This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Autoimmune disease				
Multiple sclerosis (MS)	Integrin $\alpha_4\beta_1$ (VLA-4; CD29/CD49d)	A study in mice suggests that preventing integrin $\alpha_4\beta_1$–mediated T cell adhesion may be useful for preventing MS. In an experimental autoimmune encephalomyelitis (EAE) mouse model, none of the mice injected with integrin $\alpha_4\beta_1$–deficient encephalitis-inducing T cells showed signs of MS disease pathology at 20 days, whereas mice injected with encephalitis-inducing control T cells displayed disease symptoms. EAE mice with an inducible integrin $\alpha_4\beta_1$ deficiency showed delayed development of MS disease symptoms compared with what was seen in wild-type EAE controls. The integrin $\alpha_4\beta_1$–deficient T cells were unable to adhere to the CNS endothelium, which is a known mechanism of disease progression. Next steps include identifying mechanisms of T cell infiltration into the CNS. Tysabri natalizumab, a humanized mAb against integrin α_4 from Elan Corp. plc and Biogen Idec Inc., is marketed to treat MS. At least seven other companies have integrin α_4 or integrin $\alpha_4\beta_1$ inhibitors in Phase II or earlier development for MS. *SciBX* **2**(5); doi:10.1038/scibx.2009.173 Published online Feb. 5, 2009	Work unpatented; available for licensing from Max Planck Innovation GmbH	Bauer, M. *et al. Proc. Natl. Acad. Sci. USA*; published online Jan. 26, 2009; doi:10.1073/pnas.0808909106 **Contact:** Reinhard Fässler, Max Planck Institute of Neurobiology, Martinsried, Germany e-mail: Faessler@biochem.mpg.de