This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
<i>Spectrin-β non-erythrocytic 3</i> (<i>Sptbn3</i>)-deficient mice as a model of spinocerebellar ataxia type 5 (SCA5)	Mice with a <i>Sptbn3</i> -deficiency could help identify new treatments for SCA5. Mice lacking <i>Sptbn3</i> —the murine analog of the human gene that causes disease—had greater neurodegeneration than wild-type controls. In cognitive and motor ability tests, <i>Sptbn3</i> -deficient mice showed mild ataxia, gait disturbance and muscle weakness. <i>Sptbn3</i> -deficient mice were seizure prone and expressed markers of seizure activity in the cerebellum and hippocampus. Next steps could include determining the electrophysiological effects of <i>Sptbn3</i> loss and identifying additional environmental factors that can result in more severe cerebellar degeneration and ataxia.	Patent and licensing status unavailable	Stankewich, M.C. <i>et al. Proc. Natl. Acad. Sci. USA</i> ; published online March 15, 2010; doi:10.1073/pnas.1001522107 Contact: Jon S. Morrow, Yale University, New Haven, Conn. e-mail: jon.morrow@yale.edu
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