

This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Cancer				
Cancer	v-Myc myelocytomatosis viral oncogene homolog (MYC); B-cell lymphoma 2 (BCL2; BCL-2); signal transducer and activator of transcription 3 (STAT3); STAT5; c-jun N-terminal kinase (JNK); mitogen-activated protein kinase kinase 1 (MEK1); MEK2; caspase-3 apoptosis-related cysteine peptidase (CASP3; CPP32); monophosphorylated mitogen-activated protein kinase 1 (MAPK1; ERK2)	<p><i>In vitro</i> and <i>in vivo</i> studies suggest that detecting changes in oncoprotein activation states using a nanofluidic proteomic immunoassay could improve the monitoring of cancer treatments or help develop new therapeutics. Using a few nanoliters of sample, changes in MYC levels were detectable in Burkitt's lymphoma cell lines compared with what was seen in benign lymph nodes. In a human chronic myelogenous leukemia (CML) cell line treated with a tyrosine kinase inhibitor, changes in STAT3, STAT5, JNK, MEK1, MEK2 and CASP3 were detectable following treatment. In CML patients treated with tyrosine kinase inhibitors, MAPK1 levels were lower in responding patients. Next steps include validating the method in controlled clinical trials.</p> <p>SciBX 2(17); doi:10.1038/scibx.2009.699 Published online April 30, 2009</p>	Patent pending covering findings; available for licensing through Cell BioSciences Inc.	Fan, A. <i>et al. Nat. Med.</i> ; published online March 12, 2009; doi:10.1038/nm.1903 Contact: Dean W. Felsher, Stanford University, Stanford, Calif. e-mail: dfelsher@stanford.edu