This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Acute myeloid leukemia (AML)	Fc γ-receptor IIa (CD32A; FCGR2A) IL-2 receptor α-chain (CD25)	Studies of human serum samples and of mice suggest that inhibiting CD32A and/or CD25 could help treat AML and prevent disease relapse. Leukemia stem cells are generally resistant to chemotherapy and are responsible for disease relapse. In leukemia stem cells from 32 of 61 AML patients, as compared with normal bone marrow cells, CD32A and/or CD25 were highly expressed. In mice, transplantation of $CD25^+$ stem cells caused AML. Next steps could include developing CD32A and/or CD25 inhibitors and testing them in animal models of AML. ***SciBX*** **3(8); doi:10.1038/scibx.2010.239** **Published online Feb. 25, 2010**	Patent and licensing status unavailable	Saito, Y. *et al. Sci. Transl. Med.*; published online Feb. 4, 2010; doi:10.1126/scitranslmed.3000349 **Contact:** Fumihiko Ishikawa, RIKEN Research Center for Allergy and Immunology, Yokohama, Japan e-mail: f_ishika@rcai.riken.jp