This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Drug platforms			
Specific and noncovalent binding of small molecule therapeutics to human leukocyte antigen (HLA) polymorphisms alters peptide antigen presentation	<p><i>In vitro</i> studies identified a mechanism for drug-related autoimmune reactions that could aid the design of safer therapeutics. In cells expressing the HLA-B 5701 polymorphism, which has been linked to abacavir hypersensitivity syndrome, the drug bound to the HLA antigen-binding site, prevented binding of normal antigens and induced binding of self antigens. This led to generation of T cells that targeted the self antigens. In cells expressing the HLA-B 1502 polymorphism, which has been linked to the drug reaction-associated condition Stevens-Johnson syndrome, the HLA-drug interaction and altered peptide binding also were observed. Next steps include studying other drug hypersensitivity reactions associated with HLA haplotypes.</p> <p>SciBX 5(23); doi:10.1038/scibx.2012.615 Published online June 7, 2012</p>	Findings unpatented; unavailable for licensing	<p>Illing, P.T. <i>et al. Nature</i>; published online May 23, 2012; doi:10.1038/nature11147</p> <p>Contact: Jamie Rossjohn, Monash University, Clayton, Victoria, Australia e-mail: jamie.rossjohn@monash.edu</p>