This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Acute myelogenous leukemia (AML)	<i>Cadherin-like 24 (CDH24); solute carrier family 15 (oligopeptide transporter), member 1 (SLC15A); kinase non-catalytic C-lobe domain (KIND) containing 1 (KNDC1); protein tyrosine phosphatase, receptor type, T (PTPRT); glutamate receptor, ionotropic, N-methyl D-aspartate-like 1B (GRINL1B); G protein-coupled receptor 123 (GPR123); G protein-coupled receptor 183 (GPR183; EBI2); protocadherin 24 (PCDH24; PC-LKC); fms-related tyrosine kinase 3 (FLT3); nucleophosmin (nucleolar phosphoprotein B23, numatrin) (NPM1)</i>	Whole-genome sequencing data could be used to identify new targets in AML. The complete genome of tumor cells from an AML patient was sequenced and compared against the complete genome of normal skin cells from the same subject. Mutations were found in two genes previously linked to AML (<i>FLT3</i> and <i>NPM1</i>) and eight other genes (<i>CDH24</i> , <i>SLC15A</i> , <i>KNDC1</i> , <i>PTPRT</i> , <i>GRINL1B</i> , <i>GPR123</i> , <i>GPR183</i> , <i>PCDH24</i>) with a variety of functions. Next steps include sequencing the genomes of AML cell lines from other patients to determine which of the mutations are conserved across AML tumors and testing the effect of these mutations in preclinical models of AML.	Unpatented; licensing status not applicable	Ley, T. <i>et al. Nature</i> ; published online Nov. 6, 2008; doi:10.1038/nature07485 Contact: Elaine Mardis, Washington University School of Medicine, St. Louis, Mo. e-mail: emardis@wustl.edu
		SciBX 1(43); doi:10.1038/scibx.2008.1040 Published online Dec. 4, 2008		