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This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Cancer				
Cancer	Serine/threonine kinase 33 (STK33); heat shock protein 90 (Hsp90)	Studies in cell culture suggest blocking the interaction between STK33 and Hsp90 could help treat tumors driven by activating mutations in *K-Ras*. Previous studies have shown divergent results on whether antagonizing STK33 kinase activity would be an effective cancer treatment. In lysates from cultured colon and lung tumor lines, Hsp90 coimmunoprecipitated with STK33. In *K-Ras*-mutant tumor lines, pharmacological inhibition or small hairpin RNA knockdown of Hsp90 decreased STK33 levels and increased apoptosis compared with those seen in control cells with wild-type *K-Ras*. In an *in vitro* tumor growth assay, overexpression of STK33 blocked Hsp90 inhibitor–mediated growth prevention. Next steps include understanding the kinase-independent roles of STK33 in promoting growth in *K-Ras*-mutant tumors. At least 12 companies have Hsp90 inhibitors in preclinical through Phase III development for cancer. *SciBX* 5(17); doi:10.1038/scibx.2012.436 Published online April 26, 2012	Unpatented; licensing status not applicable	Azoitei, N. *et al. J. Exp. Med.*, published online April 9, 2012; doi:10.1084/jem.20111910 **Contact:** Claudia Scholl, University of Ulm, Ulm, Germany e-mail: claudia.scholl@uni-ulm.de **Contact:** Stefan Fröhling, same affiliation as above e-mail: stefan.froehling@uni-ulm.de