

## This week in techniques

Approach	Summary	Licensing status	Publication and contact information
<b>Disease models</b>			
Knock-in mouse model of autoimmunity	<p>A knock-in mouse model of autoimmune diseases could aid the development of new therapies. The knock-in mice, which expressed the D485N mutant form of <i>TNFAIP3 interacting protein 1</i> (<i>Tnip1</i>; <i>Abin1</i>), developed lupus-like autoimmune symptoms that were suppressed by deleting the gene encoding myeloid differentiation primary response 88 (Myd88). Next steps include studying the molecular mechanism by which ABIN1 regulates MYD88 signaling.</p> <p><b>SciBX 4(23); doi:10.1038/scibx.2011.666</b>  <b>Published online June 9, 2011</b></p>	Unpatented; available for licensing	<p>Nanda, S.K. <i>et al. J. Exp. Med.</i>; published online May 23, 2011; doi:10.1084/jem.20102177</p> <p><b>Contact:</b> Philip Cohen, University of Dundee, Dundee, U.K.  e-mail: <a href="mailto:p.cohen@dundee.ac.uk">p.cohen@dundee.ac.uk</a></p> <p><b>Contact:</b> Sambit K. Nanda, same affiliation as above  e-mail: <a href="mailto:s.k.nanda@dundee.ac.uk">s.k.nanda@dundee.ac.uk</a></p>