# PANCREATIC CANCER NEWS & UPDATES – OCTOBER 2013

## PANCREATIC CANCER ACTION NETWORK AND COMMUNITY NEWS

***Pancreatic Cancer Action Network news:***

**Progress Matters: Advancing Research to Fight Pancreatic Cancer – An Informative Webinar Report**
http://www.youtube.com/watch?v=-S8waj6oglQ&feature=youtu.be
With the Pancreatic Cancer Action Network's Julie Fleshman, JD, MBA, and Lynn Matrisian, PhD, MBA; and Anirban Maitra, MBBS, University of Texas MD Anderson Cancer Center. Learn more about the progress we're making in the fight against pancreatic cancer.

**October Evening with the Stars weekend brings more strength, unity to "Community for Progress"**
http://www.pancan.org/section_about/pancreas_matters_articles/november-2013/researchers-ewts-gala.php#.UoKer6PTmAg
In addition to providing financial support for vital research, the Pancreatic Cancer Action Network is building a Community for Progress. Within this community, grant recipients receive professional advancement opportunities, including mentorship, networking, and—new this year—communication training. All of these important elements came into play in the whirlwind "An Evening with the Stars" weekend, October 18 – 19, in Beverly Hills, California.

**Pancreatic Cancer Action Network's 16th annual 'An Evening with the Stars' gala raises > $750,000**
http://pancan.org/section_about/news_press_center/2013_press_releases/10_23_13_pr.php#.UmhV56Pn-Ag
More than 400 guests attended the Pancreatic Cancer Action Network's 16th Annual 'An Evening with the Stars' gala on Saturday, October 19 at the iconic Beverly Wilshire Hotel in Beverly Hills. The fundraising event raised more than $750,000 to support the Pancreatic Cancer Action Network's effort to double the survival rate of pancreatic cancer by 2020. Celgene Corporation, Tempur-Pedic North America, LLC, and Rancho Palos Verdes Mayor (ret.) and pancreatic cancer survivor Larry Clark were honored during an evening that was dedicated to advancing research, supporting patients and creating hope for everyone affected by pancreatic cancer.

**November marks Pancreatic Cancer Awareness Month**
http://pancan.org/section_about/news_press_center/2013_press_releases/11_05_13_pr.php#.UoKhT6PTmAg
The Pancreatic Cancer Action Network is calling attention to pancreatic cancer during Pancreatic Cancer Awareness Month in November. The organization is urging Americans to join the movement to defeat pancreatic cancer and "Like the Fight" (http://www.pancan.org/fight).

***Funding opportunities:***

*New!* **Contract funding opportunities available for innovative SBIR development**

http://sbir.cancer.gov/funding/contracts/

*Deadline for receipt of all FY2014 contract topic proposals has been extended to Monday, November 25, 2013 by 4:30 p.m. ET.*

The National Cancer Institute's Small Business Innovation Research (NCI SBIR) Program announced $5 million for 8 new contract funding opportunities in a range of novel technology areas.

**2014 NIH Director's Early Independence Awards**

http://commonfund.nih.gov/earlyindependence/index.aspx

*Letters of Intent deadline: December 31, 2013*

*Application deadline: January 31, 2014*

The National Institutes of Health Common Fund announces the FY 2014 funding opportunity for the NIH Director's Early Independence Awards (EIA). The EIA initiative allows exceptional junior scientists to accelerate their transition to an independent research career by "skipping" the traditional postdoctoral training. To be eligible, candidates must be within one year (before or after) of completion of their terminal degree or clinical residency at the time of application. Each institution (as defined by a unique DUNS identifier) may submit up to two applications in response to this FOA.

**Clinical Studies of Safety and Effectiveness of Orphan Products Research Project Grant (R01)**

https://www.federalregister.gov/articles/2012/08/06/2012-19086/clinical-studies-of-safety-and-effectiveness-of-orphan-products-research-project-grant-r01#h-4

*Application deadline: February 5, 2014*

The Food and Drug Administration (FDA) is announcing the availability of grant funds for the support of FDA's Office of Orphan Products Development (OPD) grant program. The goal of FDA's OPD grant program is to support the clinical development of products for use in rare diseases or conditions (defined as a disease or condition that has a prevalence, not incidence, of fewer than 200,000 people in the US) where no current therapy exists or where the proposed product will be superior to the existing therapy.

**Scientific information request: imaging tests for diagnosis, staging of pancreatic adenocarcinoma**

http://www.gpo.gov/fdsys/pkg/FR-2013-08-27/pdf/2013-20849.pdf

*AGENCY: Agency for Healthcare Research and Quality (AHRQ), HHS*

*ACTION: Request for scientific information submissions*

The Agency for Healthcare Research and Quality (AHRQ) is seeking scientific information submissions from the public on imaging tests for the diagnosis and staging of pancreatic adenocarcinoma. Scientific information is being solicited to inform our review of Imaging Tests for the Diagnosis and Staging of Pancreatic Adenocarcinoma, which is currently being conducted by the Evidence-based Practice Centers for the AHRQ Effective Health Care Program.

**Share your federal funding experiences: Help our advocacy efforts**

http://www.pancan.org/section_research/resources_for_scientists/form_funding_experiences.php

Have you struggled to receive grants from the NCI or other federal institutions? Have you been successful? We're looking for information to help us understand what is working well for pancreatic cancer researchers and what could be improved (including, but not limited to, funding levels). We will use this information in our public policy efforts. Please click above and share your stories (they can be submitted anonymously).

**Support of the Cancer Genome Atlas Program (TCGA): Funding opportunity**
http://www.fdbdo.com/s12-335/
Financial support is available for submissions to the TCGA pancreas project. Please see the note from Kenna Shaw, PhD of the TCGA Program Office:
"Because of the poor prognosis and overall public health impact, pancreatic ductal adenocarcinoma has been selected to be studied by TCGA. However, the pancreatic cancer project might end before it even begins unless more collaborators provide tissue samples. The collapse of this research project would be a lost opportunity for the pancreatic cancer community ... If you have frozen, untreated samples in your tissue bank accompanied by blood, please help by providing them, either retrospectively or prospectively, to TCGA."

***Meetings:***
**Save the date: AACR Pancreatic Cancer special conference**
http://www.aacr.org/home/scientists/meetings--workshops/meetings--workshops-calendar.aspx
*May 18-21, 2014, New Orleans, LA*

**AACR Annual Meeting 2014: Abstracts**
http://www.aacr.org/home/scientists/meetings--workshops/aacr-annual-meeting-2014/abstracts.aspx
*Meeting: April 5-9, 2014, San Diego, CA*
*Abstract submission deadline: Tuesday, December 3, 2013*
*Early registration deadline: Friday, December 20, 2013*
*Late-breaking abstract deadline: Monday, January 27, 2014*
The 105th Annual Meeting of the American Association for Cancer Research will be held April 5-9, 2014, in San Diego, California. As always, this AACR Annual Meeting will highlight the latest and most exciting discoveries in every area of cancer research, and it will provide a unique opportunity for investigators from all over the world to meet, network, and forge new scientific interactions.

**AACR Annual Meeting 2014: Clinical research and clinical trials**
http://www.aacr.org/home/scientists/meetings--workshops/aacr-annual-meeting-2014/spotlight-on-clinical-research-and-clinical-trials.aspx
*Abbreviated abstracts deadline: January 27, 2014*
*Complete abstracts deadline: February 28, 2014*
In 2014, there will be numerous opportunities to showcase cutting-edge clinical research including the Clinical Symposia, which offer a unique presentation format. Any phase I, II, or III clinical trials abstracts submitted for either the regular or the late-breaking deadline to the Clinical Trials (CT) category will be considered by the Program Committee for oral presentation in one of these clinical trials-focused sessions.

**2014 Gastrointestinal Cancers Symposium**
http://gicasym.org/
*January 16-18, 2014, San Francisco, CA*
*Register and reserve hotel by December 11, 2013, at 11:59 PM (EST) for the best rates*
The 2014 Gastrointestinal Cancers Symposium is a specialized meeting designed to highlight the latest science in cancers of the pancreas, small bowel, and hepatobiliary tract; colon and rectum; and esophagus and stomach. Now in its 11th year, the Symposium continues to offer a fresh perspective on gastrointestinal cancers, with a special focus on the most pertinent information oncologists of all subspecialties need to know now, in order to provide the highest quality of care.

**Members only: 2014 ASCO Annual Meeting registration and hotel reservations now open**
http://am.asco.org/registration-and-hotel-information?cmpid=nm_am_rh_em_t_mem_11-01-13_amhtml
*Meeting: May 30-June 3, 2014, Chicago, IL*
*Hotel reservation and early registration rate deadline: Wednesday, April 23, 2014 at 11:59 PM (EDT)*
*General registration and hotel reservations open in early December*

***Other community news:***
**Special Issue | AGA Pancreatic Disorders**
http://pancreas.agajournals.org/Home/SpecialIssue
The American Gastroenterological Association's *Gastroenterology* journal special edition focused on pancreatic disorders is now available in full online. Many Pancreatic Cancer Action Network grant recipients and members of our Scientific and Medical Advisory Boards are featured in this special issue.

**2013 American Pancreatic Association Presidential Address: Widening the Reach of the APA**
http://journals.lww.com/pancreasjournal/Citation/2013/11000/2013_American_Pancreatic_Association_Presidential.1.aspx
**Abstracts of papers submitted to the 44th Meeting of the American Pancreatic Association**
http://journals.lww.com/pancreasjournal/Fulltext/2013/11000/Abstracts_of_Papers_Submitted_to_the_44th_Meeting.21.aspx?WT.mc_id=HPxADx20100319xMP
This year's APA Meeting took place October 30-November 2, 2013 in Miami, FL.

**Webcasts of the 2013 AACR-NCI-EORTC Conference on Molecular Targets and Cancer Therapeutics**
http://webcast.aacr.org/
This year's meeting took place October 19-23, 2013 in Boston, MA. See some of pancreatic cancer-related studies presented at this meeting in the Treatment section below.

**U.S. Cancer Statistics 1999-2010 released**
http://content.govdelivery.com/accounts/USCDC/bulletins/91396d
The *U.S. Cancer Statistics: 1999–2010 Incidence and Mortality Web-based Report* (USCS) contains the official federal statistics on cancer incidence (newly diagnosed cases) from each registry that met data quality criteria. The Centers for Disease Control and Prevention (CDC) and the National Cancer Institute have combined their cancer incidence data sources to produce these statistics. Mortality data from the National Vital Statistics System of CDC's National Center for Health Statistics are included for each state.

**Managing the Nation's Cancer Research Portfolio**
http://www.cancer.gov/aboutnci/budget_planning_leg/plan-2013
*Report: http://www.cancer.gov/PublishedContent/Files/aboutnci/budget_planning_leg/plan-archives/nci_plan_2013.pdf*
One of the goals of this annual report is to summarize some of the cancer research findings and their practical consequences, so that the NCI's many supporters and beneficiaries can better appreciate the significance of the NCI and the argument for the enhanced support that we request in the "by-pass budget."

**ASCO's 2013 top five list in oncology**
http://www.asco.org/practice-research/ascos-2013-top-five-list-oncology
ASCO recognizes the importance of evidence-based cancer care and making wise choices in the diagnosis and management of patients with cancer. After careful consideration by experienced oncologists, ASCO annually highlights five categories of tests, procedures and/or treatments annually whose common use and clinical value are not supported by available evidence as part of the American Board of Internal Medicine's (ABIM) Choosing Wisely® Campaign.

**Liquid Grids releases multiple oncology grids**
http://liquidgrids.com/press-releases/liquid-grids-releases-multiple-oncology-grids/
Liquid Grids®, Healthcare's only All-In-One Inbound marketing platform announces the release of multiple oncology disease grids. Using the company's Direct to Persona® marketing platform, the healthcare industry will be able to market directly to these online communities. The new disease grids include Breast Cancer, Prostate Cancer, Skin Cancer, Solid Tumors, Pancreatic Cancer and Esophageal Cancer.

**BIOLOGY OF CANCER**
**DCLK1 marks a morphologically distinct subpopulation of cells with stem cell properties**
http://www.ncbi.nlm.nih.gov/pubmed/24096005

- Journal: *Gastroenterology*
- Institution(s): Johns Hopkins University School of Medicine, Baltimore, MD and others
- Corresponding author(s): Steven Leach
- Pancreatic Cancer Action Network-affiliated authors:
    - Jennifer Bailey, PhD: 2011 Pathway to Leadership Grant
    - Zeshaan Rasheed, MD, PhD: 2010 Tempur-Pedic® Retailers – Pathway to Leadership Grant
    - Florencia McAllister, MD: 2012 Samuel Stroum – Fellowship
    - Nabeel Bardeesy, PhD: 2008 Randy Pausch, PhD – Pilot Grant
    - Anirban Maitra, MBBS: 2004 Career Development Award and chair, Scientific Advisory Board
    - Steven Leach, MD: member, Scientific Advisory Board
- Major finding: Human pancreatic ductal adenocarcinoma cells and pancreatic neoplasms in mice contain morphologically and functionally distinct subpopulations that have cancer stem cell-like properties. These populations can be identified at the earliest stages of pancreatic tumorigenesis, and provide new cellular and molecular targets for pancreatic cancer treatment and/or chemoprevention.

**IL-6 required for progression by promoting MAPK signaling activation and oxidative stress resistance**
http://www.ncbi.nlm.nih.gov/pubmed/24097820

- Journal: *Cancer Research*
- Institution(s): University of Michigan, Ann Arbor, MI and others
- Corresponding author(s): Marina Pasca di Magliano
- Pancreatic Cancer Action Network-affiliated authors:
    - Ben Stanger, MD, PhD: 2007 Ralph H. Hruban, MD – Career Development Award
    - Andrew Rhim, MD: 2013 Career Development Award
    - Marina Pasca di Magliano, PhD: 2009 Paul Mitchell – Career Development Award

- Major finding: Mechanistically, the authors show that interleukin-6 (IL-6) synergizes with oncogenic Kras to activate the reactive oxygen species detoxification program downstream of the mitogen-activated protein kinase/extracellular signal-regulated kinase (MAPK/ERK) signaling cascade. In addition, IL-6 regulates the inflammatory microenvironment of pancreatic cancer throughout its progression, providing several signals that are essential for carcinogenesis. Thus, IL-6 emerges as a key player at all stages of pancreatic carcinogenesis and a potential therapeutic target.

**MUC1 regulates expression of multiple microRNAs, including the miR-200c/141 cluster**
http://www.ncbi.nlm.nih.gov/pubmed/24143167

- Journal: *PLoS One*
- Institution(s): University of Nebraska Medical Center, Omaha, NE and others
- Corresponding author(s): Michael (Tony) Hollingsworth
- Pancreatic Cancer Action Network-affiliated authors:
    - Jennifer Bailey, PhD: 2011 Pathway to Leadership Grant
    - Tony Hollingsworth, PhD: member, Scientific Advisory Board
- Major finding: These data indicate that signaling through MUC1 influences cancer progression by regulating transcription of microRNAs that are associated with the process of metastasis.

**MicroRNA-200c modulates expression of MUC4, MUC16 by directly targeting their coding sequences**
http://www.ncbi.nlm.nih.gov/pubmed/24204560

- Journal: *PLoS One*
- Institution(s): University of Nebraska Medical Center, Omaha, NE
- Corresponding author(s): Michael (Tony) Hollingsworth
- Pancreatic Cancer Action Network-affiliated author: Tony Hollingsworth, PhD: member, Scientific Advisory Board
- Major finding: The authors discovered that miR-200c interacts with specific sequences within the coding sequence of the transmembrane mucins MUC4 and MUC16 mRNAs, and evaluated the regulatory nature of this association. Their data suggest that, in addition to regulating proteins that modulate epithelial-mesenchymal transition (EMT), miR-200c influences expression of cell surface mucins in pancreatic cancer.

**Isoprenylcysteine carboxylmethyltransferase deficiency exacerbates KRAS-driven pancreatic neoplasia**
http://www.ncbi.nlm.nih.gov/pubmed/24216479

- Journal: *Journal of Clinical Investigation*
- Institution(s): NYU School of Medicine, New York, NY and others
- Corresponding author(s): Mark Philips
- Pancreatic Cancer Action Network-affiliated author: Dafna Bar-Sagi, PhD: co-PI, 2013 Tempur-Pedic – Inaugural Research Acceleration Network (RAN) Grant in memory of Tim Miller, 2008 Pilot Grant, and member, Scientific Advisory Board
- Major finding: Isoprenylcysteine carboxylmethyltransferase (ICMT) methylates RAS and other CaaX-containing proteins, but its potential as a target for cancer therapy has not been fully evaluated. The authors' data suggest that ICMT behaves like a tumor suppressor in pancreatic ductal adenocarcinoma because it is required for Notch1 signaling.

**Triptolide-induced cell death is mediated by O-GlcNAc modification of transcription factor Sp1**
http://www.ncbi.nlm.nih.gov/pubmed/24129563

- Journal: *Journal of Biological Chemistry*
- Institution(s): University of Minnesota, Minneapolis, MN
- Corresponding author(s): Ashok Saluja
- Pancreatic Cancer Action Network-affiliated author: Selwyn Vickers, MD: member, Emeritus Scientific Advisory Board
- Major finding: In the current study the authors have evaluated the mechanism by which triptolide affects glycosylation of Sp1, which in turn affects downstream pathways controlling survival in pancreatic cancer cells.

**Re-directing apoptosis to aponecrosis induces selective cytotoxicity to pancreatic cancer cells**
http://www.ncbi.nlm.nih.gov/pubmed/24126434

- Journal: *Molecular Cancer Therapeutics*
- Institution(s): Columbia University, New York, NY and others
- Corresponding author(s): Robert Fine
- Pancreatic Cancer Action Network-affiliated author: Gloria Su, PhD: 2010 Innovative Grant and 2007 Pilot Grant
- Major finding: The authors concluded that early caspase-independent apoptosis was shifted to Voltage-Dependent Anion Channel (VDAC)-mediated "targeted" aponecrosis by the addition of disulfiram to arsenic trioxide and ascorbic acid. Conceptually, this work represents a paradigm shift where switching from apoptosis to aponecrosis death pathways, a.k.a. targeted aponecrosis, could be utilized to selectively kill pancreatic cancer cells resistant to apoptosis.

**HAb18G/CD147 promotes pSTAT3-mediated pancreatic cancer development via CD44s**
http://www.ncbi.nlm.nih.gov/pubmed/24132924

- Journal: *Clinical Cancer Research*
- Institution(s): Fourth Military Medical University, Shaanxi, People's Republic of China and others
- Corresponding author(s): Liang Xu
- Pancreatic Cancer Action Network-affiliated author: Diane Simeone, MD: 2010 The Randy Pausch Family – Innovative Grant and member, Scientific Advisory Board
- Major finding: The authors identified HAb18G/CD147 as a novel upstream activator of STAT3 via interacts with CD44s and plays a critical role in the development of pancreatic cancer. The data suggest HAb18G/CD147 could be a promising therapeutic target for highly aggressive pancreatic cancer and a surrogate marker in the STAT3-targeted molecular therapies.

**Down-regulation of miR-221 inhibits proliferation via up-regulation of PTEN, p27kip1, p57kip2, PUMA**
http://www.ncbi.nlm.nih.gov/pubmed/24224124

- Journal: *Clinical Cancer Research*
- Institution(s): Fourth Military Medical University, Shaanxi, People's Republic of China and others
- Corresponding author(s): Liang Xu
- Pancreatic Cancer Action Network-affiliated author: Philip Philip, MD, PhD: member, Medical Advisory Board
- Major finding: These results provide experimental evidence in support of the oncogenic role of miR-221 and also demonstrate the role of isoflavone, BR-DIM, and CDF as potential non-toxic agents that are capable of down-regulation of miR-221. Therefore, these agents combined with

conventional chemotherapeutics could be useful in designing novel targeted therapeutic strategy for the treatment of pancreatic cancer for which there is no curative therapy.

**Glycolytic ATP fuels the plasma membrane calcium pump critical for pancreatic cancer cell survival**
http://www.ncbi.nlm.nih.gov/pubmed/24158437

- Journal: *Journal of Biological Chemistry*
- Institution(s): The University of Manchester, Manchester, UK
- Corresponding author(s): Jason Bruce
- Major finding: The present study aimed to determine the relative contribution of mitochondrial vs glycolytic ATP in fueling the plasma membrane Ca2+ ATPase (PMCA) in human pancreatic cancer cells. Targeting the glycolytic regulation of the PMCA may be an effective strategy for selectively killing pancreatic cancer, whilst sparing healthy cells.

**microRNA-10b enhances invasion by suppressing TIP30 expression, promoting EGF and TGF-β actions**
http://www.ncbi.nlm.nih.gov/pubmed/24096486

- Journal: *Oncogene*
- Institution(s): Indiana University School of Medicine, Indianapolis, IN
- Corresponding author(s): Murray Korc
- Major finding: Therapeutic targeting of miR-10b in pancreatic ductal adenocarcinoma may interrupt growth-promoting deleterious EGF–TGF-β interactions and antagonize the metastatic process at various levels.

**MicroRNAs co-operatively inhibit network of tumor suppressor genes to promote growth, progression**
http://www.ncbi.nlm.nih.gov/pubmed/24120476

- Journal: *Gastroenterology*
- Institution(s): Imperial College, Hammersmith Hospital campus, London, UK and others
- Corresponding author(s): Leandro Castellano and Long Jiao
- Major finding: In an integrated data analysis, the authors identified functional miRNA-mRNA interactions that contribute to growth of pancreatic ductal adenocarcinomas. These findings indicate that miRNAs act together to promote tumor progression; therapeutic strategies might require inhibition of several miRNAs.

**Nuclear death receptor TRAILR2 inhibits maturation of let-7 and promotes proliferation**
http://www.ncbi.nlm.nih.gov/pubmed/24120475

- Journal: *Gastroenterology*
- Institution(s): University of Kiel, Kiel, Germany and others
- Corresponding author(s): Anna Trauzold
- Major finding: Nuclear TRAILR2 inhibits maturation of the microRNA let-7 in pancreatic cancer cell lines and increases their proliferation. Pancreatic tumor samples have increased levels of nuclear TRAILR2, which correlate with poor outcome of patients. These findings indicate that in the nucleus, death receptors can function as tumor promoters and might be therapeutic targets.

**Upregulation of Wnt5a promotes epithelial-to-mesenchymal transition and metastasis**
http://www.ncbi.nlm.nih.gov/pubmed/24156409

- Journal: *BMC Cancer*
- Institution(s): Second Military Medical University, Shanghai, China and others

- Corresponding author(s): Minghua Zhu
- Major finding: Upregulation of Wnt5a promotes epithelial-to-mesenchymal transition (EMT) and metastasis in pancreatic cancer models, which involves activation of β-catenin-dependent canonical Wnt signaling. These findings warrant further investigation of the clinical relevance of Wnt5 upregulation in pancreatic cancer.

**GLI1 interferes with DNA mismatch repair system through BHLHE41-mediated suppression of MLH1**
http://www.ncbi.nlm.nih.gov/pubmed/24165159

- Journal: *Cancer Research*
- Institution(s): Aichi Medical University School of Medicine, Aichi, Japan
- Corresponding author(s): Kenji Kasai
- Major finding: Based on their results, the authors propose that GLI1 depresses the mismatch repair activity and might contribute to the development and progression of pancreatic ductal adenocarcinoma.

**Synergistic interaction of novel LDH inhibitors, gemcitabine against pancreatic cancer cells in hypoxia**
http://www.ncbi.nlm.nih.gov/pubmed/24178759

- Journal: *British Journal of Cancer*
- Institution(s): VU University Medical Center, Amsterdam, the Netherlands and others
- Corresponding author(s): Elisa Giovannetti
- Major finding: All these molecular mechanisms underlying the synergism of 3-deazaneplanocin A (DZNeP)/gemcitabine combination support further studies on this novel therapeutic approach for treatment of pancreatic ductal adenocarcinomas.

**Molecular characterization of patient-derived human pancreatic tumor xenograft models**
http://www.ncbi.nlm.nih.gov/pubmed/24204193

- Journal: *Neoplasia*
- Institution(s): Agensys, Inc, Santa Monica, CA
- Corresponding author(s): Pia Challita-Eid
- Major finding: While some differences exist between the primary tumors and corresponding xenografts, the molecular profiles remain stable after extensive passaging. Evidence for stability in molecular characteristics after several rounds of passaging lends confidence to clinical relevance and allows for expansion of models to generate the requisite number of animals required for cohorts used in drug screening and development studies.

**The pancreatic expression database: recent extensions and updates**
http://www.ncbi.nlm.nih.gov/pubmed/24163255

- Journal: *Nucleic Acids Research*
- Institution(s): Barts Cancer Institute, Queen Mary University of London, London, UK and others
- Corresponding author(s): Claude Chelala
- Major finding: The Pancreatic Expression Database (PED, http://www.pancreasexpression.org) is the only device currently available for mining of pancreatic cancer literature data. Here the authors provide an update to PED, which has been previously featured in the Database issue of this journal.

**A novel regulatory mechanism of Pim-3 kinase stability, involvement in pancreatic cancer progression**
http://www.ncbi.nlm.nih.gov/pubmed/24165482

- Journal: *Molecular Cancer Research*
- Institution(s): Fudan University Shanghai Cancer Center, Shanghai, China and others
- Corresponding author(s): Ying-Yi Li
- Major finding: Translationally-controlled tumor protein (TCTP/TPT1) was identified from a yeast two-hybrid screen and shown to interact with Pim-3, a member of the proto-oncogene Pim family with serine/threonine kinase activity. The present study provides a new idea and experimental evidence for recognizing TCTP/Pim-3 pathway as a target for therapy in human pancreatic cancer.

**Knockdown of RON receptor kinase delays, doesn't prevent tumor progression, enhancing HGF/MET**
http://www.ncbi.nlm.nih.gov/pubmed/24100611

- Journal: *Oncogenesis*
- Institution(s): University of Texas Health Science Center at San Antonio, San Antonio, TX
- Corresponding author(s): James Freeman
- Major finding: This dynamic interaction of RON (receptor originated from nantes) and MET in pancreatic cancer cells suggests that dual targeting of both RON and MET will be preferable to inhibition of either target alone.

**Etoposide induces apoptosis via the mitochondrial- and caspase-dependent pathways**
http://www.ncbi.nlm.nih.gov/pubmed/24100999

- Journal: *Oncology Reports*
- Institution(s): Shanghai Jiaotong University, Shanghai, P.R. China
- Corresponding author(s): Qian Huang
- Major finding: In the present study, the authors performed several methods, including CCK-8 assay, immunofluorescence technique, western blotting and flow cytometry, to determine the effects of VP16 (etoposide) on Panc-1 pancreatic cancer cells. These data provide a new strategy for the therapy of pancreatic cancer.

**Down-regulation of PLCγ2-β-catenin pathway promotes activation and expansion of MDSCs in cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24127488

- Journal: *Journal of Experimental Medicine*
- Institution(s): Washington University School of Medicine, St. Louis, MO and others
- Corresponding author(s): Roberta Faccio
- Major finding: For the first time, the authors demonstrate that down-regulation of PLCγ2–β-catenin pathway occurs in mice and humans and leads to myeloid-derived suppressor cells (MDSC)-mediated tumor expansion, raising concerns about the efficacy of systemic β-catenin blockade as anti-cancer therapy.

**Loss of HAI-1 participates in metastatic spreading in a mouse orthotopic transplantation model**
http://www.ncbi.nlm.nih.gov/pubmed/24147538

- Journal: *Cancer Science*
- Institution(s): University of Miyazaki, Miyazaki, Japan and others
- Corresponding author(s): Hiroaki Kataoka

- Major finding: These data indicate that hepatocyte growth factor activator inhibitor type 1 (HAI-1) loss contributes to invasion and dissemination of a highly metastatic subline of the SUIT-2 human pancreatic adenocarcinoma cell line, suggesting crucial roles for the balance of pericellular serine proteases/inhibitors in pancreatic cancer progression.

**Is cancer a metabolic disease?**
http://www.ncbi.nlm.nih.gov/pubmed/24139946

- Journal: *The American Journal of Pathology*
- Institution(s): Princeton University, Princeton, NJ
- Corresponding author(s): Hilary Coller
- Major finding: This article reviews recent research in the field of cancer metabolism, raising the following questions: Why do cancer cells shift the metabolism in this way? Are the changes in metabolism in cancer cells a consequence of the changes in proliferation or a driver of cancer progression? Can cancer metabolism be targeted to benefit patients?

**ETIOLOGY**

**Pancreatitis-Diabetes-Pancreatic Cancer: Summary of an NIDDK-NCI workshop**
http://www.ncbi.nlm.nih.gov/pubmed/24152948

- Journal: *Pancreas*
- Institution(s): National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, MD and others
- Corresponding author(s): Dana Andersen
- Pancreatic Cancer Action Network-affiliated authors:
    - Michael Goggins, MD: PI, 2013 Skip Viragh – Inaugural Research Acceleration Network (RAN) Grant
    - Gloria Petersen, PhD: member, Scientific Advisory Board
- Major finding: A workshop sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) and the National Cancer Institute (NCI) on "Pancreatitis-Diabetes-Pancreatic Cancer" focused on the risk factors of chronic pancreatitis (CP) and diabetes mellitus (DM) on the development of pancreatic ductal adenocarcinoma (PDAC). A broad spectrum of expertise of the speakers and the discussants provided an unusually productive workshop, the highlights of which are summarized in the accompanying article.

**Obesity increases malignant risk in patients w/ branch-duct intraductal papillary mucinous neoplasm**
http://www.ncbi.nlm.nih.gov/pubmed/24074418

- Journal: *Surgery*
- Institution(s): Indiana University Hospital, Indianapolis, IN
- Corresponding author(s): C. Max Schmidt
- Pancreatic Cancer Action Network-affiliated author: C. Max Schmidt, MD, PhD: 2003 Career Development Award
- Major finding: The authors' findings suggest that obesity is associated with an increased frequency of malignancy in branch-duct intraductal papillary mucinous neoplasm (IPMN). Obesity is a potentially modifiable risk factor that may influence oncologic risk stratification, patient counseling, and surveillance strategy.

**Polymorphisms in metabolism/antioxidant genes may mediate the effect of dietary intake on risk**
http://www.ncbi.nlm.nih.gov/pubmed/24051964

- Journal: *Pancreas*
- Institution(s): Mayo Clinic, Rochester, MN and others
- Corresponding author(s): Gloria Petersen
- Pancreatic Cancer Action Network-affiliated author: Gloria Petersen, PhD: member, Scientific Advisory Board
- Major finding: Interindividual variation in metabolism/antioxidant genes could interact with dietary intake to influence pancreatic cancer risk.

**Genes-environment interactions in obesity- and diabetes-associated pancreatic cancer: GWAS data**
http://www.ncbi.nlm.nih.gov/pubmed/24136929

- Journal: *Cancer Epidemiology, Biomarkers & Prevention*
- Institution(s): MD Anderson Cancer Center, Houston, TX and others
- Corresponding author(s): Donghui Li
- Pancreatic Cancer Action Network-affiliated author: Gloria Petersen, PhD: member, Scientific Advisory Board
- Major finding: Genetic variations in inflammatory response and insulin resistance may affect the risk of obesity and diabetes-related pancreatic cancer. These observations should be replicated in additional large datasets. Gene-environment interaction analysis may provide new insights into the genetic susceptibility and molecular mechanisms of obesity- and diabetes-related pancreatic cancer.

**Fatty acids found in dairy, protein, unsaturated fatty acids associated with risk in case-control study**
http://onlinelibrary.wiley.com/doi/10.1002/ijc.28525/abstract;jsessionid=45520633728F9F7D0416D48E7B731F94.f01t04

- Journal: *International Journal of Cancer*
- Institution(s): Mayo Clinic, Rochester, MN and others
- Corresponding author(s): Rick Jansen
- Pancreatic Cancer Action Network-affiliated author: Gloria Petersen, PhD: member, Scientific Advisory Board
- Major finding: The authors' study suggests that eating a diet high in total protein and certain unsaturated fatty acids is associated with decreased risk of developing pancreatic cancer in a dose-dependent manner, whereas fats found in dairy increase risk.

**Nut consumption and risk of pancreatic cancer in women**
http://www.ncbi.nlm.nih.gov/pubmed/24149179

- Journal: *British Journal of Cancer*
- Institution(s): Brigham and Women's Hospital and Harvard Medical School, Boston, MA
- Corresponding author(s): Charles Fuchs
- Major finding: This study has picked up considerable media attention. Frequent nut consumption is inversely associated with risk of pancreatic cancer in this large prospective cohort of women, independent of other potential risk factors for pancreatic cancer.

**Diabetes type II, other medical conditions and pancreatic cancer risk: A prospective study**
http://www.ncbi.nlm.nih.gov/pubmed/24149173

- Journal: *British Journal of Cancer*
- Institution(s): Maastricht University Medical Centre, Maastricht, the Netherlands
- Corresponding author(s): Piet van den Brandt
- Major finding: In this prospective study, a positive association was observed between self-reported physician diagnosed diabetes mellitus type II and hepatitis and pancreatic cancer risk, whereas an inverse association was observed with hypertension.

**Risk of pancreatic cancer among individuals with hepatitis C or hepatitis B virus infection**
http://www.ncbi.nlm.nih.gov/pubmed/24178755

- Journal: *British Journal of Cancer*
- Institution(s): Karolinska Institutet, Stockholm, Sweden
- Corresponding author(s): Ann-Sofi Duberg
- Major finding: The authors' results indicated that hepatitis C virus (HCV) infection might be associated with an increased risk of pancreatic cancer but further studies are needed to verify such association. The results in the HBV cohort indicated an excess risk, however, without statistical significance due to lack of power.

**Pancreatitis before pancreatic cancer: Clinical features and influence on outcome**
http://www.ncbi.nlm.nih.gov/pubmed/24153158

- Journal: *Journal of Clinical Gastroenterology*
- Institution(s): Mayo Clinic, Scottsdale, AZ
- Corresponding author(s): Douglas Faigel
- Major finding: Patients with pancreatic adenocarcinoma (PA) and pancreatitis had more weight loss and diabetes mellitus, but had PA diagnosis at an earlier stage, were more likely to have pancreatic surgery, and therefore better survival than PA patients without pancreatitis, likely due to the earlier diagnosis. Further studies are needed to evaluate whether screening for PA in patients with pancreatitis history would provide survival benefit.

**Red meat and cancer risk in a network of case-control studies focusing on cooking practices**
http://www.ncbi.nlm.nih.gov/pubmed/24121119

- Journal: *Annals of Oncology*
- Institution(s): Centro di Riferimento Oncologico, IRCCS, Aviano, Italy and others
- Corresponding author(s): Jerry Polesel
- Major finding: The authors' analysis confirmed red meat consumption as a risk factor for several cancer sites, including pancreas, with a limited impact of cooking methods. These findings, thus, call for a limitation of its consumption in populations of Western countries.

**Seropositivity to Helicobacter pylori and risk of pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24089457

- Journal: *Cancer Epidemiology, Biomarkers & Prevention*
- Institution(s): National Cancer Institute, Bethesda, MD
- Corresponding author(s): Guoqin Yu
- Major finding: The authors' results suggest that H. pylori is not a risk factor for pancreatic cancer.

**PREVENTION**

**Beyond aspirin—cancer prevention with statins, metformin and bisphosphonates**

http://www.ncbi.nlm.nih.gov/pubmed/24080598

- Journal: *Nature Reviews Clinical Oncology*
- Institution(s): Technion–Israel Institute of Technology and Clalit National Israeli Cancer Control Center (NICCC), Haifa, Israel
- Corresponding author(s): Gad Rennert
- Major finding: This Review discusses three pharmacological agents with the most evidence for their potential as cancer chemopreventive agents: anti–hypercholesterolaemia medications (statins), an antidiabetic agent (metformin) and antiosteoporosis drugs (bisphosphonates). At the current level of evidence these potential chemopreventive drugs should be considered in high–risk situations or using the personalized approach of maximizing individual benefits and minimizing the potential for adverse effects with the aid of pharmacogenetic indicators.

**EARLY DETECTION, DIAGNOSIS, AND PROGNOSIS**

**Novel methylation biomarker panel for the early detection of pancreatic cancer**

http://www.ncbi.nlm.nih.gov/pubmed/24088737

- Journal: *Clinical Cancer Research*
- Institution(s): Johns Hopkins University, Baltimore, MD and others
- Corresponding author(s): Nita Ahuja, Stephen Baylin, and Joo Mi Yi
- Pancreatic Cancer Action Network-affiliated authors:
    - Christine Iacobuzio-Donahue, MD, PhD: 2007 Pilot Grant and member, Scientific Advisory Board
    - Michael Goggins, MD: PI, 2013 Skip Viragh – Inaugural Research Acceleration Network (RAN) Grant
- Major finding: Promoter DNA methylation of *BNC1* and *ADAMTS1* is a potential biomarker to detect early-stage pancreatic cancers. Assaying the promoter methylation status of these genes in circulating DNA from serum is a promising strategy for early detection of pancreatic cancer and has the potential to improve mortality from this disease.

**Loss of PTEN expression predicts poor prognosis in patients with IPMNs of the pancreas**

http://www.ncbi.nlm.nih.gov/pubmed/24132918

- Journal: *Clinical Cancer Research*
- Institution(s): Columbia University, New York, NY
- Corresponding author(s): Gloria Su
- Pancreatic Cancer Action Network-affiliated author: Gloria Su, PhD: 2010 Innovative Grant and 2007 Pilot Grant
- Major finding: This is the first report of AKT1 mutations in intraductal papillary mucinous neoplasms (IPMN). The authors' data indicate that oncogenic activation of the phosphatidylinositol-3 kinase pathway (PI3K) pathway can contribute to the progression of IPMN, in particular loss of PTEN expression. This finding suggests the potential employment of PI3K pathway-targeted therapies for IPMN patients. The incorporation of PTEN expression status in making surgical decisions may also benefit IPMN patients and should warrant further investigation.

**Further characterization of the target of a potential aptamer biomarker for pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24152208

- Journal: *Nucleic Acid Therapeutics*
- Institution(s): Duke University School of Medicine, Durham, NC
- Corresponding author(s): Partha Ray and Rebekah White
- Pancreatic Cancer Action Network-affiliated author: Rebekah White, PhD: 2007 Seena Magowitz – Career Development Award
- Major finding: The ability to distinguish between forms of the same protein with differing post-translational modifications is an important advantage of aptamers as tools for identification and detection of biomarkers.

**Prediagnostic body mass index and pancreatic cancer survival**
http://www.ncbi.nlm.nih.gov/pubmed/24145341

- Journal: *Journal of Clinical Oncology*
- Institution(s): Dana-Farber Cancer Institute, Boston, MA and others
- Corresponding author(s): Brian Wolpin
- Major finding: This article picked up a great deal of media attention. The authors found that higher prediagnostic body mass index (BMI) was associated with statistically significantly decreased survival among patients with pancreatic cancer from two large prospective cohorts.

**In vivo identification of pancreatic cystic neoplasms with needle-based confocal laser endomicroscopy**
http://www.ncbi.nlm.nih.gov/pubmed/24163192

- Journal: *Endoscopy*
- Institution(s): University of Chicago Medicine, Chicago, IL and others
- Corresponding author(s): Irving Waxman
- Major finding: Endoscopic ultrasound (EUS) with fine-needle aspiration (FNA) of pancreatic cystic lesions (PCL) is flawed by inadequate diagnostic yield. Needle-based confocal laser endomicroscopy (nCLE) utilizes a sub-millimeter probe that is compatible with an EUS needle and enables real-time imaging with microscopic detail of PCL. The authors' preliminary data suggested that nCLE has a high specificity in the detection of PCN, but may be limited by a low sensitivity. The safety of nCLE requires further evaluation.

**Intensity of follow-up after pancreatic cancer resection**
http://www.ncbi.nlm.nih.gov/pubmed/24092447

- Journal: *Annals of Surgical Oncology*
- Institution(s): Vanderbilt University Medical Center, Nashville, TN
- Corresponding author(s): Nipun Merchant
- Major finding: Recent therapeutic advances may have the potential to significantly alter survival after recurrence, but a careful consideration of current surveillance strategies should be undertaken to optimize existing approaches in the face of high recurrence and low survival rates.

**The relationship between multiple clinicopathological features, nerve invasion in pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24103287

- Journal: *Hepatobiliary & Pancreatic Diseases International*
- Institution(s): Fudan University, Shanghai, China

- Corresponding author(s): Liu-Bin Shi
- Major finding: The authors' data indicated that the preoperative fasting blood glucose level, serum CA19-9 level, and referred pain are novel predictive markers for neural invasion in patients with pancreatic cancer (PC). p53 and Ki67 play important roles in neural invasion of PC. Management of hyperglycemia may serve as an auxiliary treatment to curb neural invasion in PC.

**CA125 is superior to CA19-9 in predicting the resectability of pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24146342

- Journal: *Journal of Gastrointestinal Surgery*
- Institution(s): Fudan University, Shanghai, China
- Corresponding author(s): Xianjun Yu
- Major finding: CA125 is superior to CA19-9 in predicting the resectability of pancreatic cancer. Aberrant high levels of CA125 may indicate unresectable pancreatic cancer.

**False biomarker discovery due to reactivity of commercial ELISA for CUZD1 with cancer antigen CA125**
http://www.ncbi.nlm.nih.gov/pubmed/24097894

- Journal: *Clinical Chemistry*
- Institution(s): University of Toronto, Toronto, ON, Canada and others
- Corresponding author(s): Eleftherios Diamandis
- Major finding: By using proteomics and bioinformatics, the authors have previously identified a group of highly pancreas-specific proteins as candidate pancreatic ductal adenocarcinoma biomarkers. The authors conclude that poor characterization of commercial ELISA assays is a factor that could lead to false biomarker discovery. To their knowledge, this is the first report documenting that a commercial ELISA marketed for one analyte (CUZD1) may, in fact, recognize a different, nonhomologous antigen (CA125).

**Predictive value of maximum standardized uptake value (SUVmax) on 18F-FDG PET/CT**
http://www.ncbi.nlm.nih.gov/pubmed/24107806

- Journal: *Clinical Nuclear Medicine*
- Institution(s): Kyung Hee University Hospital at Gangdong, Seoul, Republic of Korea
- Corresponding author(s): You-Jung Yang
- Major finding: Higher maximum standardized uptake value ($SUV_{max}$) of primary pancreatic tumor is associated with poor progression free survival (PFS) and pretreatment $SUV_{max}$ is an independent prognostic factor for predicting PFS in patients with locally advanced or metastatic pancreatic cancer who received gemcitabine-based palliative chemotherapy. However, pretreatment $SUV_{max}$ is not associated with chemotherapeutic response.

**Retrospective analysis: early CA19-9 change in salvage chemotherapy for refractory pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24121456

- Journal: *Cancer Chemotherapy and Pharmacology*
- Institution(s): University of Tokyo, Tokyo, Japan
- Corresponding author(s): Hiroyuki Isayama
- Major finding: CA19-9 change after the first course was prognostic of progression free survival and overall survival in refractory pancreatic cancer. Early discontinuation should be considered given the palliative setting.

**Preoperative CEA and CA 19-9 are prognostic markers for survival after curative resection**
http://www.ncbi.nlm.nih.gov/pubmed/24161419

- Journal: *International Journal of Surgery*
- Institution(s): Technical University Dresden, Dresden, Germany
- Corresponding author(s): Marius Distler
- Major finding: Preoperative CEA and CA 19-9 levels correlate with patient prognosis after curative pancreatic resection due to pancreatic ductal adenocarcinoma (PDAC). This is especially true for the most frequently pT 3/4 stages of PDAC. Even if CEA and CA 19-9 might not be appropriate for screening, its serum levels should therefore be determined prior to operation and taken into account when resectability or operability is doubtful.

**The combination of serum CA19-9 and CEA is a simple and accurate predictor of mortality**
http://www.ncbi.nlm.nih.gov/pubmed/24114022

- Journal: *Surgery Today*
- Institution(s): Nagoya University Graduate School of Medicine, Nagoya, Japan
- Corresponding author(s): Tsutomu Fujii
- Major finding: The authors evaluated the prognostic impact of carbohydrate antigen 19-9 (CA19-9) and carcinoembryonic antigen (CEA), and also evaluated the indices by multiplying the values of two tumor markers (e.g., CA19-9 × CEA). The CA19-9 × CEA index is a strong prognostic biomarker that could help identify pancreatic cancer patients expected to have a poor prognosis so that they can be administered appropriate multidisciplinary treatment.

**Prognostic and predictive blood-based biomarkers: Results from CALGB 80303 (Alliance)**
http://www.ncbi.nlm.nih.gov/pubmed/24097873

- Journal: *Clinical Cancer Research*
- Institution(s): Duke University, Durham, NC and others
- Corresponding author(s): Herbert Hurwitz
- Major finding: This study identified strong prognostic markers for pancreatic cancer patients. Predictive marker analysis indicated that plasma levels of VEGF-D, Ang2, and SDF1 significantly predicted for benefit or lack of benefit from bevacizumab in this population.

**Global analysis of multitrial data investigating quality of life and symptoms as prognostic factors**
http://www.ncbi.nlm.nih.gov/pubmed/24127333

- Journal: *Cancer*
- Institution(s): European Organization for Research and Treatment of Cancer Headquarters, Brussels, Belgium and others
- Corresponding author(s): Chantal Quinten
- Major finding: The current results demonstrated that, for each cancer site, at least 1 health-related quality of life (HRQOL) domain provided prognostic information that was additive over and above clinical and sociodemographic variables. In pancreatic cancer, global health status was predictive for survival.

**Suppressed expression of *NDRG2* correlates with poor prognosis in pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24134849

- Journal: *Biochemical and Biophysical Research Communications*
- Institution(s): Tohoku University, Graduate School of Medicine, Sendai, Japan and others

- Corresponding author(s): Akira Horii
- Major finding: The authors' present results suggest that (1) N-myc downstream regulated gene 2 (*NDRG2*) is functioning as one of the candidate tumor-suppressor genes in pancreatic carcinogenesis, (2) epigenetic mechanisms such as histone modifications play an essential role in *NDRG2* silencing, and (3) the expression of NDRG2 is an independent prognostic factor in pancreatic cancer.

**Nuclear Nrf2 expression is related to a poor survival in pancreatic adenocarcinoma**
http://www.ncbi.nlm.nih.gov/pubmed/24189098

- Journal: *Pathology – Research and Practice*
- Institution(s): University of Eastern Finland, Kuopio, Finland and others
- Corresponding author(s): Ylermi Soini
- Major finding: According to the results, nuclear Nrf2 expression predicts a worse survival in pancreatic adenocarcinoma, which is in keeping with its protection of cells against oxidative or xenobiotic stress. In accordance with Nrf2's regulation of the synthesis of sulfiredoxin, there was an association between them. DJ1 had no association with Nrf2, and its expression predicted a better survival of patients.

**Is detection of circulating tumor cells in locally advanced pancreatic cancer useful prognostic marker?**
http://www.ncbi.nlm.nih.gov/pubmed/24127751
*Evaluation of: http://www.ncbi.nlm.nih.gov/pubmed/23676420*

- Journal: *Expert Review of Molecular Diagnostics*
- Institution(s): Imperial College, London, UK
- Corresponding author(s): Tamara Gall
- Major finding: The authors note that although Bidard *et al* is a well-designed study, the small number of patients with detectable circulating tumor cells means that the statistical power is not great enough to make firm conclusions. Therefore, this expensive assay needs further investigation before being used a prognostic marker in patients with locally advanced pancreatic cancer.

**Pattern and clinical predictors of lymph node involvement in nonfunctioning pancreatic NETs**
http://www.ncbi.nlm.nih.gov/pubmed/23986355

- Journal: *JAMA Surgery*
- Institution(s): University of Verona, Verona, Italy and others
- Corresponding author(s): Massimo Falconi
- Major finding: Patients with grade 1 nonfunctioning pancreatic neuroendocrine tumors (NF-PanNET-G1) have a very low risk of pathological lymph node involvement (pN+) in the absence of radiological signs of node involvement. When preoperative grading assessment is not achieved, the radiological size of the lesion is a powerful alternative predictor of pN+. The risk of pathological nodal involvement in patients with NF-PanNETs can be accurately estimated by a clinical predictive model.

**Biomarker in bile VEGF can correctly identify pancreatic cancer with high sensitivity**
http://gi.org/media/press-releases-for-acg-annual-scientific-meeting/vegf-pancreatic-cancer/
*Abstract search: http://www.eventscribe.com/2013/ACG/*

- Meeting: 78th Annual Scientific Meeting of the American College of Gastroenterology

- Institution(s): Cleveland Clinic Foundation, Cleveland, OH
- Major finding: A marker in bile known as vascular endothelial growth factor (VEGF) plays an important role in the growth of cancerous tumors. Researchers found that VEGF levels from bile aspirated from the pancreas can accurately distinguish pancreatic cancer from other causes of common problems in the bile duct. The results of this pilot study indicated that using this marker in bile can correctly identify pancreas cancer with high sensitivity, detecting pancreas cancer accurately in 93 percent of cases.

**RedPath announces results of National Pancreatic Cyst Registry at 2013 ACG Annual Meeting**
http://redpathip.com/redpath-announces-results-of-national-pancreatic-cyst-registry-at-2013-acg-annual-meeting
*Abstract search: http://www.eventscribe.com/2013/ACG/*

- Meeting: 78th Annual Scientific Meeting of the American College of Gastroenterology
- Company: RedPath Integrated Pathology, Inc., Pittsburgh, PA
- Major finding: Final data results validate the company's PathFinderTG® pancreas platform's high negative predictive value (NPV=97%) for correctly identifying patients at very low risk of developing pancreatic cancer.

**TREATMENT**

**Targeting metabolic scavenging in pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24166909
*Commentary on: http://clincancerres.aacrjournals.org/content/early/2013/09/10/1078-0432.CCR-13-0150.abstract?sid=688748e8-24d6-44ed-b09d-5ad5910f70a8*

- Journal: *Clinical Cancer Research*
- Institution(s): Weill Cornell Medical College, New York City, NY
- Corresponding author(s): Lewis Cantley
- Pancreatic Cancer Action Network-affiliated author: Costas Lyssiotis, PhD: 2013 Pathway to Leadership Grant
- Major finding: Pancreatic tumor metabolism is rewired to facilitate survival and growth in a nutrient-depleted environment. This leads to a unique dependence on metabolic recycling and scavenging pathways, including NAD salvage. Targeting this pathway in pancreatic cancer disrupts metabolic homeostasis and impairs tumor growth.

**Angiotensin inhibition enhances drug delivery and potentiates chemotherapy**
http://www.ncbi.nlm.nih.gov/pubmed/24084631

- Journal: *Nature Communications*
- Institution(s): Massachusetts General Hospital and Harvard Medical School, Boston, MA and others
- Corresponding author(s): Rakesh Jain
- Pancreatic Cancer Action Network-affiliated author: Yves Boucher, PhD: 2013 Abby Sobrato – Innovative Grant
- Major finding: Cancer and stromal cells actively exert physical forces (solid stress) to compress tumor blood vessels, thus reducing vascular perfusion. Thus, angiotensin inhibitors - inexpensive drugs with decades of safe use - could be rapidly repurposed as cancer therapeutics.

**The diagnosis and surgical treatment of pancreatoblastoma in adults: Case series, review of literature**
http://www.ncbi.nlm.nih.gov/pubmed/24081396

- Journal: *Journal of Gastrointestinal Surgery*
- Institution(s): Johns Hopkins Medical Institutions, Baltimore, MD and others
- Corresponding author(s): Christopher Wolfgang
- Pancreatic Cancer Action Network-affiliated authors:
    - Ralph Hruban, MD: member, Emeritus Scientific Advisory Board
    - Joseph Herman, MD: 2008 Blum-Kovler – Career Development Award and member, Medical Advisory Board
- Major finding: The aim of this study was to report the authors' experience with adult pancreatoblastoma as well as review the cases reported in the literature in order to provide guidelines for the management of patients with this rare neoplasm. When disease is localized, the treatment of choice is a complete surgical resection. The role of adjuvant chemotherapy or radiotherapy is still unclear based on the very small number of patients treated.

**Is surgical intervention for cystic neoplasms of the pancreas being underutilized?**
http://www.ncbi.nlm.nih.gov/pubmed/24170607

- Journal: *Journal of Gastrointestinal Surgery*
- Institution(s): Indiana University School of Medicine, Indianapolis, IN
- Corresponding author(s): C. Max Schmidt
- Pancreatic Cancer Action Network-affiliated author: C. Max Schmidt, MD, PhD: 2003 Career Development Award
- Major finding: In light of accepted parameters to guide intraductal papillary mucinous neoplasm (IPMN) surveillance/intervention being suboptimal, the pendulum may have swung too far and surgical intervention may indeed be underutilized.

**Radiographic tumor-vein interface as a predictor of intraoperative, pathologic, oncologic outcomes**
http://www.ncbi.nlm.nih.gov/pubmed/24129826

- Journal: *Journal of Gastrointestinal Surgery*
- Institution(s): University of Texas M.D. Anderson Cancer Center, Houston, TX and others
- Corresponding author(s): Matthew Katz
- Pancreatic Cancer Action Network-affiliated authors:
    - Huamin Wang, MD, PhD: 2007 Skip Viragh – Career Development Award
    - Christopher Crane, MD: member, Medical Advisory Board
    - Jason Fleming, MD: member, Medical Advisory Board
- Major finding: A tomographic classification of the tumor– superior mesenteric–portal vein (SMV-PV) interface can predict the need for venous resection, pathologic venous involvement, and survival. To assist in treatment planning, a standardized assessment of this anatomic relationship should be routinely performed.

**Morbidity and mortality after pancreaticoduodenectomy in patients with borderline resectable type C**
http://www.ncbi.nlm.nih.gov/pubmed/24129825

- Journal: *Journal of Gastrointestinal Surgery*
- Institution(s): University of Texas M.D. Anderson Cancer Center, Houston, TX
- Corresponding author(s): Thomas Aloia

- Pancreatic Cancer Action Network-affiliated author: Jason Fleming, MD: member, Medical Advisory Board
- Major finding: Nationwide, one third of patients undergoing pancreaticoduodenectomy (PD) are medically borderline. These "borderline resectable type C" (BR-C) patients are at higher risk for and less able to be rescued from postoperative major complications. Surgeons should identify and optimize comorbidities and utilize prehabilitation to address functional deficits before elective PD.

**Harnessing immune responses in the tumor microenvironment: All signals needed**
http://www.ncbi.nlm.nih.gov/pubmed/24097857
*Commentary on: http://www.ncbi.nlm.nih.gov/pubmed/23983255*

- Journal: *Clinical Cancer Research*
- Institution(s): Johns Hopkins School of Medicine, Baltimore, MD
- Corresponding author(s): Elizabeth Jaffee
- Pancreatic Cancer Action Network-affiliated author: Elizabeth Jaffee, MD: member, Emeritus Scientific Advisory Board
- Major finding: An agonist CD40 monoclonal antibody (CP-870,893) in combination with gemcitabine is well tolerated in patients with advanced pancreatic adenocarcinoma. The combination results in induction of cytokines, B cell activation, and clinical responses. These findings support testing immunotherapies in combination with other established and targeted therapies.

**Synergistic effects of concurrent blockade of PI3K and MEK pathways in preclinical models**
http://www.ncbi.nlm.nih.gov/pubmed/24130864

- Journal: *PLoS One*
- Institution(s): Washington University in St. Louis, St. Louis, MO and others
- Corresponding author(s): Andrea Wang-Gillam
- Pancreatic Cancer Action Network-affiliated author: William Hawkins, MD: 2005 Skip Viragh – Career Development Award
- Major finding: The authors' study provides the rationale for concurrent targeting of the PI3K and MEK pathways, regardless of KRAS status, and suggests that phosphorylation of the cap-dependent translational components, 4E-binding protein (p-4E-BP1) and S6 can serve as a predictive biomarker for response to treatment.

**The NCI-ASCO Cancer Trial Accrual Symposium: Summary and recommendations**
http://www.ncbi.nlm.nih.gov/pubmed/24130252

- Journal: *Journal of Oncology Practice*
- Institution(s): National Cancer Institute; Education Network to Advance Cancer Clinical Trials, Bethesda, MD and others
- Corresponding author(s): Andrea Denicoff
- Pancreatic Cancer Action Network-affiliated author: Michelle Duff, DPT, former Director of Research and Scientific Affairs, Pancreatic Cancer Action Network
- Major finding: A combination of approaches addressing both the multifactorial nature of accrual challenges and the characteristics of the target population may be needed to improve accrual to cancer clinical trials. Recommendations for best practices and for future research developed from the Cancer Trial Accrual Symposium: Science and Solutions on April 29-30, 2010

(cosponsored by The National Cancer Institute [NCI] and the American Society of Clinical Oncology [ASCO]) are provided.

**Increased survival in pancreatic cancer with nab-paclitaxel plus gemcitabine**
http://www.ncbi.nlm.nih.gov/pubmed/24131140

- Journal: *New England Journal of Medicine*
- Institution(s): Translational Genomics Research Institute, Phoenix, AZ and others
- Corresponding author(s): Daniel Von Hoff
- Major finding: In patients with metastatic pancreatic adenocarcinoma, nab-paclitaxel plus gemcitabine significantly improved overall survival, progression-free survival, and response rate, but rates of peripheral neuropathy and myelosuppression were increased.

**Adjuvant chemotherapy with gemcitabine and long-term outcomes in resected pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24104372

- Journal: *JAMA*
- Institution(s): Charité–Universitätsmedizin Berlin, Berlin, Germany and others
- Corresponding author(s): Hanno Riess
- Major finding: Among patients with macroscopic complete removal of pancreatic cancer, the use of adjuvant gemcitabine for 6 months compared with observation alone resulted in increased overall survival as well as disease-free survival. These findings provide strong support for the use of gemcitabine in this setting.

**Efficacy of nimotuzumab plus gemcitabine usage as first-line treatment in advanced pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24142531

- Journal: *Tumor Biology*
- Institution(s): Chinese PLA General Hospital, Beijing, China
- Corresponding author(s): Li Bai
- Major finding: The objective of the current study was to evaluate the contribution of a monoclonal antibody against epidermal growth factor receptor (EGFR), nimotuzumab, to standard gemcitabine therapy in patients with unresectable locally advanced or metastatic pancreatic adenocarcinoma. Nimotuzumab as a high-purity humanized monoclonal antibody with favorable safety profile, its value in the treatment of pancreatic cancer along with gemcitabine, particularly in the comprehensive treatment of advanced pancreatic cancer, is appealing for further prospective randomized large-scale clinical trials.

**Phase II clinical study of alternate-day oral therapy with S-1 as first-line chemotherapy**
http://www.ncbi.nlm.nih.gov/pubmed/24146260

- Journal: *Cancer Chemotherapy and Pharmacology*
- Institution(s): Wakayama Medical University, Wakayama, Japan
- Corresponding author(s): Hiroki Yamaue
- Major finding: The authors conducted a multicenter cooperative prospective study to compare daily with alternate-day administration of S-1 for advanced pancreatic cancer. The current data demonstrate the mitigation of adverse effects with alternate-day administration of S-1, and this appears to be a more sustainable option for advanced pancreatic cancer.

**S-1 plus CIK as second-line treatment for advanced pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24122257

- Journal: *Medical Oncology*
- Institution(s): Shan Dong Ji Ning First People's Hospital, Jining, People's Republic of China
- Corresponding author(s): Jie-lin Qi
- Major finding: This study aimed to evaluate the efficacy and tolerability of S-1 (Tegafur, Gimeracil, and Oteracil Potassium Capsules) plus CIK (Cytokine-induced killer cells) in patients with advanced pancreatic cancer who had previously received gemcitabine-based therapy. The S-1 plus CIK regimen was well tolerated in a second-line setting in patients with gemcitabine-refractory and advanced pancreatic cancer.

**Randomized double-blinded, placebo-controlled phase II trial of simvastatin and gemcitabine**
http://www.ncbi.nlm.nih.gov/pubmed/24162380

- Journal: *Cancer Chemotherapy and Pharmacology*
- Institution(s): Sungkyunkwan University School of Medicine, Seoul, Korea and others
- Corresponding author(s): Young Suk Park
- Major finding: Adding low-dose statin drug simvastatin to gemcitabine in advanced pancreatic cancer does not provide clinical benefit, although it also does not result in increased toxicity. Given the emerging role of statins in overcoming resistance to anti-EGFR treatment, further studies are justified to evaluate the efficacy and safety of combined simvastatin and anti-EGFR agents, such as erlotinib or cetuximab, plus gemcitabine for treating advanced pancreatic cancer.

**Two immune faces of pancreatic adenocarcinoma: possible implication for immunotherapy**
http://www.ncbi.nlm.nih.gov/pubmed/24129765

- Journal: *Cancer Immunology, Immunotherapy*
- Institution(s): University of Heidelberg, Heidelberg, Germany
- Corresponding author(s): Alexandr Bazhin
- Major finding: These results allowed the authors to conclude that pancreatic ductal adenocarcinoma (PDAC) provokes not only an anti-tumor immune response, but also strong immune suppression. Thus, the authors supposed that new immunotherapeutical strategies should involve not only stimulation of the immune system of PDAC patients, but also exert control over the tumor immune suppressive milieu.

**Current advances in immunotherapy for pancreatic cancer**
http://www.sciencedirect.com/science/article/pii/S0147027213000925

- Journal: *Current Problems in Cancer*
- Institution(s): Johns Hopkins University, School of Medicine, Baltimore, MD
- Corresponding author(s): Dung Le
- Major finding: In this review, the authors will discuss the most recent immunotherapy failures and successes, as well as future treatment strategies for pancreatic ductal adenocarcinoma.

**Combining two strategies to improve perfusion and drug delivery in solid tumors**
http://www.ncbi.nlm.nih.gov/pubmed/24167277

- Journal: *PNAS*
- Institution(s): University of Cyprus, Nicosia, Cyprus and others

- Corresponding author(s): Rakesh Jain
- Major finding: Therapeutic strategies to improve perfusion include reduction in vascular permeability by vascular normalization and vascular decompression by alleviating physical forces (solid stress) inside tumors. Solid stress alleviation is more beneficial for compressed but less-permeable vessels (e.g., pancreatic ductal adenocarcinomas).

**The stromal compartments in pancreatic cancer: Are there any therapeutic targets?**
http://www.ncbi.nlm.nih.gov/pubmed/24141189

- Journal: *Cancer Letters*
- Institution(s): University of Oxford, Oxford, UK and others
- Corresponding author(s): Thomas Brunner
- Major finding: In this review the authors described the main actors of the desmoplastic reaction within pancreatic ductal adenocarcinoma and novel therapeutic approaches that are being tested to block the detrimental function of the stroma.

**Two-wave nanotherapy to target the stroma and optimize gemcitabine delivery to a model in mice**
http://www.ncbi.nlm.nih.gov/pubmed/24143858

- Journal: *ACS Nano*
- Institution(s): University of California, Los Angeles, Los Angeles, CA and others
- Corresponding author(s): Andre Nel and Huan Meng
- Major finding: Not only does the authors' approach overcome stromal resistance to drug delivery in pancreatic ductal adenocarcinoma, but it also introduces the concept of using a stepwise engineered approach to address a range of biological impediments that interfere in nanocancer therapy in a spectrum of cancers.

**Genetic and molecular alterations in pancreatic cancer: Implications for personalized medicine**
http://www.ncbi.nlm.nih.gov/pubmed/24172537

- Journal: *Medical Science Monitor*
- Institution(s): Baylor College of Medicine, Houston, TX and others
- Corresponding author(s): Changyi (Johnny) Chen
- Major finding: This review aims to summarize recent advances of important genes, proteins, and microRNAs that play a critical role in the pathogenesis of pancreatic cancer, and to provide implications for personalized medicine in pancreatic cancer.

**Pancreatic cancer genomes: toward molecular subtyping and novel approaches to diagnosis, therapy**
http://www.ncbi.nlm.nih.gov/pubmed/23757204

- Journal: *Molecular Diagnosis & Therapy*
- Institution(s): Johns Hopkins University School of Medicine, Baltimore, MD
- Corresponding author(s): Laura Wood
- Major finding: This review discusses the molecular alterations underlying pancreatic neoplasms as well as the clinical impact of these alterations for diagnosis and treatment.

**Overview of the pancreatic toxicity and carcinogenesis session**
http://www.ncbi.nlm.nih.gov/pubmed/24159055

- Journal: *Toxicologic Pathology*
- Institution(s): Experimental Pathology Laboratories, Inc., Research Triangle Park, NC and others

- Corresponding author(s): Arun Pandiri
- Major finding: The theme of the Society of Toxicologic Pathology Annual Symposium 2013 was "Toxicologic Pathology of the Digestive Tract and Pancreas." The last session focused on pancreatic toxicity and carcinogenesis. This overview highlights the various presentations in this session, focusing on pancreatic toxicologic pathology, responses of the pancreas to xenobiotics, and current understanding on pancreatic carcinogenesis. The objective of this symposium overview and the subsequent articles from this session is to enable the audience to develop a better appreciation for the pancreas as a target organ in toxicological studies.

**Improvement of surgical results for pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24079875

- Journal: *The Lancet Oncology*
- Institution(s): University of Heidelberg, Heidelberg, Germany
- Corresponding author(s): Markus W Büchler
- Major finding: With increasing evidence on surgical and perioperative aspects of pancreatic cancer therapy, short-term and long-term outcomes of resectable and borderline resectable pancreatic cancer are improving.

**Crizotinib inhibits metabolic inactivation of gemcitabine in c-Met-driven pancreatic carcinoma**
http://www.ncbi.nlm.nih.gov/pubmed/24085787

- Journal: *Cancer Research*
- Institution(s): VU University Medical Center, Amsterdam, the Netherlands and others
- Corresponding author(s): Elisa Giovannetti
- Major finding: Together, these more readily imaged orthotopic pancreatic ductal adenocarcinoma (PDAC) models displayed genetic, histopathologic, and metastatic features similar to their human tumors of origin. Moreover, their use pointed to c-Met as a candidate therapeutic target in PDAC and highlighted crizotinib and gemcitabine as a synergistic combination of drugs warranting clinical evaluation for PDAC treatment.

**Antitumor effects of immunotoxins are enhanced by lowering HCK or treatment with Src inhibitors**
http://www.ncbi.nlm.nih.gov/pubmed/24145282

- Journal: *Molecular Cancer Therapeutics*
- Institution(s): National Cancer Institute, National Institutes of Health, Bethesda, MD
- Corresponding author(s): Ira Pastan
- Major finding: The authors' data suggest that the combination of immunotoxin with tyrosine kinase inhibitors may be an effective way to treat some cancers.

**Selective disruption of Rb-Raf-1 kinase interaction inhibits pancreatic adenocarcinoma growth**
http://www.ncbi.nlm.nih.gov/pubmed/24107447

- Journal: *Molecular Cancer Therapeutics*
- Institution(s): University of Florida, Gainesville, FL and others
- Corresponding author(s): Srikumar Chellappan
- Major finding: Inactivation of the Retinoblastoma tumor suppressor protein Rb is thought to be initiated by association with Raf-1 (C-Raf) kinase, and here the authors determined how RRD-251, a disruptor of the Rb-Raf-1 interaction, affects pancreatic tumor progression. Disruption of the Rb-Raf-1 interaction significantly reduces the malignant properties of pancreatic cancer cells

irrespective of their gemcitabine sensitivity. Selective targeting of Rb-Raf-1 interaction might be a promising strategy targeting pancreatic cancer.

**Erlotinib, gefitinib and vandetanib inhibit human nucleoside transporters and protect cancer cells**
http://www.ncbi.nlm.nih.gov/pubmed/24170548

- Journal: *Clinical Cancer Research*
- Institution(s): Cross Cancer Institute, Edmonton, AB, Canada and others
- Corresponding author(s): Michael Sawyer
- Major finding: Tyrosine kinase inhibitor (TKI) inhibition of uridine transport was studied with recombinant human (h) equilibrative (E) and concentrative (C) nucleoside transporters (hENTs, hCNTs) produced individually in yeast. Vandetanib inhibited hENT1, hENT2, hCNT1, hCNT2 and hCNT3 whereas erlotinib inhibited hENT1 and hCNT3 and gefitinib inhibited hENT1 and hCNT1. The potential for reduced accumulation of nucleoside chemotherapy drugs in tumor tissues due to inhibition of hENTs and/or hCNTs by TKIs indicates that pharmacokinetic properties of these agents must be considered when scheduling TKIs and nucleoside chemotherapy in combination.

**Combination treatment of human pancreatic cancer xenograft models with erlotinib and HF10**
http://www.ncbi.nlm.nih.gov/pubmed/24170435

- Journal: *Annals of Surgical Oncology*
- Institution(s): Nagoya University Graduate School of Medicine, Nagoya, Aichi, Japan and others
- Corresponding author(s): Hideki Kasuya
- Major finding: Combination therapy with HF10 (a herpes simplex virus type 1 [HSV-1] mutant), and erlotinib warrants further investigation to establish a new treatment strategy against human pancreatic cancers.

**Targeting and cytotoxicity of SapC-DOPS nanovesicles in pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24124494

- Journal: *PLoS One*
- Institution(s): University of Cincinnati College of Medicine, Cincinnati, OH
- Corresponding author(s): Xiaoyang Qi
- Major finding: The authors' data suggest that the acidic phospholipid phosphatidylserine (PS) is a biomarker for pancreatic cancer that can be effectively targeted for therapy utilizing cancer-selective SapC-DOPS nanovesicles. This study provides convincing evidence in support of developing a new therapeutic approach to pancreatic cancer.

**Synergistic combination of valproic acid and oncolytic parvovirus H-1PV against pancreatic carcinomas**
http://www.ncbi.nlm.nih.gov/pubmed/24092664

- Journal: *EMBO Molecular Medicine*
- Institution(s): German Cancer Research Center (DKFZ), Heidelberg, Germany and others
- Corresponding author(s): Antonio Marchini
- Major finding: The rat parvovirus H-1PV has oncolytic and tumor-suppressive properties potentially exploitable in cancer therapy. Here the authors show that this can be achieved by co-treating cancer cells with H-1PV and histone deacetylase inhibitors (HDACIs) such as valproic acid (VPA). These results warrant clinical evaluation of H-1PV/VPA co-treatment against cervical and pancreatic ductal carcinomas.

**The FAK scaffold inhibitor C4 disrupts FAK-VEGFR-3 signaling and inhibits pancreatic cancer growth**
http://www.ncbi.nlm.nih.gov/pubmed/24142503

- Journal: *Oncotarget*
- Institution(s): Roswell Park Cancer Institute, Buffalo, NY and others
- Corresponding author(s): Elena Kurenova and William Cance
- Major finding: Taken together, these results demonstrate that targeting the scaffolding function of focal adhesion kinase (FAK) with a small-molecule FAK-VEGFR-3 (vascular endothelial growth factor receptor 3) inhibitor can be an effective therapeutic strategy against pancreatic ductal adenocarcinoma.

**Effect of combined treatment with recombinant interleukin-2 and allicin on pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24135803

- Journal: *Molecular Biology Reports*
- Institution(s): Tongji University School of Medicine, Shanghai, China and others
- Corresponding author(s): Rong-Hua Xu and Hui Zhang
- Major finding: Combined treatment of xenograft mice with allicin and recombinant interleukin-2 (rIL-2) resulted in suppression of tumor growth and prolonged survival time possibly through activation of CD4+T, CD8+T and NK cell.

**Proteomic strategy for probing complementary lethality of kinase inhibitors against pancreatic cancer**
http://www.ncbi.nlm.nih.gov/pubmed/24167056

- Journal: *Proteomics*
- Institution(s): Carolinas HealthCare System, Charlotte, NC
- Corresponding author(s): Sun-Il Hwang
- Major finding: The results suggest that induction of chemosensitization by manipulating specific molecular targets can potentiate synergistic chemotherapeutic effects at lower, better tolerated doses, and in turn reduce the toxicity of multidrug treatment of pancreatic cancer.

**New drug combinations may benefit patients with pancreatic cancer**
http://www.aacr.org/home/public--media/aacr-in-the-news.aspx?d=3187
*Abstract: http://www.abstractsonline.com/Plan/ViewAbstract.aspx?mID=3429&sKey=b0b6cb86-622e-4564-a771-a80454a4f820&cKey=d94d019c-bc23-4260-8bec-40f2557e4085&mKey=18fa2242-fd0d-4689-82a0-e4d68ac8a74a*

- Meeting: AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics
- Institution: Johns Hopkins School of Medicine, Baltimore, MD and others
- Pancreatic Cancer Action Network-affiliated author: Anirban Maitra, MBBS: 2004 Career Development Award and chair, Scientific Advisory Board
- Major finding: The striking results obtained in these models appear to validate the authors' hypothesis that blocking multiple effector pathways downstream from KRAS, including the Ral effector pathway, may provide increased efficacy in pancreatic cancer. Thus, the combinations of Dinaciclib (CDK5 inhibitor) with the pan-AKT inhibitor MK2206 or the ERK inhibitor SCH772984 are novel, highly promising potential therapies against pancreatic cancer. Based on these data, an NCI-CTEP approved Phase I clinical trial for pancreatic cancer of the combination of Dinaciclib and MK2206 has now opened at Johns Hopkins.

**New idea for targeting the common cancer protein KRAS**
http://www.aacr.org/home/public--media/aacr-press-releases.aspx?d=3185
*http://www.abstractsonline.com/Plan/ViewAbstract.aspx?mID=3429&sKey=0ddcadfa-2cdb-4cb4-81af-ea91dc87ec44&cKey=4687bcb8-cb68-4e43-bf03-e4cc9f587ba2&mKey=18fa2242-fd0d-4689-82a0-e4d68ac8a74a*

- Meeting: AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics
- Institution: Vanderbilt University School of Medicine, Nashville, TN and others
- Major finding: Here the authors report the discovery of small molecules that bind to a unique pocket on the Ras:SOS:Ras complex, increase SOScat-catalyzed nucleotide exchange, and perturb Ras signaling pathways in cells. The authors' studies suggest a novel way to target K-Ras and offer possible starting points for the discovery of compounds that could be used to treat Ras-driven tumors.

**Antibody-drug conjugate may provide new treatment option for pancreatic cancer patients**
http://www.aacr.org/home/public--media/aacr-in-the-news.aspx?d=3188
*Abstract: http://www.abstractsonline.com/Plan/ViewAbstract.aspx?mID=3429&sKey=923a9946-24c0-4e78-a65f-91a5c89ef339&cKey=cd1fe313-c6de-466b-8b9e-d02bd39add0a&mKey=18fa2242-fd0d-4689-82a0-e4d68ac8a74a*

- Meeting: AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics
- Company: Millennium: the Takeda Oncology Company, Cambridge, MA
- Major finding: The authors' findings indicate that MLN0264 (a fully human anti-GCC [guanylyl cyclase C] monoclonal antibody linked to the microtubule-disrupting agent monomethyl auristatin E [MMAE] via a protease-cleavable linker) has antitumor activity as a single agent and in combination with gemcitabine in GCC-expressing pancreatic cancer xenograft models, and support clinical evaluation of MLN0264 in patients with pancreatic cancer.

**Preclinical data on TH-302 with gemcitabine and nab-paclitaxel in models of pancreatic cancer**
http://investor.thresholdpharm.com/releasedetail.cfm?ReleaseID=795328
*Abstract: http://www.abstractsonline.com/Plan/ViewAbstract.aspx?mID=3429&sKey=9bb824c9-d8b0-468c-8870-db76d30fed63&cKey=3e88ee57-7957-4b02-a067-c7ac4664667a&mKey=18fa2242-fd0d-4689-82a0-e4d68ac8a74a*

- Meeting: AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics
- Company: Threshold Pharmaceuticals, South San Francisco, CA
- Major finding: Preclinical data on the combination of TH-302 with Gemzar® (gemcitabine) and Abraxane® (nab-paclitaxel) in models of pancreatic cancer will be presented showing in xenograft models greater anti-tumor activity associated with the "triplet" (TH-302 plus gemcitabine plus nab-paclitaxel) compared with that of the doublet (gemcitabine plus nab-paclitaxel), and without additive hematological toxicity or peripheral neuropathy.

**OXiGENE presents preclinical data demonstrating significant antitumor activity of ZYBRESTAT®**
http://investor.oxigene.com/releasedetail.cfm?ReleaseID=798633
*Poster: http://www.oxigene.com/files/uploads/CA4P_posterDBRev.pdf*

*Albert Einstein College of Medicine press release:*
*http://www.einstein.yu.edu/news/releases/948/preclinical-study-finds-drug-helps-against-pancreatic-cancer/*
*Abstract: http://www.abstractsonline.com/Plan/ViewAbstract.aspx?mID=3429&sKey=43335476-b933-4b8a-9b96-57f8f9e50225&cKey=32eab685-06c7-4a5d-a097-2495aa76bd31&mKey=18fa2242-fd0d-4689-82a0-e4d68ac8a74a*

- Meeting: AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics
- Institution: Albert Einstein College of Medicine, Bronx, NY and others
- Major finding: Systemic administration of the systemically delivered vascular disrupting agent combretastatin A-4 phosphate (CA4P) is effective in treating functional insulinomas in a transgenic mouse model. Both circulating hormone and tumor size was significantly reduced without obvious toxicity. Further evaluation of the vascular disrupting agent CA4P as a therapy for patients with enteropancreatic neuroendocrine tumors is warranted.

**Eleison announces Phase III trial of glufosfamide for second-line treatment of pancreatic cancer**
http://eleison-pharma.com/cushy_uploads/news_16_4041856779.pdf

- Company: Eleison Pharmaceuticals LLC, St. Petersburg, FL
- Major finding: Eleison Pharmaceuticals, a specialty pharmaceutical company developing life-saving therapeutics for rare cancers, announced it has initiated a Phase III study of Glufosfamide for the second-line treatment of patients with pancreatic cancer. Glufosfamide, a new chemical entity, is a third-generation alkylating agent designed for greater specificity and tumor uptake, with reduced systemic toxicities and side effects.

**Immunomedics reports $^{90}$Y-clivatuzumab tetraxetan combination with low-dose gemcitabine is active**
http://www.immunomedics.com/pdfs/news/2013/pr10232013.pdf

- Company: Immunomedics, Inc., Lyon, France
- Major finding: Immunomedics, a biopharmaceutical company primarily focused on the development of monoclonal antibody-based products for the targeted treatment of cancer, autoimmune and other serious diseases, reported that multiple cycles of therapy with the Company's pancreatic cancer therapeutic, clivatuzumab tetraxetan labeled with yttrium-90 ($^{90}$Y), in combination with low-dose gemcitabine, produced a median overall survival (OS) of more than 5 months in patients with metastatic pancreatic cancer who had received at least 2 prior treatments.

**SWOG initiates randomized Phase 2 clinical trial of Halozyme's PEGPH20 with modified FOLFIRINOX**
http://www.halozyme.com/Investors/News-Releases/News-Release-Details/2013/SWOG-Initiates-Randomized-Phase-2-Clinical-Trial-of-Halozymes-PEGPH20-in-Combination-with-modified-FOLFIRINOX-for-Advanced-Pancreatic-Cancer/default.aspx

- Company: Halozyme Therapeutics, Inc., San Diego, CA
- Major finding: Halozyme Therapeutics announced the initiation of a Phase 1b/2 randomized clinical trial (S1313) of Halozyme's investigational drug PEGPH20 (PEGylated Recombinant Human Hyaluronidase) in combination with modified FOLFIRINOX chemotherapy (mFOLFIRINOX) compared to mFOLFIRINOX treatment alone in patients with metastatic pancreatic adenocarcinoma. The trial, which will enroll approximately 144 patients, is being sponsored by SWOG.

**Cornerstone Pharmaceuticals announces initiation of Phase II clinical trial of CPI-613**
http://www.cornerstonepharma.com/wp-content/uploads/CPI-Press-Rel-31October2013-Cornerstone-Pharamceuticals-Announces-Initiation-of-Phase-II-Clinical-Trial-of-CPI-613-in-Patients-with-Locally-Advanced-or-metastatic-Pancreatic-Cancer1.pdf

- Company: Cornerstone Pharmaceuticals, Inc., Cranbury, NJ
- Major finding: Cornerstone Pharmaceuticals, Inc., a leader in the growing field of cancer metabolism-based therapeutics, announced the initiation of a pilot Phase II clinical trial of CPI-613 for the treatment of patients with locally advanced or metastatic pancreatic cancer. CPI-613 is the Company's lead Altered Energy Metabolism Directed (AEMD) drug candidate, which is designed to disrupt the altered energy-production pathways in cancer cells.

**Aduro receives orphan drug designation for CRS-207 for pancreatic cancer**
http://www.businesswire.com/news/home/20131022005404/en

- Company: Aduro BioTech, Inc., Berkeley, CA
- Major finding: Aduro BioTech, announced that the Office of Orphan Products Development of the Food and Drug Administration (FDA) has granted orphan drug designation for CRS-207, a novel immunotherapy, for the treatment of pancreatic cancer. CRS-207 is a live-attenuated Listeria monocytogenes strain that has been genetically engineered to be safe for clinical use and to induce a potent immune response specific for the tumor-associated antigen mesothelin.

**Ganymed's IMAB362 receives orphan drug designation from FDA & EMA for pancreatic cancer**
http://www.dddmag.com/news/2013/10/fda-ema-grant-orphan-designation-pancreatic-cancer-drug

- Company: Ganymed Pharmaceuticals AG, Mainz, Germany
- Major finding: Ganymed Pharmaceuticals AG announced that the Food and Drug Administration (FDA) and the European Medicines Agency (EMA) have granted orphan drug designation to IMAB362 for the treatment of pancreatic cancer. IMAB362 is a monoclonal antibody currently in Phase 2b clinical trial for gastroesophageal cancer. IMAB362 is a first-in-class monoclonal antibody selectively binding to the tight junction protein CLDN18.2 which is expressed in approximately 60 percent of primary and metastatic pancreatic cancers.

**OncoSil Medical awarded key U.S. patent, treatment of pancreatic cancer**
http://www.proactiveinvestors.com.au/companies/news/49249/oncosil-medical-awarded-key-us-patent-treatment-of-pancreatic-cancer-49249.html
*OncoSil press release:*
*http://www.oncosil.com.au/pdf/36204813OncoSil18%2010%2013%20US%20Patents%20Granted.pdf*

- Company: OncoSil Medical Limited, Sydney, Australia
- Major finding: OncoSil Medical has been awarded a key U.S. patent covering its OncoSil™ nuclear medical device that is being developed for treatment of locally advanced pancreatic cancer. The company plans to initiate a pivotal study for the device in the first quarter of 2014 that would enroll 100-300 patients. OncoSil™ is an active implantable medical device that provides a localized radiation therapy for tumors, avoiding the systemic side effects of external radiation treatment.

## CANCER CONTROL, SURVIVORSHIP, AND OUTCOMES RESEARCH

**Depression, cytokines, and pancreatic cancer**

http://www.ncbi.nlm.nih.gov/pubmed/24136882

- Journal: *Psycho-Oncology*
- Institution(s): Memorial Sloan Kettering Cancer Center, New York, NY and others
- Corresponding author(s): William Breitbart
- Major finding: This study demonstrated an association between depression and IL-6, but not with other cytokines. Moreover, IL-6 was not significantly associated with other measures of psychological distress (anxiety and hopelessness) or with symptom distress (pain, fatigue, and sleep quality), although some cytokines assayed were associated with specific symptoms. The implications of these findings for the etiology and treatment of depression in pancreatic cancer patients are discussed.

**Treatment of pancreatic cancer: A narrative review of cost-effectiveness studies**

http://www.ncbi.nlm.nih.gov/pubmed/24182608

- Journal: *Best Practice & Research Clinical Gastroenterology*
- Institution(s): CHU Dijon, Dijon Cedex, France
- Corresponding author(s): Isabelle Le Ray
- Major finding: Limitations in healthcare resources, burden of treatment, and uncertainty of the net clinical benefit of adjuvant therapy, underline the need to identify the cost-effectiveness of different therapeutic approaches, as well as a need to establish patient groups who benefit most from these treatments. The present paper reviews cost-effectiveness studies published on pancreatic cancer treatment.

**Healthcare costs, treatment patterns, and resource utilization in a managed care population**

http://www.ncbi.nlm.nih.gov/pubmed/24074258

- Journal: *Journal of Medical Economics*
- Institution(s): OptumInsight, Eden Prairie, MN and others
- Corresponding author(s): Stacey DaCosta Byfield
- Major finding: These results indicate that pancreatic cancer imposes a substantial burden on the US healthcare system, and that treatment of more advanced disease is significantly more costly than initial treatment of non-metastatic disease.

**The quality of supportive cancer care in Veterans Affairs Health System and targets for improvement**

http://www.ncbi.nlm.nih.gov/pubmed/24126685

- Journal: *JAMA Internal Medicine*
- Institution(s): VA Greater Los Angeles Healthcare System, Los Angeles, CA and others
- Corresponding author(s): Karl Lorenz
- Major finding: Using a retrospective cohort study design, the authors measured evidence-based cancer care processes using previously validated indicators of care quality in patients with advanced cancer, addressing pain, nonpain symptoms, and information and care planning among 719 veterans with a 2008 Veterans Affairs Central Cancer Registry diagnosis of stage IV colorectal (37.0%), pancreatic (29.8%), or lung (33.2%) cancer. The authors identified care gaps that reflect important targets for improving the patient and family experience of cancer care.

**SCIENTIFIC MODEL SYSTEMS**

**Pathogenesis of pancreatic cancer: Lessons from animal models**

http://www.ncbi.nlm.nih.gov/pubmed/24178582

- Journal: *Toxicologic Pathology*
- Institution(s): University of Utah, Salt Lake City, UT
- Corresponding author(s): L. Charles Murtaugh
- Major finding: Together, animal models enable diverse approaches to basic and preclinical research on pancreatic cancer, the results of which will accelerate progress against this currently intractable cancer.