This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Neurology				
Alzheimer's disease (AD); cognitive dysfunction	β -Site APP-cleaving enzyme 1 (BACE1); amyloid precursor protein (APP)	Human genetic, cell culture and <i>in vitro</i> assay studies suggest the A673T substitution in <i>APP</i> may protect against AD and normal cognitive decline. In multiple elderly AD and non-AD cohorts, human genetic and cognitive performance studies showed that the A673T mutation in <i>APP</i> was associated with greater conserved cognition than that in noncarriers and suggested the mutation may protect against AD and normal cognitive decline. In an <i>in vitro</i> BACE1 cleavage assay, mutant A673T <i>APP</i> was processed 50% less efficiently than wild-type <i>APP</i> . Next steps could include developing A673T mutant <i>APP</i> mouse models.	Patent and licensing status unavailable	Jonsson, T. <i>et al. Nature</i> ; published online July 11, 2012; doi:10.1038/nature11283 Contact: Kari Stefansson, deCODE genetics ehf, Reykjavik, Iceland e-mail: kstefans@decode.is
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