This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Neurology				
Alzheimer's disease (AD)	Ribosomal protein S23 retroposition 1 (RPS23R1)	<p>A study in mice and in cell culture suggests that increasing expression of the transmembrane protein RPS23R1 could help treat AD. In a mouse model of AD, overexpression of <i>Rps23r1</i> in the brain reduced AD-associated τ-protein phosphorylation and β-amyloid ($A\beta$) levels compared with those in mice that had normal <i>Rps23r1</i> expression. In mouse and human cells, overexpression of <i>Rps23r1</i> led to lower τ-protein phosphorylation and $A\beta$ levels than were seen in cells treated with a control expression vector. Next steps include identifying compounds that could increase RPS23R1 expression and developing a conditional gene knockout mouse for proof-of-principle studies.</p> <p>SciBX 2(46); doi:10.1038/scibx.2009.1704 Published online Dec. 3, 2009</p>	Patent application filed; available for licensing from the Burnham Institute for Medical Research	<p>Zhang, Y.-W. <i>et al. Neuron</i>; published online Nov. 11, 2009; doi:10.1016/j.neuron.2009.08.036</p> <p>Contact: Huaxi Xu, Burnham Institute for Medical Research, La Jolla, Calif. e-mail: xuh@burnham.org</p> <p>Contact: Limin Li, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing, China e-mail: limin@liminli.com</p>