This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Prostate cancer	Androgen receptor; protein kinase DNA-activated catalytic polypeptide (PRKDC; DNAPK)	Cell culture and mouse studies suggest inhibiting components of the DNA damage response could help treat prostate cancer. In cell culture and mouse xenograft models of prostate cancer, anti–androgen receptor therapies increased sensitivity to DNA damage, and DNA-damaging therapies such as radiation increased androgen receptor–dependent expression of genes encoding DNA repair proteins. In these models, radiation plus anti–androgen receptor therapy decreased cell growth compared with either treatment alone. In cultured prostate cancer cells, dihydrotestosterone (DHT) partially reversed the growth inhibitory effect of a DNAPK inhibitor plus radiation. Next steps include screening DNAPK inhibitors in disease models. *SciBX* 6(41); doi:10.1038/scibx.2013.1160 Published online Oct. 24, 2013	Work from first study unpatented; licensing status not applicable Patent and licensing status unavailable for second study	Goodwin, J.F. *et al. Cancer Discov.*; published online Sept. 11, 2013; doi:10.1158/2159-8290.CD-13-0108 **Contact:** Karen E. Knudsen, Thomas Jefferson University, Philadelphia, Pa. e-mail: karen.knudsen@jefferson.edu Polkinghorn, W.R. *et al. Cancer Discov.*; published online Sept. 11, 2013; doi:10.1158/2159-8290.CD-13-0172 **Contact:** Charles L. Sawyers, Memorial Sloan-Kettering Cancer Center, New York, N.Y. e-mail: sawyersc@mskcc.org