Nutritional Links to Plausible Mechanisms Underlying Pancreatic Cancer

Agenda

8:30-8:35	**R J. Hine, NSRG** Introduction and Purpose
8:35-8:40	**P. Greenwald** Welcome
8:40-9:00	What clues do alterations in genes and gene expression patterns provide about how nutrients or bioactive food components might affect the pathogenesis of pancreatic cancer? 1 **S. Kern** - Department of Oncology, Johns Hopkins University, and Co-Chair, NCI Pancreatic Cancer Progress Review Group "Activation of the K-ras2 oncogene, and inactivation of tumor suppressor and specific DNA repair genes in pancreatic cancer"
9:00-9:10	Discussion
9:10-10:10	Is insulin resistance a unifying hypothesis that explains the relationship between hyperglycemia, diabetes, obesity and dietary macronutrients and pancreatic cancer development? 2 **V. L. Go** -Department of Medicine and Center for Human Nutrition, University of California-Los Angeles, "Metabolic hypothesis for pancreatic cancer" 1 **D. Silverman** - Occupational Epidemiology Branch, Division of Cancer Epidemiology and Genetics, "Roles of energy balance and elevated Body Mass Index in the etiology of pancreatic cancer" 2 **D. Michaud** - Nutritional Epidemiology Branch, Division of Cancer Epidemiology and Genetics, National Cancer Institute "Dietary sugars, glycemic load and pancreatic cancer risk"
10:10-10:20	Discussion
10:20-10:40	Break

10:40-11:20	What emerging data from experimental studies highlight the complexity of dietary effects on pancreatic carcinogenesis? 1 **P. Pour** - Department of Pathology and Microbiology, Eppley Cancer Center, University of Nebraska Medical Center, "Interaction among the dietary constituents and between diets and intrinsic factors" 3 **H Schuller** - Department of Pathology, College of Veterinary Medicine, University of Tennessee, "Epigenetic effects of alcohol intake and NNK associated with pancreatic carcinogenesis"
11:20-11:30	Discussion
11:30-12:30	How strong and consistent are data demonstrating that folate and other dietary methyl donors modify methylation status and cellular differentiation in pancreatic carcinogenesis? 1 **M Goggins** - Department of Pathology, Johns Hopkins Medical Institutions, "Do polymorphisms in methylenetetrahydrofolate reductase and methionine synthase protect against pancreatic cancer?" 2 **R Stolzenberg-Solomons** - Nutritional Epidemiology Branch, Division of Cancer Epidemiology and Genetics, "Dietary methyl donors, biochemical indicators of methyl group availability and pancreatic cancer risk" 3 **D S Longnecker** - Department of Pathology, Dartmouth Medical School, "Abnormal methylation patterns enhance the pathogenesis of pancreatic cancer"
12:30-1:30	Working Lunch Future Directions
1:30- 2:10	Oxidative damage to DNA is a common finding in pancreatitis that may lead to mutations that cause pancreatic cancer. Do data from experimental studies suggest that dietary antioxidants can reduce oxidative damage? Which candidate biomarkers are most sensitive to the effects of these agents? 1 **L.K. Gates Jr** - Gastroenterology Associates, Salem OR "Antioxidants suppress transcriptional factor NF-kappaB activation and inhibit IL6 and IL8 secretion" 2 **D.W. McFadden** Department of Surgery, West Virginia, University School of Medicine "Peptide YY augments inhibition of pancreatic cell growth by vitamin E succinate"
2:10-2:20	Discussion
2:20-2:50	**R. J Hine**- Setting priorities for future research about nutrition and pancreatic cancer