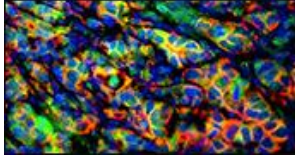


Poster Session C
Sunday, September 8
4:00 p.m.-6:30 p.m.

- C01 Porcupine inhibition redirects mitochondrial metabolism in Wnt-dependent pancreatic cancer.** Kristina Y. Aguilera, University of California Los Angeles, Los Angeles, CA
- C02 Establishment of a human PDAC explant culture model for treatment prediction and characterization of the tumor microenvironment.** Azaz Ahmed, National Center for Tumor Diseases & German Cancer Research Center, Heidelberg, Germany
- C03 Evaluation of the implementation of genetic cancer screening guidelines in African American pancreatic cancer patients in a safety net hospital.** Taiwo A. Ajose, Morehouse School of Medicine, Atlanta, GA
- C04 Treating hepatic metastasis of pancreatic cancer through targeting cancer cell metabolism.** Amer Alasadi, Rutgers University, Piscataway, NJ
- C05 Comparative quantitative proteomic profiling of neoadjuvantly treated versus treatment-naive human pancreatic ductal adenocarcinoma.** Manoj Amrutkar, Department of Pharmacology and Department of Hepato-Pancreato-Biliary Surgery, Institute of Clinical Medicine, University of Oslo, Oslo, Norway
- C06 Cholesterol deprivation induces TGF β signaling to promote basal differentiation in pancreatic cancer.** Igor Astsaturov, Fox Chase Cancer Center, Philadelphia, PA
- C07 Ciliogenesis and Hh signaling are suppressed downstream of KRas during ADM.** Fiona Bangs, University of Oxford, Oxford, United Kingdom
- C08 SM08502, a novel, small-molecule CDC-like kinase (CLK) inhibitor, demonstrates activity against cancer stem cell (CSC)-enriched pancreatic cancer cells and suppresses stemness in vitro.** Carine Bossard, Samumed, LLC, San Diego, CA
- C09 Inhibition of tumor growth and post-treatment regrowth by SM08502, a novel, small-molecule CDC-like kinase (CLK) inhibitor, in combination with standard of care in pancreatic cancer models.** Carine Bossard, Samumed, LLC, San Diego, CA
- C10 Glutamine deprivation triggers hexosamine salvage in pancreatic cancer cells.** Sydney L. Campbell, University of Pennsylvania, Philadelphia, PA
- C11 Linoleic acid elevates sirtuin 6 expression and modulates glycolysis and epithelial-mesenchymal transition of pancreatic cancer with loss of Krüppel like factor 10.** Hui-Ju Ch'ang, National Institute of Cancer Research, National Health Research Institutes, Miaoli, Taiwan
- C12 Lipids signaling contributes to glucose-independent metabolic adaptation in pancreatic ductal adenocarcinoma.** Ziheng Chen, The University of Texas MD Anderson Cancer Center, Houston, TX



Poster Session C
Sunday, September 8
4:00 p.m.-6:30 p.m.

C13 A “mouse hospital” for preclinical testing of diagnostic and treatment modalities in pancreatic ductal adenocarcinoma (PDA). Cynthia Clendenin, Abramson Cancer Center, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA

C14 A novel RAD51 inhibitor, CYT-0851, shows anticancer activity in preclinical models of pancreatic cancer. Melinda Day, Cyteir Therapeutics, Lexington, MA

C16 The glycan CA19-9 promotes pancreatitis and pancreatic cancer in mice. Dannielle Engle, Salk Institute, La Jolla, CA

C17 Preclinical models to dissect immune escape in pancreatic cancer. William A. Freed-Pastor, Koch Institute at MIT, Cambridge, MA

C18 TGF-beta blockade paradoxically activates non-SMAD signaling. Evan S. Glazer, University of Tennessee Health Science Center, Memphis, TN

C19 Impaired adaptation to negative energy balance in pancreatic cancer-associated wasting. Aaron J. Grossberg, Oregon Health & Science University, Portland, OR

C20 Smpd3 augments chemotherapy and thwarts PDA progression. Audrey M. Hendley, University of California San Francisco, San Francisco, CA

C21 KRAS confers resistance to mechanical compression in pancreatic cancer cells. Liam J. Holt, New York University, New York, NY

C22 Loss of KDM6A promotes pancreatic cancer progression by upregulating epithelial-mesenchymal transition pathway. Sivakumar Jeyarajan, Department of Pathology, University of Michigan, Ann Arbor, MI

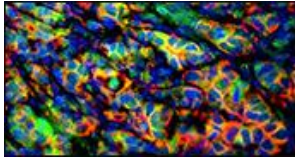
C25 Aspiration liquid biopsy (ALB) of pancreatic cancer (PC). Aleksei Kashintsev, National Bioservice, Saint-Petersburg, Russia

C26 Oncogenic mechanism of soluble keratin 17 offers potential therapeutic vulnerability in pancreatic cancer. Ryan R. Kawalerski, Stony Brook Medicine, Stony Brook, NY

C27 Association of mutant KRAS isoforms with weight loss in pancreatic cancer. Haesoo Kim, Cedars-Sinai Medical Center, Los Angeles, CA

C28 KMT2D mediates TGF- β -induced epithelial-to-mesenchymal transition to promote more aggressive pancreatic cancer. Hong S. Kim, University of Michigan, Ann Arbor, MI

C29 Development of a circulating tumor DNA assay in pancreas cancer. Dan King, Stanford University, Palo Alto, CA



Poster Session C
Sunday, September 8
4:00 p.m.-6:30 p.m.

C30 Myc amplification is a negative biomarker and a resistance mechanism to trametinib/HCQ treatment, but can be overcome by combined palbociclib/HCQ treatment. Conan Kinsey, Huntsman Cancer Institute, Salt Lake City, UT

C31 The glycosaminoglycan syndecan-4 facilitates pancreatic cancer progression and biologic aggressiveness. Murray Korc, University of California Irvine, Irvine, CA

C32 Therapeutic targeting of keratin 17 and nuclear export uncover therapeutic vulnerabilities of pancreatic cancer. Cindy V. Leiton, Stony Brook University, Stony Brook, NY

C33 Effects of curcumin on pancreatic ductal adenocarcinoma. Adrianna M. Vaskas, Mansfield University, Mansfield, PA

C34 Signaling regulation of epithelial-mesenchymal transition in pancreas cancer cells cultured under hypoxic conditions. Brooke A. McGirr, University of Virginia, Charlottesville, VA

C36 A roadmap for targeting cysteine dependency in a subset of pancreatic cancer. Zeribe C. Nwosu, University of Michigan, Ann Arbor, MI

C37 Setdb1 is required for formation of pancreatic ductal adenocarcinoma by inhibiting apoptosis through regulation of p53 expression. Satoshi Ogawa, Department of Gastroenterology and Hepatology, Kyoto University Graduate School of Medicine, Kyoto, Kyoto, Japan

C38 HNF4a regulates progression and molecular subtype of pancreatic ductal adenocarcinoma. Eric L. Snyder, Huntsman Cancer Institute, University of Utah, Salt Lake City, UT

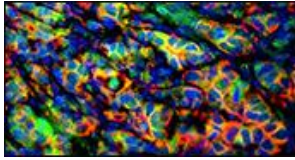
C39 A novel rewired pathway of nucleotide metabolism drives chemoresistance in pancreatic cancer. Chun-Hao Pan, Department of Pathology, Renaissance School of Medicine at Stony Brook University, Stony Brook, New York, NY

C40 Dissecting key biologic processes in pancreatic cancer metastasis using a genetically defined 3D organoid model. Fong Cheng Pan, Weill Cornell Medicine, New York, NY

C41 Establishment of a novel mouse model of pancreatic squamous carcinoma. Filipa Pinto, Boston University, Boston, MA

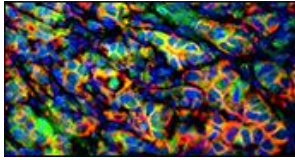
C42 Cell culture under perfusion conditions reduces cellular metabolic stress and mimics the in vivo physiologic environment in pancreatic cancer. Daniel Hughes, Department of Oncology, University of Oxford, Oxford, United Kingdom

C43 Assessment of tumor heterogeneity, clonal evolution, and the stromal microenvironment in metastatic pancreatic ductal adenocarcinoma and matched patient-derived organoids. Srivatsan Raghavan, Dana-Farber Cancer Institute, Boston, MA



Poster Session C
Sunday, September 8
4:00 p.m.-6:30 p.m.

- C44 MBD3 stabilizes MYC, leading to metastatic outgrowth of pancreatic cancer in the liver.** Alok Ranjan, National Institute of Health, Bethesda, MD
- C45 NT219, a novel bispecific inhibitor of STAT3 and IRS1/2, combined with chemotherapy or MEK inhibitor in gemcitabine-resistant pancreatic tumors, induced tumor regression.** Hadas Reuveni, TyrNovo Ltd., Tel Aviv, Israel
- C46 Establishing a living biobank of patient-derived organoids of intraductal papillary mucinous neoplasms.** Dayana Reverón, Moffitt Cancer Center, Tampa, FL
- C47 Plasma ANP and soluble cell adhesion molecule X are novel risk factors for pancreatic cancer-associated thrombosis.** Makoto Sano, The University of Tokyo, Tokyo, Japan
- C48 Defining mechanisms of adaptation to KRAS G12C inhibitors: Using quantitative proteomics to design combinatorial strategies in pancreatic cancer.** Naiara Santana-Codina, Department of Radiation Oncology, Dana-Farber Cancer Institute, Boston, MA
- C49 Knockdown of FOXN2 enhances adhesion and reduces migration in pancreatic cancer cells.** Blanca Santibanez, University of New Hampshire - Manchester, Manchester, NH
- C50 Dissecting vulnerabilities of pancreatic tumors' silent fraction unravels secrets of an alternative cell state.** Yogev Sela, University of Pennsylvania, Philadelphia, PA
- C51 Targeted inhibition of DUSP1 and DUSP6 suppresses pancreatic adenocarcinoma cells' growth and glucose metabolism via SAPK/JNK pathway activation.** Vanessa S. Silveira, University of Sao Paulo, Ribeirao Preto, Sao Paulo, Brazil
- C52 The radioprotector GC4419 enhances the response of PDAC tumors to high dose per fraction radiation exposure.** Brock J. Sishc, UT Southwestern Medical Center, Dallas, TX
- C53 Liver X receptor ligand targets glutamine metabolism in pancreatic cancer.** Shivangi Srivastava, University of Houston, Houston, TX
- C54 GATA6 level is a prognostic biomarker for pancreatic cancer in African American patients.** Haoyu Tang, Yale University, New Haven, CT
- C55 Mapping the pharmacotranscriptomic landscape of pancreatic circulating tumor cell organoids.** Fredrik I. Thege, The University of Texas MD Anderson Cancer Center, Houston, TX
- C56 Bmi1 is widely expressed in acini and regulates Kras-driven transcription factor networks in early pancreatic neoplasia.** Joyce K. Thompson, University of Michigan, Ann Arbor, MI
- C57 Organoid profiling identifies common responders to chemotherapy in pancreatic cancer.** Herve Tiriac, University of California San Diego, La Jolla, CA



Poster Session C
Sunday, September 8
4:00 p.m.-6:30 p.m.

C58 p53/p16-independent cyto-reduction of chemoresistant pancreatic adenocarcinoma by metabolically optimized epigenetic therapy. Rita Tohme, Cleveland Clinic, Cleveland, OH

C59 NRF2 drives metabolic reprogramming in irradiated pancreatic cancer cells and promotes radioresistance. Erina Vlashi, University of California Los Angeles, Los Angeles, CA

C60 Combination of MEK and autophagy inhibition promotes tumor regression in the KPC mouse model of pancreatic cancer. Urszula N. Wasko, Columbia University, New York, NY

C61 Dysregulation of HNF1B/Clusterin axis enhances disease progression in a highly aggressive subset of pancreatic cancer. Shouhui Yang, Laboratory of Human Carcinogenesis, CCR, NCI, Bethesda, MD

C62 The Alliance of Pancreatic Cancer Consortia for Biomarkers for Early Detection. Matthew R. Young, National Cancer Institute, Rockville, MD

C63 The analysis of microbiota composition between tumor and normal tissues in pancreatic cancer and the comparison with the public data of NCBI. Eunsung Jun, Asan Medical Center, Seoul, South Korea

C64 C4BPA identified as a novel biomarker is associated with favorable outcome of patients with pancreatic cancer. Kosuke Sasaki, Chiba University, Chiba, Japan

C65 Identifying mechanisms of macrophage-mediated metastasis and therapy resistance in PDAC. Tony J. Wu, Cancer Research UK Cambridge Institute, University of Cambridge, Cambridge, United Kingdom, ²Memorial Sloan Kettering Cancer Center, New York, NY