GRANT SNAPSHOT

2012 Blum-Kovler – Pancreatic Cancer Action Network – AACR Innovative Grant

Grantee:	Tyler Jacks, PhD
Institution:	Massachusetts Institute of Technology
Research Project:	*Mechanisms of K-RAS Independent Growth in Pancreatic Cancer*
Award Period:	July 1, 2012 – June 30, 2014
Amount:	$200,000

Biographical Highlights

Dr. Jacks has been a member of the Massachusetts Institute of Technology (MIT) faculty since 1992 and has trained over 45 postdoctoral fellows and graduate students, including Dave Tuveson, MD, PhD (recipient of a 2003 Pancreatic Cancer Action Network Career Development Award and Emeritus Scientific Advisory Board member) and Ken Olive, PhD (recipient of a 2011 Pancreatic Cancer Action Network Career Development Award, funded by Tempur-Pedic® Retailers). Dr. Jacks is currently a Professor of Biology in the Department of Biology and Center for Cancer Research and Director of the David Koch Institute for Integrative Cancer Research at MIT. He received his undergraduate degree at Harvard, PhD at University of California, San Francisco, and did his postdoctoral studies at the Whitehead Institute for Biomedical Research in Cambridge, MA.

The focus of Dr. Jacks' research is centered on the use of gene targeting to create more accurate mouse models of human cancer and to explore the pathways regulated by cancer-associated genes. These mouse models are being evaluated with cutting-edge tools in genetics, genomics, and imaging, as well as with various chemotherapeutic agents.

Project Overview

The vast majority of pancreatic tumors are known to express a mutant version of the protein K-Ras. When mutated, K-Ras becomes constantly active and signals cells to continue growing and ignore cues to stop growing. K-Ras is thought to be an early event in the development of pancreatic cancer, and mutation of this protein alone can lead to precancerous abnormalities.

Dr. Jacks' expertise in mouse modeling of human disease will allow him to establish a novel model of pancreatic cancer development. Here, the mice will express mutant K-Ras specifically in the pancreas, so that early pancreatic tumors will form. Then, the expression of K-Ras will be turned off to study other protein signaling pathways that are involved in the progression of pancreatic cancer, and independent from signaling originating from mutant K-Ras. Dr. Jacks' goals are to investigate the mechanisms of K-Ras-independent growth in mouse models of pancreatic cancer, and validate these findings in human and mouse cells.