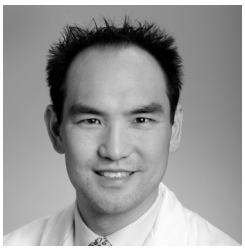
GRANT SNAPSHOT

2012 Pancreatic Cancer Action Network – AACR Career Development Award

Grantee:	David Yu, MD, PhD
Institution:	Emory University
Research Project:	*Exploiting the Replication Stress Response in Pancreatic Cancer*
Award Period:	July 1, 2012 – June 30, 2014
Amount:	$200,000

Biographical Highlights



Dr. Yu is an Assistant Professor in the Department of Radiation Oncology at Emory University School of Medicine. He received his MD from the University of Texas at Southwestern Medical School and PhD from the University of Cambridge, and then completed residency training in Radiation Oncology at Vanderbilt University Medical Center. He graduated with honors from Stanford University with a BS in Biology. Dr. Yu is actively engaged in both clinical and basic science research. He is interested in understanding how cells respond to replication stress and how we can utilize this knowledge for improvements in cancer diagnosis and treatment. The ultimate goal of his work is to translate insights gained from his interactions with his patients and the laboratory to innovative therapies to improve the quality of care for patients with cancer.

Dr. Yu is a Georgia Cancer Coalition Distinguished Cancer Scholar. He has been awarded funding as Principal Investigator of grants from the National Institutes of Health (NIH)/National Cancer Institute (NCI) and Department of Defense (DOD), and has authored a number of publications in high impact journals, including *Nature*, *Cell*, *Molecular Cell*, and *EMBO*.

Project Overview

The standard of care chemotherapy for pancreatic cancer, gemcitabine, acts by inducing damage to cancer cells' DNA. However, all cells have complex mechanisms to recognize and repair DNA damage. The protein ATR (ATM and Rad3 related) checkpoint kinase behaves by identifying improperly repaired DNA. ATR finds single-strand breaks of DNA (the molecule is supposed to exist as a double strand), and activates a cascade of proteins to fix the damage.

Dr. Yu proposes to find proteins involved in the ATR signaling pathway, and determine whether they could be viable drug targets to sensitize pancreatic cancer cells to gemcitabine and other DNA damage-inducing treatments. Expression and activity of proteins can be positively and negatively regulated by drug intervention, so Dr. Yu and his colleagues will perform screens to identify which genes and proteins' alterations cause pancreatic cancer cells to respond better to gemcitabine. He will also study whether the presence and expression level of ATR and its related proteins may predict which patients will respond best to certain treatment strategies.