This week in techniques

THE DISTILLERY brings you this week's most essential scientific findings in techniques, distilled by *SciBX* editors from a weekly review of more than 400 papers in 41 of the highest-impact journals in the fields of biotechnology, the life sciences and chemistry. The Distillery goes beyond the abstracts to explain the commercial relevance of featured research, including licensing status and companies working in the field, where applicable.

This week in techniques includes findings about research tools, disease models and manufacturing processes that have the potential to enable or improve all stages of drug discovery and development.

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Alzheimer's disease (AD) mouse model	Transgenic mice expressing amyloid-β (A4) 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. 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. 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. Rember methylthioninium chloride (methylene blue), a TAU-modulating small molecule from TauRx Therapeutics Ltd. has completed a Phase II AD trial. ***SciBX* 2(26); doi:10.1038/scibx.2009.1056** **Published online July 9, 2009**	Transgenic mice described in paper are patented; available for licensing from Duke University	Wilcock, D. *et al. J. Neurosci.*; published online June 24, 2009; doi:10.1523/JNEUROSCI.1339-09.2009 **Contact:** Donna M. Wilcock, Duke University Medical Center, Durham, N.C. e-mail: donna.wilcock@duke.edu
Locust model of cortical spreading depression	The locust (*Locusta migratoria*) could provide a model of cortical spreading depression, a depolarization of cortical neurons associated with stroke, migraine and head trauma. Electrophysiological measurements showed that injection of potassium or a sodium-potassium pump inhibitor into the meta-thoracic ganglion of locusts mimicked a spreading depression–like event that occurs in response to heat or anoxic stress. Inhibition of nitric oxide (NO) or protein kinase G (PKG) accelerated the insects' recovery from the spreading depression–like events. Future studies in locusts will examine the role of signaling events downstream of NO and PKG in spreading depression–like events. ***SciBX* 2(26); doi:10.1038/scibx.2009.1057** **Published online July 9, 2009**	Patented; available for licensing	Armstrong, G. *et al. J. Neurosci.*; published online June 24, 2009; doi:10.1523/JNEUROSCI.1652-09.2009 **Contact:** R. Meldrum Robertson, Queen's University, Kingston, Ontario, Canada e-mail: robertrm@queensu.ca
Parkinson's disease (PD) mouse model	Mice with a 95% reduction in vesicular monoamine transporter 2 (VMAT2; SLC18A2) could be useful PD models. The VMAT2-deficient mice showed degeneration in multiple parts of the brain and disrupted dopamine and norepinephrine signaling. The mice also showed defective olfactory function, sleeping disturbances, delayed gastric emptying and anxiety- and depression-like behaviors compared with wild-type mice. Next steps include using the model to test PD therapeutics. ***SciBX* 2(26); doi:10.1038/scibx.2009.1058** **Published online July 9, 2009**	Mouse model unpatented; model available for licensing through Emory University	Taylor, T. *et al. J. Neurosci.*; published online June 24, 2009; doi:10.1523/JNEUROSCI.1495-09.2009 **Contact:** Gary W. Miller, Emory University, Atlanta, Ga. e-mail: gary.miller@emory.edu