

### This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
<b>Cancer</b>				
Non-small cell lung cancer (NSCLC)	MEK; epidermal growth factor receptor (EGFR)	<p>Cell culture studies suggest antagonizing MEK or EGFR could be useful for treating <i>BRAF</i> mutation-positive NSCLC. About 6%–8% of NSCLC tumors have activating mutations in <i>BRAF</i>. In cultured NSCLC cells and human tissue samples with activating <i>BRAF</i> mutations, acquired resistance to BRAF inhibitors was associated with elevated MEK activation or constitutive EGFR signaling. In cell culture, small molecule MEK or EGFR inhibitors decreased treatment-acquired BRAF inhibitor resistance compared with vehicle controls. Next steps could include testing combinations of MEK or EGFR inhibitors with BRAF inhibitors in animal models of NSCLC.</p> <p>Tafinlar dabrafenib from GlaxoSmithKline plc and Zelboraf vemurafenib from Roche and Daiichi Sankyo Co. Ltd. are BRAF inhibitors marketed to treat BRAF-mutant melanoma. Tafinlar is also in Phase II testing for NSCLC. GSK also markets Mekinist trametinib, a small molecule MEK inhibitor, to treat BRAF-mutant melanoma.</p> <p>At least 12 companies have MEK inhibitors in Phase III or earlier testing to treat various cancers.</p> <p>More than a dozen EGFR inhibitors are marketed or in late-stage clinical development for a range of cancers.</p> <p><b>SciBX 7(9); doi:10.1038/scibx.2014.253</b>  <b>Published online March 6, 2014</b></p>	Patent and licensing status undisclosed	<p>Lin, L. <i>et al. Proc. Natl. Acad. Sci. USA</i>; published online Feb. 3, 2014; doi:10.1073/pnas.1320956111</p> <p><b>Contact:</b> Trever G. Bivona, University of California, San Francisco, Calif.  e-mail:  <a href="mailto:trever.bivona@ucsf.edu">trever.bivona@ucsf.edu</a></p>