This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Dermatology				
Chronic dermatitis	SHANK-associated RH domain interactor (SHARPIN); tumor necrosis factor- α (TNF- α)	<p>Three separate <i>in vitro</i> and mouse studies suggest that inhibiting TNF-α and/or the linear ubiquitin chain assembly complex (LUBAC) could help treat chronic dermatitis. In one study, coimmunoprecipitation assays showed that SHARPIN, a component of the LUBAC complex, induced linear ubiquitination. In mice, <i>Sharpin</i> deficiency caused chronic proliferative dermatitis (CPDM) compared with what was seen in wild-type mice. In a second study, TNF-α induced cell death in embryonic fibroblasts from <i>Sharpin</i>-deficient CPDM mice compared with in cells from wild-type mice. In a third study, crossing the <i>Sharpin</i>-deficient CPDM mice with heterozygous TNF-α knockout mice prevented the inflammatory skin lesions compared with those in mice expressing both copies of TNF-α. Next steps include testing anti-TNF-α therapeutics for the treatment of chronic dermatitis in humans and identifying inhibitors of polyubiquitination that could help treat other diseases caused by increased levels of TNF-α.</p> <p>SciBX 4(15); doi:10.1038/scibx.2011.426 Published online April 14, 2011</p>	<p>For findings in the first study, patent application filed for a truncated version of LUBAC that can be used to develop inhibitors of linear polyubiquitination; licensed by some pharmaceutical companies; LUBAC screening tool available for licensing</p> <p>Findings in the second study unpatented; unavailable for licensing</p> <p>For findings in the third study, priority patent application filed covering therapeutic approaches to treat autoimmune diseases and cancer-related inflammation; available for licensing</p>	<p>Tokunaga, F. <i>et al. Nature</i>; published online March 30, 2011; doi:10.1038/nature09815 Contact: Kazuhiro Iwai, Osaka University, Osaka, Japan e-mail: kiwai@cellbio.med.osaka-u.ac.jp</p> <p>Ikeda, F. <i>et al. Nature</i>; published online March 30, 2011; doi:10.1038/nature09814 Contact: Ivan Dikic, Goethe University School of Medicine, Frankfurt, Germany e-mail: Ivan.Dikic@biochem2.de</p> <p>Gerlach, B. <i>et al. Nature</i>; published online March 30, 2011; doi:10.1038/nature09816 Contact: Henning Walczak, Imperial College London, London, U.K. e-mail: h.walczak@imperial.ac.uk</p>