This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Cancer				
Melanoma	Mitochondrial oxidative phosphorylation	Cell culture and mouse studies suggest inhibiting oxidative phosphorylation could help treat drug-resistant melanoma. In cultured, drug-resistant melanoma cells that highly expressed jumonji AT rich interactive domain 1B (JARID1B; PLU-1), multiple proteins involved in oxidative phosphorylation were overexpressed compared with expression in control cells. A series of compounds that inhibit mitochondrial ATP production increased cell death compared with vehicle. In xenograft mouse models for melanoma, combining inhibitors of oxidative phosphorylation with anticancer drugs decreased tumor growth compared with either set of compounds alone. Next steps include understanding the basis for mitochondria-mediated drug resistance in JARID1B-overexpressing cells and could include a clinical trial of Zelboraf vemurafenib plus phenformin in melanoma. Roche, Daiichi Sankyo Co. Ltd. and Chugai Pharmaceutical Co. Ltd. market Zelboraf vemurafenib, a small molecule inhibitor of BRAF V600E, for melanoma. Phenformin, a biguanide that inhibits oxidative phosphorylation, is a generic diabetes drug. *SciBX* 6(28); doi:10.1038/scibx.2013.718 Published online July 25, 2013	Patent and licensing status undisclosed	Roesch, A. *et al. Cancer Cell*; published online June 10, 2013; doi:10.1016/j.ccr.2013.05.003 **Contact:** Meenhard Herlyn, The Wistar Institute, Philadelphia, Pa. e-mail: herlynm@wistar.org **Contact:** Alexander Roesch, Saarland University Hospital, Homburg, Germany e-mail: alexander.roesch@uks.eu