



This week in therapeutics

| ndication             | Target/marker/<br>pathway  | Summary  | Licensing<br>status     | Publication and contact information  |
|-----------------------|--|--|-------------------------|--|
| Cardiovascula         | ar disease   |  |                         |  |
| Arterial<br>hrombosis | S100 calcium binding<br>protein A9 (S100A9;<br>calgranulin B; MRP14) | Studies in mice and patients suggest \$100A9 inhibitors could help prevent arterial thrombosis. Patients with acute myocardial infarction (MI) had higher \$100A9 levels in arterial thrombithan patients with stable coronary artery disease (CAD). In mouse models of arterial thrombosis, \$100a9 deficiency decreased thrombin-induced platelet activation and platelet accumulation on arterial walls and increased time to thrombus formation compared with wild-type \$100a9 expression. In a thrombosis assay using human whole blood, an anti-\$100A9 antibody decreased thrombus formation compared with an inactive control antibody. Next steps include investigating the role of \$100A9 in venous thrombosis. Active Biotech AB and Teva Pharmaceutical Industries Ltd. have Nerventra laquinimod, an oral quinoline-3-carboxamide immunomodulator that targets \$100A9, under EMA review to treat multiple sclerosis (MS). The compound also is in Phase II testing to treat Crohn's disease, lupus and Huntington's disease (HD).  Active Biotech and Ipsen Group have tasquinimod (ABR-215050), an oral quinoline-3-carboxamide derivative that binds \$100A9, in Phase II trials to treat gastric, liver, ovarian and renal cancers. Active Biotech's paquinimod (ABR-215757), a small molecule quinoline-3-carboxamide immunomodulator that targets \$100A9, is in Phase II testing to treat lupus. | Patented;<br>unlicensed | Wang, Y. et al. J. Clin. Invest.; publishe online April 1, 2014; doi:10.1172/JCI70966 Contact: Daniel I. Simon, University Hospitals Case Medical Center, Cleveland, Ohio e-mail: daniel.simon@uhhospitals.org |