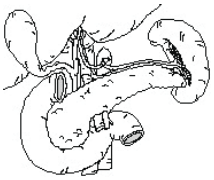

Welcome to the 44th Annual Meeting of the Pancreas Club. The Mission of the Pancreas Club, since its founding in 1966, is to promote the interchange of ideas between pancreatologists throughout the world and to maintain an informal "club" atmosphere.

This year the Pancreas Club received over 110 abstracts which were reviewed by the Program Committee. We know that you will be fully engaged in both listening to the excellent presentations and in the discussions which follow. Poster authors will be available posterside during the two Poster Sessions.

This meeting will offer continuing medical education credits through a joint sponsorship with the American College of Surgeons. We thank them for their support of this important meeting. We hope this provides a benefit to your CME needs and appreciate you support of this meeting

The abstracts selected for oral and poster presentation are included in this program book and are available on our website.

The Pancreas Club is pleased to announce two awards for 2010 which will be presented at the Sunday evening dinner.

PanCan Research Award. $1,000 for the best oral presentation of clinical or basic science pancreatic cancer research by a resident or fellow. This award is funded by the Pancreatic Cancer Action Network

Kenneth Warren Research Award. $1,000 for the best oral presentation of clinical or basic science pancreatitis or pancreatic cancer research by a resident or fellow. This award is funded by the Pancreas Club and the Kenneth Warren Foundation

Pancreas Club Directors

Bill Traverso

Bill Nealon

Doug Evans

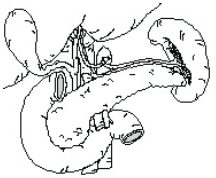

Table of Contents

44th Annual Pancreas Club Meeting

Location

Pan American Life Conference and Media Center
601 Poydras Street, 11th Floor
New Orleans, LA 70130

Antoine's Restaurant
713 Rue Saint Louis
New Orleans, LA 70130

Registration: Pan American Life Conference and Media Center
Saturday, May 1 12 noon – 5:00 pm
Sunday, May 2 7:00 am – 3:00 pm

Scientific Sessions: Pan American Life Auditorium
Saturday, May 1 1:00 – 5:00 pm
Sunday, May 2 8:00 am – 5:00 pm

Dinner
7:00 pm – 10:00 pm - Antoine's Restaurant

Program Committee Members

William Nealon, Chair	Sharona Ross	Jennifer Tseng
Hwayda Arafat	Kay Reid-Lombardo	Casper van Eijck
Gerard Aranha	Sara Thayer	Santhi Swaroop Vege
Hein Gooszen	William Traverso	Chris Wolfgang

Continuing Medical Education

Meeting Objectives:

- Elucidate the current clinical and basic science research in pancreatic cancer and pancreatitis
- Discuss the results of clinical trials in pancreatology
- Discuss laparoscopic pancreatic resection and open pancreatic resection
- Understand new diagnostic and therapeutic modalities for diseases of the pancreas and be able to make a decision regarding their use in professional practice

American College of Surgeons
Division of Education

Accreditation Statement:
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the American College of Surgeons and the Pancreas Club. The American College Surgeons is accredited by the ACCME to provide continuing medical education for physicians.

AMA PRA Category 1 Credits™
The American College of Surgeons designates this educational activity for a maximum of 9.25 *AMA PRA Category 1 Credits™.* Physicians should only claim credit commensurate with the extent of their participation in the activity.

Disclosure information found on page 4

Program At a Glance

Saturday, May 1, 2010

12:00 pm—5:00 pm	Registration & Exhibitor Displays
1:00 pm—2:45 pm	Scientific Session 1
3:00 pm—4:00 pm	Professor Rounds w/ Posters
4:00 pm—5:00 pm	Scientific Session 2
5:00 pm—6:00 pm	Welcome Reception & Poster viewing

Sunday, May 2, 2010

7:00 am—5:00 pm	Registration
7:00 am—3:00 pm	Exhibitor Displays
7:00 am—7:45 am	Continental Breakfast
8:00 am—9:45 am	Scientific Session 3
10:00 am—11:00 am	Scientific Session 4
11:00 am—12:00 pm	Professor Rounds w/ Posters
12:00 pm—1:00 pm	Lunch
1:00 pm—2:45 pm	Scientific Session 5
3:00 pm – 3:45 pm	How I Do It Session
3:45 pm – 5:00 pm	Scientific Session 6
5:00 pm – 5:15 pm	Business Meeting
7:00 pm—10:00 pm	Cocktails and Dinner Antoine's Restaurant
	Abstract Award Presentations Dinner Honoree Presentation

2010 Dinner Honoree

Hans G. Beger

Hans Gunter Beger is internationally recognized as a true pioneer in pancreatic surgery. He has been one of the most innovative and influential pancreatic surgeons in this era and he is credited, in many ways with carrying a torch to follow to a new generation of practices and strategies, particularly relating to the management of acute necrotizing pancreatitis and chronic pancreatitis. His contributions to pancreatic diseases and surgery are recognized worldwide. From the University of Ulm he designed and implemented the duodenum preserving pancreatic head resection for chronic pancreatitis (the Beger Procedure) and gathered a database which after more than 30 years had accrued the largest single experience in this disease with superb and thorough data and follow-up. At a time when severe acute pancreatitis was often a fatal disease and was further a disease for which surgery was discouraged as a catastrophic error Hans Beger established the significance of defining the presence of necrosis and he advocated "necrosectomy" to manage operatively either infected necrosis or selected patient with sterile necrosis. For 20 years he was Chairman and Head of the Department of General Surgery, University of Ulm while authoring over 750 articles and training many surgeons who have become department chairs through Germany. For years Hans held a course, open to all surgeons throughout Europe, in which didactic sessions were combined with actual demonstrations of complex pancreatic procedures in actual operating room settings. He has organized so many international congresses on pancreatic diseases that he has touched most of us involved with these diseases – either personally or by facilitating opportunities for individuals to meet and network with other pancreatic surgeons. Each of us owe Hans Beger a great debt of gratitude.

Disclosure Information

In compliance with ACCME regulations, the American College of Surgeons, as the accredited provider of this activity, must ensure that anyone in a position to control the content of the educational activity has disclosed all relevant financial relationships with any commercial interest. All reported conflicts are managed by a designated official to ensure a bias-free presentation. Please see the insert to this program for the complete disclosure list.

The requirement for disclosure is not intended to imply any impropriety of such relationships, but simply to identify such relationships through full disclosure, and to allow the audience to form its own judgments regarding the presentation.

Thank you

The Pancreas Club gratefully acknowledges support for the 44th Annual Pancreas Club meeting from the following:

Educational Grant in support of the meeting

New Link Genetics
WL Gore & Associates

Resident award support

Kenneth Warren Foundation
Pancreatic Cancer Action Network

Exhibitors

New Link Genetics
Solvay Pharmaceuticals, Inc.
Boston Scientific Endoscopy
Alnara Pharmaceuticals, Inc
Karl Storz Endoscopy

Information as available at the time of printing.

Program Schedule

Saturday, May 1, 2010

12:55 pm **Welcome and Introductory Remarks**
Doug Evans,Froedtert & Medical College of Wisconsin
William H. Nealon, Vanderbilt University
William Traverso,Virginia Mason Clinic

SESSION I
Neo-adjuvant vs. Adjuvant Therapy and Other Controversies
Clinical and Basic Science
1:00 pm – 3:00 pm

Moderators: William Traverso, Seattle and Roberto Coppola, Rome

1:00 pm **DOWNSTAGING CHEMOTHERAPY (DCTX) MAY ALTER THE CLASSIC CT/MRI SIGNS OF VASCULAR INVOLVEMENT IN PATIENTS WITH PANCREATICOBILIARY CANCERS. THIS SHOULD INFLUENCE PATIENT SELECTION FOR SURGERY**
Timothy R. Donahue MD1, O. Joe Hines MD1, James S. Tomlinson MD PhD2,5, James J. Farrell MD3, Yasser M. Bhat MD3, William H. Isacoff MD4, Edward Garon MD4, Barbara Clerkin NP MPH1, Howard A. Reber MD1
Departments of Surgery, [1]Division of General Surgery and [2]Surgical Oncology, and Medicine, [3]Division of Digestive Diseases and [4]Hematology and Oncology, David Geffen School of Medicine at University of California Los Angeles, Los Angeles, CA (UCLA), and [5]Greater Los Angeles Veterans Healthcare System, Los Angeles, CA

1:15 pm **SIGNIFICANCE OF PATHOLOGIC RESPONSE TO PREOPERATIVE THERAPY IN PANCREATIC CANCER**
Y. S. Chun[1], H. S. Cooper[2], J. C. Watson[1], P. B. Rao[1], J. P. Hoffman[1]
[1]Department of Surgical Oncology; [2]Department of Pathology, Fox Chase Cancer Center, Philadelphia, PA

1:30 pm **PATTERNS AND PREDICTORS OF FAILURE AFTER CURATIVE RESECTIONS OF PANCREATIC ENDOCRINE CARCINOMA**
Massimo Falconi, Letizia Boninsegna[1], Rossella Bettini[1], Francesco Panzuto[2], Stefano Partelli, Paola Capelli†, Aldo Scarpa†, Gianfranco Dalle Fave[2], Claudio Bassi, Paolo Pederzoli.
[1]Department of Surgery, "S.Cuore-Don Calabria" Hospital, Negrar, [2]Department of Gastroenterology, Sant'Andrea Hospital, Rome, Departments of Surgery, Chirurgia "B" and †Pathology , "G.B. Rossi" Hospital, University of Verona (Italy).

1:45 pm **CT STAGING SYSTEM FOR PANCREATIC CANCER**
C. J. Clark[1], D. L. Coy[2], L. W. Traverso[1]
[1]Department of General Surgery, Virginia Mason Medical Center, Seattle, WA; [2] Department of Radiology, Virginia Mason Medical Center, Seattle, WA

2:00 pm **DOES NEOADJUVANT THERAPY IMPROVE SURVIVAL IN PATIENTS WITH RESECTABLE PANCREATIC CANCER?**
K.T. Papalezova, D.S. Tyler, T.N. Pappas, D.G. Blazer III, B.M. Clary, R.R. White
Department of Surgery, Duke University, Durham, NC

2:15 pm **MOLECULAR MECHANISMS UNDERLYING THE SYNERGISTIC INTERACTION OF THE NOVEL ANTICANCER DRUG UKRAIN WITH GEMCITABINE IN PRECLINICAL MODELS OF PANCREATIC CANCER**
Niccola Funel[1], Elisa Giovannetti[2], Wassil Nowicky[3], Luca Emanuele Pollina[4], Marco Del Chiaro[1], Franco Mosca[1], G.J. Peters[2], Daniela Campani[5], Ugo Boggi[1]
[1]Division of General and Transplantation Surgery, University of Pisa and Pisa University Hospital, Italy ; [2]VU University Medical Center, Amsterdam, The Netherlands; [3]Nowicky Pharma, Vienna, Austria; [4]Department of Medicine Laboratory and Molecular Diagnoses, Hospital-University of Pisa, Italy; [5]Department of Surgery, University of Pisa, Italy

2:30 pm **EFFICACY OF ADJUVANT VERSUS NEOADJUVANT THERAPY FOR RESECTABLE PANCREATIC ADENOCARCINOMA: A DECISION ANALYSIS**
Hiromichi Ito, Dan Ruan, Edward E. Whang
Department of Surgery, Brigham and Women's Hospital, Boston, MA

2:45 pm **Break**

3:00 – 3:50pm **POSTER ROUNDS VIEWING AND POSTER-SIDE PROFESSOR ROUNDS**
Moderator: Michael Farnell, Rochester, MN
Invited Professors: Keita Wada, Tokyo and Atilla Nakeeb, Indianapolis
Posters of Note: 1-5 and 31-35

Authors will be by their posters to discuss their research poster presentations. Abstracts of note are identified in the program and will be part of the *Poster-side Professor Rounds*. Each invited Professor will discuss several posters. Short oral presentations which have accompanying posters will be identified on the poster board with a red dot.

SESSION II
Technologies – Clinical and Basic Science
4:00 – 5:00 PM

Moderators: Keith Lillemoe, Indianapolis and Casper van Eijck, Rotterdam

4:00 pm **PRELIMINARY DATA ON SURVIVAL AFTER RADIOFREQUENCY ABLATION OF STAGE III PANCREATIC CANCER: A WIND OF CHANGE?**
Girelli R., Frigerio I., Giardino A., Partelli S., Crippa S., Armatura G., Bacchion M., Salvia R., Butturini G. and Bassi C.
Surgical Department – HPB Unit, Pederzoli Clinic – University of Verona, Verona – Italy

4:15 pm **FEASIBILITY AND SAFETY OF ROBOTIC PANCREATECTOMIES: ANALYSIS OF TWENTY-NINE CONSECUTIVE OPERATIONS**
M. Del Chiaro1, C. Moretto1, S. D'Imporzano1, N. De Lio1, V.G. Perrone1, S. Signori1, C. Croce1, F. Vistoli1, U. Boggi1.
1Pisa University Hospital – Division of General and Transplant Surgery – Pisa, Italy

4:30 pm Short Oral **ROBOT-ASSISTED MAJOR PANCREATIC RESECTIONS: A RETROSPECTIVE ANALYSIS OF 30 CONSECUTIVE PATIENTS**
Zeh HJ III, Zureikat AH, Plate JF, Bartlett DL, Moser AJ.
Division of Surgical Oncology, University of Pittsburgh School of Medicine and Cancer Institute, Pittsburgh, PA.

4:35 pm Short Oral **PERIOPERATIVE OUTCOMES FOR OPEN DISTAL PANCREATECTOMY: CURRENT BENCHMARKS FOR COMPARISON?**
W.H. Tseng, D.Muilenburg, S.L. Chen, S.R. Martinez, R.J. Canter, and R.J. Bold, *Division of Surgical Oncology, University of California, DavisCancer Center, Sacramento, California*

4:40 pm **A NOVEL EXPLANT CULTURE SYSTEM FOR THE IN VITRO STUDY OF MURINE PANCREATIC INTRAEPITHELIAL NEOPLASIA (PANIN)**
S. Karhadkar, M. Rovira, S.D. Leach, C.L. Wolfgang, *Department of Surgery, The Sol Goldman Pancreatic Cancer Research Center, The JohnsHopkins Medical Institutions, Baltimore, MD.*

4:55 pm
Short
Oral

PREOPERATIVE CT MEASUREMENT OF PANCREATIC STEATOSIS AND VISCERAL FAT; PROGNOSTIC MARKERS FOR DISSEMINATION AND LETHALITY OF PANCREATIC ADENOCARCINOMA

A. Mathur[1], J. Hernandez[1], F. Shaheen[2], M. Shroff [3], S. Dahal[1], C. Morton[1], R. Kedar[2], A. Rosemurgy[1]
[1]Department of Surgery, University of South Florida;
[2]Department of Radiology, University of South Florida;
[3]University of South Florida

5:00pm **Welcome Reception**

Sunday, May 2, 2010

7:45 am **Welcome and Introductory Remarks**
Doug Evans, Froedtert & Medical College of Wisconsin
William H. Nealon, Vanderbilt University
William Traverso, Virginia Mason Clinic

SESSION III
Cancer Translational Studies –Basic Science
8:00 am – 9:45 am

Moderators: Charles J. Yeo, Philadelphia and Masao Tanaka, Fukuoka

8:00 am **A TRANSLATIONAL CLINICAL STUDY OF A PANCREATIC CANCER VACCINE AS NEOADJUVANT TREATMENT AND ITS EFFECT ON THE TUMOR MICROENVIRONMENT**
K.M.Bever[1], B.H.Edil[2], C.Judkins[1], A.Yager[1], R.Sharma[3], T.Nguyen[1], R.A.
Anders[3], E.Lutz[1], G.Mo[1], H.Xu[1], L.Chen[1], E.Sugar[5], K.Olino[2], R.H.Hruban[3],
J.Herman[4], D.Le[1], C. L. Wolfgang[2], J. L. Cameron[2], R. D. Schulick[2],
D.Laheru[1], E.M.Jaffee[1], L.Zheng[1].
Depts of [1]Oncology, [2]Surgery, [3]Pathology, [4]Rad Oncology, Johns Hopkins University School of Medicine and [5]School of Public Health, Baltimore, MD

8:15 am **CLINICAL IMPLICATIONS OF THE STATUS OF MAJOR FOUR GENES IN PANCREATIC CANCER**
ANALYSES OF MUTATIONS AND EXPRESSION OF THE KRAS, TP53, P16, AND SMAD4 GENES IN AUTOPSY CASES
S. Yachida[1], C. White[1], R. Patrascu[1], Y. Naito[1], H. Abe[1], B. Fu[1], R. H. Hruban[1,2], J. L. Cameron[3], C. J. Yeo[4], T. M. Pawlik[3], B. H. Edil[3], R. D. Schulick[3], C. L. Wolfgang[3], C. A. Iacobuzio Donahue[1,2,3]
Departments of [1]Pathology, [2]Oncology and [3]Surgery, The Sol Goldman Pancreatic Cancer Research Center, Johns Hopkins Medical Institutions, Baltimore, MD; [4]Department of Surgery and the Jefferson Pancreas, Biliary and Related Cancer Center, Thomas Jefferson University, Philadelphia, PA

8:30 am **MICRORNA-21 FROM BENCH TO BEDSIDE AND BACK: A POTENTIAL MARKER OF CLINICAL OUTCOME AND A TARGET TO OVERCOME RESISTANCE TO GEMCITABINE IN PANCREATIC CANCER**

E. Giovannetti,[1,2] N. Funel,[3,4] M. Del Chiaro,[3] L.A. Erozenci,[2] E. Vasile,[5] L.G. Leon,[2] L.E. Pollina,[6] A. Falcone,[5,7] R. Danesi,[1] D. Campani,[4] G.J. Peters,[2] H.M. Verheul,[2] U. Boggi[3]

[1]Dipartimento di Medicina Interna, Università di Pisa, Pisa, Italy; [2]VU University Medical Center, Amsterdam, The Netherlands; [3]U.O. Chirurgia Generale e Trapianti nell'Uremico e nel Diabetico, Azienda Ospedaliera-Universitaria Pisana; Pisa, Italy; [4]U.O. Anatomia Patologica 3, Azienda Ospedaliera-Universitaria Pisana; Pisa, Italy; [5]U.O. Oncologia 2 Universitaria, Azienda Ospedaliera-Universitaria Pisana; Polo Oncologico Area Vasta Nord-Ovest, Istituto Toscano Tumori; Pisa, Italy; [6]Dipartimento di Medicina di laboratorio e diagnosi molecolari, Ospedale-Università di Pisa; [7]Dipartimento di Oncologia, dei Trapianti e delle Nuove Tecnologie in Medicina, Università di Pisa, Pisa, Italy

8:45 am **OVEREXPRESSION OF EPIDERMAL GROWTH FACTOR RECEPTOR (EGFR) DETECTED BY ANTIBODY BINDING EGFR INTERNAL DOMAIN PREDICTS POOR SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA**

Jessica Kline[1], Mary McDonald[1], Charles J Yeo[2], Jonathan R Brody[2], Agnieszka K Witkiewicz[1]

[1]Pathology, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, United States; [2]Surgery, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania

9:00 am **HUR STATUS IS A POWERFUL CLINICAL MARKER FOR RESECTED PANCREATIC DUCTAL ADENOCARCINOMA PATIENTS AND CAN BIND TO VEGF AND HIF-1ALPHA MRNA**

Nathan G. Richards[1], Agnes K. Witkiewicz[2], Christina L. Costantino[1], Dane R. Grenda[1], David W. Rittenhouse[1], Eugene P. Kennedy[1], Charles J Yeo[1], Jonathan R Brody[1]

[1]Surgery, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, United States; [2]Pathology, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

9:15 am **DPC4 STATUS IS CORRELATED WITH TUBULAR MORPHOLOGY OF INVASIVE CARCINOMA ASSOCIATED WITH INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM OF THE PANCREAS, BUT NOT WITH LYMPH NODE STATUS**

Yoshiki Naito[1], John L. Cameron[3], Barish H. Edil[3], Richard D. Schulick[3], Christopher L. Wolfgang[3], Christine A Iacobuzio-Donahue [1, 2, 3]

Departments of Pathology[1], Oncology[2] and Surgery[3], The Sol Goldman Pancreatic Cancer Research Center, Johns Hopkins Medical Institutions, Baltimore, MD, USA

9:30 am Short Oral **REPRESSION OF E-CADHERIN BY THE POLYCOMB GROUP PROTEIN EZH2 IN PANCREATIC CANCER**
J Kline[1], C Kleer[2], CJ Yeo[3], JR Brody[3] and AK Witkiewicz[1].
[1]Pathology, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, United States; [3]Surgery, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, and [2]Pathology, University of Michigan, Ann Arbor, Michigan, United States

9:35 am Short Oral **INTRADUCTAL MUCINOUS PAPILLARY NEOPLASMS: GENETIC CHARACTERIZATION OF LESION PROGRESSION**
R. P. Jury[1], T. J. Geddes[2], L. E. Fortier[2], M. A. Farinola[3], B. L. Pruetz[2], G. D. Wilson[4]
[1] Department of General Surgery, [2]Beaumont BioBank, [3] Department of Anatomic Pathology, [4] Department of Radiation Oncology, William Beaumont Hospital, Royal Oak, Michigan, USA

9:40 am Short Oral **LOSS OF HETEROZYGOSITY (LOH) STATUS OF D9S105 MARKER IS ASSOCIATED WITH DOWN-REGULATION OF KRUPPEL-LIKE FACTOR 4 (KLF4) EXPRESSION IN PANCREATIC DUCTAL ADENOCARCINOMA AND PANINS**
Niccola Funel[1], Mariangela Morelli[2], Elisa Giovannetti[3], Luca Emanuele Pollina[4], Marco Del Chiaro[1], Generoso Bevilacqua[2], Franco Mosca[1], Andrea Cavazzana[2], Daniela Campani[5], Ugo Boggi[1]
[1]Division of General and Transplantation Surgery, University of Pisa and Pisa University Hospital, Italy; [2]Department of Oncology, Division o f Surgical, Molecular and Ultrastructural Patholoy University of Pisa and Pisa University Hospital, Italy; [3]VU University Medical Center, Amsterdam, The Netherlands; [4]Department of Medicine Laboratory and Molecular Diagnoses, Hospital-University of Pisa, Italy [5]Department of Surgery, University of Pisa, Italy

9:45 am **Break. Visit with exhibitors and view the posters**

SESSION IV
Outcomes
10:00 am – 11:00 am

Moderators: Gerard Aranha, Chicago and Hein Gooszen, Utrecht

10:00 am **PREOPERATIVE FACTORS PREDICT MORBIDITY AFTER PANCREATICODUODENECTOMY: CREATION OF A NSQIP NOMOGRAM**
D.Y. Greenblatt, E. Winslow, V. Rajamanickam, Y. Wan, R. Rettammel, C.S. Cho, S.M. Weber
Department of Surgery, University of Wisconsin, Madison, Wisconsin

10:15 am **PANCREATECTOMY RISK CALCULATOR: AN ACS-NSQIP RESOURCE**
Parikh, Purvi, Shiloach, Mira, Cohen, Mark E, Bilimoria, Karl Y, Ko, Clifford Y, Hall, Bruce L, Pitt, Henry A

10:30 am Short Oral

BRAIN NATRIURETIC PEPTIDE (BNP) AND POSTOPERATIVE FLUID BALANCE IN THE MANAGEMENT OF PATIENTS UNDERGOING PANCREATECTOMY

Berri RN, Lin H, Folloder J , Pisters PWT, Abdalla EK, Vauthey JN, Lee JE, and Fleming JB

10:35 am

DIFFERENCES IN METHYLATION OF CELL-FREE CIRCULATING DNA IN PATIENTS WITH PANCREATIC CANCER AND CHRONIC PANCREATITIS

Thomas Liggett[1], Anatoliy Melnikov[2], Qi-long Yi[3], Charles Replogle[3], Randall Brand[4], Karen Kaul[5], Mark Talamonti[6], Ross A. Abrams[2], Victor Levenson[2]

[1] Department of Neurological Sciences, and [2]Department of Radiation Oncology, Rush University Medical Center, Chicago, IL 60612; [3]ScienceDocs, Inc, Portland, OR 97225;

[4]Division of Gastroenterology, Hepatology & Nutrition, University of Pittsburgh Medical Center, Pittsburgh, PA 15232; [5]Department of Pathology and Laboratory Medicine, NorthShore University HealthSystem, Evanston, IL 60201; [6]Department of Surgery, NorthShore University HealthSystem, Evanston IL 60201

10:50 am Short Oral

THE BURDEN OF INFECTION FOR ELECTIVE PANCREATIC RESECTIONS

TS Kent[1], S Gautam[2], MP Callery[1], CM Vollmer[1]

Beth Israel Deaconess Medical Center/Harvard Medical School, Department of Surgery, Boston, MA, USA

10:55 am Short Oral

SUPPORT FOR A POSTRESECTION PROGNOSTIC SCORE FOR PANCREATIC ENDOCRINE TUMORS

M.G. Hurtuk[1], A. Godambe[2], M. Shoup[1], S. Yong[2], G.V. Aranha[1]

[1] Department of Surgery, Division of Surgical Oncology, Loyola University Medical Center, Maywood, IL

[2] Department of Pathology, Loyola University Medical Center, Maywood, IL

11:00 am

POSTER VIEWING AND POSTER-SIDE PROFESSOR ROUNDS

Moderator: Sean Mulvihill, Salt Lake City

Invited Professors: Santhi Vege, Rochester, MN and Jennifer Tseng, Worcester

Posters of Note: 1-5 & 31-35

Authors will be by their posters to discuss their research poster presentations. Abstracts of note are identified in the program and will be part of the *Poster-side Professor Rounds*. Each invited Professor will discuss several posters. Short oral presentations which have accompanying posters will be identified on the poster board with a red dot.

12:00 pm

LUNCH

Highlight Beverlee Anderson

Recognition of exhibitors and supporters:
William Traverso

SESSION V
Cancer – Basic Science
1:00 pm – 2:45 pm

Moderators: Hwyda Arafat, Philadelphia and Robert Grutzmann, Dresden

1:00 pm **ADIPOCYTES IN THE TUMOR MICROENVIRONMENT PROMOTE DISSEMINATION OF HUMAN PANCREATIC CANCER**

P.B White[1], J-H. Chen[2], N. J. Zyromski[1], A. Mathur[1], K. D. Lillemoe[1], H.A. Pitt[1]
1. Department of Surgery, Indiana University School of Medicine, Indianapolis, IN, US. 2. Department of Pathology and Laboratory Medicine, Indiana University School of Medicine, Indianapolis, IN, US.

1:15 pm **LOW DOSE METRONOMIC GEMCITABINE HAS HIGH ANTIMETASTATIC EFFICACY IN AN ORTHOTOPIC MOUSE MODEL OF PANCREATIC CANCER**

H.S. Tran Cao[1], M. Bouvet[1,2], S. Kaushal[2], A. Keleman[3], E. Romney[3], S. Dalal[3], D.K. Imagawa[3], R.M. Hoffman[1,4], M.H.G. Katz[3]
[1] Department of Surgery, University of California San Diego, San Diego, CA
[2] Moores Cancer Center, UCSD, San Diego, CA [3] Department of Surgery, University of California Irvine, Orange, CA [4] AntiCancer Inc., San Diego, CA

1:30 pm **TUMOR SUPPRESSOR, ANP32A, DISRUPTS HUR'S REGULATION OF DEOXYCYTIDINE KINASE IN PANCREATIC CANCER: IMPLICATIONS FOR GEMCITABINE THERAPY**

Agnieszka Witkiewicz[1,2,3] Timothy K. Williams[1], Christina L. Costantino[1], Nathan G. Richards[1], Nikolai A. Bildzukewicz[1], Lisa Einstein[1], Joseph Cozzitorto[1], Christine Hostetter[2], Judith C. Keen[2], Abhijit Dasgupta[3], Charles J. Yeo[1], Jonathan R. Brody[1,2,3]
[1]Department of Surgery, [2]Jefferson Center for Pancreatic, Biliary and Related Cancers, Department of Pathology, [3]Kimmel Cancer Center, Thomas Jefferson University, Philadelphia, PA Virginia Mason Medical Center, Seattle, WA

1:45 pm **INDUCTION OF MONOCYTE CHEMOATTRACTANT PROTEIN-1 BY NICOTINE IN PANCREATIC DUCTAL ADENOCARCINOMA CELLS: ROLE OF OSTEOPONTIN**

Melissa Lazar[1], Jennifer Sullivan[1], Galina Chipitsyna[1], Tamer Aziz, Ahmed F Salem, Qiaoke Gong[1], Agnes Witkiewicz[2], David T. Denhardt[3], Charles J. Yeo[1], Hwyda A. Arafat[1,2]
[1]Departments of Surgery, Jefferson Pancreatic, Biliary and Related Cancer Center, [2]Pathology Anatomy and Cell Biology, Thomas Jefferson University, Philadelphia, PA, and [3]Department of Cell Biology and Neuroscience, Rutgers University, New Brunswick, NJ

2:00 pm **A MOLECULAR LINK BETWEEN EPITHELIAL-MESENCHYMAL TRANSITION AND CANCER STEM CELL PROPERTIES IN PANCREATIC CANCER**
Wellner U., Schubert J., Burk U. C., Hopt U.T., Brabletz T., Keck, T., *University Hospital Freiburg, Frieburg, Germany*

2:15 pm **ADIPOCYTES PROMOTE PANCREATIC CANCER PROLIFERATION VIA A HEPATOCYTE GROWTH FACTOR-MEDIATED MECHANISM**
Kathryn M Dalbec, MD[1], Robert V Considine[2], PhD, Eben True, BS[1], Sue Wang, MD[1], Deborah A Swartz-Basile, PhD[1], Henry A Pitt, MD[1], Nicholas J Zyromski, MD[1]
[1]*Department of General Surgery, Indiana University*
[2]*Division of Endocrinology, Department of Medicine, Indiana University*

2:30 pm Short Oral **DEREGULATION OF THE RB/E2F PATHWAY AND P16 EXPRESSION IN PANCREATIC ADENOCARCINOMA**
Jonathan M Hernandez MD*, Abul Elahi MD^, Stephen Brantley MD*, Connor Morton BS*, Leighann Humphries*, M. AnneTimmel*, Sharona Ross MD*, Suresh C. Jhanwar PhD#, Alexander S. Rosemurgy MD
University of South Florida Department of Surgery,^Moffitt Cancer Center and Research Institute, #Memorial Sloan-Kettering Cancer Center

2:35 pm Short Oral **A NOVEL MURINE MODEL FOR THE STUDY OF METASTATIC PANCREATIC ADENOCARCINOMA**
Kelly Olino[1], Kiyoshi Yoshimura[1,2], Elizabeth Jaffee[2], Kelly Foley[2], Ashley Leubner[2], Xiaoyu Pan[2], Drew Pardoll[2], Richard Schulick[1,2], Lei Zheng[1], Barish Edil[1,2]
[1] Department of Surgery and [2] Immunology and Hematopoiesis Division, Department of Medical Oncology, Sidney Kimmel Cancer Center, Johns Hopkins Medical Institutions, Baltimore, Maryland

2:40 pm Short Oral **BLOOD PRESSURE LOWERING MEDICATIONS DISRUPT FATTY ACID METABOLISM IN PANCREATIC CANCER**
Maheshwaran Sivarajah[1], SuhYueh Lim[1], Galina Chipitsyna[1], Qiaoke Gong[1], Tamer Aziz[1], Agnes Witkiewicz[2], Charles J. Yeo[1], Hwyda A. Arafat[1,2]
[1]Departments of Surgery, Jefferson Pancreatic, Biliary and Related Cancer Center; [2]Pathology Anatomy and Cell Biology, Thomas Jefferson University, Philadelphia, PA

2:45 pm **Break: Visit with exhibitors and view the posters**

How I Do It Session

Adjuvant Therapy for Resected Pancreas Cancer –

Is there a Role for Radiation Therapy?

3:00 pm – 3:45 pm

Moderator: Howard Reber, Los Angeles and Markus W. Büchler, Heidelberg

Presentors: Yes – Douglas Evans, Milwaukee

No – John Neoptolemos, Liverpool

This session will be available after the meeting on our website.

SESSION VI
Pancreatitis
3:45 pm – 5:00 pm

Moderators: William Nealon, Nashville and Hans Beger, Ulm

3:45 pm **RANDOMIZED TRIAL COMPARING EUS AND SURGERY FOR PANCREATIC PSEUDOCYST DRAINAGE**
Varadarajulu S[1], Trevino JM[1], Wilcox CM[1], Sutton B[2], Christein JD[3]
University of Alabama at Birmingham Division of Gastroenterology[1], School of Public Health[2], Department of Surgery[3]

4:00 pm **DOES INCREASING INSURANCE IMPROVE OUTCOMES FOR U.S. PANCREATIC CANCER PATIENTS?**
J. K. Smith, J. E. Carroll, S. C. Ng, S. A. Shah, T. P. McDade, J. F. Tseng
University of Massachusetts Medical School, Surgical Outcomes Analysis & Research, Worcester, MA

4:15 pm **AUTO-ISLET TRANSPLANTATION FOR CHRONIC PANCREATITIS IN DIABETIC PATIENTS: WHY BOTHER?**
Katherine A. Morgan, David B. Adams
Medical University of South Carolina, Charleston, SC

4:30 pm **ABDOMINAL COMPARTMENT SYNDROME: AN EARLY LETHAL COMPLICATION OF ACUTE PANCREATITIS**
B. Boone[1], A. Zureikat[1], J. Breaux[1], S. J. Hughes[1], A. J. Moser[1], H. J. Zeh[1], and K. K.W. Lee[1]
Departments of Surgery[1] and Anesthesia[2], University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

4:45 pm **LIVE ANIMAL MOLECULAR IMAGING OF PROTEASE ACTIVITY IN ACUTE PANCREATITIS**

V. Lyo[1], F. Cattaruzza[2], T. Kim[1], J. Cloyd[1], D. Cox[1], A. Walker[2], J. Buxbaum[3], K. Bagatelos[3], M. Paulick[4], M. Bogyo[4], J. Ostroff[3], E. Grady[2], N. W. Bunnett[2], K. S. Kirkwood[2]

[1] School of Medicine, University of California San Francisco, San Francisco, CA; [2] Dept of Surgery, University of California San Francisco, San Francisco, CA; [3] Dept of Gastroenterology, University of California San Francisco, San Francisco, CA [4] Dept of Pathology Stanford University, Stanford, CA.

5:00 pm **Pancreas Club Business Meeting and Planning for 2011**

7:00pm **Pancreas Club Dinner at Antoine's Restaurant**

Dinner Honoree: Hans Beger, MD

Plus:

- *Presentation of two $1,000 resident/fellow awards*

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Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
1 Saturday Poster of Note	IMPACT OF PREOPERATIVE HYPERGLYCEMIA ON MORBIDITY AFTER PANCREATICODUODENECTOMY	Spencer Liles *University of Alabama at Birmingham*
2 Saturday Poster of Note	PROGNOSTIC IMPACT OF DIHYDROPYRIMIDINE DEHYDROGENASE EXPRESSION IN ADJUVANT GEMCITABINE PLUS S-1 CHEMOTHERAPY AFTER SURGICAL RESECTION FOR PANCREATIC ADENOCARCINOMA	Naru Kondo *Department of Surgery, Division of Clinical Medical Science, Graduate School of Biomedical Sciences, Hiroshima University,*
3 Saturday Poster of Note	SURVIVIN EXPRESSION IN CARCINOGENESIS OF INTRADUTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS	Akira Nakashima *Department of Surgery, Graduate School of Biomedical Sciences, Hiroshima University*
4 Saturday Poster of Note	TUMOR LOCATION DOES NOT EFFECT SURVIVAL OF PATIENTS UNDERGOING RESECTION FOR PANCREATIC ADENOCARCINOMA DESPITE LARGER DISTAL PANCREATIC TUMORS	Paul Toomey *University of South Florida and Tampa General Hospital Digestive Disorders Center*
5 Saturday Poster of Note	PP INFUSION IN TYPE 1 DIABETIC PATIENTS (T1DM) REDUCES INSULIN REQUIRMENTS: A PILOT STUDY	Atoosa Rabiee *Johns Hopkins University School of Medicine*
6 Saturday	PERIOPERATIVE OUTCOMES FOR OPEN DISTAL PANCREATECTOMY: CURRENT BENCHMARKS FOR COMPARISON?	Warren Tseng** *University of California, Davis*
7 Saturday	PREOPERATIVE CT MEASUREMENT OF PANCREATIC STEATOSIS AND VISCERAL FAT; PROGNOSTIC MARKERS FOR DISSEMINATION AND LETHALITY OF PANCREATIC ADENOCARCINOMA	Abhishek Mathur** *University of South Florida*
8 Saturday	PERIOPERATIVE AND LONG TERM OUTCOME AFTER MULTIVISCERAL RESECTION FOR PANCREATIC CANCER	Tobias Keck *Department of Surgery, University of Freiburg*
9 Saturday	PERIOPERATIVE BLOOD TRANSFUSION AND OPERATIVE TIME ARE QUALITY INDICATORS FOR PANCREATICODUODENECTOMY	Chad Ball *Indiana University*

Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
10 Saturday	ROBOT-ASSISTED MAJOR PANCREATIC RESECTIONS: A Retrospective ANALYSIS OF 30 CONSECUTIVE PATIENTS.	Amer Zureikat** *University of Pittsburgh Medical Center*
11 Saturday	FIRST 162 WHIPPLES: DISSECTION OF THE LEARNING CURVE	William Fisher *Baylor College of Medicine*
12 Saturday	OBESITY INCREASES COMPLICATIONS FOLLOWING WHIPPLE	Courtney Ballentine *Baylor College of Medicine*
13 Saturday	A NEW STANDARDIZE TECHNIQUE FOR PANCREATICOJEJUNOSTOMY CAN AVOID PANCREATIC FISTULA WITHOUT PANCREATIC DUCT STENTING	Yoshinori Azumi *MIe university Graduate School of Medicine*
14 Saturday	QUALITY OF LIFE OUTCOMES AFTER THE WHIPPLE PROCEDURE FOR PARADUODENAL PANCREATITIS	Elliot Tapper *Beth Israel Deaconess Medical Center*
15 Saturday	CHARACTERIZATION OF THE UNDEREXPRESSED GENE MGC45438 IN PANCREATIC CANCER CELL LINES	Carolin Zimmermann *Department of visceral, thoracic and vascular Surgery, University Hospital Dresden, Germany*
16 Saturday	LAPAROSCOPIC DISTAL PANCREATECTOMY: AN ASSESSMENT OF ONCOLOGIC PRINCIPLES	Eric Jensen *University of Minnesota*
17 Saturday	FACTORS INFLUENCING SURVIVAL IN PATIENTS UNDERGOING A PALLIATIVE BYPASS PROCEDURE FOR PANCREATIC ADENOCARCINOMA	Joseph Herma *Johns Hopkins*
18 Saturday	ROUTINE USE OF A VESSEL SEALING SYSTEM DURING PANCREATICODUODENECTOMY REDUCES INTRAOPERATIVE BLOOD LOSS AND OPERATIVE TIME.	Keita Wada *Teikyo University School of Medicine, Japan*
19 Saturday	PREDICTIVE FACTORS OF MALIGNANCY IN PATIENTS AFFECTED BY INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS	Courtney Ballentine *Baylor College of Medicine* Sergio Valeri *General Surgery, University of Campus Bio-Medico, Rome*
20 Saturday	IS THERE A LEARNING CURVE FOR PANCREATICODUODENECTOMY AFTER FELLOWSHIP TRAINING?	Jeffrey Hardacre *University Hospitals Case Medical Center*
21 Saturday	MULTIDISCIPLINARY EVALUATION FOR PANCREATIC CANCER IN AN ACADEMIC COMPREHENSIVE CANCER CENTER	Amer Zureikat *3550 Terrace St*

Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
22 Saturday	THE ROLE OF CHEMORADIATION IN THE MANAGEMENT OF LOCALLY ADVANCED PANCREATIC ENDOCRINE NEOPLASMS	Trevor Ellison *The Johns Hopkins Hospital*
23 Saturday	DIAGNOSTIC LAPAROSCOPY FOR PANCREATIC CANCER IN AN MRI DRIVEN PRACTICE: WHAT'S IT WORTH?	Elliot Tapper *Beth Israel Deaconess Medical Center*
24 Saturday	TIMING OF CHOLECYSTECTOMY FOR MILD BILIARY PANCREATITIS: A SYSTEMATIC REVIEW	Mark van Baal *University Medical Center Utrecht*
25 Saturday	FLUORESCENCE LAPAROSCOPY AS A NOVEL TECHNIQUE FOR PANCREATIC TUMOR DETECTION IN AN ORTHOTOPIC MOUSE MODEL	Hop Tran Cao *University of California, San Diego*
26 Saturday	PANCREATIC SURGERY: EVOLUTION AT A HIGH-VOLUME CENTER	Kathryn Dalbec *Indiana University*
27 Saturday	PATIENTS DELAY AND ETIOLOGY IN THE DEVELOPMENT OF SEVERE PANCREATITIS. A RETROSPECTIVE STUDY FROM A REFERRAL HOSPITAL	Jonas Dale *Department of Surgery, Haukeland University Hospital*
28 Saturday	DELAYED GASTRIC EMPTYING AFTER PYLORUS-PRESERVING PANCREATICODUODENECTOMY. VALIDATION OF ISGPS CLASSIFICATION AND ANALYSIS OF RISK FACTORS	Stefano Partelli *General Surgery B, G.B. Rossi Hospital*
29 Saturday	ADJUVANT CHEMORADIATION FOR ADENOCARCINOMA OF THE BODY AND TAIL OF THE PANCREAS: THE JOHNS HOPKINS EXPERIENCE	Joseph Herman *Johns Hopkins University*
30 Saturday	PREOPERATIVE MECHANICAL BOWEL PREPARATION DOES NOT OFFER A BENEFIT FOR PATIENTS UNDERGOING PANCREATICODUODENECTOMY	Harish Lavu *Thomas Jefferson University*
31 Saturday Poster of Note	CHARACTERISATION OF A NEWLY ESTABLISHED PATIENT-DERIVED PANCREATIC ADENOCARCINOMA XENOGRAFT COLLECTION	Tobias Keck *University of Freiburg, Department of Visceral Surgery*
32 Saturday Poster of Note	INTRAVENOUS DELIVERY OF THE PLASMA FRACTION OF STORED PACKED RED BLOOD CELLS PROMOTES PANCREATIC CANCER GROWTH IN IMMUNOCOMPETENT MICE	Carlton Barnett *University of Colorado at Denver, Denver Health*

Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
33 Saturday Poster of Note	HYPERINSULINEMIC HYPOGLYCEMIA AFTER ROUX-EN-Y GASTRIC BYPASS: WHAT DRIVES THE PANCREAS?	Atoosa Rabiee *Johns Hopkins University School of Medicine*
34 Saturday Poster of Note	PANCREATIC CANCER CELLS STIMULATE STROMAL CELLS TO PRODUCE PINCH	Jill Shea *University of Utah*
35 Saturday Poster of Note	POSTOPERATIVE ACUTE PANCREATITIS AFTER PANCREATICODUODENRCTOMY IS ASSOCIATED WITH POSTOPERATIVE PANCREATIC FISTULA	Kenichiro Uemura *Hiroshima University Hospital*
36 Saturday	REPRESSION OF E-CADHERIN BY THE POLYCOMB GROUP PROTEIN EZH2 IN PANCREATIC CANCER	Jessica Kline** *Thomas Jefferson University Hospital*
1 Sunday Poster of Note	TOTAL PANCREATECTOMY: INDICATIONS, DIFFERENT TIMING, PERIOPERATIVE AND LONG-TERM OUTCOMES	Stefano Crippa *Department of Surgery University of Verona*
2 Sunday Poster of Note	THE NOVEL SIGMA-2 RECEPTOR LIGAND SW43 INDUCES APOPTOSIS IN PANCREAS CANCER	John Hornick *Washington University at St Louis School of Medicine*
3 Sunday Poster of Note	GENE EXPRESSION PROFILING FOR PREDICTION OF PROGNOSIS IN PATIENTS WITH PANCREATIC DUCTAL ADENOCARCINOMA	Robert Grützmann *University hospital Dresden, Department of Surgery*
4 Sunday Poster of Note	NATURAL HISTORY OF ASYMPTOMATIC PANCREATIC CYSTIC NEOPLASMS	Gareth Morris-Stiff *Cleveland Clinic Foundation*
5 Sunday Poster of Note	LOWER INCOME IMPACTS CARE FOR PANCREATIC CANCER	James Carroll *University of Massachusetts Medical School, Surgical Outcomes Analysis & Research*
6 Sunday	BRAIN NATRIURETIC PEPTIDE (BNP) AND POSTOPERATIVE FLUID BALANCE IN THE MANAGEMENT OF PATIENTS UNDERGOING PANCREATECTOMY	Richard Berri** *MD Anderson Cancer Center*

Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
7 Sunday	LOSS OF HETEROZYGOSITY (LOH) STATUS OF D9S105 MARKER IS ASSOCIATED WITH DOWN-REGULATION OF KRUPPEL-LIKE FACTOR 4 (KLF4) EXPRESSION IN PANCREATIC DUCTAL ADENOCARCINOMA AND PANINS	Niccola Funel** *Division of General and Transplantation Surgery, University of Pisa and Pisa University Hospital, Italy;*
8 Sunday	DEREGULATION OF THE RB/E2F PATHWAY AND P16 EXPRESSION IN PANCREATIC ADENOCARCINOMA	Jonathan Hernandez** *University of South Florida*
9 Sunday	THE BURDEN OF INFECTION FOR ELECTIVE PANCREATIC RESECTIONS	Tara Kent** *Beth Israel Deaconess Medical Center*
10 Sunday	A NOVEL MURINE MODEL FOR THE STUDY OF METASTATIC PANCREATIC ADENOCARCINOMA	Kelly Olino** *The Johns Hopkins Medical Institutions*
11 Sunday	SUPPORT FOR A POSTRESECTION PROGNOSTIC SCORE FOR PANCREATIC ENDOCRINE TUMORS	Michael Hurtuk** *Loyola University Medical Center: Department of Surgery; Division of Surgical Oncology*
12 Sunday	HUMAN PANCREATIC JUICE ENZYME ACTIVITY PREDICTS DISEASE SEVERITY IN CHRONIC PANCREATITIS	Victoria Lyo *UCSF*
13 Sunday	GIANT SPLENIC ARTERY PSEUDOANEURYSM: CASE REPORT AND REVIEW OF THE LITERATURE	Ross Goldberg *Thomas Jefferson University*
14 Sunday	CENTRAL PANCREATECTOMY WITH INFRAMESOCOLIC INTRAPERITONEAL PANCREATOJEJUNOSTOMY	Simone D'imporzano *U.O. Chirurgia Generale e Trapianti nell'Uremico e Diabetico*
15 Sunday	BLOOD PRESSURE LOWERING MEDICATIONS DISRUPT FATTY ACID METABOLISM IN PANCREATIC CANCER	Hwyda Arafat** *Thomas Jefferson University*
16 Sunday	PRIMARY PREVENTION OF ACUTE PANCREATITIS DURING EXTENDED DURATION SPACEFLIGHT: SHOULD THE GALLBLADDER BE REMOVED IN ALL ASTRONAUTS?	Chad Ball *Indiana University*
17 Sunday	LIGATION VS RECONSTRUCTION OF THE SOFT PANCREATIC REMNANT FOLLOWING PANCREATICODUODENECTOMY – REVISITING AN OLD FRIEND	Jason Denbo *University of Tennessee, Memphis*

Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
18 Sunday	DUPLICATE PANCREAS MEETS GASTRIC DUPLICATION CYST: A TALE OF TWO ANOMALIES	Kathleen Christians *Medical College of Wisconsin*
19 Sunday	A SAFE RECONSTRUCTION TECHNIQUE AFTER PANCREATICODUODENECTOMY	Marcel Machado *University of Sao Paulo*
20 Sunday	EFFECT OF ABERRANT HEPATIC ARTERIAL ANATOMY ON OVERALL SURVIVAL IN PATIENTS WITH ADENOCARCINOMA OF THE PANCREAS WHO UNDERGO PANCREATICODUODENECTOMY	Christiana Shaw *Fox Chase Cancer Center*
21 Sunday	NORMAL LIVER ENZYMES ON DAY #1 OF ACUTE PANCREATITIS (AP) PREDICT HIGH RECURRENCE RATES OF PANCREASTITIS AFTER CHOLECYSTECTOMY : A POPULATION-BASED STUDY	Santhi Vege *Mayo Clinic*
22 Sunday	PANCREATIC HETEROTOPIA OF THE DUODENUM: ANATOMIC ANOMALY OR CLINICAL CHALLENGE?	Marius Distler *University Hospital TU-Dresden, Germany*
23 Sunday	OUTCOMES OF DISTAL PANCREATECTOMY AS PART OF MULTIORGAN TUMOR RESECTION	Ioannis Konstantinidis *Massachusetts General Hospital*
24 Sunday	CYST SIZE AND MINOR SYMPTOMS ALONE ARE NOT ASSOCIATED WITH MALIGNANCY IN BRANCH-DUCT IPMNS	Stefano Crippa *Department of Surgery University of Verona*
25 Sunday	ANTI-INFLAMMATORY EFFECTS OF PERITONEAL LAVAGE IN ACUTE PANCREATITIS	Marcel Machado *University of Sao Paulo*
26 Sunday	PREOPERATIVE THERAPY DOES NOT ADVERSELY AFFECT NUTRITIONAL ANTHROPOMETRICS IN PANCREATIC ADENOCARCINOMA PATIENTS.	Maria Petzel *The University of Texas M. D. Anderson Cancer Center*
27 Sunday	(IN)ACCURACY OF PREOPERATIVE CLASSIFICATION OF PANCREATIC CYSTIC NEOPLASMS	Andrew Russ *University of Wisconsin School of Medicine and Public Health*
28 Sunday	RADIOPAQUE BIODEGRADABLE STENT FOR PANCREATOBILIARY APPLICATIONS – THE FIRST HUMAN PHASE I STUDY IN PANCREATICO-JEJUNOSTOMY	Juhani Sand *Tampere University Hospital*

Board # and Day	TITLE OF POSTER	Corresponding Author & Institution
29 Sunday	THE NUMBER OF METASTATIC LYMPH NODES BUT NOT LYMPH NODE RATIO IS AN INDEPENDENT PROGNOSTIC FACTOR AFTER RESECTION OF PANCREATIC CARCINOMA	Yasushi Hashimoto *Department of Surgery, Division of Clinical Medical Science, Graduate School of Biomedical Sciences, Hiroshima University*
30 Sunday	THE CYCLOOXYGENASE-2 (COX-2) DEPENDENT EXPRESSION OF ANGIOGENIC CXC CHEMOKINES LIGAND (CXCL) IN HUMAN PANCREATIC CANCER CELL LINES.	Makoto Satake *Hyogo College of Medicine, Epartment of Surgery*
31 Sunday Poster of Note	LAPAROSCOPIC PANCREATICODUODENECTOMY CAN BE SAFELY IMPLEMENTED IN A HIGH-VOLUME PANCREATIC SURGERY CENTER.	Amer Zureikat *University of Pittsburgh Medical Center*
32 Sunday Poster of Note	DOES RESECTION FOR AN INVASIVE INTRADUCTAL PANCREATIC MUCINOUS NEOPLASM PROVIDE A BETTER PROGNOSIS THAN FOR A PANCREATIC ADENOCARCINOMA? COMPARISON OF CASES MATCHED BY TMN STAGE AND TUMOR LOCATION.	Toshiyuki Moriya *Virginia Mason Medical Center*
33 Sunday Poster of Note	SELECTIVE USE OF ARTERIAL RESECTION DURING PANCREATICODUODENECTOMY FOR PANCREATIC CANCER IS EFFECTIVE.	Carlo Contreras *MD Anderson Cancer Center*
34 Sunday Poster of Note	SURGICAL MANAGEMENT OF SOLID-PSEUODOPAPILLARY NEOPLASMS OF THE PANCREAS (FRANZ OR HAMOUDI TUMORS): A LARGE SINGLE-INSTITUTIONAL SERIES	Sushanth Reddy *The Johns Hopkins University*
35 Sunday Poster of Note	INHIBITION OF SIRT1 AS A NOVEL THERAPEUTIC STRATEGY FOR PANCREATIC CANCER	Vikas Dudeja *University of Minnesota*
36 Sunday	INTRADUCTAL MUCINOUS PAPILLARY NEOPLASMS: GENETIC CHARACTERIZATION OF LESION PROGRESSION.	Robert Jury** *William Beaumont Hospital*

** Indicates Short Oral Presentation Also

Session 1: 1:00 PM

DOWNSTAGING CHEMOTHERAPY (DCTX) MAY ALTER THE CLASSIC CT/MRI SIGNS OF VASCULAR INVOLVEMENT IN PATIENTS WITH PANCREATICOBILIARY CANCERS. THIS SHOULD INFLUENCE PATIENT SELECTION FOR SURGERY

Timothy R. Donahue MD1, O. Joe Hines MD1, James S. Tomlinson MD PhD2,5, James J. Farrell MD3, Yasser M. Bhat MD3, William H. Isacoff MD4, Edward Garon MD4, Barbara Clerkin NP MPH1, Howard A. Reber MD1

Departments of Surgery, [1]Division of General Surgery and [2]Surgical Oncology, and Medicine, [3]Division of Digestive Diseases and [4]Hematology and Oncology, David Geffen School of Medicine at University of California Los Angeles, Los Angeles, CA (UCLA), and [5]Greater Los Angeles Veterans Healthcare System, Los Angeles, CA

Introduction/Background: Surgery is the only treatment associated with cure in patients with pancreaticobiliary cancers. However, by virtue of the anatomic location and aggressive tumor biology, patients often present with locally advanced disease with major vascular involvement that precludes resection. Unresectable patients are treated with chemotherapy and/or radiation, which can occasionally result in impressive tumor responses. In fact, some patients are downstaged and become candidates for possible resection. The preoperative clinical and radiographic factors that predict resectablility after DCTx and the efficacy of this treatment strategy are still not well defined.

Methods: We reviewed a retrospective case series of 41 patients with locally advanced pancreaticobiliary cancers (39 pancreas, 2 bile duct) who underwent reoperation after completing a course of DCTx.

Results: We operated on 41 patients (1992-2009) with locally advanced pancreaticobiliary cancers after 8.5 months (median) of DCTx. Before DCTx, most (78%) had classic signs of major vessel involvement on CT/MRI (e.g. vessel distortion, encasement). Criteria for exploration after DCTx were 1) CT/MRI evidence of tumor shrinkage and/or change in signs of vascular involvement, 2) CA 19-9 decrease, and 3) good functional status. None had progressive disease. At operation, we resected 34/41 (83%) patients; 7 remained unresectable because of persistent vascular involvement. Surprisingly, CT/MRI scan was only 72% sensitive and 57% specific to detect vascular involvement *after* DCTx. The positive and negative predictive values were 31% and 88% respectively. Apparent persistent "involvement" on imaging was often from tumor fibrosis rather than viable cancer. Radiographic decrease in tumor size also did not predict resectability ($p>0.05$). Resected patients had a mean 87% decrease in CA19-9 ($p=0.04$) during DCTx. Median follow-up (all survivors) was 31 months. On subgroup analysis, the median disease-specific survival (DSS) of the 32 patients with pancreatic cancer (PDAC) who underwent resection was 52 months. Interestingly, 81% (26/32) of these patients had lymph node negative disease. There were 9 patients with $\geq$ 5 year survival (median follow-up 85 months), one of whom died of another cause and 8 who are alive without evidence of disease, yielding a minimum 5-year survival rate of 28% (9/32). Furthermore, if one considers the 8 patients with $<$ 5 year survival with no evidence of disease at last followup, then the maximum possible 5-year survival is 53% (17/32).

Conclusions: In resectable patients with initially unresectable pancreaticobiliary cancers, original CT/MRI signs of vascular involvement may persist even after successful DCTx. Thus, patients should be chosen for surgery based on lack of disease progression, good functional status, and decrease in CA 19-9, rather than evidence that vessel involvement has disappeared. In our series, this treatment strategy led to a high rate of resectability after DCTx and an excellent survival of patients who underwent resection.

Session 1: 1:15 PM

SIGNIFICANCE OF PATHOLOGIC RESPONSE TO PREOPERATIVE THERAPY IN PANCREATIC CANCER

Y. S. Chun[1], H. S. Cooper[2], J. C. Watson[1], P. B. Rao[1], J. P. Hoffman[1]
[1]Department of Surgical Oncology; [2]Department of Pathology, Fox Chase Cancer Center, Philadelphia, PA

Background: Pathologic response to preoperative therapy is increasingly recognized as an important prognostic factor in solid tumors. The impact of pathologic response on survival in pancreatic adenocarcinoma is not well-established.
Methods: Data on 135 consecutive patients treated with gemcitabine or 5-fluorouracil-based chemoradiation followed by pancreatectomy for adenocarcinoma of the pancreatic head and/or body between July 1987 and May 2009 were reviewed. Prospective histopathologic examination was performed in 108 patients to determine pathologic response, defined as minor (<50% fibrosis relative to residual neoplastic cells), partial (50–94% fibrosis), and major (95–100% fibrosis).
Results: Minor, partial, and major pathologic response rates were 17% (n=18), 64% (n=69), and 19% (n=21), including a 7% (n=8) complete pathologic response rate. Pathologic response correlated with R0 resection (*P*=.019), positive lymph nodes (*P*=.006), and tumor size (*P*=.001, Table). Median survival rates by pathologic response were as follows: 10 months (95% confidence interval [CI], 0-25 months) minor response, 14 months (95% CI, 11-17 months) partial response, and 51 months (95% CI, 29-73 months) major response (minor *v* partial response, *P*=not significant; partial *v* major response, *P*=.011). Multivariate analysis revealed that major pathologic response (*P*=.029; hazard ratio [HR], 2.51) and R0 resection (*P*=.001; HR, 2.33) were independent predictors of survival.
Conclusions: Major pathologic response to preoperative therapy occurs in a minority of patients with pancreatic adenocarcinoma and is independently associated with prolonged survival.

Table

Pathologic response	R0 resection, n (%)	Positive lymph nodes, n (%)	Median tumor size (range), cm
Minor, n=18	12 (67%)	4 (22%)	3.5 (1–5.5)
Partial, n=69	36 (52%)	24 (35%)	2.5 (1–5.7)
Major, n=21	18 (86%)	0	0.3 (0–6.5)

Session 1: 1:30 PM

PATTERNS AND PREDICTORS OF FAILURE AFTER CURATIVE RESECTIONS OF PANCREATIC ENDOCRINE CARCINOMA

Massimo Falconi, Letizia Boninsegna[1], Rossella Bettini[1], Francesco Panzuto[2], Stefano Partelli, Paola Capelli†, Aldo Scarpa†, Gianfranco Dalle Fave[2], Claudio Bassi, Paolo Pederzoli.

[1]Department of Surgery, "S.Cuore-Don Calabria" Hospital, Negrar, [2]Department of Gastroenterology, Sant'Andrea Hospital, Rome, Departments of Surgery, Chirurgia "B" and †Pathology , "G.B. Rossi" Hospital, University of Verona (Italy).

Introduction. Pancreatic endocrine carcinomas (PECs) are generally associated with a good prognosis above all after radical resection. In other pancreatic malignancies predictors of failure and the role of lymph node ratio (LNR) are well known, but prognostic factors and the value of LNR as well as patterns of recurrence after surgery for PEC has never been
investigated.

Methods. The prospective database from the Department of Surgery of the University of Verona was queried and all clinical and pathological data of patients with radically resected, pathologically confirmed, PECs between 1990 and 2007 were reviewed. Univariate and multivariate analyses were performed.

Results. Fifty-six patients (28 males and 28 females) with median age of 58 years entered in the study. Postoperative mortality was nil and 23 (41%) patients faced a complication. At a median follow up of 43 months, the median overall survival and the median disease specific survival (DSS) were 125 and 76 months, respectively. After a median interval time of 19 months from surgery, recurrent disease was identified in 25 patients (44.6%) and the 2 and 5-year DSS were 69.8% and 52.1%, respectively. There were 19 (44%) patients who had negative lymph nodes (N0), whereas 37 (66%) had lymph node metastases (N1). Patients with lymph node metastases had a lymph node ratio (LNR) ≤0.15 in 25 cases whereas 12 (32.4) had a LNR >0.15. The frequency of microvascular (76.8% versus 23.2%, p=0.002) and peripancreatic fat (54.3% versus 35.7%, p=0.0007) invasion as well as the median value of Ki67 (8% versus 3%, p=0.003) were significantly higher in those patients who faced recurrence. On multivariate analysis, LNR >0.15 (HR:) and a value of Ki67 >5% (HR 3.71) were significant predictors of recurrence (P <0.002).

Conclusions. After curative resection for PECs, LNR and a value of Ki67 >5% are the most powerful predictors of recurrence. The presence of these factors should be considered in the choice of addressing patients to adjuvant treatment in future clinical trials.

Session 1: 1:45 PM

CT STAGING SYSTEM FOR PANCREATIC CANCER

C. J. Clark[1], D. L. Coy[2], L. W. Traverso[1]
[1]Department of General Surgery, Virginia Mason Medical Center, Seattle, WA; [2] Department of Radiology, Virginia Mason Medical Center, Seattle, WA

Introduction: To accurately stage cases with locally-extending disease (unresectable and borderline lesions) a staging system based on high quality CT imaging is required. This system should be validated by survival. We used a large number of patients with biopsy-proven pancreatic cancer involving the head of the gland to investigate such a system.

Methods: Between 2000 and 2008, 220 patients with adenocarcinoma of the pancreas were felt to be currently not resectable due to locally-extending disease by CT (T3 or T4). We excluded cases if a high quality CT was not available at their initial staging and those who underwent surgical resection. We included only cases involving the head of the gland. A radiologist blinded to the initial clinical stage independently reviewed high quality contrast enhanced, thin-cut, multi-detector CT studies and characterized tumor burden based on AJCC T3 and T4 staging (7th Ed.). Primary tumor T4 was defined as major arterial abutment or encasement. Using the Kaplan-Meier method, these CT findings were assessed for correlation with survival. Staging was completed with outpatient 5mm laparoscopy and peritoneal lavage, and if positive were considered M1.

Results: Exclusion and inclusion criteria were met for 110 cases where the median tumor size was 38 mm. At least one major mesenteric vessel was involved in 98% (n=108) of patients.

	Overall Survival			
AJCC Stage 7th Ed.		**2 year**		**Log-rank p-value**
cT3	63%	25%	12%	0.132
cT4	46%	13%	6%	
pM0	55%	16%	10%	0.027
pM1	30%	12%	0%	
cT3,pM0	69%	27%	13%	0.017
cT3,pM1	0%	0%	0%	
cT4,pM0	50%	13%	9%	0.186
cT4,pM1	33%	13%	0%	

Conclusion: Although high quality CT imaging can detect aggressive tumor behavior in the form of tumor extension to adjacent mesenteric arteries, our study suggests that current CT imaging is not able to discern a survival difference for T3 vs T4 disease. However, positive peritoneal cytology (M1 disease) in this group of patients demonstrates worse overall survival. Adjunctive staging procedures, such as diagnostic laparoscopy and peritoneal lavage for cytology, may be useful in identifying patients with worse survival when CT demonstrates no evidence of metastatic disease but are considered to be locally-extending disease.

Session 1: 2:00 PM

DOES NEOADJUVANT THERAPY IMPROVE SURVIVAL IN PATIENTS WITH RESECTABLE PANCREATIC CANCER?

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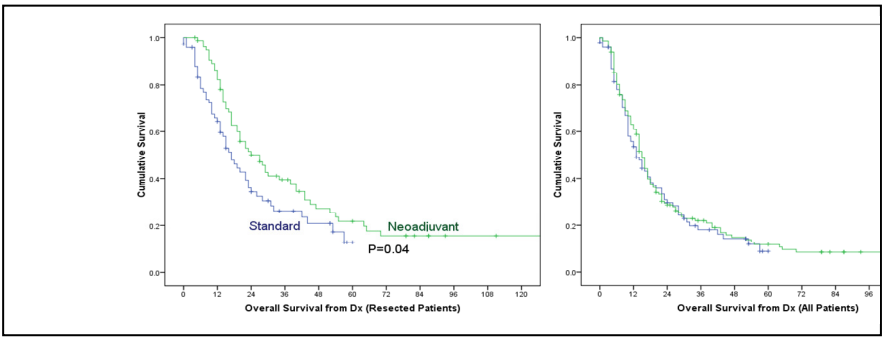
Introduction: Retrospective series show pancreatic cancer pts who undergo resection after neoadjuvant chemoradiation therapy (NEOCRT) live longer than pts who undergo resection without NEOCRT, a difference that may be attributable to patient selection. Our goal was to compare survival between all pts with resectable disease who underwent NEOCRT or surgical exploration with "intent to resect".
Methods: We retrospectively identified 237 pts with pancreatic head adenocarcinoma seen between 1999-2007 with enough data to be confirmed medically and radiographically resectable by NCCN criteria.
Results: Ninety-three pts (39%) proceeded directly to exploration (SURGERY) due to lack of tissue diagnosis or patient/surgeon preference, and 144 pts (61%) initiated NEOCRT. The groups were similar in age and tumor size on imaging, but the NEOCRT group was more likely to have venous abutment (47 vs. 25%, $p<0.01$) and trended toward having a higher Charlson comorbidity index ($p=0.09$). In the NEOCRT group, 76 pts (53%) underwent resection, 29 (20%) had metastatic and 16 (11%) locally unresectable disease after NEOCRT, and 23 (16%) were not explored due to performance status or loss to follow-up. In the SURGERY group, 68 pts (73%) underwent resection (of whom 66% received adjuvant therapy), 17 (18%) had metastatic and 8 (9%) locally unresectable disease. In resected patients, the NEOCRT group had smaller path tumor size and lower incidence of positive lymph nodes than the SURGERY group but no difference in positive margins or need for vascular resection. Median follow-up in surviving pts was 30 mos. from diagnosis. Median overall survival (OS) in resected pts was 27 mos. in the NEOCRT group and 17 mos. in the SURGERY group ($p=0.04$, Figure left). Median OS in all pts was 15 and 13 mos., respectively, with superimposable survival curves (Figure right).
Conclusions: Despite a lower resection rate, the NEOCRT group had similar survival to the SURGERY group, suggesting that NEOCRT allows better patient selection for resection. To demonstrate an effect on survival, a randomized trial is necessary to control for differences in patient/tumor characteristics that influence the decision to offer NEOCRT.



Session 1: 2:15 PM

MOLECULAR MECHANISMS UNDERLYING THE SYNERGISTIC INTERACTION OF THE NOVEL ANTICANCER DRUG UKRAIN WITH GEMCITABINE IN PRECLINICAL MODELS OF PANCREATIC CANCER

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Introduction/Background. Current therapy for pancreatic ductal adenocarcinoma (PDAC) is surgery followed by adjuvant radiotherapy and chemotherapy for early-stage, and palliative chemotherapy for advanced disease. Gemcitabine is the standard drug in both adjuvant and palliative treatment, but yields a marginal impact on disease outcome. Several attempts to improve the efficacy of gemcitabine by addition of a second cytotoxic or targeted agent have not shown a significant survival advantage. A new drug, NSC-631570 (Ukrain), used, showed greater median survival in combination with gemcitabine compared to gemcitabine alone (10.4 months vs 5.2 months; $p<0.001$) in the palliative treatment of unresectable PDAC (Gansauge et al.; Langenbecks Arch Surg 2002). However, the authors did not study the interactions between ukrain and the molecular determinant expressions involved in the metabolism of gemcitabine. There is compelling evidence that gene transcripts of determinants of gemcitabine activity, such as hENT1, could be used to tailor chemotherapy in PDAC (Giovannetti et al., Cancer Res 2006). Therefore, the aim of present study was to evaluate the modulation of the expression of two pivotal genes (hENT1 and dCK) involved in gemcitabine activity
Methods. In vitro studies were performed in 2 ATCC cell lines (PL45 and Mia PaCa-2) and 2 Primary Cell Cultures obtained from PDAC patients underwent surgical resections (PPTCC78 and PPTCC109). Cells were treated with Ukrain at IC_{50} concentration levels for 48h. The total RNA extraction was performed with Trizol protocol. All the amplifications were carried out with normalization of gene expression against the glyceraldehyde 3-phosphate dehydrogenase (GAPDH) housekeeping control gene, and the quantitation of gene expression was performed using the direct ratio, the standard curve method and the $\Delta\Delta$CT calculation, in which the amount of target, normalized to the endogenous control and relative to the calibrator (untreated control cells) was calculated as $2^{(-\Delta\Delta Ct)}$.
Results. Ukrain positively modulates the expression of hENT1 mRNA in all PDAC cell cultures treated with IC50 ($p<0.001$). The $2^{(-\Delta\Delta Ct)}$ analysis revealed a mean increase of 2.8 fold ($p=0.001$) with respect to untreated control cells. In PL45 and Mia PaCa-2 cells Ukrain positively affects mRNA expression of dCK gene as well.
Conclusions. To date a few options based on gemcitabine are available for treatment of PDAC. Most gemcitabine-based chemotherapy regimens resulted in a very limited disease control, and studies attempting to widen the therapeutic armamentarium against this disease are warranted. Based on the previous clinical data the Ukrain-gemcitabine combination appears a promising regimen and the results of the present study provide the experimental basis for the further clinical testing of the Ukrain-gemcitabine schedule in PDAC patients

Session 1: 2:30 PM

EFFICACY OF ADJUVANT VERSUS NEOADJUVANT THERAPY FOR RESECTABLE PANCREATIC ADENOCARCINOMA: A DECISION ANALYSIS

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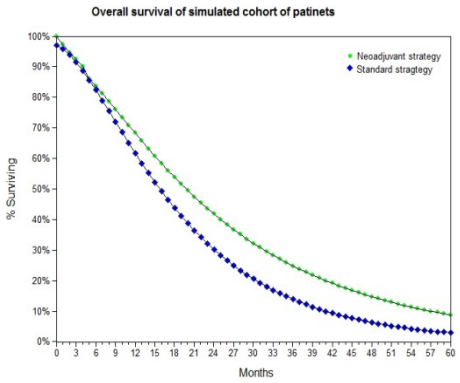
Background: Neoadjuvant therapy-based protocols for potentially resectable pancreatic adenocarcinoma offer theoretical advantages over standard adjuvant therapy-based management. However, these advantages are unproven. The aim of this study was to compare the efficacy of neoadjuvant therapy- and adjuvant therapy-based management using formal decision analysis.
Methods: A decision analytic Markov model was created to compare two management strategies for simulated cohorts of patients with potentially resectable pancreatic adenocarcinoma. In the standard strategy, patients undergo surgical resection and subsequently are treated with either adjuvant systemic chemotherapy (CT), chemoradiation therapy (CRT) or both, as tolerated. In the neoadjuvant strategy, patients are treated with an average of 3 months of neoadjuvant therapy (CT, CRT or both) first and then undergo surgical resection (unless disease progression renders them unresectable). Base-case probabilities were derived from published data derived from phase II and III trials (a total 3302 patients with potentially resectable pancreatic cancer were analyzed). The outcome measures were overall and quality-adjusted survival, with survival calculated from date of surgery (adjuvant group) or date when neoadjuvant therapy was initiated. Intention-to-treat analysis was used. Sensitivity analysis was performed to assess the effects of model uncertainty on outcomes.
Results: The median overall survivals and 2-year OS rates were 16 months and 30% for patients managed by the standard strategy and 20 months and 42% for those managed by the neoadjuvant strategy, respectively (Figure). Quality-adjusted overall survivals for these patients were 13.8 months and 19.6 months, respectively. Sensitivity analysis indicated the benefits of the neoadjuvant strategy over the standard strategy in terms of both OS and quality-adjusted survival are robust: stability of findings is maintained over a wide range of plausible baseline estimates.
Conclusions: Our analysis suggests that neoadjuvant therapy-based management improves outcomes of patients with potentially resectable pancreatic cancer. A randomized trial designed to evaluate the advantage of this strategy is warranted.



Session 2: 4:00 PM

PRELIMINARY DATA ON SURVIVAL AFTER RADIOFREQUENCY ABLATION OF STAGE III PANCREATIC CANCER: A WIND OF CHANGE?

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BACKGROUND AND AIM

Radiofrequency ablation (RFA) is an accepted treatment for different not resectable solid tumours but experiences in pancreatic cancer are still very limited. We recently demonstrated [1] feasibility and safety of RFA in locally advanced (Stage III) pancreatic cancer (LAPC).

Aim of the present study is to evaluate survival after RFA plus different regimens of chemo-radiotherapy (Ch+TR) in pts with LAPC.

MATHERIALS AND METHODS

Only pts with histologically proven LAPC were consecutively enrolled. RFA was carried out during laparotomy. CT scan was performed at discharge and follow up (consisting in clinical examination, CT and serum Ca19.9) was planned at 1-3-6-12 months. Overall survival (OS), disease specific survival (DSS) and progression free survival (PFS) were analysed.

RESULTS

Between February 2007 and December 2008, 56 patients with LAPC underwent RFA and were enrolled for a minimum 12 months follow up. In 7 pts a diagnosis was made before January 2007 and they were excluded from the study. Male/female ratio was 24/25 with a median age of 61. Tumor was located in the head in 29 pts (59%) and in body-tail in 20 pts (41%). Median tumor diameter was 37 mm. Thirty seven pts (76%) received RFA as up front treatment while 12 pts (24%) received different associations of Ch-TR before RFA (3 pts only Ch, 7 pts Ch+RT, 2 pts Ch + RT + locoregional Ch).

Palliative surgery was associated to RFA in 30 pts (61%): digestive bypass in 7 pts (14%), biliary bypass in 3 pts (6%), double bypass in 19 pts (39%) and pancreo-jejunum anastomosis in one case. Postoperative mortality occurred in 2 pts (4%).

The 1 - and 2 year OS was 67% and 45% respectively with a median survival of 20 months. The 1 - and 2 year DSS was 74% and 51% respectively with a median survival of 28 months. In all, 34 pts had recurrence of disease and 20 of them eventually died of disease. The 6 - and 12 months PFS was 62% and 24% respectively with a median of 10 months. Patients who received RFA as up front treatment (#37) had 1 – and 2 year OS of 67.4% and 52.6% respectively with a median survival of 28 months, not significantly different for patients undergone to RFA after Ch-RT.

CONCLUSIONS

LAPC is a very aggressive entity with a median survival range from 5.3 to 14.5 months after different regimens of Ch-RT [2]. To confirm this trend, in the same period of the present study, we treated 70 pts suffering from LAPC with Ch-RT without RFA: in these patients the 1- and 2 year OS was 46% and 23% respectively ($p<0.041$ when compared to the OS in the RFA group). A significant difference is also achieved in favor of RFA considering only the 49 pts out of 70 without RFA who undergone palliative or explorative surgery.

In conclusion the impact on survival achieved in the present study is tempting and seems to be independent by the RFA timing.

On the bases of this experience RFA could be considered as part of future new multimodal therapy\ for LAPC.

1] Girelli R. et al. Br J Surg 2010, in press. (Published online in Wiley InterScience (www.bjs.co.uk). DOI: 10.1002/bjs 6800). 2] Huguet F. et al. Chemoradiotherapy in the Management of Locally Advanced Pancreatic Carcinoma:

Session 2: 4:15 PM

FEASIBILITY AND SAFETY OF ROBOTIC PANCREATECTOMIES: ANALYSIS OF TWENTY-NINE CONSECUTIVE OPERATIONS

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Background: The use of the da Vinci Surgical System (dVSS) (Intuitive Surgical®, Sunnyvale, CA, USA) in minimally invasive surgery, maximizes surgeon power by providing he or she with real 3D vision, use of three robotic arms equipped with wristed instruments, and tremor filtration. These enhanced operative abilities might be particularly useful in pancreatectomies. The aim of this study is to evaluate feasibility and safety of robotic pancreatectomies.

Methods: From April 2008 to December 2009, 29 consecutive robotic pancreatectomies were performed at our Institution. Nine patients were male (31%) and 20 (69%) female, with a mean age of 58.2 yrs (range 24-80 yrs) and a mean BMI of 25 kg/m^2. Patients were selected for robotic pancreatectomy when diagnosed with benign or low-grade malignancy and when otherwise suitable for a minimally invasive approach.

Results: No procedure was converted to either conventional laparoscopy or open surgery. The procedures performed were: 10 pancreatoduodenectomies (34.5%), 13 (44.8%) distal pancreatectomies (including 10 with preservation of splenic vessels and spleen), 3 (10.3%) central pancreatectomies, 2 (6.9%) tumor enucleations, and 1 (3.5%) total pancreatectomy. Final pathology demonstrated intraductal papillary mucinous neoplasm in 6 patients (20.6%), neuroendocrine tumor in 5 patients (17.2%), mucinous cystadenoma in 4 patients (13.8%), serous cystadenoma in 4 patients (13.8%), ampullary tumor in 4 patients (13.8%), ductal adenocarcinoma in 2 patients (6.9%), solid pseudopapillary tumor in 2 patients (6.9%), chronic pancreatitis in 1 patient (3.5%), and duodenal cancer in 1 patient (3.5%). Mean operative time was 438 minutes (range 120-800). There were no post-operative deaths, but 14 patients developed post-operative complications (48.3%). Reoperation was required in a patient, because of bleeding caused by a small artery behind the pancreatico-jejunostomy. Although the anastomosis was seemingly intact, and there were no signs of overt retroperitoneal infection, we decided to manage this patient by completion pancreatectomy. Overall, based on the recommendations of the international study group on pancreatic fistula, there were 11 pancreatic fistulas (39.3%). Mean post-operative stay was 14.1 days (10.5 days for patients with an uneventful course vs 17.9 days for patients with post-operative complications; p=0.01). Four patients (13.8%) required peri-operative transfusions.

Conclusions: Our initial experience shows that robotic pancreatectomies, including pancreatoduodenectomy, are feasible with an acceptable operative risk. Because of the paucity of information on robotic pancreatectomies, there is a price to pay to the learning curve. Subjectively, the dVSS allows surgeons to reproduce laparoscopically the same manoeuvres that would have been performed during open surgery. Whether or not this is real, and if it eventually translates in better outcome as compared with conventional laparoscopy, remains to be determined. In this initial experience, we acknowledge a rather high incidence of pancreatic fistula. Some of these fistulas, however, were grade A. Further, pancreatic remnants were all soft and with small ducts, because of patients' selection criteria.

Session 2: 4:30 PM

ROBOT-ASSISTED MAJOR PANCREATIC RESECTIONS: A RETROSPECTIVE ANALYSIS OF 30 CONSECUTIVE PATIENTS.

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Introduction: Despite widespread adaptation of minimally invasive techniques to complex surgical procedures, limitations inherent in laparoscopic techniques have reduced their application to pancreaticoduodenectomy (PD). Open PD has significant postoperative morbidity and a complication rate approaching 45% in large series with a 10 day median length of stay. Robotically-assisted minimally invasive surgery offers several significant advantages compared to traditional laparoscopic approaches, including enhanced visualization in three dimensions with near 360 degree range of motion of the surgical instruments. The robotic technique allows the complex reconstruction following PD to be performed using a technique identical to the open procedure.

Methods: We conducted a retrospective review of the first thirty robotic-assisted complex pancreatic resections at the University of Pittsburgh Medical Center. All procedures were conducted by a single surgical team possessing a combination of advanced laparoscopic skills and extensive prior experience with pancreatic surgery. Cases were selected and approved by the multidisciplinary robotic surgical oncology committee. Patients with borderline-resectable pancreatic malignancy were excluded. We evaluated the perioperative events, final pathology, and complications occurring within the first 30 days on an intention to treat basis.

Results: Between October 2008 and December 2009, we performed 26 robotic pancreatoduodenectomies (PD), 3 central pancreatectomies, and 1 duodenum-preserving pancreatic head resection. Final pathology included 17 periampullary adenocarcinomas (9 pancreatic, 8 ampullary), 9 pancreatic cystic neoplasms, 3 pancreatic neuroendocrine tumors, and one chronic pancreatitis. Conversion to open was only necessary for patients undergoing PD. The PD completion rate was 74%. Reasons for conversion (7/26) to open PD included unsuspected venous involvement (3) and failure to progress (4). For the 26 patients undergoing intended robotic PD (11 male, 15 female), median age was 76.5 (range 47-85). Median operative time was 590 minutes (range 327-848 min) with 500 cc median estimated blood loss (100-2000 cc). The R0 resection rate following PD for malignancy was 85% (17/20). Median lymph node harvest was 16 (5-31). Median length of stay was 10 days (4-87 days). The rate of pancreatic fistula was 23% (6/26), but only 2 (8%) were grade C. 8 patients (38%) developed minor complications (Clavien grade 1/2), whereas 7 had major complications (33%). There was one late death (3.8%) on postoperative day 87 as the result of multiple precipitating factors.

Conclusions: This early experience suggests that robotic assisted minimally invasive pancreaticoduodenctomy can be performed with equivalent safety and oncologic outcomes to open procedures.

Session 2: 4:35 PM

PERIOPERATIVE OUTCOMES FOR OPEN DISTAL PANCREATECTOMY: CURRENT BENCHMARKS FOR COMPARISON?

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Cancer Center, Sacramento, California

Background: There exists increasing enthusiasm for laparoscopic distal pancreatectomy (LDP) for benign and malignant lesions of the pancreatic tail. Comparisons to open distal pancreatectomy (ODP) outcomes have largely relied on single institution databases primarily from high-volume, tertiary centers. Therefore, to provide an appropriate national benchmark of ODP outcomes, we examined the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) database (2005-07) to accurately describe 30-day morbidity and mortality, operative time, transfusion requirement, and hospital length-of-stay (LOS) for patients undergoing ODP.
Methods: The NSQIP database was used to identify 1,313 cases of distal pancreatectomy (CPT code 48140). Cases with extra-pancreatic procedures or any laparoscopic component were excluded leaving a final study cohort of 868 patients. Central tendencies of operative time, intraoperative transfusion requirement, and LOS were calculated. Univariate analyses were performed using all 53 preoperative NSQIP variables and malignant vs. benign diagnosis for likelihood of any postoperative complications and severe complications (composite endpoint: death, organ space surgical site infection, or reoperation). Multivariate analyses were performed using a logistic regression likelihood model, adjusting for age, body mass index (BMI), diagnosis type, creatinine, albumin, hematocrit, and American Society of Anesthesiologists classification (ASA).
Results: Any complication, severe complication, and mortality rates were 27.2%, 11.6%, and 1%, respectively. Mean operative time was 206 minutes (±86); 18.1% patients required intraoperative red blood cell transfusion (median 2 units; range 1-15), and median LOS was 6 days. Predictors of any complication or severe complication were renal insufficiency, hypoalbuminemia, and worsening ASA classification. Malignant diagnosis was not associated with increased likelihood of morbidity or mortality.
Discussion: Open distal pancreatectomy remains the gold standard to which laparoscopic distal pancreatectomy is compared. Our analysis of patients undergoing ODP reflect nationwide data that may serve as current benchmarks to which patients undergoing LDP should be compared.

Table. Multivariate analysis of predictors of any complications and severe complications, n=868; Odds Ratio/ (95% Confidence Interval)

Variable	Any Complication	Severe Complication
Age (median 59 yrs)	1.00 (0.99-1.02)	1.00 (0.99-1.02)
BMI (median 27)	1.02 (1.00-1.05)	1.02 (0.98-1.05)
Diagnosis Type		
Benign	1.00 (referent)	1.00 (referent)
Malignant	1.06 (0.75-1.49)	1.04 (0.65-1.68)
Unknown	0.81 (0.50-1.32)	0.82 (0.42-1.62)
Creatinine		
<1.2	1.00 (referent)	1.00 (referent)
1.2-2.0	1.43 (0.88-2.33)	***1.89 (1.02-3.49)**
>2.0	0.89 (0.22-3.63)	0.82 (0.10-6.71)
Unknown	0.93 (0.38-2.23)	1.35 (0.45-4.07)
Albumin		
>3.5	1.00 (referent)	1.00 (referent)
2.5-3.5	***1.94 (1.12-3.38)**	1.57 (0.74-3.34)
<2.5	3.23 (0.75-13.84)	***6.63 (1.39-31.57)**
Unknown	1.04 (0.68-1.57)	1.06 (0.60-1.89)
Hematocrit		
36-49	1.00 (referent)	1.00 (referent)
>49	2.23 (0.29-17.22)	2.18 (0.21-23.05)
<36	0.89 (0.57-1.41)	0.76 (0.39-1.45)
Unknown	1.83 (0.77-4.36)	1.71 (0.58-5.04)
ASA Classification		
1/2	1.00 (referent)	1.00 (referent)
3	***1.67 (1.19-2.33)**	1.52 (0.95-2.44)
4	2.54 (0.91-7.08)	1.74 (0.45-6.75)
*** Denotes significance, P≤0.05**		

Session 2: 4:40 PM

A NOVEL EXPLANT CULTURE SYSTEM FOR THE IN VITRO STUDY OF MURINE PANCREATIC INTRAEPITHELIAL NEOPLASIA (PANIN)

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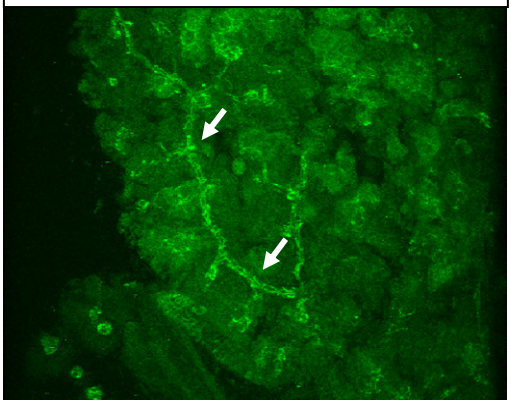
Background: Recent work has demonstrated a complex interaction between stromal and parenchymal cells in the development of pancreatic adenocarcinoma. The mechanism by which stromal derived factors such as retinoids and sonic hedgehog interact with cancer progenitor cells to form pancreatic intraepithelial neoplasia (PanIN) is unknown. Studying these events in real-time is not possible *in vivo* and cell culture fails to recapitulate the local microenvironment that is present during tumor initiation. In order to overcome these limitations, we have developed an *in vitro* system that allows the long term maintenance of intact pancreatic sections.

Methods: Adult mouse pancreata were isolated and sectioned using a 1000 Plus Vibratome sectioning system to a thickness of 200 micrometers. Pancreatic microslices were maintained in culture for an average of 5 days and were treated with 10nM cerulein. 5-ethynyl-2´-deoxyuridine (EdU; Invitrogen) to assess proliferation in culture. Microslices were fixed and stained for amylase, insulin, E-cadherin, and Dolichos Biflorus Agglutinin by immunofluorescence. Stained microslices were analyzed using fluorescent and confocal microscopy.

Results: Pancreatic microslices were successfully maintained in culture for up to 4 weeks after isolation. Metabolic activity was verified with methyl methanethio-sulfonate (MMTS). Through the use of immunostaining, we demonstrate that microslices maintain an intact pancreatic architecture exhibiting separate ductal (E-cadherin; Figure), acinar (amylase), and endocrine (insulin) compartments. In addition, we analyzed proliferation *in vitro* with EdU and observed that treatment of microslices with 10nM cerulein induced proliferation in ductal and acinar compartments.

2-Photon Image of Pancreatic Microslice Immunolabeled for E-cadherin, 20X: Arrows denote



Conclusion: We present a novel technique for studying pancreatic biology in cultured pancreatic microslices. This work lays the foundation for an innovative model to study the microenvironment effects on the development, regeneration, and neoplasia of *in vitro* pancreatic tissue in transgenic mice such as the K_{ras} mouse model of pancreatic adenocarcinoma.

Session 2: 4:55 PM

PREOPERATIVE CT MEASUREMENT OF PANCREATIC STEATOSIS AND VISCERAL FAT; PROGNOSTIC MARKERS FOR DISSEMINATION AND LETHALITY OF PANCREATIC ADENOCARCINOMA

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INTRODUCTION
Increased visceral fat amplifies the risk of developing pancreatic cancer, while pancreatic steatosis promotes lymphatic metastases and decreased survival for patients with pancreatic adenocarcinoma after pancreaticoduodenectomy (PD). However, a correlation between preoperative adipose measurements by CT scanning and patient outcomes remains ill defined. We aim to determine the utility of preoperative CT measurements of pancreatic steatosis and visceral fat as prognostic indicators for patients with pancreatic adenocarcinoma.

METHODS
High resolution CT scans of 42 patients undergoing PD for pancreatic adenocarcinoma were reviewed. CT attenuation of the pancreas, liver, and spleen were measured in Hounsfield units and scored by two blinded investigators. Perirenal adipose tissue (an indicator of visceral fat) was measured in mm. Pathology slides were reviewed for tumor differentiation and invasion. Data are presented as mean ± SD.

RESULTS
Lymphatic metastases were absent (N0) in 43% and present (N1) in 57% of patients. Age, gender, tumor size, and margin status were similar between patients with and without nodal metastases. Outcomes for patients stratified by nodal status and perirenal adiposity are depicted below. All patients received adjuvant therapy.

Nodal Status & Fat Pad Depth	Perirenal Adipocity (mm)	Pancreatic Body (HU)	Liver (HU)	Peripancreatic Fat Invasion (%)	Survival (months)
N0	13±2	35±4	58±4	53	21±4
N1	18±1*	23±2*	50±2*	90*	11±2*
N1 & <10mm	9±1	27±2	53±4	80	16±2
N1 & >10mm	21±1†	25±2	48±2	91	7±1†

*p<0.01 vs. N0, †p<0.01 vs. N1 & fat pad<10mm

CONCLUSIONS
With resected pancreatic adenocarcinoma, increased pancreatic and liver steatosis, as well as increased visceral fat stores are associated with lymphatic metastases. Furthermore, increased visceral fat is associated with an abbreviated survival for patients with lymphatic metastases, and thereby may serve a prognostic role for patients with pancreatic malignancies. Therefore, we conclude that CT measurements of visceral fat predict the dissemination and lethality of pancreatic adenocarcinoma.

Session 3: 8:00 AM

A TRANSLATIONAL CLINICAL STUDY OF A PANCREATIC CANCER VACCINE AS NEOADJUVANT TREATMENT AND ITS EFFECT ON THE TUMOR MICROENVIRONMENT

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Introduction
Pancreatic ductal adenocarcinoma (PDA) is the 4th leading cause of cancer death and is highly resistant to chemotherapy. An irradiated GM-CSF secreting allogeneic pancreatic tumor vaccine was developed and shown in phase I/II studies to be safe and effective in inducing an anti-tumor immune response and to enhance survival in PDA patients. Low dose cyclophosphamide may further enhance the anti-tumor immune response by depleting immunosuppressive $FoxP3^+$ regulatory T cells (Tregs). It has been suggested that an examination of immune infiltration in solid tumors may be used to assess the immune response to tumor and may also have prognostic value. In this study we investigate the immunologic response to vaccination within the tumor microenvironment of resected whipple specimens after neoadjuvant vaccine treatement with or without cyclophosphamide**.**

Methods
Eligible patients will be enrolled to a target accrual of 39 evaluable patients and will receive one vaccine prior to and 5 additional vaccines following pancreaticoduodenectomy in addition to standard adjuvant chemotherapy/chemoradiation. Patients are randomized to one of three arms: Arm A – vaccine alone, Arm B – vaccine with IV cyclophosphamide, Arm C – vaccine with oral metronomic cyclophosphamide. Immunohistochemistry is used to investigate immune cells infiltrating resected tumors.

Results
Between 7/08 and 1/10, 19 patients (Arm A: 7; Arm B: 6; Arm C: 6) received the neoadjuvant vaccination and two weeks later underwent pancreaticoduodenectomy. Immunohistochemical analysis of resected PDAs reveals tertiary lymphoid aggregates (LAs) residing within and peripheral to the tumor. These LAs are characterized by the presence of organized B and T cell zones and $CD21^+$ germinal centers. When compared to age- and sex-matched unvaccinated controls, the amount of intratumoral LAs appears to be increased by vaccination (mean=$4.4x10^{-08}$ LAs per um^2 tumor in vaccinated patients versus $2.3x10^{-08}$ per um^2 tumor in unvaccinated controls, P=0.096). The number of intratumoral LAs in vaccinated patients is inversely correlated with $FoxP3^+$ Tregs infiltrating the tumor (P=0.027). Intratumoral LAs have more proliferative activity (as measured by Ki67) than peritumoral LAs, and may function in the generation of anti-tumor adaptive immune responses. PD-1 and its ligand, B7-H1, are present on immune cells in the germinal centers of the majority of LAs. $B7\text{-}H1^+$ cells in the germinal center have a morphology similar to that of monocytes and may represent a mechanism of suppression of the anti-tumor adaptive response.

Conclusion
The resected tumors of patients receiving neoadjuvant vaccination demonstrate previously unreported tertiary LAs within PDA, which may differ in function from peritumoral LAs. The PD-1/B7-H1 pathway appears to be functioning in these LAs, and may represent a target for blockade in future vaccine trials. Further analyses and correlation with clinical outcomes is on-going.

Session 3: 8:15 AM

CLINICAL IMPLICATIONS OF THE STATUS OF MAJOR FOUR GENES IN PANCREATIC CANCER
ANALYSES OF MUTATIONS AND EXPRESSION OF THE KRAS, TP53, P16, AND SMAD4 GENES IN AUTOPSY CASES

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Introduction: Pancreatic cancer is a devastating disease and 80% of patients with pancreatic cancer have advanced disease at diagnosis. To expand our understanding of the clinical and molecular features characteristic of advanced stage disease, we established a rapid autopsy program for patients with end stage gastrointestinal malignancies with an emphasis on patients with pancreatic cancer. The alterations of major four genes (*KRAS*, *p16*, *TP53* and *SMAD4[DPC4]*) have been identified in pancreatic cancer carcinogenesis. The goal of this study was to compare the status of these four genes to clinicopathological features at autopsy in pancreatic cancers.
Patients and Methods: Rapid autopsies were performed on 91 patients (mean age, 62.7 years) with documented pancreatic cancer. Twenty-six patients previously underwent operation. Two of them had no evidence of disease at autopsy (died of other causes). Snap frozen samples (total 1067 lesions) were sequenced for *KRAS2* and *TP53*. Paraffin-embedded samples (total 564 slides) were immunostained for p16 and Smad4, for which immunolabeling is a sensitive marker of genetic status. The clinicopathological features including survival and metastatic burden at autopsy (4 groups: no metastases, 1 to10, 11 to 99 and 100s to 1000s) were determined and compared to the status of these four genes.
Results: Activating mutations in the *KRAS2* gene were identified in 92% of the cancers. Inactivating mutations in the *TP53* gene were identified in 67%. There was no heterogeneity in the status of *KRAS2* and *TP53* within each primary cancer, and there were no significant differences between the status of these genes in the primary cancer and in the patient's matched metastases. Loss of Smad4 and p16 immunolabeling was identified in 58% and 90% of the primary cancers, respectively. Interestingly, intratumoral heterogeneity of p16 and Smad4 immunolabeling was observed in 22% and 10% of the patients, respectively. Kaplan-Meier survival analysis in all 91 patients showed that the tumor size at diagnosis ($P = 0.0456$) and the status of *SMAD4* gene ($P = 0.0354$) were significantly associated with shorter survival. Significant correlations were also found between mutations of *TP53* ($P = 0.0482$), loss of Smad4 ($P = 0.0169$) and increasing number of altered genes ($P = 0.0022$) to metastatic burden. Two-thirds of the patients with all 4 altered genes had 100s to 1000s of metastases. Kaplan-Meier survival analysis in 26 patients who underwent surgical resection showed loss of Smad4 immunolabeling was significantly associated with shorter survival (median survival: 36 months for intact Smad4 *vs* 24 months for loss of Smad4; $P = 0.0325$). The increasing number of altered genes ($P = 0.0019$) and loss of Smad4 immunolabeling ($P = 0.0487$) were significantly correlated with extensive metastatic burden at autopsy in these patients.
Conclusions: Genetic alterations of *KRAS2* and *TP53* occur in pancreatic carcinogenesis prior to the development of metastatic spread and these mutations are universally present in both primary and metastatic cancers within a single patient. By contrast, genetic alterations of *p16* and *SMAD4* might occur not only in the development of an invasive carcinoma, but also during disease progression, inducing the heterogeneity within the same tumor. Genetic alteration of all 4 genes in the same carcinoma is highly correlated with extensive metastatic burden. Immunohistochemical analysis of Smad4 could provide prognostic information for predicting survival and patterns of failure, especially in patients with surgically-resected pancreatic cancer.

Session 3: 8:30 AM

MICRORNA-21 FROM BENCH TO BEDSIDE AND BACK: A POTENTIAL MARKER OF CLINICAL OUTCOME AND A TARGET TO OVERCOME RESISTANCE TO GEMCITABINE IN PANCREATIC CANCER

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Introduction/Background. MicroRNAs are small noncoding RNAs with pivotal functions in development, cell differentiation and apoptosis. Recently, microRNA-21 (miR-21) miR-21 was reported to be overexpressed in pancreatic ductal adenocarcinoma (PDAC). and contributed to tumor invasion and resistance to gemcitabine. The aim of this study was to evaluate whether miR-21 expression was associated with the overall survival (OS) of PDAC patients treated with gemcitabine and provide mechanistic insights for new therapeutic targets.

Methods. MiR-21 expression was evaluated in pancreatic cells (including 7 PDAC cells lines, 7 primary PDAC cultures, fibroblasts and a normal pancreatic ductal cell line) and tissues (neoplastic specimens from 77 PDAC patients in the adjuvant (N=45) or in the inoperable/metastatic (N=32) setting, and normal ductal samples) isolated by laser microdissection, using the Leica LMD7000 instrument. Association with OS was estimated using Kaplan-Meier method. The role of miR-21 on the pharmacological effects of gemcitabine was studied in cells transfected with a specific miR-21 precursor (pre-miR-21). Modulation of apoptosis and Akt activation was analyzed by Annexin V and ELISA assays. Quantitative PCR and ELISA assays evaluated the correlation of miR-21 with invasion-related genes (metalloproteinase-2/-9), and VEGF. Inhibitors of PI3K and mTOR (LY294002 and rapamycin) were used to test whether modulation of these signalling pathways affected molecular mechanisms activated by miR-21 and gemcitabine activity.

Results. MiR-21 overexpression correlated with worse outcome in PDAC patients treated with gemcitabine both in the palliative and in the adjuvant setting. In the latter the patients with miR-21 expression below the median (i.e. low expression) had a median OS of 23.7 months (95% CI, 12.3-35.0), while the remaining patients (high expression) had a median OS of 13.2 months (95% CI, 8.3-18.0), P=0.009. Similar data were reported for disease-free survival, and multivariate analysis confirmed the prognostic significance of miR-21 (HR=2.6, with P=0.001 for miR-21 high expression). MiR-21 expression in the primary cultures was significantly correlated to the expression detected in their respective tissues, and with gemcitabine resistance. Moreover, the treatment with gemcitabine resulted in a significant increase of miR-21 expression, ranging from 2 to 19-fold, in 13 PDAC cells. Pre-miR-21 transfection significantly decreased apoptosis induction by gemcitabine and up-regulated metalloproteinase-2/-9, and VEGF expression. In contrast, addition of LY294002 and rapamycin resulted in decrease of phospho-Akt and prevented pre-miR-21 induced resistance to the pro-apoptotic effects of gemcitabine.

Conclusions. MiR-21 expression correlated with clinical outcome in PDAC patients treated with gemcitabine. Modulation of apoptosis, Akt phosphorylation, and expression of genes involved in invasive behaviour, may contribute to miR-21 role in gemcitabine chemoresistance and provide mechanistic insights for the rational development of new targeted combinations against PDAC.

Session 3: 8:45 AM

OVEREXPRESSION OF EPIDERMAL GROWTH FACTOR RECEPTOR (EGFR) DETECTED BY ANTIBODY BINDING EGFR INTERNAL DOMAIN PREDICTS POOR SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA

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INTRODUCTION: Pancreatic ductal adenocarcinoma (PDA) is the fourth leading cause of cancer-related deaths in the United States. Monoclonal antibodies and small molecule inhibitors targeting epidermal growth factor receptor (EGFR) have been approved by Food and Drug Administration in combination with gemcitabine for pancreatic cancer treatment. EGFR/HER1 is a transmembrane glycoprotein from the HER family, with tyrosine kinase activity. When the ligand (epidermal growth factor) binds the receptor, dimerization and autophosphorylation of the receptor results in activation of signaling pathways regulating cell proliferation, migration and angiogenesis. Although EGFR mutations are rare in pancreatic cancer, overexpression of EGFR has been reported in as many as 30-70% of all cases. Recent studies evaluating EGFR expression by immunohistochemistry have used antibody detecting the external domain of EGFR. The aim of this study was to evaluate expression of EGFR in PDA using a novel antibody binding internal domain of EGFR.

METHODS: Eighteen cases of PDA from patients with long (>3 years) and 19 cases with short (<1 year) survival were included in the study. Representative sections were stained with anti-EGFR primary antibody (clone 5B7, an internal domain binding antibody, Ventana Medical Systems, Tucson, AZ, USA). Immunohistochemistry was performed following the manufacturer's recommendations. Immunohistochemical semiquantitative assessment of EGFR protein expression was based on the fraction of stained cells. Cases were scored as negative if there was no staining, 1+ if there was incomplete membranous staining, 2+ if there was complete membranous staining present in <30% and score 3+ was given to cases with complete membranous staining present in >30% of tumor cells. Score 3+ was considered to represent EGFR overexpression. Statistical analysis was performed using Fisher's exact test.
Gene expression profiling was performed on stromal PDA tissue from 6 patients with long and 7 patients with short survival. For those cases expression of epidermal growth factor (EGF) in tumor stroma was correlated with EGFR expression in tumor epithelium.

RESULTS: There was a statistically significant correlation between EGFR expression and shorter survival (p value=0.0081). There were no statistically significant differences between EGFR positivity and tumor grade, size, extrapancreatic extension or presence of lymph node metastases. Interestingly, 2 of 2 patients with EGFR overexpression and long survival had low EGF gene expression in profiled tumor associated stroma. All profiled cases with short survival had high EGF gene expression in stroma.

CONCLUSION: Our study suggests that EGFR overexpression in PDA, evaluated with novel internal domain binding antibody, is associated with poor prognosis. Evaluation of both EGFR and EGF may select patients who best respond to targeted therapies with EGFR inhibitors.

Session 3: 9:00 AM

HUR STATUS IS A POWERFUL CLINICAL MARKER FOR RESECTED PANCREATIC DUCTAL ADENOCARCINOMA PATIENTS AND CAN BIND TO VEGF AND HIF-1ALPHA MRNA

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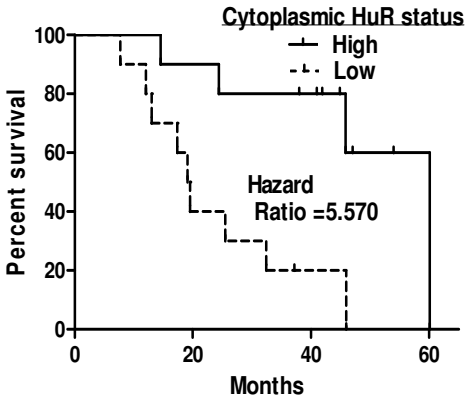
Introduction: Treatment of pancreatic ductal adenocarcinoma (PDA) typically includes chemotherapy with gemcitabine. No reliable biomarker exists for overall prognosis or response to chemotherapy. Two previously proposed prognostic markers, COX-2 and VEGF, are regulated by HuR, an mRNA binding protein that we demonstrated is a promising predictive marker of gemcitabine response (*Cancer Research* 2009, 69:4567-72). This study evaluates a clinically useful biomarker for PDA and explores the association of HuR to oncogenic mRNA target genes, HIF-1α and VEGF.

Methods: A tissue microarray of 53 PDA specimens, who underwent potentially curative resection, was analyzed. HuR, COX-2, and VEGF status were compared and correlated with clinical data. Human pancreatic cancer cells were treated with gemcitabine for 6 hours and cytoplasmic extracts were collected. RT-qPCR was performed using bound RNA immunoprecipitated (RNP-IP) from an HuR or IgG antibody. Relative quantification of VEGF and HIF-1α mRNA was assessed.

Results: Roughly 50% (27/53) of patients had elevated cytoplasmic HuR expression (HuR+). These patients had worse pathologic features (i.e. positive lymph nodes (75%) and AJCC pathologic stage 2 or greater (94%)) compared to HuR- patients. Cytoplasmic HuR status correlated with staging better than VEGF or COX-2 expression alone. HuR cellular positivity with VEGF+ status yielded 100% lymph node positivity. HuR status was a robust positive predictive marker for overall survival in patients treated with gemcitabine pushing median survival over 40 months in the HuR+ patient population (p-value 0.0049). RT-qPCR and RNP-IP demonstrated that after gemcitabine treatment, VEGF and HIF-1α mRNA transcripts are selectively bound to HuR compared to a control by over 20 and 14 fold, respectively.



Conclusion: HuR associates with VEGF and HIF-1α, key proteins in pancreatic tumorigenesis, and likely regulates their expression. HuR status is a robust predictor of outcome for patients with resected PDA and should be used by clinicians to individualize treatment.

Session 3: 9:15 AM

DPC4 STATUS IS CORRELATED WITH TUBULAR MORPHOLOGY OF INVASIVE CARCINOMA ASSOCIATED WITH INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM OF THE PANCREAS, BUT NOT WITH LYMPH NODE STATUS.

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Introduction/Background
Intraductal papillary mucinous neoplasms (IPMNs) are neoplasms of the pancreas with clear malignant potential but with an improved 5-year survival compared to conventional duct adenocarcinomas. Two distinct types of invasive carcinoma commonly occur in association with IPMN: the tubular type, which typically arises from pancreatobiliary type IPMN and resembles standard pancreatic adenocarcinoma; and the colloid (mucinous noncystic) type, which is typically arises from intestinal type IPMN, and is characterized by extensive stromal pools of extracellular mucin. The goal of this study was to compare clinicopathologic features and genetic status of Dpc4 with adenocarcinomas associated with IPMN.
Methods
Fifty-five patients who underwent surgical resection for tubular adenocarcinoma or colloid carcinoma arising in association with an IPMN at the Johns Hopkins Hospital were used. DPC4 immunohistochemistry was performed on paraffin sections of each carcinoma, and correlated to the clinicopathologic features of each patient.
Results
The mean age of all patients was 68.1±10.1 years and twenty-six patients (47%) were male. The mean IPMN size was 4.3±2.7cm and for infiltrating carcinomas was 3.0±2.0cm. Thirty-seven patients (67%) had lymph node metastases. Infiltrating carcinomas showed a tubular morphology in 75% of patients (41/55, 67.8±9.0 years and male in 44%) and colloid morphology in 25% (14/55, 68.4±13.0 years and male in 57%) of patients. While there was a significant difference between the size of IPMNs associated with a tubular versus colloid carcinoma (P=0.0124), there was no difference in the size of tubular versus colloid carcinomas specifically in this group of patients (P=0.2331). Lymph node metastases were found in 73% (30/41) and 50% (7/7) of tubular and colloid carcinomas respectively (P= 0.1106). Loss of DPC4 was more frequent among tubular versus colloid carcinomas (24/41 tubular versus 0/14 colloid, P= 0.00006). However, among tubular carcinomas specifically, there was no relationship of DPC4 to the presence of lymph node metastasis.

Conclusion
DPC4 loss is associated with the development of tubular carcinoma arising in an IPMN, but additional factors may be related to the development of lymph node metastasis specifically from IPMN associated tubular carcinomas.

Session 3: 9:30 AM

REPRESSION OF E-CADHERIN BY THE POLYCOMB GROUP PROTEIN EZH2 IN PANCREATIC CANCER

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Background: Pancreatic ductal adenocarcinoma (PDA) is the fourth leading cause of cancer deaths in the United States. Single-agent gemcitabine remains the standard treatment for advanced PDA, which has shown improvement in disease-related symptoms and a modest benefit in survival. A recently discovered histone methyltransferase termed enhancer of zeste homologue 2 (EZH2) was found to be overexpressed in a variety of carcinomas including PDA. Silencing of E-cadherin was proposed as a mechanism by which EZH2 mediates tumor aggressiveness. Furthermore, in-vitro studies showed EZH2 depletion sensitizes pancreatic cancer cells to gemcitabine. In this study we correlated EZH2 with E-cadherin expression in PDA, and evaluated response to gemcitabine in relation to EZH2 expression.

Methods: 43 PDAs, 14 intraductal papillary mucinous neoplasms (IPMNs), and 5 chronic pancreatitis (CP) cases were stained with EZH2 (BD Bioscience; 1:25) and E-cadherin (Zymed; 1:1,000). Cases with diffuse weak staining, or strong staining in less than 30% of tumor nuclei were considered to have low EZH2 expression. High EZH2 expression was defined as strong nuclear staining in >30% of tumor cells. E-cadherin expression was scored on membrane positivity as follows: 0 (0-10%); 1 (10–25%); 2 (25-75%), and 3 (>75%). E-cadherin scores were considered normal at 3, reduced at 2, and negative at 1 or 0. Statistical analysis was performed using Fisher's exact and Kruskal-Wallis tests, depending on the discrete or continuous nature of the other factors. A Kaplan-Meier curve was stratified by EZH2 expression to assess survival.

Results: High EZH2 expression in PDA was significantly associated with decreased E-cadherin expression (70% vs. 35%), node-positivity (82% vs. 40%), and larger tumor size (4 cm vs. 2.4 cm). There was a trend for longer survival (35 vs. 15 months) in gemcitabine treated patients with low vs. high EZH2 expression. High EZH2 expression was detected in IPMN with moderate-severe dysplasia, however not in CP.

Conclusion: Our study suggests that E-cadherin downregulation may lead to EZH2-mediated invasion and metastasis. While strong diffuse EZH2 expression is seen in PDA, overexpression may be present in IPMN.

Session 3: 9:35 AM

INTRADUCTAL MUCINOUS PAPILLARY NEOPLASMS: GENETIC CHARACTERIZATION OF LESION PROGRESSION.

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Background: The timing and choice of appropriate surgical treatment for intraductal papillary mucinous neoplasm (IPMN) remains a major clinical challenge. Current evidence clearly supports the dysplasia to carcinoma sequence of malignant transformation in IPMN at unknown rates of progression. This study investigates the changes in gene expression that occur in IPMNs during their progression from low grade to high grade dysplasia and on to invasive carcinoma.

Methods: IPMN cases were identified from the pathology archives treated by a single surgeon (RJ). Foci of different grades of dysplasia and invasion, from the same specimens where possible, were identified by a pathologist and marked on the cover slip of the slide. Serial sections were cut and processed for laser capture microdissection. Areas of interest ($1 – 2\ mm^2$), were dissected representing ~5,000 cells captured. Captured tissue was subjected to RNA extraction using the NuGEN WT-Ovation™ FFPE System. The extracted RNA was analyzed for integrity and hybridized to Affymetrix Human Exon 1.0 ST arrays using proprietary procedures. Gene expression data were normalized and filtered using GCOS software and analyzed using Expression Console software and statistical analysis.

Results: 96 genes were identified that were differentially expressed within lesions of varying dysplastic change and invasion from the same IPMN patient. Table 1 shows the top 20 genes overexpressed in areas of invasive IPMN compared to low grade disease.

Discussion: This study identifies a set of pancreas-specific genes associated with the progression of IPMN to malignancy within the same patient as well as changes in some unrelated genes. Many of the overexpressed genes lead to production of enzymes with capacity to break down connective tissue potentially allowing invasion. Further analysis of different mucinous lesions to uncover the genes most commonly associated malignant progression will ultimately help in the choice of appropriate and timely surgical therapy.

Gene symbol	Gene product
PLA2G1B	phospholipase A2, group IB (pancreas)
GP2	glycoprotein 2 (zymogen granule membrane)
CPA1	carboxypeptidase A1
PNLIPRP2	pancreatic lipase-related protein 2
INS	insulin
ZNF30	zinc finger protein 30
CLPS	colipase, pancreatic (CLPS)
REG3A	regenerating islet-derived 3 alpha
ELA2B	elastase 2B
REG1P	regenerating islet-derived 1 pseudogene
CPA2	carboxypeptidase A2 (pancreatic)
REG1A	regenerating islet-derived 1 alpha
ELA2A	elastase 2A
SFRP2	secreted frizzled-related protein 2
PNLIPRP1	pancreatic lipase-related protein 1
APCS	amyloid P component, serum
GABRP	gamma-aminobutyric acid (GABA) A receptor, pi
CRISP3	cysteine-rich secretory protein 3
CTRC	chymotrypsin C (caldecrin)
CD163	CD163 molecule

Table 1. Top 20 genes associated with progression to invasive lesions

Session 3: 9:40 AM

LOSS OF HETEROZYGOSITY (LOH) STATUS OF D9S105 MARKER IS ASSOCIATED WITH DOWN-REGULATION OF KRUPPEL-LIKE FACTOR 4 (KLF4) EXPRESSION IN PANCREATIC DUCTAL ADENOCARCINOMA AND PANINS

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Introduction/Background. Homozygosis deletion of 9q31-32 has been associated with Kruppel-*Like Factor* 4 (KLF4) suppression placing this gene as putative tumor suppressor gene in gastric, bladder and colorectal cancer. However, Ectopic $Kras^{V12}$ gene mutation can promote KLF4 as oncogene *in vitro*. Furthermore, experimental data suggest that KLF4 may be over-expressed or down-regulated in Pancreatic Ductal Adenocarcinoma (PDAC) and its role in this pathology is unclear. This study was aimed at evaluating the association between loss of 9q31-32 region and gene expression of KLF4 and to evaluate the role of this gene in PDAC.

Methods. We investigated the Loss of Heterozygosity (LOH) in the 9q region and the expression of KFL4 gene in PDAC, PanINs, Normal Ducts and Primary Cell Culture of PDAC (PCC). Epithelial cells from 35 PDAC, 6 Pancreatic Intraepithelial Lesions (PanINs) and 6 normal ducts were isolated by laser microdissection (Leica LMD 7000) for molecular analyses. We used 4 microsatellite markers (D9S127, D9S53, D9S105, D9S106), flanking KLF4 locus to test the LOH, while mRNA gene expression was performed by RT-PCR, both in PDAC and PanINs.

Results. LOH in at least one locus was observed in *25/35* PDAC cases and in *5/6* PanINs respectively. We identified a Small Region of Deletion Overlap (SRDO) in 52.9% of PDAC that presented LOH in both D9S53 and D9S105 markers. In 46.9% of PDAC and 83.3% of PanIN lesions there was a loss of the D9S105 marker, which resulted to be the most deleted marker. Five cases showing homozygosis deletion of D9S105 had not amplified PCR product for KLF4. In contrast, no LOH in D9S105 marker was observed in normal pancreatic duct cells and in PCC. The PCC wild-type for D9S105 marker had mutated in K-ras gene at codon 12 and expressed KLF4 and. Lack of KLF4 expression was found to be significantly associated with: 1) Genomic deletion flanking KLF4 in PDAC ($p=0.018$) and in PanINs ($p<0.01$); 2) LOH of D9S105 marker ($p=0.014$); 3) Presence of low–grade of PDAC-associated PanIN ($p=0.021$).

Conclusion. We identified a relation between D9S105 deletion and loss of KLF4 expression in PDAC. Our results suggested that the KLF4 gene can switch its role between tumor suppressor gene and oncogene depending on the biological context of PDAC.

Session 4: 10:00 AM

PREOPERATIVE FACTORS PREDICT MORBIDITY AFTER PANCREATICODUODENECTOMY: CREATION OF A NSQIP NOMOGRAM

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Introduction: Pancreaticoduodenectomy (PD) has long been associated with high rates of morbidity and mortality. Several high-volume centers have reported markedly improved outcomes with the procedure. The objective of this study was to identify preoperative risk factors for serious complications after PD, and to construct a risk-stratification nomogram.

Methods: Patients who underwent elective PD from 2005-2007 were identified from the American College of Surgeons National Surgical Quality Improvement Project (NSQIP) Participant Use Data Files. Multivariate logistic regression identified predictors of serious complications and adjusted odds ratios (ORs) were calculated. A risk-stratification nomogram was created and validated using 2008 NSQIP data.

Results: Of 2,750 patients who underwent PD, 746 (27.1%) suffered a serious complication within 30-days. The most frequent complications were sepsis (16.9%), surgical site infections (12.5%), and respiratory complications (9.9%). Rates of 30-day reoperation and mortality were 7.4% and 2.8%, respectively. The mortality rate was significantly higher in patients who had a serious complication (9.4% vs. 0.4%, $p<0.001$). After adjusting for preoperative risk factors, significant predictors of morbidity included old age, high body mass index, chronic obstructive pulmonary disease, peripheral vascular disease, bleeding disorder, disseminated cancer, and low serum albumin. These variables were used to construct a preoperative risk-stratification prediction tool.

Conclusion: In this sample drawn from over 180 academic and community hospitals nationwide, more than one in four patients suffered a serious complication after PD. Complications were associated with high rates of 30-day reoperation and mortality. Preoperatively-determined patient-specific risk factors predicted perioperative morbidity. The resulting nomogram may be used to help estimate the risk of complications for patients undergoing PD, as well as for risk adjustment when comparing surgical outcomes.

Table: Significant predictors of 30-day serious complication after pancreaticoduodenectomy. ORs are adjusted for all variables which differed significantly in univariate analysis between the complication and no complication groups.

Characteristic	Adjusted OR (95% CI) for Serious Complication
Age 80 years and older	1.89 (1.25 - 2.86)
BMI 30 to 49 kg/m^2 (obese)	1.46 (1.14 - 1.88)
COPD	1.67 (1.05 - 2.65)
PVD	2.62 (1.19 - 5.79)
Bleeding disorder	2.42 (1.34 - 4.39)
Disseminated cancer	1.89 (1.10 - 3.26)
Albumin < 2.5 g/dL	2.02 (1.23 - 3.32)

Session 4: 10:15 AM

PANCREATECTOMY RISK CALCULATOR: AN ACS-NSQIP RESOURCE

Parikh, Purvi, Shiloach, Mira, Cohen, Mark E, Bilimoria, Karl Y, Ko, Clifford Y, Hall, Bruce L, Pitt, Henry A

BACKGROUND: The morbidity of pancreatoduodenectomy remains high, and the mortality may be doubled or tripled in high-risk patients. However, a method to predict postoperative adverse outcomes based on readily available clinical data has not been available.

OBJECTIVE: To create a "Pancreatectomy Risk Calculator" using the American College of Surgeons-National Surgical Quality Improvement Program (ACS-NSQIP) database.

METHODS: The 2005-08 ACS-NSQIP data on 7,571 patients undergoing proximal (n=4,621), distal (n=2,552), total pancreatectomy (n=177) or enucleation (n=221) were analyzed. Preoperative variables (n=31) were assessed for prediction of postoperative mortality, serious morbidity, and overall morbidity using a logistic regression model. Statistically significant variables were ranked and weighted to create a risk model for all three outcomes.

RESULTS: Twenty preoperative variables were statistically significant predictors of postoperative mortality (2.5%), serious morbidity (21%), or overall morbidity (32%). Ten of 20 significant preoperative variables were employed to produce a mortality and morbidity risk model (see Table).

Table. Pancreatectomy Risk Calculator

Variable	30-Day Mortality*	Serious Morbidity*	Overall Morbidity*
ASA Class 3	2.33	1.18	1.19
Functional Status	3.27	1.73	1.75
Preop Sepsis	2.26	2.26	2.10
Surgery Extent†	1.62	1.29	1.36
Age > 74	2.28	1.30	1.26
Dyspnea §	1.72	1.38	1.36
BMI > 40	2.32	1.83	1.60
Cardiac Surgery△	1.18*	1.20	1.36
Gender – Male	1.16*	1.22	1.13
Bleeding Disorder	1.15*	1.43	1.68

* Odds Ratios all p<0.05 except three forced variables indicated
† Proximal vs distal pancreatectomy
§ With moderate exertion △ Prior major procedure

CONCLUSIONS: The ACS-NSQIP "Pancreatectomy Risk Calculator" employs ten easily assessable clinical parameters to assist patients and surgeons in making an informed decision regarding the advisability of undergoing pancreatic resection. This model will become available as on online ACS-NSQIP resource.

Session 4: 10:30 AM

BRAIN NATRIURETIC PEPTIDE (BNP) AND POSTOPERATIVE FLUID BALANCE IN THE MANAGEMENT OF PATIENTS UNDERGOING PANCREATECTOMY

BERRI RN, LIN H, FOLLODER J , PISTERS PWT, ABDALLA EK, VAUTHEY JN, LEE JE, AND FLEMING JB.

Background: Postoperative fluid management in patients receiving major intra-abdominal operative procedures remains a clinical dilemma. Conventional methods of fluid management are rarely endpoint driven which can lead to errors resulting in preventable morbidity. Recent publications have suggested that the serum levels of Brain Natriuretic Peptide (BNP) reflect the effective blood volume during short-term changes in fluid status. We hypothesized that serial measurements of BNP would accurately reflect daily changes in intravascular fluid status after pancreatectomy.
Methods: We prospectively collected data including, age, gender, serum BNP, and serum creatinine. Using immunoassay methods, serum BNP was measured at baseline in sequential patients receiving pancreatectomy at a high volume center. Serial BNP measurements were then obtained every 24 hours for seven days postoperatively. The total fluid intake and total output (measured in cc) were measured and values obtained from patient care records. Preoperative medical and cardiac evaluations performed at our institution were also recorded. Linear mixed effect statistical models were used to study the change of BNP and fluid balance over time separately and to then take the correlation between these measures at different time points within each individual patient. Bivariate random effect models were used to examine the correlation between the time trend of the BNP and the time trend of fluid balance.
Results: Serial BNP measurements were obtained in 44 patients receiving pancreatectomy. A reproducible absolute and percent decline in serum BNP from the day of operation to postoperative day 7was observed in all patients. Furthermore, a reproducible pattern of BNP decline in postoperative days one to two and three to seven was present. When comparing this decline in BNP to fluid balance specifically, the changes in BNP levels accurately reflected the positive fluid balance that occurs after pancreatectomy and the % change in BNP was highly correlated ($\rho = 0.91$, p-value = 0.039) with fluid balance over the first three postoperative days. A predictable daily % change in BNP was observed during the postoperative period for all patients, but those with evidence of diastolic cardiac dysfunction on preoperative echo were less likely to follow the anticipated pattern of BNP change.
Conclusions: The results demonstrated a predictable pattern of serum BNP in postoperative period. Furthermore, his pattern accurately reflects the fluid balance of the patient. Together, these observations, suggest that daily serum BNP measures could be used to measure the effective intravascular volume after pancreatectomy and cardiac dysfunction may affect the observed patterns of BNP change.

DIFFERENCES IN METHYLATION OF CELL-FREE CIRCULATING DNA IN PATIENTS WITH PANCREATIC CANCER AND CHRONIC PANCREATITIS

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Background. Patients with chronic pancreatitis (CP) have higher risk of pancreatic cancer (PanCa), but the timely detection of PanCa is often difficult because of the similarity of symptoms of CP and PanCa. In addition, secondary inflammation may be present in PanCa, making diagnosis even more difficult. A reliable test to differentiate CP from PanCa may improve survival of PanCa patients by allowing quicker detection of PanCa. In this communication, we describe a DNA methylation pattern in cell-free plasma DNA that can distinguish CP from PanCa with over 90% accuracy.

Methods. Methylation in cell-free plasma DNA from blood was compared among 30 samples from CP patients, 30 samples from patients with PanCa, and 30 healthy controls (N) using a microarray-mediated methylation analysis of 56 fragments in each sample (MethDet56). Statistical analysis was done by Fisher's Exact test, naïve Bayes algorithm, and 25 rounds of five-fold cross-validation.

Results. Using MethDet56 technique for methylation analysis, 17 gene promoters were found to be informative (8 for distinguishing normal from CP; 14 for distinguishing CP from PanCa), achieving 81.7% sensitivity and 78.0% specificity ($p<0.01$) in detection of CP (N vs. CP), and 91.2% sensitivity and a 90.8% specificity ($p<0.01$) in differential detection of PanCa (PanCa vs. CP).

Conclusions. Our data suggest that patients with pancreatic illness have different methylation patterns for inflammatory disease and cancer that can be detected in blood. New and improved biomarkers for differential diagnosis of these diseases appear to be possible, and further investigation of diagnostic biomarkers for pancreatic cancer based on methylation in cell-free circulating DNA is warranted.

Session 4: 10:50 AM

THE BURDEN OF INFECTION FOR ELECTIVE PANCREATIC RESECTIONS

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Background: Because mortality rates for pancreatic resection have fallen, other valid measures of surgical quality are needed. While infection control is a critical surrogate quality indicator, it remains incompletely understood, especially in high-acuity GI surgery. We therefore evaluated the incidence and impact of infections after our elective pancreatic resections.
Methods: All pancreatic resections followed standardized perioperative care, including timely administration of antibiotics. Infections were classified according to NSQIP definitions, while complication severity was based on Clavien criteria. Clinical and economic outcomes were evaluated and predictors of infection were identified by regression analysis.
Results: Of 550 pancreatic resections (356 Whipple, 167 Distal, 11 Total, 16 Other), 288 (53%) had some complication, of which 167 (31%) were infectious. Rates of infection differed by type of resection (p=0.029) but not by the presence of malignancy. There was a trend toward increased infection in patients stented preoperatively (41.4% vs 32.8%, p=0.054). While most infections were minor (n=105, Clavien 1-2), major infections (n=62, Clavien 3-5), occurred in 11%. Patients with infection did significantly worse, with longer LOS and OR time, more transfusions, ICU use, and greater EBL. They were readmitted more often (34% vs 12%). Common organisms were Staphylococcus, Enterococcus, and E. coli. By category, wound infection (14%) was most common, followed by infected fistula (9%), UTI (7%), pneumonia (6%), and sepsis (2%). 48/72 clinically relevant fistulae involved polymicrobial infection and occurred equivalently for Whipple and distal fistulae. TPN use (Odds Ratio 7.3), coronary artery disease (OR 2.1), and perioperative hypotension (OR 1.6) predicted any infection, but specific categories of infection had different predictors. Total costs increased grade-for-grade across the Clavien scale, with infection accounting for 50% of the cost differential.

Table 1: Cost of care by severity of infectious complication.

	Infectious Complications (%)	**Total cost** Non-infected Cases	**Total cost** Infected Cases	**Cost differential**
All resections	58.0	$25,197	$40,533	$15,336
No complication	N/A	$25,082	N/A	
Clavien 1	56.7	$26,831	$27,599	$768
Clavien 2	41.8	$24,406	$26,094	$1,688
Clavien 3	65.2	$27,085	$36,224	$9,139
Clavien 4	80.6	$20,763	$101,253	$80,490
Clavien 5 (Death)	37.5	$24,082	$76,755	$52,673
p-value	0.001		<0.0001	

Conclusion: Nearly one-third of patients undergoing pancreatic resections experienced infections. Depending on severity, clinical outcomes suffered and costs rose significantly. These data are guiding process evaluations and initiatives for infection control in our unit

SUPPORT FOR A POSTRESECTION PROGNOSTIC SCORE FOR PANCREATIC ENDOCRINE TUMORS

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Introduction/Background: While the incidence of pancreatic adenocarcinoma has remained stable, the incidence of pancreatic neuroendocrine tumors (PNETs) has increased. The natural history of these tumors is poorly defined, and limited information is available with regards to factors affecting survival after resection. Recently, prognostic scores predicting long term survival of patients with PNETS have been created. The purpose of this study was to validate a currently accepted prognostic scoring scheme at a single institution.

Methods: All patients who underwent resection for pancreatic tumors at a single institution from 1996 – 2004 were reviewed. Tumors in which the final pathological diagnosis was of a PNET were further studied. Clinicopathological and survival data were collected on each patient. A prognostic scores based on patient age, tumor grade, and presences of metastases was calculated for each patient. Survival was compared to an established post-resection prognostic score for PNETs.

Results: A total of 34 PNETs were identified. Since the beginning of 2000, a 50% increase in surgical resections for PNETs was observed. Although, increasing in incidence, PNETs were relatively rare, and accounted for approximately 3% of all pancreatic neoplasms found. Prognostic scores are found in table 1.

Table 1. PNET Postresection Prognostic Score Categories.

Prognostic Score	Raw Score	No. Patients	5-yr Survival actual	5-yr Survival (predicted)
1*	0	13 (38.2‰)	100‰	76.7‰
2*	1-2	18 (52.9‰)	66.7‰	50.9‰
3*	≥3	3 (8.8‰)	66.7‰	35.7‰

*score calculated based on age, grade, and metastases. Scoring for age: <55 years = 0 points, 55-75 years =1 point, >75 years = 2 points. Scoring for grade: well/moderately differentiated = 0 points, poorly differentiated = 1 point. Scoring for distant metastases: none = 0 points, liver = 1 pointt, distant = 3 points.

Discussion/Conclusion: Patients with PNET prognostic scores of 1 had better overall survival when compared to those with prognostic scores of 2 or 3. PNET post- resection prognostic score categories recently established may be a useful tool in prediction of long term survival of patients suffering from PNETs.

Session 5: 1:00 PM

ADIPOCYTES IN THE TUMOR MICROENVIRONMENT PROMOTE DISSEMINATION OF HUMAN PANCREATIC CANCER

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Background: Epidemiologic studies have established associations between obesity and several malignancies. Human studies also have demonstrated that obesity accelerates pancreatic cancer development and progression. We recently reported that increased pancreatic neck fat is correlated with increased mortality and dissemination in human pancreatic cancer (JACS 2009, 208: 989-94). However, the adipocyte content of the pancreatic tumors themselves was not analyzed. Therefore, we hypothesized that adipocytes in the tumor microenvironment would be associated with increased dissemination and reduced survival in patients with resected pancreatic cancer.
Methods: A case-control analysis was conducted in patients who had undergone resection for pancreatic adenocarcinoma. Twenty lymph node positive patients and twenty node negative patients were matched for age (59 vs. 63 years), gender (70% male vs. 60% male), BMI (24.5 vs. 25.6 kg/m2), medical comorbidities (hypertension, diabetes, hyperlipidemia), tumor size (2.8 vs. 2.6 cm), resection status (Ro-80% vs. 85%), and neural invasion (100% vs. 85%). Pancreatic tumor sections were reviewed for percentage fat, tumor, and fibrosis (including vessels and nerves) in a blinded fashion by two trained investigators (percentage of field/ 10 high-powered fields). Furthermore, the percentage of patients with adipocytes in the tumor microenvironment was determined. Data were analyzed by Wilcoxian rank-sum and Fisher's exact test as appropriate.
Results: Percentages of fat, tumor, and fibrosis in the two groups are presented in the table. More patients with adipocytes in the tumor microenvironment were observed in the node positive than in the node negative group (79% vs. 37%, $p<0.03$).

Lymph Node	% Fat	% Tumor	% Fibrosis
Negative	2.4±0.8	41.8±2.4	56.1±2.5
Positive	4.7±0.7 *	39.7±2.5	55.5±2.4

*P<0.05 vs. Node Negative

Conclusion: These data suggest that adipocytes within the tumor microenvironment promote the dissemination and lethality of pancreatic cancer. This analysis confirms and extends our previous observation that pancreatic steatosis enhances tumor spread and contributes to increased mortality in patients with pancreatic adenocarcinoma.

Session 5: 1:15 PM

LOW DOSE METRONOMIC GEMCITABINE HAS HIGH ANTIMETASTATIC EFFICACY IN AN ORTHOTOPIC MOUSE MODEL OF PANCREATIC CANCER

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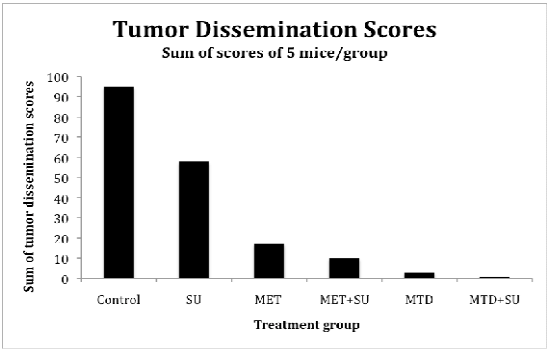
Introduction: Therapies targeting metastasis of pancreatic cancer (PC) are ineffective. Low dose metronomic chemotherapy suppresses primary tumor growth, but its antimetastatic efficacy is unknown. We report the efficacy of metronomic Gemcitabine with and without tyrosine kinase inhibitor Sunitinib on metastasis in an orthotopic model of PC

Methods: Mice with highly metastatic, orthotopic PC tumorgrafts expressing red fluorescent protein were treated with intraperitoneal Gemcitabine on a metronomic (1 mg/kg daily, MET) or maximum tolerated dose (150 mg/kg twice weekly, MTD) schedule, with or without oral Sunitinib (SU). Rates of metastasis and primary tumor growth were quantified by fluorescence imaging. Survival was also evaluated.

Results: Control mice with orthotopically-implanted Mia-PaCa-2-RFP fluorescent tumorgrafts died of extensive disease within 4 weeks. Treatment of mice with established tumorgrafts with MET for 2 weeks suppressed metastasis at multiple sites – an effect enhanced by SU ($p<0.05$). In contrast, primary tumor growth was inhibited by MET+SU ($p<0.05$) but not by either MET or SU alone. In a survival study, both MET and SU had a modest effect on survival compared to control, but survival was limited by extensive primary tumor growth (med. survival 32d, 31d, 25d, respectively). MET+SU had a more pronounced effect on survival (44 days, $p<0.05$). MTD, with or without SU, had the most pronounced effects on primary tumor growth and survival, but its antimetastatic effect was similar to that of MET+SU ($p<0.05$). Staining of primary tumors for vWF revealed an antiangiogenic effect of therapy.

Conclusions: Antimetastatic activity approaching that of standard MTD Gemcitabine is achieved with a total Gemcitabine dose reduced 42 times using MET and is further enhanced by Sunitinib. Our results suggest the clinical potential of this well-tolerated regimen against PC in the adjuvant and maintenance settings.



Scored by evaluating metastases as 1 (microscopic) to 4 (large, macroscopic) at multiple sites.

Session 5: 1:30 PM

TUMOR SUPPRESSOR, ANP32A, DISRUPTS HUR'S REGULATION OF DEOXYCYTIDINE KINASE IN PANCREATIC CANCER: IMPLICATIONS FOR GEMCITABINE THERAPY.

Agnieszka Witkiewicz[1,2,3] Timothy K. Williams[1], Christina L. Costantino[1], Nathan G. Richards[1], Nikolai A. Bildzukewicz[1], Lisa Einstein[1], Joseph Cozzitorto[1], Christine Hostetter[2], Judith C. Keen[2], Abhijit Dasgupta[3], Charles J. Yeo[1], Jonathan R. Brody[1,2,3]
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Introduction: pp32 (ANP32A) has reduced expression in poorly differentiated pancreatic cancers and can interact with HuR (ELAV1), a predictive marker for gemcitabine response.
Methods: pp32 cDNA under a strong CMV promoter were stably transfected into multiple human pancreatic cancer cell lines. Confirmation of pp32 exogenous overexpression was performed by semi-quantitative RT-PCR and western blot analysis. Cell growth and drug sensitivity assays were used to test the effect of pp32 overexpression on pancreatic cancer cells. Immunofluorescence and immunoblots assessed subcellular localization (cytoplasmic vs. nuclear) of HuR and pp32 after drug exposure. Ribonucleoprotein immunoprecipitation (RNP-IP) with HuR antibody on overexpressing pp32 cells surveyed the binding of HuR with deoxycytodine kinase (dCK) mRNA, the gemcitabine activating enzyme. Immunohistochemistry was performed on 37 resected PDA specimens who had received either gemcitabine alone or in combination with other treatments or no treatment. Clinical data was collected and correlated with pp32 and HuR subcellular localization and expression levels.
Results: In pancreatic cancer cells (MiaPaCa2 cells), exogenous overexpression of pp32 caused an inhibition of growth compared to control cells. We were unable to maintain stable pp32 overexpression in other pancreatic cancer cell lines, further validating pp32's role as a tumor suppressor in pancreatic cancer. Nuclear to cytoplasmic transport of pp32 occurred upon certain stressors, including gemcitabine. In chemotherapeutic sensitivity assays, overexpressing pp32 cells demonstrated a dramatic resistance to nucleoside analogs, gemcitabine (ten-fold resistance) and cytarabine (ARA-C), yet a sensitivity to 5-fluorouracil. Accordingly, pp32 siRNA silencing in the pancreatic cancer cell line, PL5, caused gemcitabine sensitivity compared to control cells. We sensitized overexpressing pp32 cells to gemcitabine by irradiating cells prior to chemotherapeutic exposure. Mechanistically, in pp32 overexpressing cells, we detected a reduced association of HuR to the mRNA of dCK. Thus, pp32 overexpression directly reduced dCK protein levels. In tumor specimens from patients, we found that low pp32 nuclear expression in pancreatic cancer cells correlated with high-grade tumors and the presence of lymph node metastasis, as compared to patients with high nuclear pp32 expression. Yet, pp32 expression levels did not enhance the predictive power of HuR cytoplasmic status. We also found a statistically significant correlation between pp32 and HuR expression levels in PDA specimens.
Conclusion: These data (a) provide the first evidence that HuR and pp32 collaborate to regulate gemcitabine efficacy and (b) aid in our understanding of pp32's tumor suppressive function. Thus, therapeutic disruption of the pp32/HuR mRNA stability network may be a logical approach for the treatment of pancreatic cancer.

Session 5: 1:45 PM

INDUCTION OF MONOCYTE CHEMOATTRACTANT PROTEIN-1 BY NICOTINE IN PANCREATIC DUCTAL ADENOCARCINOMA CELLS: ROLE OF OSTEOPONTIN

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Introduction/Background: Cigarette smoke and nicotine are among the leading environmental risk factors for developing pancreatic ductal adenocarcinoma (PDA). We showed recently that nicotine induces osteopontin (OPN), a protein that plays critical roles in inflammation and tumor metastasis. We identified an OPN isoform, OPNc, that is selectively inducible by nicotine and highly expressed in PDA tissue from smokers. In this study, we explored the potential proinflammatory role of nicotine in PDA through studying its effect on the expression of monocyte chemoattractant protein- (MCP)-1 and evaluated the role of OPN in mediating these effects.

Methods: MCP-1 mRNA and protein in PDA cells treated with or without nicotine (3-300 nM) or OPN (0.15-15 nM) were analyzed by real time PCR and ELISA. Luciferase-labeled promoter studies evaluated the effects of nicotine and OPN on MCP-1 transcription. Intracellular and tissue colocalization of OPN and MCP-1 were examined by immunofluorescence and immunohistochemistry.

Results: Nicotine treatment significantly increased MCP-1 expression in PDA cells. Interestingly, blocking OPN with siRNA or OPN antibody abolished these effects. Transient transfection of the OPNc gene in PDA cells or their treatment with recombinant OPN protein significantly ($P<0.05$) increased MCP-1 mRNA and protein and induced its promoter activity. MCP-1 was found in 60% of invasive PDA lesions, of which 66% were smokers. MCP-1 colocalized with OPN in PDA cells and in the malignant ducts, and correlated well with higher expression levels of OPN in the tissue from patients with invasive PDA.

Discussion/Conclusions: Our study suggests that cigarette smoking and nicotine may contribute to PDA inflammation through inducing MCP-1 and provides a novel insight into a unique role for OPN in mediating these effects. Although the role of OPN-mediated induction of MCP-1 in pancreatic carcinogenesis remains to be defined, the existence of OPN as a downstream effector of nicotine, capable of mediating proinflammatory effects in PDA cells is novel and could provide a unique potential target to control pancreatic cancer aggressiveness, especially in the cigarette smoking population.

Session 5: 2:00 PM

A MOLECULAR LINK BETWEEN EPITHELIAL-MESENCHYMAL TRANSITION AND CANCER STEM CELL PROPERTIES IN PANCREATIC CANCER

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Introduction: The process of epithelial-mesenchymal transition (EMT) has been implicated in cancer cell invasion and metastasis. Recent studies have also suggested a link between EMT and the acquisition of Cancer Stem Cell (CSC) properties.

Methods: Human pancreatic cancer cell lines were obtained from the ATCC. Pancreatic cancer tissue and follow-up data was obtained from patients operated at our institution. Gene and micro-RNA expression analysis was done by RT-PCR, Immunofluorescence and Western Blot. In vitro assays comprise tumor sphere formation, FACS, matrigel invasion, growth inhibition by cytotoxic agents, and reporter assays for functional gene regulation studies. In vivo experiments were orthotopic xenograft model and limiting dilution assay with s.c. injection.

Results: We show that in human pancreatic cancer, the transcriptional repressor ZEB1 can mediate EMT, which is also associated with the acquisition of some important properties of CSC and a worse prognosis for patients. Furthermore, we propose one molecular mechanism for this link between EMT and CSC phenotype, namely the repression of stemness-inhibiting micro-RNAs by ZEB1.

Conclusion: EMT seems to be associated with the acquisition of a CSC phenotype also in human pancreatic cancer. On the molecular level, suppression of stemness-inhibiting microRNAs by ZEB1 may play an important role in this process.

Session 5: 2:15 PM

ADIPOCYTES PROMOTE PANCREATIC CANCER PROLIFERATION VIA A HEPATOCYTE GROWTH FACTOR-MEDIATED MECHANISM

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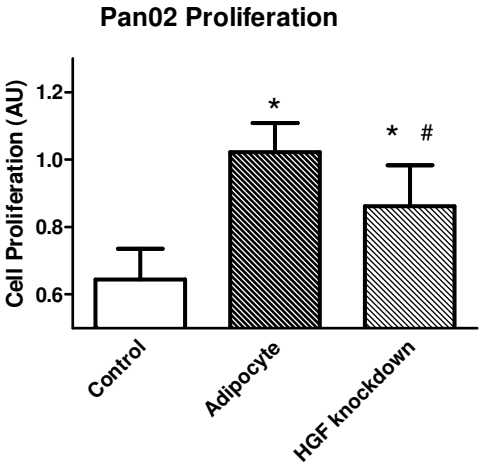
BACKGROUND: Obesity clearly accelerates the development and progression of pancreatic cancer, though the mechanisms underlying this association are unclear. Adipocytes are biologically active, producing factors such as hepatocyte growth factor (HGF) that may influence tumor proliferation. We hypothesized that adipocyte-secreted factors would accelerate pancreatic cancer proliferation.
METHODS: Murine pancreatic cancer (Pan02) cells were exposed to conditioned media (CM) from a) wild type F442A adipocytes, b) adipocytes with the HGF gene knocked down using siRNA, or c) Pan02 (control). Cellular proliferation was evaluated using the standard MTT assay. The response of Pan02 cells to increasing doses of exogenous HGF was also measured. Real Time (RT)-PCR was performed to evaluate the presence of the HGF receptor, c-met. Finally, the HGF receptor, c-met, was blocked with the specific inhibitor, PF-2341066, and Pan02 proliferation in response to adipocyte CM was measured by MTT. ANOVA and t-test were applied where appropriate, with $p<0.05$ considered significant.
RESULTS: Wild-type adipocyte CM increased Pan02 proliferation by 68% relative to control ($p<0.05$); adipocyte HGF knockdown CM decreased this proliferation by 49% (Figure). Exogenous HGF dose-dependently accelerated PAN02 proliferation ($p<0.05$). RT-PCR confirmed the presence of the HGF receptor, c-met, in PAN02 cells, and inhibition of c-met significantly decreased Pan02 proliferation ($p<0.05$).
CONCLUSIONS: These data show that 1) adipocytes significantly increase Pan02 pancreatic cancer proliferation; 2) this excess proliferation is significantly decreased with siRNA knockdown of adipocyte HGF; 3) exogenous HGF dose-dependently increases Pan02 proliferation; 4) the HGF receptor, c-met, is present in Pan02 cells; and 5) inhibition of Pan02 HGF receptor c-met significantly decreases Pan02 pancreatic cancer proliferation. We conclude that adipocytes promote pancreatic cancer proliferation, in part, via a HGF-mediated mechanism.



* $p<0.05$ vs. Control
# $p<0.05$ vs. Adipocyte

Session 5: 2:30 PM

DEREGULATION OF THE RB/E2F PATHWAY AND P16 EXPRESSION IN PANCREATIC ADENOCARCINOMA

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BACKGROUND: Deregulation of the Rb/E2F pathway has recently been shown to lead to epigenetic plasticity, particularly in the background of constitutive activation of *ras*, an oncogene mutated in the majority of pancreatic neoplasias. Moreover, cells in the plasticity state are programmable via signals from the microenvironment, resulting in epithelial to mesenchymal transition (EMT). EMT is believed to be responsible for generating metastatic cells as well as drug resistant phenotypes for the majority of epithelial cancers. We therefore sought to determine the relationship between Rb/E2F deregulation and outcomes for patients with pancreatic adenocarcinoma undergoing pancreaticoduodenectomy and adjuvant gemcitabine-based chemotherapy.
METHODS: Cytogenetic analysis was undertaken on ten pancreatic adenocarcinoma cell lines, four of which were established in the laboratory. PCR analysis, single-strand conformation polymorphism (SSCP) analysis, and northern blotting were used to determine the status of CDKN2A (p16), as well as other genes regulating the Rb/E2F pathway. Immunohistochemical staining for p16 was performed on FFPE tissues, and correlated with clinical outcomes. Data is presented as median, mean ± SD where appropriate.
RESULTS: Pancreatic adenocarcinoma cell lines commonly possessed chromosomal abnormalities in the following locations; 3p, 6p, 8p, 9p, 17p, and 18q. We sequenced the 3 exons of CDKN2A (located on chromosome 9p) and found homozygous deletions in 7/10 cell lines, suggesting frequent deregulation of the Rb/E2F pathway via loss of p16 among pancreatic cancers. In order to determine the clinical significance of Rb/E2F pathway deregulation, p16 immunohistochemical staining was performed on 26 patients who underwent resections for pancreatic adenocarcinoma (AJCC Stage IB (3), IIA (8), and IIB (15)) and completed adjuvant gemcitabine-based chemotherapy. Overall survival for 26 patients was 20 months, 27 months ± 17.5. p16 protein expression was absent in 25/26 (96%) patients. Of note, the single patient with positive p16 staining had lymphatic metastasis on final pathology and an overall survival consistent with the mean survival for the 26-patient cohort. Additionally, Pan-INs were identified in association with 12/26 adenocarcinomas; 2/12 Pan-INs stained positive for p16. Finally, we re-examined the 3 pancreatic adenocarcinoma cell lines expressing CDKN2A mRNA by DNA sequence analysis and identified mutations in the coding region of the gene for each of these cell lines.
Conclusions: Deregulation of the Rb/E2f pathway, specifically via deletions or mutations of the tumor suppressor gene CDKN2A (p16), appears to be a ubiquitous finding among pancreatic adenocarcinomas, and an early event in the tumorigenesis of this cancer. Although the Rb/E2F pathway does not appear to directly contribute to EMT and thereby metastasis and drug resistance given its early loss in the oncogenic process, deregulation may contribute to an environment conducive to EMT promoting events. Further work to identify the downstream targets of Rb/E2F signaling is warranted.

Session 5: 2:35 PM

A NOVEL MURINE MODEL FOR THE STUDY OF METASTATIC PANCREATIC ADENOCARCINOMA

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Introduction: The overall survival for patients with pancreatic cancer is poor with the liver being the most common site of metastatic disease. Novel, reproducible, translational models utilizing immune-competent mice are needed to test treatments and to study interactions between pancreatic adenocarcinoma and the surrounding metastatic microenvironment.
Methods: We injected tumor cell lines via a hemi-splenectomy model to create hepatic metastases. The chemically carcinogen induced Panc02 murine tumor cell line or pancreatic adenocarcinoma (PDA4969) cells derived from a genetically engineered Kras/p53 mutation knock in mouse were injected into syngeneic mice with or without C3H10T1/2 mesenchymal stem cells, representative of stromal cells. Histopathology and ultrasound images were obtained for metastatic lesions.
Results: Doses of $2x10^6$ Panc02 or PDA4969 cells led to the formation of tumors in the liver with some mice also exhibiting malignant ascites or peritoneal disease within sixty days (Figure 1). Results also indicate co-injection of mouse mesenchymal stem cells facilitate tumor formation in this model.

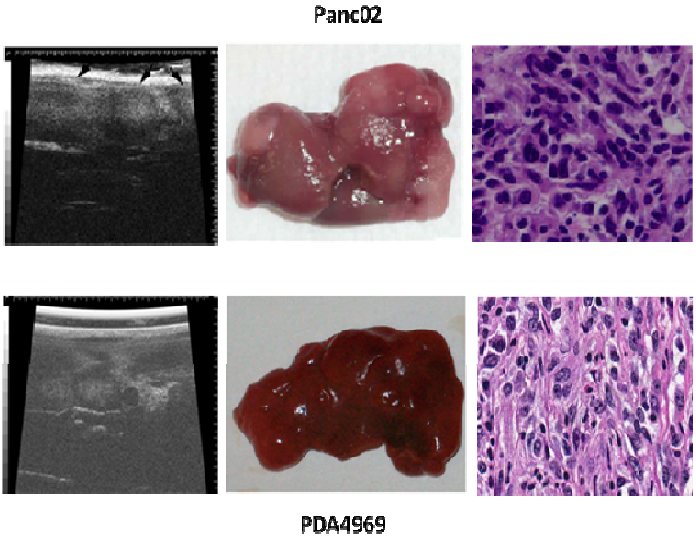

Figure 1. Liver ultrasound, gross necropsy and histopathology for pancreatic liver metastases given through hemi-splenectomy model. Images from representative mice shown

Conclusion: We successfully developed a pre-clinical model of metastatic pancreatic cancer. To our knowledge, this is the first mouse pancreatic tumor model that can be used to reliably produce liver metastases at rates approaching 100%. This mouse model will facilitate both the development of treatments and the study of interactions between pancreatic cancer and the components of the tumor microenvironment including infiltrating lymphocytes and stromal cells.

Session 5: 2:40 PM

BLOOD PRESSURE LOWERING MEDICATIONS DISRUPT FATTY ACID METABOLISM IN PANCREATIC CANCER

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Introduction/Background: Despite the routine use of chemotherapy and radiotherapy, survival has not significantly improved in patients with pancreatic ductal adenocarcinoma (PDA), a situation that signifies the urgent need for novel therapeutic approaches. Angiotensin II (AngII), the principal hormone of the renin angiotensin system, is actively generated in the pancreas and has been suggested as a key mediator of PDA cell survival. Fatty acid synthase (FAS) is a key enzyme involved in synthesis of fatty acids and its high levels have been correlated with poor prognosis in many cancers. In this study, we investigated the molecular basis for the role of AngII in pancreatic carcinogenesis through studying its effect on FAS.

Methods: FAS mRNA and protein in Panc-1 and PK-9 PDA cells, treated with or without AngII (10^{8}-10^{6} mol/L) in presence or absence of AngII receptor type 1 (ATR1) or type 2 (ATR2) blockers, were analyzed by real time PCR and ELISA. Luciferase-labeled promoter studies evaluated the effects of AngII on FAS transcription. MAPkinase and AKT phosphorylation was analyzed by Western immunoblotting. In human premalignant (n=6) and invasive PDA lesions (n=25), mRNA levels and localization of ACE (AngII converting enzyme), the enzyme responsible for AngII generation, and FAS were examined by real time PCR and immunohistochemistry. Mice with established PANC-1 or PK-9 cells tumor xenografts were treated orally with the AT1R blocker, losartan of or vehicle control (n = 5-10 mice per group) and were used to evaluate effects on tumor growth and FAS pathway activity.

Results: AngII significantly increased the expression of FAS mRNA and protein in PDA cells lines and induced FAS promoter activity. AngII-FAS mRNA induction was inhibited by an AngII type 1 receptor (AT1R) antagonist, losartan, and to a lesser extent by an AT2R antagonist. AngII activated the phosphorylation of ERK1/2, but not p38 or c-Jun NH2-terminal MAP kinases. Inhibition of ERK1/2 activation reduced the AngII-induced FAS synthesis. AngII activated the phosphorylation of AKT. Inhibition of AKT activation prevented the AngII-mediated increase of FAS. In human tissue, high mRNA levels of FAS correlated well with tumor stage and invasion status, and with high expression levels of ACE. Immunohistochemical staining of PDA serial sections showed co localization of ACE with FAS in the malignant ducts and stromal cells. Oral administration of losartan significantly ($p< 0.05$) decreased the growth of PANC-1 and PK-9 cells xenografted on the flank of nude mice and significantly ($p<0.05$) reduced the expression of FAS mRNA and protein in the xenografts.

Discussion/conclusions: Our data suggest a positive autocrine/paracrine action for the local pancreatic AngII generating system during pancreatic carcinogenesis, and provide the first insight into an AngII-initiated signal transduction pathway that involves AT1R/ERK1/2/AKT to regulate fatty acid metabolism and modulate lipogenesis in PDA. These results raise the possibility that AngII blockade therapies could be potential candidates in novel treatment strategies of PDA.

Session 6: 3:45 PM

RANDOMIZED TRIAL COMPARING EUS AND SURGERY FOR PANCREATIC PSEUDOCYST DRAINAGE

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BACKGROUND Although EUS-guided cystgastrostomy is increasingly performed, surgery is still considered the gold standard technique for management of symptomatic uncomplicated pancreatic pseudocysts (PC). No study has randomized the treatment of PC between EUS and surgery and compared outcomes, quality of life (QOL) and costs.
METHODS Symptomatic patients with PC measuring > 6cm were evaluated by both the surgeon and gastroenterologist in order to determine inclusion into the study. Patients were then prospectively randomized to undergo EUS-guided or surgical cystgastrostomy. Pancreatic abscess or necrosis, multiple PC and those PC located distant from the stomach were excluded. The primary endpoint was PC recurrence at 18 months. The secondary endpoints were pain pattern (measured by brief pain inventory), quality of life (assessed by SF-36), total costs, length of post-procedure hospital stay (LOS), complications and reinterventions at end of follow-up. Technical success was defined as the ability to perform the intervention. Treatment success was defined as symptom relief without need for repeat intervention during follow-up.
RESULTS Of 90 screened patients, 36 met inclusion criteria and underwent randomization (19 to EUS and 17 to surgery). There was no difference in patient demographics, clinical presentation, pseudocyst size, rates of technical (both cohorts, 100%) or treatment success (94.4% vs. 100%, p=1) and procedural complications (none in both cohorts) between EUS and surgery, respectively. At a median follow-up of 18 months, there was no difference in rates of PC recurrence (0% vs. 5.8%, p=0.48) or reinterventions (5.2% vs. 0%, p=1) between EUS and surgery, respectively. Although there were no long-term differences, when compared to surgery, the average scores for pain (3.3 vs. 1.6, p=0.009) and interference of general activity (4.9 vs. 2.2, p=0.007) and mood (4.1 vs. 1.8, p=0.005) were significantly better at 1 week for the EUS cohort. Compared to surgery, the mean QOL scores for general health (50.4 [SD=19.7] vs. 71.4 [19.7], p = 0.007), general vitality (47.1 [14.5] vs. 63.8 [12.5], p = 0.004) and physical function (57.2 [27.9] vs. 80 [23.9], p = 0.008) were significantly better for EUS from post-procedure and up to 3 months. After 3 months, the improvement in QOL was similar between both groups. Compared to surgery, the median LOS (6 vs. 2 days, p <0.0001) and average costs ($22,475 vs. $8,195, p<0.0001) were significantly less for EUS-guided cystgastrostomy.
CONCLUSIONS When surgical and endoscopic evaluations concur, EUS-guided cystgastrostomy should be the preferred treatment approach for patients with uncomplicated symptomatic pancreatic pseudocysts. The procedure is less costly, yields quick pain relief, is associated with shorter length of hospital stay and has long-term clinical outcomes and quality of life comparable to that of surgery. A multidisciplinary approach is important to achieving safe outcomes and optimal results.

Session 6: 4:00 PM

DOES INCREASING INSURANCE IMPROVE OUTCOMES FOR U.S. PANCREATIC CANCER PATIENTS?

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Background: While debate continues surrounding health care reform and increasing access to insurance across the U.S., there is a lack of data on the impact of insurance coverage when confronted with pancreatic cancer. Using national data, we examined insurance coverage rates and cancer mortality on a state-by-state level.
Methods: Data from the U.S. Census Bureau Current Population Survey, 2002-2004, was used to examine rates of uninsured populations across states. Data were compared with incidence and mortality rates from National Cancer Institute State Cancer profiles (2002-2006). Proxies for cancer lethality rates were established by comparing mortality to incidence. Rates and directional trends of % uninsured and cancer lethality for each state were evaluated.
Results: Overall rate of being uninsured in the U.S. over the years 2002-2004 was 15.5%. Overall mortality for all identified cancers as a whole 2002-2006 decreased; however hepatopancreaticobiliary, melanoma, and thyroid cancer mortality rates demonstrated an increase. Furthermore, comparing mortality and incidence among cancers with leading mortality rates, pancreatic cancer was the most lethal. Examining the states with the highest and lowest uninsured rates, higher uninsurance was associated with higher pancreatic cancer lethality rates than the national average, whereas better-insured states were more likely to have lower than national average rates.
Conclusions: At the state level, across the U.S., insurance coverage correlated with improved cancer outcomes. Further investigations are needed to examine whether these associations hold true within the individual community, and how any barriers to cancer care in specific communities can be surmounted.

Table: Pancreatic cancer lethality among states with lowest and highest rates of uninsurance

	U.S.	/	**MN**	**HI**	**IO**	**VT**	**RI**	/	**LA**	**NV**	**OK**	**NM**	**TX**
%Uninsured	15.5		8.5	9.9	10.1	10.5	10.5		**18.8**	**19.1**	**19.2**	**21.4**	**25.1**
%Lethality	93.9		**100**	84.3	93.6	76.6	92.0		90.2	86.4	**98.1**	**96.1**	**96.2**
Mortality Rate (per 100,000)	10.8		10.7	10.2	10.3	8.2	10.4		**11.9**	10.8	10.1	9.9	10.1
Incidence Rate (per 100,000)	11.0		10.2	**12.1**	11.0	10.7	**11.3**		**13.2**	**12.5**	10.3	10.3	10.5

(bold = higher than national average)

Session 6: 4:15 PM

AUTO-ISLET TRANSPLANTATION FOR CHRONIC PANCREATITIS IN DIABETIC PATIENTS: WHY BOTHER?

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BACKGROUND: In select patients with chronic pancreatitis, pancreatectomy with simultaneous islet auto-transplantation can be an effective means of pain control while ameliorating the attendant complications of severe diabetes. Expected outcomes include improved blood glucose control, with insulin independence in 25-40% of patients, and hypoglycemic awareness. Whether patients with preoperative diabetes benefit from islet auto-transplantation after extensive pancreatectomy has not been well examined.

METHODS: With the approval of the Institutional Review Board for the evaluation of human subjects, a prospectively collected database of all patients undergoing pancreatectomy with islet autotransplantation at a single institution between March and December of 2009 was retrospectively reviewed. Data, including demographics, preoperative and postoperative clinical course and outcomes were examined, with particular attention to patients that were diabetic preoperatively.

RESULTS: Twenty-six patients (18 women, median age 46 years, median BMI 27.7kg/m^2) underwent extensive pancreatectomy with immediate islet auto-transplantation for pancreatitis. Of these patients, 6 (5 women, median age 34, median BMI 28.6kg/m^2) were insulin-requiring preoperatively. All patients underwent total (4) or completion (2) pancreatectomy. A median of 40,003 IEQ (range 3,667 to 675,350) and 546.4 IEQ/kg (range 54.2 to 13,782.7) were harvested from these diabetic patients. Median preoperative daily insulin requirement was 21 units (range 10 to 86), with discharge daily requirement of median 10 units (range 2 to 44 units). In follow up at a mean 3.5 months, median daily insulin requirement was 12 units (range 2 to 53 units), with a median decrease of 9 units per patient (range -3 to 41 units). All patients had hypoglycemic awareness.

CONCLUSIONS: Diabetic patients undergoing extensive pancreatectomy demonstrate lower insulin requirements and hypoglycemic awareness after islet autotransplantation. Larger experience and longer term follow up are needed to determine the true benefits of islet autotransplantation in the diabetic patient.

Session 6: 4:30 PM

ABDOMINAL COMPARTMENT SYNDROME: AN EARLY LETHAL COMPLICATION OF ACUTE PANCREATITIS

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Introduction: Increases in intraabdominal pressure may result in a syndrome of organ dysfunction referred to as the abdominal compartment syndrome. Although usually occurring after emergency abdominal surgery or abdominal trauma, abdominal compartment syndrome may also occur in acute pancreatitis. We compared the operative management and results of patients with acute pancreatitis who underwent surgery for treatment of abdominal compartment syndrome with patients who underwent surgery for debridement of the pancreas.
Methods: An electronic database was searched to identify patients with acute pancreatitis who underwent surgery between 1/1/2000 and 12/31/2009 for treatment of abdominal compartment syndrome or pancreatic debridement.
Results: Twelve patients with acute pancreatitis underwent decompressive laparotomies for the clinical diagnosis of abdominal compartment syndrome in the setting of acute pancreatitis. The interval between onset of pancreatitis and surgery ranged from 1 to 75 days (median 5 days, mean 13.75 days). 9 patients underwent decompressive laparotomies within 7 days, and 11 within 16 days, of onset of pancreatitis. Due to cardiopulmonary instability 4 decompressive laparotomies were performed at the bedside in the intensive care unit. In 11 of the 12 patients, cardiopulmonary improvement was initially observed. Nine patients underwent subsequent pancreatic debridement procedures. Six of the 12 patients, including 2 of the 4 patients undergoing ICU laparotomies, recovered with lengths of stay after the decompressive laparotomy ranging from 32 to 100 days; these patients were transferred to skilled nursing, long term acute care, or rehabilitation facilities. Six (50 %) patients died from multisystem organ failure between 2 and 30 days after the decompressive laparotomies.

During the same time period, 99 patients underwent pancreatic debridement procedures following an episode of acute pancreatitis. The interval between onset of pancreatitis and an initial debridement procedure ranged from 0 to 120 days (median 32 days, mean 42 days; $p<0.05$). In only 6 patients was a debridement procedure performed within 7 days of onset of pancreatitis, and in only 17 was a debridement procedure performed within 16 days. The most common indications for pancreatic debridement were infected necrosis (73 patients), multisystem organ failure (9), and gastrointestinal complications such as perforation or obstruction (6). Multiple debridement procedures were performed in 59 (59 %) patients. 87 patients survived and were discharged after lengths of stay ranging from 6 to 180 days. Twenty patients were discharged to home. Twelve patients (12 %; $p<0.05$) died between 1 and 120 days after undergoing pancreatic debridement procedures as a result of multisystem organ failure (8), sepsis (1), bleeding (1), or cardiac arrhythmias (2).
Conclusion: Abdominal compartment syndrome is an uncommon but highly lethal complication that may arise in acute pancreatitis and should particularly be considered in patients who become critically ill early in the course of their pancreatitis. Although abdominal exploration in patients with acute pancreatitis is most often reserved for patients who develop infected necrosis or progressive multisystem organ failure, recognition and prompt treatment of abdominal compartment syndrome by means of a decompressive laparotomy may rescue some of these critically ill patients.

Session 6: 4:45 PM

LIVE ANIMAL MOLECULAR IMAGING OF PROTEASE ACTIVITY IN ACUTE PANCREATITIS

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Introduction:
Proteases promote inflammation and pain in animal models of acute pancreatitis. Traditional biochemical techniques to study proteases such as fluorogenic assays, protein expression and immunostaining fail to provide concurrent information on the post-translational regulation, localization, and activity levels during the onset and progression of pancreatic inflammation. We determined the feasibility of detecting increased protease activity *in situ* with both fiberoptic confocal fluorescence microscopy (FOCM) and two photon fluorescence excited microscopy (2PM) using fluorescent probes activated by trypsin and cathepsins in cerulein-induced pancreatitis.

Methods and Results:
Activity-based probes (ABPs) and activatable probes (APs) are novel tools that give a direct read-out of active protease activity in whole organisms via *in vivo* near-infrared imaging, in tissue sections, and in cell lysates for biochemical analysis. To identify cathepsins that are activated in the inflamed pancreas, we used **ProSense680** (VisEn), an AP with a Cy5.5 fluorophore activated by trypsin and cathepsins for imaging and **MGP75** a biotin-labeled ABP that covalently binds to activated serine proteases for biochemical studies. C57/Bl6 mice were treated with cerulein or vehicle (12 hourly injections, 50 µg/kg SQ) to induce acute pancreatitis. Mice then received ProSense680 intravenously and were studied 24 h later. *In vivo* pancreas imaging with FOCM (CellVizio) was performed through a small midline incision. In living animals with acute pancreatitis, FOCM detected diffuse protease activity throughout the inflamed pancreas with acinar-level resolution in real-time. Inflamed tissue had significantly more activity (trypsin, cathepsin B, L, and S) compared to non-inflamed controls. Excised pancreas from these animals were fixed, cryoprotected, and then imaged as whole organs using 2PM or via tissue sections using traditional confocal microscopy. *In situ* 2PM detected protease activity within acinar cells with high resolution and excellent preservation of tissue architecture. Confocal microscopy with immunstaining on fixed tissue confirmed that protease activity was localized to acinar cells and infiltrating macrophages in interlobular septae.
To determine if protease activity is increased in patients with chronic pancreatitis, we measured serine protease activity using ABPs and a conventional fluorogenic trypsin assay, MPO, and amylase in pancreatic juice from patients with chronic pancreatitis undergoing ERCP. PJ was reacted with MGP75, run on SDS-PAGE and analyzed by Western blot. We found that MGP75 binds to the following serine proteases: trypsin, □-trypase, thrombin, kallikrein14, and the inhibitor-resistent isoforms of trypsin, human mesotrypsin and rat p23. Patients with elevated PJ protease activity also had increased MPO activity, and conversely PJ with low protease activity had low MPO activity while amylase activity was similar among all samples.
Conclusions: Elevated protease activity can be imaged using minimally invasive methods in live animals with acute pancreatitis. FOCM offers a rapid means of quantifying protease signal whereas 2PM provides enhanced subcellular resolution. Confocal microscopy complements *in situ* techniques to facilitate immunolocalization of protease activity. Fiberoptic fluorescence imaging could be used with ERCP in conjunction with protease activated probes to measure inflammation and disease severity.

Saturday- Poster #1- Professor Rounds 3:00 – 3:25 pm

IMPACT OF PREOPERATIVE HYPERGLYCEMIA ON MORBIDITY AFTER PANCREATICODUODENECTOMY

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Introduction High volume centers have achieved acceptable rates of morbidity and mortality after pancreaticoduodenectomy (PD) for both benign and malignant disease. These rates continue to vary based on preoperative patient debilitation and comorbidities. Most studies have examined complications after PD based on operative and clinical data but none have specifically analyzed the effect of preoperative glucose levels.
Methods Patients undergoing PD by the senior author between September 2005 and June 2009 were analyzed. The American Diabetes Association defines diabetes mellitus (DM) as fasting blood glucose > 125 mg/dL on two occasions. Patients were divided into 4 groups (non-DM, untreated DM, poorly-controlled DM, and well-controlled DM). Obesity was defined as body mass index (BMI) > 30. Pancreatic fistula and delayed gastric emptying (DGE) were defined according to the International Study Group of Pancreatic Surgery.
Results 231 consecutive patients (mean age 61 years, 52% male) underwent PD for benign (chronic pancreatitis, n=53; IPMN or cystic neoplasm, n = 32; ampullary or duodenal adenoma, n = 14) and malignant (pancreatic adenocarcinoma, n = 89; ampullary and duodenal adenocarcinoma, n = 17; neuroendocrine, n = 14; cholangiocarcinoma, n = 10; duodenal GIST, n = 2) pathology. Complications occurred in 48% of patients and the overall 30-day and in-hospital mortality rate was 2.2%. Patients with newly-diagnosed untreated DM (n = 24) experienced more complications compared to non-DM patients (n = 142; $p < 0.01$). Similarly, patients with poorly-controlled DM (n = 41) had more complications when compared to those with well-controlled DM (n = 24; $p < 0.002$). Patients with newly-diagnosed untreated DM and poorly-controlled DM had higher rates of DGE (26.0% vs. 12.0%, $p < 0.05$), wound infection (26.0% vs. 4.4%, $p < 0.001$), and longer mean length of stay (11.4 vs. 9.6 days, $p = 0.05$) than normoglycemic patients. All other systemic infections were more frequent in the untreated and poorly-controlled groups (30.0% vs. 9.9%, $p < 0.005$). Pancreatic fistula rate (18.2%) did not correlate with preoperative glycemic control. Obesity (23%) did not correlate with a higher rate of overall ($p=0.46$) or infectious complications ($p=0.54$). Univariate analysis identified age ≥ 65 years ($p < 0.02$), untreated or poorly controlled DM ($p < 0.001$), Charlson Comorbidity Index Score ≥ 4 ($p < 0.001$), and malignant pathology ($p < 0.005$) as risk factors for post-operative complications. Multivariate analysis of these factors identified untreated or poorly controlled DM ($p < 0.008$) as the most powerful independent predictor of operative complications after PD.
Conclusion Patients with hyperglycemia before PD, whether known to be diabetic or not, have significantly more complications after PD than those with normoglycemia. In order to minimize post-operative morbidity, our findings support a role for aggressive glycemic control in the preoperative setting for all patients scheduled for PD.

Saturday-Poster #2- Professor Rounds 3:00 – 3:25 pm

PROGNOSTIC IMPACT OF DIHYDROPYRIMIDINE DEHYDROGENASE EXPRESSION IN ADJUVANT GEMCITABINE PLUS S-1 CHEMOTHERAPY AFTER SURGICAL RESECTION FOR PANCREATIC ADENOCARCINOMA

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Short title: Prognostic Impact of DPD in Pancreatic Cancer

Objective: Although the prognosis in patients with pancreatic adenocarcinoma remains poor, adjuvant gemcitabine plus S-1 chemotherapy (GEM + S-1) after surgical resection for pancreatic adenocarcinoma has been shown to improve survival. S-1 is a novel oral fluoropyrimidine combination including tegafur (a prodrug of 5-fluorouracil; 5-FU), dihydropyrimidine dehydrogenase (DPD) inhibitor (5-chloro-2,4-dihydroxypyrimidine), and orotate phosphoribosyltransferase (OPRT) inhibitor (potassium oxonate). To clarify the relationship between expression of intratumoral enzymes related to the metabolism of 5-FU and its derivatives and response to adjuvant chemotherapy with GEM + S-1 for pancreatic adenocarcinoma, we evaluated thymidylate synthase (TS), DPD, and OPRT expression immunohistochemically in resected pancreatic adenocarcinoma tissues.
Methods: Polyclonal antibodies were used to immunostain sections of 106 formalin-fixed paraffin-embedded specimens of pancreatic adenocarcinoma resected between 1998 and 2009. The relationship between intratumoral TS, DPD, and OPRT expression and prognosis was evaluated statistically.
Results: Out of 106 patients, 75 (70.1%) received adjuvant GEM + S-1 chemotherapy. High intratumoral TS, DPD, and OPRT expression was present in 68 (64.1%), 39 (36.8%), and 70 (66.0%) cases, respectively. Comparison of overall survival between High and Low intratumoral TS or OPRT expression revealed no significant difference regardless the application of adjuvant GEM + S-1 chemotherapy. In the GEM + S-1 (+) group, overall survival was significantly longer in the Low DPD subgroup than in the High DPD subgroup (hazard ratio [HR], 0.661: 95% confidence interval [CI], 0.447 – 0.978; $P = 0.031$), whereas in the GEM + S-1 (–) group, there was no significant difference between the High DPD and Low DPD subgroups. Moreover, in the High DPD group, there was no significant difference in overall survival between the GEM + S-1 (+) and GEM + S-1 (–) subgroups, whereas in the Low DPD group overall survival was significantly higher in the GEM + S-1 (+) subgroup by univariate analysis (HR, 0.456; 95% CI, 0.295 – 0.683; $P < 0.001$).
Conclusion: Low intratumoral DPD expression was associated with increased overall survival in patients with pancreatic adenocarcinoma who received adjuvant GEM + S-1 chemotherapy. DPD is a relevant predictive marker of benefit from adjuvant GEM + S-1 chemotherapy in patients with resected pancreatic adenocarcinoma.

Saturday- Poster #3- Professor Rounds 3:00 – 3:25 pm

SURVIVIN EXPRESSION IN CARCINOGENESIS OF INTRADUTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS

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Backgrounds: Intraductal papillary mucinous neoplasms (IPMNs) of the pancreas are indicated the possibility to be a model of multistep carcinogenesis of pancreatic cancer, because they include a spectrum of neoplasms like adenoma, borderline lesion, carcinoma in situ (CIS), and invasive adenocarcinoma (invasive). Survivin, a member of the apoptosis inhibitor family, shows increased expression in human cancers of various origins. It has been demonstrated that survivin inhibits apoptosis via caspase inhibition and promotes mitosis via aurora-B kinase activation.
Objectives: To evaluate survivin and aurora-B kinase expression in IPMNs, which have potential to clarify the carcinogenesis of the pancreatic neoplasms and to be a biomarker of earlier diagnosis of pancreatic malignancy.
Methods: Survivin and aurora-B kinase expression were evaluated by immunohistochemistry in a total of 168 lesions from 85 patients who underwent pancreatic resection for IPMNs which included invasive IPMNs (n=22), CIS IPMNs (n=32), borderline IPMNs (n=14), and adenoma IPMNs (n=17).
Results: The nuclear overexpression of survivin was observed in 15 of 15 (100%) invasive IPMNs, 20 of 43 (47%) CIS IPMNs, 2 of 14 (14%) borderline IPMNs, and 9 of 77 (12%) adenoma IPMNs, respectively. The nuclear overexpression of aurora-B kinase was observed in 6 of 15 (40%) invasive IPMNs, 17 of 43 (40%) CIS IPMNs, non of 14 (0%) borderline IPMNs, and 2 of 62 (3%) adenoma IPMNs, respectively, while no hyperlastic lesion had overexpression of survivin and aurora-B kinase. There were significant differences in the expression of survivin between invasive IPMNs and CIS IPMNs ($p<0.001$), and in the expression of survivin and aurora-B kinase between CIS IPMNs and adenoma IPMNs ($p<0.001$ for both). There were also significant differences in the expression of survivin and aurora-B kinase between malignant IPMNs and benign IPMNs ($p<0.001$ for both).
Conclusions: These results suggested that survivin may play important roles in the transition from CIS IPMNs to invasive IPMNs of the pancreas. Moreover, survivin and aurora-B kinase overexpression may indicate the carcinogenesis of IPMNs of the pancreas and they may be useful biomarkers for earlier diagnosis of pancreatic malignancy.

Saturday- Poster # 4- Professor Rounds 3:00 – 3:25 pm

TUMOR LOCATION DOES NOT EFFECT SURVIVAL OF PATIENTS UNDERGOING RESECTION FOR PANCREATIC ADENOCARCINOMA DESPITE LARGER DISTAL PANCREATIC TUMORS

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Introduction: The only hope of cure for pancreatic adenocarcinoma is surgical resection. Convention dictates that patients with pancreatic adenocarcinoma in the tail of the pancreas have shorter survival than patients with pancreatic adenocarcinoma in the head of the pancreas; this is presumed to be due to the lack of symptoms early in the disease course and presentation with more advanced tumors. This study was undertaken to determine differences in tumor characteristics and survival between resectable adenocarcinomas of the pancreatic tail and head.
Methods: Since 1992, data of patients undergoing pancreaticoduodenectomy (PD) or distal pancreatectomy with splenectomy for pancreatic adenocarcinoma were collected. Tumor characteristics were analyzed using the Mann-Whitney U-Test; survival comparisons were undertaken with Mantel-Cox survival curve analysis. Data are presented as median, mean ± SD, where appropriate.
Results: 220 patients underwent PD and 33 patients underwent distal pancreatectomy with splenectomy for pancreatic adenocarcinoma. Tumors leading to distal pancreatectomy with splenectomy were larger (4 cm, 5 cm ± 2.3) than tumors leading to PD (3 cm, 3 cm ± 1.4; p=0.005). Overall survival was similar after PD (17 months, 26 months ± 25.9) and distal pancreatectomy with splenectomy (15 months, 20 months ± 18.6; p=0.74). When stratified by margin status, survival was not different for PD versus distal pancreatectomy with splenectomy (Table). Survival was impacted by T stage, nodal status, and AJCC stage; survival was improved with complete tumor extirpation (i.e., R0 resection) in patients undergoing PD (p=0.004).

Margin	Distal Pancreatectomy with Splenectomy	PD	p-value
R0	16 months, 20 ± 19.8 (n=26)	20 months, 26 ± 23.5 (n=55)	0.90
R1	11 months, 17 ± 14.4 (n=7)	13 months, 20 ± 27.6 (n=165)	0.95

Conclusions: By most descriptors, adenocarcinomas of the tail or head of the pancreas are similar, though tumors in the tail are larger. Survival is unsatisfactory after resection of proximal or distal cancers, while it is improved by complete tumor extirpation. Few factors impact survival after pancreatectomy for adenocarcinoma, but margin status is vital and should be aggressively sought.

Saturday –Poster # 5- Professor Rounds 3:00 – 3:25 pm

PP INFUSION IN TYPE 1 DIABETIC PATIENTS (T1DM) REDUCES INSULIN REQUIRMENTS: A PILOT STUDY.

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Introduction: Pancreatic Polypeptide (PP) deficiency has been shown to be associated with hepatic insulin resistance in chronic pancreatitis (CP) and after pancreatectomy. Studies in CP and pancreatic resection patients have suggested that hepatic sensitivity to insulin is increased, and glucose tolerance improved, after an 8 hr infusion of PP. We sought to determine whether the addition of a continuous infusion of PP in T1DM on insulin pump treatment would reduce their insulin requirement.
Methods: We studied 7 patients (5 T1DM, 2 post pancreatectomy) who had stable glycemia on insulin pump therapy (variable basal rate with bolus additions based on frequent finger-stick glucose determinations). In a single-blind cross-over design, a continuous, fixed subcutaneous infusion of 2 $pmol \cdot kg^{-1} \cdot min^{-1}$ PP or saline was begun, and insulin requirements and glucose levels were monitored for 72 h. Mean plasma glucose [G] determination and mean insulin infusion [Ii] requirements were assessed during each 24 h of the infusion period.
Results: There were no adverse outcomes or side effects associated with the 72 h PP infusion in any patient. The insulin need, corrected for [G], or [Ii]/[G], decreased 38.4% during the second day of PP infusion compared to saline and 26.6% during the third day of PP infusion ($p<0.02$ for both). Plasma PP responses to a standard test meal were measured, and the reduction in insulin requirements correlated with the degree of PP deficiency.
Conclusion: These data demonstrate that subcutaneous infusion of PP in patients with presumed PP deficiency reduces their insulin need. PP deficiency invariably follows after proximal or total pancreatectomy, but is also present in some T1DM patients. This observation corroborates earlier findings, and suggests that PP administration may be beneficial in selected insulin-dependent, PP deficient patients. Longer infusion studies are warranted.

Saturday Poster 6

PERIOPERATIVE OUTCOMES FOR OPEN DISTAL PANCREATECTOMY: CURRENT BENCHMARKS FOR COMPARISON?

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Background: There exists increasing enthusiasm for laparoscopic distal pancreatectomy (LDP) for benign and malignant lesions of the pancreatic tail. Comparisons to open distal pancreatectomy (ODP) outcomes have largely relied on single institution databases primarily from high-volume, tertiary centers. Therefore, to provide an appropriate national benchmark of ODP outcomes, we examined the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) database (2005-07) to accurately describe 30-day morbidity and mortality, operative time, transfusion requirement, and hospital length-of-stay (LOS) for patients undergoing ODP.
Methods: The NSQIP database was used to identify 1,313 cases of distal pancreatectomy (CPT code 48140). Cases with extra-pancreatic procedures or any laparoscopic component were excluded leaving a final study cohort of 868 patients. Central tendencies of operative time, intraoperative transfusion requirement, and LOS were calculated. Univariate analyses were performed using all 53 preoperative NSQIP variables and malignant vs. benign diagnosis for likelihood of any postoperative complications and severe complications (composite endpoint: death, organ space surgical site infection, or reoperation). Multivariate analyses were performed using a logistic regression likelihood model, adjusting for age, body mass index (BMI), diagnosis type, creatinine, albumin, hematocrit, and American Society of Anesthesiologists classification (ASA).
Results: Any complication, severe complication, and mortality rates were 27.2%, 11.6%, and 1%, respectively. Mean operative time was 206 minutes (±86); 18.1% patients required intraoperative red blood cell transfusion (median 2 units; range 1-15), and median LOS was 6 days. Predictors of any complication or severe complication were renal insufficiency, hypoalbuminemia, and worsening ASA classification. Malignant diagnosis was not associated with increased likelihood of morbidity or mortality.
Discussion: Open distal pancreatectomy remains the gold standard to which laparoscopic distal pancreatectomy is compared. Our analysis of patients undergoing ODP reflect nationwide data that may serve as current benchmarks to which patients undergoing LDP should be compared.

Table. Multivariate analysis of predictors of any complications and severe complications, n=868; Odds Ratio/ (95% Confidence Interval)

Variable	Any Complication	Severe Complication
Age (median 59 yrs)	1.00 (0.99-1.02)	1.00 (0.99-1.02)
BMI (median 27)	1.02 (1.00-1.05)	1.02 (0.98-1.05)
Diagnosis Type		
Benign	1.00 (referent)	1.00 (referent)
Malignant	1.06 (0.75-1.49)	1.04 (0.65-1.68)
Unknown	0.81 (0.50-1.32)	0.82 (0.42-1.62)
Creatinine		
<1.2	1.00 (referent)	1.00 (referent)
1.2-2.0	1.43 (0.88-2.33)	***1.89 (1.02-3.49)**
>2.0	0.89 (0.22-3.63)	0.82 (0.10-6.71)
Unknown	0.93 (0.38-2.23)	1.35 (0.45-4.07)
Albumin		
>3.5	1.00 (referent)	1.00 (referent)
2.5-3.5	***1.94 (1.12-3.38)**	1.57 (0.74-3.34)
<2.5	3.23 (0.75-13.84)	***6.63 (1.39-31.57)**
Unknown	1.04 (0.68-1.57)	1.06 (0.60-1.89)
Hematocrit		
36-49	1.00 (referent)	1.00 (referent)
>49	2.23 (0.29-17.22)	2.18 (0.21-23.05)
<36	0.89 (0.57-1.41)	0.76 (0.39-1.45)
Unknown	1.83 (0.77-4.36)	1.71 (0.58-5.04)
ASA Classification		
1/2	1.00 (referent)	1.00 (referent)
3	***1.67 (1.19-2.33)**	1.52 (0.95-2.44)
4	2.54 (0.91-7.08)	1.74 (0.45-6.75)
*** Denotes significance, P≤0.05**		

Saturday Poster 7

PREOPERATIVE CT MEASUREMENT OF PANCREATIC STEATOSIS AND VISCERAL FAT; PROGNOSTIC MARKERS FOR DISSEMINATION AND LETHALITY OF PANCREATIC ADENOCARCINOMA

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INTRODUCTION
Increased visceral fat amplifies the risk of developing pancreatic cancer, while pancreatic steatosis promotes lymphatic metastases and decreased survival for patients with pancreatic adenocarcinoma after pancreaticoduodenectomy (PD). However, a correlation between preoperative adipose measurements by CT scanning and patient outcomes remains ill defined. We aim to determine the utility of preoperative CT measurements of pancreatic steatosis and visceral fat as prognostic indicators for patients with pancreatic adenocarcinoma.

METHODS
High resolution CT scans of 42 patients undergoing PD for pancreatic adenocarcinoma were reviewed. CT attenuation of the pancreas, liver, and spleen were measured in Hounsfield units and scored by two blinded investigators. Perirenal adipose tissue (an indicator of visceral fat) was measured in mm. Pathology slides were reviewed for tumor differentiation and invasion. Data are presented as mean ± SD.

RESULTS
Lymphatic metastases were absent (N0) in 43% and present (N1) in 57% of patients. Age, gender, tumor size, and margin status were similar between patients with and without nodal metastases. Outcomes for patients stratified by nodal status and perirenal adiposity are depicted below. All patients received adjuvant therapy.

Nodal Status & Fat Pad Depth	Perirenal Adipocity (mm)	Pancreatic Body (HU)	Liver (HU)	Peripancreatic Fat Invasion (%)	Survival (months)
N0	13±2	35±4	58±4	53	21±4
N1	18±1*	23±2*	50±2*	90*	11±2*
N1 & <10mm	9±1	27±2	53±4	80	16±2
N1 & >10mm	21±1†	25±2	48±2	91	7±1†

*p<0.01 vs. N0, †p<0.01 vs. N1 & fat pad<10mm

CONCLUSIONS
With resected pancreatic adenocarcinoma, increased pancreatic and liver steatosis, as well as increased visceral fat stores are associated with lymphatic metastases. Furthermore, increased visceral fat is associated with an abbreviated survival for patients with lymphatic metastases, and thereby may serve a prognostic role for patients with pancreatic malignancies. Therefore, we conclude that CT measurements of visceral fat predict the dissemination and lethality of pancreatic adenocarcinoma.

Saturday Poster 8

PERIOPERATIVE AND LONG TERM OUTCOME AFTER MULTIVISCERAL RESECTION FOR PANCREATIC CANCER

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Decreasing mortality and morbidity rates after resection for pancreatic cancer (PaCa) and advances in perioperative (neo-) adjuvant treatment might suggest the inclusion of patients with locally advanced or even limited metastasized PaCa as candidates for surgery.
Methods: During a 15-year period 272 patients underwent resection for PaCa (80% PD, 14% distal, 6% total pancreatectomy). 28% (n=77) of the operations were performed with venous resection (group PVRes), 13% (n=35) with resection of one or more further organ (group OrgRes; in part with vein resection, spleen not included; 16 stomach, 12 adrenal, 8 colon, 7 other) and 59% without vein or further organ resection (group LimRes). Histopathological, perioperative and outcome data were compared by retrospective analysis of our prospective pancreatic database.
Results: The 3 groups did not differ significantly regarding age, grading, nodal disease (66%) or margin status (positive margins 30%, R2 6%), but tumors in the LimRes group were significantly smaller ($p<0.001$). Mortality was significantly higher in the OrgRes than in the LimRes or PVRes-groups (11.4% vs 5% / 0%; $p=0.02$). Patients with OrgRes had higher rates of any complication (71% vs 50% in LimRes/VPRes; $p<0.03$), intraabdominal abscess (20% vs 6%; $p=0.01$) and relaparotomy (20% vs 7%; $p=0.02$). Actuarial 5-year survival (5-ys) was 16% (17 actual 5-year survivors). In multivariate (Cox) survival analysis outcome was independently worse with positive nodes ($p<0.04$; RR 1.4), positive margins ($p<0.01$; RR 1.7) and after OrgRes ($p<0.01$; RR 2.1). As compared to the group with LimRes, patients with additional PV-resection had comparable mortality, morbidity and survival rates.
Conclusion: In our series multivisceral resections of pancreatic cancer (other than vein resections) were associated with increased mortality and morbidity rates and a lower survival rate. This poor long-term outcome did not directly correlate with node or margin positivity. Patients with PaCa probably requiring multivisceral resection, therefore, should be well selected and/or considered to undergo neoadjuvant treatment.

PERIOPERATIVE BLOOD TRANSFUSION AND OPERATIVE TIME ARE QUALITY INDICATORS FOR PANCREATICODUODENECTOMY

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Introduction: Allogenic blood transfusions increase the risk of infection, acute lung injury, immunosuppression and potentially cancer recurrence. Minimization of blood loss during pancreaticoduodenectomy requires precise dissection and careful surgical technique. Therefore, packed red blood cell (PRBC) transfusions and operative time are potential surgical quality indicators for pancreaticoduodenectomy. The goal of this study was to identify the associations between both perioperative PRBC transfusion and operative time with 30-day morbidity/mortality following pancreaticoduodenectomy.
Methods: All patients who underwent a pancreaticoduodenectomy were identified using the American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) database. PRBC transfusions (within 72 hours) and operative time were correlated with 30-day morbidity and mortality. Correlation coefficients (R) were calculated.
Results: Pancreaticoduodenectomy was completed in 4817 patients. PRBC transfusions were given in 1559 (32%) patients (range: 1-35 units). Most (59%) operative times were between 4 and 7 hours. Overall morbidity and mortality rates were 37% and 3%, respectively. Overall 30-day morbidity increased in a stepwise manner with the number of PRBC transfusions ($R=0.69$, $p<0.01$). Although PRBC transfusions and operative times were not statistically linked ($p=0.87$), longer operative times were linearly associated with increased 30-day morbidity ($R=0.79$, $p<0.001$) and mortality ($R=0.65$, $p<0.01$). Patients who were not transfused also displayed less morbidity (33%) and mortality (1.9%) than patients receiving any volume of PRBCs ($p<0.05$).
Conclusion: Perioperative PRBC transfusion following pancreaticoduodenectomy is associated with 30-day morbidity in a stepwise manner. Longer operative time also correlates with increased morbidity and mortality. Therefore, avoidance of blood transfusions and prolonged operative time should be considered quality indicators for pancreaticoduodenectomy.

Saturday Poster 10

ROBOT-ASSISTED MAJOR PANCREATIC RESECTIONS: A RETROSPECTIVE ANALYSIS OF 30 CONSECUTIVE PATIENTS.

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Introduction: Despite widespread adaptation of minimally invasive techniques to complex surgical procedures, limitations inherent in laparoscopic techniques have reduced their application to pancreaticoduodenectomy (PD). Open PD has significant postoperative morbidity and a complication rate approaching 45% in large series with a 10 day median length of stay. Robotically-assisted minimally invasive surgery offers several significant advantages compared to traditional laparoscopic approaches, including enhanced visualization in three dimensions with near 360 degree range of motion of the surgical instruments. The robotic technique allows the complex reconstruction following PD to be performed using a technique identical to the open procedure.

Methods: We conducted a retrospective review of the first thirty robotic-assisted complex pancreatic resections at the University of Pittsburgh Medical Center. All procedures were conducted by a single surgical team possessing a combination of advanced laparoscopic skills and extensive prior experience with pancreatic surgery. Cases were selected and approved by the multidisciplinary robotic surgical oncology committee. Patients with borderline-resectable pancreatic malignancy were excluded. We evaluated the perioperative events, final pathology, and complications occurring within the first 30 days on an intention to treat basis.

Results: Between October 2008 and December 2009, we performed 26 robotic pancreatoduodenectomies (PD), 3 central pancreatectomies, and 1 duodenum-preserving pancreatic head resection. Final pathology included 17 periampullary adenocarcinomas (9 pancreatic, 8 ampullary), 9 pancreatic cystic neoplasms, 3 pancreatic neuroendocrine tumors, and one chronic pancreatitis. Conversion to open was only necessary for patients undergoing PD. The PD completion rate was 74%. Reasons for conversion (7/26) to open PD included unsuspected venous involvement (3) and failure to progress (4). For the 26 patients undergoing intended robotic PD (11 male, 15 female), median age was 76.5 (range 47-85). Median operative time was 590 minutes (range 327-848 min) with 500 cc median estimated blood loss (100-2000 cc). The R0 resection rate following PD for malignancy was 85% (17/20). Median lymph node harvest was 16 (5-31). Median length of stay was 10 days (4-87 days). The rate of pancreatic fistula was 23% (6/26), but only 2 (8%) were grade C. 8 patients (38%) developed minor complications (Clavien grade 1/2), whereas 7 had major complications (33%). There was one late death (3.8%) on postoperative day 87 as the result of multiple precipitating factors.

Conclusions: This early experience suggests that robotic assisted minimally invasive pancreaticoduodenctomy can be performed with equivalent safety and oncologic outcomes to open procedures.

Saturday Poster 11

FIRST 162 WHIPPLES: DISSECTION OF THE LEARNING CURVE

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Background: The operative mortality after pancreaticoduodenectomy has decreased from 25% in the 1940's to currently less than 3%. Improved outcomes have been associated with high-volume institutions and surgeons. However, it is still unknown how a single surgeon performs immediately after training and the learning curve associated with this complex operation as he progresses from a low-volume to a high-volume pancreas surgeon.

Objective: To evaluate the learning curve of pancreaticoduodenectomy through the experience of one surgeon doing his first 162 consecutive operations.

Methods: Between July 1, 1998 and June 30, 2009, 162 consecutive Whipple procedures were performed by a single surgeon in the first 11 years of academic practice immediately after finishing residency training. A retrospective review of a prospectively maintained database was performed to determine the outcome of these patients, and to examine the evolution or learning curve of this operative experience. The study period was divided into low volume (≤ 11/year, 1998-2004, n =19 cases) and high volume (≥ 23/year, 2005-2009, n =143 cases) and comparisons were made between these two eras. Comparison of continuous variables was performed using the Wilcoxon rank sum test, and comparison of categorical variables was performed using Fisher's exact test.

Results: Patients in the low-volume era were more likely to be younger, 58 vs. 64 p=0.03, male, 12 (63%) vs. 58 (41%) p=0.08, and have coronary artery disease, 7 (37%) vs. 21 (15%) p=0.025. Otherwise, there were no significant differences in the demographics, comorbidities, or indications for surgery between the low and high volume eras. A pancreatic duct stent was used for the pancreaticojejunostomy less frequently in the low-volume era, 1 (6%) vs. 47 (33%). Mean EBL was higher in the low-volume era, 942 ml vs. 570 ml p=0.001, and more patients were transfused, 8 (44%) vs. 26 (18%) p=0.027. Patients in the low-volume era were more likely to have one or more complications, 11 (58%) vs. 48 (34%) p=0.045. The incidence of specific complications was similar in the low-volume and high-volume eras with the exception of wound infection, 6 (32%) vs. 13 (9%) p=0.012, and bile leak, 2 (11%) vs. 1 (0.7%) p=0.037. Postoperative length of stay was higher in the low-volume era, 16 days vs. 9 days p=0.006. There was only one death in the series (high-volume era) for a mortality rate of 0.66% for the entire time period. For patients with adenocarcinoma, there was no difference in disease free or overall survival between the two eras.

Conclusions: Examination of the learning curve of a single surgeon indicates that experience is associated with decreased blood loss, transfusions, complications, and a faster recovery. However, a low-volume surgeon can perform this procedure with acceptable outcomes during the learning curve. Identification of specific practice changes associated with improved outcomes may shorten the learning curve for others.

Saturday Poster 12

OBESITY INCREASES COMPLICATIONS FOLLOWING WHIPPLE

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Background: Many surgeons believe that obesity increases complications following pancreatic resection, but the literature has offered conflicting results. One possible explanation for the varied results is that obesity measured by body mass index (BMI) fails to account for fat distribution. We hypothesized that intra-abdominal/visceral fat (VF) may be a better predictor of postoperative complications than BMI.
Methods: VF measurements from preoperative CT scans and outcome data from patients undergoing a pancreaticoduodenectomy were reviewed. VF was retrospectively recorded as distance from the posterior aspect of the kidney to the abdominal wall. Logistic regression was used to determine independent predictors of complications.
Results: Among 160 patients who underwent pancreaticoduodenectomy, 132 had preoperative CT/MRI imaging available for review. Mean patient age was 62.9±1 years and mean VF was 15.5±1mm. Eighty-one patients (51%) were overweight with preoperative BMI ≥ 25kg/m^2. Overall, 55 patients (34%) had one or more postoperative complications, and fistula (ISGPF Grade B/C, 17.5%), ileus (12.5%), and wound infection (10.6%) were the most frequent. Overweight patients experienced a two-fold increase in risk of complication compared to those with BMI < 25kg/m^2 ($p< 0.029$, Table 1). Increasing quantity of VF was associated with a trend toward increased complications, but the difference was not significant even in patients with the greatest amount of VF (highest tercile).

Multivariate Odds Ratio for Predicting Postoperative Complications

Factor		95% C.I.for OR		
	Odds Ratio[1]	Lower	Upper	p
Visceral Fat (overall)				.516
Visceral Fat Tercile 1 (<7.85mm)	1 (reference)			
Visceral Fat Tercile 2 (7.86-19.4mm)	1.248	.499	3.117	.636
Visceral Fat Tercile 3 (>19.4mm)	1.849	.639	5.344	.257
BMI <25kg/m^2	1 (reference)			
Overweight(BMI>=25kg/m^2)	2.189	1.085	4.416	**.029**

[1]Odds ratios adjusted for demographics and comorbidities

Conclusion: Obesity may increase the risk of pancreaticoduodenectomy regardless of the visceral distribution of fat.

Saturday Poster 13

A NEW STANDARDIZED TECHNIQUE FOR PANCREATICOJEJUNOSTOMY CAN AVOID PANCREATIC FISTULA WITHOUT PANCREATIC DUCT STENTING

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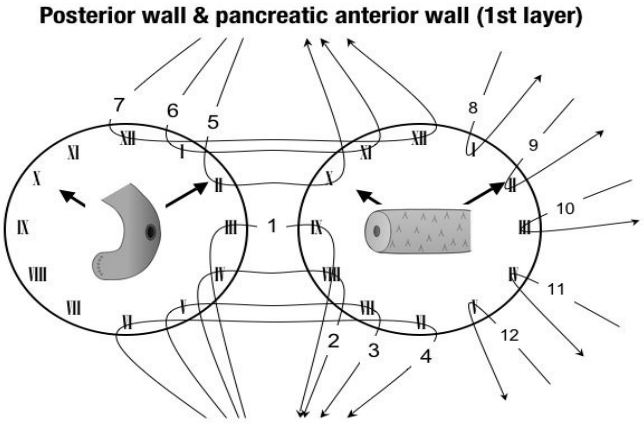
<Introduction> Pancreaticojejunostomy after pancreaticoduodenectomy is still the Achilles heel of pancreatic surgery. To prevent pancreatic fistula after pancreaticojejunostomy, we developed a new standardized technique that we term “pair watch suturing technique (PWST)” three years ago, resulting in very good results. Here we report the results of prospective study to evaluate the validity of PWST focusing on the effect of pancreatic tube insertion.
<Patients and Methods> During anastomosis, we image the faces of pair watches on the jejunal hole and pancreatic duct as shown in the figure. By using PWST we can always put 12 stitches regardless of the caliber of main pancreatic duct. From April 2007 to April 2009, the total of 55 cases undergoing PWST were nonrandomly prospectively divided into the following two groups: stent group (a stent tube was inserted in 28 cases) and no-stent group (27 cases). In all cases amylase levels in drain solution was measured on postoperative day 3 and 7. All cases were evaluated by the grade definition of postoperative pancreatic fistula (ISGPF). We performed several statistics analyzes on operation time, blood loss, the caliber of main pancreatic duct, pancreatic consistency, serous/drain amylase ratio, incidence rate of pancreatic fistula and other complications, and postoperative hospital stay between the two groups.
<Results> The caliber of main pancreatic duct and pancreatic consistency did not differ in both group. There were no cases belonging to Grades B and C pancreatic fistula. The incidence of Grade A was 10.7% in stent group and 14.8% in no-stent group, showing no significant difference. The median postoperative hospital stay was significantly shorter in no-stent group. Other factors showed no significant differences.
<Conclusion> The pancreatic duct stenting has been generally believed to decrease the rate of pancreatic fistula. However, our study reveals that PWST provide a safe pancreaticojejunostomy regardless of pancreatic duct stenting.



Saturday Poster 14

QUALITY OF LIFE OUTCOMES AFTER THE WHIPPLE PROCEDURE FOR PARADUODENAL PANCREATITIS

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Introduction
"Paraduodenal Pancreatitis" (PDP) is the recently described clinico-pathologic entity that unifies cystic dystrophy of heterotopic pancreas, groove pancreatitis, pancreatic hamartoma of the duodenum, and paraduodenal wall cyst. PDP afflicts a predominantly male population, aged 40-50 with a history of alcohol abuse and a predisposing common anatomic variant of the pancreatic minor papilla. It is unknown how patients with PDP fare with intraoperative management compared to those with non-PDP chronic pancreatitis. Nor is it known which operation should be offered. We compared quality of life (QOL) scores for patients with PDP after pancreaticoduodenectomy compared to a control population with non-PDP chronic pancreatitis.

Methods
Data including basic demographics and clinical characteristics were collected for all patients who received a histopathological diagnosis of PDP and clinically matched controls with chronic pancreatitis, all of whom received Whipple procedures. All patients were operated on by the same surgeon and diagnosed by the same pathologist. Patients were then contacted by phone and asked to participate in the EORTC-Q30 and PAN26 quality of life surveys by one investigator. The phone interviews were uniformly conducted during evenings and weekends. Statistical analysis was univariate and p-values were derived from Mann-Whitney testing.

Results
Ten patients with PDP were identified and matched with 9 controls. There were no significant differences in age, gender, length of stay, and time of interview from operation. The PDP group had more patients with multiple comorbidities. PDP had consistent preoperative imaging features: a poorly vascular 'mass' involving the head and uncinate process of the pancreas.

Using the Q30, PDP patients had global QOL scores of 69.2 +/- 14.7 while controls had scores of 45.4 + 7.3 ($p = 0.0004$). PDP patients also rated their physical and role functions higher ($p = 0.0001$ and 0.0007) and experienced fewer symptoms of fatigue, nausea and vomiting, appetite loss, and pain. By the PAN 26, PDP patients scored better for pancreatic pain, digestive symptom and sexuality questions.

Discussion
This is the first study to evaluate QOL after surgery in Paraduodenal Pancreatitis and the first to compare it to outcomes for patients with chronic pancreatitis. PDP is a meaningful diagnosis which suggests favourable outcomes after a pancreaticoduodenectomy when compared to patients with non-PDP chronic pancreatitis.

Saturday Poster 15

CHARACTERIZATION OF THE UNDEREXPRESSED GENE MGC45438 IN PANCREATIC CANCER CELL LINES

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Pancreatic cancer (PDAC) is a devastating disease. It is the third leading cancer death in Germany. The incidence in the years 2003/2004 counts 16 diseases per 100.000 inhabitants. Of all carcinomas pancreatic cancer has the highest mortality rate with one- and five-year survival rates of 25% and less 5%, irrespective of initial stage. This shows the poor prognosis of the carcinoma. So it is necessary to increase the knowledge about the molecular biology of the carcinoma to find new diagnostic and therapeutic approaches. Within our gene expression profiling experiments on microdissected PDAC and normal ductal cells we identified MGC45438 as underexpressed in pancreatic cancer tissue. Hence, we were interested to characterize the role of MGC45438 in PDAC.
At first the gene full length cDNA of MGC45438 was transferred into the transfer vector pcDNAmycHIS-A and MiaPaCa-2 and Panc-1 cells were transfected. After selection of clones with Zeocin we performed quantitative RT-PCR, Western Blot, WST, treatment with Gemcitabine, FACS-Analysis and experiments on the extra cellular matrix Matrigel.
Growth rate determination of cells on Matrigel, showed that the cell clone of Panc-1 with the highest protein expression of MGC45438 showed the least growth rate compared to the other with a lower amount of protein and controls.
In conclusion it is possible that this gene influences migration and invasion in pancreatic cancer cell lines. So the activation of MGC45438 might be an interesting approach for the therapy in pancreatic cancer. We are now in process to characterize the function of MGC45438 in pancreatic cancer in more detail.

Saturday Poster 16

LAPAROSCOPIC DISTAL PANCREATECTOMY: AN ASSESSMENT OF ONCOLOGIC PRINCIPLES

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Background: It is unclear whether laparoscopic distal pancreatectomy (LDP) is appropriate for the treatment of malignancy. Our aim was to evaluate whether LDP provides adequate oncologic assessment of tumor and lymph node (LN) status when performed for pre-malignant or malignant conditions.

Methods: We performed a retrospective review of a prospectively collected database including individuals who underwent LDP for malignant or pre-malignant conditions at our institution between 1/2007 and 1/2010. We collected patient and tumor characteristics with particular attention to factors that relate to oncologic outcomes, including margin status and LN evaluation.

Results: We identified 21 patients who underwent LDP between 1/2007 and 1/2010. 62% of the cohort was female. The average patient age was 57 years. Spleen preservation was performed in 16 (76%) cases, while en bloc splenectomy was performed in 5 (24%). One case was converted to open due to failure to progress due to body habitus. Estimated blood loss during spleen preserving LDP was 72.2 cc and for en bloc LDP and splenectomy 170 cc. Malignancy was present in 12 cases. The most common malignancy was neuroendocrine (10 cases), followed by pancreatic adenocarcinoma (1 exocrine, 1 mucinous). Non-invasive intraductal papillary mucinous neoplasms (IPMN) were identified in 5 patients and mucinous cystic neoplasms (MCN) in 4. Several patients had multiple pathologies identified. Mean tumor size was 2.5cm. Mean specimen length was 8.3cm. Margins were negative in all cases. The mean tumor margin was 1.1cm. The median number of lymph nodes evaluated overall was 2 (range 0-23). For individuals who had splenectomy, the median number of LN evaluated was 3. In cases of malignancy, the median number of nodes evaluated was 2, which was not statistically different than cases identifying benign pathology. Overall, 8 patients had no LN evaluated. For all cases with LN evaluated, the median number was 5. Pancreas fistula, as defined by the International Study Group of Pancreatic Fistula, occurred in 13 (62%) cases, 10 were Grade A (elevated drain amylase on post-operative day 3), and 3 were Grade B (requiring percutaneous drain replacement after initial operative drain was removed). There were no mortalities. Median length of stay was 4 days.

Conclusions: LDP provides a safe alternative to open resection and provides excellent control of surgical margins, however LN counts are less than expected. The use of LDP for potential malignancy should proceed with careful attention to LN evaluation to ensure optimal outcomes.

Saturday Poster 17

FACTORS INFLUENCING SURVIVAL IN PATIENTS UNDERGOING A PALLIATIVE BYPASS PROCEDURE FOR PANCREATIC ADENOCARCINOMA

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Introduction: The purpose of this study is to identify factors predictive of early mortality following palliative bypass in patients with unsuspected or known advanced pancreatic adenocarcinoma in an attempt to identify a cohort of patients who may benefit from preoperative chemoradiation therapy as opposed to upfront palliative surgery.

Methods: All patients with pancreatic adenocarcinoma who underwent a bypass procedure between 9/30/1994 and 1/31/2006 were reviewed. Patients with perioperative mortality and pathology other than pancreatic adenocarcinoma were excluded from the analysis. Univariate analysis was performed on peri-operative data to identify factors associated with early mortality. Patients having multiple factors were assigned an overall prognostic score based on the sum of these factors.

Results: Of the 397 patients with pancreatic adenocarcinoma analyzed, four factors were found to predict for early mortality (defined as death within 6 months) following palliative bypass. These factors were presence of distant metastatic disease (RR 2.59, $p<0.0001$), poor tumor differentiation (RR 1.71, $p=0.009$), severe pre-operative nausea and vomiting (RR 1.48, $p=0.013$) and previous placement of a biliary stent (RR 0.737, $p=0.048$). Patients with a sum score of 0 were significantly more likely to survive past 6 months than patients with a sum score of 1 (RR 2.71, $p<0.0001$), 2 (RR 3.70, $p<0.0001$) or 3+ (RR 5.63, $p<0.0001$).

Conclusions: In a cohort of patients undergoing a palliative bypass procedure, specific pre-operative factors can be used to identify patients who are at risk of early mortality. Patients with these characteristics may benefit from preoperative therapy as opposed to upfront palliative surgery.

Saturday Poster 18

ROUTINE USE OF A VESSEL SEALING SYSTEM DURING PANCREATICODUODENECTOMY REDUCES INTRAOPERATIVE BLOOD LOSS AND OPERATIVE TIME.

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Introduction: Recent advances in surgical instruments make complex surgical procedures safer. We tested if routine use of LigaSure™ vessel sealing system (LVSS) during pancreaticoduodenectomy (PD) would contribute to reduction of intraoperative blood loss and transfusion.
Methods: A total of 168 patients who underwent PD between 2005/10 and 2009/12 were enrolled in this study. Since 2007/04 we have been using LVSS during PD routinely. A retrospective comparison is made between 84 consecutive patients who underwent PD with LVSS and historical cohort of 84 consecutive patients without LVSS. Perioperative data were collected from the prospectively maintained database.
Results: Of 168 patients two groups were comparable in terms of patient's demographics, type of disease (malignant/benign), type of procedure (PD/PPPD). OR time, blood loss and transfusion required were significantly different between the two groups. This trend is more significant in subgroup of malignant disease than in benign disease. Overall morbidity, POPF (any grade) and LOS were not different (Table).

	PD w/o LVSS (N=68)	PD w/o LVSS (N=68)
OR time (min.)*	443 (240-845)	401 (218-829)
Blood loss (g)*	1663 (270-6760)	1185 (150-6730)
Transfusion*	82%	56%
Morbidity	49%	42%
POPF (any grade)	29%	41%
LOS (days)	34	29

*$p<0.05$
Conclusions: Using LigaSure™ vessel sealing system during PD contributes to reduction of surgical time, blood loss, and transfusion rate, making surgical procedure safer. However, postoperative morbidity and LOS were not affected.

Saturday Poster 19

PREDICTIVE FACTORS OF MALIGNANCY IN PATIENTS AFFECTED BY INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS

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Introduction

Intraductal Papillary Mucinous Neoplasms (IPMNs) are tumors with an intraductal proliferation producing mucin with a cystic dilation of main pancreatic duct and/or secondary ducts. This kind of tumor accounts for 0.5-9.8% of all the tumors of the exocrine pancreas. According to the literature, the predictive factors of malignancy are symptoms, the involvement of main/secondary pancreatic duct, mural nodes and the grade of main pancreatic duct dilation. In the last years the role of new prognostic factors for patients affected by IPMNs such as the intracystic CEA level have been investigated. The goal of our study was to evaluate the role of clinicopathological features and intracystic CEA and CA 19-9 level as preoperative predictive factors of malignancy in a series of 22 patients affected by IPMN.

Methods

Twenty-two patients affected by IPMN were consecutively observed and surgically treated at the Campus Bio-Medico University of Rome. In 14 cases, the level of CEA and Ca 19-9 levels in the pancreatic juice was evaluated. The clinicopathological features (age, presence of symptoms, diameter of main pancreatic duct, mural nodes and dimension of the tumor) and the level of CEA and Ca 19-9 levels of pancreatic juice were correlated with histological findings.

Results

Our results showed that 86.4% of our patients were symptomatic. The histological findings revealed IPMN adenoma in 2 patients (9.1%), IPMN borderline in 5 patients (22.7%), IPMN carcinoma in situ in 1 case (4.6%) and IPMN carcinoma in 14 patients (63.6%). Among the clinicopathological features only the presence of mural nodes and tumor diameter resulted significantly predictive factors of malignancy. On the opposite intracystic CEA and Ca 19-9 levels did not result affecting patients' prognosis.

Conclusion

Our findings suggest that tumor size and the presence of mural nodule resulted predictive prognostic factors for diagnosis of malignancy in patients affected by main duct/combined IPMN. It is necessary to perform further studies to identify other parameters able to obtain an earlier diagnosis with possible improved results of surgical treatment.

Saturday Poster 20

IS THERE A LEARNING CURVE FOR PANCREATICODUODENECTOMY AFTER FELLOWSHIP TRAINING?

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Introduction: There are a number of studies that examine learning curves for advanced laparoscopic procedures. However, limited data exist regarding a learning curve for open pancreaticoduodenectomy, a complex procedure with potentially significant morbidity and mortality. This study was undertaken to examine whether a learning curve exists for the performance of a pancreaticoduodenectomy by a fellowship-trained surgeon.

Methods: Review of the outcomes of a single surgeon's first 60 pancreaticoduodenectomies after completion of specialty training in pancreatic surgery (61 pancreaticoduodenectomies). Comparisons were made between the first 30 and last 30 cases.

Results: From July of 2004 through June of 2009, 60 pancreaticoduodenectomies were performed, with 30 being completed in the final 15 months. There were no differences in median age, 69 vs. 65 years, or gender, 47% vs. 50% female, between the two groups. Median estimated blood loss did not differ between the groups, 778 vs. 800 ml, but median operative time dropped from 463 minutes to 388 minutes from the first 30 cases to the second 30, $p = 0.002$. Two perioperative mortalities (6.7%) occurred in the first group and none in the second group, but the difference was not significant. Major complications (Clavien grade 3-4) occurred in 27% and 17% of cases, respectively, and clinically relevant pancreatic fistulae (ISGPF grade B/C) occurred in 23% and 13% of cases, respectively; but the differences were not significant. Median length of stay fell from 10 days in the first group to 7 days in the second group, $p = 0.02$, but readmission rate did not change, 18% vs. 20%, respectively. Final pathology was malignant in 93% of patients in the first group and 77% in the second group, $p = 0.15$. The R1 resection rate for patients with pancreatic ductal adenocarcinoma was 25% in the first group and 24% in the second group. The percentage of patients with periampullary adenocarcinoma who received adjuvant therapy increased from 58% in the first group to 91% in the second group, $p = 0.02$. A separate analysis comparing the first 20 cases to the last 20 cases revealed similar, significant changes in operative time, length of stay, and receipt of adjuvant therapy.

Conclusion: Even with extensive training in pancreatic surgery, a learning curve exists for the performance of pancreaticoduodenectomy. With experience, improvements were made in operative time, but more importantly in patient outcomes including length of stay and receipt of adjuvant therapy

Saturday Poster 21

MULTIDISCIPLINARY EVALUATION FOR PANCREATIC CANCER IN AN ACADEMIC COMPREHENSIVE CANCER CENTER

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INTRODUCTION: Pancreatic cancer is a therapeutic dilemma requiring multidisciplinary specialty care. The spectrum of evidence-based management includes variable combinations of surgical intervention, neoadjuvant and/or adjuvant chemotherapy, radiotherapy, palliation of pain, psychological counseling, prevention of malnutrition, and evaluation for clinical research. Traditional approaches to evaluation often require multiple visits to different specialists that delay definitive treatment, create heterogeneous care plans within the same institution, and raise barriers to data collection and clinical trial enrollment. The purpose of this study was to review the performance of multidisciplinary evaluation at an academic comprehensive cancer center organized to care for complex diseases in a hub-and-spoke network of 19 local cancer centers. METHODS: New patients were evaluated in a single clinic visit by specialists from surgical, medical, and radiation oncology, diagnostic radiology, gastroenterology, palliative care, nutrition, research, and social work. Pertinent findings were reviewed comprehensively by the multidisciplinary panel. The eventual treatment plan was generated according to an evidence-based clinical pathway established by the specialist team and implemented across the extended network. RESULTS: 154 new patients were evaluated between March and December of 2009 by the UPMC Specialty Care Center (SCC) for pancreatic cancer. Patients traveled a mean distance of 44 miles (IQR 9-61 miles), and 19 patients (12%) traveled in excess of 100 miles. Clinical stage at presentation included: stage I (0.6%), stage II (26%), stage III (34%), and stage IV (31%). Treatment was initiated in 124 patients (81%), with 70 patients treated at a UPMC facility (45%) and 54 patients referred locally (35%). Twenty patients (13%) received no treatment; eight patients (5%) were seen for a second opinion, and one patient (1%) is awaiting a phase I trial. 148 patients (96%) entered the research registry; 50% of eligible patients were subsequently enrolled in a clinical trial. According to patient satisfaction surveys, 96% of patients were satisfied with their evaluation and the efficiency of the multidisciplinary process. The average time from initial evaluation to initiation of definitive oncologic care was 17 days, as compared to an average delay of 49 days before the implementation of the multidisciplinary clinic. DISCUSSION: Specialty care for complex malignancies can be effectively administered at great distances by means of a hub-and-spoke academic tertiary cancer center. This approach maximizes patient satisfaction, reduces delays in therapy, and promotes evidence-based specialty care and clinical research for pancreatic cancer.

Saturday Poster 22

THE ROLE OF CHEMORADIATION IN THE MANAGEMENT OF LOCALLY ADVANCED PANCREATIC ENDOCRINE NEOPLASMS

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Introduction: Radiation therapy has historically not played a prominent role in the treatment regimen for pancreatic neuroendocrine neoplasm. We provide a single-institutional series of patients with locally advanced pancreatic neuroendocrine neoplasm who were treated with radiation therapy in an attempt to down-stage for surgery or for recurrent post-surgical disease.

Methods: We identified 6 patients and used the institution's electronic patient record to evaluate patient and disease history characteristics; including age, gender, race, chemoradiation protocol, down-staging success, surgical intervention and survival.

Results: The group of patients consisted of 3 women and 3 men with a mean age of 57 years (range 38-67 years). Of the three who were taken to the operating room, one was down-staged neo-adjuvantly with chemoradiation therapy and the two others were treated with chemoradiation therapy due to local recurrence. The patient who was downsized before surgery eventually passed away from cancer within 15 months of diagnosis. Of the two who received chemoradiation therapy for recurrence, one has no evidence of disease 19 months out from presentation and the other is being treated for hepatic metastases 30 months out from surgery. Of the three other patients who received chemoradiation in an attempt to downsize, one was lost to follow-up and the other two are alive at 3 and 16 months out from radiation with evidence of downsized tumor. The chemoradiation therapy consisted of approximately 50 Gy, with or without oral Xeloda. This regimen was associated with only grade 1/2 toxicities.

Conclusions: While radiation therapy is frequently used in pancreatic adenocarcinoma, there is still no experience published in the literature on radiation therapy for locally advance pancreatic neuroendocrine neoplasms. While our experience is small, it demonstrates that there may be a potential role for radiation therapy in selected patients either to down-stage their disease for surgery or to palliate surgical recurrence.

Saturday Poster 23

DIAGNOSTIC LAPAROSCOPY FOR PANCREATIC CANCER IN AN MRI DRIVEN PRACTICE: WHAT'S IT WORTH?

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Introduction:
For many patients with pancreatic cancer, CT is inadequate in determining unresectability; 10-48% of patients deemed resectable receive an unnecessary laparotomy. Accordingly, many groups have studied the role for diagnostic laparoscopy (DL) though none have evaluated it in an MRI driven practice.
Methods: All MRI's administered for suspected pancreatic cancer between December 2004 and 2008 were evaluated. Radiographic diagnoses were prospective judged resectability based on the presence of metastases and relationship of the tumor with the surrounding vasculature. Unresectable disease received endoscopic biliary and duodenal stenting. Resectable and borderline disease received Whipples and double bypasses if unresectable intraoperatively.

We performed a decision analysis for the cost-effectiveness of incorporating DL. We queried our billing database to render average costs for all inpatients with pancreatic cancer who received Whipples, double-bypasses and double-stenting procedures. We did not include professional fees. The marginal cost of DL was derived from the itemized costs of the materials, space and ancillary staff, presuming routine utilization, no missed metastases, and no complications.

Results
Preoperative MRI deemed 94 patients' tumors resectable; 86 agreed to a laparotomy. Six patients were found to have metastases intraoperatively and 15 patients had unresectable disease (vascular involvement or benign pancreatitis) and thus received double-bypass procedures for which the average total cost of the hospitalization was $21,957.18. Whipples were provided to 65 patients at an average cost of $26,122.43.

DL would thus be offered to 86 patients. For the 6 patients with metastases, it would be the only operation ($3604.07). This would be added to the cost of endoscopic stenting procedures, which results in an average total cost of hospitalization of $18,451.41. For the patients without metastases, the marginal cost of DL before a laparotomy would be $2651.71, which we added to the total costs above.

Conclusions
For DL to be cost-effective, it would have to increase the rate at which we diverted patients to the GI lab for palliative stenting. In our model, DL would increase our costs by $191,072.18, equivalent to the total cost of hospitalization for treating 7 patients with Whipple procedures. Given our rate of missed metastases – 6% - and presuming perfect yield from DL, 15 patients would have unnecessary DL for every patient with occult metastases. For DL to be cost-effective, its intraoperative yield would have to be 70%.

Saturday Poster 24

TIMING OF CHOLECYSTECTOMY FOR MILD BILIARY PANCREATITIS: A SYSTEMATIC REVIEW

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Background: Current guidelines recommend early cholecystectomy following mild biliary pancreatitis. There is, however, no consensus on the definition of "early": during index admission, within 2 weeks or within 4 weeks after discharge. In recent years, many cohort studies have been published addressing this topic. The current study represents the first systematic review.

Methods: We searched the MEDLINE library since 1992. Inclusion criteria were: 1) cohort of patients undergoing cholecystectomy after mild biliary pancreatitis; 2) essential outcomes reported: time between hospital discharge and cholecystectomy, number of readmissions during waiting period, number or recurrent acute biliary pancreatitis during waiting period, surgical complications (bile duct injury, bleeding) and mortality. Exclusion criteria were: 1) cohort <5 patients; 2) cohort including severe pancreatitis without results for mild pancreatitis reported separately.

Results: 6 out of 811 studies were included, describing 11 different "timing" cohorts in 791 patients. Cholecystectomy was performed during index admission in 385 patients (49%), without any readmissions for biliary complications. In 7 cohorts, consisting of 406 patients, the median time between discharge and cholecystectomy was 44 days (range 14-68 days). Readmission rate was 76/406 (19% vs 0%, P<0.0001); 40 patients (10%) with recurrent biliary pancreatitis, 11 patients (3%) with acute cholecystitis and 25 patients (6%) with biliary colics. Complications occurred in 6 patients (2%) in the early group and in 11 patients (3%) in the delayed group. In both groups, no mortality after cholecystectomy was described.

Conclusion: This systematic review suggests that patients with mild biliary pancreatitis, should undergo cholecystectomy during the index admission as delayed cholecystectomy increases the risk for readmissions, especially for recurrent biliary pancreatitis.

Saturday Poster 25

FLUORESCENCE LAPAROSCOPY AS A NOVEL TECHNIQUE FOR PANCREATIC TUMOR DETECTION IN AN ORTHOTOPIC MOUSE MODEL

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Background: Fluorescent labeling of tumors affords easier identification of both primary and metastatic lesions, which may bear significant diagnostic, staging, and therapeutic implications. Accurate staging of pancreatic cancer, in particular, is of paramount importance. We report here the usefulness of fluorescence laparoscopy in identifying green fluorescent protein (GFP)-expressing tumors in an orthotopic mouse model of human pancreatic cancer.
Methods: An orthotopic mouse model of human pancreatic cancer was established by injecting GFP-expressing MiaPaca-2 human pancreatic cancer cells into the pancreas of 6-week old female athymic mice. On post-operative day 14, a diagnostic laparoscopy using both white and fluorescent lights was performed in the animals. Our goal was to achieve a fluorescent mode that would allow both easy detection of the fluorescent tumor and the ability to maintain a clear view of the surrounding tissue. To achieve this, a standard laparoscopic system was modified in the following manner: a 480-nm short pass excitation filter was placed between the light cable and the laparoscope, and a 2-mm thick glass emission filter that allows leakage of 1% of the background was placed between the laparoscope and the camera. A set of camera and recording system that allows variable exposure time and gain setting in the controlling software was used. For the purpose of our experiment, the exposure time was set to 110 msec and the gain to 97. A 3-mm 0-degree laparoscope was used in the mice, and their abdomen was gently insufflated to 2 mm Hg via a 22-gauge angiocatheter that was secured via a suture. The animals were then sacrificed, and the identified tumors were collected and processed for histology. The experiments were performed in triplicate.
Results: Fluorescence laparoscopy allowed easy and fast identification of a brightly fluorescent tumor in the pancreatic body. By employing the specific parameters above mentioned, a clear background was visible along with the fluorescent tumor under the fluorescent light mode. This could proffer an advantage in allowing exact localization of the lesions, eliminating the need to flip back and forth between white and fluorescent lighting, under which the background is usually so darkened that it is difficult to maintain spatial orientation.
Conclusion: The use of fluorescence laparoscopy permits the easy, clear, and rapid identification and localization of tumors that are labeled with fluorescent proteins or other fluorophores. Fluorescence laparoscopy may thus play a useful role in the diagnosis and staging of pancreatic and other aggressive gastrointestinal cancers.

PANCREATIC SURGERY: EVOLUTION AT A HIGH-VOLUME CENTER

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Background: Over the past decade, advances in imaging have identified more asymptomatic pancreatic cysts. More recently, minimally invasive pancreatic procedures have been developed. In addition, volume/outcome data for pancreatectomy have led to increased referrals to regional centers. However, no analysis has been performed to determine whether the indications for surgery or the types of pancreatic operations have changed at a high-volume center. Therefore, the aims of this analysis were to determine whether the spectrum of disease or the types of pancreatic procedures have evolved with regionalization.
METHODS: From 1996 through 2009, 1,884 pancreatic procedures were performed at our high-volume center. These procedures included 1,006 pancreatoduodenectomies, 513 distal pancreatectomies, 166 pancreatojejunostomies, 61 Beger/Frey procedures, 53 central pancreatectomies, 47 total pancreatectomies, and 38 enucleations. The underlying diagnosis, the pancreatic procedure, and the 30-day mortality were ascertained. Outcomes for 1996-2003 were compared to those for 2004-09.
RESULTS: The number of patients undergoing pancreatic operations increased significantly ($p<0.01$) from 90/yr in 1996-03 to 212/yr in 2004-09. Interestingly, no change was observed in the spectrum of disease: perimampullary cancer 37 vs 38%, chronic pancreatitis 20 vs 17%, and cystic neoplasms 20 vs 17%. However, the types of procedures changed with an increase in pancreatoduodenectomy (50 vs 55%, $p<0.03$) and a decrease in pancreas preservation procedures (pancreatojejunostomy, Beger/Frey, central, enucleation) (21 vs 14%, $p<0.01$). In addition, pylorus preservation (64 vs 82%, $p<0.01$), splenic preservation (2 vs 23%, $p<0.001$), and distal pancreatectomy performed laparoscopically (0 vs 32%, $p<0.001$) all increased in 2004-09. Moreover, 33% of 98 distal pancreatectomies, pancreatojejunostomies, enucleations and central pancreatectomies as well as 5% of 131 pancreatoduodenectomies were performed laparoscopically this past year. Finally, 30-day mortality has improved from 2.5% in 1996-2003 to 1.7% in 2004-09.
CONCLUSIONS: These data suggest that in the modern era of pancreatic surgery 1) the spectrum of disease has not changed but 2) relatively more pylorus preservation with pancreatic head resections, splenic preservation with distal pancreatectomy and laparoscopic pancreas preserving procedures are being performed. We conclude that patients with pancreatic disease requiring surgery have benefited from regionalization.

PATIENTS DELAY AND ETIOLOGY IN THE DEVELOPMENT OF SEVERE PANCREATITIS. A RETROSPECTIVE STUDY FROM A REFERRAL HOSPITAL.

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Background: Acute pancreatitis is associated with high mortality and morbidity rates and several factors predispose for complications and death during an episode with acute pancreatits. This study addresses the effect of prehospital time from symptom onset to hospital admission on the development of severity of acute pancreatitis.
Method: Data from an ongoing study in a referral hospital in Norway were analyzed. Patients admitted with acute pancreatitis between 01.01.2000 and 31.12 2007 were identified and their records reviewed. 373 of 470 (79%) patient records contained sufficient data for inclusion in this study. Time from onset of symptoms to admission at hospital was recorded. 43(11.5%) patients had a severe course of the disease based on admission to intensive care unit or death in hospital. 330(88.5%) patients had a mild course of the disease. The median age of patients with a mild or severe course of disease was 57 (range 7 – 98) and 62 (31 – 95) years respectively ($p< 0.05$). Women represented 37% of patients with severe and 49% with mild pancreatitis ($p=0.133$).
Results: Time between debut of symptom and hospital admission was less than 24 hours in 133(36.0%) patients, whereas 240(64.0%) had a delay of more than 24 hours. The incidence of severe pancreatitis was 8.3 % amongst early and 13.3% amongst late admitted patients ($p=0.143$). Potential confounders to treatment delay were entered into a multivariate logistic regression model. The OR for severe pancreatitis amongst patient with a delay of more than 24 hours was 1.85 (95% CI: 0.84 – 4.1) ($p=0.125$). Age with an OR 1.023 (95% CI: 1.003 – 1.045) ($p=0.027$) and etiology ($p=0.003$) were the only significant factors that predicted sever pancreatitis. With gallstones as reference (1), patients with alcohol OR 4.822 (95% CI: 1.468 – 15.833) ($p=0.010$) or miscellaneous etiologies OR 4.366 (95% CI: 1.800 – 10.592) ($p=0.001$) for acute pancreatitis were at a higher risk of developing severe pancreatitis. Gender OR 1.412 (95% CI: 0.683 – 2.918), former episodes of acute pancreatitis OR 0.206 (95% CI: 0.026 – 1.625), comorbidity OR 0.840 (95% CI: 0.321 – 2.199), and smoking OR 1,206(95% CI: 0.571 – 2.548) were not significantly associated with a severe course.
Conclusion: Our preliminary data show that increasing age and different etiologies are factors that increase the risk of developing severe acute pancreatitis. Alcohol and miscellaneous etiologies show a four-fold increased risk of developing severe acute pancreatitis when compared to gallstones associated disease. Prehospital treatment delay was not associated with severe pancreatitis.

DELAYED GASTRIC EMPTYING AFTER PYLORUS-PRESERVING PANCREATICODUODENECTOMY. VALIDATION OF ISGPS CLASSIFICATION AND ANALYSIS OF RISK FACTORS

0G. Malleo, S. Partelli, S. Crippa, G. Butturini, R. Salvia, R. Rossini, M. Bacchion, C. Bassi From The General Surgery B Unit, G.B. Rossi Hospital, University of Verona – Verona, Italy

BACKGROUND: delayed gastric emptying (DGE) is a major complication after pancreaticoduodenectomy (PD), and leads in turn to patient discomfort, prolonged hospitalization and increased costs. Its incidence, clinical presentation and risk factors widely varied in past studies due to the lack of an objective definition.

AIM: To evaluate our experience with DGE after PD employing the ISGPS consensus definition, and to clinically validate the definition itself and the grading system.

METHODS: Demographic, pathological and surgical details from 260 consecutive patients undergone PD at our Institution between November 2007 and October 2009 were analyzed. Clinical presentation and grading of DGE, postoperative parameters associated with DGE and risk factors were evaluated with uni- and multivariate models.

RESULTS: Perioperative mortality was nil, surgical morbidity was 41.5%. The incidence of DGE was 13.8%. Among DGE patients, 44% were grade A, 50% grade B and 5.6% grade C. Discontinuation of nasogastric intubation ($p<0.0001$), resumption of a solid diet ($p<0.0001$), time to passage of stool ($p=0.002$) and hospital discharge ($p<0.0001$) occurred significantly later in DGE patients. Total parenteral nutrition was mostly required in grades B/C. In the univariate analysis, abdominal collections ($p=0.0001$), pancreatic fistula ($p<0.0001$), biliary fistula ($p=0.002$), pulmonary complications ($p<0.0001$), and sepsis ($p=0.002$), were associated with DGE. Only abdominal collections ($p=0.009$), pancreatic fistula ($p<0.0001$) and sepsis ($p=0.024$) were associated to clinically relevant DGE. There was no association with the route of duodeno-jejunal anastomosis (antecolic versus retrocolic). In the multivariate analysis, pancreatic ($p=0.004$) and biliary fistula ($p=0.039$) were independent risk factors for DGE.

CONCLUSIONS: ISGPS classification and the grading system well correlate with clinical course and are feasible for patients management. Main risk factors for DGE seem to be pancreatic and biliary fistulas.

ADJUVANT CHEMORADIATION FOR ADENOCARCINOMA OF THE BODY AND TAIL OF THE PANCREAS: THE JOHNS HOPKINS EXPERIENCE.

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Department of Surgery[4], Department of Medical Oncology[5] , Department of Pathology[6]
The Sol Goldman Pancreatic Cancer Research Center
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Abstract:
Background: To examine the effect of adjuvant 5-FU-based chemoradiation therapy (CRT) after distal pancreatectomy in a prospectively collected database of patients with adenocarcinoma of the distal pancreas.

Methods: All patients underwent curative resection for adenocarcinoma of the distal pancreas between December 1985 and June 2006. Patients who received adjuvant CRT were compared with those who underwent surgery alone. Kaplan-Meier techniques were used to determine survival estimates and log-rank test were used to make comparisons between groups.

Results: 123 patients underwent distal pancreatectomy. 29 patients were excluded for either: distant metastases at the time of surgery (N= 12, 9.8%) or prior to adjuvant therapy (N=11, 8.9%), death within 2 months of surgery (N=2, 1.6%), or CRT treatment status was unknown (N=4, 3.2%). Of the remaining 94 patients, 72% received adjuvant 5-FU based CRT and 28% underwent surgery alone. Overall median survival was 16.7 months (95% CI, 13.7 to 21.9 months). The groups were similar with respect to tumor size, nodal status, margin status. Patients with a margin-positive resection (p=0.01) or lymph node metastases (p=0.05) had worse overall survival. There was no significant difference in overall survival between patients treated with adjuvant CRT versus surgery alone (p=0.79). An exploratory sub-group analysis suggested a potential survival benefit of adjuvant CRT in patients with lymph node metastases ($p < 0.01$).

Conclusions: Nodal metastases or margin positive disease resulted in poor survival. Adjuvant CRT did not increase survival when compared to surgery alone, however patients with node positive disease appear to benefit from CRT.

Saturday Poster 30

PREOPERATIVE MECHANICAL BOWEL PREPARATION DOES NOT OFFER A BENEFIT FOR PATIENTS UNDERGOING PANCREATICODUODENECTOMY

H. Lavu, E. P. Kennedy, R. E. Mazo, R. J. Stewart, C. Greenleaf, D. R. Grenda, P. K. Sauter, B. E. Leiby, S. P. Croker, C. J. Yeo
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Introduction:
Mechanical bowel preparations (MBP) are commonly administered preoperatively to patients undergoing pancreaticoduodenectomy (PD), however their effectiveness over a clear liquid diet preparation (CLD) remains unclear. The aim of this study was to determine if any differences exist in the outcomes of patients undergoing PD based upon the administration of a preoperative MBP.
Methods:
In this retrospective review, we analyzed the clinical data from 100 consecutive PDs performed on patients receiving preoperative MBP from March 2006 to April 2007, and compared them to 100 consecutive patients who received a preoperative CLD from May 2007 to March 2008.
Results:
There were no significant differences between the MBP and CLD groups in the rates of pancreatic fistula (13% versus 14%, $p = 1.0$), intra-abdominal abscess (11% versus 13%, $p = .83$), or wound infection (9% versus 8%, $p = 1.0$) (Table 1). There were trends toward increased urinary tract infections (13% versus 5%, $p < .08$) and Clostridium difficile infections in the MBP group (6% versus 1%, $p = .12$). Median length of postoperative hospital stay was 7 days in each group and the 12-month survival rates were equivalent (74% versus 75%, $p = 1.0$).
Conclusion:
There appears to be no clinical benefit to the administration of a preoperative MBP for patients undergoing PD. Patients who receive MBP show no significant differences in the rates of pancreatic fistula formation, intra-abdominal abscess, wound infection, delayed gastric emptying, length of stay, or one year survival. Patients undergoing MBP did have a trend toward higher rates of urinary tract and Clostridium difficile infections, suggesting that MBP may have a deleterious effect. We no longer use MBP in our patients.

Table 1- Mechanical Bowel Prep versus Clear Liquid Diet in Patients Undergoing

				***p*- Value**
Total	200(100)	100	100	
Complications n(%) *	77 (38.5)	40	37	0.77
P. Fistula	27 (13.5)	13	14	1.00
Intra-abdominal Abscess	24 (12)	11	13	0.82
Cardiac	21 (10.5)	11	10	1.00
UTI	18 (9)	13	5	0.08
Wound infection	17 (8.5)	9	8	1.00
C. diff. colitis	7 (3.5)	6	1	0.12
DGE	3 (1.5)	2	1	0.59
DVT	2 (1)	1	1	1.00
Length of Stay (days)				
Median	7	7	7	0.66
Range	3 to 59	3 to 45	5 to 59	----
Readmissions n (%)	24 (12)	10	14	0.39
1 Year Survival (%)	149 (74.5)	74	75	1.00

MBP, Mechanical Bowel Preparation Group; **CLD**, Clear Liquid Diet Group;
PPPD, Pylorus Preserving Pancreaticoduodenectomy; **EBL**, Estimated Blood Loss;
DGE, Delayed Gastric Emptying; **P. Fistula**, Pancreatic Fistula; **UTI**, Urinary Tract Infection;
C. diff. colitis, Clostridium difficile colitis; **DVT**, Deep Venous Thrombosis.
* Number of patients with one or more complication

Saturday- Poster #31- Professor Rounds 3:25 – 3:50 pm

CHARACTERISATION OF A NEWLY ESTABLISHED PATIENT-DERIVED PANCREATIC ADENOCARCINOMA XENOGRAFT COLLECTION

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Introduction: At lot of studies in the field of pancreatic cancer research are using the few commercially available pancreatic cancer cell lines. Over the time and with growing passage number, changes in the biology of these cell lines might occur. Aim of this study was to establish new, patient derived xenograft modells and cell lines of pancreatic cancers.

Methods: **In this study, more than 80 primary pancreatic carcinoma samples from patients were transplanted subcutaneously (s.c.) into NMRI nude mice. In most cases, tumor material from chemonaive patients with defined histology and staging was used for implantation. Tumor clonogenic assay and in vivo testing was used to evaluate chemosensitivity of the tumor models. Histological stains, RNA expression analysis (Affymetrix) and mutational analysis were performed for further characterisation of the tumor models.**

Results: **Up to now more than 20 new xenograft models and 4 cell lines could be established. In general, the histology of the primary tumor was comparable to that of established xenograft. Chemosensitivity *in vivo* was evaluated by treatment of tumor bearing nude mice with 5-FU, Gemcitabine, Erlotinib and RAD 001. In most studies, tumor growth was inhibited with best T/C values > 50%, highlighting the general chemoresistance of pancreatic cancer. Only for PAXF 1872 and PAXF 1998, a high sensitivity towards Gemcitabine was evident with best T/C values of 8% and 3.8%, respectively. There was no correlation to the RNA-Expression of gemcitabine related proteins (transporters, metabolisation). In general, there was also no correlation of response to Erlotinib with EGFR expression status. A more broad chemosensitivity profile was established with the *ex-vivo* clonogenic assay. Interestingly, several tumors responded strongly to treatment with Rapamycin. Mutational analysis of p53 (exons 4 to 10) revealed only 4 out of 13 models as p53 wild-type. Nevertheless, the p53 pathway is dysregulated in all tumor models and for selected tumors, an aneuploid cell population was identified by *ex vivo* cell cycle analysis.**

Conclusion: In summary, a unique collection of patient-derived pancreatic xenograft models has been established. These models probably are closer to the clinic than the usually used cell lines, they are available for translational research as well as in vivo and in vitro testing of new compounds.

Saturday- Poster #32- Professor Rounds 3:25 – 3:50 pm

INTRAVENOUS DELIVERY OF THE PLASMA FRACTION OF STORED PACKED RED BLOOD CELLS PROMOTES PANCREATIC CANCER GROWTH IN IMMUNOCOMPETENT MICE

C. C. Barnett, Jr.[1], A .W. Beck[2], S. E. Holloway[3], M. Kehler[1], M. K. Schluterman[3], R. A. Brekken[3], J. B. Fleming[4], C. C. Silliman[1].

[1]Department of Surgery, University of Colorado at Denver, Denver Colorado. [2] Department of Surgery, University of Florida, Gainesville Florida. [3] Department of Surgery, UT Southwestern Medical Center at Dallas, Dallas Texas, [4] Division of Surgical, UT MD Anderson Cancer Center, Houston Texas.

Background: Perioperative blood transfusion in pancreatic cancer patients is linked to decreased survival; however, a causal mechanism has not been determined. During processing and storage of packed red blood cells (pRBCs) biologically active molecules accumulate in the acellular plasma fraction; therefore, we hypothesize that the plasma fraction of pRBCs promotes tumor progression.

Methods: Proliferation and migration of murine pancreas cancer and control cells were determined *in vitro* in response to the plasma fraction from leukocyte and non-leukocyte reduced, fresh versus stored pRBCs. Lastly, an immunocompetent murine model was used to assess the affect of the plasma fraction of pRBCs on pancreas cancer progression.

Results: Incubation of pancreatic cancer cells with the plasma fraction of pRBCs increased proliferation and migration. Intravenous delivery of the acellular plasma fraction to mice with pancreatic cancer significantly increased the tumor weight in both leukocyte reduced and non-leukocyte reduced pRBCs groups ($p<0.01$) although tumor growth and morbidity was greatest in the non-leukocyte reduced group.

Conclusions: The plasma fraction of stored pRBCs promotes murine pancreatic cancer proliferation and migration *in vitro* and when administered intravenously significantly augments pancreatic cancer growth in immunocompetent mice.

Saturday- Poster #33- Professor Rounds 3:25 – 3:50 pm

HYPERINSULINEMIC HYPOGLYCEMIA AFTER ROUX-EN-Y GASTRIC BYPASS: WHAT DRIVES THE PANCREAS?

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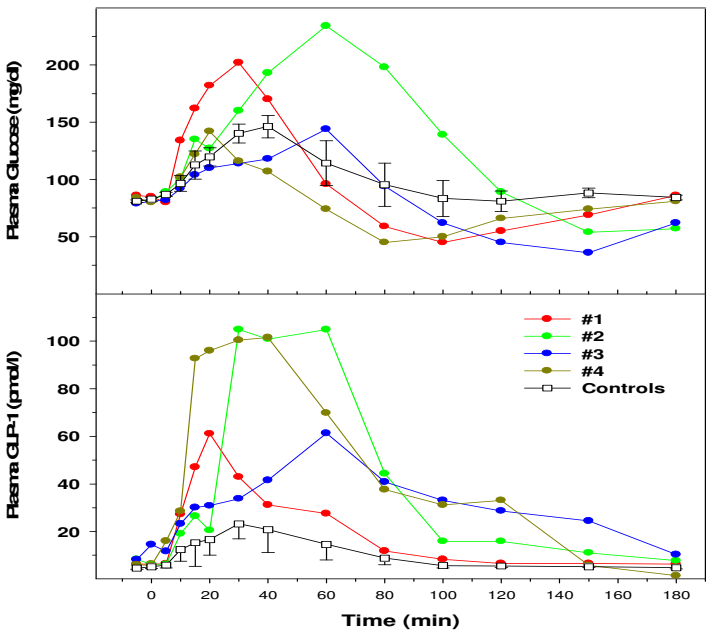
Introduction: Hyperinsulinemic hypoglycemia (hypoG) after Roux-en-Y gastric bypass (RYGB) is a rare condition, the cause of which is unknown. In some cases nesideoblastosis has been described, and several treatment modalities have been used including low carbohydrate diet, acarbose, diazoxide and octreotide, with variable success. When medical treatment fails, the prevailing treatment is partial or total pancreatectomy.

Methods: To determine the cause of post-RYGB hypoG, we examined four patients with this syndrome with a standardized test meal (STM) (475 cc Ensure plus®) and compared the hormonal and glucose profiles to five asymptomatic patients who had undergone RYGB one year before. HypoG patients underwent RYGB 2-3 years prior to symptom development.

Results: After STM ingestion, a robust increase in glucose followed by a sharp decrease to 58 ± 8 mg/dL was uniformly observed in hypoG patients. The time to nadir was not uniform among the patients (Fig 1). This was accompanied by a striking increase in the peak level of GLP-1 (70 ± 18 pmol/L compared to 24 ± 5 pmol/L in controls, $p<0.05$). This resulted in a dramatic increase in insulin in all four subjects. In one hypoG patient, glucose clamp studies revealed normal beta cell sensitivity to glucose and GLP-1; after 85% distal pancreatectomy, hypersecretion of GLP-1 persisted, and hypoG recurred.

Conclusion: The hypoG that occurs following RYGB is due, in part, to the insulinotropic and insulinomimetic actions of elevated levels of GLP-1, and not to primary pancreatic endocrine dysfunction. Whether pancreatectomy should be considered as the only surgical treatment for these patients, whose hyperinsulinemia is driven by gut hormone hypersecretion, requires careful scrutiny.

Graph Listed Below.



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Saturday- Poster #34- Professor Rounds 3:25 – 3:50 pm

PANCREATIC CANCER CELLS STIMULATE STROMAL CELLS TO PRODUCE PINCH

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INTRODUCTION: One of the hallmarks of pancreatic ductal adenocarcinoma (PDA) is a strong desmoplastic response. The local microenvironment within the tumor, including the interactions of the stromal and cancer cells, is thought to be an active participant in the progression of PDA. The stromal cells within more aggressive cancers express higher levels of an integrin-associated protein, PINCH and increased PINCH expression is associated with greater cell motility and survival. There is little data available that explains why PINCH is expressed to a higher degree in the stromal cells of cancers. We hypothesized that pancreatic cancer cells stimulate PINCH expression within stromal cells.

MATERIALS AND METHODS: Conditioned media (CM) was collected from the supernatants of the cultured human PDA cell line MiaPaCa-2 after 48 hrs under serum-free conditions. Human primary fibroblasts or pancreatic stellate cells (PSC) were plated and exposed to DMEM or varying concentrations of CM (0.1mg/kg, 0.2mg/kg, 0.4mg/kg total protein) for 48 hours. PINCH expression in cultured fibroblasts or PSCs was determined by western blot and values normalized to GAPDH protein expression. Cell growth of the stromal cells upon exposure to the varying conditions was also determined using an alamarBlue assay. Groups were compared with an ANOVA.

RESULTS: There was a CM dose-dependent increase in PINCH protein expression in both fibroblasts and PSCs relative to expression under serum-free conditions (Table1). CM did not alter cell growth as all groups had values equivalent to serum-free conditions (data not shown).

DISCUSSION: The data indicate that a soluble factor(s) is produced by PDA tumor cells that stimulates stromal cells to produce PINCH. Since greater PINCH expression within PDA stromal cells is associated with poorer patient outcomes, understanding the mechanisms associated with this tumor/stromal interaction may provide intervention opportunities.

Table 1: Relative PINCH protein expression in fibroblasts and PSCs after exposure to varying protein concentrations of pancreatic cancer cell conditioned media.

Group	Fibroblasts	Pancreatic Stellate Cells
DMEM Serum Free	0.15 ± 0.05	0.25 ± 0.1
0.1mg/kg CM	0.25 ± 0.1	0.29 ± 0.1
0.2mg/kg CM	0.99 ± 0.2+	0.34 ± 0.1+
0.4mg/kg CM	1.32 ± 0.1+	0.41 ± 0.2+

•Data reported as mean ± standard deviation of the PINCH/GAPDH ratio from 3 independent experiments; + statistically different from DMEM serum free group.

Saturday- Poster #35- Professor Rounds 3:25 – 3:50 pm

POSTOPERATIVE ACUTE PANCREATITIS AFTER PANCREATICODUODENRCTOMY IS ASSOCIATED WITH POSATAOPERATIVE PANCREATIC FISTULA

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[Background and aims]
The urine trypsinogen-2 has been used successfully in the diagnosis of pancreatitis of various etiologies, but has been rarely studied in postoperative pancreatitis following pancreatic surgery. The aim of this study was to reveal the risk factors for the postoperative acute pancreatitis after pancreaticoduodenectomy, and also to analyze the possible association of postoperative acute pancreatitis and postoperative pancreatic fistula.
[Methods]
131 patients undergoing pancreaticoduodenectomy who measured postoperative urine trypsinogen-2 were included in this study: 56 females and 75 males (median age 68 years; range 19-88). The pancreatic anastomosis was reconstructed with a two-layered duct-to-mucosa pancreaticogastrostomy with internal stent into the posterior wall of the stomach. The concentration of trypsinogen-2 in the urine samples on the postoperative day 3 was measured by a quantitative immunofluorometric assay. Postoperative acute pancreatitis was defined chemically as the elevation of urine trypsinogen-2 levels more than 50 μg/l. Levels of drain amylase was also measured daily, and postoperative pancreatic fistula was classified into three categories by International Study Group Pancreatic Fistula (ISGPF) criteria. We analyzed the risk factors of postoperative acute pancreatitis by logistic regression analysis, and also analyze the association of postoperative acute pancreatitis and postoperative pancreatic fistula.
[Results]
The incidence of postoperative acute pancreatitis was 35/131 (27%). Univariate analysis revealed that the soft pancreatic parenchyma, non pancreatic adenocarcinoma, main pancreatic duct diameter less than 3mm, and preoperative biliary drainage were significant risk factors for the development of postoperative acute pancreatitis. Multivariate analysis demonstrated that the independent risk factor for postoperative acute pancreatitis is soft pancreatic parenchyma. The rate of ISGPF was 18 of 131 patients (14%). Of these, 14 patients (11%) had grade A, three patients (2%) had grade B, and one patient (1%) had grade C by ISGPF criteria. Postoperative acute pancreatitis was significantly associated with postoperative pancreatic fistula (ISGPF grade A+B+C, $p< 0.01$) and it was also associated with clinically relevant pancreatic fistula (ISGPF grade B+C, $p<0.05$).
[Conclusion]
Soft pancreatic parenchyma is an independent risk factor for postoperative acute pancreatitis defined by urine trypsinogen-2 levels. Postoperative acute pancreatitis might play an important role in the pathogenesis of postoperative pancreatic fistula following pancreaticoduodenectomy.

Saturday Poster 36

REPRESSION OF E-CADHERIN BY THE POLYCOMB GROUP PROTEIN EZH2 IN PANCREATIC CANCER

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Background: Pancreatic ductal adenocarcinoma (PDA) is the fourth leading cause of cancer deaths in the United States. Single-agent gemcitabine remains the standard treatment for advanced PDA, which has shown improvement in disease-related symptoms and a modest benefit in survival. A recently discovered histone methyltransferase termed enhancer of zeste homologue 2 (EZH2) was found to be overexpressed in a variety of carcinomas including PDA. Silencing of E-cadherin was proposed as a mechanism by which EZH2 mediates tumor aggressiveness. Furthermore, in-vitro studies showed EZH2 depletion sensitizes pancreatic cancer cells to gemcitabine. In this study we correlated EZH2 with E-cadherin expression in PDA, and evaluated response to gemcitabine in relation to EZH2 expression.

Methods: 43 PDAs, 14 intraductal papillary mucinous neoplasms (IPMNs), and 5 chronic pancreatitis (CP) cases were stained with EZH2 (BD Bioscience; 1:25) and E-cadherin (Zymed; 1:1,000). Cases with diffuse weak staining, or strong staining in less than 30% of tumor nuclei were considered to have low EZH2 expression. High EZH2 expression was defined as strong nuclear staining in >30% of tumor cells. E-cadherin expression was scored on membrane positivity as follows: 0 (0-10%); 1 (10–25%); 2 (25-75%), and 3 (>75%). E-cadherin scores were considered normal at 3, reduced at 2, and negative at 1 or 0. Statistical analysis was performed using Fisher's exact and Kruskal-Wallis tests, depending on the discrete or continuous nature of the other factors. A Kaplan-Meier curve was stratified by EZH2 expression to assess survival.

Results: High EZH2 expression in PDA was significantly associated with decreased E-cadherin expression (70% vs. 35%), node-positivity (82% vs. 40%), and larger tumor size (4 cm vs. 2.4 cm). There was a trend for longer survival (35 vs. 15 months) in gemcitabine treated patients with low vs. high EZH2 expression. High EZH2 expression was detected in IPMN with moderate-severe dysplasia, however not in CP.

Conclusion: Our study suggests that E-cadherin downregulation may lead to EZH2-mediated invasion and metastasis. While strong diffuse EZH2 expression is seen in PDA, overexpression may be present in IPMN.

Sunday- Poster #1- Professor Rounds 11:00 – 11:25 am

TOTAL PANCREATECTOMY: INDICATIONS, DIFFERENT TIMING, PERIOPERATIVE AND LONG-TERM OUTCOMES

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Background: Total pancreatectomy (TP) was rarely performed in the past because of its high morbidity and mortality. Since outcome of pancreatic surgery as well as management of pancreatic insufficiency have significantly improved, TP has had an increased reappraisal.

Methods: Between 1996 and 2008, 65 patients (33 females, 32 males, median age 63 years) underwent TP at a single, high-volume centre. Indications, timing, perioperative and long-term results were analyzed.

Results: 25 patients (38.5%) underwent a planned elective TP, 25 patients had a single-stage unplanned TP after an initial partial pancreatectomy that turned into TP because of intraoperative haemorrhage (n=1) or positive pancreatic resection margin (n=24). The remaining 15 patients (23%) underwent a two-stage pancreatectomy for tumor recurrence in the remnant. No completion TP for postoperative complications were performed. There was no mortality, overall morbidity was 38.5% and reoperation rate 4.5%. Overall, 48% of patients had intraductal papillary mucinous neoplasms (IPMNs), and 29.5% pancreatic ductal adenocarcinoma (PDA). R1 resections rate was 12%. 4/23 patients (17%) who underwent single-stage unplanned TP for positive resection margin had R1 resection (positive retroperitoneal margin). Median follow-up was 30 months. Five-years overall survival was 71%. No deaths due to hypoglycaemia were observed. Median insulin units/day was 32, median lipase units/day 80000.

Conclusions: TP can be performed safely with no mortality and acceptable morbidity. Postoperative pancreatic insufficiency can be well-managed nowadays. In order to achieve an R0 TP, both the resection and retroperitoneal margin should be evaluated intraoperatively. TP is an effective operation is selected patients.

Sunday- Poster #2- Professor Rounds 11:00 – 11:25 am

THE NOVEL SIGMA-2 RECEPTOR LIGAND SW43 INDUCES APOPTOSIS IN PANCREAS CANCER

John R Hornick[1], Dirk Spitzer[1], Peter S Goedegebuure[1], Robert H Mach[3], William G Hawkins[1,2]

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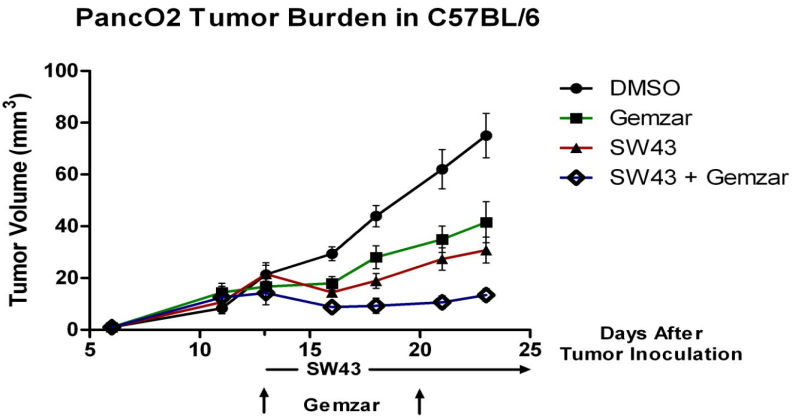
Introduction: Pancreatic cancer has limited treatment options. We have previously shown that sigma-2 selective ligands preferentially bind to pancreatic cancers and induce apoptosis with limited systemic toxicity. In order to indentify the best candidates for a clinical study, we created and systematically tested multiple derivatives of these ligands. We identified a candidate compound (SW43) which has superior apoptotic activity.
Methods: Multiple human pancreas cancer lines were treated with 25uM of the sigma-2 receptor ligands SV119 or SW43, with or without gemcitibine. Following four hours viability was determined with CellTiter-Glo® Luminescent Cell Viability Assay (Promega). Apoptosis in PancO2 was observed by caspase-3 cleavage of Z-DEVD-AMC. SW43 induced oxidation was quantified by flow cytometry for the fluorescence of ethidine. PancO2 cells were inoculated into C57BL/6 mice (n=15/group) and when tumors reached ~5mm in diameter, they were treated daily with sigma ligands (0.05mg/kg) with or without gemcitibine (0.15mg/kg) for ten days.
Results: Compared to control cells the viability of cells in vitro treated SV119 was not significantly decreased, while SW43 decreased to 59-66%. Caspase-3 activity increased after SW43 compared to SV119 treatment. The fluorescence intensity of ethidine was increased from 7.5 to 63% after treatment with SW43, indicating an increase in oxidative stress in the cell. A decrease in viability due to oxidative stress (43%) was partially relieved by treatment of the antioxidant alpha-tocopherol (67%). In vivo, mice treated with SW43 have significantly smaller tumors (31mm^3) than mice treated with vehicle (75mm^3). SW43 combined with gemcitibine enhanced the effect (14mm^3).
Conclusions: SW43 is a more potent inducer of apoptosis in pancreas cancer cell lines when compared with other selective S2-ligands. We are currently testing the in vivo efficacy and toxicity profile of SW43 to see if this new S2-ligand has potential as a novel chemotherapeutic for the treatment of pancreas cancer.



Sunday- Poster #3- Professor Rounds 11:00 – 11:25 am

GENE EXPRESSION PROFILING FOR PREDICTION OF PROGNOSIS IN PATIENTS WITH PANCREATIC DUCTAL ADENOCARCINOMA

Robert Grützmann[1], Marius Distler[1], Moritz Wente[2], Marco Niedergethmann[3], Helmut Friess[4], Marcus Bahra[5], Petra Rümmele[6], Glen Kristiansen[7], Christof Winter[8], Daniela Aust[9], Hans Detlev Saeger[1], Christian Pilarsky[1]

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Background: Prognosis for patients with pancreatic carcinoma (PDAC) remains poor. Despite of increasing knowledge about the molecular basis of PDAC no specific marker for early diagnosis nor a target protein for a new therapeutic approach have been identified so far. Surgery is the only potentially curative option. But even after R0-resection of PDAC, the prognosis is bad and most patients suffer from recurrences and metastases. Moreover, the individual prognosis is not known. This might be of interest for indication of adjuvant therapy. Therefore we were interested in the analysis of differential gene expression between PDAC with relatively good and poor prognosis.

Methods: We used fresh frozen tissue from 30 patients with pancreatic ductal adenocarcinoma. From every single patient, the clinical characteristics, pathological data as well as follow up has been collected prospectively. The tissues were obtained during surgery and freshly frozen. The type of each frozen tissue sample was reevaluated pathologically. The RNA was extracted using the RNeasy Mini Kit. The quality of the RNA was assessed using the Agilent Lab on a Chip System and only samples displaying a RIN > 4 were used. For hybridization we used 100 ng of total RNA, and samples were prepared according the Affymetrix two cycle amplification labelling protocol. Samples were hybridised to Affymetrix U133 2.0 plus GeneChips. The obtained data from the microarray were normalized using RMA. Expression profiles were classified according the survival time of the patients and signature genes were identified by a combination of a transcription network-based ranking algorithm and repeated cross validation. Q-RT-PCR was used to verify the potential of each signature gene. Immunohistochemistry was performed on tissue microarrays comprising more than 400 patient samples using automated staining protocols. Staining intensities were analysed by trained pathologists.

Results: Median survival of the patients was 17.5 (4-53) months. Using this time point we classified the samples into two groups. GeneRank analysis identified seven signature genes resulting prediction accuracy of approx. 70% after cross validation. Quantitative RT-PCR confirmed the accuracy of the signature genes. Immunohistochemistry for selected signature genes resulted in the identification of prognostic marker for the survival of pancreatic cancer patients

Conclusion: In conclusion, gene expression analysis of the tumor tissue of PDAC enables prediction of prognosis with reasonable accuracy. The role of adjuvant treatment has to be elucidated. Moreover, using the set of differentially expressed genes we might identify new markers and therapeutic targets for pancreatic cancer.

Sunday- Poster #4- Professor Rounds 11:00 – 11:25 am

NATURAL HISTORY OF ASYMPTOMATIC PANCREATIC CYSTIC NEOPLASMS

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Objectives: Management of asymptomatic pancreatic cysts is controversial, with need for excision based on pathology and natural history. The aim of this study was to examine the outcome of asymptomatic lesions using our protocol based on size and cyst fluid analysis.
Methods: A prospective database was queried to identify asymptomatic pancreatic cysts. Sequential cross-sectional imaging studies were assessed, and for those ≥1.5cm, results of endoscopic ultrasound-guided aspiration were co-analysed. Definitive histopathology was available for resected specimens.
Results: During the period 1998-2008, 338 patients underwent evaluation (228 females) with a median age of 67 years (IQR [Inter-quartile range]: 57-75). 84 cysts were <1.5cm and 254 were ≥1.5cm in diameter. Median patient follow-up was 5.1 years (IQR: 4.1-6.9). In the <1.5cm group, median cyst size at presentation was 1cm (IQR: 0.6-1.2) increasing to 1.2cm (0.7-1.6) during follow-up. 5 (6.0%) patients underwent resection, all within 2 months of presentation, 4 of these also underwent cyst aspiration which was positive for mucin. In the ≥ 1.5cm group, the median cyst size at presentation was 2.5 cm (IQR: 2.0-3.4) increasing to 2.7cm (IQR: 3.0-4.2). 63 (24.8%) patients underwent resection. 53 (84.1%) cases were performed within 2 months, 4 (6.3%) within 12 months, and 6 (9.6%) >12 months post presentation. Histopathology of resected specimens revealed 71% were malignancies or had malignant potential.
Conclusions: Asymptomatic cysts < 1.5cm can safely be followed only by imaging with little change expected. A quarter of all asymptomatic cysts ≥ 1.5cm are appropriately resected based on imaging and cyst fluid analysis.

Sunday- Poster #5- Professor Rounds 11:00 – 11:25 am

LOWER INCOME IMPACTS CARE FOR PANCREATIC CANCER

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Background: Socioeconomic status (SES) has been associated with survival differences for pancreatic adenocarcinoma (PC). The identification of factors impeding delivery of care and impacting outcomes for PC is needed.

Methods: Linked SEER-Medicare databases 1991-2005 were queried for PC patients (pts) with locoregional, non-metastatic disease and then stratified into SES quintiles based hierarchically upon census tract, zip code median income, and per capita income data. Quintiles were then dichotomized into "low SES" and "high SES," with the lowest quintile (≈2 x U.S. mean poverty threshold or mean income = $23,254) employed as a proxy for low SES. Univariate and Kaplan-Meier (KM) analyses characterized associations between SES, patient characteristics, and therapy. SES was then assessed as a predictor of specialist consultation and receipt of therapy with logistic regression models adjusting for age, gender, race, marital status, and comorbidity.

Results: 7655 pts were identified with locoregional disease; 1597 (21%) of these were low SES. Low SES pts had worse overall survival on KM curves (log-rank, p<0.0001). On univariate analyses, low SES pts tended to be older (p<0.0001), female (p=0.014), black (p<0.0001), unmarried (p<0.0001), and had more comorbidities (p<0.0001). Low SES pts had less frequent specialist consultation, chemotherapy, radiation, and resection than high SES pts (all p<0.0001). Multivariate analyses demonstrated that low SES negatively predicted consultation with a medical oncologist (Odds Ratio (OR) 0.87, 95% Confidence Interval (CI) 0.78-0.98) and surgeon (OR 0.82, CI 0.71-0.94). Overall, low SES pts were less likely to receive chemotherapy (OR 0.81, CI 0.72-0.92) and resection (OR 0.80, CI 0.70 0.91). Furthermore, a subset of low SES pts receiving surgical consultation was less likely to undergo resection (OR 0.84, CI 0.73-0.97).

Conclusions: Socioeconomic disparities exist in specialist consultation and therapy utilization for potentially resectable PC. These barriers may represent discrete points for intervention with specialist referral and therapy. Table Listed Below

Table: cancer specialist consultation and subsequent therapy for local-regional pancreatic adenocarcinoma
(Multivariate analyses controlling for age, sex, marital status, statuses, and comorbidity)

	Univariate Analyses			Multivariate Analyses		
	OR	**95% CI**	**p**	**AOR**	**95% CI**	**p**
Low SES (ref=high)						
Specialist Consultation						
Medical oncologist	**0.79**	**0.71-0.82**	**<0.0001**	**0.87**	**0.78-0.98**	**0.03**
Radiation oncologist	**0.78**	**0.69-0.88**	**<0.0001**	**0.88**	**0.78-1.01**	**0.06**
Surgeon	**0.79**	**0.70-0.89**	**<0.0001**	**0.87**	**0.76-0.98**	**0.03**
Subsequent therapy						
Chemotherapy	**0.70**	**0.61-0.81**	**<0.0001**	**0.88**	**0.75-1.02**	**0.09**
Radiation	**1.17**	**0.90-1.53**	**0.25**	**1.27**	**0.96-1.68**	**0.91**
Resection	**0.71**	**0.61-0.82**	**<0.0001**	**0.84**	**0.72-0.97**	

Sunday Poster 6

BRAIN NATRIURETIC PEPTIDE (BNP) AND POSTOPERATIVE FLUID BALANCE IN THE MANAGEMENT OF PATIENTS UNDERGOING PANCREATECTOMY

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Background: Postoperative fluid management in patients receiving major intra-abdominal operative procedures remains a clinical dilemma. Conventional methods of fluid management are rarely endpoint driven which can lead to errors resulting in preventable morbidity. Recent publications have suggested that the serum levels of Brain Natriuretic Peptide (BNP) reflect the effective blood volume during short-term changes in fluid status. We hypothesized that serial measurements of BNP would accurately reflect daily changes in intravascular fluid status after pancreatectomy.
Methods: We prospectively collected data including, age, gender, serum BNP, and serum creatinine. Using immunoassay methods, serum BNP was measured at baseline in sequential patients receiving pancreatectomy at a high volume center. Serial BNP measurements were then obtained every 24 hours for seven days postoperatively. The total fluid intake and total output (measured in cc) were measured and values obtained from patient care records. Preoperative medical and cardiac evaluations performed at our institution were also recorded. Linear mixed effect statistical models were used to study the change of BNP and fluid balance over time separately and to then take the correlation between these measures at different time points within each individual patient. Bivariate random effect models were used to examine the correlation between the time trend of the BNP and the time trend of fluid balance.
Results: Serial BNP measurements were obtained in 44 patients receiving pancreatectomy. A reproducible absolute and percent decline in serum BNP from the day of operation to postoperative day 7was observed in all patients. Furthermore, a reproducible pattern of BNP decline in postoperative days one to two and three to seven was present. When comparing this decline in BNP to fluid balance specifically, the changes in BNP levels accurately reflected the positive fluid balance that occurs after pancreatectomy and the % change in BNP was highly correlated ($\rho = 0.91$, p-value = 0.039) with fluid balance over the first three postoperative days. A predictable daily % change in BNP was observed during the postoperative period for all patients, but those with evidence of diastolic cardiac dysfunction on preoperative echo were less likely to follow the anticipated pattern of BNP change.
Conclusions: The results demonstrated a predictable pattern of serum BNP in postoperative period. Furthermore, his pattern accurately reflects the fluid balance of the patient. Together, these observations, suggest that daily serum BNP measures could be used to measure the effective intravascular volume after pancreatectomy and cardiac dysfunction may affect the observed patterns of BNP change.

Sunday Poster 7

LOSS OF HETEROZYGOSITY (LOH) STATUS OF D9S105 MARKER IS ASSOCIATED WITH DOWN-REGULATION OF KRUPPEL-LIKE FACTOR 4 (KLF4) EXPRESSION IN PANCREATIC DUCTAL ADENOCARCINOMA AND PANINS

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Introduction/Background. Homozygosis deletion of 9q31-32 has been associated with Kruppel-*Like Factor* 4 (KLF4) suppression placing this gene as putative tumor suppressor gene in gastric, bladder and colorectal cancer. However, Ectopic $Kras^{V12}$ gene mutation can promote KLF4 as oncogene *in vitro*. Furthermore, experimental data suggest that KLF4 may be over-expressed or down-regulated in Pancreatic Ductal Adenocarcinoma (PDAC) and its role in this pathology is unclear. This study was aimed at evaluating the association between loss of 9q31-32 region and gene expression of KLF4 and to evaluate the role of this gene in PDAC.
Methods. We investigated the Loss of Heterozygosity (LOH) in the 9q region and the expression of KFL4 gene in PDAC, PanINs, Normal Ducts and Primary Cell Culture of PDAC (PCC). Epithelial cells from 35 PDAC, 6 Pancreatic Intraepithelial Lesions (PanINs) and 6 normal ducts were isolated by laser microdissection (Leica LMD 7000) for molecular analyses. We used 4 microsatellite markers (D9S127, D9S53, D9S105, D9S106), flanking KLF4 locus to test the LOH, while mRNA gene expression was performed by RT-PCR, both in PDAC and PanINs.
Results. LOH in at least one locus was observed in *25/35* PDAC cases and in *5/6* PanINs respectively. We identified a Small Region of Deletion Overlap (SRDO) in 52.9% of PDAC that presented LOH in both D9S53 and D9S105 markers. In 46.9% of PDAC and 83.3% of PanIN lesions there was a loss of the D9S105 marker, which resulted to be the most deleted marker. Five cases showing homozygosis deletion of D9S105 had not amplified PCR product for KLF4. In contrast, no LOH in D9S105 marker was observed in normal pancreatic duct cells and in PCC. The PCC wild-type for D9S105 marker had mutated in K-ras gene at codon 12 and expressed KLF4 and. Lack of KLF4 expression was found to be significantly associated with: 1) Genomic deletion flanking KLF4 in PDAC ($p=0.018$) and in PanINs ($p<0.01$); 2) LOH of D9S105 marker ($p=0.014$); 3) Presence of low–grade of PDAC-associated PanIN ($p=0.021$).
Conclusion. We identified a relation between D9S105 deletion and loss of KLF4 expression in PDAC. Our results suggested that the KLF4 gene can switch its role between tumor suppressor gene and oncogene depending on the biological context of PDAC.

Sunday Poster 8

DEREGULATION OF THE RB/E2F PATHWAY AND P16 EXPRESSION IN PANCREATIC ADENOCARCINOMA

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BACKGROUND: Deregulation of the Rb/E2F pathway has recently been shown to lead to epigenetic plasticity, particularly in the background of constitutive activation of *ras*, an oncogene mutated in the majority of pancreatic neoplasias. Moreover, cells in the plasticity state are programmable via signals from the microenvironment, resulting in epithelial to mesenchymal transition (EMT). EMT is believed to be responsible for generating metastatic cells as well as drug resistant phenotypes for the majority of epithelial cancers. We therefore sought to determine the relationship between Rb/E2F deregulation and outcomes for patients with pancreatic adenocarcinoma undergoing pancreaticoduodenectomy and adjuvant gemcitabine-based chemotherapy.
METHODS: Cytogenetic analysis was undertaken on ten pancreatic adenocarcinoma cell lines, four of which were established in the laboratory. PCR analysis, single-strand conformation polymorphism (SSCP) analysis, and northern blotting were used to determine the status of CDKN2A (p16), as well as other genes regulating the Rb/E2F pathway. Immunohistochemical staining for p16 was performed on FFPE tissues, and correlated with clinical outcomes. Data is presented as median, mean ± SD where appropriate.
RESULTS: Pancreatic adenocarcinoma cell lines commonly possessed chromosomal abnormalities in the following locations; 3p, 6p, 8p, 9p, 17p, and 18q. We sequenced the 3 exons of CDKN2A (located on chromosome 9p) and found homozygous deletions in 7/10 cell lines, suggesting frequent deregulation of the Rb/E2F pathway via loss of p16 among pancreatic cancers. In order to determine the clinical significance of Rb/E2F pathway deregulation, p16 immunohistochemical staining was performed on 26 patients who underwent resections for pancreatic adenocarcinoma (AJCC Stage IB (3), IIA (8), and IIB (15)) and completed adjuvant gemcitabine-based chemotherapy. Overall survival for 26 patients was 20 months, 27 months ± 17.5. p16 protein expression was absent in 25/26 (96%) patients. Of note, the single patient with positive p16 staining had lymphatic metastasis on final pathology and an overall survival consistent with the mean survival for the 26-patient cohort. Additionally, Pan-INs were identified in association with 12/26 adenocarcinomas; 2/12 Pan-INs stained positive for p16. Finally, we re-examined the 3 pancreatic adenocarcinoma cell lines expressing CDKN2A mRNA by DNA sequence analysis and identified mutations in the coding region of the gene for each of these cell lines.
Conclusions: Deregulation of the Rb/E2f pathway, specifically via deletions or mutations of the tumor suppressor gene CDKN2A (p16), appears to be a ubiquitous finding among pancreatic adenocarcinomas, and an early event in the tumorigenesis of this cancer. Although the Rb/E2F pathway does not appear to directly contribute to EMT and thereby metastasis and drug resistance given its early loss in the oncogenic process, deregulation may contribute to an environment conducive to EMT promoting events. Further work to identify the downstream targets of Rb/E2F signaling is warranted.

Sunday Poster 9

THE BURDEN OF INFECTION FOR ELECTIVE PANCREATIC RESECTIONS

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Background: Because mortality rates for pancreatic resection have fallen, other valid measures of surgical quality are needed. While infection control is a critical surrogate quality indicator, it remains incompletely understood, especially in high-acuity GI surgery. We therefore evaluated the incidence and impact of infections after our elective pancreatic resections.
Methods: All pancreatic resections followed standardized perioperative care, including timely administration of antibiotics. Infections were classified according to NSQIP definitions, while complication severity was based on Clavien criteria. Clinical and economic outcomes were evaluated and predictors of infection were identified by regression analysis.
Results: Of 550 pancreatic resections (356 Whipple, 167 Distal, 11 Total, 16 Other), 288 (53%) had some complication, of which 167 (31%) were infectious. Rates of infection differed by type of resection (p=0.029) but not by the presence of malignancy. There was a trend toward increased infection in patients stented preoperatively (41.4% vs 32.8%, p=0.054). While most infections were minor (n=105, Clavien 1-2), major infections (n=62, Clavien 3-5), occurred in 11%. Patients with infection did significantly worse, with longer LOS and OR time, more transfusions, ICU use, and greater EBL. They were readmitted more often (34% vs 12%). Common organisms were Staphylococcus, Enterococcus, and E. coli. By category, wound infection (14%) was most common, followed by infected fistula (9%), UTI (7%), pneumonia (6%), and sepsis (2%). 48/72 clinically relevant fistulae involved polymicrobial infection and occurred equivalently for Whipple and distal fistulae. TPN use (Odds Ratio 7.3), coronary artery disease (OR 2.1), and perioperative hypotension (OR 1.6) predicted any infection, but specific categories of infection had different predictors. Total costs increased grade-for-grade across the Clavien scale, with infection accounting for 50% of the cost differential.

Table 1: Cost of care by severity of infectious complication.

	Infectious Complications (%)	**Total cost** Non-infected Cases	**Total cost** Infected Cases	**Cost differential**
All resections	58.0	$25,197	$40,533	$15,336
No complication	N/A	$25,082	N/A	
Clavien 1	56.7	$26,831	$27,599	$768
Clavien 2	41.8	$24,406	$26,094	$1,688
Clavien 3	65.2	$27,085	$36,224	$9,139
Clavien 4	80.6	$20,763	$101,253	$80,490
Clavien 5 (Death)	37.5	$24,082	$76,755	$52,673
p-value	0.001		<0.0001	

Conclusion: Nearly one-third of patients undergoing pancreatic resections experienced infections. Depending on severity, clinical outcomes suffered and costs rose significantly. These data are guiding process evaluations and initiatives for infection control in our unit

Sunday Poster 10

A NOVEL MURINE MODEL FOR THE STUDY OF METASTATIC PANCREATIC ADENOCARCINOMA

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Introduction: The overall survival for patients with pancreatic cancer is poor with the liver being the most common site of metastatic disease. Novel, reproducible, translational models utilizing immune-competent mice are needed to test treatments and to study interactions between pancreatic adenocarcinoma and the surrounding metastatic microenvironment.
Methods: We injected tumor cell lines via a hemi-splenectomy model to create hepatic metastases. The chemically carcinogen induced Panc02 murine tumor cell line or pancreatic adenocarcinoma (PDA4969) cells derived from a genetically engineered Kras/p53 mutation knock in mouse were injected into syngeneic mice with or without C3H10T1/2 mesenchymal stem cells, representative of stromal cells. Histopathology and ultrasound images were obtained for metastatic lesions.
Results: Doses of $2x10^6$ Panc02 or PDA4969 cells led to the formation of tumors in the liver with some mice also exhibiting malignant ascites or peritoneal disease within sixty days (Figure 1). Results also indicate co-injection of mouse mesenchymal stem cells facilitate tumor formation in this model.

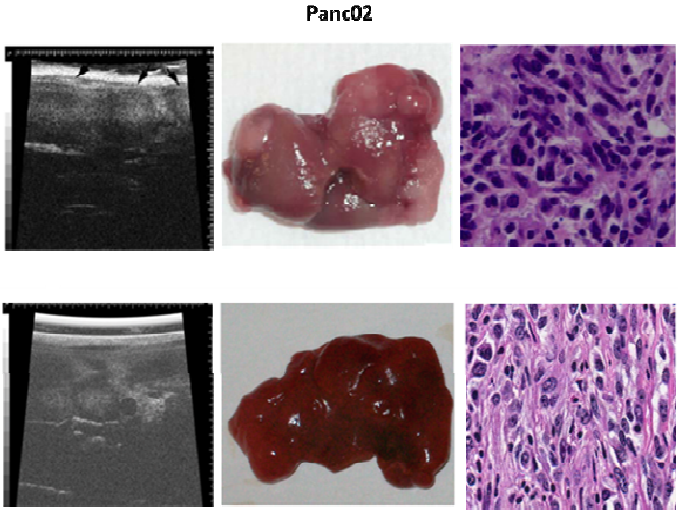

Figure 1. Liver ultrasound, gross necropsy and histopathology for pancreatic liver metastases given through hemi-splenectomy model. Images from representative mice shown.

Conclusion: We successfully developed a pre-clinical model of metastatic pancreatic cancer. To our knowledge, this is the first mouse pancreatic tumor model that can be used to reliably produce liver metastases at rates approaching 100%. This mouse model will facilitate both the development of treatments and the study of interactions between pancreatic cancer and the components of the tumor microenvironment including infiltrating lymphocytes and stromal cells.

Sunday Poster 11

SUPPORT FOR A POSTRESECTION PROGNOSTIC SCORE FOR PANCREATIC ENDOCRINE TUMORS

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Introduction/Background: While the incidence of pancreatic adenocarcinoma has remained stable, the incidence of pancreatic neuroendocrine tumors (PNETs) has increased. The natural history of these tumors is poorly defined, and limited information is available with regards to factors affecting survival after resection. Recently, prognostic scores predicting long term survival of patients with PNETS have been created. The purpose of this study was to validate a currently accepted prognostic scoring scheme at a single institution.

Methods: All patients who underwent resection for pancreatic tumors at a single institution from 1996 – 2004 were reviewed. Tumors in which the final pathological diagnosis was of a PNET were further studied. Clinicopathological and survival data were collected on each patient. A prognostic scores based on patient age, tumor grade, and presences of metastases was calculated for each patient. Survival was compared to an established post-resection prognostic score for PNETs.

Results: A total of 34 PNETs were identified. Since the beginning of 2000, a 50% increase in surgical resections for PNETs was observed. Although, increasing in incidence, PNETs were relatively rare, and accounted for approximately 3% of all pancreatic neoplasms found. Prognostic scores are found in table 1.

Table 1. PNET Postresection Prognostic Score Categories.

Prognostic Score	Raw Score	No. Patients	5-yr Survival actual	5-yr Survival (predicted)
1*	0	13 (38.2‰)	100‰	76.7‰
2*	1-2	18 (52.9‰)	66.7‰	50.9‰
3*	≥3	3 (8.8‰)	66.7‰	35.7‰

*score calculated based on age, grade, and metastases. Scoring for age: <55 years = 0 points, 55-75 years =1 point, >75 years = 2 points. Scoring for grade: well/moderately differentiated = 0 points, poorly differentiated = 1 point. Scoring for distant metastases: none = 0 points, liver = 1 pointt, distant = 3 points.

Discussion/Conclusion: Patients with PNET prognostic scores of 1 had better overall survival when compared to those with prognostic scores of 2 or 3. PNET post- resection prognostic score categories recently established may be a useful tool in prediction of long term survival of patients suffering from PNETs.

Sunday Poster 12

HUMAN PANCREATIC JUICE ENZYME ACTIVITY PREDICTS DISEASE SEVERITY IN CHRONIC PANCREATITIS

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Introduction: Chronic pancreatitis (CP) confers significant morbidity and is responsible for considerable direct and indirect health care costs. However, current modalities for assessing disease severity are inadequate, partly related to our incomplete understanding of the pathophysiology of pancreatitis pain. Serine proteases, including trypsin, are prematurely active in the pancreas and are thought to be critical in the pathogenesis of pancreatitis. Therefore, we set out to determine if levels of pancreatic juice enzymes could serve as potential quantifiable markers for disease severity.

Methods: All patients with CP undergoing endoscopic retrograde cholangiopancreatography (ERCP) with pancreatic examination were included. Demographics, information on pain, disease history and risk factors were obtained via a standardized questionnaire, based on the validated EUROPAC survey, before the procedure. During ERCP, the pancreatic duct was cannulated and 1-3mL of PJ was removed and immediately frozen and stored at -80^{o}C. Trypsin activity was measured using a fluorogenic substrate assay (BOC-QAR-MCA) while myeloperoxidase (MPO) and amylase levels were measured using standard colorimetric assays. All samples were run in batch. MPO and trypsin activity were normalized to protein content (mU/ug protein and ug trypsin/ug protein, respectively) and amylase was calculated as U/L. Univariate logistic regression was used to determine the association between clinical factors and enzyme activity levels.

Results: 44 patients with CP were enrolled and 34 patients had all endpoints collected. Patients with more than 10 prior ERCPs had mean trypsin and amylase levels of 1.98±2.56 and 4975±3608, respectively compared to 0.33±0.43 and 3254±3234, respectively for those with 10 or fewer ERCPs. Patients diagnosed with CP for at least 10 years had a mean trypsin of 1.20±1.38 compared to 0.75±2.09 in those with shorter disease duration, but similar amylase levels were observed. The number of previous ERCPs was predictive of both high trypsin [OR 1.06 (95% CI 1.0-1.11)] as well as high amylase [OR 1.06 (95% CI 1.01-1.13)] activity. In addition, the duration of disease also demonstrated a trend towards predicting high trypsin activity [OR 1.08 (95% CI 0.99-1.17, p=0.06]. Multivariate analysis with larger sample sizes is needed to determine if these are inter-dependent factors. Finally, higher trypsin levels were also independently associated with a history of 10 or more ERCPs [OR 3.86 (95% CI 1.08-13.8)]. Patients with a history of alcohol abuse consistently had low levels of trypsin activation with a mean of 0.16±0.06ug/ug compared to 1.23±2.05ug/ug in those with minimal alcohol use. Amylase levels were similar between drinkers and non-drinkers. MPO levels were not significantly associated with clinical markers.

Conclusions: Pancreatic juice trypsin and amylase activity were associated with the number of previous ERCPs as well as duration of disease burden, and therefore may serve as quantifiable markers of disease severity. The range of enzyme activity seen among patients may be a reflection of the considerable diversity within this cohort of patients with chronic pancreatitis. The lower levels of PJ trypsin activity in patients with significant alcohol abuse may be due to pancreatic “burnout”. Further studies are needed to clarify the mechanisms of trypsin activation among patients with chronic pancreatitis.

Sunday Poster 13

GIANT SPLENIC ARTERY PSEUDOANEURYSM: CASE REPORT AND REVIEW OF THE LITERATURE

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Introduction: Giant splenic artery pseudoaneurysms (≥5 cm in size) are rare entities. We document the successful operative management of one of the largest splenic artery pseudoaneurysms (18 cm) ever reported, as well as review the world literature on the subject.

Methods: The available PubMed literature was searched from 1966-2009 for reports of splenic artery pseudoaneurysms, focusing our review on giant pseudoaneurysms ≥5 cm in size.

Results: In our case, a 68 year old male patient developed an 18 cm splenic artery pseudoaneurysm 17 years after celiac artery reconstruction, and presented with acute abdominal pain from aneurismal leak. Definitive surgical treatment was via splenic artery ligation and debridement of the aneurysm cavity. Blood loss was 250 mL and the patient was discharged on POD #5. Our literature search identified 157 cases of splenic artery pseudoaneurysm in the last 43 years. These ranged in size from 0.3 to 17 cm, and of these, 15 (10%) were ≥5 cm. The majority of patients underwent treatment, either endovascularly or with open surgery, and their outcomes were independent of presenting symptoms or size.

Discussion: Giant splenic artery pseudoaneurysms are uncommon and typically are caused by pancreatitis, trauma, or iatrogenic etiologies. Many can be treated via an endovascular approach, though in our case, the presence of celiac artery occlusion with retrograde pseudoaneurysm filling via superior mesenteric artery collaterals precluded this. Therapeutic alternatives considered included: endovascular balloon catheter placement near the collateral inflow site for temporary control of hemorrhage during dissection, open distal splenic artery control with the placement of a sheath for retrograde endovascular repair, and pseudoaneurysm thrombin-collagen injection. Ultimately, we opted for an open technique, with supra-celiac aortic control prior to manipulation and resection of the pseudoaneurysm. Our recommendation is that splenic artery pseudoaneurysms should be repaired when encountered, regardless of aneurysm size at presentation.

Table 1: Giant Splenic Artery Pseudoaneurysms Reported in the Literature

Age (range)	Gender	Size (range)	Cause	Treatment
Mean 53 years (41-73)	11 M, 4 F	Mean 7.5 cm (5-17 cm)	14 Pancreatitis 1 Iatrogenic	6 Embolization 2 Thrombin injection 7 Distal pancreatectomy/splenectomy

Sunday Poster 14

CENTRAL PANCREATECTOMY WITH INFRAMESOCOLIC INTRAPERITONEAL PANCREATOJEJUNOSTOMY

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Background: The high rate of pancreatic fistula (PF) after central pancreatectomy (CP) is believed to be the compounded effect of handling two divided edges of the pancreas. Since CP is indicated for benign or low grade tumors, the risk of PF is further increased by the soft consistency of pancreatic parenchyma and the small caliber of the main pancreatic duct. Although in the setting of CP PF usually pursues a benign course, severe local complications can occur. The aim of this study is to analyze the short term result of five CP with inframesocolic intraperitoneal pancreatojejunostomy performed at our Institution.
Methods: From October 2008 to October 2009, 112 patients underwent pancreatectomy at our Institution, including 5 (4.5%) CP. One patient was male and 4 females. The mean age was 42.2 years (range: 27-59 years). In three of them the entire operation was carried out laparoscopically, with the assistance of the da VinciS surgical system®. In every patient the margin of the distal pancreatic stump was brought down in the inframesocolic intraperitoneal space through the defect in the transverse mesocolon that is usually employed for the upward passage of the Roux-en-Y jejunal loop. Pancreatojejunostomy was hence constructed using standard techniques.
Results: The mean operative time was 315.0 minutes (range 275-355 minutes) for open procedures, and 426.7 minutes (range 390-450 minutes) for robot-assisted laparoscopic operations. There was no post-operative mortality, but four patients developed post-operative complications (4/5; 80%). Each of these patients developed a PF. According to the recommendations of the international study group on pancreatic fistula (10), there were 2 grade A and 2 grade B PF. No patient required reoperation and/or interventional radiology procedures. The mean post-operative stay was 21.4 days (range 11-40 days).
Conclusions: Construction of pancreatojejunostomy in the inframescolic intraperitoneal space does not increase the complexity of CP and segregates the two pancreatic stumps into different body districts. Leaving in the retroperitoneum only the right-sided pancreatic stump, a possible source of pure PF with limited digestive power, might reduce the severity of local complications after CP.

Sunday Poster 15

BLOOD PRESSURE LOWERING MEDICATIONS DISRUPT FATTY ACID METABOLISM IN PANCREATIC CANCER

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Introduction/Background: Despite the routine use of chemotherapy and radiotherapy, survival has not significantly improved in patients with pancreatic ductal adenocarcinoma (PDA), a situation that signifies the urgent need for novel therapeutic approaches. Angiotensin II (AngII), the principal hormone of the renin angiotensin system, is actively generated in the pancreas and has been suggested as a key mediator of PDA cell survival. Fatty acid synthase (FAS) is a key enzyme involved in synthesis of fatty acids and its high levels have been correlated with poor prognosis in many cancers. In this study, we investigated the molecular basis for the role of AngII in pancreatic carcinogenesis through studying its effect on FAS.

Methods: FAS mRNA and protein in Panc-1 and PK-9 PDA cells, treated with or without AngII (10^{8}-10^{6} mol/L) in presence or absence of AngII receptor type 1 (ATR1) or type 2 (ATR2) blockers, were analyzed by real time PCR and ELISA. Luciferase-labeled promoter studies evaluated the effects of AngII on FAS transcription. MAPkinase and AKT phosphorylation was analyzed by Western immunoblotting. In human premalignant (n=6) and invasive PDA lesions (n=25), mRNA levels and localization of ACE (AngII converting enzyme), the enzyme responsible for AngII generation, and FAS were examined by real time PCR and immunohistochemistry. Mice with established PANC-1 or PK-9 cells tumor xenografts were treated orally with the AT1R blocker, losartan of or vehicle control (n = 5-10 mice per group) and were used to evaluate effects on tumor growth and FAS pathway activity.

Results: AngII significantly increased the expression of FAS mRNA and protein in PDA cells lines and induced FAS promoter activity. AngII-FAS mRNA induction was inhibited by an AngII type 1 receptor (AT1R) antagonist, losartan, and to a lesser extent by an AT2R antagonist. AngII activated the phosphorylation of ERK1/2, but not p38 or c-Jun NH2-terminal MAP kinases. Inhibition of ERK1/2 activation reduced the AngII-induced FAS synthesis. AngII activated the phosphorylation of AKT. Inhibition of AKT activation prevented the AngII-mediated increase of FAS. In human tissue, high mRNA levels of FAS correlated well with tumor stage and invasion status, and with high expression levels of ACE. Immunohistochemical staining of PDA serial sections showed co localization of ACE with FAS in the malignant ducts and stromal cells. Oral administration of losartan significantly ($p< 0.05$) decreased the growth of PANC-1 and PK-9 cells xenografted on the flank of nude mice and significantly ($p<0.05$) reduced the expression of FAS mRNA and protein in the xenografts.

Discussion/conclusions: Our data suggest a positive autocrine/paracrine action for the local pancreatic AngII generating system during pancreatic carcinogenesis, and provide the first insight into an AngII-initiated signal transduction pathway that involves AT1R/ERK1/2/AKT to regulate fatty acid metabolism and modulate lipogenesis in PDA. These results raise the possibility that AngII blockade therapies could be potential candidates in novel treatment strategies of PDA.

Sunday Poster 16

PRIMARY PREVENTION OF ACUTE PANCREATITIS DURING EXTENDED DURATION SPACEFLIGHT: SHOULD THE GALLBLADDER BE REMOVED IN ALL ASTRONAUTS?

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Introduction: Extending human spaceflight beyond Earth's orbit is a current NASA goal. A return to the Moon with anticipation of lunar inhabitance, and the human exploration of Mars, is in the planning stages. Other nations, as well as private industry, are also rapidly developing space-faring technologies. Space exploration has always been associated with a significant human cost, as illness and injury have been responsible for more expedition-related delays/failures than either engineering or weather issues. In addition to injury, nephrolithiasis and appendicitis, the potential occurrence of acute pancreatitis in healthy crewmembers remains a significant concern for long duration space travel.
Although the treatment of pancreatitis is well defined, the inherent hostility of the spaceflight environment offers significant challenges to providing medical care. These include: (1) restricted on-board imaging and laboratory capabilities, (2) limited medical/surgical equipment due to payload weight constraints, (3) non-surgeon crew medical officers, and (4) the potential inability to offer basic critical care or perioperative anesthesia given equipment/manpower constraints. Mission objectives must also be considered when assessing the response to an ill crewmember. The primary goals of this study were to: (1) identify the probable incidence of acute pancreatitis and its associated impact on mission and crewmember health, and (2) develop a primary prevention consensus statement for astronauts tasked to extended duration spaceflight.

Methods: A systematic PRISMA review of all literature outlining the risk of acute pancreatitis, physiologic impact of spaceflight, and cholecystectomy was completed.

Results: Of the numerous etiologies that cause acute pancreatitis, most can be identified through the health screening process for astronaut selection and flight-ready maintenance. While current selection protocols do not mandate cholecystectomy in astronauts without symptomatic cholelithiasis, the effect of spaceflight-related alterations in physiology (relative hypovolemia, immunosuppression) on gallstone behavior (cholesterol/lipid biochemistry, gallbladder contractility/absorption) is unknown. With missions of increasing distance, in-flight diagnosis/treatment becomes more problematic. Time to definitive medical care will also become much longer (Mars = 2-4 year voyage; evacuation time = 9-12 months; delayed communication transmissions of up to 50 minutes). Given the poor sensitivity of screening ultrasonography for detecting biliary sludge (<55%) coupled with both the potential for microlithiasis to cause "iatrogenic" acute pancreatitis (60% of 4.8-24.2 cases per 100,000 people per year) and spaceflight-associated gastrointestinal dysmotility, primary prevention via cholecystectomy should be contemplated prior to extended duration missions. Considering the relatively low risk of bile duct injury (0.4%) and significant hemorrhage (0.1%) during laparoscopic cholecystectomy, contrasted with the higher risks (1-4% bleeding/pancreatitis) associated with an ERCP (bile aspiration to detect microlithiasis), prophylactic removal of astronaut gallbladders is recommended. This strategy would also eliminate the risk of acute cholecystitis (progression to symptomatic disease approximates 1-4% per year).

Conclusion: The theoretic incidence of acute pancreatitis in a healthy astronaut during extended duration spaceflight is unclear. The impact of altered human physiology, anatomy and immunology during spaceflight on the natural history of this disease is also unknown. As a consequence of the immense potential risk for loss of mission and human life, prophylactic laparoscopic cholecystectomy should be considered to prevent microlithiasis-induced pancreatitis.

Sunday Poster 17

LIGATION VS RECONSTRUCTION OF THE SOFT PANCREATIC REMNANT FOLLOWING PANCREATICODUODENECTOMY – REVISITING AN OLD FRIEND

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INTRODUCTION: Leakage from a pancreaticoenteric anastomosis following pancreaticoduodenectomy most commonly occurs following reconstruction to a soft gland. Frank disruption may lead to sepsis and death. Ligation of the remnant pancreas has been previously described in unselected series as a means to mitigate the morbidity and mortality of anastomotic failure with equivocal results. Prior studies have failed to compare results of reconstruction versus ligation specific to those with a soft pancreatic remnant.
METHODS: We retrospectively reviewed patients having Whipple procedures and management of both a hard and soft pancreatic remnant at the University of Tennessee, Memphis from 1997-2009. Factors examined included the presence or absence of peritoneal drainage at the index procedure, the development of a pancreatic fistula (PF) or an intra-abdominal abscess (IAA) and procedure related mortality. The magnitude of exocrine insufficiency following ligation was noted. Pancreatic fistulas were graded according to the ISGPF classification. Drains were used selectively in those with reconstruction whereas all patients with ligation had drainage of the operative bed.
RESULTS: During the study period 179 patients had Whipple procedures 79% of who had carcinoma. Sixty-four of 76 patients with a soft pancreatic remnant following resection had reconstruction via pancreaticojejunostomy and 12 had ligation. Of 103 patients with reconstruction to a hard gland 12 (11.6%) developed either a PF (5 (Class A-3, B-2)) or an IAA (7). Four of 5 developing a PF and 2 of 7 with an IAA had peritoneal drainage. Three (2.9%) patients died secondary to jejunostomy tube complications (2) and pulmonary embolism.
Twenty-six (40.6%) of 64 patients having reconstruction to a soft gland developed either a PF (9 (Class A-3, B-2, C-4)) or an IAA (17). Five of 9 developing a PF and 8 of 17 with an IAA had peritoneal drainage. Three (4.6%) of these 64 patients died. In contradistinction to those with reconstruction to a hard gland, all deaths were a result of Class C PF.
Three patients developed a Class A PF following ligation. Two closed spontaneously on post-operative days 22 and 47. One low output PF continues to drain 5 months after resection. One patient developed an IAA for an overall major complication rate of 33% in those having ligation. Eleven of these twelve patients required only 1 enzyme tablet with each meal with no clinical signs of steatorrhea and negative fecal fat studies. There was no procedure related mortality in this group.
CONCLUSIONS: We conclude the following with respect to pancreaticoduodenectomy: 1) The vast majority of major morbidity is related to management of a soft remnant. 2) The role of peritoneal drainage in this cohort requires further study. 3) While numbers are small and acknowledging a potentially higher rate of Class A PF, our data suggests that ligation rather than reconstruction of a soft pancreatic remnant may reduce the incidence of septic morbidity and procedure related mortality. 4) Exocrine insufficiency following ligation is minimal and easily managed.

Sunday Poster 18

DUPLICATE PANCREAS MEETS GASTRIC DUPLICATION CYST: A TALE OF TWO ANOMALIES

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Introduction/Background: To report a rare cause of pancreatitis due to a duplicate pancreas and gastric duplication cyst that communicated with the pancreatic duct.

Case Report: A 43 year-old male presented with 29 years of progressive epigastric pain that ended in a hospitalization for pancreatitis. He is the product of a premature twin birth weighing 21/2lbs. All usual causes of pancreatitis were ruled out. Cholecystectomy and proton pump inhibitors failed to relieve symptoms. CT revealed a heterotopic pancreas with two pseudocysts and extrinsic mass effect on the antrum. EGD showed extrinsic antral compression. ERCP revealed a pancreatic duct bifurcation within the body of the pancreas. The duct looped back across midline and filled a pseudocyst. Exploratory laparotomy revealed a duplicate pancreas emanating from the body of the pancreas and ending in a pseudocyst that communicated with a gastric duplication cyst. The gastric duplication was contained within the antrum of the stomach, but did not communicate with it. The aberrant pancreas, pseudocyst, and gastric duplication cyst were resected with complete symptom resolution.

Literature Review: Gastric duplication cysts are the rarest of enteric duplications and even rarer in the setting of a duplicate pancreas. These anomalies are believed to occur as a result of a neuroenteric band causing traction diverticula of the foregut. The majority of reported cases occur in females (65%) aged 9 days to 46 years (median 9 years) and all present with recurrent abdominal pain. Patients whose gastric duplication communicated with the pancreatic duct had pain caused by pancreatitis from peptic bleeds or mucoid secretions blocking the duct. Thirteen (57%) of the gastric duplications were contiguous with the stomach and at least 14 (61%) communicated with the main pancreatic duct. ERCP and CT /MRI were keys to preoperative recognition and appropriate therapy. Ten patients underwent >1 operation before correcting the problem (ave. 1.61, range 1-4) and one even underwent a Whipple. The majority can be surgically corrected with excision of the gastric duplication and aberrant pancreas with associated duplicate duct.

Conclusion: Recurrent abdominal pain and pancreatitis in young adults devoid of risk factors should lead to consideration of congenital anomalies as a cause. ERCP and CT/MRI provide critical information for diagnosis and therapy. Not all cysts near the pancreas and stomach are pseudocysts. This dual anomaly is successfully treated by simple excision of the gastric duplication and aberrant pancreas.

A SAFE RECONSTRUCTION TECHNIQUE AFTER PANCREATICODUODENECTOMY

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Background: High volume centers report low mortality and morbidity rates after pancreaticoduodenectomy (PD). However, low volumes centers perioperative complications and mortality rates are still considerable. Despite the various approaches devised to reduce the rate of postoperative pancreatic fistulas (POPF) rate after PD, none of them were able to accomplish this intent. In fact, in some series, the occurrence of pancreatic fistula is greater than 30%. It is therefore important to develop a technique that could be useful in dimishing the morbidity and mortality of patients of patients with PF following PD.
Methods: One-hundred and twenty nine patients with an average age of 58.7 years (12 – 83 years) were submitted to pylorus-preserving PD (PPPD). Indications for resection were as follows: 84 pancreatic head tumors, 33 ampullary tumors, 5 chronic pancreatitis, 3 distal bile duct tumors and 4 duodenal tumors. Duct-to-mucosa pancreato-jejunostomy and invagination of the pancreatic stump were used in all cases. Reconstruction of the alimentary tract was performed using the double intestinal loop technique. **Results:** There was no perioperative mortality in the present series. Immediate postoperative complications are shown in Table 1. Twenty seven patients (20.9%) presented with PF. Fistulas grade A were present in 26 patients. One patient developing a combined pancreatic and biliary fistula was reoperated for pancreatic abscess drainage. Two other patients were reoperated for bowel ischemia. The three patients had uneventful recoveries.
Discussion: Biliary pancreatic secretion activation that occurs in POPF in after PD using a single intestinal loop for reconstruction is a major factor responsible for the severity of POFP. Diverting the pancreatic secretion from bile diminishes considerably the harsh effect of this complication.
Conclusion: The technique for reconstruction of the alimentary tract after PD described here in represents a safe method that could used at surgical centers with lower pancreatic surgery volume.

Table 1.

Complications	n (%)
Pancreatic fistula	**27 (20.9)**
Biliary fistula	**2**
Acute pancreatitis	**3**
Reoperation	**3**
Other*	**3**

*** cardiac failure and bowel ischemia**

Sunday Poster 20

EFFECT OF ABERRANT HEPATIC ARTERIAL ANATOMY ON OVERALL SURVIVAL IN PATIENTS WITH ADENOCARCINOMA OF THE PANCREAS WHO UNDERGO PANCREATICODUODENECTOMY

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BACKGROUND
Complete resection (R0) is crucial to achieving long-term survival in patients with resectable adenocarcinoma of the pancreatic head. Aberrant anatomy is common among the vasculature of the hepatoduodenal ligament, imparting a potential technical challenge in achieving R0 resection. The objective of this study was to determine whether presence of aberrant hepatic arterial anatomy affects overall survival in patients with resectable pancreatic adenocarcinoma in patients undergoing pancreaticoduodenectomy.

METHODS
A retrospective chart review was conducted using patients with pancreatic adenocarcinoma who underwent pancreaticoduodenectomy at our institution from 2000-2008. One hundred forty-eight patients had complete medical records and were included. Hepatic arterial anatomy was determined through angiography (47%) or operative note dictations (53%). Data were analyzed using Wilcoxon rank-sum testing, equality of survivor functions, and Cox proportional hazards models. The Kaplan-Meier method was used to graphically examine data.

RESULTS
Median survival was 19 months (range 2-101). 81.8% of patients had conventional anatomy, 12.8% had a replaced right hepatic artery, 2.7% had a replaced common hepatic artery, and 2.7% had other variants of aberrant hepatic arterial anatomy. On univariate analysis, a statistically significant increase in survival was seen in patients with negative margins ($p = .004$), absence of nodal metastases ($p = .009$), and those who had undergone neoadjuvant therapy ($p = .037$). No difference in survival was seen in patients with a replaced right hepatic artery compared to conventional anatomy ($p = .6$) or when any aberrant anatomy was identified ($p = .9$). Presence of a replaced right hepatic artery did not alter ability to achieve a negative margin ($p = .57$). On multivariate analysis, only margin status remained statistically significant. Overall survival did not vary by other factors including age, sex, race, smoking history, tumor size, nor year treatment began.

CONCLUSION
Patients with a replaced right hepatic artery have survival that is comparable to patients with conventional anatomy. R0 resection remains a crucial factor to a favorable prognosis in patients with resectable pancreatic cancer.

Sunday Poster 21

NORMAL LIVER ENZYMES ON DAY #1 OF ACUTE PANCREATITIS (AP) PREDICT HIGH RECURRENCE RATES OF PANCREASTITIS AFTER CHOLECYSTECTOMY : A Population-based Study

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Background/Aims: To prevent recurrence of acute biliary pancreatitis, current recommendations are to perform cholecystectomy (CCX) after the first episode of gallstone pancreatitis and 2 or more attacks of idiopathic AP. In a population-based study we examined recurrence rates after CCX for AP.

Methods: We retrospectively abstracted data of all Olmsted county residents diagnosed with AP at Mayo Clinic between 1990 and 2005 (n=1049); 239 patients (22.8%) underwent CCX after AP to prevent recurrence. We examined recurrence of AP in 4 groups classified based on the presence or absence of criteria A (elevated liver tests on day 1 of AP) and B (presence of gallstones/sludge on US): Group I: A + B, Group II: only A, Group III: only B and Group IV: neither A nor B.

Results: After a median follow-up of 99 months, AP recurred in 41/239 (17%) patients who underwent CCX. Recurrences of AP in groups I through IV were 13/142 (9.2%), 1/17(5.9%), 13/57(22.8%) and 15/23 (60.9%), respectively. The recurrence rates were higher in IV than all the other groups and higher in III than I. Recurrences after CCX done for $\geq$2 AP attacks were higher in IV than II (87.5%vs20%).
Conclusions: While recurrence of AP after CCX was low in AP associated with elevated liver enzymes on day 1, it was common in those with normal liver enzymes on day 1 of AP, especially in those without gallbladder stones/sludge. Recurrences were high in patients with $\geq$2 attacks of idiopathic attacks of AP undergoing CCX.

Sunday Poster 22

PANCREATIC HETEROTOPIA OF THE DUODENUM: ANATOMIC ANOMALY OR CLINICAL CHALLENGE?

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Background
Pancreatic heterotopia is a relatively common congenital anomaly and can occur anywhere in the gastrointestinal tract (GIT). Localizations in the upper GIT are dominating. In most cases these heterotopia stay asymtomatic and are only detected at pathohistological examination or autopsy. Against this background we analyzed our case loads concerning clinical relevance and impact.

Methods
On the basis of our prospective pancreatic database we retrospectively analyzed the period from 01/2000 to 06/2009, cooperating with the institute of pathology. All specimens with duodenal pancreatic heterotopia collected during pancreatic- or duodenal resections were reviewed. Classification was performed according to Heinrich (***Type I***: both pancreatic components: acini, ducts and islet cells, ***Type II:*** pancreatic tissue without islet cells and ***Type III:*** mainly pancreatic ducts, poor acini, no islet cells).

Results
In the above mentioned period a total of 657 pancreatic resections were performed in our department. A number of 33 patients (5.0%) showed pancreatic heterotopia of the duodenum. In this panel of patients pancreatic resections (*PPPD n=26, Whipple n=6, left resection with partial duodenal resection n=1*) were performed due to various indications (*chronic pancreatitis n=16, malignancies n=9, cystic neoplasms n=5, neuroendocrine tumors n= 2, pancreatic heterotopy n=1*). The patient with heterotopic pancreatic tissue was treated (*Whipple*) due to a suspicious duodenal tumor; pathohistological examination showed a pancreatic heterotopy. Furthermore indication for a *partial duodenal resection* (*n=1*) was an unclear symptomatic stenosis of the duodenum. Specimen of that case also showed pancreatic heterotopy. Overall according to Heinrich`s classification the following types of heterotopia were found: ***Type I*** n=12, ***Type II*** n=17 and ***Type III*** n=5 (Total n=34).

Conclusion
Pancreatic heterotopia still is a rare clinical diagnosis. In most of the cases heterotopic pancreatic tissue is detected accidentally during pathohistological examination of the specimen. However, own data shows that in two of our patients surgery was performed immediate to symptomatic pancreatic heterotopia of the duodenum. Types I and II according to Heinrich`s classification seem to be most frequently. Therefore in cases with unclear pancreatoduodenal lesions heteropic pancreatic tissue should be considered. If there is any impact of symptoms surgery is indicated.

Sunday Poster 23

OUTCOMES OF DISTAL PANCREATECTOMY AS PART OF MULTIORGAN TUMOR RESECTION

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Introduction/Background: Distal pancreatectomy with splenectomy for a pancreatic lesion is associated with a mortality rate of <1% in high volume institutions. Its utility as part of a multiorgan resection for pancreatic and non-pancreatic malignancies is not well-established.

Methods: Patients who underwent distal pancreatectomy as part of a multiorgan oncologic resection between 11/1992-3/2009. Multiorgan resection is defined as at least one additional organ resected other than the spleen. Patients who underwent only distal pancreatectomy with or without splenectomy during the same period served as a control group.

Results: We identified 623 patients who underwent a distal pancreatectomy of whom 109 patients underwent multiorgan tumor resection. The latter group consisted of 52% female with a median age of 63 years (range 24-92). Splenectomy was performed in 91%, partial gastrectomy in 51%, partial colectomy in 34%, adrenalectomy in 24%, nephrectomy in 18%, and/or hepatectomy in 7% of patients. The pathologies for which the resections were performed included pancreatic adenocarcinoma (25%), gastric adenocarcinoma (18%), sarcoma (14%), colorectal cancer (9%), and other tumors (34%). The median survival for patients with pancreatic adenocarcinoma and gastric adenocarcinoma was 21 and 15 months respectively. The overall mortality rate was 5.5% whereas the mortality rate in the control group was 0.8%. The most common complications were pancreatic fistula (23%), abdominal abscess (19%), and pneumonia (11%). Readmission and reoperation rates were 14.7% and 2.8%, respectively. Median in-hospital stay was 9 days (range 3-82).

Discussion/Conclusion: Distal pancreatectomy can be performed as part of a multiorgan resection for oncologic purposes with acceptable mortality and morbidity irrespective of the organ of origin of the tumor.

Sunday Poster 24

CYST SIZE AND MINOR SYMPTOMS ALONE ARE NOT ASSOCIATED WITH MALIGNANCY IN BRANCH-DUCT IPMNS

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Background: The 2006 Sendai Consensus Guidelines recommended surgical resection for all bench-duct intraductal papillary mucinous neoplasms (BD-IPMNs) of the pancreas with symptoms, size>3cm, nodules. However size>3cm alone and the presence of minor symptoms (i.e. vague abdominal pain) are debated. Aim of the preset study was to retrospectively evaluate these guidelines in a series of resected, pathologically confirmed BD-IPMNs.

Methods: All patients who underwent a pancreatic resection at our Institution between 1990 and 2007 with a final histological diagnosis of BD-IPMNs were identified. The Sendai criteria for surgical resection (presence and type of symptoms, cyst size, presence of nodules) were retrospectively applied and correlated with pathology.

Results: 58 patients (25 males, 33 females, median age 64 years, range: 35-78) were identified. Overall, 26 patients (45%) had BD-IPMNs with adenoma, 18 with borderline neoplasms (31%), 5 with carcinoma-in-situ (8.5%) and 9 with invasive carcinoma (15.5%). Factors associated with the presence of malignancy were nodules and “major” symptoms such as jaundice. Cyst size > 30 mm and the presence of “minor symptoms” such as vague abdominal pain were not associated with malignancy unless other worrisome features were present. There were no cases of malignant BD-IPMNs in patients without symptoms who had a BD-IPMNs < 30 mm without nodules. In the present series only one patient had synchronous ductal adenocarcinoma distinct from IPMN (1.5%) and no patient developed metachronous pancreatic cancer during follow-up.

Conclusions: Cyst size and minor symptoms in the absence of other worrisome features should not be considered as absolute criteria for surgical resection.

Sunday Poster 25

ANTI-INFLAMMATORY EFFECTS OF PERITONEAL LAVAGE IN ACUTE PANCREATITIS

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Introduction/Background: Previous studies have demonstrated that intraperitoneal administration of trypsin stimulates the production of cytokines from peritoneal macrophages. Therefore, removing the pancreatitis-associated ascitic fluid from the peritoneal cavity may decrease the systemic inflammatory response in acute pancreatitis (AP). The present study was designed to investigate the effect of peritoneal lavage on the systemic inflammatory response in a rat model of severe AP.
Methods: AP was induced in Wistar rats by intraductal injection of 5% sodium taurocholate. Peritoneal lavage (PL) was performed by infusion of 200 ml of warm peritoneal dialysis fluid at constant flow (50 ml/h) for 4h after onset of AP. Animals were divided in: Group I: Sham-operated rats, with PL; Group II: No treatment after the induction of AP; and Group III: PL beginning immediately after induction of AP. At 4 hours after induction of AP, serum samples were assayed for amylase and inflammatory cytokines (TNF-α, IL-6, and IL-10). Expression of pancreatic cyclooxygenase 2 (COX-2) and inducible nitric oxide synthase (iNOS), liver mitochondrial function and pulmonary myeloperoxidase (MPO) activities were determined.
Results: When compared to Group II (no PL), use of PL after experimental AP (Group III) led to a decrease in serum levels of TNF- α and IL-6, an increase in IL-10, and a decrease in liver mitochondrial dysfunction and pancreatic COX-2 and iNOS expression. There were no differences on serum amylase levels and pulmonary MPO between groups with AP.
Conclusion: Peritoneal lavage has a systemic anti-inflammatory effect in severe AP and may be able to decrease the severity of severe AP.

Sunday Poster 26

PREOPERATIVE THERAPY DOES NOT ADVERSELY AFFECT NUTRITIONAL ANTHROPOMETRICS IN PANCREATIC ADENOCARCINOMA PATIENTS.

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Background
Although preoperative therapy for patients with localized pancreatic adenocarcinoma is currently being evaluated, its influence on host factors is unknown. No published data exists examining the nutritional impact of preoperative therapy. Body mass index ((BMI) kg/m^2) is a readily available anthropometric measure. Based on anecdotal experience, we hypothesized that preoperative therapy would not adversely affect patient BMI.

Methods
We reviewed and extracted prospectively collected data from our existing IRB approved database of patients receiving pancreatectomy from September 2002 to October 2006. BMI was calculated from measures obtained at initial evaluation and immediately prior to pancreatectomy. A change in BMI was calculated as the difference between preoperative BMI and initial BMI. For statistical evaluation, the patients were stratified into two groups: those who received preoperative therapy and the surgery first group. The differences in BMI change were compared using a two-sided t-test for unequal variances. Subsequent exploratory analyses were conducted by further stratifying patients into categories of obese (BMI >30) and non-obese (BMI <30) based on initial BMI.

Results
A total of 170 patients (median age: 63, range 25-86; 55% male) were identified. We found no statistically significant difference in BMI change between the preoperative and surgery first groups. Patients who were obese experienced significant reductions in BMI in both the preoperative and surgery first groups when compared to non-obese patients. Additional statistical comparisons are presented below.

	All patients		**Surgery first**		**Preoperative treatment**	
	Surgery first	Preoperative treatment	BMI<30	BMI>30	BMI<30	BMI>30
# Patients	55 (32%)	115 (68%)	38	17	96	19
SD	0.93	1.92	1.27	2.71	1.70	2.56
Mean Δ BMI	0.1	-0.2	0.5	-1.3	0.1	-1.3
Median Δ BMI	0.0	0.0	0.3	-0.4	0.1	-1.1
Max Δ BMI	3.0	4.2	4.2	2.9	4.2	2.9
Min Δ BMI	-2.8	-9.0	-3.2	-7.2	-9.0	-7.2
p	0.26		0.02		0.04	
Difference in Mean Δ BMI	n/a		-1.8		-1.4	

Discussion
In this cohort, preoperative treatment did not negatively impact change in BMI. Though obese patients had a significant decrease in BMI, this was not limited to the preoperative treatment group alone. These data suggest that the nutritional status of patients with localized pancreatic adenocarcinoma is not adversely affected during preoperative therapy.

Sunday Poster 27

(IN)ACCURACY OF PREOPERATIVE CLASSIFICATION OF PANCREATIC CYSTIC NEOPLASMS

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Introduction. The diagnosis of pancreatic cystic neoplasm (PCN) is being made with increasing frequency. The decision to pursue operative therapy is driven by the clinical characteristics and biological behavior of various PCN classifications. Thus, appropriate preoperative classification of PCN is critical. Advancements in radiologic and endoscopic assessment have refined the diagnostic evaluation, but the accuracy with which we preoperatively classify PCN remains undefined.

Methods. We reviewed a prospectively maintained single institution database to identify contemporary patients who underwent surgical resection from 1999 through 2009 for a preoperative diagnosis of pancreatic cystic neoplasm (serous cystadenoma (SC), mucinous cystadenoma (MC), intraductal papillary mucinous neoplasm (IPMN), or cystadenocarcinoma (CA)). All patients were evaluated in a multidisciplinary hepatopancreaticobiliary clinic. Medical reports were analyzed to compare preoperative and pathological PCN classification and to identify factors affecting the accuracy of preoperative diagnosis. To evaluate for temporal trends, patients were categorized into two eras by date of operation (1999-2004 and 2005-2009).

Results. Fifty-one patients underwent surgical resection for the preoperative indication of PCN between 1999 and 2009. Preoperative endoscopic ultrasonography was performed in 33% of cases before 2005 and in 70% after 2005. Operative management consisted of left pancreatectomy in 67%. Preoperative diagnoses were MC in 40%, IPMN in 27%, CA in 12%, SC in 6%, and indeterminate PCN in 6%; pathological diagnoses were MC in 33%, IPMN in 29%, SC in 21%, CA in 6%, and other in 4% (one cystic neuroendocrine neoplasm and one solid pseudopapillary tumor). Preoperative PCN classification was correct in 53% and preoperative classification of mucinous versus non-mucinous PCN was correct in 75%; the accuracy of preoperative classification did not differ between the two eras. Among cases with incorrect preoperative PCN classification, the pathological diagnosis was more likely to be less aggressive (46%) than more aggressive (13%) compared with the preoperative diagnosis ($p=0.043$). The likelihood of identifying occult invasive malignancy not confirmed preoperatively was 6%.

Conclusion. Despite ongoing advancements in preoperative diagnostic techniques, the ability to accurately classify PCN remains suboptimal. When incorrect, preoperative classification tends to overestimate the severity of diagnosis. The prevalence of occult invasive malignancy among PCN patients undergoing resection in a multidisciplinary setting is low.

Sunday Poster 28

RADIOPAQUE BIODEGRADABLE STENT FOR PANCREATOBILIARY APPLICATIONS – THE FIRST HUMAN PHASE I STUDY IN PANCREATICO-JEJUNOSTOMY

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Introduction. Previously we have experimentally tested biodegradable stents for pancreatobiliary applications. These stents may be used to treat benign strictures, or to secure the flow of bile, pancreatic juice or fluid collection after endoscopic or surgical procedures. The lack of suitable inserting device has delayed clinical endoscopic or percutaneous studies. However, we described a modified pancreatico-jejunostomy anastomosis, where a biodegradable stent was used to prevent pancreatic duct obstruction. The peripancreatically anchored jejunum was tightened over the pancreas with a purse-string suture. Although the use of any stent in pancreatico-jejunostomy anastomosis remains controversial, we used the novel pancreatico-jejunal anastomosis to study the usage of a biodegradable stent. The aim was to perform the first phase I study with a biodegradable stent in the human pancreatic duct.

Methods. In 29 patients who underwent Whipple operations with a novel purse-string type pancreatico-duodenectomy, a braided, gamma-sterilized, radiopaque 96L/4D polylactide stent was introduced into the pancreatic duct at the anastomosis. Complications, stent disappearance and late anastomotic patency with MRI were monitored.

Results. Hospital mortality was zero. Fifteen patients (52%) developed complications during the follow-up. There were ten patients with Grade A and five patients with Grade B/C (17%) delayed gastric emptying (DGE). Of these five patients, two patients also had Grade B-C haemorrhage and one patient had Grade C fistula (there were no Grade A/B fistulas, overall fistula rate 3%). Nine out of 26 patients (35%) with negative preoperative trypsinogen test, developed post-operative trypsinogen release suggesting development of pancreatitis. Within 12 months four patients died and one quitted the study. The stents disappeared in median three months. MRI interpretation of the anastomosis failed in one patient having ascites from metastasized disease. Of the 23 patients, 13 (57%) had the anastomosis well open, three (13%) had narrowing, while seven (30%) had the anastomosis obstructed. Hard pancreas, but not the duct size, was a risk for late anastomotic obstruction.

Conclusion. The rate for early and late complications with the novel anastomotic technique with a biodegradable stent is low when compared to our previous experience, and that indicates the biodegradable stent is well tolerated in the human pancreatic duct and encourages randomized trials in the future.

Sunday Poster 29

THE NUMBER OF METASTATIC LYMPH NODES BUT NOT LYMPH NODE RATIO IS AN INDEPENDENT PROGNOSTIC FACTOR AFTER RESECTION OF PANCREATIC CARCINOMA

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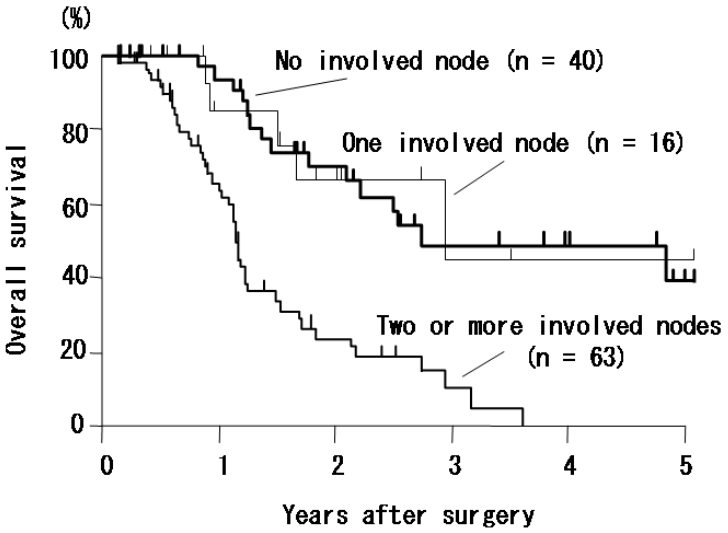
BACKGROUND: This study evaluated the prognostic significance of the number of metastatic lymph nodes and the ratio of metastatic nodes to total number of examined lymph nodes (lymph node ratio, LNR) after resection of pancreatic carcinoma.
STUDY DESIGN: Records of 119 consecutive patients with pancreatic ductal adenocarcinoma who underwent R0 or R1 pancreatectomy with regional node dissection were reviewed retrospectively. Clinical factors, pathological factors including number of metastatic nodes and LNR, and survival were analyzed by univariate and multivariate analysis.
RESULTS: Overall survival rates were 78%, 28%, and 20% at one, three, and five years, respectively. The median number of evaluated lymph nodes and involved nodes were 28 and 3, respectively. Univariate analysis revealed that tumor location, postoperative adjuvant chemotherapy, tumor differentiation, choledochal invasion, portal or splenic vein invasion, extrapancreatic nerve plexus invasion, resection margin status, node status, number of involved nodes, LNR, UICC pT factor, and UICC stage correlated significantly ($P < 0.05$) with increased survival. By multivariate analysis, negative node metastasis ($P = 0.008$) and zero or one involved node ($P = 0.004$), but not LNR, correlated independently with longer survival. The one, three, five-year survival rates of patients with zero or one metastatic node and patients with two or more metastatic nodes were 91%, 48%, and 40% and 66%, 10%, and 0%, respectively.
CONCLUSIONS: The number of metastatic nodes but not LNR is one of the most powerful prognostic factors after resection of pancreatic carcinoma.



Sunday Poster 30

THE CYCLOOXYGENASE-2 (COX-2) DEPENDENT EXPRESSION OF ANGIOGENIC CXC CHEMOKINES LIGAND (CXCL) IN HUMAN PANCREATIC CANCER CELL LINES.

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Background: Cyclooxygenase (COXs) is the limiting enzyme for production of prostaglandins (PGs). COX-2 expressed in response to cytokine, growth factor and other stimuli. It is well demonstrated that the growth of COX-2 positive pancreatic cancer (PaCa) cell lines are related with PGE2 production from arachidonic acid. On the other hand, CXC chemokines (CXCLs) are the crucial mediators on pancreatic cancer (PaCa) growth and invasion, too. ELR-positive-CXCL (ELR+CXCL) show angiogenic response via CXC-receptor-2 (CXCR2), in contrast, ELR-negative-CXCL (ELR-CXCL) show angiostatic response via CXCR3. The current study of cancer of other organs defines a role of COX-2 in the expression of angiogenic CXCL8 and CXCL5. From this standpoint, the progression of COX-2 positive PaCa is promoted by CXCLs and there receptors. The aim of this study is to establish the expression profiles of COX-2, CXCLs and receptors in PaCa cell lines.
Methods: COX-2 positive PaCa cell line, BxPc-3(B), KMP-4 (K4) and COX-2 negative cell lines, Panc-1 (P) and KMP-5 (K5) are were analyzed for ELR+CXCL (CXCL8, CXCL5), CXCR2 and CXCR3 by ELISA, western blot analysis (W/B) and Immunofluorescence studies (IMF).
Results: In COX-2 positive PaCa cell lines (B, K4) CXCL5 and CXCL8 are expressing significantly higher when compared with COX-2 negative cell lines (P, K5). IMF demonstrated that CXCL5 and CXCL8 are strongly expressed in B and K4, when compared with P and K5. CXCR2 were highly expressed in COX-2 positive cell lines. In COX-2 negative cell lines, the expression of CXCL5, CXCL8 and CXCR2 are weak but detectable level. The expression of the angiostatic receptor: CXCR3 are significantly weaker both in COX-2 positive and COX-2 negative cell lines.
Conclusion: Our data demonstrate that angiogenic CXCLs and CXCR2 are high in COX-2 positive PaCa cell lines. However, the CXCR3 expressions are weak in both COX-2 positive and COX-2 negative cell lines. Therefore, we concern that angiogenic CXCL are activated with COX-2 dependent manner. However, the progression of PaCa may have possibilities that depending on the imbalance of CXCRs. Since angiogenic CXCL regulated PaCa growth, and CXCR2 expression is higher than CXCR3, targeting CXCR2 may serve as a potentially powerful strategy to prevent progression of PaCa.

Sunday- Poster #31- Professor Rounds 11:25 – 11:50 am

LAPAROSCOPIC PANCREATICODUODENECTOMY CAN BE SAFELY IMPLEMENTED IN A HIGH-VOLUME PANCREATIC SURGERY CENTER.

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Introduction: Implementation of laparoscopic radical pancreaticoduodenectomy (LPD) has been appropriately met with apprehension by most high-volume pancreatic surgeons. We report our initial experience in performing LPD.

Methods: Patient selection was determined using the UPMC image-based mathematical model predictive of R0 resection and multidisciplinary review. Patients that received neoadjuvant therapy were excluded. We report a retrospective review of all patients that went to surgery with the intent for LPD in the first year of this program. A brief video companion to this presentation focusing on our evolving technique will be presented.

Results: Prior to performing this procedure, LPD was performed in 4 fresh-frozen cadavers, focusing upon trochar placement, methods of exposure, and anastomotic technique. Laparoscopic dissection with intentional conversion to open technique for anastomosis was subsequently performed on 2 patients. Eleven patients underwent planned LPD with intra-corporal reconstruction over a period of 14 months (see table). The pre-operative presumed diagnoses included duodenal GIST (n=1), IPMN (n=1), chronic pancreatitis (n=2), duodenal carcinoma (n=1), cholangiocarcinoma (n=3), and pancreatic adenocarcinoma (n=3). Conversion to an open procedure was necessary in 18.5% of patients (1 case for bleeding and 1 case for failure to progress). An R0 resection was achieved in all cases of malignancy. The mean number of retrieved lymph nodes was 17.3 (range 12-31). Mean operative time was 458 minutes (range 334-583 mins). Average blood loss was 385 ml. Mean length of stay was 12 days (range 6-28). Pancreatic leak occurred in 5 patients and all were Grade A by ISGPF criteria. There was no peri-operative mortality. The minor complication rate (Clavien score I-II) was 54 %. The major complication rate (Clavien score III-V) was 45%.

Conclusions: LPD can be implemented by high-volume pancreatic surgeons with advanced laparoscopic skills with acceptable morbidity, mortality, and cancer-related surgical outcomes. Table Listed Below.

Table 1. First Eleven Laparoscopic Whipple procedures at UPMC

Case#	Primary	Conversion	Margin	LN	OR time (min)	Pancreatic leak	Major complications
1	PAC	-	R0	21	583	-	-
2	D-GIST	-	R0	-	418	-	-
3	DAC	-	R0	22	392	Yes	DGE, bile gastritis
4	PAC	-	R0	16	474	-	-
5	PAC	-	R0	12	493	Yes	GJ anastomosis bleed
6	CP	Yes / Bleed	-	16	536	Yes	PV thrombosis, Ascites
7	IPMN	Yes / FTP	-	6	431	Yes	PE, Sepsis
8	CCA	-	R0	16	557	-	Reoperation - bleed
9	PAC	-	R0	31	360	-	-
10	PAC	-	R0	21	468	-	Sepsis
11	PAC	-	R0	17	334	Yes	-

CCA, Cholangiocarcinoma; CP, Chronic pancreatitis; D-GIST, Duodenal gastrointestinal stromal tumor; DAC, Duodenal adenocracinoma; DGE, Delayed gastric emptying; GJ, Gastro-jejunustomy; IPMN, Intrapapillary mucinous neoplasm; LN, Lymph nodes; FTP, Failure to progress; PAC, Pancreatic adenocarcinoma; PE, Pulmonary embolism; PV, Portal vein

Sunday- Poster #32- Professor Rounds 11:25 – 11:50 am

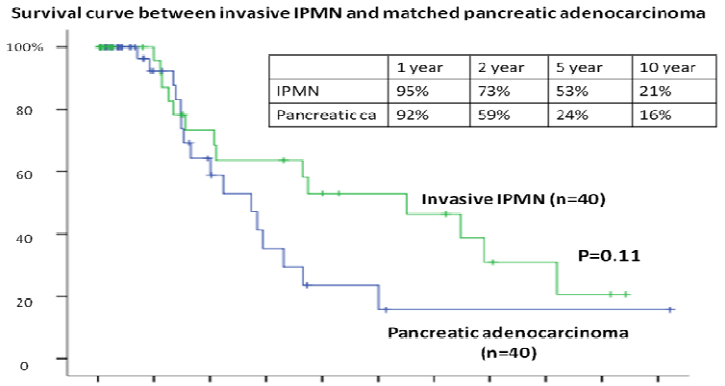
DOES RESECTION FOR AN INVASIVE INTRADUCTAL PANCREATIC MUCINOUS NEOPLASM PROVIDE A BETTER PROGNOSIS THAN FOR A PANCREATIC ADENOCARCINOMA? COMPARISON OF CASES MATCHED BY TMN STAGE AND TUMOR LOCATION.

Toshiyuki Moriya, M.D., Yasushi Hashimoto, M.D, L William Traverso, M.D. Department of General Surgery, Virginia Mason Medical Center, Seattle, WA

Background: After resection many feel that the prognosis is better for an invasive intraductal papillary mucinous neoplasm (IPMN) of the pancreas than for a pancreatic adenocarcinoma (PanCa). To support this concept we compared cases matched by pTNM and location.
Methods: Between 1989 and 2009 we analyzed 210 patients who underwent pancreatic resection for IPMN of which 40 had invasive IPMN. Excluded were cases of diffuse IPMN requiring total pancreatectomy (n=8). These 40 cases were matched for stage (AJCC pTNM 7th edition) and location with 40 PanCa cases resected during the same time period.
Results:

	Invasive IPMN	PanCA	P
N	40	40	-
Head, Body/tail	33,7	33,7	-
Mean number + nodes	1.8	3.5	<0.05
Lymphatic invasion	5	13	<0.05
Venous invasion	5	12	<0.05
Perineural invasion	24	28	NS

There was no significant survival difference (figure) between the invasive IPMN vs. PanCa groups (p=0.11). However, in patients with invasive IPMN there were less number of positive nodes, lymphatic and venous invasion versus the matched PanCa.
Discussion/Conclusion: Cases of invasive IPMN had less aggressive histology and a similar survival even though the IPMN malignancies tended to have more survivors beyond 4 years than the PanCa patients.



Sunday- Poster #33- Professor Rounds 11:25 – 11:50 am

SELECTIVE USE OF ARTERIAL RESECTION DURING PANCREATICODUODENECTOMY FOR PANCREATIC CANCER IS EFFECTIVE.

Contreras CM, Katz M, Tamm EP, Wang H, Pisters PWT, Abdalla EA, Vauthey JN, Lee JE, Evans DB and Fleming JB.

Background: **Portal and superior mesenteric venous (SMV) resection to achieve a negative margin resection (R0) during pancreatico-duodenectomy (PD) for pancreatic cancer is an accepted surgical approach. However, the use of arterial resection for the same purpose remains controversial.**

Hypothesis: We hypothesized that a very selective use of arterial resection can be applied to achieve R0 resection with acceptable surgical risks and cancer-related outcomes.
Design: Review and analysis of data collected from a prospective patient database.
Setting: A comprehensive cancer center with a multidisciplinary pancreatic cancer program.
Patients and Methods: The database was queried for all patients who received PD for cancer between October 1999 and August 2006 and the subset of patients who received arterial resection during PD. Descriptive clinicopathologic data and cancer-related outcomes were analyzed.
Results: Of 543 patients who underwent PD, 18 (3%) received arterial resection. All 18 were due to tumor involvement of the common hepatic artery or its branches, and 11(61%) were performed in conjunction with SMV resection. The median blood loss and operative time were 1,875ml and 716 minutes, respectively; there were no postoperative deaths. Pathologic examination identified a positive resection margin in 4(22%) specimens and microscopic lymphovascular and perineural invasion in 7(39%) and 12(67%) of the tumors. Notably, no positive lymph nodes were identified in 7(39%) specimens. During follow up 13 patients have died of disease, 2 are alive with disease and 3 are alive and free of disease. The Kaplan-Meier estimation of overall and disease-free survival was 17 and 14.7 months, respectively.
Conclusion: Pancreatic cancer involving arterial vessels is associated with an aggressive tumor biology making arterial resection during PD a rare event (3%). However, this series demonstrates that hepatic arterial resection can safely achieve an R0 resection and acceptable long-term results when carefully applied by experienced pancreatic surgeons.

Sunday- Poster #34- Professor Rounds 11:25 – 11:50 am

SURGICAL MANAGEMENT OF SOLID-PSEUODOPAPILLARY NEOPLASMS OF THE PANCREAS (FRANZ OR HAMOUDI TUMORS): A LARGE SINGLE-INSTITUTIONAL SERIES

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INTRODUCTION/BACKGROUND: Solid-pseudopapillary neoplasms (SPNs) are rare pancreatic tumors with malignant potential. Clinico-pathologic characteristics and outcomes of patients with SPN were reviewed.
METHODS: Long-term outcomes were evaluated in patients with an SPN who were followed from 1970 to 2008.
RESULTS: Thirty-seven patients were identified with an SPN. Thirty-three (89%) were women, and median age at diagnosis was 32 years. Most patients were symptomatic; the most common symptom was abdominal pain (81%). Thirty-six patients underwent resection; one patient with distant metastases was not operated on. There were no 30-day mortalities. Median tumor size was 4.5 cm. Thirty-four patients underwent an R0 resection, 1 had an R1 resection, and 1 had an R2 resection. Two patients had lymph node metastases, and one patient had perineural invasion. After resection, 34 (94%) patients remain alive. One patient died of unknown causes 9.4 years after resection, and another died of unrelated causes 25.6 years after operation. The patient with widespread disease who did not have resection died 11 months after diagnosis. Thirty-five of the 36 patients having resection remained disease free, including those who died of unrelated causes (median follow-up, 4.8 years). One patient developed a recurrence 7.7 years after complete resection. She was treated with gemcitabine based therapy and remains alive 13.6 months after recurrence.
DISCUSSION/CONCLUSIONS: SPNs are rare neoplasms with malignant potential found primarily in young women. Formal surgical resection may be performed safely and is associated with long-term survival.

Sunday- Poster #35- Professor Rounds 11:25 – 11:50 am

INHIBITION OF SIRT1 AS A NOVEL THERAPEUTIC STRATEGY FOR PANCREATIC CANCER

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SIRT1 is the human orthologue of SIR2, a conserved NAD-dependent protein deacetylase that regulates life span in accord with nutritional provision and under conditions of stress. Thus SIRT1 has been proposed as a regulator of aging and senescence. Due to its pro-survival function, SIRT1 may play a role in the pathogenesis of cancer. The **aim** of this study was to evaluate the role of SIRT1 in the pathogenesis of pancreatic cancer.

Methods: SIRT1 expression was reduced in pancreatic cancer cell lines (MiaPaCa-2 & S2VP10) and cholangiocarcinoma cell line KMBC by SIRT1 siRNA. Two unique sequences of SIRT1 siRNA were used to rule out any off-target effects of siRNA. Cell viability was measured by an MTT assay. The effect of SIRT1 down-regulation on cell cycle and annexin V staining (marker for apoptosis) was evaluated by flow cytometry. LC3 II protein expression, a marker for autophagy, was evaluated by western blot. Autophagy was further evaluated by immunofluorescence by evaluating the pattern of distribution of LC3II.

Results: Inhibition of SIRT1 expression by SIRT1 siRNA markedly reduced the viability of both the pancreatic cancer cell lines tested at 96h. Viability (% of control) expressed as mean ± SEM: MiaPaCa-2: 22.6±3.2, S2VP10 42.3±7.61. Down-regulation of SIRT1 also reduced viability in cholangiocarcinoma cell lines suggesting that the pro-survival effects of SIRT1 may be generalized to other cancers as well. To evaluate the mechanism by which SIRT1 induces cell death in pancreatic cancer cells, we evaluated the impact of SIRT1 downregulation on three common pathways of cell death and inhibition of proliferation: apoptosis, cell cycle arrest and autophagy. Inhibition of SIRT1 expression is not associated with annexin V positivity thus suggesting that SIRT1-inducedcell death is not through apoptosis. Furthermore, SIRT1 down-regulation does not induce cell cycle arrest and the distribution of cancer cells in different phases of cell cycle remains unchanged with SIRT1 down-regulation. Remarkably inhibition of SIRT1 is associated with marked increase in LC3 II protein, suggesting activation of autophagy. Downregulation of SIRT1 in pancreatic cancer cells led to a punctuate distribution of LC3 as compared to diffuse pattern in control cells, suggesting localization of LC3 to autophagosomes and further supporting activation of autophagy.

Conclusion: Silencing of SIRT1 expression activates cell death by autophagy in pancreatobiliary cancer cells. SIRT1 holds a great promise as a potential candidate for the drug development.

Sunday Poster 36

INTRADUCTAL MUCINOUS PAPILLARY NEOPLASMS: GENETIC CHARACTERIZATION OF LESION PROGRESSION.

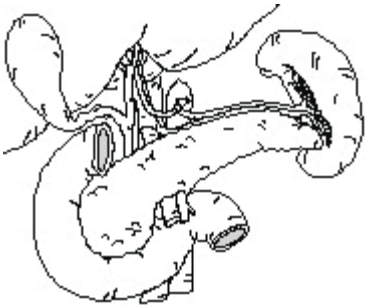
R. P. Jury[1], T. J. Geddes[2], L. E. Fortier[2], M. A. Farinola[3], B. L. Pruetz[2], G. D. Wilson[4]
[1] Department of General Surgery, [2]Beaumont BioBank, [3] Department of Anatomic Pathology, [4] Department of Radiation Oncology, William Beaumont Hospital, Royal Oak, Michigan, USA

Background: The timing and choice of appropriate surgical treatment for intraductal papillary mucinous neoplasm (IPMN) remains a major clinical challenge. Current evidence clearly supports the dysplasia to carcinoma sequence of malignant transformation in IPMN at unknown rates of progression. This study investigates the changes in gene expression that occur in IPMNs during their progression from low grade to high grade dysplasia and on to invasive carcinoma.
Methods: IPMN cases were identified from the pathology archives treated by a single surgeon (RJ). Foci of different grades of dysplasia and invasion, from the same specimens where possible, were identified by a pathologist and marked on the cover slip of the slide. Serial sections were cut and processed for laser capture microdissection. Areas of interest (1 – 2 mm^2), were dissected representing ~5,000 cells captured. Captured tissue was subjected to RNA extraction using the NuGEN WT-Ovation™ FFPE System. The extracted RNA was analyzed for integrity and hybridized to Affymetrix Human Exon 1.0 ST arrays using proprietary procedures. Gene expression data were normalized and filtered using GCOS software and analyzed using Expression Console software and statistical analysis.
Results: 96 genes were identified that were differentially expressed within lesions of varying dysplastic change and invasion from the same IPMN patient. Table 1 shows the top 20 genes overexpressed in areas of invasive IPMN compared to low grade disease.
Discussion: This study identifies a set of pancreas-specific genes associated with the progression of IPMN to malignancy within the same patient as well as changes in some unrelated genes. Many of the overexpressed genes lead to production of enzymes with capacity to break down connective tissue potentially allowing invasion. Further analysis of different mucinous lesions to uncover the genes most commonly associated malignant progression will ultimately help in the choice of appropriate and timely surgical therapy.

Table Listed Below

Gene symbol	Gene product
PLA2G1B	phospholipase A2, group IB (pancreas)
GP2	glycoprotein 2 (zymogen granule membrane)
CPA1	carboxypeptidase A1
PNLIPRP2	pancreatic lipase-related protein 2
INS	insulin
ZNF30	zinc finger protein 30
CLPS	colipase, pancreatic (CLPS)
REG3A	regenerating islet-derived 3 alpha
ELA2B	elastase 2B
REG1P	regenerating islet-derived 1 pseudogene
CPA2	carboxypeptidase A2 (pancreatic)
REG1A	regenerating islet-derived 1 alpha
ELA2A	elastase 2A
SFRP2	secreted frizzled-related protein 2
PNLIPRP1	pancreatic lipase-related protein 1
APCS	amyloid P component, serum
GABRP	gamma-aminobutyric acid (GABA) A receptor, pi
CRISP3	cysteine-rich secretory protein 3
CTRC	chymotrypsin C (caldecrin)
CD163	CD163 molecule

Table 1. Top 20 genes associated with progression to i



Pancreas Club, Inc.

2009/2010 Membership & Meeting Attendees

Please advise the Registration Desk of any corrections or changes.

List is current as of 4/12/10
An updated list will be available on our website.

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Past Meetings of the Pancreas Club

Date & Location	Host
1966 Northwestern	Marion Anderson
1967 Philadelphia	John Howard
1968 University of California SF	Leon Goldman
1969 Mt. Sinai Hospital	David Dreiling
1970 University of Chicago	Edward Paloyan
1971 Sheraton Hotel – Philadelphia	John Howard
1972 University of California SF	Englebert Dunphy
1973 Mt. Sinai Hospital	David Dreiling
1974 - No Meeting	
1975 Univ. of Texas-San Antonio	Bradley Aust
1976 Doral on the Ocean – Miami	Robert Zeppa
1977 Toronto, Canada	Roger Keith
1978 Jockey Club-Las Vegas	Charles Frey
1979 LSU Med Center – New Orleans	Isadore Cohn
1980 Salt Lake City	Frank Moody
1981 Alumni Hall-NYU	John Ranson
1982 University of Chicago	A.R. Moosa
1983 Washington Hilton	Francis Milligan
1984 LSU Med Center – New Orleans	Francis Nance
1985 Mt. Sinai Hospital	David Dreiling
1986 Ft. Miley VA-San Francisco	Carlos Pellegrini
1987 University of IL- Chicago	Phillip Donahue
1988 Tulane Univ.- New Orleans	Elmo Cerise
1989 Washington Hilton	Gregory Bulkley, Frances Milligan John Cameron
1990 Univ. of Texas-San Antonio	Bradley Aust
1991 Tulane Univ.- LSU	Elmo Cerise J.Patrick O'Leary
1992 University of California SF	Carlos Pellegrini
1993 Mass. General Hospital-Boston	Andrew Warshaw

1994 Tulane University	Elmo Cerise J. Patrick O'Leary
1995 University of California SD	A.R. Moosa
1996 Laurel Heights-UCSF	Sean Mulvihill
1997 Univ. Health Sci.-Bethesda	John W. Harmon
1998 LSU-Tulane	J.Patrick O'Leary Elmo Cerise
1999 Peabody-Orlando	Michael M. Murr, James G. Norman
2000 University of California SD	A.R. Moosa
2001 Hilton Atlanta	Aaron Fink
2002 San Francisco	Kimberly Kirkwood
2003 Orlando FL	Michel Murr
2004 New Orleans	Alton Ochsner
2005 Chicago IL	Gerard V. Aranha Richard Bell
2006 Los Angeles	Howard A. Reber
2007 Children's Medical Center DC	Dana Anderson
2008 San Diego CA	Mark Talamini Mike Bouvet
2009 Chicago IL	Gerry Aranha Mark Talamonti David Bentrem

2011 Meeting

May 7 & 8, 2011
Chicago, IL