

PANCREATIC CANCER ACTION NETWORK

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GRANT SNAPSHOT

2010 Samuel Stroum – Pancreatic Cancer Action Network – AACR Fellowship Award

Grantee: Vikram Bhattacharjee, PhD Institution: Fox Chase Cancer Center

Research Project: Candidate Gene Validation of Sensitizers of Pancreatic Cancer to Gemcitabine

Award Period: July 1, 2010 – June 30, 2011

Amount: \$45,000



Biographical Highlights

Dr. Bhattacharjee received his PhD in Biological Sciences from University of South Carolina in Columbia. He joined the Fox Chase Cancer Center in 2009 as a Postdoctoral Fellow. Dr. Bhattacharjee's interest in oncology dates back to college when he became aware of the efforts that were being made to combat the disease. He thought that looking at the whole genomes of different cancers could provide critical information that can help in our fight against the disease. Currently, Dr. Bhattacharjee is working to identify protein targets

for established and novel drug therapies that can sensitize pancreatic cancer cells in patients and contribute to improving patient survival. He has co-authored several articles published in *Molecular Biology and Evolution*, *Journal of Molecular Evolution* and *Molecular Cellular Biology*.

Project Overview

Gemcitabine remains the standard of care chemotherapy for pancreatic cancer patients, despite limited clinical responses. Dr. Bhattacharjee's goal is to identify drugs that could be administered in combination with gemcitabine, to increase the drug's effectiveness.

Dr. Bhattacharjee's experimental strategy was to conduct a large-scale screen for genes that are responsible for a patient's responsiveness to gemcitabine. All 23,000 genes in the human genome were individually turned off, and outcomes were determined. Results suggested that the expression of 122 genes affected the cells' reaction to gemcitabine. Among these, they found multiple genes involved in the cell cycle and DNA replication. Gemcitabine is known to function by inhibiting DNA replication, but Dr. Bhattacharjee observed that gemcitabine also causes cells to pause in their cell cycle, allowing time for DNA replication to recover. Therefore, he is proposing to combine gemcitabine with a drug to bypass the cell cycle arrest, forcing the cells to prematurely start replicating before their DNA is duplicated, leading to cell death. Dr. Bhattacharjee is analyzing this and other mechanisms by which to increase pancreatic cancer cells' sensitization to gemcitabine. If successful, this approach could greatly increase the effectiveness of gemcitabine in treating pancreatic cancer patients.