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Exploring the response of *BRAF* or *PIK3CA* mutated cancers to pathway-targeted inhibition using mouse models

Martin McMahon
University of California, USA

This lecture will address the utility of genetically engineered (GEM) and patient-derived mouse xenograft (PDX) models of cancer to explore mechanisms of oncogene-tumor suppressor cooperation in cancer initiation, progression and therapy. The lecture will emphasize the importance of the quality and quantity of signal pathway activation in sustaining cancer cell proliferation and how such phenomenon may be utilized to prevent the onset of drug resistant disease.

Biography

Martin McMahon research program focuses on the mechanisms underlying the development and treatment of metastatic melanoma, lung and thyroid cancer. Although these malignancies are derived from distinct cell types, they share a striking number of common genetic alterations especially activating mutations in *KRAS, BRAF or PIK3CA*. To do this, McMahon's laboratory works with cultured human cancer-derived cells and with genetically engineered mouse models of human cancer. He has served on the editorial boards for the *Molecular & Cellular Biology* and as a Senior Editor of *Molecular Cancer Research*. He is Assistant Director for Professional Education and Co-Leader of the Developmental Therapeutics Program at the U.C. San Francisco, Helen Diller Family Comprehensive Cancer Center. He is also the President-Elect of the Society for Melanoma Research and the Chair of the Basic Mechanisms of Cancer Therapy NIH study section.

mcmahon@cc.ucsf.edu