



Research

PANCREATIC CANCER ACTION NETWORK

ADVANCE RESEARCH. SUPPORT PATIENTS. CREATE HOPE.

www.pancan.org | 877.272.6226

PANCREATIC CANCER NEWS & UPDATES – FEBRUARY 2013

PANCREATIC CANCER ACTION NETWORK AND COMMUNITY NEWS

AACR Annual Meeting 2013 Online Proceedings and Itinerary Planner

<http://www.abstractsonline.com/plan/start.aspx?mkey={9B2D28E7-24A0-466F-A3C9-07C21F6E9BC9}>

Looking forward to seeing many of you at this year's AACR Annual Meeting in Washington, DC. With the Itinerary Planner, you can search or browse all Annual Meeting presentations by author/speaker, title, topic track, or organ site track and create a personal itinerary for the meeting.

**The Pancreatic Cancer Action Network – AACR Grants Poster Presentation and Discussion Special Session will take place Tuesday, April 9, 10:30 am – 12:30 pm, at Renaissance Washington Hotel, Congressional Hall A. This session is moderated by members of the Pancreatic Cancer Action Network Scientific Advisory Board and spotlights research by 2012 grant recipients.*

The Pancreatic Cancer Action Network welcomes new members to its Medical Advisory Board

http://pancan.org/section_about/news_press_center/2013_press_releases/02_05_13_pr.php

The Pancreatic Cancer Action Network has named four new members to the organization's Medical Advisory Board. The newest advisors are leading clinicians in the field of pancreatic cancer: Joseph M. Herman, MD, MSc, Johns Hopkins University (recipient of the 2008 Blum-Kovler Career Development Award); George A. Fisher Jr., MD, PhD, Stanford University; James Farrell, MD, Yale University; and Philip Agop Philip, MD, PhD, Karmanos Cancer Center.

The Ruth Leff Siegel Award for Excellence in Pancreatic Cancer Research

http://pancreasmd.org/guide_news_siegel.html

The Pancreas Center at Columbia University and The Herbert Irving Comprehensive Cancer Center have been entrusted by the Siegel family to identify the investigator who has made the most significant contribution to the understanding and/or treatment of pancreatic cancer over the past year. The selection committee will notify the successful nominee by June 1, 2013. The awardee will be expected to accept the \$50,000 cash prize at a ceremony attended by the family and friends of Ruth Siegel. If you know of a candidate whom you believe meets the criteria described above, please complete an online form or the downloadable application on or before April 15, 2013.

Levine Cancer Institute, UNC Charlotte announce innovative pancreatic cancer research partnership

<http://inside.uncc.edu/news/item/levine-cancer-institute-unc-charlotte-announce-innovative-pancreatic-cancer-research-partn>

Carolina HealthCare System's Levine Cancer Institute and UNC Charlotte will enter into a joint project to advance translational and clinical research in the field of pancreatic cancer. The collaborative effort, called the Charlotte Pancreatic Cancer Project (CPCP), aims to foster more working relationships between physicians and scientists at both institutions by offering funding for innovative research ideas, submitted to and reviewed by a committee of their peers. Pinku Mukherjee, PhD, recipient of a 2007

Pancreatic Cancer Action Network Pilot Grant is pictured and identified as one of the University's leading scientists studying pancreatic and breast cancers.

At \$3 million, new award gives medical researchers a dose of celebrity

http://www.nytimes.com/2013/02/20/science/new-3-million-prizes-awarded-to-11-in-life-sciences.html?_r=1&

Eleven scientists, most of them American, were scheduled to be named as the first winners of the world's richest academic prize for medicine and biology — \$3 million each. The award, the Breakthrough Prize in Life Sciences, was meant to reward scientists who think big, take risks and have made a significant impact on our lives. One of the recipients is Dr. Bert Vogelstein at Johns Hopkins, whose work will continue to focus on pancreatic cancer in addition to other cancer types.

ASCO 2013 registration and housing information

<http://chicago2013.asco.org/registration-and-housing-information>

Housing reservation and early registration rate deadline: Wednesday, April 24, 2013 at 11:59 PM (EDT).

Explore an interactive history of cancer research advances

http://www.cancerprogress.net/?cmpid=ma_cancprog_gen_etoc_all_02-20-13_cancprog

CancerProgress.Net is developed and curated by the American Society of Clinical Oncology. This site was launched in 2011 to mark the 40th anniversary of the U.S. National Cancer Act, which led to major new investments in cancer research and significant increases in cancer survival. The site is intended to provide a dynamic and interactive history of progress against cancer, expert perspective on remaining challenges and other useful tools.

Now available from ASCO: patientACCESS

http://www.cancer.net/publications-and-resources/patientaccess?cmpid=kh_net_pataccess_etoc_all_02-20-13_ascohtml

The American Society of Clinical Oncology (ASCO) and the Conquer Cancer Foundation are pleased to provide patients being treated for cancer and their caregivers with free access to medical research articles published in ASCO's two journals: *Journal of Clinical Oncology (JCO)* and *Journal of Oncology Practice (JOP)*. This initiative – known as patientACCESS – offers access to full-text *JCO* and *JOP* articles to patients and caregivers for their own use, or to bring to their doctor.

Funding opportunity: Clinical Assay Development Program (CADP)

<http://cadp.cancer.gov/>

The NCI Clinical Assay Development Program (CADP) is requesting project applications from investigators in academia, government and industry seeking clinical assay validation resources. These resources are designed to assist with the development of assays that may predict therapy response or prognostic behavior of a diagnosed cancer, primarily for use in clinical trials. Remaining 2013 application deadlines: June 15 and October 15.

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).

2012 RFA Links and Provocative Questions

<http://provocativequestions.nci.nih.gov/rfa>

The provocative questions (PQ) project is intended to assemble a list of important but non-obvious questions that will stimulate the NCI's research communities to use laboratory, clinical, and population sciences in especially effective and imaginative ways. For the current issuance of the PQ Program, the original list of PQs is now updated to a set of 24 PQs. The new/updated PQs have been divided into four groups, resulting in four R01 FOAs and four R21 FOAs, with LOI deadlines of May 20, 2013.

Pancreas Cancer Research Fellowship at Virginia Mason Cancer Center

<http://jobs.virginiamason.org/job/Seattle-Pancreas-Cancer-Research-Fellowship-Job-WA-98101/1913701/>

Virginia Mason Cancer Center in Seattle is now accepting applications for a Pancreas Cancer Research Fellowship (PCRF) program and hopes to have their first PCRF fellow start on July 1, 2013 (the beginning of the next academic year). Vincent J. Picozzi, Jr., MD (Medical Advisory Board) is the Fellowship Director for this program. More information about the Digestive Disease Institute can be found here: <https://www.virginiamason.org/ddi>.

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.”

BIOLOGY OF CANCER

WNT7B mediates autocrine Wnt/ β -catenin signaling and anchorage-independent growth

<http://www.ncbi.nlm.nih.gov/pubmed/23416978>

- Journal: *Oncogene*
- Institution(s): David Geffen School of Medicine at UCLA, Los Angeles, CA
- Corresponding author(s): David Dawson
- PanCAN affiliated author: Dave Dawson, MD, PhD: 2008 Seena Magowitz – Career Development Award
- Major finding: The authors' findings indicate WNT7B can serve as a primary determinant of differential Wnt/ β -catenin activation in pancreatic ductal adenocarcinoma. Disrupting the interaction between Wnt ligands and their receptors may be a particularly suitable approach for therapeutic modulation of Wnt/ β -catenin signaling in pancreatic cancer and other cancer

contexts where Wnt activation is mediated by ligand expression rather than mutations in canonical pathway members.

Hippo signaling regulates differentiation and maintenance in the exocrine pancreas

<http://www.ncbi.nlm.nih.gov/pubmed/23454691>

- Journal: *Gastroenterology*
- Institution(s): University of Pennsylvania School of Medicine, Philadelphia, PA and others
- Corresponding author(s): Ben Stanger
- PanCAN affiliated authors:
 - Nabeel Bardeesy, PhD: 2008 Randy Pausch, PhD – Pilot Grant
 - Ben Stanger, MD, PhD: 2007 Ralph H. Hruban, MD – Career Development Award
- Major finding: In this study, the authors investigated the role of the core Hippo kinases-Mst1 and Mst2-in pancreatic development and homeostasis. The mammalian Hippo kinases, and YAP, maintain postnatal pancreatic acinar differentiation in mice.

Notch signaling pathway targeted therapy suppresses tumor progression and metastatic spread

<http://www.ncbi.nlm.nih.gov/pubmed/23402814>

- Journal: *Cancer Letters*
- Institution(s): Johns Hopkins School of Medicine, Baltimore, MD and others
- Corresponding author(s): N.V. Rajeshkumar
- PanCAN affiliated authors:
 - Zeshaan Rasheed, MD, PhD: 2010 Tempur-Pedic Retailers – Pathway to Leadership Grant
 - Anirban Maitra, MBBS: 2004 Career Development Award and Chair, Scientific Advisory Board
- Major finding: The authors' preclinical data suggest that PF-03084014, a selective γ -secretase inhibitor, has greater anti-tumor activity in combination with gemcitabine in pancreatic ductal adenocarcinoma and provides rationale for further investigation of this combination in PDA.

PARI overexpression promotes genomic instability and pancreatic tumorigenesis

<http://www.ncbi.nlm.nih.gov/pubmed/23436799>

- Journal: *Cancer Research*
- Institution(s): Dana-Farber Cancer Institute, Boston, MA and others
- Corresponding author(s): George-Lucian Moldovan
- PanCAN affiliated author: Alec Kimmelman, MD, PhD: 2010 Career Development Award
- Major finding: Taken together, the authors' findings offered a preclinical proof-of-concept for the PARP-binding protein C12orf48/PARI as candidate therapeutic target to treat pancreatic ductal adenocarcinoma.

Vitamin E δ -tocotrienol induces p27(Kip1)-dependent cell-cycle arrest, E2F-1-dependent mechanism

<http://www.ncbi.nlm.nih.gov/pubmed/23393547>

- Journal: *PLoS One*
- Institution(s): H. Lee Moffitt Cancer Center and Research Institute, Tampa, FL and others
- Corresponding author(s): Mokenge Malafa
- PanCAN affiliated author: Mo Malafa, MD: Medical Advisory Board

- **Major finding:** The authors' findings reveal a new mechanism, dependent on p27(Kip1) induction, by which δ -tocotrienol can inhibit proliferation in pancreatic ductal cancer cells, providing a new rationale for p27(Kip1) as a biomarker for δ -tocotrienol efficacy in pancreatic cancer prevention and therapy.

Inflammatory networks and immune surveillance of pancreatic carcinoma

<http://www.ncbi.nlm.nih.gov/pubmed/23422836>

- **Journal:** *Current Opinion in Immunology*
- **Institution(s):** University of Pennsylvania, Philadelphia, PA
- **Corresponding author(s):** Robert Vonderheide
- **PanCAN affiliated author:** Bob Vonderheide, MD, DPhil: Scientific Advisory Board
- **Major finding:** Recent studies demonstrate that derailing immune suppressive pathways in the pancreatic ductal adenocarcinoma microenvironment, such as tumor derived GM-CSF, facilitates T-cell mediated tumor rejection. These findings carry major implications for the development of novel, combination immunotherapies for pancreatic cancer.

Evolution and dynamics of pancreatic cancer progression

<http://www.ncbi.nlm.nih.gov/pubmed/23416985>

- **Journal:** *Oncogene*
- **Institution(s):** Johns Hopkins Medical Institutions, Baltimore, MD and others
- **Corresponding author(s):** Christine Iacobuzio-Donahue
- **PanCAN affiliated author:** Christine Iacobuzio-Donahue, MD, PhD: 2007 Pilot Grant and Scientific Advisory Board
- **Major finding:** This review summarizes current knowledge of the genetics of pancreatic cancer from its initiation within a normal cell until the time that it has disseminated to distant organs, many features of which can be extrapolated to other solid tumor types. The implications of these findings to personalize genome analyses of an individual patient's tumor are also discussed.

Aberrant expression of mucin core proteins and O-linked glycans associated with progression

<http://www.ncbi.nlm.nih.gov/pubmed/23446997>

- **Journal:** *Clinical Cancer Research*
- **Institution(s):** University of Nebraska Medical Center, Omaha, NE and others
- **Corresponding author(s):** Michael (Tony) Hollingsworth
- **PanCAN affiliated author:** Tony Hollingsworth, PhD: Scientific Advisory Board
- **Major finding:** There are significant alterations in mucin expression and post-translational processing during progression of pancreatic cancer from early lesions to metastasis. The results are presented in the context of how mucins influence the biology of tumor cells and their microenvironment during progression of pancreatic cancer.

Targeting the NF- κ B and mTOR pathways with a quinoxaline urea analog that inhibits IKK β

<http://www.ncbi.nlm.nih.gov/pubmed/23444213>

- **Journal:** *Clinical Cancer Research*
- **Institution(s):** University of Nebraska Medical Center, Omaha, NE
- **Corresponding author(s):** Amarnath Natarajan

- PanCAN affiliated author: Tony Hollingsworth, PhD: Scientific Advisory Board
- Major finding: A kinome screen identified a quinoxaline urea analog 13-197 as an I κ B kinase β (IKK β) inhibitor. The results suggest that 13-197 targets IKK β and thereby inhibits mTOR and NF- κ B pathways. Oral availability along with in vivo efficacy without obvious toxicities makes this quinoxaline urea chemotype, a viable cancer therapeutic.

Bmi1 enhances tumorigenicity and cancer stem cell function in pancreatic adenocarcinoma

<http://www.ncbi.nlm.nih.gov/pubmed/23437065>

- Journal: *PLoS One*
- Institution(s): University of Michigan Medical School, Ann Arbor, MI
- Corresponding author(s): Diane Simeone
- PanCAN affiliated author: Diane Simeone, MD: 2010 The Randy Pausch Family – Innovative Grant and member, Scientific Advisory Board
- Major finding: The authors' results implicate Bmi1, an integral component of the Polycomb Repressive Complex 1 (PRC1), in the invasiveness and growth of pancreatic cancer and demonstrate its key role in the regulation of pancreatic cancer stem cells.

Outlier kinase expression by RNA sequencing as targets for precision therapy

<http://www.ncbi.nlm.nih.gov/pubmed/23384775>

- Journal: *Cancer Discovery*
- Institution(s): University of Michigan Medical School, Ann Arbor, MI and others
- Corresponding author(s): Chandan Kumar-Sinha
- PanCAN affiliated author: Diane Simeone, MD: 2010 The Randy Pausch Family – Innovative Grant and member, Scientific Advisory Board
- Major finding: The authors analyzed transcriptome sequencing data from a compendium of cancer and benign samples, and defined distinct "outlier kinases" in individual breast and pancreatic cancer samples, based on highest levels of absolute and differential expression. Their results suggest that outlier kinases represent effective precision therapeutic targets that are readily identifiable through RNA sequencing of tumors.

Pancreatic cancer-associated cathepsin E as a drug activator

<http://www.ncbi.nlm.nih.gov/pubmed/23422726>

- Journal: *Journal of Controlled Release*
- Institution(s): MD Anderson Cancer Center, Houston, TX and others
- Corresponding author(s): Ching-Hsuan Tung
- PanCAN affiliated authors:
 - Huamin Wang, MD, PhD: 2007 Skip Viragh – Career Development Award
 - Craig Logsdon, PhD: Scientific Advisory Board
- Major finding: In this study, a novel approach using Cathepsin E (Cath E) activation of a Cath E-specific prodrug was demonstrated. This treatment could result in fewer side effects than the non-specific treatments currently in use. Cath E is a specific and effective drug activator for pancreatic ductal adenocarcinoma treatment.

Selective requirement of PI3K/PDK1 signaling for Kras oncogene-driven pancreatic cell plasticity

<http://www.ncbi.nlm.nih.gov/pubmed/23453624>

- Journal: *Cancer Cell*
- Institution(s): Technische Universität München, München, Germany and others
- Corresponding author(s): Dieter Saur
- Major finding: The authors show that cell-autonomous phosphoinositide 3-kinase (PI3K) and 3-phosphoinositide-dependent protein kinase 1 (PDK1), but not Raf, are key effectors of oncogenic Kras in the pancreas, mediating cell plasticity, acinar-to-ductal metaplasia (ADM), and pancreatic ductal adenocarcinoma (PDAC) formation. These in vivo genetic studies together with pharmacologic treatment studies in models of human acinar-to-ductal metaplasia and pancreatic ductal adenocarcinoma demonstrate tissue-specific differences of oncogenic Kras signaling and define PI3K/PDK1 as a suitable target for therapeutic intervention specifically in PDAC.

Targeting tumor macrophages decreases tumor-initiating cells, relieves immunosuppression

<http://www.ncbi.nlm.nih.gov/pubmed/23221383>

- Journal: *Cancer Research*
- Institution(s): Washington University School of Medicine, St. Louis, MO and others
- Corresponding author(s): David DeNardo
- Major finding: The authors' findings show how targeting tumor-infiltrating macrophages can effectively overcome therapeutic resistance mediated by tumor-initiating cells.

Exploiting inflammation for therapeutic gain in pancreatic cancer

<http://www.ncbi.nlm.nih.gov/pubmed/23385734>

- Journal: *British Journal of Cancer*
- Institution(s): The Beatson Institute for Cancer Research, Glasgow, UK and others
- Corresponding author(s): Christopher Ross Carter
- Major finding: This review explores the pathways known to modulate inflammation at different stages of tumor development, drawing conclusions on their potential as therapeutic targets in pancreatic ductal adenocarcinoma.

Senescence in pancreatic carcinogenesis: from signalling to chromatin remodelling and epigenetics

<http://www.ncbi.nlm.nih.gov/pubmed/23408353>

- Journal: *Gut*
- Institution(s): Philipps University of Marburg, Marburg, Germany
- Corresponding author(s): Volker Ellenrieder
- Major finding: The importance of cellular senescence, a permanent cell growth arrest, is increasingly being recognized as a critical fail-safe program in pancreatic carcinogenesis.

3D pancreatic carcinoma spheroids induce matrix-rich, chemoresistant phenotype, better model

<http://www.ncbi.nlm.nih.gov/pubmed/23446043>

- Journal: *BMC Cancer*
- Institution(s): Karolinska Institutet, Stockholm, Sweden and others
- Corresponding author(s): Matthias Löhr

- **Major finding:** The authors developed a high-throughput 3D cell culture drug screening system for pancreatic cancer, which displays a strongly increased chemoresistance. Features associated to the 3D cell model are increased expression of matrix proteins and miRNA as well as stromal markers such as PPP1R1B and SNED1. This is supporting the concept of cell adhesion mediated drug resistance.

Erlotinib prolongs survival in pancreatic cancer by blocking gemcitabine-induced MAPK signals

<http://www.ncbi.nlm.nih.gov/pubmed/23378339>

- **Journal:** *Cancer Research*
- **Institution(s):** University of Tokyo, Tokyo, Japan and others
- **Corresponding author(s):** Hideaki Ijichi
- **Major finding:** The authors found that gemcitabine induced MAPK signaling, which was dramatically inhibited by erlotinib even in the Kras-activated pancreatic cancer cells in the mouse model. Mechanistic investigations suggested that gemcitabine induces EGFR ligand expression and ERBB2 activation by increasing heterodimer formation with EGFR, thereby maintaining high levels of ERBB2 protein in PDAC cells. Overall, these findings suggest a significant role of ERBB in PDAC treatment.

Heparin-binding epidermal growth factor-like growth factor eliminates constraints on activated Kras

<http://www.ncbi.nlm.nih.gov/pubmed/23376846>

- **Journal:** *Oncogene*
- **Institution(s):** Vanderbilt University Medical Center, Nashville, TN
- **Corresponding author(s):** Anna Means
- **Major finding:** The authors demonstrate that two oncogenic events, mutation of Kras and production of the growth factor heparin-binding epidermal growth factor-like growth factor (HB-EGF), are sufficient for rapid and complete neoplastic transformation of the exocrine pancreas. These findings establish the importance of oncogenic synergy in cancer initiation and promotion, and establish a molecular link between inflammation and the earliest stages of tumor induction.

Inhibition of growth of patient-derived pancreatic cancer xenografts with MEK inhibitor trametinib

<http://www.ncbi.nlm.nih.gov/pubmed/23441129>

- **Journal:** *Neoplasia*
- **Institution(s):** University of Virginia Health System, Charlottesville, VA and others
- **Corresponding author(s):** Todd Bauer
- **Major finding:** These data indicate that inhibition of the EGFR family receptor signaling may contribute to the effectiveness of MEK1/2 inhibition of tumor growth possibly through the inhibition of feedback activation of receptor tyrosine kinases in response to inhibition of the RAS-RAF-MEK-ERK pathway. These studies provide a rationale for assessing the co-inhibition of these pathways in the treatment of pancreatic cancer patients.

Suppression of AKT phosphorylation restores rapamycin-based synthetic lethality in SMAD4-defective

<http://www.ncbi.nlm.nih.gov/pubmed/23443316>

- **Journal:** *Molecular Cancer Research*
- **Institution(s):** The Hunter College of the City University of New York, New York, NY

- **Corresponding author(s):** David Foster
- **Major finding:** This study demonstrates that the synthetic lethality to rapamycin in pancreatic cancers with defective TGF- β signaling is masked by rapamycin-induced increases in Akt phosphorylation. The implication is that a combination of approaches that suppress both Akt phosphorylation and mTOR could be effective in targeting pancreatic cancers with defective TGF- β signaling.

Different patterns of Akt, ERK feedback activation response to rapamycin, mTOR inhibitor, metformin

<http://www.ncbi.nlm.nih.gov/pubmed/23437362>

- **Journal:** *PLoS One*
- **Institution(s):** University of California, Los Angeles, Los Angeles, CA
- **Corresponding author(s):** Enrique Rozengurt
- **Major finding:** The effects of metformin on Akt and ERK activation are strikingly different from allosteric or active-site mTOR inhibitors in pancreatic ductal adenocarcinoma cells, though all these agents potently inhibited the mTORC1/S6K axis.

ETIOLOGY

ABO blood groups and pancreatic cancer risk and survival: Results from the PANDORA consortium

<http://www.ncbi.nlm.nih.gov/pubmed/23403949>

- **Journal:** *Oncology Reports*
- **Institution(s):** German Cancer Research Center, Heidelberg, Germany and others
- **Corresponding author(s):** Federico Canzian
- **Major finding:** In this analysis, carriers of the A1 ABO blood group were indeed at greater risk of developing the disease. In addition, the authors investigated the possible influence that genetic variability at the ABO locus may have in pancreatic cancer survival, but they observed no effect in their population.

The association between self-reported diabetes & cancer incidence in NIH-AARP diet and health study

<http://www.ncbi.nlm.nih.gov/pubmed/23408570>

- **Journal:** *The Journal of Clinical Endocrinology and Metabolism*
- **Institution(s):** National Cancer Institute, Bethesda, MD and others
- **Corresponding author(s):** Gabriel Lai
- **Major finding:** The authors' results suggest an etiological role for diabetes in a number of cancers, including pancreatic, independent of obesity, and that preventing diabetes may contribute to reduced cancer risk.

Cancer risk associated with STK11/LKB1 germline mutations in Peutz-Jeghers syndrome patients

<http://www.ncbi.nlm.nih.gov/pubmed/23415580>

- **Journal:** *Digestive and Liver Disease*
- **Institution(s):** "Aldo Moro" University of Bari, Italy and others
- **Corresponding author(s):** Nicoletta Resta
- **Major finding:** Peutz-Jeghers syndrome entails markedly elevated cancer risks, mainly for pancreatic and cervical cancers. This Italian multicenter study provides a helpful reference for improving current surveillance protocols.

Alcohol consumption on pancreatic diseases

<http://www.ncbi.nlm.nih.gov/pubmed/23429423>

- Journal: *World Journal of Gastroenterology*
- Institution(s): Mayo Clinic, Rochester, MN and others
- Corresponding author(s): Luis Bujanda
- Major finding: The objective of this study was to review the epidemiology described in the literature for pancreatic diseases as a consequence of alcoholic behavior trying to understand the association between dose, type, and frequency of alcohol consumption and risk of pancreatitis and pancreatic cancer. With regard to PC, the role of alcohol consumption remains less clear, and low to moderate alcohol consumption do not appear to be associated with PC risk, and only chronic heavy drinking increase the risk compared with lightly drinkers.

PREVENTION

The molecular mechanism of action of aspirin, curcumin and sulforaphane combinations

<http://www.ncbi.nlm.nih.gov/pubmed/23404329>

- Journal: *Oncology Reports*
- Institution(s): Western University of Health Sciences, Pomona, CA
- Corresponding author(s): Sunil Prabhu
- Major finding: The present study evaluated the chemopreventive potential of a combination of aspirin, curcumin, and sulforaphane in low doses to human pancreatic cancer cells. Data from this study demonstrate that a low-dose ACS combination inhibits cell growth by inducing cell apoptosis, and proposes sustained activation of the ERK1/2 signaling pathway as one of the possible mechanisms.

EARLY DETECTION, DIAGNOSIS, AND PROGNOSIS

CA 19-9 nonproduction is associated with poor survival after resection of pancreatic adenocarcinoma

<http://www.ncbi.nlm.nih.gov/pubmed/23428954>

- Journal: *American Journal of Clinical Oncology*
- Institution(s): Northwestern University Feinberg School of Medicine, Chicago, IL and others
- Corresponding author(s): Mark Talamonti
- PanCAN affiliated author: Mark Talamonti, MD: Medical Advisory Board
- Major finding: CA 19-9 nonproduction is not associated with improved survival after pancreatic cancer resection, as has previously been asserted, when compared with patients with normal and elevated levels.

Prognosis of minimally invasive carcinoma arising in mucinous cystic neoplasms of the pancreas

<http://www.ncbi.nlm.nih.gov/pubmed/23388125>

- Journal: *American Journal of Surgical Pathology*
- Institution(s): The Johns Hopkins University School of Medicine, Baltimore, MD and others
- Corresponding author(s): Ralph Hruban
- PanCAN affiliated authors:
 - Huamin Wang, MD, PhD: 2007 Skip Viragh – Career Development Award
 - Eileen O'Reilly, MD: Medical Advisory Board
 - Ralph Hruban, MD: Emeritus Scientific Advisory Board

- **Major finding:** This study demonstrates that the majority of patients with minimally invasive adenocarcinoma arising in mucinous cystic neoplasms are cured by surgery, particularly if the neoplasms are completely examined histologically.

Is it necessary to follow patients after resection of a benign pancreatic IPMN?

<http://www.ncbi.nlm.nih.gov/pubmed/23395158>

- **Journal:** *Journal of the American College of Surgeons*
- **Institution(s):** Johns Hopkins Medical Institutions, Baltimore, MD
- **Corresponding author(s):** Christopher Wolfgang
- **PanCAN affiliated author:** Ralph Hruban, MD: Emeritus Scientific Advisory Board
- **Major finding:** Patients who have undergone resection for noninvasive intraductal papillary mucinous neoplasm (IPMN) require indefinite close surveillance because of the risks of developing a new IPMN, of requiring surgery, and of developing cancer. A family history of pancreatic cancer, but not margin status or degree of dysplasia, is associated with a risk of development of a new or progressive IPMN.

Classification, morphology and molecular pathology of premalignant lesions of the pancreas

<http://www.ncbi.nlm.nih.gov/pubmed/23442735>

- **Journal:** *Pathology*
- **Institution(s):** Royal Prince Alfred Hospital, Camperdown, Australia and others
- **Corresponding author(s):** James Kench
- **Major finding:** Over the past few years there have been substantial advances in our knowledge of premalignant lesions of the pancreas.

Cyst features and risk of malignancy in intraductal papillary mucinous neoplasms of the pancreas

<http://www.ncbi.nlm.nih.gov/pubmed/23416279>

- **Journal:** *Clinical Gastroenterology and Hepatology*
- **Institution(s):** Kaiser Permanente Los Angeles Medical Center, Los Angeles, CA
- **Corresponding author(s):** Bechien Wu
- **Major finding:** Based on a meta-analysis, cyst features proposed by the international guidelines for resection of intraductal papillary mucinous neoplasms (IPMN) were highly associated with malignancy. However, based on our findings, not all cyst features should be weighted equally when considering risk of malignancy; cyst size >3 cm was most strongly associated with malignant IPMN.

Diagnosis and management of cystic pancreatic lesions

<http://www.ncbi.nlm.nih.gov/pubmed/23345356>

- **Journal:** *American Journal of Roentgenology*
- **Institution(s):** Massachusetts General Hospital, Harvard Medical School, Boston, MA and others
- **Corresponding author(s):** Carlos Fernandez-del Castillo
- **Major finding:** The purpose of this review is to outline the management guidelines for the care of patients with cystic pancreatic lesions.

European experts consensus statement on cystic tumours of the pancreas

<http://www.ncbi.nlm.nih.gov/pubmed/23415799>

- Journal: *Digestive and Liver Disease*
- Institution(s): Karolinska Institutet at Karolinska University Hospital, Stockholm, Sweden and others
- Corresponding author(s): Marco Del Chiaro
- Major finding: European expert pancreatologists provide updated recommendations: diagnostic computerized tomography and/or magnetic resonance imaging are indicated in all patients with cystic lesion of the pancreas. Resection should be considered in all symptomatic lesions, in mucinous cystic neoplasm, main duct intraductal papillary mucinous neoplasm and solid pseudo-papillary neoplasm as well as in branch duct intraductal papillary mucinous neoplasm with mural nodules, dilated main pancreatic duct >6mm and possibly if rapidly increasing in size.

Combined serum CA19-9 and miR-27a-3p in peripheral blood mononuclear cells to diagnose

<http://www.ncbi.nlm.nih.gov/pubmed/23430754>

- Journal: *Cancer Prevention Research*
- Institution(s): Fudan University, Shanghai, China and others
- Corresponding author(s): Xiao-Lin Wang
- Major finding: A panel combining serum CA19-9 and peripheral blood mononuclear cell (PBMC) miR-27a-3p level could have considerable clinical value in diagnosing pancreatic cancer.

Circulating tumor cells in pancreatic cancer patients: Methods of detection and clinical implications

<http://www.ncbi.nlm.nih.gov/pubmed/23447365>

- Journal: *International Journal of Cancer*
- Institution(s): Stavanger University Hospital, Norway
- Corresponding author(s): Kjersti Tjensvoll
- Major finding: This review highlights various enrichment procedures and methods for the detection of circulating tumor cells (CTCs). Furthermore, the authors systematically review previously reported studies of the clinical relevance of CTC detection in pancreatic cancer patients. Larger studies, as well as characterization of the CTC population, are required to achieve further insight into the clinical implications of CTC detection in pancreatic cancer patients.

Circulating molecular markers in pancreatic cancer: ready for clinical use?

<http://www.ncbi.nlm.nih.gov/pubmed/23414463>

- Journal: *Future Oncology*
- Institution(s): Imperial College, Hammersmith Hospital Campus, London, UK
- Corresponding author(s): Long Jiao
- Major finding: Issues remain surrounding the specificity and sensitivity of CA 19-9 as a pancreatic cancer biomarker, which have led to increased attention in novel blood biomarkers for pancreatic ductal adenocarcinoma, including circulating tumor cells (CTCs), circulating cell-free DNA (cfDNA), and circulating miRNAs.

Whole blood interferon- γ levels predict the therapeutic effects of adoptive T-cell therapy

<http://www.ncbi.nlm.nih.gov/pubmed/23420507>

- Journal: *International Journal of Cancer*
- Institution(s): Kyoto Prefectural University of Medicine, Kyoto, Japan
- Corresponding author(s): Satoshi Kokura
- Major finding: The findings of this study indicate that the assay of whole blood IFN- γ production offers promise for evaluating the clinical response of patients to cancer immunotherapy.

Immune cell infiltration as an indicator of the immune microenvironment of pancreatic cancer

<http://www.ncbi.nlm.nih.gov/pubmed/23385730>

- Journal: *British Journal of Cancer*
- Institution(s): National Cancer Center Research Institute, Tokyo, Japan
- Corresponding author(s): Nobuyoshi Hiraoka
- Major finding: Tumor-infiltrating CD4T/CD8T/%Treg and %M1/M2 are independent prognosticators useful for evaluating the immune microenvironment of pancreatic ductal carcinoma.

Arginase II expressed in cancer-associated fibroblasts indicates tissue hypoxia, predicts poor outcome

<http://www.ncbi.nlm.nih.gov/pubmed/23424623>

- Journal: *PLoS One*
- Institution(s): National Cancer Center Research Institute, Tokyo, Japan and others
- Corresponding author(s): Nobuyoshi Hiraoka
- Major finding: These results indicate that cancer cell-mediated immune suppression through arginase II (ARG2) expression is not a general event and that the presence of ARG2-expressing cancer-associated fibroblasts is an indicator of poor prognosis, as well as hypoxia, in pancreatic ductal carcinoma tissue.

Loss of TRAIL-receptors is a recurrent feature in pancreatic cancer and determines the prognosis

<http://www.ncbi.nlm.nih.gov/pubmed/23460812>

- Journal: *PLoS One*
- Institution(s): University of Munich, Munich, Germany
- Corresponding author(s): Enrico De Toni
- Major finding: This is a first report on the prognostic significance of TRAIL-receptors expression in pancreatic cancer showing that TRAIL-R2 might represent a prognostic marker for patients with early stage disease. In addition, the authors' data suggest that loss of membrane-bound TRAIL-receptors could represent a molecular mechanism for therapeutic failure upon administration of TRAIL-receptors-targeting antibodies in pancreatic cancer. This hypothesis should be evaluated in future clinical trials.

KRAS mutation status is not predictive for objective response to anti-EGFR treatment with erlotinib

<http://www.ncbi.nlm.nih.gov/pubmed/23435671>

- Journal: *Journal of Gastroenterology*
- Institution(s): Ludwig-Maximilians-University of Munich, Munich, Germany
- Corresponding author(s): Stefan Boeck

- **Major finding:** AIO-PK0104 was a phase III trial comparing gemcitabine/erlotinib followed by capecitabine with capecitabine/erlotinib followed by gemcitabine in advanced pancreatic cancer. This post hoc analysis of AIO-PK0104 supports the assumption that KRAS is rather a prognostic than a predictive biomarker in pancreatic cancer.

MTA2 expression is a novel prognostic marker for pancreatic ductal adenocarcinoma

<http://www.ncbi.nlm.nih.gov/pubmed/23400716>

- **Journal:** *Tumor Biology*
- **Institution(s):** The PLA 117 Hospital, Hangzhou, China and others
- **Corresponding author(s):** Xiao-Xiao Jiang
- **Major finding:** This study suggests that overexpression of MTA2 may play an important role in the progression of pancreatic ductal adenocarcinoma and MTA2 expression may serve as a biomarker for poor prognosis for PDA.

TREATMENT

Borderline resectable: need for standardization and methods for optimal clinical trial design

<http://www.ncbi.nlm.nih.gov/pubmed/23435609>

- **Journal:** *Annals of Surgical Oncology*
- **Institution(s):** University of Texas MD Anderson Cancer Center, Houston, TX and many others
- **Corresponding author(s):** Matthew Katz
- **PanCAN-affiliated authors:**
 - Joseph Herman, MD: 2008 Blum-Kovler – Career Development Award and member, Medical Advisory Board
 - Eric Collisson, MD: 2012 Skip Viragh – Career Development Award
 - Philip Philip, MD: Medical Advisory Board
 - Jordan Berlin, MD: Chair, Medical Advisory Board
- **Major finding:** Rigorous standards of clinical trial design incorporated into trials of other disease stages must be adopted in all future studies of borderline resectable pancreatic cancer. The Intergroup trial should serve as a paradigm for such investigations.

Global, multicenter, randomized, phase II trial of gemcitabine and gemcitabine plus AGS-1C4D4

<http://www.ncbi.nlm.nih.gov/pubmed/23448807>

- **Journal:** *Annals of Oncology*
- **Institution(s):** Dana-Farber Cancer Institute, Boston, MA and many others
- **Corresponding author(s):** Brian Wolpin
- **PanCAN-affiliated author:** Eileen O'Reilly, MD: Medical Advisory Board
- **Major finding:** This randomized, phase II study achieved its primary end point, demonstrating an improved 6-month survival rate with addition of AGS-1C4D4, a fully human monoclonal antibody to prostate stem cell antigen (PSCA), to gemcitabine among patients with previously untreated, metastatic pancreatic adenocarcinoma.

Phase II randomised proof-of-concept study of the urokinase inhibitor upamostat (WX-671)

<http://www.ncbi.nlm.nih.gov/pubmed/23412098>

- **Journal:** *British Journal of Cancer*
- **Institution(s):** Ludwig-Maximilians-University of Munich, Munich, Germany and others

- **Corresponding author(s):** Volker Heinemann
- **Major finding:** This prospective multicenter study evaluated the efficacy and tolerability of the urokinase plasminogen activator (uPA) inhibitor upamostat in combination with gemcitabine in locally advanced pancreatic adenocarcinoma (LAPC). In this proof-of-concept study targeting the uPA system in LAPC, the addition of upamostat to gemcitabine was tolerated well; similar survival results were observed for the three treatment arms.

Phase II study of gemcitabine in combination with regional arterial infusion of nafamostat mesilate

<http://www.ncbi.nlm.nih.gov/pubmed/22157216>

- **Journal:** *American Journal of Clinical Oncology*
- **Institution(s):** The Jikei University School of Medicine, Tokyo, Japan
- **Corresponding author(s):** Tadashi Uwagawa
- **Major finding:** The purpose of this phase II study was to evaluate the efficacy of regional arterial infusion of the synthetic serine protease inhibitor nafamostat mesilate combined with gemcitabine for the treatment of patients with unresectable locally advanced or metastatic pancreatic cancer. The regimen was found to be an alternative regimen for unresectable pancreatic cancer, especially for metastatic pancreatic cancer based on acceptable survival time, clinical benefit, and cost advantage.

Pharmacological ascorbate with gemcitabine for metastatic and node-positive pancreatic cancer

<http://www.ncbi.nlm.nih.gov/pubmed/23381814>

- **Journal:** *Cancer Chemotherapy and Pharmacology*
- **Institution(s):** The University of Iowa Carver College of Medicine, Iowa City, IA
- **Corresponding author(s):** Joseph Cullen
- **Major finding:** A phase I clinical trial was performed to establish safety and tolerability of pharmacological ascorbate combined with gemcitabine in patients with biopsy-proven stage IV pancreatic adenocarcinoma. Data suggest pharmacologic ascorbate administered concurrently with gemcitabine is well tolerated. Initial data from this small sampling suggest some efficacy.

Phase II trial of erlotinib plus capecitabine as first-line treatment for metastatic pancreatic cancer

<http://www.ncbi.nlm.nih.gov/pubmed/23393373>

- **Journal:** *Anticancer Research*
- **Institution(s):** University Hospital Complex of Santiago, Santiago de Compostela, Spain and others
- **Corresponding author(s):** Rafael López
- **Major finding:** The combination of capecitabine with erlotinib is an active regimen with a favorable safety profile for patients with metastatic pancreatic cancer.

Disrupting cytokine signaling in pancreatic cancer: A phase I/II study of etanercept with gemcitabine

<http://www.ncbi.nlm.nih.gov/pubmed/23429495>

- **Journal:** *Pancreas*
- **Institution(s):** Ohio State University, Columbus, OH
- **Corresponding author(s):** Tanios Bekaii-Saab
- **Major finding:** Etanercept blocks tumor necrosis factor α (TNF- α), a proinflammatory cytokine that plays a role in cancer-related cachexia and tumor growth. A phase I/II study was conducted to assess the tolerability and efficacy of gemcitabine and etanercept in advanced pancreatic

cancer. Etanercept added to gemcitabine is safe but did not show significant enhancement of gemcitabine in patients with advanced pancreatic cancer.

Restoration of mannose-binding lectin complement activity is associated with improved outcome

<http://www.ncbi.nlm.nih.gov/pubmed/23423329>

- **Journal:** *Journal of Parenteral and Enteral Nutrition*
- **Institution(s):** Leicester General Hospital, Leicester, UK
- **Corresponding author(s):** Ali Arshad
- **Major finding:** Mannose-binding lectin (MBL) restoration had an association with improved outcome in the cohort of patients with low MBL activity at baseline. The independent contribution of ω -3 fatty acids to this effect warrants further investigation in the form of randomized clinical trials.

Comparative effectiveness of minimally invasive and open distal pancreatectomy

<http://www.ncbi.nlm.nih.gov/pubmed/23426503>

- **Journal:** *JAMA Surgery*
- **Institution(s):** Beth Israel Deaconess Medical Center, Boston, MA and others
- **Corresponding author(s):** A. James Moser
- **Major finding:** The authors detected no evidence that minimally invasive distal pancreatectomy was inferior to open distal pancreatectomy based on postoperative outcomes or overall survival. This conclusion was verified by propensity score analysis with adjustment for factors affecting selection of operative technique.

Pancreatic surgery in the very old: face to face with a challenge of the near future

<http://www.ncbi.nlm.nih.gov/pubmed/23435699>

- **Journal:** *World Journal of Surgery*
- **Institution(s):** Ruhr University of Bochum, Bochum, Germany
- **Corresponding author(s):** Orlin Belyaev
- **Major finding:** Despite high mortality and morbidity rates, surgery remains the only chance for cure in most octogenarians with pancreatic disease. Careful patient selection is the key to success and improved long-term survival in this group, which will represent a substantial fraction of the population in the near future.

Personalizing pancreatic cancer medicine

http://www.medscape.com/viewarticle/776794?nlid=28300_484&src=wnl_edit_medp_honc&spon=7&pa=oDtMoIkcoU6OH092GIF26khJdke9cB1L9lmccLY3bTSSA15v%2BQib%2BBHdoBc3wTy8Sivl8zjYv73GUYW5rsbWA%3D%3D

This *Medscape Oncology News* article summarizes the basic molecular biology of pancreatic tumors and the current state of pancreatic cancer treatment, as well as targeted treatments in the pipeline that might enable future personalized pancreatic cancer treatment and prediction of response to treatment. It also discusses possible directions for screening patients at high risk of developing the disease, detecting tumors at earlier stages, and increasing patient involvement in designing treatment.

Everolimus in advanced pancreatic neuroendocrine tumors: the clinical experience

<http://www.ncbi.nlm.nih.gov/pubmed/23436795>

- Journal: *Cancer Research*
- Institution(s): The University of Texas MD Anderson Cancer Center, Houston, TX and others
- Corresponding author(s): James Yao
- Major finding: The authors review the clinical studies showing the efficacy of the oral mTor inhibitor everolimus in pancreatic neuroendocrine tumors (pNET) and summarize the translational science from these studies.

PI3K/Akt/mTOR pathway inhibitors in the therapy of pancreatic neuroendocrine tumors

<http://www.ncbi.nlm.nih.gov/pubmed/23419523>

- Journal: *Cancer Letters*
- Institution(s): Cedars-Sinai Medical Center, Los Angeles, CA
- Corresponding author(s): Edward Wolin
- Major finding: The phosphatidylinositol 3-kinase (PI3K)/Akt/mammalian target of rapamycin (mTOR) pathway is implicated in the pathogenesis of pancreatic neuroendocrine tumors (pNETs). Clinical studies are underway evaluating individual node and dual node inhibitors.

Systemic therapy for advanced pancreatic neuroendocrine tumors

<http://www.ncbi.nlm.nih.gov/pubmed/23391115>

- Journal: *Seminars in Oncology*
- Institution(s): Dana-Farber/Brigham and Women's Cancer Center, Boston, MA
- Corresponding author(s): Matthew Kulke
- Major finding: It has become increasingly evident that pancreatic neuroendocrine tumors (NETs) tend to respond differently to therapeutic agents than do other NET subtypes. In most cases, systemic therapy has been more effective in NETs of pancreatic origin than in NETs arising from other locations.

Peregrine Pharmaceuticals announces results from Phase II clinical trial of bavituximab

<http://ir.peregrineinc.com/releasedetail.cfm?ReleaseID=740017>

- Company: Peregrine Pharmaceuticals, Tustin, CA
- Major finding: Peregrine Pharmaceuticals announced results from its 70 patient open-label, randomized Phase II clinical trial of bavituximab (phosphatidylserine-targeting monoclonal antibody) used in combination with gemcitabine in patients with previously untreated, advanced Stage IV pancreatic cancer. Results showed that the combination of bavituximab and gemcitabine resulted in more than a doubling of overall response rates and an improvement in overall survival when compared with gemcitabine alone.

VAXIMM reports positive topline data from first oral cancer vaccine trial

http://www.vaximm.com/stuff/downloads/130207_Vaximm-positive-results-final.pdf

- Company: VAXIMM AG, Basel, Switzerland and Mannheim, Germany
- Major finding: VAXIMM AG, a Swiss-German biotech company focusing on oral cancer vaccines, announced topline data from the first clinical trial of its investigational oral cancer vaccine VXMO1, a therapeutic vaccine targeting the tumor vasculature (VEGFR2). The randomized,

placebo-controlled, double-blind Phase I/II dose escalation study met all key endpoints and demonstrated safety and tolerability.

Nuvilex reports cannabinoid-based pancreatic cancer treatments to be developed by its subsidiary

http://www.nuvilex.com/news/preview.php?id=260&cat_id=6&p=#ontitle

- **Company:** Nuvilex, Inc., Silver Spring, MD
- **Major finding:** Nuvilex, Inc., international biotechnology and clinical stage provider of natural products and cell and gene therapy solutions for the treatment of diseases, announced that its subsidiary, Medical Marijuana Sciences, Inc., is planning to develop treatments for pancreatic cancer based on cannabinoids from *Cannabis sativa*.

Clinical trial looks to improve pancreatic cancer survival rates

<http://www.ncbi.nlm.nih.gov/pubmed/23408570>

- **Institution:** Georgia Regents University Cancer Center, Augusta, GA
- **Major finding:** Researchers at Georgia Regents University Cancer Center are investigating a new avenue of treatment to help boost poor pancreatic cancer survival rates. The treatment combines Gemcitabine with a monoclonal antibody called CT-011 (targets PD1 and its related proteins) in certain pancreatic cancer patients who have been treated with surgery.

CANCER CONTROL, SURVIVORSHIP, AND OUTCOMES RESEARCH

The cost-effectiveness of neoadjuvant chemoradiation is superior to a surgery-first approach

<http://www.ncbi.nlm.nih.gov/pubmed/23397153>

- **Journal:** *Annals of Surgical Oncology*
- **Institution(s):** University of Cincinnati School of Medicine, Cincinnati, OH
- **Corresponding author(s):** Jason Fleming
- **PanCAN affiliated authors:**
 - Chris Crane, MD: Medical Advisory Board
 - Jason Fleming, MD: Medical Advisory Board
- **Major finding:** Neoadjuvant chemoradiation for pancreatic cancer identifies patients with early metastases or poor performance status, who can be spared an ineffective or prohibitively morbid operation, and is associated with improved survival at significantly lower cost than a surgery-first approach. Neoadjuvant chemoradiation followed by surgery is a strategy that provides more cost-effective care than a surgery-first approach.

Frequency and intensity of postoperative surveillance after curative treatment of pancreatic cancer

<http://www.ncbi.nlm.nih.gov/pubmed/23408126>

- **Journal:** *Annals of Surgical Oncology*
- **Institution(s):** The University of Texas MD Anderson Cancer Center, Houston, TX
- **Corresponding author(s):** Ching-Wei Tzeng
- **PanCAN affiliated author:** Jason Fleming, MD: Medical Advisory Board
- **Major finding:** Increasing the frequency and intensity of postoperative surveillance of patients after curative therapy for pancreatic cancer beyond clinical evaluation and CA 19-9 testing every 6 months increases cost but confers no clinically significant survival benefit.

Pancreatic cancer and supportive care-pancreatic exocrine insufficiency negatively impacts quality of life

<http://www.ncbi.nlm.nih.gov/pubmed/23397095>

- Journal: *Supportive Care in Cancer*
- Institution(s): University of Sydney, Camperdown, Australia
- Corresponding author(s): Helen Gooden
- Major finding: Participants expressed distress relating to the effects of pancreatic exocrine insufficiency. Pancreatic enzyme supplement therapy with clear dosage guidelines and associated dietary advice could resolve symptoms of malabsorption and markedly improve quality of life. For people affected by pancreatic cancer, this is an essential supportive care.

Effects of psycho-oncologic interventions on emotional distress and quality of life

<http://www.ncbi.nlm.nih.gov/pubmed/23319686>

- Journal: *Journal of Clinical Oncology*
- Institution(s): Tumor Biology Center, University of Freiburg, Freiburg, Germany
- Corresponding author(s): Hermann Faller
- Major finding: This systematic review and meta-analysis aimed to evaluate the effects of psycho-oncologic interventions on emotional distress and quality of life in adult patients with cancer. The authors conclude that various types of psycho-oncologic interventions are associated with significant, small-to-medium effects on emotional distress and quality of life. These results should be interpreted with caution, however, because of the low quality of reporting in many of the trials.

Cancer statistics for African Americans, 2013

<http://www.ncbi.nlm.nih.gov/pubmed/23386565>

- Journal: *CA: A Cancer Journal for Clinicians*
- Institution(s): American Cancer Society, Atlanta, GA
- Corresponding author(s): Carol DeSantis
- Major finding: Overall, progress in reducing cancer death rates has been made, although more can and should be done to accelerate this progress through ensuring equitable access to cancer prevention, early detection, and state-of-the-art treatments.