This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Autoimmune disease				
Arthritis; ankylosing spondylitis	IL-23; IL-23 receptor	<p>Mouse studies suggest inhibiting IL-23 signaling in a T cell subpopulation could help treat nonrheumatoid arthritis spondyloarthropathies, such as ankylosing spondylitis. In a mouse model of passive collagen-induced arthritis, IL-23 led to joint inflammation. In the same model, an antibody targeting the IL-23 p19 subunit decreased disease severity and inflammation at tendon-bone attachment sites compared with an isotype control. In those mice, the inflammatory effects of IL-23 were mediated by a subpopulation of IL-23 receptor-positive T cells at tendon-bone attachment sites. Next steps could include evaluating inhibition of IL-23 signaling in additional spondyloarthropathy models.</p> <p>SciBX 5(30); doi:10.1038/scibx.2012.778 Published online Aug. 2, 2012</p>	Patent application filed covering methods to modulate IL-23 activity; licensing status unavailable	<p>Sherlock, J.P. <i>et al. Nat. Med.</i>; published online July 1, 2012; doi:10.1038/nm.2817 Contact: Daniel J. Cua, Merck Research Laboratories, Palo Alto, Calif. e-mail: daniel.cua@merck.com</p>