GRANT SNAPSHOT

2013 Blum-Kovler Foundation – Pancreatic Cancer Action Network – AACR Innovative Grant

Grantee:	Valerie Weaver, PhD
Institution:	University of California, San Francisco
Research Project:	*Interplay between tension and inflammation in pancreatic tumor progression*
Award Period:	July 1, 2013 – June 30, 2015
Amount:	$200,000

Biographical Highlights

Dr. Weaver is currently the Director of the Center for Bioengineering and Tissue Regeneration, and Professor of Surgery, Anatomy, and Bioengineering and Therapeutic Sciences at UCSF. Her education took place in Canada, with a bachelor's, honors bachelor's, and PhD degrees from University of Waterloo, and postdoctoral training at the Institute for Biological Sciences, National Research Council of Canada. She did a second postdoctoral fellowship at the Lawrence Berkeley National Laboratory at UC Berkeley, and then joined the faculty of the University of Pennsylvania as an Assistant Professor. She moved to UCSF in 2006 and became the Director of the Center for Bioengineering & Tissue Regeneration. Dr. Weaver has over 20 years of experience in leading interdisciplinary research in oncology, including leadership of significant program projects merging approaches in the physical/engineering sciences and biology. Her research focuses on the contribution of force, cell-intrinsic as well as extracellular matrix, to oncogenesis and tumor development.

Project Overview

Dr. Weaver's project will be in collaboration with Matthias Hebrok, PhD, a two-time Pancreatic Cancer Action Network grant recipient (a Pilot Grant in 2008, funded in memory of Michael C. Sandler, and an Innovative Grant awarded in 2011, in memory of Abby Sobrato). Preliminary data generated in the Weaver laboratory indicate that pancreatic cancer progression is accompanied by deposition of collagen in the surrounding tissue, leading to a thick, stiff barrier around the tumor. The extracellular matrix also contracts and becomes stiffer due to inflammation, such as pancreatitis, which is a known risk factor for pancreatic cancer.

Dr. Weaver hypothesizes that the contractility of the matrix, due to inflammation and the tumor itself, is responsible for the dense fibrosis, progression, and aggressiveness of pancreatic cancer. Using genetically engineered mouse models, Dr. Weaver will establish whether tissue tension increases with the progression of the disease. She will also test the influence of cellular contractility on pancreatic tumor aggression and inflammation, and finally determine whether inhibiting cellular contractility increases the efficacy of chemotherapy in pancreatic cancer treatment.

This two-year project will focus on characterizing and understanding the interplay between cellular contractility and inflammation in driving pancreatic cancer progression, a perspective currently underappreciated in cancer research. This work will elucidate new mechanisms underlying pancreatic cancer progression and aggression, providing a solid foundation for downstream clinical impact including the identification of novel pathways for the development of targeted therapies, as well as for the diagnosis and early detection of the disease.