



Research

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GRANT SNAPSHOT

2014 Pancreatic Cancer Action Network – AACR Research Acceleration Network Grant

Grantees: PI: Giulio Draetta, MD, PhD
Institutions: MD Anderson Cancer Center

Co-PI: Lewis Cantley, PhD
Weill Cornell Medical College



Research Project: *Developing a novel oxidative phosphorylation inhibitor in pancreatic cancer*
Award Period: July 1, 2014 – June 30, 2017
Amount: \$1,000,000

Biographical Highlights

Dr. Draetta earned his medical and graduate degrees from the University of Naples Medical School. He then moved to industry where he co-founded the biotechnology company Mitotix prior to accepting executive appointments with Pharmacia and Merck Research Laboratories. Prior to joining MD Anderson, Dr. Draetta served as Chief Research Business Development Officer and Deputy Director of the Belfer Institute for Applied Cancer Science at the Dana-Farber Cancer Institute. Dr. Draetta currently serves as the Director of the MD Anderson Institute for Applied Cancer Science and is a Professor of Molecular and Cellular Oncology.

Dr. Cantley is the Margaret and Herman Sokol Professor and Director of the Cancer Center at Weill Cornell Medical College/New York Presbyterian Hospital. Prior to joining Cornell, Dr. Cantley was a Professor of Systems Biology at Harvard Medical School and the Director of Beth Israel Deaconess Cancer Center. Dr. Cantley's laboratory discovered the PI 3-Kinase pathway that plays a critical role in insulin signaling and in cancers. Two postdoctoral fellows in Dr. Cantley's laboratory, Costas Lyssiotis, PhD and Gina DeNicola, PhD, were awarded Pathway to Leadership grants from the Pancreatic Cancer Action Network in 2013 and 2014, respectively.

Project Overview

Mutation in KRAS is well known as an early and critical event in the initiation and progression of pancreatic tumors. In addition to its role in activating cell growth and promoting survival, mutant KRAS has also been shown to be involved in cellular metabolism pathways (the ways that cells break down nutrients as a source of energy). Drs. Draetta and Cantley observed that pancreatic cancer cells that are dependent on mutant KRAS signaling require a specific type of metabolic process, called oxidative phosphorylation, or OXPHOS. In contrast, noncancerous cells get their energy from the breakdown of sugar by a completely different method. Encouragingly, the research team has already developed novel drugs that block OXPHOS, and these drugs have shown progress in stopping the growth of pancreatic tumors in mice. Therefore, the goal of this grant proposal is to combine OXPHOS inhibition with drugs that block signaling generated by mutant KRAS, with the intention of initiating a clinical trial of this combination regimen.