This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Amyotrophic lateral sclerosis (ALS)	X-box binding protein 1 (XBP1); ER-associated protein degradation (ERAD); superoxide dismutase 1 (SOD1)	Studies in cell culture and in mice suggest that inhibiting XBP1 could help treat ALS. In a murine neuronal cell line expressing an ALS-associated mutant *SOD1*, knockdown of *Xbp1* led to greater autophagic activity and better clearance of mutant SOD1 than that in controls. Similar results were seen in a mouse model of ALS, including longer lifespan for knockdown mice than for *Xbp1*-expressing controls. Ongoing work includes testing compounds that inhibit XBP1 or enhance autophagy in animal models of ALS. ***SciBX*** **2(38); doi:10.1038/scibx.2009.1446** **Published online Oct. 1, 2009**	Patented by the Harvard School of Public Health; available for licensing	Hetz, C. *et al. Genes Dev.*; published online Sept. 17, 2009; doi:10.1101/gad.1830709 **Contact:** Laurie H. Glimcher, Harvard Medical School, Boston, Mass. e-mail: lglimche@hsph.harvard.edu **Contact:** Claudio Hetz, University of Chile, Santiago, Chile e-mail: chetz@med.uchile.cl