

### This week in therapeutics

| Indication  | Target/marker/<br>pathway     | Summary   | Licensing status                         | Publication and contact<br>information  |
|---|-------------------------------|---|--|---|
| <b>Neurology</b>  |                               |   |  |   |
| Guillain-Barré syndrome (GBS); chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) | Myelin protein zero (MPZ; P0) | <p>Studies in mice suggest that targeting P0 could be useful for treating GBS or CIDP. CD4<sup>+</sup> T cells generated from the CD4<sup>+</sup> T cells of neuropathic mice were highly specific for P0, suggesting that P0 is a dominant self-antigen recognized by T cells and autoantibodies. T cells from a transgenic mouse specific for a P0 epitope were sufficient to induce a fulminant form of autoimmune peripheral neuropathy resembling GBS and CIDP. Next steps include validating the P0-specific response in humans and exploring its therapeutic potential in animal models of GBS and CIDP.</p> <p><b>SciBX 2(9); doi:10.1038/scibx.2009.371</b><br/>Published online March 5, 2009</p> | Patent status not applicable; unlicensed | <p>Louvet, C. <i>et al. J. Exp. Med.</i>; published online Feb. 16, 2009; doi:10.1084/jem.20082113<br/> <b>Contact:</b> Jeffrey A. Bluestone, University of California, San Francisco, Calif.<br/>                     e-mail: <a href="mailto:jbluest@diabetes.ucsf.edu">jbluest@diabetes.ucsf.edu</a></p> |