Elizabeth Allen et al., Madridge J Cancer Stud Res. 2016
http://dx.doi.org/10.18689/2640-5180.a1.004

madridge CONFERENCES

International
Cancer Study & Therapy Conference

April 4-6, 2016 Baltimore, USA

# Metabolic adaptation to anti-angiogenic therapy

**Elizabeth Allen[1], Pascal Miéville[2], Carmen Warren[3], Leanne Li[1], Mei-Wen Peng[1]** and **Douglas Hanahan[1,4,5]**
[1]The Swiss Institute for Experimental Cancer Research (ISREC), Switzerland
[2]The Institute of Chemical Sciences & Engineering (ISIC-SB-EPFL), Switzerland
[3]Department of Molecular, Cell and Developmental Biology; University of California, USA
[4]The Swiss Cancer Center Lausanne (SCCL), Swiss Federal Institute of Technology Lausanne, Switzerland

Although anti-angiogenic therapy has demonstrable efficacy in mouse models of cancer and in certain types of human cancer, responses are typically transitory, followed by resumption of malignant progression that limits survival benefit. Unconventional modes of evasive/adaptive resistance underlay the eventual failures of anti-angiogenic therapy. Functionally established resistance mechanisms include revascularization mediated by alternative pro-angiogenic signals, protection by peri-vascular macrophages and monocytes, and normal vessel cooption via heightened local invasion and distant metastasis. All three mechanisms have been documented in the prototypical RIP-Tag2 mouse model of multistep tumorigenesis, in which the angiogenic switch is a discrete step in the pathway to invasive pancreatic neuroendocrine cancer. New results to be presented identify a fourth mode of adaptive resistance to potent anti-angiogenic therapy in this model: metabolic adaptation, whereby cancer cells adopt distinctive metabolic states, such that they can be alternatively fueled by glucose or lactate, in a form of metabolic symbiosis.

**Biography:**

Elizabeth Allen received a BS in biology, and an MS/PhD in Human Genetics at the University of Michigan. She worked as part of the Sharpiro's team that cloned the steroid sulfatase gene, and performed structure-function studies of the in von Willebrand Factor protein in the Ginsburg lab. Her postdoctoral research in the Fuchs lab involved engineering mouse models with desmosomal mutations in order to potentially identify associated human skin diseases. She became a staff scientist at Stanford University, where she worked with the Yang lab to help identify the role of the proteasome in Giant Axonal Neuropathy. She joined the Hanahan group at UCSF and EPFL to use mouse PanNET models to investigate the mechanisms of resistance to antiangiogenic therapy.