

# Pretreatment with rituximab prevents subsequent ischemia-reperfusion injury in mice kidney

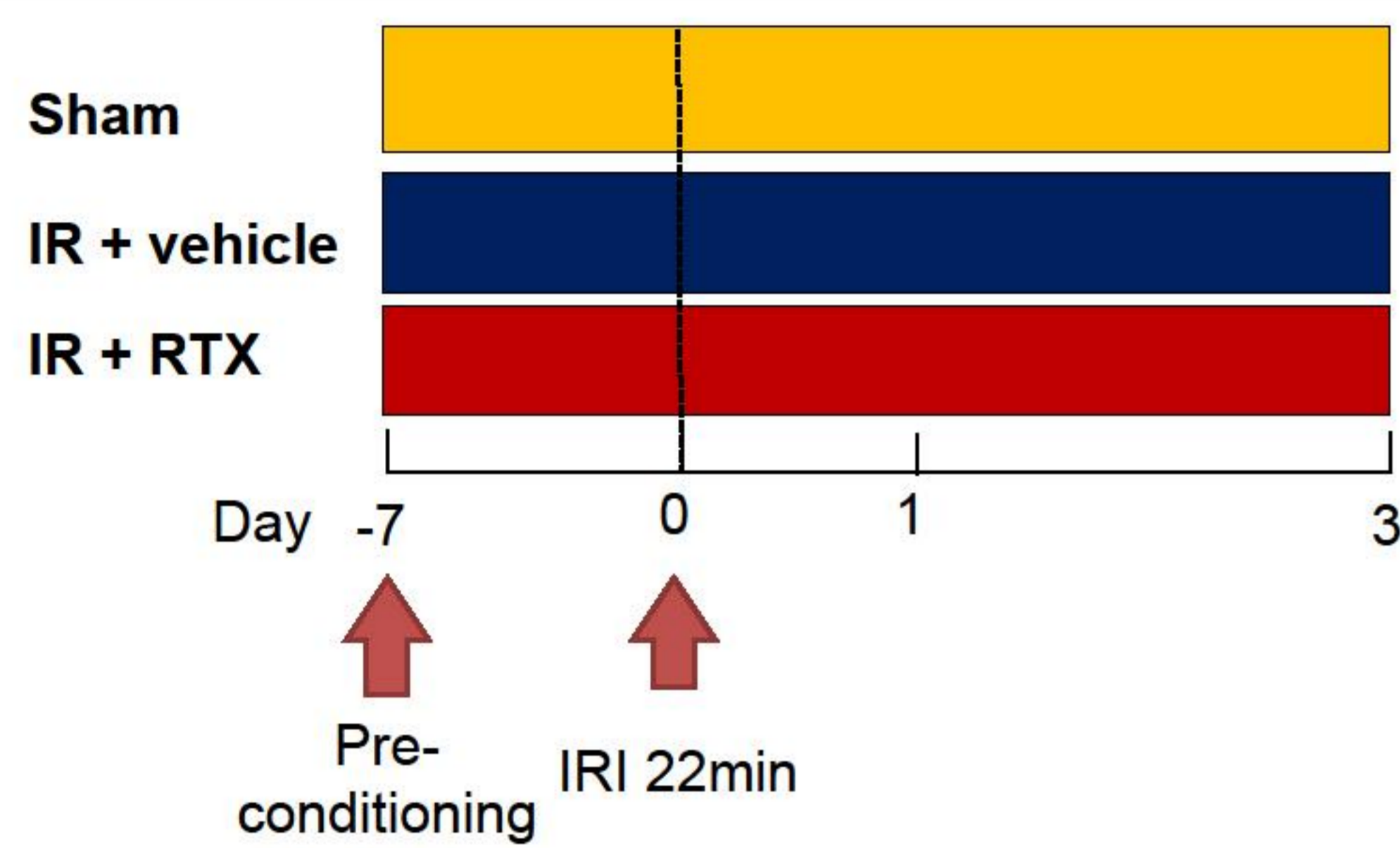
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## Introduction

Ischemia-reperfusion injury (IRI) is unavoidable event in renal transplantation, causing the delayed graft function and increased immunogenicity. We investigated whether rituximab is renoprotective in a mouse model of IRI, and whether potential mechanism is related with modulation of renal inflammatory infiltration.

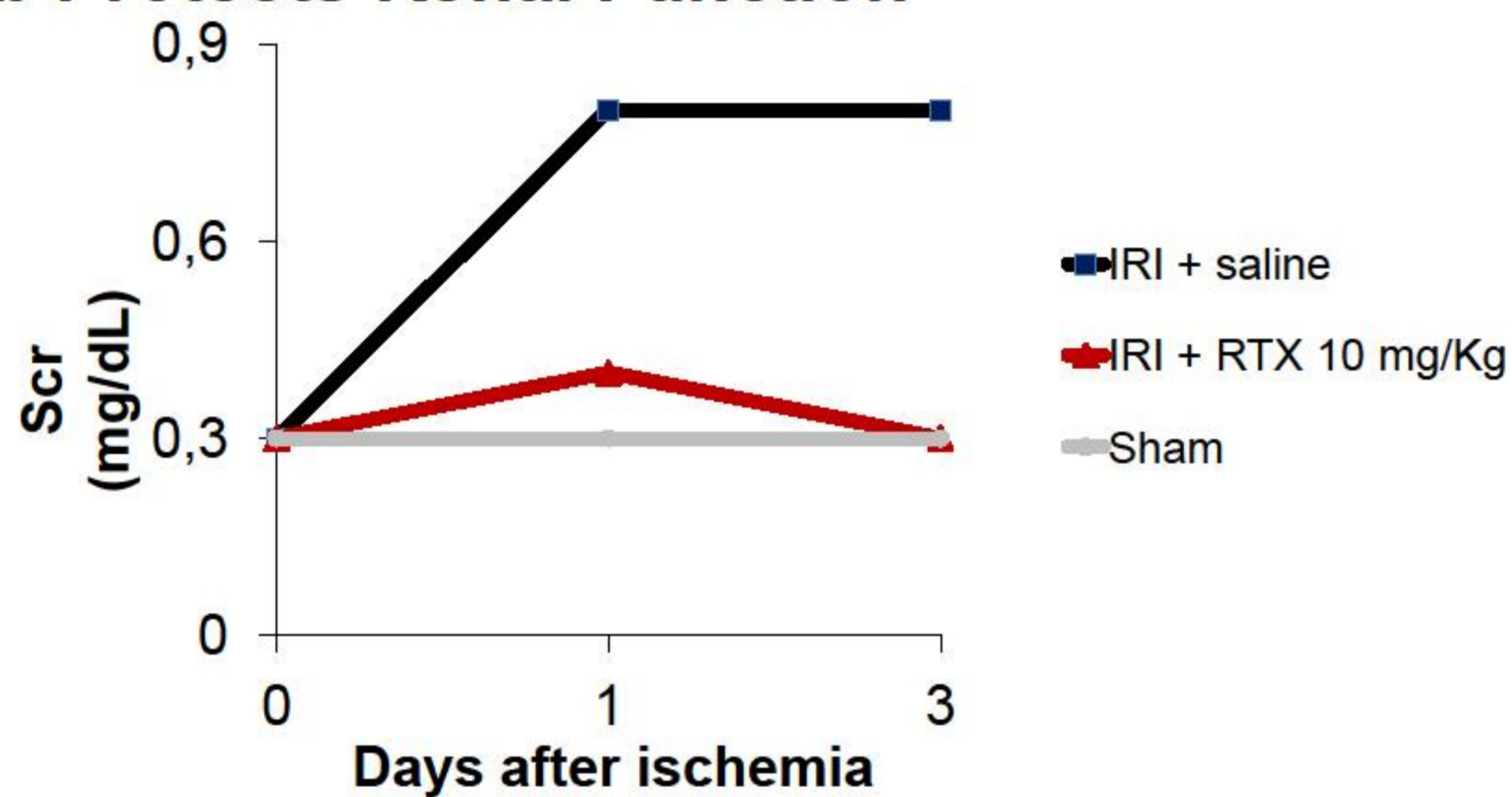
## Methods



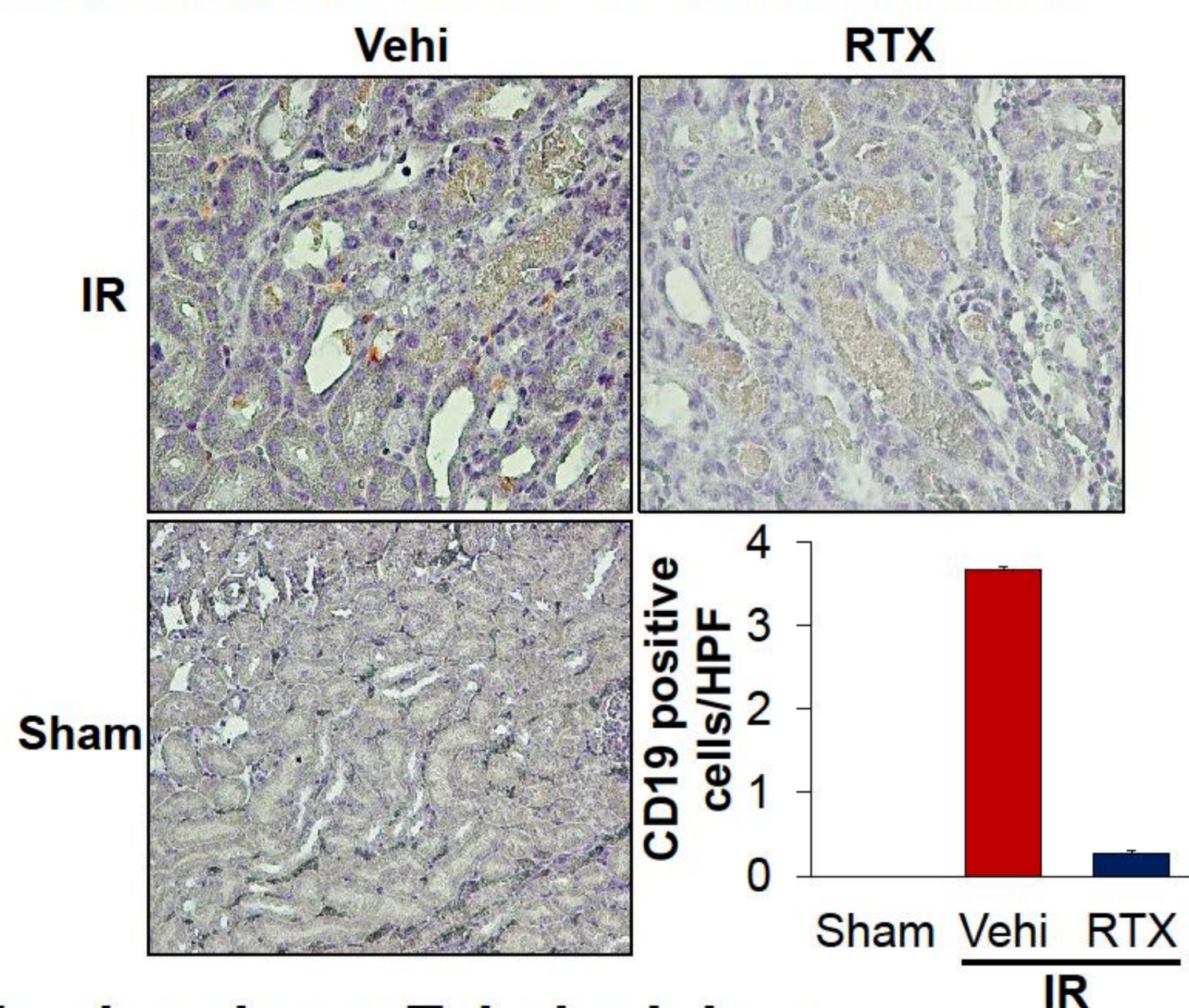
Rituximab (10 mg/kg) was administered to male C57BL/6 mice 7 days before IRI, and mice were killed at 72 hours after IRI

## Results

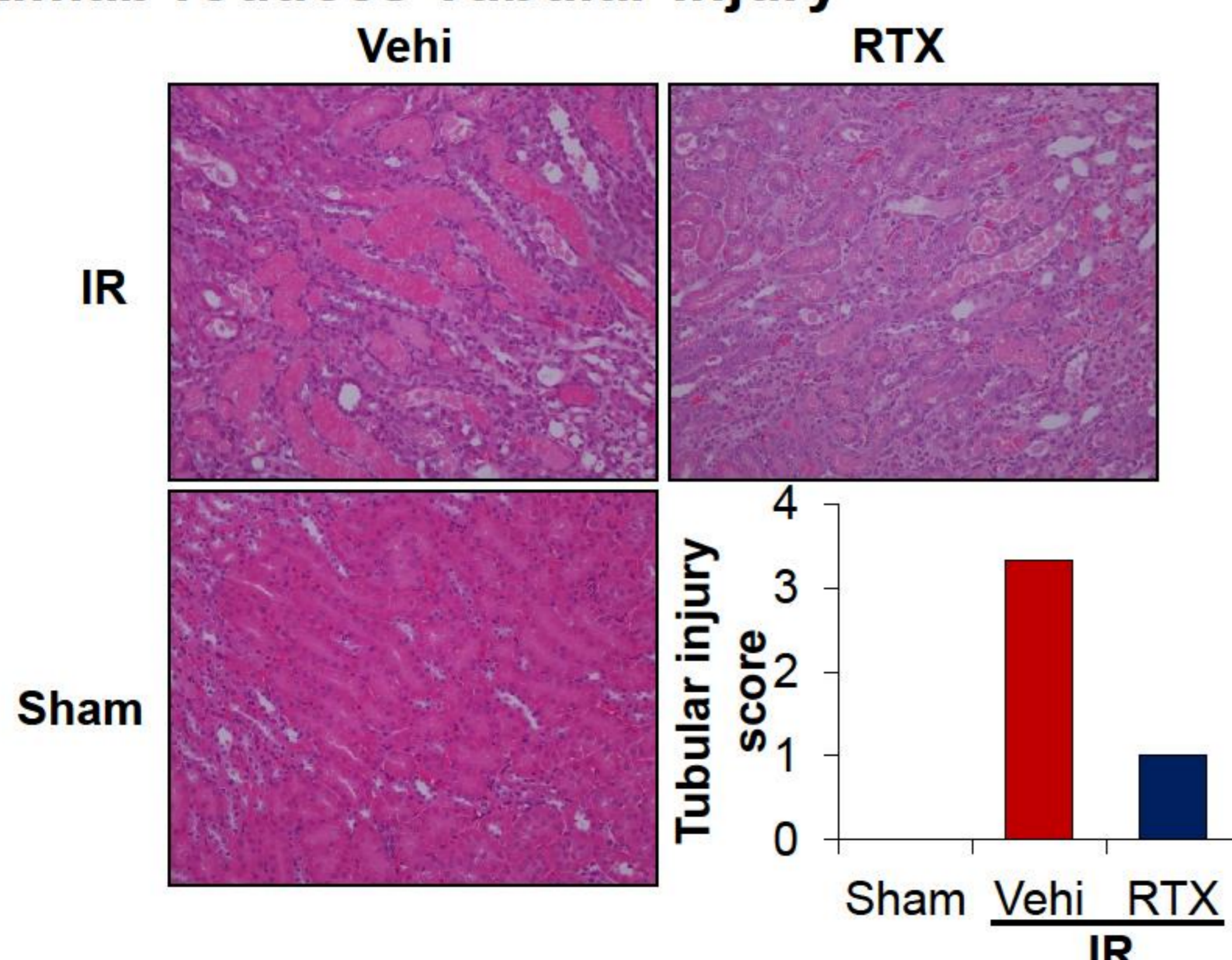
### Rituximab Protects Renal Function



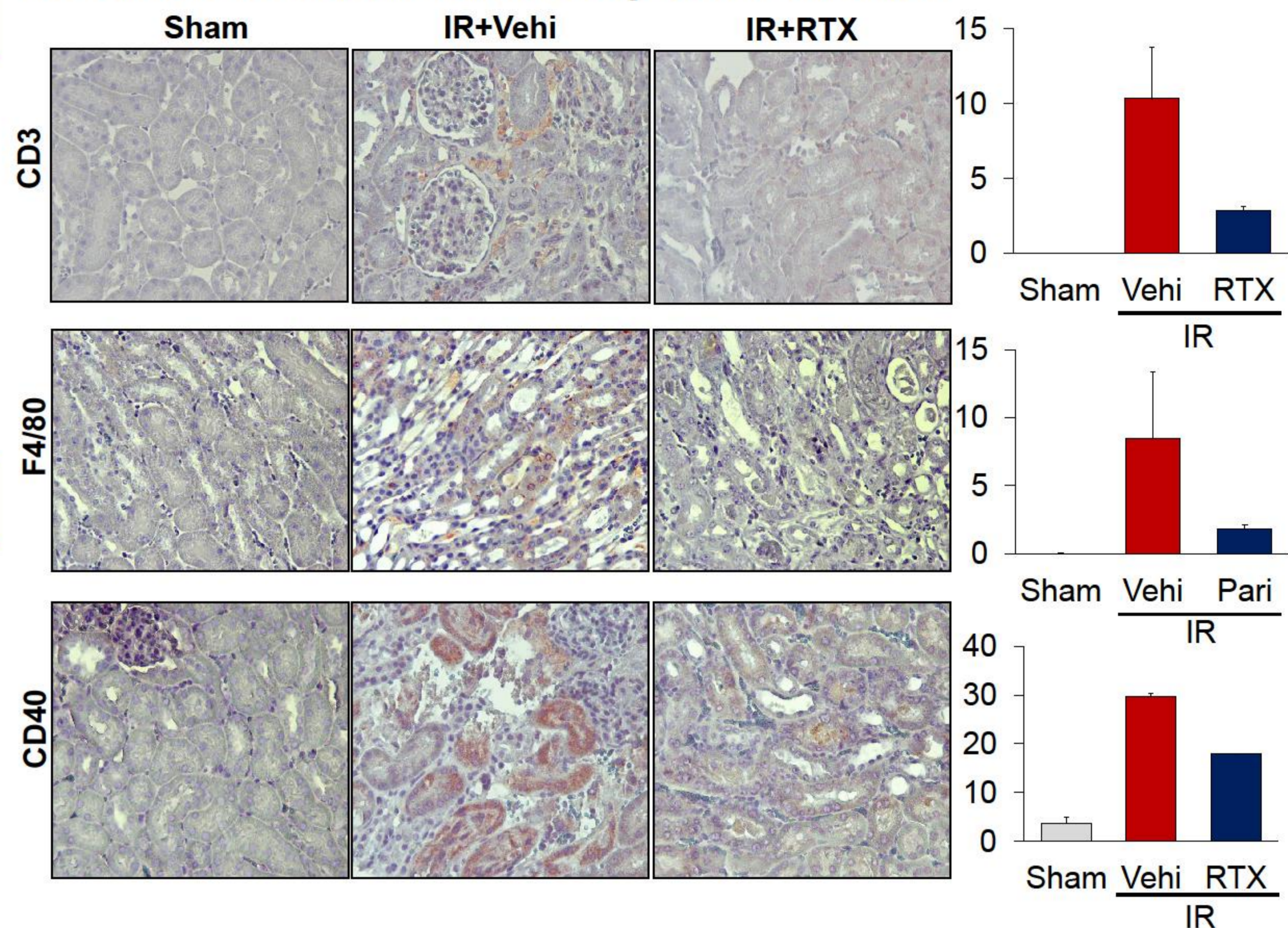
### Rituximab Inhibits CD19+ B-cell Infiltration



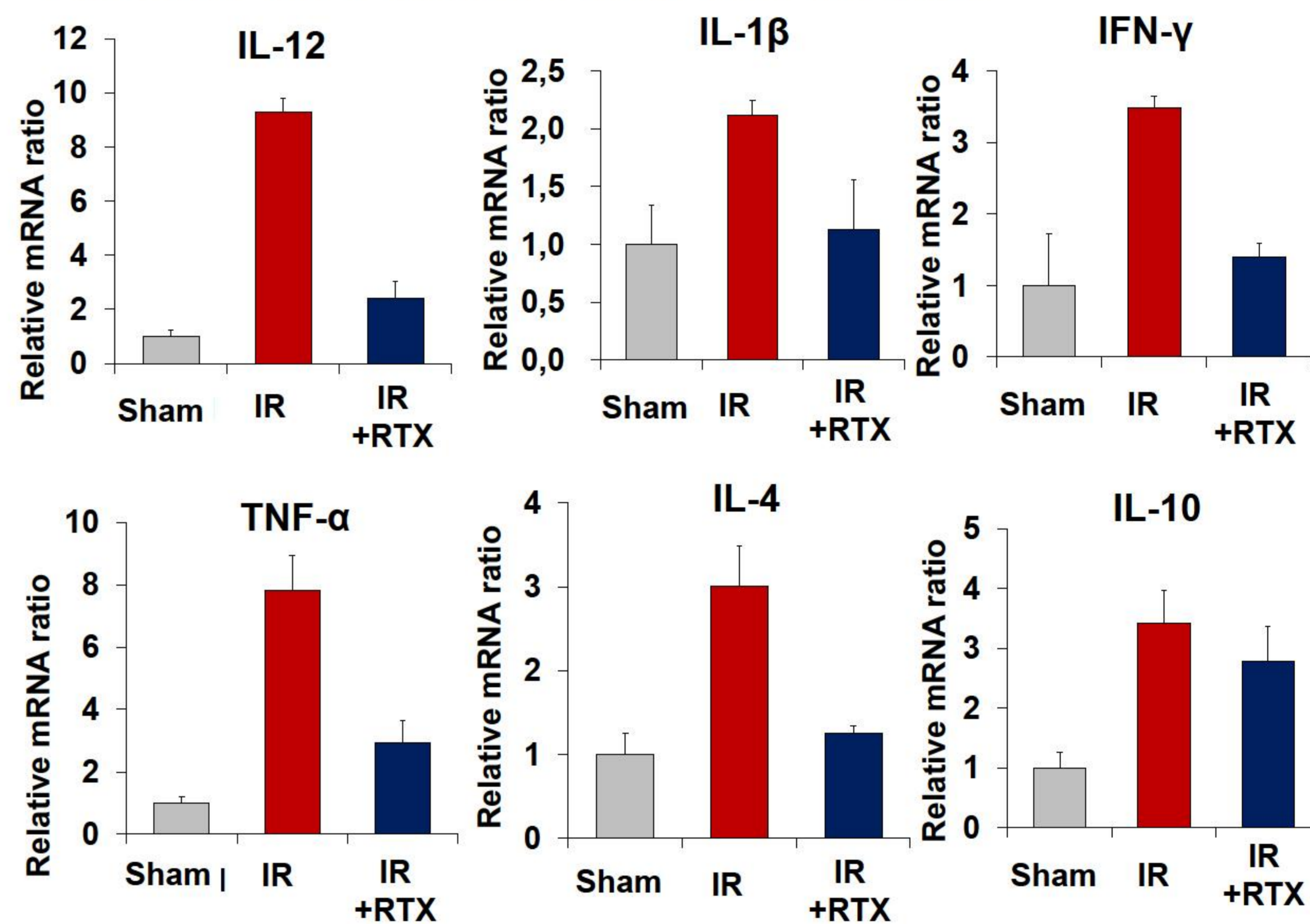
### Rituximab reduces Tubular Injury



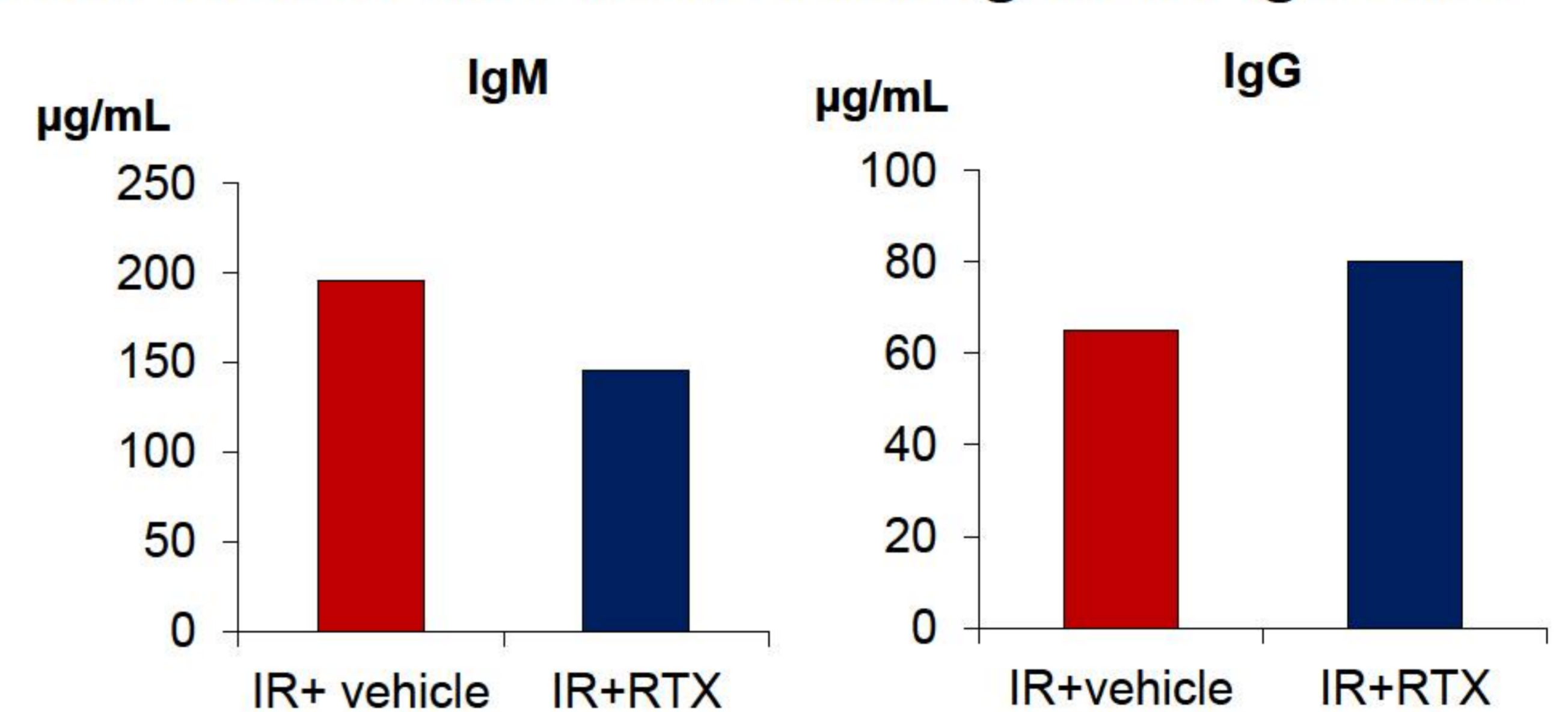
### Rituximab Inhibits Inflammatory cell infiltration



### Rituximab Inhibits Inflammatory cell infiltration



### Rituximab dose not inhibit serum IgM and IgG level



## Summary and Conclusion

- The infiltration of CD19-positive B-cells and CD40-positive antigen-presenting cells were decreased in the rituximab-treated kidneys, which was accompanied with decreased production of IL-12
- Rituximab inhibited the infiltration of T-cells and macrophages and also affected the decreased production of Th1 cytokines IL-1β, IFN-γ and TNF-α.
- Of the Th2 cytokines, IL-4 expression was decreased in rituximab-treated kidneys, but rituximab had little effect on IL-10 expression in the mice kidneys with IRI.
- The IgM and IgG was not expected to be protective mechanism of rituximab in IRI
- Rituximab has a protective effect, which is associated with reduced antigen-presentation and decreased activation of inflammatory cytokine associated with Th1-pathway.

