This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Various				
Autoimmune glomerulonephritis (GN)	Bruton's tyrosine kinase (BTK); leukocyte immunoglobulin-like receptor, subfamily B (LILRB3; PIRB); toll-like receptor 9 (TLR9)	<p>Studies in mice and in cell culture suggest that activating PIRB or inhibiting TLR9-induced BTK activation could help prevent autoimmune GN. In <i>Pirb</i>-deficient mice, the TLR9 ligand CpG increased levels of autoantibodies compared with those in wild-type controls. In mice with a mutation associated with autoimmune GN, <i>Pirb</i> deficiency led to increased production of disease-associated autoantibodies compared with that in controls expressing normal <i>Pirb</i>. In peritoneal cells, <i>PirB</i> signaling inhibited TLR9-mediated activation of <i>Btk</i>. Next steps include studying how PIRB regulates the TLR9 signaling cascade.</p> <p>SciBX 2(33); doi:10.1038/scibx.2009.1287 Published online Aug. 27, 2009</p>	Work unpatented; available for licensing from the Tohoku University Department of Industrialization	<p>Kubo, T. <i>et al. J. Exp. Med.</i>; published online Aug. 17, 2009; doi:10.1084/jem.20082392</p> <p>Contact: Toshiyuki Takai, Tohoku University, Sendai, Japan e-mail: tostakai@idac.tohoku.ac.jp</p>