



Research

PANCREATIC CANCER ACTION NETWORK

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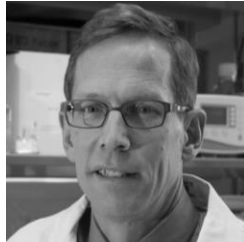
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GRANT SNAPSHOT

2013 Tempur-Pedic – Pancreatic Cancer Action Network – AACR Inaugural Research Acceleration Network Grant in Memory of Tim Miller

Grantees: PI: Robert Vonderheide, MD, DPhil
Institutions: University of Pennsylvania

Co-PI: Dafna Bar-Sagi, PhD
New York University



Research Project: *Accelerating Development of CD40 Therapy for Pancreatic Cancer*
Award Period: July 1, 2013 – June 30, 2016
Amount: \$1,000,000

Biographical Highlights

Dr. Vonderheide is an Associate Professor of Medicine, and Associate Director of Translational Research for the Abramson Cancer Center at University of Pennsylvania. Dr. Vonderheide is a medical oncologist whose research is focused on the immunobiology of pancreatic cancer. He is a national expert in his field and serves as a member of the Scientific Advisory Board for the Pancreatic Cancer Action Network.

Dr. Bar-Sagi is the Senior Vice President and Vice Dean for Science, Chief Scientific Officer of NYU Langone Medical Center. She is a leading cancer biologist and is widely known for her research which is focused on the molecular and cellular mechanisms involved in pancreatic cancer development. She has published over 100 peer-reviewed articles in leading scientific journals. She serves on the Scientific Advisory Board of the Pancreatic Cancer Action Network.

Project Overview

The immune system is the body's natural biological mechanism that protects against disease. An immune response is a complicated biological process that is strategically and elegantly activated to destroy foreign invaders (such as bacteria, viruses, and cancer cells). CD40 is a key molecule found on the cell surface of immune cells called antigen presenting cells (APC), which facilitate the activation of the immune response by binding to a specific CD40-receptor found on helper T-cells. Helper T-cells subsequently drive the immune response by recruiting other cells that have specific functions in the process. The downstream processes eventually lead to the destruction of the foreign invaders. This mechanism is disrupted in pancreatic cancer by inhibitory cells that infiltrate the surrounding area of the tumor, the tumor microenvironment, not allowing immune cells to reach the tumor and destroy it. During this immune suppression process, helper T-cells are not available to bind to CD40 on the APC and prevent an immune response. Drs. Vonderheide and Bar-Sagi propose to reinstate an immune response by using an antibody against CD40 as a therapeutic. The antibody acts as a surrogate to the helper T-cells by binding to CD40 on the APC and activating an immune response. This therapeutic antibody against CD40 can be added to the standard of care for patients with pancreatic cancer to propel the immune system to attack the tumor.