This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Cancer				
Cancer	β -Catenin (CTNNB1); B cell CLL lymphoma 9 (BCL9); wingless-type MMTV integration site (WNT)	<p>Mouse studies suggest disrupting the interaction between CTNNB1 and the WNT pathway coactivator BCL9 could help treat WNT-driven cancers. The stapled peptide SAH-BCL9 was generated by incorporating a stabilizing hydrocarbon staple into a peptide derived from the helical domain of BCL9 that interacts with CTNNB1. In xenograft mouse models of WNT-driven colorectal cancer and multiple myeloma (MM), SAH-BCL9 decreased tumor angiogenesis and primary or metastatic tumor burden compared with a control peptide. Next steps include determining the range of cancer subtypes driven by the BCL9-CTNNB1 interaction.</p> <p>SciBX 5(37); doi:10.1038/scibx.2012.977 Published online Sept. 20, 2012</p>	Patent application filed; available for licensing	<p>Takada, K. <i>et al. Sci. Transl. Med.</i>; published online Aug. 22, 2012; doi:10.1126/scitranslmed.3003808 Contact: Daniel Ruben Carrasco, Dana-Farber Cancer Institute, Boston, Mass. e-mail: ruben_carrasco@dfci.harvard.edu Contact: Loren D. Walensky, same affiliation as above e-mail: loren_walensky@dfci.harvard.edu</p>