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THE DISTILLERY

This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Renal disease				
Renal damage	Transient receptor potential cation channel subfamily M member 2 (TRPM2); Ras-related C3 botulinum toxin substrate 1 (rho family, small GTP binding protein Rac1) (RAC1)	Mouse studies suggest inhibiting TRPM2 or RAC1 could help treat ischemic kidney injury. In renal cells from a mouse model of ischemic kidney injury, activity of Rac1—which promotes oxidative stress—was higher than that in renal cells from *Trpm2* knockout models. In the model of ischemic kidney injury, *Trpm2* knockout decreased kidney inflammation and Rac1-mediated oxidative stress and increased kidney function compared with normal *Trpm2* expression. Also in the model, inhibitors of TRPM2 or RAC1 decreased oxidative stress and increased kidney function compared with vehicle. Next steps could include testing TRPM2 or RAC1 inhibitors in additional models of kidney injury.	Patent and licensing status unavailable	Gao, G. *et al. J. Clin. Invest.*; published online Oct. 8, 2014; doi:10.1172/JCI76042 **Contact:** W. Brian Reeves, Pennsylvania State University Hershey College of Medicine, Hershey, Pa. e-mail: wreeves@psu.edu

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