This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Neurology				
Alzheimer's disease (AD)	Tumor necrosis factor receptor superfamily member 21 (TNFRSF21; DR6)	Mouse studies suggest antagonizing DR6 may not be useful for treating AD. Previous cell culture studies suggested DR6 promoted neuronal apoptosis by interacting with AD-associated amyloid precursor protein (APP) (*see* Osherovich, L., *SciBX* 2(8); doi:10.1038/scibx.2009.300). In two new studies, deletion of Dr6 in mouse models of AD did not improve cellular, cognitive or behavioral deficits. Next steps include identifying other potential AD targets. Roche's Genentech Inc. unit has discontinued its discovery stage programs targeting DR6 in AD. ***SciBX*** **7(21); doi:10.1038/scibx.2014.619** **Published online May 29, 2014**	Patents on modulating DR6 in AD previously filed by Genentech; licensing status undisclosed	Kallop, D.Y. *et al. J. Neurosci.*; published online May 7, 2014; doi:10.1523/JNEUROSCI.4963-13.2014 **Contact:** Robby M. Weimer, Genentech Inc., South San Francisco, Calif. e-mail: weimer.robby@gene.com Olsen, O. *et al. J. Neurosci.*; published online May 7, 2014; doi:10.1523/JNEUROSCI.3522-13.2014 **Contact:** Marc Tessier-Lavigne, The Rockefeller University, New York, N.Y. e-mail: marctl@rockefeller.edu **Contact:** Robby M. Weimer, Genentech Inc., South San Francisco, Calif. e-mail: weimer.robby@gene.com