This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Neurology				
Alzheimer's disease (AD)	AMP-activated protein kinase (AMPK); β -amyloid 42 (A β 42); calcium calmodulin-dependent protein kinase kinase 2 (CAMKK2)	<p>Mouse and cell culture studies suggest inhibiting the CAMKK2/AMPK pathway could help prevent Aβ42 toxicity in AD. In cultured mouse hippocampal neurons, knockout or pharmacological inhibition of Camkk2 decreased Aβ42 oligomer-induced synaptotoxicity compared with no knockout or inhibition. In cell culture studies in neurons, the Aβ42 oligomers induced AMPK-mediated phosphorylation of microtubule-associated protein-τ (MAPT; TAU; FTDP-17). In mice overexpressing Aβ42 in the hippocampus, inhibiting Camkk2/Ampk signaling decreased the loss of dendritic spines compared with no inhibition. Next steps include validating the long-term protective effects of blocking CAMKK2/AMPK signaling on additional measures of AD histopathology.</p> <p>SciBX 6(17); doi:10.1038/scibx.2013.418 Published online May 2, 2013</p>	Unpatented; licensing status not applicable	<p>Mairet-Coello, G. <i>et al. Neuron</i>; published online April 10, 2013; doi:10.1016/j.neuron.2013.02.003 Contact: Franck Polleux, The Scripps Research Institute, La Jolla, Calif. e-mail: polleux@scripps.edu</p>