This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Mouse model of nickel-associated allergic contact dermatitis	<p>A mouse model of nickel-associated allergic dermatitis could help guide the development of dermatitis therapies. In a human embryonic kidney cell line, mutations in two histidine residues of <i>toll-like receptor 4</i> (<i>TLR4</i>) lowered nickel-induced proinflammatory gene expression compared with nonmutated <i>TLR4</i>, confirming that <i>TLR4</i> is a key mediator of nickel-associated dermatitis. Transgenic mice expressing human <i>TLR4</i> developed nickel-induced contact dermatitis, whereas those expressing only mouse <i>Tlr4</i>, which lacks a nickel-responsive region, did not. Next steps include using the transgenic mice to identify specific inhibitors of the nickel-responsive region of human <i>TLR4</i>.</p> <p>SciBX 3(32); doi:10.1038/scibx.2010.998 Published online Aug. 19, 2010</p>	<p>Patent application filed covering contact allergies in humans; licensing inquiries should be directed to the University of Giessen</p>	<p>Schmidt, M. <i>et al. Nat. Immunol.</i>; published online Aug. 15, 2010; doi:10.1038/ni.1919 Contact: Matthias Goebeler, University of Giessen, Giessen, Germany e-mail: matthias.goebeler@derma.med.uni-giessen.de</p>