Pretreatment with paricalcitol attenuates inflammation in ischemia-reperfusion injury via upregulation of cyclooxygenase-2 and prostaglandin E2

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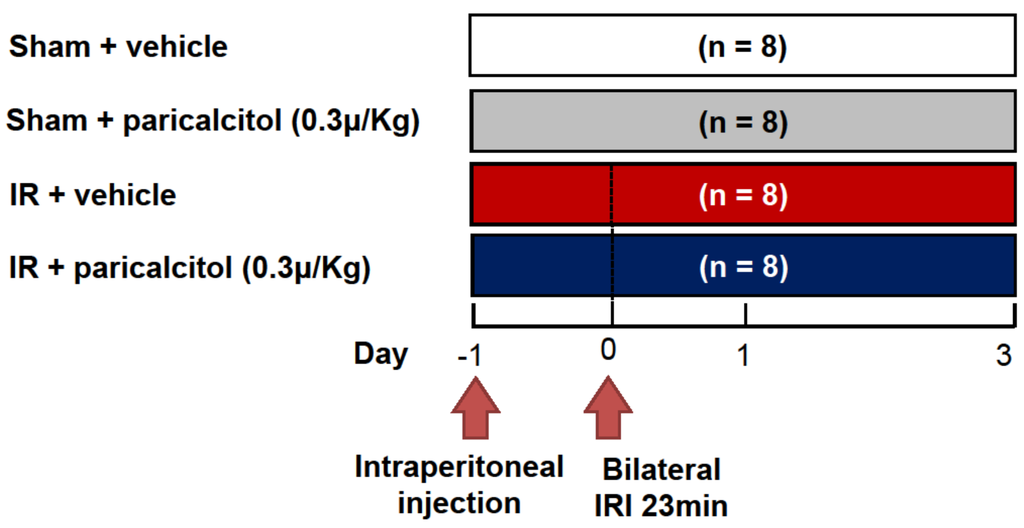
Introduction

Ischemia–reperfusion injury (IRI) is unavoidable event in renal transplantation, causing the delayed graft function and increased immunogenicity. We investigated whether paricalcitol is renoprotective in a mouse model of IRI, and whether potential mechanism is related with modulation of renal inflammation and prostaglandin E2 (PGE2) synthesis.

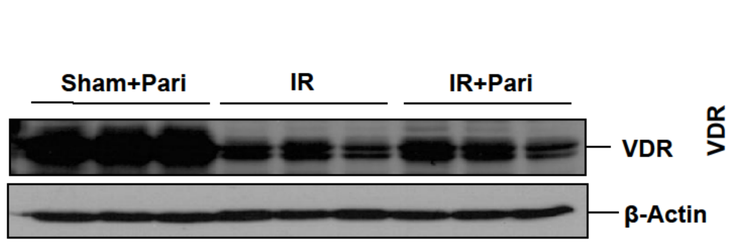
Methods

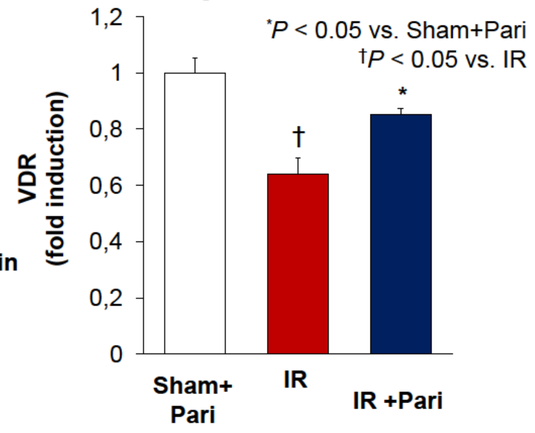


Paricalcitol (0.3 μ/Kg) was administered to male C57BL/6 mice 24 hours before IRI, and mice were killed at 72 hours after IRI

Results

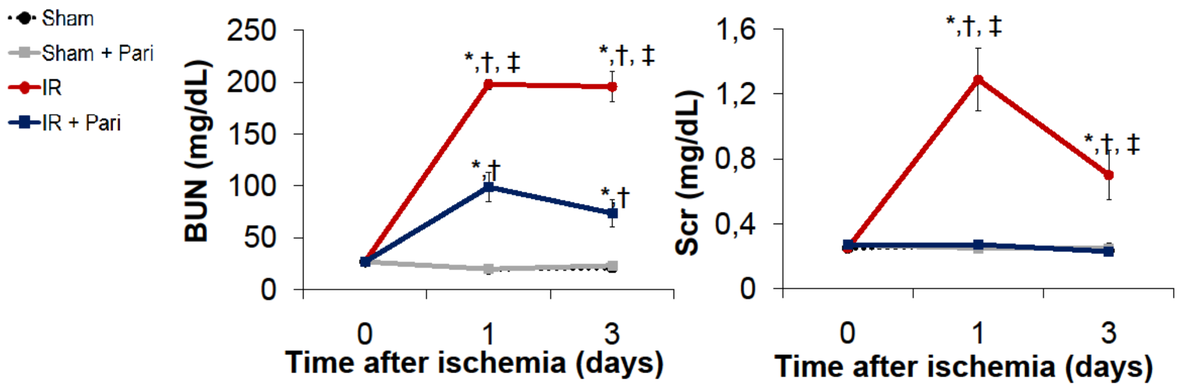
Parcicalcitol Restores the Decreased VDR Expression after IRI



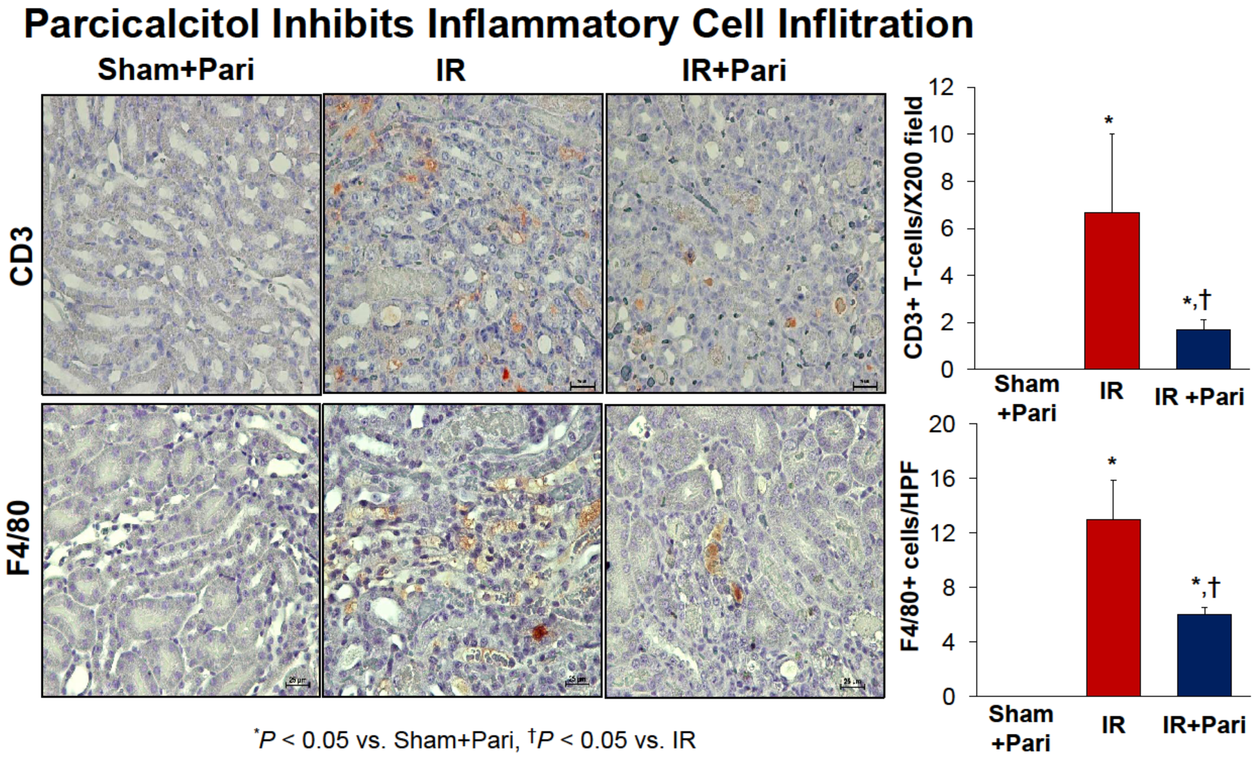


Parcicalcitol Protects Renal Function without Hypercalcemia

*P < 0.05 vs. Sham, †P < 0.05 vs. Sham+Pari, ‡P < 0.05 vs. IR

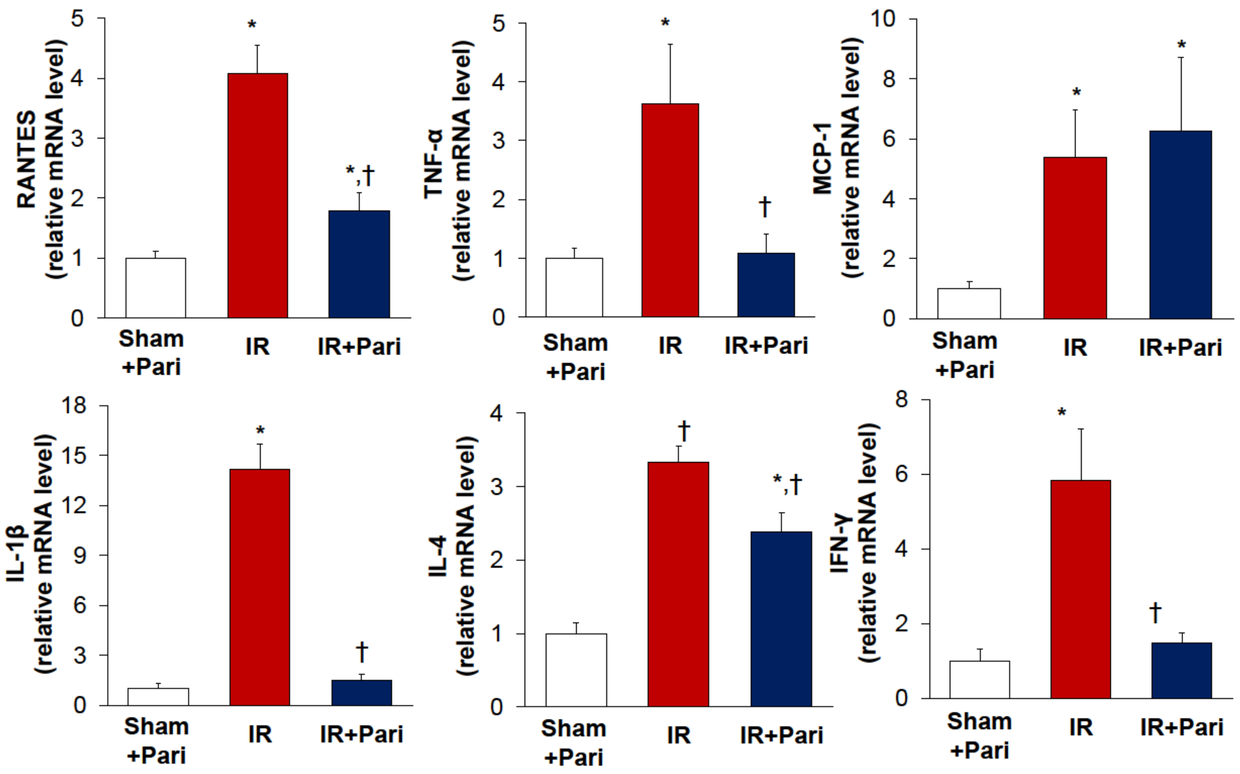


Parcicalcitol Inhibits Inflammatory Cell Inflitration

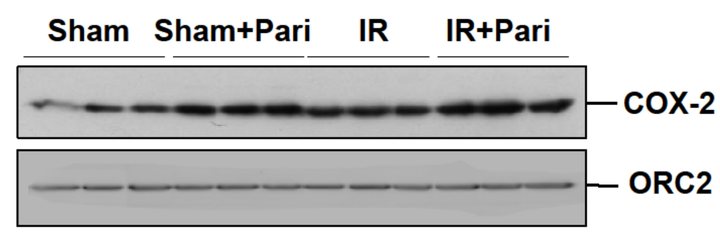


*P < 0.05 vs. Sham+Pari, †P < 0.05 vs. IR

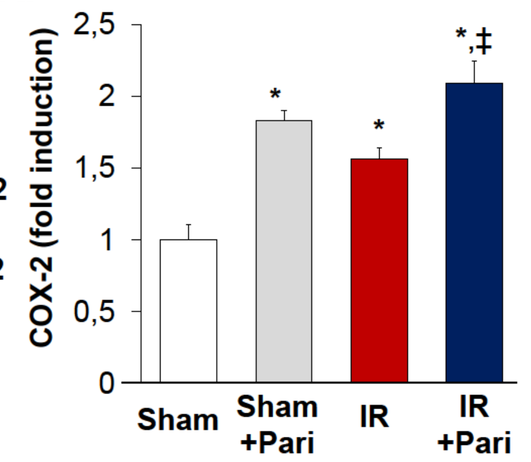
Parcicalcitol Inhibits Inflammatory Cytokine Expression

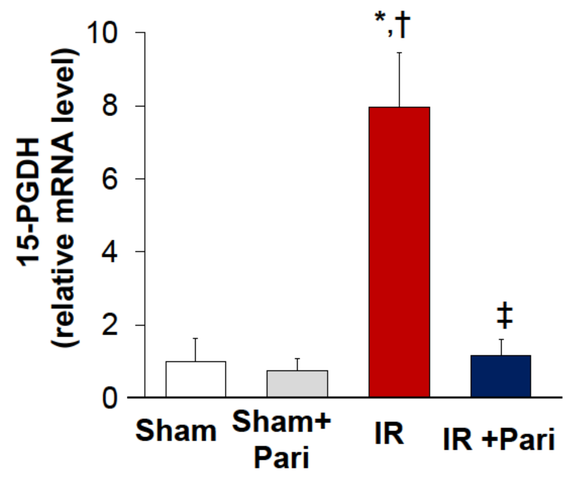


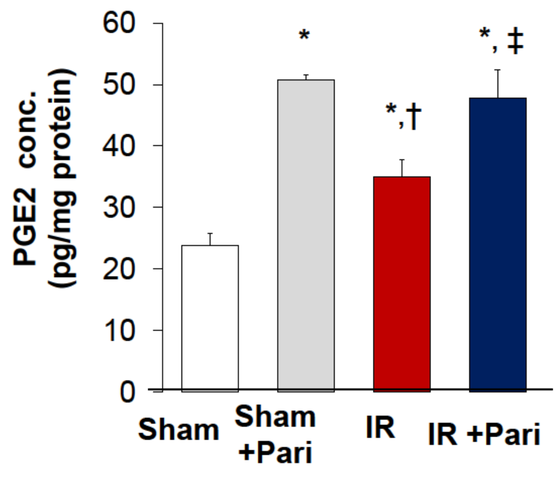
Up-regulation of COX-2 and PGE2 is one of the protective mechanisms of paricalcitol in renal IRI

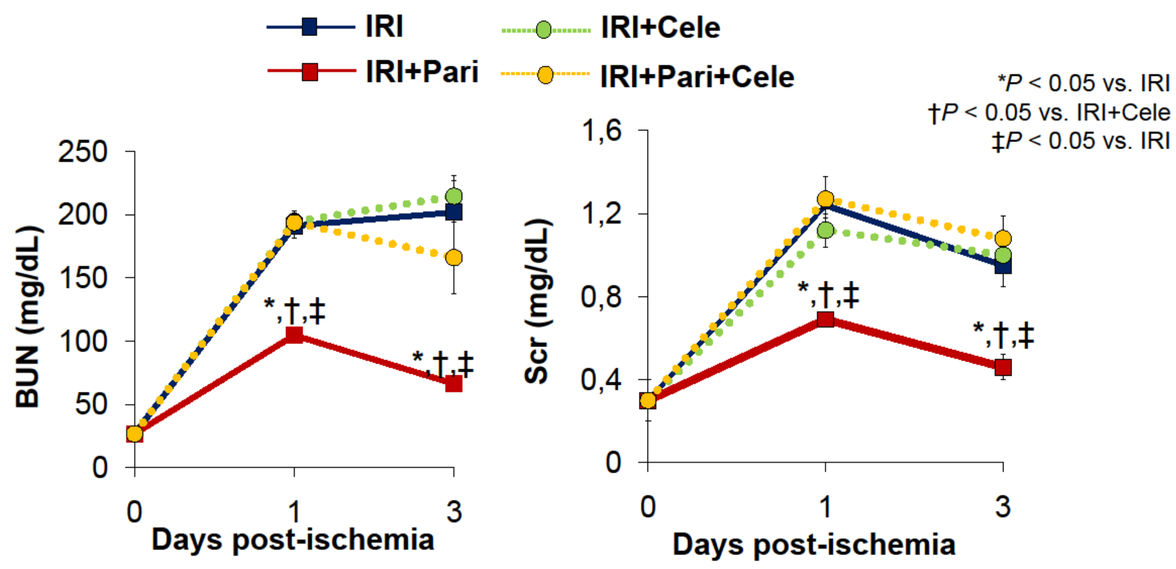


*P < 0.05 vs. Sham
†P < 0.05 vs. Sham+Pari
‡P < 0.05 vs. IR









Summary and Conclusion

- Paricalcitol reduced inflammatory cytokine expression, T-cell and macrophage infiltration in mice kidneys with IRI
- Paricalcitol increased the COX-2, PGE2 and EP4 expression in mice kidneys without IRI
- Up-regulation of COX-2, PGE2 and EP4 expression in IRI kidneys was enhanced by paricalcitol
- Paricalcitol attenuates renal IRI via inhibition of renal inflammation and activation of PGE2 synthesis and its receptor expression

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Hyeon Seok Hwang
DOI: 10.3252/pso.eu.50era.2013
Poster presented at: ERA-EDTA 50th Congress Istanbul, Turkey May 15-21, 2013
PosterSessionOnline.com