This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Cancer				
Skin cancer	BRAF; c-jun N-terminal kinase (JNK)	<p><i>In vitro</i> and mouse studies suggest the BRAF inhibitor Zelboraf vemurafenib could induce cutaneous squamous cell carcinoma (cSCC) by inhibiting JNK signaling. Treatment of melanoma with BRAF inhibitors can increase the incidence of cSCC. In cSCC cells and keratinocytes, Zelboraf suppressed JNK activation and apoptosis by inhibiting kinases upstream of JNK including sterile α-motif and leucine zipper containing kinase AZK (ZAK). Next steps include using this information to design melanoma therapeutics that do not reduce JNK signaling. Daiichi Sankyo Co. Ltd., Chugai Pharmaceutical Co. Ltd. and Roche market Zelboraf to treat melanoma. GlaxoSmithKline plc markets the BRAF inhibitor Tafinlar dabrafenib to treat melanoma. At least four other companies have BRAF inhibitors in Phase III or earlier testing to treat cancers.</p> <p>SciBX 6(47); doi:10.1038/scibx.2013.1347 Published online Dec. 12, 2013</p>	Patent status not applicable; unavailable for licensing	<p>Vin, H. <i>et al. eLife</i>; published online Nov. 5, 2013; doi:10.7554/eLife.00969 Contact: Kenneth Y. Tsai, The University of Texas MD Anderson Cancer Center, Houston, Texas e-mail: kytsai@mdanderson.org</p>