This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Cognitive dysfunction; Alzheimer's disease (AD)	Heat shock protein 90 (Hsp90)	<p>Mouse and <i>in vitro</i> studies suggest inhibiting Hsp90 could enhance cognitive function. In cultured primary rat neurons, an Hsp90 inhibitor increased heat shock transcription factor 1 (HSF1)-dependent expression of pre- and postsynaptic proteins compared with vehicle control, leading to elevated synaptic transmission and protection against spine loss after treatment with β-amyloid (Aβ). In mice, intracerebroventricular injection of Aβ plus the inhibitor rescued synaptic protein expression and decreased memory loss in a fear conditioning test compared with injection of Aβ alone. Ongoing work includes testing an undisclosed proprietary brain-permeable Hsp90 inhibitor in a mouse model of AD.</p> <p>SciBX 7(9); doi:10.1038/scibx.2014.264 Published online March 6, 2014</p>	Unpatented; unlicensed	<p>Chen, Y. <i>et al. J. Neurosci.</i>; published online Feb. 12, 2014; doi:10.1523/JNEUROSCI.0151-13.2014 Contact: Francesca-Fang Liao, The University of Tennessee Health Science Center College of Medicine, Memphis, Tenn. e-mail: fliao@uthsc.edu</p>