This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Acute myelogenous leukemia (AML)	Nucleophosmin (nucleolar phosphoprotein B23, numatrin) (NPM1; NPM); F-box and WD-40 domain protein 7 (Fbxw7)	*In vivo* studies suggest that targeting mutated NPM could help treat AML and other leukemias. In mouse embryos, NPM knockout led to downstream c-Myc stabilization in the cytoplasm. High c-Myc levels and NPM mutations that lead to aberrant cytoplasmic localization of NPM have been previously associated with AML. In NPM knockout mouse embryos, absence of the protein led to nuclear localization and degradation of the Fbxw7 isoform Fbxw7γ, leading to c-Myc stabilization in the cytoplasm. Future studies in AML animal models with the NPM mutation are necessary to determine if blocking NPM degradation or NPM nuclear export can treat the cancer.	Research not patented; unavailable for licensing	Bonetti, P. *et al. J. Clin. Biol.*; published online July 14, 2008; doi:10.1083/jcb.200711040 **Contact:** Emanuela Colombo, The FIRC Institute of Molecular Oncology Foundation, Milan, Italy e-mail: emanuela.colombo@ifom-ieo-campus.it **Contact:** Pier G. Pelicci, same affiliation as above e-mail: piergiuseppe.pelicci@ifom-ieo-campus.it