This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Musculoskeletal disease				
Bone repair; osteoporosis	Hypoxia-inducible factor 1α (HIF1A; HIF1α); notch 1 (NOTCH1)	Mouse studies suggest stimulating expansion of a vascular endothelial vessel subtype in bone could help treat fractures or age-dependent osteoporosis. In mice, immunohistochemical analysis and lineage tracing showed that a specific type of branched, proliferative vasculature in a portion of the bone was associated with osteoprogenitor cells. In aged mice suffering from a loss of this specific type of vasculature and bone mass, activation of Hif1a transcriptional activity led to expansion of this vasculature type and increased both osteoprogenitor cell numbers and bone mass compared with vehicle. In mice, inactivation of notch signaling in endothelial cells decreased endothelial cell proliferation and bone formation compared with what was seen in wild-type controls. In mice with inactivated notch signaling, recombinant noggin (Nog), a secreted bone morphogenetic protein (Bmp) antagonist induced by Notch1, improved vascularization and formation of bone. Next steps could include developing targeted approaches for activating HIF1A or NOTCH1 signaling in bone. *SciBX* 7(16); doi:10.1038/scibx.2014.465 Published online April 24, 2014	Patent and licensing status unavailable	Kusumbe, A.P. *et al. Nature*; published online March 12, 2014; doi:10.1038/nature13145 Ramasamy, S.K. *et al. Nature*; published online March 12, 2014; doi:10.1038/nature13146 **Contact:** Ralf H. Adams, University of Muenster, Muenster, Germany e-mail: ralf.adams@mpi-muenster.mpg.de