



**Research**

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## GRANT SNAPSHOT

### 2014 Pancreatic Cancer Action Network – AACR Career Development Award

Grantee:	Kenneth Scott, PhD
Institution:	Baylor College of Medicine
Research Project:	<i>Functionalizing Metabolic Pathway Driver Aberrations in Pancreatic Cancer</i>
Award Period:	July 1, 2014 – June 30, 2016
Amount:	\$200,000

### Biographical Highlights



Dr. Scott underwent his undergraduate studies at Texas State University, then did his PhD work at Baylor College of Medicine. From there, he moved to the Dana-Farber Cancer Institute in Boston for his postdoctoral training. His work at Dana-Farber centered on cancer gene discovery through the use of comparative oncogenomics, genetic screens, and downstream functional and clinicopathological target validation.

Dr. Scott returned to Baylor in 2010 as an Assistant Professor in the Department of Molecular and Human Genetics. Since returning to Baylor, Dr. Scott has continued his work in cancer gene discovery for which he has developed innovative genetic screening tools permitting functional characterizations of large numbers of gene mutations found in pancreatic tumors.

### Project Overview

The vast majority of pancreatic tumors have a mutated version of a gene called KRAS, which is thought to play an important role in tumor initiation and progression. Inhibiting KRAS activity or expression has not been successful to date. Therefore, there is a strong need to identify other abnormalities within pancreatic tumors that could be exploited to treat this devastating disease.

Dr. Scott and his colleagues utilized data gathered from large-scale efforts to sequence pancreatic tumors, and have found other genetic abnormalities present in these tumors. One gene, NADK, was found to be mutated in pancreatic cancer samples. Mutations in NADK had not previously been discovered in pancreatic cancer, and Dr. Scott's preliminary experiments suggested that NADK mutation can drive progression of pancreatic tumors. NADK is involved in cells' ability to produce metabolites (byproducts of breaking down nutrients) necessary for defense against cellular damage among other important cell processes. Based on his findings so far, Dr. Scott proposes to use a panel of pancreatic cancer cell lines for a series of functional and metabolic assays to better understand the mechanisms-of-action for both normal and mutated NADK. He and his colleagues will also assess whether NADK could serve as a new drug target by examining tumor growth and signaling through metabolic pathways following inhibiting NADK expression or activity. Finally, Dr. Scott and his research team will use large scale genomics data collected from pancreatic cancer tumors to create and examine collections of other mutated genes with high probability of impacting metabolic pathways. The goal of this project is to lead to new drug targets and/or detection biomarkers critically needed for pancreatic cancer patients who currently have no other effective treatment options.