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This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Drug platforms			
Bone marrow niche–sensitizing chemotherapy to enhance antibody-based acute lymphoblastic leukemia (ALL) elimination	Mouse and human sample studies suggest targeting tumor type–specific factors that suppress antitumor immunity could help eliminate residual ALL. In a humanized mouse model of ALL treated with Campath alemtuzumab, shRNA screening of residual leukemia cells identified secreted factors that suppressed antitumor macrophage activity in the bone marrow. In the same mice, Campath plus low doses of cyclophosphamide led to a synergistic, near-complete elimination of tumor cells in the bone marrow caused by cyclophosphamide-dependent blockade of a tumor cell secretory program that suppressed bone marrow–resident macrophages. Next steps include testing low-dose cyclophosphamide inhibition with therapeutic antibodies as treatment for refractory B cell malignancies and investigating specific tumor-secreted, macrophage-suppressing factors. Campath, an anti-CD52 antibody, is marketed by Sanofi to treat chronic lymphocytic leukemia (CLL) and multiple sclerosis (MS). Cyclophosphamide is a generic chemotherapeutic used to treat cancers including lymphoma and leukemia. *SciBX* 7(7); doi:10.1038/scibx.2014.208 Published online Feb. 20, 2014	Patents filed covering the humanized ALL model and treatment with low-dose cyclophosphamide as an antibody-sensitizing agent; available for licensing	Pallasch, D.P. *et al. Cell*; published online Jan. 30, 2014; doi:10.1016/j.cell.2013.12.041 **Contact:** Michael T. Hemann, Massachusetts Institute of Technology, Cambridge, Mass. e-mail: hemann@mit.edu **Contact:** Jianzhu Chen, same affiliation as above e-mail: jchen@mit.edu