

## This week in therapeutics

| Indication       | Target/marker/pathway                                    | Summary  | Licensing status                        | Publication and contact information   |
|------------------|--|--|---|---|
| <b>Neurology</b> |  |  |   |   |
| Nerve damage     | Mothers against decapentaplegic homolog 1 (MADH1; SMAD1) | <p>Studies in mice suggest that enhancing SMAD1 signaling could help treat nerve damage. In mice, dorsal root ganglia with peripheral injury showed greater SMAD1 expression than uninjured neurons. In neuronal cell culture, small interfering RNA-mediated knockdown of SMAD1 decreased neurite length compared with control siRNA treatment (<math>p &lt; 0.001</math>). Transduction with an RNAi-resistant SMAD1 rescued the decreased neurite outgrowth caused by siRNA knockdown. Next steps could include studying the functional role of SMAD1 signaling in nerve repair.</p> <p><b>SciBX 2(24); doi:10.1038/scibx.2009.982</b><br/> <b>Published online June 18, 2009</b></p> | Patent and licensing status unavailable | <p>Zou, H. <i>et al. J. Neurosci.</i>; published online June 3, 2009; doi:10.1523/JNEUROSCI.5397-08.2009<br/> <b>Contact:</b> Marc Tessier-Lavigne, Genentech Inc., South San Francisco, Calif.<br/>                     e-mail: <a href="mailto:marctl@gene.com">marctl@gene.com</a></p> |