

This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Neurology				
Alzheimer's disease (AD)	Solute carrier family 25 member 38 (SLC25A38); β -amyloid 42	Human and rodent studies suggest inhibiting SLC25A38 could prevent neurodegeneration in AD. In the brains of patients with AD, SLC25A38 levels were higher than those in healthy controls. In primary rat and mouse neurons and in a human neuroblastoma cell line, β -amyloid 42 increased SLC25A38 levels and caspase-induced apoptosis compared with no treatment. In the rodent primary neurons and the cell line, small interfering RNA against <i>SLC25A38</i> decreased β -amyloid 42-induced apoptosis compared with control siRNA. Ongoing work includes evaluating the effects of knocking out <i>Slc25a38</i> in animal AD models.	Unpatented; available for licensing or partnering	Zhang, H. <i>et al. J. Neurosci.</i> ; published online Oct. 31, 2012; doi:10.1523/JNEUROSCI.3668-12.2012 Contact: Huaxi Xu, Sanford-Burnham Medical Research Institute, La Jolla, Calif. e-mail: xuh@sanfordburnham.org Contact: Ye-Guang Chen, Tsinghua University, Beijing, China e-mail: ygchen@tsinghua.edu.cn
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