This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Alzheimer's disease (AD) mouse model	<p>Transgenic mice expressing amyloid-β (Aβ) precursor protein (APP) mutations and lacking inducible nitric oxide synthase 2 (NOS2; iNOS) recapitulate key features of human AD that are absent from current AD models. The mice developed β-amyloid (Aβ) deposits and two features not present in previous models: aggregates of microtubule-associated protein-τ (MAPT; TAU; FTDP-17) and neuron loss. In the mice, mAbs targeting Aβ decreased Aβ deposition, TAU pathology and neuron loss and improved memory compared with no treatment. Next steps include looking at the downstream effects of Aβ deposition and testing TAU-targeted therapeutics in the mice.</p> <p>Compounds in Phase III testing for AD include Aβ-targeting mAbs bapineuzumab, from Elan Corp. plc and Wyeth, and LY2062430, from Eli Lilly and Co.</p> <p>Anti-Aβ mAbs in Phase I for AD include R1450 from MorphoSys AG and Roche, RN1219 from Pfizer Inc.'s Rinat Neuroscience Corp., MABT5102 from Roche's Genentech Inc. unit and GlaxoSmithKline plc's GSK933776A.</p> <p>Rember methylthioninium chloride (methylene blue), a TAU-modulating small molecule from TauRx Therapeutics Ltd. has completed a Phase II AD trial.</p> <p>SciBX 2(26); doi:10.1038/scibx.2009.1056 Published online July 9, 2009</p>	Transgenic mice described in paper are patented; available for licensing from Duke University	<p>Wilcock, D. <i>et al. J. Neurosci.</i>; published online June 24, 2009; doi:10.1523/JNEUROSCI.1339-09.2009</p> <p>Contact: Donna M. Wilcock, Duke University Medical Center, Durham, N.C. e-mail: donna.wilcock@duke.edu</p>