

### This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
<b>Cardiovascular disease</b>				
Myocardial infarction (MI)	Alcohol dehydrogenase 5 (ADH5; GSNOR)	<p>Studies in mice suggest that inhibiting ADH5 could be useful for protecting against myocardial injury and preserving cardiac function. In mice, deletion of <i>adh5</i> decreased myocardial infarct size, preserved ventricular systolic and diastolic function and maintained tissue oxygenation following acute coronary artery ligation compared with what was seen in wild-type mice. <i>Adh5</i> deletion also triggered an increase in myocardial capillary density and S-nitrosylation of hypoxia-inducible factor 1 (Hif1A; Hif1), which promotes angiogenesis. Next steps include developing ADH5 inhibitors to prevent protein denitrosylation and designing HIF1A S-nitrosylating compounds to induce angiogenesis.</p> <p><b>SciBX 2(13); doi:10.1038/scibx.2009.533</b>  <b>Published online April 2, 2009</b></p>	Patent application filed; unlicensed	<p>Lima, B. <i>et al. Proc. Natl. Acad. Sci. USA</i>; published online March 23, 2009;            doi:10.1073/pnas.0901043106  <b>Contact:</b> Howard A. Rockman,            Duke University Medical Center,            Durham, N.C.            e-mail:  <a href="mailto:h.rockman@duke.edu">h.rockman@duke.edu</a></p>