This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Pain	Nav1.9 (SCN11A)	Gene sequencing, cell culture and mouse studies suggest antagonizing Nav1.9 could help treat congenital analgesia. Sequencing of patients identified a missense gain-of-function mutation in *SCN11A*, which encodes the voltage-gated sodium ion channel Nav1.9. In a dorsal root ganglion neuronal cell line, expression of mutant Nav1.9 decreased the depolarization required to activate or inactivate voltage-dependent sodium currents compared with wild-type Nav1.9 expression. In heterozygous knock-in mice expressing the mutant *Scn11a* allele, thermal and mechanical stimuli produced fewer pain responses and less protective activity than what was seen in mice expressing wild-type *Scn11a*. Next steps include testing Nav1.9 inhibitors in congenital analgesia and testing whether compounds mimicking mutant channel behavior could help suppress pain. *SciBX* 6(41); doi:10.1038/scibx.2013.1166 Published online Oct. 24, 2013	Patent application filed; available for licensing	Leipold, E. *et al. Nat. Genet.*; published online Sept. 15, 2013; doi:10.1038/ng.2767 **Contact:** Ingo Kurth, Institute of Human Genetics, Jena University Hospital, Jena, Germany e-mail: ingo.kurth@med.uni-jena.de