SciBX
Science-Business eXchange

THE DISTILLERY

This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Pain	Sodium channel, voltage-gated, type IX, α subunit (SCN9A; NaV1.7)	A cell-culture study suggests that inhibiting variants of NaV1.7 may help treat chronic pain. Gain-of-function mutations in the NaV1.7 channel are known to generate hyperpolarizing neuronal currents that cause inherited erythromyalgia (IEM) or paroxysmal extreme pain disorder (PEPD). Electrophysiology studies showed that cultured dorsal root and trigeminal ganglion neurons expressing NaV1.7 channels with a certain mutation were hyperexcitable compared with wild-type neurons. The A1632E mutation of NaV1.7 was found in a patient who showed a mixed clinical phenotype of IEM and PEPD. Next steps include identifying specific antagonists of NaV1.7. *SciBX* 1(40); doi:10.1038/scibx.2008.981 Published online Nov. 6, 2008	Unpatented; licensing status not applicable	Estacion, M. *et al. J. Neurosci.*; published online Oct. 22, 2008; doi:10.1523/JNEUROSCI.3443-08.2008 **Contact:** S.G. Waxman, Yale Medical School, New Haven, Conn. e-mail: stephen.waxman@yale.edu