This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Cancer	Fibroblast growth factor receptor (FGFR); IL-6; JAK kinase (JAK); phosphoinositide 3-kinase (PI3K); signal transducer and activator of transcription 3 (STAT3)	Mouse and *in vitro* studies suggest inhibiting STAT3 signaling could help circumvent cancer resistance to tyrosine kinase inhibitors. In a panel of human cancer cell lines, multiple classes of tyrosine kinase inhibitors increased STAT3 activation compared with vehicle, and STAT3 activation was associated with drug resistance. STAT3 activation was shown to occur via the IL-6/JAK and FGFR/PI3K signaling pathways. In a mouse xenograft model of non–small cell lung cancer (NSCLC), combined treatment with a tyrosine kinase inhibitor plus inhibitors of JAK or PI3K led to tumor regression, whereas treatment with individual agents or vehicle did not. Next steps could include developing and validating a diagnostic assay to detect emergence of tyrosine kinase inhibitor resistance via STAT3 activation.	Patent and licensing status unavailable	Lee, H.-J. *et al. Cancer Cell*; published online July 24, 2014; doi:10.1016/j.ccr.2014.05.019 **Contact:** Jeff Settleman, Genentech Inc., South San Francisco, Calif. e-mail: settleman.jeffrey@gene.com

SciBX 7(34); doi:10.1038/scibx.2014.1009
Published online Sept. 4, 2014