This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Melanoma	BRAF; checkpoint kinase 1 (Chk1; CHEK1); Chk2 (CHEK2); ataxia telangiectasia mutated (ATM); VEGF	*In vitro* and mouse studies suggest inhibiting the DNA damage repair pathway could help treat melanoma. In mice, transplantation of melanoma cells expressing shRNA against *DNA damage repair* (*DDR*) genes *CHEK1*, *CHEK2* or *ATM* led to delayed tumor growth versus transplantation of cells expressing control shRNA. In mice, activation of *DDR* genes was higher during hypoxia. In mouse models of BRAF inhibitor–resistant and BRAF inhibitor–sensitive melanomas, a CHEK1 and CHEK2 inhibitor plus the anti-VEGF antibody Avastin bevacizumab, which increases tumor hypoxia, synergistically inhibited tumor growth. Next steps include reproducing the animal model results and performing clinical testing. Pfizer Inc. has the ATM inhibitor CP-466722 in preclinical development for cancer. Chugai Pharmaceutical Co. Ltd., Roche and its Genentech Inc. unit market Avastin to treat various cancers.	Patent and licensing status unavailable	Possik, P.A. *et al. Cell Rep.*; published online Nov. 6, 2014; doi:10.1016/j.celrep.2014.10.024 **Contact:** Daniel S. Peeper, The Netherlands Cancer Institute, Amsterdam, the Netherlands e-mail: d.peeper@nki.nl

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