Abstract (600 word limits)

Recruitment of β-arrestin to G protein coupled receptors (GPCRs), initially described to cause receptor desensitization, has recently been shown to take active roles in cell signaling. We investigated the effects of TRV027, an angiotensin AT1 receptor β-arrestin-biased ligand, as well as losartan and valsartan on cisplatin-induced kidney injury. Male Sprague dawley rats were treated with angiotensin receptor ligands (1 or 10 mg/kg/day) with or without cisplatin, and kidney variables were monitored using animal SPECT, histopathology, and serum variables. TRV027, losartan and valsartan did not alter renal DMSA uptake, histopathological manifestations of kidney injury, BUN and creatinine or Na and K levels, per se. However, when rats co-treated with cisplatin and either of the AT1 receptor blockers at higher doses, we observed aggravation of cisplatin-induced reduction of radiotracer uptake but improvement of cisplatin-induced hypokalemia, and insignificant effect on histological findings. Furthermore, we noted an additional increase in cisplatin-induced augmentation of BUN and creatinine levels in cisplatin plus valsartan group. Meanwhile, TRV027 inhibited cisplatin adverse effects on radiotracer uptake, kidney histology, BUN and creatinine as well as electrolyte levels. Taken together, TRV027 may offer potential benefits in kidney injury due to cisplatin.

Recent Publications (minimum 5)

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Biography [100 words]

Mohammad Seyedabadi is assistant professor of pharmacology in Bushehr university of medical sciences. He has received his PharmD from Isfahan university of medical sciences, and his Phd from Tehran university of medical sciences. His main area of interest is GPCRs signaling and drug discovery.

References (With Hyperlink)

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