This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Cancer				
Chronic myelogenous leukemia (CML)	BCR-ABL tyrosine kinase; macrophage inflammatory protein-1 α (CCL3; MIP1A)	<p>Mouse studies suggest inhibiting CCL3 signaling could help treat CML. In a newly developed, nonirradiated mouse model of CML using BCR-ABL-expressing, leukemia-initiating cells, <i>Ccl3</i> knockout delayed disease relapse following cessation of Gleevec imatinib therapy and increased survival compared with no knockout. Next steps include studies to identify progenitor cell subsets that compete with leukemia-initiating cells and to understand how such cells are maintained in the bone marrow niche. Novartis AG markets Gleevec to treat CML and gastrointestinal stromal tumors.</p> <p>SciBX 6(46); doi:10.1038/scibx.2013.1316 Published online Dec. 5, 2013</p>	Patent application filed; licensing details available from Kanazawa University	<p>Baba, T. <i>et al. J. Exp. Med.</i>; published online Oct. 28, 2013; doi:10.1084/jem.20130112 Contact: Tomohisa Baba, Kanazawa University, Ishikawa, Japan e-mail: sergenti@staff.kanazawa-u.ac.jp</p>