This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Neurology				
Alzheimer's disease (AD)	G protein–coupled receptor 3 (GPR3)	A study in cell culture and in mice suggests inhibiting GPR3 could help treat AD. In cell culture, overexpression of GPR3 increased production of AD-associated β-amyloid (Aβ) compared with that seen in mock-treated controls, whereas small interfering RNA knockdown of GPR3 decreased Aβ production. In cultured mouse neurons, GPR3 overexpression increased levels of γ-secretase, an enzyme that converts amyloid precursor protein (APP) to Aβ, compared with those seen in untreated controls. In a mouse model of AD, *Gpr3* deletion lowered Aβ levels in the hippocampus compared with those seen in wild-type controls. Next steps include developing small molecule antagonists of GPR3 and testing them in mouse models of AD-associated cognitive impairment. Galapagos N.V. has a preclinical program targeting GPR3 to treat AD. ***SciBX* 2(8); doi:10.1038/scibx.2009.327** **Published online Feb. 26, 2009**	Patented by Galapagos N.V.; available for partnering	Thathiah, A. *et al. Science*; published online Feb.12, 2009; doi:10.1126/science.1160649 **Contact:** Bart De Strooper, Catholic University Leuven, Leuven, Belgium e-mail: bart.destrooper@med.kuleuven.be **Contact:** David Fischer, Galapagos N.V., Mechelen, Belgium e-mail: david.fischer@glpg.com