This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Transgenic mouse model of Alzheimer's disease (AD) expressing human β -site APP cleaving enzyme 1 (<i>BACE1</i>)	Mice with forebrain-specific expression of human <i>BACE1</i> could be useful as models for evaluating new treatments for AD. In mice, human <i>BACE1</i> under control of the calcium calmodulin-dependent protein kinase II (CAMK2) promoter was knocked in to the <i>hypoxanthine phosphoribosyltransferase 1</i> (<i>Hprt1</i> ; <i>Hgppt</i>) locus to yield forebrain-specific expression of the transgene. The transgenic mice showed decreased motor activity compared with wild-type controls and impaired spatial acquisition and long-term memory retrieval. In the transgenic mice, amyloid precursor protein (App) processing was faster and β -amyloid (A β) oligomer levels were higher than those in wild-type controls. Next steps include exploring mechanisms of dementia in the model.	Unpatented; mouse model available for licensing	Plucińska, K. <i>et al. J. Neurosci.</i> ; published online Aug. 6, 2014; doi:10.1523/JNEUROSCI.0433-14.2014 Contact: Bettina Platt, The University of Aberdeen, Aberdeen, U.K. e-mail: b.platt@abdn.ac.uk Contact: Gernot Riedel, same affiliation as above e-mail: g.riedel@abdn.ac.uk
	SciBX 7(34); doi:10.1038/scibx.2014.1024 Published online Sept. 4, 2014		