This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Neurology				
Pain	Calcium channel voltage-dependent N type- α 1B subunit (CACNA1B; CaV2.2); collapsin response mediator protein-2 (DPYSL2; CRMP-2)	<p><i>In vitro</i> and rat studies suggest inhibiting the interaction between CaV2.2 and CRMP-2 could help treat neuropathic pain. CaV2.2 signaling is involved in pain pathways; however, completely blocking the channel affects normal neuron function. In rat dorsal root ganglion neurons, a peptide derived from a Crmp2-binding region of CaV2.2 blocked the Crmp2-CaV2.2 interaction and decreased calcium ion influx and neurotransmitter release compared with a nontargeting peptide. In two rat models of peripheral neuropathy, intraperitoneal administration of the peptide decreased pain behaviors compared with a nontargeting peptide or saline. Next steps include clinical development of a lead molecule.</p> <p>UCB Group markets Vimpat lacosamide, an anticonvulsant that modulates CRMP-2 to slow inactivation of sodium channels, to treat epilepsy and seizures.</p> <p>SciBX 5(36); doi:10.1038/scibx.2012.958 Published online Sept. 13, 2012</p>	Patent application filed covering peptides and their use in preventing neuropathic pain; exclusively licensed to Sophia Therapeutics LLC	<p>Wilson, S.M. <i>et al. J. Biol. Chem.</i>; published online Aug. 13, 2012; doi:10.1074/jbc.M112.378695</p> <p>Contact: Rajesh Khanna, Indiana University School of Medicine, Indianapolis, Ind. e-mail: khanna5@iupui.edu</p>