This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Disease models			
Transgenic mouse models of Netherton syndrome expressing human <i>kallikrein-related peptidase 5</i> (<i>KLK5</i>)	<p>Transgenic mice expressing human <i>KLK5</i> in the epidermis could be used to study the biology of Netherton syndrome and screen for drugs to treat the indication. Netherton syndrome is a severe form of ichthyosis caused by mutations in serine peptidase inhibitor Kazal type 5 (<i>SPINK5</i>; <i>LEKT1</i>), an inhibitor of <i>KLK5</i>. The transgenic mice survived longer than <i>Spink5</i>-deficient mice and recapitulated symptoms of Netherton syndrome including reddened, scaling skin; hyperkeratosis; detachment of the skin's outer epidermal layer from the underlying granular layer; and skin barrier and hair defects. The transgenic mice also had enlarged lymph nodes and increased levels of allergy-associated cells and T helper type 2 (Th2) proinflammatory molecules in the skin and increased serum levels of IgE compared with wild-type controls. Future studies could include evaluating potential Netherton syndrome therapies in the model.</p> <p>SciBX 7(10); doi:10.1038/scibx.2014.297 Published online March 13, 2014</p>	Patent and licensing status unavailable	<p>Furio, L. <i>et al. J. Exp. Med.</i>; published online Feb. 17, 2014; doi:10.1084/jem.20131797 Contact: Alain Hovnanian, University Paris Descartes–Sorbonne, Paris, France e-mail: alain.hovnanian@inserm.fr</p>