## This week in therapeutics

| Indication | Target/marker/ pathway | Summary | Licensing status | Publication and contact information |
|---|---|---|---|---|
| **Cancer** | | | | |
| Acute myelogenous leukemia (AML) | FMS-like tyrosine kinase 3 (FLT3; CD135) | *In vitro* and mouse studies suggest the selective small molecule FLT3 inhibitor G-749 could help treat AML with resistance mutations in *FLT3*. In leukemia cell lines expressing wild-type or drug-resistant *FLT3* mutants, G-749 decreased proliferation compared with the FLT3 inhibitor quizartinib or midostaurin, which both inhibit FLT3 activity. In mouse xenograft models of leukemia, the inhibitor caused complete tumor regression, and it increased survival compared with vehicle. In bone marrow blasts from patients with *FLT3*-mutant AML, G-749 increased cell death compared with quizartinib or midostaurin. Next steps include preclinical toxicity testing of G-749 and starting a Phase I trial in patients with *FLT3*-mutant AML.<br>Genosco's G-749 is in preclinical development for AML.<br>Ambit Biosciences Corp.'s quizartinib is in Phase II testing to treat AML.<br>Novartis AG has the protein kinase C (PKC) inhibitor midostaurin in Phase III testing to treat AML and other hematological malignancies.<br>Ariad Pharmaceuticals Inc. markets the FLT3 inhibitor Iclusig ponatinib to treat leukemia.<br>At least six additional companies have FLT3 inhibitors in Phase II or earlier testing to treat cancer. | Patented; available for licensing | Lee, H.K. *et al. Blood*; published online Feb. 14, 2014; doi:10.1182/blood-2013-04-493916<br>**Contact:** Ho-Juhn Song, Genosco, Cambridge, Mass.<br>e-mail: hsong@genosco.com |

*SciBX* 7(12); doi:10.1038/scibx.2014.337
Published online March 27, 2014