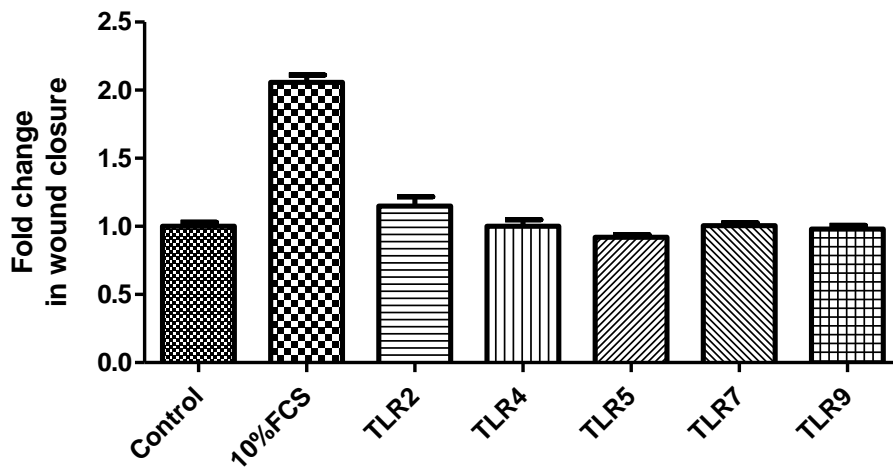
A



B



C



Supplementary figure 4. (A) Necrotic supernatant induced many factors in primary bone marrow derived dendritic cells. (B) TLR ligands stimulation of primary tubular epithelial cells, did not induced proliferation. (C) TLR ligands stimulation did not affect wound re-epithelisation on monolayer of primary tubular epithelial cells.