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THE DISTILLERY

This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Various				
Tissue damage	Receptor-interacting serine-threonine kinase 3 (RIPK3; RIP3)	Studies in cell culture and in mice suggest that inhibiting RIP3 could prevent tissue necrosis associated with inflammation. In RIP3-deficient human cell lines, a necrosis-promoting caspase inhibitor failed to induce necrosis. Overexpression of RIP3 in the same cells restored the necrotic response to the inhibitor. In a mouse model of acute pancreatitis, RIP3 knockouts showed less pancreatic necrosis and tissue damage than controls. In a mouse model of vaccinia virus infection, RIP3-knockouts showed fewer signs of infection-induced inflammation and liver necrosis than controls. Next steps include ongoing work on elucidating the molecular basis for defective inflammatory responses in RIP3 knockout mice. *SciBX* 2(24); doi:10.1038/scibx.2009.987 Published online June 18, 2009	For the first paper: patent and licensing status unavailable For the second paper: unpatented; unlicensed	He, S. *et al. Cell*; published online June 11, 2009; doi:10.1016/j.cell.2009.05.021 **Contact:** Xiaodong Wang, The University of Texas Southwestern Medical Center at Dallas, Dallas, Texas e-mail: xiaodong.wang@utsouthwestern.edu Cho, Y. *et al. Cell*; published online June 11, 2009; doi:10.1016/j.cell.2009.05.037 **Contact:** Francis Ka-Ming Chan, University of Massachusetts Medical School, Worcester, Mass. e-mail: francis.chan@umassmed.edu