GRANT SNAPSHOT

2003 Pancreatic Cancer Action Network – AACR Career Development Award

Grantee:	C. Max Schmidt, MD, PhD
Institution:	Indiana University, Indianapolis
Project Title:	*The Chemopreventive Role of Cyclooxgenase Inhibitors in Pancreatic Tumorigenesis*
Award Period:	July 1, 2003 – June 30, 2005
Amount:	$100,000

Biographical Highlights

Dr. Schmidt is an Associate Professor of Surgery, Associate Professor of Biochemistry/Molecular Biology at Indiana University School of Medicine, Department of Surgery. He completed a residency and fellowship at Johns Hopkins Hospital. His clinical interests focus on pancreaticobillary and he has a research track record in pancreatic and hepatic neoplasia.

Project Description

Some people are at increased risk of pancreatic cancer or may have pre-cancerous cells in the pancreas that predispose them to later pancreatic cancer development. In these individuals, preventive measures with chemotherapy (chemoprevention) may block future development of pancreatic cancer. Dr. Schmidt's previous research suggested that cyclooxygenase (COX), an enzyme in the body that helps maintain normal functioning of the stomach, intestines, platelets and kidneys, may be a viable target for chemoprevention. He had shown that one COX enzyme, COX-2, is overproduced in human pancreatic cancers and pre-cancerous cells in the pancreas compared to normal tissue in the pancreas. In the funded project, Dr. Schmidt hoped to determine the chemopreventive effects of treatments that inhibit the production of COX enzymes in mouse and hamster models of pancreatic cancer. The results of the study were expected to provide guidance for the analyses of pancreatic tumor and pancreatic duct juice before and after COX enzyme inhibitor treatment in a phase II trial of patients with premalignant pancreatic lesions that was under IRB review at Indiana University. The study had the potential to provide preclinical evidence in support of a multi-institutional study of these compounds for chemoprevention in patients at high risk for the development of pancreatic cancer.

Next Steps

Plans are to evaluate the chemotherapeutic and /or chemopreventative potential of NF-KB targeted agents alone or in combination with gemcitabine in the hamster developmental model of pancreatic cancer. These studies also will be performed in a transgenic mouse model, Kras/Cre, since the Kras oncogene is frequently mutated in human pancreatic cancer. By using both of these models and comparing the effects of treatments, environmental as well as genetic influences on pancreatic cancer will hopefully be addressed.