This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Nerve damage	Nicotinamide nucleotide adenylyltransferase 1 (NMNAT1)	A study in mice and in cell culture suggests that increasing cytoplasmic and axonal NMNAT1 expression could help delay nerve degeneration. In mice, neurons that overexpressed cytoplasmic NMNAT1 showed no signs of structural axonal degeneration at 72 hours postinjury, whereas wild-type neurons showed such signs within 24 hours ($p<0.001$). Injured neurons overexpressing NMNAT1 had delayed decreases in function compared with wild-type neurons. Next steps include identifying the molecular pathway downstream of NMNAT1 that could provide targets to block the degeneration process. *SciBX* 2(23); doi:10.1038/scibx.2009.950 Published online June 11, 2009	Patent pending; licensed to the Sirtris Pharmaceuticals Inc. unit of GlaxoSmithKline plc	Sasaki, Y. *et al. J. Neurosci.*; published online May 20, 2009; doi:10.1523/JNEUROSCI.1429-09.2009 **Contact:** Jeffrey Milbrandt, Washington University School of Medicine, St. Louis, Mo. e-mail: jmilbrandt@wustl.edu