47th Annual Meeting of the Pancreas Club

MAY 17-18, 2013
Walt Disney World Swan & Dolphin Hotel
in Lake Buena Vista, Florida
Welcome to the 47th 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 200 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 your 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 three awards for 2013 which will be presented at the Saturday 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/Pancreas Club 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.

- **John Howard Annual Research Award:** $1,000 for the best presentation from someone within 5 years of their end of residency.
MEETING LOCATION

Walt Disney World Swan & Dolphin Hotel
1500 Epcot Resorts Boulevard, Lake Buena Vista, FL 32830

MEETING HOURS

**Registration** *Southern Foyer I-II*
Friday, May 17, 2013 • 12:00pm – 5:30pm
Saturday, May 18, 2013 • 6:45am – 6:00pm

**Scientific Sessions** *Southern IV-V w/ Posters in Southern II*
Friday, May 17, 2013 • 1:00pm – 5:15pm
Saturday, May 18, 2013 • 7:00am – 5:30pm

**Exhibit Hours** *Exhibits located in Southern I*
Friday, May 17
1:00pm – 6:30pm  Exhibits Open
2:45pm – 3:00pm  Refreshment Break in Exhibit Area
5:15pm – 6:30pm  Welcome Reception in Exhibit Area

Saturday, May 18
7:00am – 1:00pm  Exhibits Open
7:00am – 7:45am  Continental Breakfast in Exhibit Hall
9:45am – 10:00am Refreshment Break in Exhibit Hall
12:00pm – 1:00pm  Luncheon at Hotel

**Annual Dinner Reception** *Southern III Foyer and Dinner in Southern III*
Saturday, May 18, 2013 • 7:00pm – 10:00pm
Walt Disney World Swan & Dolphin Hotel
1500 Epcot Resorts Boulevard, Lake Buena Vista, FL 32830
General Information/Accreditation

CONTINUING MEDICAL EDUCATION

Meeting Objectives

At the conclusion of this meeting, participants should be able to:

- Address the challenges of the management of all of the complexities of pancreatic diseases with considerably greater insight and evidence-based decision making
- List additional/different treatment options for patients based on evidence provided in abstract presentations

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 join sponsorship of the American College of Surgeons and the Pancreas Club. The American College of 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 live activity for a maximum of 10.25 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

DISCLOSURE INFORMATION

In compliance with ACCME Accreditation Criteria, 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.

PROGRAM COMMITTEE MEMBERS

William H. Nealon, Chair
David Adams, MD
Gerard Aranha, MD
Richard Bold, MD
John Christein, MD
Michael B. Farnell, MD
Jason Fleming, MD
Nipun Merchant, MD

James Moser, MD
L. William Traverso, MD
Santhi Swarrop Vege, MD
Charles M. Vollmer, MD
Sharon Weber, MD
John Windsor, MD
Christopher Wolfgang, MD
Nicholas Zyromski, MD
47TH ANNUAL MEETING OF THE PANCREAS CLUB
Program-at-a-Glance

Meeting Rooms
Scientific Sessions: Southern IV-V
Registration: Southern Foyer I-II
Posters: Southern II
Exhibits: Southern I

FRIDAY, MAY 17, 2013

12:00pm – 5:30pm  Registration
1:00pm – 6:30pm  Exhibits Open
1:00pm – 2:45pm  Scientific Session I: Cancer Clinical/Readmissions/
Complications/Outcomes
2:45pm – 3:00pm  Break with Exhibitors & View Posters
3:00pm – 3:50pm  Professor Rounds w/ Posters (see page 15 for list of posters)
4:00pm – 5:15pm  Scientific Session II: Cancer Clinical/Translational Studies/
NET/IPMN
5:15pm – 6:30pm  Welcome Reception & Poster Viewing (Southern I-II)

SATURDAY, MAY 18, 2013

6:45am – 6:00pm  Registration
7:00am – 1:00pm  Exhibits Open
7:00am – 7:45am  Continental Breakfast
7:45am – 8:00am  Welcome and Introductory Remarks
8:00am – 9:45am  Scientific Session III: Basic Science Studies in Pancreas Cancer
9:45am – 10:00am  Break with Exhibitors & Poster Viewing
10:00am – 11:00am  Scientific Session IV: Surgical Techniques
11:00am – 12:00pm  Professor Rounds with Posters
12:00pm – 1:00pm  Lunch (Southern III)
1:00pm – 1:45pm  How I Do It Session: Minimally Invasive
Pancreatoduodenectomy-Ready For Primetime?
1:45pm – 3:35pm  Scientific Session V: Pancreatitis
3:40pm – 5:30pm  Scientific Session VI: Cancer Clinical/Timing of Therapy/
Preoperative Evaluation/Lymph Node Status
5:30pm – 6:00pm  Pancreas Club Brief Business Meeting (Southern IV-V)
7:00pm – 10:00pm  Pancreas Club Annual Dinner
Swan/Dolphin Hotel-Reception from 7:00pm-8:00pm
in Southern Foyer III followed by dinner in Southern III

Schedule-at-a-Glance 3
Sponsors & Exhibitors

The Pancreas Club gratefully acknowledges support for the 47th Annual Pancreas Club Meeting from the following:

EDUCATIONAL GRANT SUPPORT

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Covidien
Digestive Care, Inc.

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RESIDENT AWARD SUPPORT

Arpa Foundation
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The Pancreas Club

EXHIBITORS

Aptalis Pharma
ChiRhoClin, Inc.
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Digestive Care, Inc.
NewLink Genetics Corporation
12:00pm – 5:30pm  Registration  
*Southern Foyer I-II/Southern I*

1:00pm – 6:30pm  Exhibits Open  
*Southern I*

12:55pm – 1:00pm  Welcome & Introductory Remarks  
*Southern IV-V*
William H. Nealon, MD, Vanderbilt University, Nashville, TN
William Traverso, MD, St. Luke’s Hospital, Boise, ID
Michael Farnell, MD, Mayo Clinic, Rochester, MN

1:00pm – 2:45pm  Scientific Session I: Cancer  
*Southern IV-V*
**Cancer Clinical/Readmissions/Complications/Outcomes**
MODERATOR: Charles M. Vollmer, MD

1:00pm – **S001** COMPARING EARLY AND DELAYED READMISSION AFTER SURGERY FOR PANCREAS CANCER: A SEER-MEDICARE STUDY  
*Marquita R Decker, MD, MPH, David Y Greenblatt, MD, MSPH, Chee P Lin, MS, Jeffery A Havlena, MS, Sharon M Weber, MD, Noelle Loconte, MD, Maureen A Smith, MD, MPH, PhD, Caprice C Greenberg, MD, MPH, Emily Winslow, MD University of Wisconsin: Department of Surgery - Wisconsin Surgical Outcomes Research (WISOR), Department of Medicine - Division of Hematology-Oncology, UW Comprehensive Cancer Center, UW Health Innovation Program (LONG)*

1:15pm – **S002** REDUCING READMISSIONS FOLLOWING PANCREATECTOMY: COORDINATION OF THE CARE CONTINUUM  
*Eugene P Ceppa, MD, Henry A Pitt, MD, Attila Nakeeb, MD, C Max Schmidt, MD, Nicholas J Zyromski, MD, Michael G House, MD, Alisha George-Minkner, RN, Elizabeth W Brand, BSN, Denise J Weidert, MSN, Keith D Lillemoe, MD Indiana University; Indiana University Health (LONG)*

1:30pm – **S003** READMISSIONS AFTER PANCREAS SURGERY: COMPLEX AND COSTLY  
*Zeling Chau, MD, Sing Chau Ng, BA, MS, Elan R Witkowski, MD, Tara S Kent, MD, Arthur J Moser, MD, Mark P Callery, MD, Jennifer F Tseng, MD, MPH Beth Israel Medical Center- Harvard Medical School; University of Massachusetts Medical School (SHORT)*

1:35pm – **S004** LIMITATIONS OF NSQIP IN REPORTING COMPLICATIONS FOR PATIENTS UNDERGOING PANCREATECTOMY: UNDERSCORING THE NEED FOR A PANCREAS-SPECIFIC MODULE  
*Irene Epelboym, MD, Irmina Gawlas, BA, James A Lee, MD, Beth A Schrope, MD, PHD, John A Chabot, MD, John D Allendorf, MD Columbia University Medical Center, Department of Surgery (LONG)*

1:50pm – **S005** TRENDS IN THE SURGICAL TREATMENT OF PANCREATIC ADENOCARCINOMA  
*Siaavash Raigani *, John Ammori ^, MD, FACS, Julian Kim ^, MD, FACS, Jeffrey Hardacre *, MD, FACS Case Western Reserve University School of Medicine*, University Hospitals Case Medical Center Department of Surgery^ (LONG)
2:05pm – **S006** NINETY-DAY MORTALITY RATE AFTER RESECTION OF CANCER OF THE PANCREAS IS NEARLY DOUBLE THIRTY-DAY MORTALITY: ANALYSIS OF 20,000 PANCREATECTOMIES IN THE NATIONAL CANCER DATA (NCDB) Richard S Swanson*, MD, Kathy Mallin^, PhD, Christopher M Pezzi**, MD, Andrew Stewart^, MA, Bryan Palis^, MA, David P Winchester^, MD *Department of Surgery, Brigham and Women’s Hospital, Boston, MA, ^Commission on Cancer of the American College of Surgeons, Chicago, IL, and **Department of Surgery, Abington Memorial Hospital, Abington, PA (LONG)

2:20pm – **S007** PATTERNS OF FAILURE FOLLOWING WHIPPLE PROCEDURE FOR RESECTABLE PANCREATIC DUCTAL ADENOCARCINOMA Avani S Dholakia, BS, Rachit Kumar, MD, Aaron T Wild, BA, Amy Hacker-Price, MS, PAC, Susannah Ellsworth, MD, Siva P Raman, MD, Dung T Le, MD, Ana De Jesus-Acosta, MD, Le Zheng, MD, PhD, Elliot K Fishman, MD, Ralph H Hruban, MD, Matthew J Weiss, MD, Johns Hopkins School of Medicine (SHORT)

2:25pm – **S008** PERIOPERATIVE BLOOD TRANSFUSION REDUCES SURVIVAL IN PATIENTS WITH PANCREATIC ADENOCARCINOMA: A MULTI-INSTITUTIONAL STUDY OF 698 PATIENTS Jeffrey M Sutton, MD, David A Kooby, MD, Gregory C Wilson, MD, Dennis J Hanseman, PhD, Shishir K Maithel, MD, David J Bentrem, MD, Sharon M Weber, MD, Clifford S Cho, MD, Emily R Winslow, MD, Charles R Scoggins, MD, Robert C Martin, MD, Hong J Kim, Authors are from Departments of Surgery from Institutions Representing the Central Pancreas Consortium. (Please contact primary author if accepted for specific institutions as they will not all fit within the character limits.) (LONG)

2:30pm – **S009** COMPARING THE IMPACT OF COMPLICATIONS AFTER MAJOR PANCREATECTOMIES USING THE POSTOPERATIVE MORBIDITY INDEX Charles M Vollmer, MD, Russell S Lewis, BS, Bruce L Hall, MD, PhD, John D Allendorf, MD, Joal P Beane, MD, Stephen W Behrman, MD, Mark P Callery, MD, John D Christein, MD, Jeffrey A Drebin, MD, PhD, Irene Epelboym, MD, Jin He, MD, Henry A Pitt, MD, The University of Pennsylvania; Washington University of St. Louis; BIDMC - Harvard University; Columbia University; Indiana University; University of Wisconsin; Johns Hopkins University; University of Alabama at Birmingham; University of Tennessee (LONG)

2:45pm – 3:00pm Break with Exhibitors & Poster Viewing

3:00pm – 3:50pm Professor Rounds with Posters (see page 15 for list of posters)

**Southern I-II**

MODERATORS: Santhi Swarrop Vege, MD & Gerard Aranha, MD

Posters P001-P025: Authors will be by their posters to discuss their research poster presentations. Abstracts of note are identified in the program will be part of the *Poster-side Professor Rounds*. Each invited Professor will discuss several posters. Posters of disctinctions will be identified on the poster board with a gold star.
4:00pm – 5:15pm

Scientific Session II: Cancer Clinical/Translational Studies/NET/IPMN

MODERATORS: L. William Traverso, MD & Kyoichi Takaori, MD

4:00pm – S010 COEXPRESSION OF MUC16 AND MESOTHELIN IS RELATED TO THE INVASION PROCESS AND SURVIVAL FOR PANCREATIC DUCTAL ADENOCARCINOMA Seiko Hirono, Masaji Tani, Manabu Kawai, Kein-ichi Okada, Motoki Miyazawa, Atushi Shimizu, Yuji Kitahata, Hiroki Yamae Wa Kayama Medical University (LONG)

4:15pm – S011 DCK IS A PROGNOSTIC MARKER AND CORRELATES WITH 5-FLUOROURACIL RESPONSE AND HUR STATUS IN PANCREATIC CANCER: ANALYSIS FROM THE RTOG 9704 TRIAL Florecia McAllister, Jennifer Moughan, Kathryn A Winter, Ana de Jesus Acosta, Rachana H Lankapalli, Shruti Lal, Charles J Yeo, Agnieszka K Witkiewicz, Christine Iacobuzio-Donahue, Daniel Laheru, Jonathan R Brody 1Department of Medical Oncology– Johns Hopkins Hospital, Baltimore, MD, 2 RTOG Statistical Center, Philadelphia, PA, 3 Department of Pathology– Johns Hopkins Hospital, Baltimore, MD, 4 Department of Surgery - Division of Surgical Research, The Jefferson (SHORT)

4:20pm – S012 PROGNOSTIC AND BIOLOGICAL ROLE OF MIR-101, MIR-155 AND MIR-21 IN PANCREATIC INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS E Giovannetti 1, MD, PhD, S Caponi 2, MD, N De Lio 2, MD, V Perrone 2, MD, E Vasile 2, MD, N Funel 2, PhD, G Kazemier 1, MD, G J Peters 1, MD, D Campani 2, MD, U Boggi 2, MD University of Pisa, Italy (LONG)

4:25pm – S013 DOES PREOPERATIVE IMAGING ACCURATELY PREDICT MAIN DUCT INVOLVEMENT IN INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM? Morgan Barron*, Joshua Waters*, MD, Janak Parikh*, MD, John DeWitt^, MD, Mohammad Ali Al-Haddad^, MD, Eugene Ceppa*, MD, Michael House*, MD, Nicholas Zyromski*, MD, Attila Nakeeb*, MD, C. Max Schmidt*, MD Indiana University School of Medicine, Department of Surgery*; Indiana University School of Medicine, Department of Gastroenterology^ (LONG)

4:40pm – S014 CONSERVATIVE MANAGEMENT OF BRANCH DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM OF THE PANCREAS WITH WORRISOME FEATURES Hiroyuki Hisai*, Yutaka Okagawa*, Hironori Wada*, Yutaka Koshiba*, Yusuke Kanari*, Etsu Miyazaki*, Yoshiharu Maeda^, Masafumi Sato^, Ryosuke Kawasaki^, Hiroshi Gyobo^, Seiichiro Nakajima^ *Department of Gastroenterology, Japan Red Cross Date General Hospital, Date, Japan, ^Department of Surgery, Japan Red Cross Date General Hospital, Date, Japan (LONG)

4:45pm – S015 SMALL NON-FUNCTIONAL PANCREATIC NEUROENDOCRINE TUMORS ARE ASSOCIATED WITH A LOW INCIDENCE OF NODAL METASTASIS AND AN EXCELLENT OVERALL SURVIVAL P A Tostes, MD, S F Tatishchev, MD, D W Dawson, MD, PhD, B M Clerkim, RN, MPH, J S Tomlinson, MD, PhD, O J Hines, MD, H A Reber, MD, T R Donahue, MD University of California, Los Angeles (LONG)

5:00pm – S016 PREDICTORS OF LYMPH NODE METASTASES AND IMPACT ON SURVIVAL IN RESECTED PANCREATIC NEUROENDOCRINE TUMORS, A SINGLE CENTER EXPERIENCE Joyce Wong, MD, William Fulp, PhD, Jonathan R Strosberg, MD, Larry K Kvols, MD, Pamela J Hodul, MD Moffitt Cancer Center (LONG)
SATURDAY, MAY 18, 2013

6:45am – 6:00pm  **Registration**  
**Southern Foyer I-II**

7:00am – 1:00pm  **Exhibits Open**  
**Southern I**

7:00am – 7:45am  **Continental Breakfast**  
**Southern I**

7:45am – 8:00am  **Welcome & Introductory Remarks**  
**Southern IV-V**
William H. Nealon, MD, Vanderbilt University, Nashville, TN
William Traverso, MD, St. Luke’s Hospital, Boise, ID
Michael Farnell, MD, Mayo Clinic, Rochester, MN

8:00am – 9:45am  **Scientific Session III:**
**Basic Science Studies in Pancreas Cancer**  
**Southern IV-V**
MODERATOR: Jim Moser, MD

8:00am – **S017**  **STAT3 INHIBITION ATTENUATES CHEMoresistance and ENHANCES Drug Delivery in Pancreatic Cancer**  
Jason Castellanos, MD, Ngaraj Nagathihalli, PhD, Nagaraj Nagathihalli, PhD, Yughander Beesetty, MS, Michelle Reyzer, PhD, Chanjuan Shi, MD, Richard Caprioli, PhD, Nipun Merchant, MD Vanderbilt University Medical Center (LONG)

8:15am – **S018**  **Tumor Associated Fibroblasts Promote Pancreatic Tumor Progression and Chemoresistance Through a Potential C-Met Dependent-ID1 Signaling Axis**  
Adrian C Vlada, MB, BCh, Dongyu Zhang, MD, PhD, Song Han, PhD, George A Sarosi, MD, Kevin E Behrens, MD, Steven J Hughes, MD, Jose G Trevino, MD University of Florida (LONG)

8:30am – **S019**  **Biophysical Markers Derived from Standard Pre-Treatment Imaging Quantitatively Describe Gemcitabine Delivery and Chemoradiation Response in Human Pancreatic Adenocarcinoma**  
Mark Truty, MD, Eugene Koay, MD, Vittorio Cristini, PhD, Varadhachary Gauri, MD, William Plunkett, PhD, Matthew Katz, MD, Jeffrey E Lee, MD, Jason B Fleming, MD MD Anderson Cancer Center, University of New Mexico (LONG)

8:45am – **S020**  **Implications for Pancreatic Cancer Cell Resistance and Survival: Critical Cancer-Related Genes are Selectively Regulated by HUR When Exposed to Chemotherapeutics and Nutrient Deprivation**  
Richard A Burkhart, MD, Danielle Pineda, MD, Joseph A Cozziorto, Charles J Yeo, MD, Jordan M Winter, MD, Judith C Keen, PhD, Jonathan R Brody, PhD Department of Surgery and the Jefferson Pancreas, Biliary and Related Cancer Center, Thomas Jefferson University, Philadelphia, PA (LONG)

9:00am – **S021**  **CXCR2 Inhibition Provides Protection Against Metastases in Pancreatic Ductal Adenocarcinoma**  
Colin Steele, MD, Jennifer Morton, PhD, Colin McKay, MD, Jeffry Evans, MD, Ross Carter, MD, Owen Sansom, PhD Beatson Institute for Cancer Research, Glasgow, UK. West of Scotland Pancreatic Unit, Glasgow Royal Infirmary, Glasgow, UK (LONG)
9:15am – S022 MULTITARGETED APPROACHES IN THE TREATMENT OF PANCREATIC DUCTAL ADENOCARCINOMA (PDAC) Brett L Broussard*, MD, Juan P Arnoletti^, MD, Alevtina Mikhaylina*, MS, Martin J Heslin*, MD, Andrey Frolov*, MD, PhD *Department of Surgery, University of Alabama at Birmingham, Birmingham, Alabama; ^Department of Surgery, Florida Hospital, Orlando, Florida (LONG)

9:30am – S023 DEVELOPMENT OF A MUC1-DRIVEN DIPHTHERIA TOXIN-A NANTHERAPY FOR THE SELECTIVE KILLING OF AGGRESSIVE PANCREATIC CANCER CELLS Renée M Tholey, MD, Richard A Burkhart, MD, Joseph A Cozzitorto, Charles J Yeo, MD, Janet A Sawicki, PhD, Jonathan R Brody, PhD, Jordan M Winter, MD Department of Surgery and the Jefferson Pancreas, Biliary, and Related Cancer Center, Thomas Jefferson University, Philadelphia. Lankenau Institute for Medical Research; Wynnewood (SHORT)

9:35am – S024 INHIBITION OF CENTROSOME DUPLICATION AS A THERAPEUTIC APPROACH TO PANCREATIC CANCER WITH POTENTIALLY FEW SIDE EFFECTS Shrutika Mehta, MS, Chaozhong Zou, PhD, Mark S Talamonti, MD, Qingshen Gao, MD Division of Hematology/Oncology, Department of Medicine, Department of Surgery, NorthShore University HealthSystem, Affiliate of the University of Chicago Pritzker School of Medicine, MBP Program, Northwestern University, Evanston, IL (SHORT)

9:40am – S025 PINCH EXPRESSION IN PANCREATIC NEUROENDOCRINE TUMORS Kelly C Hewitt, MD, Jill Shea, PhD, W. Cory Johnston, MD, Lyska Emerson, MD, Courtney L Scaife, MD University of Utah (SHORT)

9:45am – 10:00am Break with Exhibitors & Poster Viewing Southern I-II

10:00am – 11:00am Scientific Session IV: Surgical Techniques Southern IV-V MODERATOR: William H. Nealon, MD

10:00am – S026 2000 CONSECUTIVE PANCREATICODUODENECTOMIES John L Cameron, MD, Jin He, MD Johns Hopkins Hospital (LONG)

10:15am – S027 A MULTI-INSTITUTIONAL EXTERNAL VALIDATION OF THE FISTULA RISK SCORE FOR PANCREATICODUODENECTOMY Benjamin C Miller, BA, John D Christein, MD, Stephen W Behrman, MD, Jeffrey A Drebin, MD, PhD, Wande B Pratt, MD, MPH, Mark P Callery, MD, Charles M Vollmer, MD Hospital of the University of Pennsylvania, University of Alabama, Birmingham Medical Center, University of Tennessee Health Science Center, Beth Israel Deaconess Medical Center (LONG)

10:30am – S028 250 ROBOTIC ASSISTED MAJOR PANCREATIC RESECTIONS Herbert J Zeh, MD, Brian A Boone, MD, David L Bartlett, MD, A. James Moser, MD, Amer Zureikat, MD University of Pittsburgh Medical Center, Pittsburgh, Pa (LONG)

10:45am – S029 A STANDARDIZED RADIOGRAPHIC ASSESSMENT OF THE TUMOR-VEIN INTERFACE PREDICTS THE NEED FOR VENOUS RESECTION AND THE PRESENCE OF HISTOLOGIC VENOUS INVASION IN BORDERLINE RESECTABLE PANCREATIC CANCER Hop S Tran Cao, MD, Aparna Balachandran, MD, Huamin Wang, MD, PhD, Jason B Fleming, MD, Jeffrey E Lee, MD, Peter W Pisters, MD, Matthew H Katz, MD Department of Surgical Oncology, U.T. M.D. Anderson Cancer Center; Department of Diagnostic Radiology, U.T. M.D. Anderson Cancer Center; Department of Pathology, U.T. M.D. Anderson Cancer Center (SHORT)
10:50am – **S030** MARGIN STATUS IMPACTS SURVIVAL AFTER PANCREATICODUODENECTOMY; BUT NEGATIVE MARGINS SHOULD NOT BE CHASED Alexander S Rosemurgy, MD, Abhishek Mathur, MD, Michelle Vice, Tony Kurian, BS, Paul G Toomey, MD, Kenneth Luberice, BS, Sharona B Ross, MD Florida Hospital Tampa (SHORT)

10:55am – **S031** A SINGLE CENTER EXPERIENCE OF 129 Pancreatic Enucleations: Indications, Short and Long-Term Outcome Sebastien Gaujoux, Francois Faitot, Safi Dokmak, Benjamin Blanc, David Fuks, Philippe Ruszniewski, Jacques Belghiti, Alain Sauvanet Department of HPB Surgery – PMAD - Hopital Beaujon - AP-HP - Clichy, France (SHORT)

11:00am – 12:00pm **Professor Rounds with Posters**

(see page 15 for list of posters)
MODERATORS: Horacio Asbun, MD & Christopher Wolfgang, MD

Posters P071-P095: Authors will be by their posters to discuss their research poster presentations. Abstracts of note are identified in the program will be part of the Poster-side Professor Rounds. Each invited Professor will discuss several posters. Posters of distinctions will be identified on the poster board with a gold star.

12:00pm – 1:00pm **Lunch**

1:00pm – 1:45pm **How I Do It Session:**

**Surgical Management of Chronic Pancreatitis**
MODERATORS: William Traverso and David Adams

- Classic approach: the Gold Standard – Indications and Outcomes — *William Nealon, Nashville, TN*
- Role of Total Pancreatectomy – Indications and Outcomes Birmingham Experience — *John Christein, Birmingham, AL*
- Role of Total Pancreatectomy – Indications and Outcomes Minnesota Experience — *Gregory Beilman, Minneapolis, MN*

The purpose of the How I Do It Session is to examine the efficacy of the surgical treatment of chronic pancreatitis particularly with the advent of newer procedures such as total pancreatectomy followed by islet cell transplantation.

1:45pm – 3:35pm **Scientific Session V: Pancreatitis**

MODERATOR: David Adams, MD

1:45pm – **S032** AGING IS RELATED TO INCREASED INTESTINAL DAMAGE AND BACTERIAL TRANSLLOCATION IN ACUTE PANCREATITIS IN RATS Ana Maria M Coelho, PhD, Marcel C Machado, MD, PhD, Sandra N Sampietre, Nilza T Molan, Inneke M Heijden, PhD, Jose Eduardo M Cunha, MD, PhD, Luiz Carneiro D’Albuquerque, MD, PhD Department of Gastroenterology, University of Sao Paulo, Sao Paulo, Brazil (LONG)
2:00pm – **S033** **PANCRETIPO-JEJUNOSTOMY LIMITED TO THE BODY AND TAIL OF THE PANCREAS; A PROCEDURE PERFORMED WITH GROWING FREQUENCY AS A RESULT OF TWO EMERGING PATIENT POPULATIONS** William H Nealon, MD, Nipun B Merchant, MD, Alexander A Parikh, MD, Christopher D Lind, MD, Patrick Yachimsky, MD Vanderbilt University Medical Center  (LONG)

2:15pm – **S034** **PERCUTANEOUS DRAINAGE OF PANCREATIC NECROSIS-BEYOND THE PANTER TRIAL** Gregory S Flint, Cody J Boyce, MD, John C Kirkham, MD, Sean M Carr, MD, Brent D Nelson, MD, Don A Bell, MD, R. Taylor Handley, MD, Stephen M Schutz, MD, Joshua G Barton, MD, L. William Traverso, MD St. Luke’s Health System  (LONG)

2:30pm – **S035** **EVOLVING TREATMENT STRATEGIES IN THE ENDOSCOPIC MANAGEMENT OF WALLED-OFF PANCREATIC NECROSIS (WOPN)** Ji Young Bang, MBBS, MPH, Muhammad Hasan*, MD, Jayapal Ramesh^, MD, Jessica Trevino^, MD, C. Mel Wilcox^, MD, Robert Hawes*, MD, Shyam Varadarajulu*, MD Center for Interventional Endoscopy, Florida Hospital, Orlando, FL, USA*; Division of Gastroenterology-Hepatology, University of Alabama at Birmingham, Birmingham, AL, USA^  (LONG)

2:45pm – **S036** **SALVAGE DUAL MODALITY DRAINAGE FOR PERSISTENT WALLED OFF PANCREATIC NECROSIS ELIMINATES EXTERNAL PANCREATIC FISTULAE BUT DOES NOT REDUCE LENGTH OF HOSPITALIZATION NOR USE OF RADIOLOGIC RESOURCES** Michael Gluck, MD, Flavio G Rocha, MD, Andrew R Ross, MD, Shayan Irani, MD, Seng I Gan, MD, Richard A Kozarek, MD Virginia Mason Medical Center  (SHORT)

2:50pm – **S037** **DOES ACUTE PANCREATITIS CHANGE THE NATURAL HISTORY OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN)?** Sejoon Lee*, MD, Joshua A Waters, MD, C M Schmidt, MD, Henry A Pitt, MD, Nicholas J Zyromski, MD Department of Gastroenterology, Yonsei University College of Medicine, Seoul, Korea* and Department of Surgery, Indiana University School of Medicine, Indianapolis, IN  (LONG)

3:05pm – **S038** **DOES WEIGHT AFFECT OUTCOMES FOLLOWING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION?** Stefanie Owczarski, PAC, MPAS, Katherine Morgan, MD, FACS, David Adams, MD, FACS, Kelley Martin, MPH, RD, LD, Hongjun Wang, PHD, Jeffrey Borckardt, PHD, Alok Madan, PHD, Joseph Romagnuolo, MD, MSC, FRCP Medical University of South Carolina  (SHORT)

3:10pm – **S039** **MULTIVARIABLE LOGISTIC REGRESSION ANALYSIS OF ALCOHOL CONSUMPTION, CIGARETTE SMOKING AND PANCREAS DIVISUM IN THE RISK OF RECURRENT ACUTE AND CHRONIC PANCREATITIS** Giulia Martina Cavestro, MD, PhD, Elisabetta Goni, MD, Raffaella Alessia Zuppardo, MD, PhD, Paolo Giorgio Arcidiacono, MD, Silvia Carrara, MD, Alberto Mariani, MD, Maria Chiara Petrone, MD, Gioacchino Leandro, MD, Pier Alberto Testoni, MD Vita-Salute San Raffaele University, San Raffaele Scientific Institute, Milan  (SHORT)

3:15pm – **S040** **DOES RESIDENT EXPERIENCE AFFECT OUTCOMES IN COMPLEX ABDOMINAL SURGERY?** Daniel Relles, MD, Richard A Burkhart, MD, Jocelyn Sendecky, MS, Michael Pucci, MD, Renee Tholey, MD, Ross Drueing, BS, Patricia K Sauter, CRNP, Eugene P Kennedy, MD, Jordan M Winter, MD, Harish Lavu, MD, Charles J Yeo, MD Thomas Jefferson University  (LONG)
3:30pm – S041 POSITRON EMISSION TOMOGRAPHY (PET) HAS LIMITED UTILITY IN PREOPERATIVE STAGING OF PANCREATIC ADENOCARCINOMA
Peter Einersen, BA, Irene Epelboym, MD, Megan Winner, MD, David Leung, MD, John A Chabot, MD, John D Allendorf, MD Columbia University Medical Center  (LONG)

3:45pm – S042 THE VALUE OF (18)FDG-PET/CT IN PATIENTS WITH RESECTABLE PanCREATIC CANCER: A PROSPECTIVE STUDY  
Stefano Crippa, MD, Matteo Salgarello, MD, Silvia Laiti, MD, Stefano Partelli, MD, Giuliano Barugola, MD, Paola Castelli, MD, Giuseppe Zamboni, MD, Massimo Falconi, MD Departments of Surgery, Universita’ Politecnica delle Marche, Ancona and Ospedale Sacro Cuore Negrar, Italy and Departments of Nuclear Medicine and of Pathology, Ospedale Sacro Cuore Negrar, Italy  (SHORT)

3:50pm – S043 A STANDARDIZED REPORTING SYSTEM FOR EUS/FNA CYTOPATHOLOGY OF SOLID PanCREATIC MASSES  
Giuseppe Perrone*, MD, Domenico Borzomati**, MD, PhD, Francesco Di Matteo^, MD, Chiara Brunelli^^, MD, Francesco Panzera^, MD, Gennaro Nappo**, MD, Andrea Onetti Muda*, MD, Roberto Coppola**, MD Units of Pathology, General Surgery and Digestive Endoscopy, Campus Bio-Medico University of Rome, Rome; Department of Pathology, Catholic University of Rome, Rome  (LONG)

4:05pm – S044 A LOW LYMPH NODE RATIO IS ASSOCIATED WITH IMPROVED SURVIVAL, DECREASED RECURRENCE AND POSTOPERATIVE CHEMOTHERAPY BENEFIT AFTER NEOADJUVANT CHEMOREXITATION FOR PanCREATIC DUCTAL ADENOCARCINOMA  
Christina L Roland, MD, Ching-Wei D Tzeng, MD, Matthew H Katz, MD, Anthony D Yang, MD, Heathere Lin, PhD, Jean-Nicolas Vauthey, MD, Peter W Pisters, MD, Robert A Wolff, MD, Christopher H Crane, MD, Jeffrey E Lee, MD, Jason B Fleming, MD The University of Texas MD Anderson Cancer Center, Houston, Texas  (LONG)

4:20pm – S045 LocALLY ADVANCED PanCREATIC CANCER: PROLONGED PREOPERATIVE TREATMENT IS ASSOCIATED WITH LYMPH NODE NEGATIVITY AND EXCELLENT OVERALL SURVIVAL  
Brian E Kadera, MD, Dharma Sunjaya, BS, William Isacoff, MD, Luyi Li, MS, Oscar J Hines, MD, James Tomlinson, MD, PhD, David Dawson, MD, PhD, Matthew Rochefort, MD, Graham Donald, MD, James Farrell, MD, Barbara Clerkin, RN, MPH, Howard Reber, MD, T University of California, Los Angeles  (LONG)

4:35pm – S046 FREQUENCY AND INTENSITY OF POSTOPERATIVE SURVEILLANCE AFTER CURATIVE TREATMENT OF PanCREATIC CANCER: A COST-EFFECTIVENESS ANALYSIS  
Daniel E Abbott, MD, Ching-Wei D Tzeng, MD, Scott B Cantor, PhD, Jason B Fleming, MD, Jeffrey E Lee, MD, Peter W Pisters, MD, Gauri R Varadhachary, MD, James L Abbruzzese, MD, Robert A Wolff, MD, Syed A Ahmad, MD, Matthew H Katz, MD University of Cincinnati, The University of Texas MD Anderson Cancer Center  (LONG)
4:50pm – S047 TREATMENT SEQUENCING FOR RESECTABLE PANCREATIC CANCER: INFLUENCE OF EARLY METASTASES AND SURGICAL COMPLICATIONS ON MULTIMODALITY THERAPY COMPLETION RATES AND SURVIVAL
Ching-Wei D Tzeng, MD, Daniel E Abbott, MD, Jeffrey D Lee, MD, Peter W Pisters, MD, Jason B Fleming, MD, Jean-Nicolas Vauthey, MD, Matthew H Katz, MD The University of Texas MD Anderson Cancer Center; University of Cincinnati (SHORT)

4:55pm – S048 A COMPARATIVE ANALYSIS OF PLASTIC VERSUS METAL ENDOSCOPIC BILIARY STENTS IN BORDERLINE RESECTABLE PANCREATIC CANCER PATIENTS UNDERGOING EXTENDED NEOADJUVANT CHEMOTHERAPY
R E Heneghan, MD, J B Rose, MD, A Alseidi, T R Biehl, MD, R Moonka, MD, F Rocha, MD, S I Gan, MD, M Gluck, MD, S Irani, MD, V Piccozzi, MD, R A Kozarek, MD, S Helton, MD Virginia Mason Medical Center, Seattle, WA, United States (SHORT)

5:00pm – S049 EXTENDED NEOADJUVANT CHEMOTHERAPY FOR LOCALLY ADVANCED, RESECTABLE PANCREATIC CANCER DEMONSTRATES PROMISING POSTOPERATIVE OUTCOMES AND SURVIVAL
J B Rose, F Rocha, A Alseidi, T Biehl, R Moonka, J Ryan, B Lin, V Piccozzi, S Helton Virginia Mason Medical Center (SHORT)

5:05pm – S050 GEMCITABINE-BASED CHEMORADIOThERAPY FOLLOWED BY SURGERY FOR RESECTABLE, BORDERLINE RESECTABLE AND LOCALLY UNRESECTABLE PANCREATIC ADENOCARCINOMA
Masashi Kishiwada, MD, PhD, Motoyuki Kobayashi, MD, Akihiro Tanemura, MD, PhD, Naohisa Kuriyama, MD, PhD, Yoshinori Azumi, MD, PhD, Ichiro Osawa, MD, PhD, Shugo Mizuno, MD, PhD, Masanobu Usui, MD, PhD, Hiroyuki Sakurai, MD, PhD, Masami Tabata, MD, Hepatobiliary Pancreatic and Transplant Surgery, Mie University School of Medicine (SHORT)

5:10pm – S051 RESECTION OF LOCALLY ADVANCED PANCREATIC CANCER AFTER NEOADJUVANT CHEMOTHERAPY WITH MODIFIED FOLFIRINOX: A PROSPECTIVE PHASE II STUDY
Enrico Vasile, MD, Nelide De Lio, MD, Mario Antonio Belluomini, MD, Francesca Costa, MD, Carla Cappelli, MD, Daniela Campani, Alfredo Falcone, Ugo Boggi, FACS Division of General and Transplant Surgery, University of Pisa, Pisa, Italy 1. Division of Oncology, University of Pisa, Pisa, Italy 2. Division of Radiology, University of Pisa, Pisa, Italy 3. Division of Pathology, University of Pisa, Pisa, Italy (SHORT)

5:25pm – S052 IMPACT OF MARGIN CLEARANCE ON SURVIVAL AFTER PANCREATICODUODENECTOMY FOR PANCREATIC DUCTAL ADENOCARCINOMA
Yasushi Hashimoto, MD, Yoshiaki Murakami, MD, Kenichiro Uemura, MD, Takeshi Sudo, MD, Naru Kondo, MD, Hayato Sasaki, MD, Taijiro Sueda, MD Department of Surgery, Applied Life Sciences Institute of Biomedical (SHORT)

5:30pm – 6:00pm Pancreas Club Brief Business Meeting
Southern IV-V

7:00pm – 10:00pm Pancreas Club Annual Dinner and Reception
Reception in Southern III Foyer followed by dinner in Southern III

Award Presentations
Presentation of two $1,000 resident/fellow awards
Poster Listing

All posters located in Southern II. The ★ symbol indicates Poster of Distinction. Complete Poster Abstract descriptions are available online at www.pancreasclub.com.

★ P001 HOSPITAL AND MEDICAL CARE DAYS IN PANCREATIC CANCER
Casey B Duncan, MD MS, Daniel W Branch, MS, Kristin M Sheffield, PhD, Yimei Han, MS, Yong-Fang Kuo, PhD, James S Goodwin, MD, Taylor S Riall, MD PhD; University of Texas-Medical Branch

★ P002 CLINICO-PATHOLOGICAL FEATURES AND SURGICAL MANAGEMENT OF SOLID PSEUDOPAPILLARY NEOPLASMS OF THE PANCREAS
Pablo E Serrano, Hassan Al-Ali, Steve Gallinger, Ian D McGilvray, Carol-anne Moulton, Alice C Wei, Stefano Serra, Sean Cleary; University of Toronto

★ P003 INTRAOPERATIVE PANCREATOSCOPY: A VALUABLE TOOL FOR PANCREATIC SURGEONS
Michael J Pucci, MD, Caitlyn Johnson, MD, Kelly Lopez, Jordan M Winter, MD, Harish Lavu, MD, Charles J Yeo, MD; Jefferson Pancreas, Biliary, and Related Cancer Center at Thomas Jefferson University Hospital

★ P004 THE RISK FACTOR OF PANCREATIC FISTULA FOLLOWING STUMP CLOSURE USING STAPLER CLOSURE DURING DISTAL PANCREATECTOMY
Manabu Kawai, MD PhD, Masaji Tani, MD PhD, Ken-ichi Okada, MD PhD, Seiko Hirono, MD PhD, Motoki Miyazawa, Astusi Shimizu, MD PhD, Yuji Kitahata, MD, Hiroki Yamaue, MD PhD; The Second Department of Surgery, Wakayama Medical University

★ P005 CLINICOPATHOLOGIC FEATURES INFLUENCING SURVIVAL IN PATIENTS UNDERGOING PANCREATICODUODENECTOMY FOR PANCREATIC ADENOCARCINOMA
Cynthia Weber, MD, Eileen Bock, MD, Michael Hurtuk, MD, Gerard Abood, MD, Margo Shoup, MD, Gerard Aranha, MD; Loyola University Medical Center, Central DuPage Hospital

★ P006 FIRST JEJUNAL VEIN-ORIENTED MESENTERIC EXCISION DECREASES BLEEDING DURING PANCREATODUODENECTOMY
Masafumi Nakamura, MD PhD, Kosuke Tsutsumi, MD PhD, Hiroshi Nakashima, MD PhD; Kawasaki Medical College

★ P007 QUALITY-OF-LIFE FOR PANCREATIC CANCER PATIENTS BEFORE DIAGNOSIS: A POPULATION-BASED STUDY
Clancy J Clark, MD, Victor Zaydfudim, MD MPH, Scott Harmsen, MS, Kaye M Reid Lombardo, MD; Wake Forest Baptist Health; Mayo Clinic
★ P008 PERIOPERATIVE OUTCOME AFTER PANCREATIC HEAD RESECTIONS: CONSECUTIVE SINGLE SURGEON SERIES IN A SPECIALIZED UNIVERSITY HOSPITAL AND IN A COMMUNITY HOSPITAL Ulrich Adam, MD, Hartwig Riediger, MD, Tobias Keck, MD, Ulrich T Hopt, MD, Frank Makowiec, MD; Dept. of Surgery, Vivantes-Humboldtstabinum, Berlin, Germany and Dept. of Surgery, University of Freiburg, Freiburg, Germany

★ P009 THE ROLE OF ADJUVANT CHEMORADIOThERAPY IN PANCREATOBILIARY VERSUS INTESTINAL SUBTYPES OF AMPULLARY CANCERS Sanjay S Reddy, MD, Harry S Cooper, MD, Karen J Ruth, MS, James C Watson, MD, Yun Shin Chun, MD, John P Hoffman, MD; Fox Chase Cancer Center

★ P010 MORPHO-HISTOLOGICAL FEATURES OF PANCREATIC STUMP PREDICT POSTOPERATIVE PANCREATIC FISTULA AFTER PANCREATOCODUODENECTOMY Alessandro Zerbi, MD, Francesca Gavazzi, MD, Maria Rachele Angiolini, MD, Cristina Ridolfi, MD, Marco Madonini, MD, Paola Spaggiari*, MD, Marco Montorsi, MD; Section of Pancreatic Surgery, General Surgery Department; *Pathology Department; Humanitas Clinical Institute - Rozzano (Milan)

★ P011 TRANSGASTROMURAL INTERVENTIONAL ENDOSCOPIC THERAPY OF POSTOPERATIVE COMPLICATIONS AFTER PANCREATIC RESECTION Björn Dahl, Peter Troschel, Dietfried Scholz-Brand, Martin Reuther, Hans Seifert; Klinikum Oldenburg

★ P012 UTILITY OF PRETREATMENT SERUM CARCINOEMBRYONIC ANTIGEN (CEA) LEVEL IN PATIENTS WITH LOCALIZED PANCREATIC CANCER (LPCA) Ben George, MD, Paul S Ritch, MD, James P Thomas, MD PhD, Lauren A Wiebe, MD, Anna Mahmoud, Kathleen K Christians, MD, Sam G Pappas, MD, Kiran Turaga, MD, Edward J Quebbeman, MD PhD, Thomas C Gamblin, MD, Beth A Erickson-Wittmann, MD, Tracy R; Medical College of Wisconsin

★ P013 RAISED INTESTINAL FATTY ACID BINDING PROTEIN AND CLINICAL HYPOVOLUMIA EARLY IN SEVERE ACUTE PANCREATITIS Hannes Hartman, MD, Tomi Sippola, MD, Juozas Kupcinskas, MD, Outi Lindström, MD, Colin D Johnson, MS, Sara Regner, PhD; Lund University, Sweden. Tampere University Hospital,, Finland. Seinäjoki Central Hospital, Seinäjoki, Finland. LSMU, Kaunas, Lithuania. Helsinki University Central Hospital, Finland. University Hospital Southampton UK
P014 Diagnostic Accuracy of Contrast-Enhanced Computed Tomography in Assessing Extra-Regional Lymphadenopathy in Pancreatic and Peri-Ampullary Cancer: A Systematic Review

Dorine S Tseng, MD, Hjalmar C van Santvoort, MD PhD, Samira Fegrachi, MD, Marc G Besselink, MD PhD, Maarten S van Leeuwen, MD PhD, Quintus I Molenaar, MD PhD; University Medical Center Utrecht; Academic Medical Center Amsterdam

P015 The Role of Preoperative EUS in Addition to CT in Patients Suspected of Pancreatic or Peri-Ampullary Cancer

Kasia P Cieslak^, MD, Hjalmar C van Santvoort*, MD PhD, Frank P Vleggaar^, MD PhD, Maarten S van Leeuwen^, MD PhD, Fibon J ten Kate**, MD PhD, Marc G Besselink*, MD PhD, I Quitus Molenaar*, MD PhD; *Department of Surgery, ^Department of Gastroenterology and Hepatology, **Department of Pathology, University Medical Center Utrecht, The Netherlands

P016 Prognostic Significance and Functional Relevance of HERG1 Potassium Channel Expression in Pancreatic Ductal Adenocarcinoma

Annarosa Arcangeli*, MD, Giuseppe Perrone**, Elena Lastraioli*, Olivia Crociani*, Angelica Sette*, Sagar Manoli^, Domenico Borzomati^, Gennaro Nappo^, Marcella Callea**, Francesco Di Costanzo#, Andrea Onetti Muda**, Roberto Coppola^; *Experimental and Clinical Medicine, University of Florence, Italy; ^IonTraC-Marie Curie fellow; #Medical Oncology, Azienda Ospedaliero-Universitaria Careggi, Florence, Italy; Units of **Pathology and ^General Surgery, Campus Bio-Medico University

P017 Correlation of Computed Tomography with Histopathology in T4 Pancreatic Cancer: Prognostic Implications

Carla Cappelli, PhD MD, Ugo Boggi, FEBS MD, Rosa Cervelli, MD, Salvatore Mazzeo, MD, Niccolà Funel, PhD, Luca Emanuele Pollina, MD, Daniela Campani, MD, Nelide De Lio, MD, Vittorio Grazio Perrone, MD, Fabio Caniglia, MD, Carlo Bartolozzi, MD; Diagnostic and Interventional Radiology, University of Pisa

P018 Usefulness of Early Prediction for Local and Systemic Complications of Severe Acute Pancreatitis Using Perfusion CT

Yoshihisa Tsuji, MD, Yuzo Kodama, MD, Tsutomu Chiba; Department of Gastroenterology and Hepatology, Kyoto University Hospital

P019 Pancreatectomy with Major Arterial Resection

Nelide De Lio, MD, Mario Antonio Belluomini, MD, Francesca Costa, MD, Stefano Signori, MD, Fabio Vistoli, MD, Franco Mosca, Ugo Boggi, FACS; Division of General and Transplant Surgery, University of Pisa, Pisa - Italy 1. Division of General Surgery 1, University of Pisa, Pisa - Italy
P020 METFORMIN DOES NOT INCREASE SURVIVAL FOR PATIENTS WITH DIABETES AND RESECTABLE PANCREATIC ADENOCARCINOMA
Paul G Toomey, MD, Sharona B Ross, MD, Ashley Joseph, Anthony Teta, BS, Harold Paul, MS, Kenneth Luberice, BS, Kimmerle Cohen, MD, Alexander S Rosemurgy, MD; Florida Hospital Tampa

P021 CLINICAL MONITORING OF FLUOROPYRIMIDINE ACTIVITY IN PANCRERATICODUODENECTOMY SPECIMENS: PROOF OF PRINCIPLE
Kalpesh Patel, Christine A Iacobuzio-Donahue, Paul Gormley, Scott E Kern, Steven C Cunningham; Johns Hopkins and Saint Agnes Hospitals

P022 PRECISE MEASURES OF VISCERAL FAT EMPHASIZE THE ROLE OF VISCERAL FAT IN THE BIOLOGY OF PANCREATIC CANCER
Whalen Clark, MD, Sharona B Ross, MD, Ty A Bowman, BS, Julia Francoeur, BS, Kenneth Luberice, BS, Charles Tkatch, BS, Alexander S Rosemurgy, MD; Florida Hospital Tampa

P023 SERIAL IN VIVO PASSAGING OF HUMAN PANCREATIC CANCER WITH WILD TYPE OR MUTANT KRAS IN NUDE MICE RESULTS IN GREATER METASTATIC DISEASE
Cristina A Metildi, MD, Sharmeela Kaushal, PhD, Robert M Hoffman, PhD, Michael Bouvet, MD; University of California San Diego and AntiCancer, Inc.

P024 COVERED SELF-EXPANDABLE METAL STENT DEPLOYMENT PROMISES SAFE NEOADJUVANT CHEMORADIATION THERAPY IN PATIENTS WITH BORDERLINE RESECTABLE PANCREATIC HEAD CANCER
Jun Arimoto, Takamitsu Sato, Seitaro Watanabe, Shin Maeda, Atsushi Nakajima, Kensuke Kubota; Yokohama City University Hospital

P025 EARLY OUTCOMES FOR A PROSPECTIVE TRIAL OF PROTON THERAPY AND CONCOMITANT CAPECITABINE FOR PATIENTS WITH NON-METASTATIC UNRESECTABLE PANCREATIC ADENOCARCINOMA
Romaine C Nichols, MD, Christopher G Morris, MS, Thomas J George, MD, Robert A Zaiden, MD, Horacio J Asbun, MD, Ziad T Awad, MD, Meng Wei Ho, MSc, Soon Huh, PhD, Nancy P Mendenhall, MD, Bradford S Hoppe, MD; University of Florida Proton Therapy Institute, Jacksonville, FL; Departments of Medical Oncology and Surgery, University of Florida, Gainesville and Jacksonville, FL; Department of Surgery, Mayo Clinic, Jacksonville, FL

P026 LAPAROSCOPIC WHIPPLE: FEASIBILITY AND OUTCOMES
Martin A Makary, MD MPH, Heather G Lyu, BA, Michol A Cooper, MD PhD, Neda Rezaee, BS, John L Cameron, MD, Barish H Edil, MD; Johns Hopkins University School of Medicine, Johns Hopkins University School of Public Health
P027 UNIVERSAL MODERN METHOD OF SURGICAL TREATMENT FOR CHRONIC PANCREATITIS Andrii V Klymenko, MD, Volodymyr N Klymenko, MD, Andrii A Steshenko, MD, Valerii A Tumansky, MD; Surgery Faculty, Zaporizhzhya State Medical University, Ukraine

P028 LAPAROSCOPIC SURGERY FOR MUCINOUS CYSTIC NEOPLASM (MCN) OF THE PANCREAS Takao Ohtsuka, Shunichi Takahata, Junji Ueda, Kazuhiro Mizumoto, Shuji Shimizu, Masao Tanaka; Kyushu University

P029 PREDICTORS OF RECURRENT AND POST RECURRENT SURVIVAL IN PATIENTS WITH RESEDCTED AMPULLARY ADENOCARCINOMA Irene Epelboym, MD, Susan J Hsiao, MD, James A Lee, MD, Beth A Schroepe, MD PHD, John A Chabot, MD, Helen Remotti, MD, John A Allendorf, MD; Columbia University Medical Center

P030 THE INCIDENCE OF PANCREATIC FISTULA COULD BE PREDICTABLE ON POD4 AFTER PANCREATODUODENECTOMY Hisashi Kosaka, Nobukazu Kuroda, Kazuhiro Suzumura, Yasukane Asano, Toshihiro Okada, Tadamichi Hirano, Yuji Iimuro, Jiro Fujimoto; Hyogo College of Medicine

P031 PRESERVING A LEFT GASTRIC ARTERY REDUCED THE INCIDENCE OF DELAYED GASTRIC EMPTYING IN DISTAL PANCREATECTOMY WITH CELIAC AXIS EN-BLOC RESECTION Ken-ichi Okada, MD, Masaji Tani, MD, Manabu Kawai, MD, Seiko Hirono, MD, Motoki Miyazawa, MD, Atsushi Shimizu, MD, Yuji Kitahata, MD, Masaki Ueno, MD, Shinya Hayami, MD; Second Department of Surgery, Wakayama Medical University

P032 LOW FISTULA RATE WITH HAND-SEWN CLOSURE TECHNIQUE AFTER DISTAL PANCREATECTOMY AND ANALYSIS OF RISKFACTORS FOR PANCREATIC FISTULA Marius Distler, MD, Stephan Kersting, MD, Felix Rueckert, MD, Hans-Detlev Saege, MD, Robert Gruetzmann, MD; Department of General, Thoracic and Vascular Surgery, University Hospital Carl Gustav Carus, TU Dresden, Germany

P033 PHASE I/II TRIAL OF AUTOPHAGY INHIBITION IN COMBINATION WITH NEOADJUVANT GEMCITABINE IN HIGH RISK PANCREATIC ADENOCARCINOMA: SAFETY AND RESPONSE TO TREATMENT Brian A Boone, MD, Amer Zureikat, MD, Nathan Bahary, MD, David Bartlett, MD, Ravi Amaravadi, MD, Michael T Lotze, MD, Herbert J Zeh, MD; University of Pittsburgh Medical Center, Pittsburgh, Pa; University of Pennsylvania, Philadelphia, Pa

P034 PALLIATIVE REOPERATION FOR RECURRENT PERIAMPUPLARY ADENOCARCINOMA: PRIMUM NON NOCERE? Brian A Boone, MD, A. James Moser, MD, Paul J Johnson, MD, Brady K Mock, MD, Igor Dvorchik, Herbert J Zeh, MD, J. Wallis Marsh, MD; University of Pittsburgh Medical Center, Pittsburgh, Pa
P035 A TWO-PHASE STRATEGY FOR LONG-TERM IN VITRO MAINTENANCE OF FUNCTIONALLY COMPETENT HUMAN PANCREATIC ACINAR CELLS Merja Bläuer, PhD, Juhani Sand, MD PhD, Isto Nordback, MD PhD, Johanna Laukkaninen, MD PhD; Tampere Pancreas Laboratory and Department of Gastroenterology and Alimentary Tract Surgery, Tampere University Hospital, Tampere, Finland.

P036 A CD166 NEGATIVE SUBPOPULATION OF PANCREATIC CANCER CELLS HAS STRONG INVASIVE AND MIGRATORY ACTIVITY Kenji Fujiwara, MD, Kenoki Ohuchida, MD PhD, Koji Shindo, MD, Daiki Eguchi, MD, Shingo Kozono, MD, Takao Ohtsuka, MD PhD, Shunichi Takahata, MD PhD, Shinichi Aishima, MD PhD, Kazuhiro Mizumoto, MD PhD, Masao Tanaka, MD PhD; Departments of Surgery and Oncology and Department of Anatomic Pathology, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.

P037 LAPAROSCOPIC TRANSGASTRIC NECROSECTOMY IS A SAFE AND EFFECTIVE APPROACH FOR SELECT PATIENTS WITH NECROTIZING PANCREATITIS David J Worhunsky, MD, Motaz Qadan, MD PhD, George A Poultsides, MD MS, Walter G Park, MD, Jeffrey A Norton, MD, Brendan C Visser, MD; Stanford University Medical Center

P038 PROGNOSTIC FACTORS FOR PATIENTS WITH BORDERLINE RESECTABLE Pancreatic Cancer AFTER NEOADJUVANT CHEMOTHERAPY Toshihiko Masui, MD PhD, Ryuichiro Doi, MD PhD, Yoshiya Kawaguchi, MD PhD, Masaki Mizumoto, MD PhD, Yasuhiro Iwanaga, MD PhD, Michiya Kawaguchi, MD PhD, Kyoichi Takaori, MD PhD, Shinji Uemoto, MD PhD; Kyoto University, Department of Surgery


P040 SPLEEN PRESERVING LAPAROSCOPIC DISTAL PanCREATECTOMY Heather G Lyu, BA, Michol A Cooper, MD PhD, Barish H Edil, MD, Neda Rezaee, BS, Christopher L Wolfgang, MD PhD, John L Cameron, MD, Martin A Makary, MD MPH; Johns Hopkins University School of Medicine, Johns Hopkins University School of Public Health

P041 ENDOSCOPIC AND SURGICAL ALTERNATIVES TO Pancreaticoduodenectomy and Distal Pancreatectomy K Plichta, MD MS, E A Bock, MD, M G Hurtuk, MD, G J Abood, MD MS, G V Aranha, MD; Department of Surgery, Loyola University Medical Center, Maywood, Illinois
P042 IMPACT OF MEDIATED DECISION SUPPORT ON PARTICIPATION IN A PANCREAS CANCER REGISTRY Harish Lavu, MD, Scott Keith, PhD, Heidi Swan, MS, Nadine O’Rourke, James Cocroft, MA, Charles J Yeo, MD, Vishnu Potluri, MD, Ronald Myers, PhD; Thomas Jefferson University, University of Pennsylvania

P043 FIRST YEAR RESULTS FROM A FELLOWSHIP TRAINED PANCREATIC SURGEON John A Stauffer, MD, Justin H Nguyen, MD, J. Kirk Martin, MD, Horacio J Asbun; Mayo Clinic Florida

P044 ROBOTIC ASSISTED SURGERY FOR PANCREATIC NEUROENDOCRINE TUMORS Melissa E Hogg, MD, Mustapha Daoaudi, MD, Brian A Boone, MD, Haroon M Choudry, MD, Kenneth K Lee, MD, Wallis Marsh, MD, James F Pingpank, MD, Michael T Stang, MD, Allan Tsung, MD, A J Moser, MD, David L Bartlett, MD, Herbert J Zeh, MD, Amer H; UPMC

P045 USE OF THE DA VINCI ROBOT TO REMOVE PRE-MALIGNANT PANCREATIC LESIONS Melissa E Hogg, MD, Mustapha Daoaudi, MD, Brian A Boone, MD, Haroon M Choudry, MD, Kenneth K Lee, MD, Allan Tsung, MD, A J Moser, MD, David L Bartlett, MD, Herbert J Zeh, MD, Amer H Zureikat, MD; UPMC

P046 RETROSPECTIVE ANALYSIS OF PROGNOSTIC FACTORS IN PATIENTS WITH PANCREATIC CANCER AND INDICATION OF GEMCITABINE-BASED NEOADJUVANT CHEMORADIATION THERAPY WITH IMRT Masaki Mizumoto, MD, Kyoichi Takaori, MD, Toshihiko Masui, MD, Michiya Kawaguchi, MD, Yasuhiro Iwanaga, MD, Shinji Uemoto, MD; Department of Hepatobiliary-Pancreatic Surgery and Transplantation, Kyoto University Hospital

P047 IMMEDIATE AND LONG-TERM OUTCOMES OF PANCREATICOJEJUNOSTOMY USING THE PAIR-WATCH SUTURING TECHNIQUE AFTER PANCREATICODUODENECTOMY Yoshinori Azumi, MD PhD, Rie Sato, Akihiro Tanemura, Naohisa Kuriyama, Masashi Kishiwada, Ichiro Osawa, Shugo Mizuno, Masanobu Usui, Hiroyuki Sakurai, Masami Tabata, Shuji Isaji, Prof; Department of Hepatobiliary pancreatic and transplant Surgery, Mie University, Mie, Japan

P048 PANCREATIC CYST PREVALENCE AND THE RISK OF MUCIN-PRODUCING ADENOCARCINOMA IN UNITED STATES ADULTS Kerrington Smith, MD, Timothy Gardner, MD; Dartmouth-Hitchcock Medical Center

P049 REDUCTION OF SPLENIC VOLUME BY STEROID THERAPY IN CASES WITH AUTOIMMUNE PANCREATITIS Hiroyuki Matsubayashi, MD PhD, Naomi Kakushima, MD PhD, Kohei Takizawa, MD, Masaki Tanaka, MD, Kinichi Hotta, MD, Toshitatsu Takao, MD, Kenichiro Imai, MD, Yuichiro Yamaguchi, MD, Hiroyuki Ono, MD PhD; Shizuoka Cancer Center, Japan
P050 TUMOR RECURRENCE IS INDEPENDENT OF PANCREATIC FISTULA (PF) IN PATIENTS FOLLOWING PANCREATICODUODENECTOMY (PD) FOR PANCREATIC ADENOCARCINOMA

M M Assifi, MD, Sarah Zhang, BA, Ernest L Rosato, MD FACS, Harish Lavu, MD FACS, Eugene P Kennedy, MD FACS, Charles J Yeo, MD FACS, Adam C Berger, MD FACS; Department of Surgery and Jefferson Pancreas, Biliary and Related Cancer Center, Thomas Jefferson University, Philadelphia, PA 19107

P051 AN ANTEGRADE EN BLOC PANCREATICODUODENECTOMY INCLUDING MESOPANCREAS FOR BORDERLINE RESECTABLE PANCREATIC DUCTAL ADENOCARCINOMA WITH OR WITHOUT ABUTMENT OF THE SUPERIOR MESENTERIC ARTERY AND/OR HEPATIC ARTERY

Shugo Mizuno, Shuji Isaji, Masashi Kishiwada, Akihiro Tanemura, Naohisa Kuriyama, Yoshinori Azumi, Ichiro Ohsawa, Masanobu Usui, Hiroyuki Sakurai, Masami Tabata; Mie University

P052 THE EVALUATION OF SURGICAL OUTCOMES OF PANCREATEODUODENECTOMY FOR ELDERLY PATIENTS OVER 80 YEARS OLD

Hayato Sasaki, Yoshiaki Murakami, Kenichiro Uemura, Takeshi Sudo, Yasushi Hashimoto, Naru Kondo, Naoya Nakagawa, Taijiro Sueda; Department of Surgery, Institute of Biochemical

P053 PILOT STUDY EVALUATING AN ALLOGENEIC GM-CSF-TRANSUCED PANCREATIC TUMOR CELL VACCINE (GVAX) AND LOW DOSE CYCLOPHOSPHAMIDE INTEGRATED WITH FRACTIONATED STEREOTACTIC BODY RADIATION THERAPY AND FOLFIRINOX CHEMOTHERAPY FOR RESECTED PANCREATIC ADENOCARCINOMA

Joseph M Herman, MD MSc, Aaron T Wild, BA, Daniel A Laheru, MD, Avani S Dholakia, BS, Katie Y Fan, BS, Lei Zheng, MD PhD, Dung T Le, MD, Frederick Eckhauser, MD, Ross Donehower, MD, Mark Duncan, MD, Ana De Jesus-Acosta, MD, Eric Lutz, PhD, Ral; Johns Hopkins University School of Medicine

P054 ROLE OF ADDITIONAL LOCO-REGIONAL THERAPY FOR LONG-TERM CHEMO-RESPONDER BY GEMCITABINE WITH S1 FOR ADVANCED PANCREATIC CANCER

Keita Wada, MD, Keiji Sano, MD, Hodaka Amano, MD, Fumihiko Miura, MD, Naoyuki Toyota, MD, Yoshiko Aoyagi, MD, Tadahiro Takada, MD; Teikyo University School of Medicine, Tokyo, JAPAN

P055 INTRADUCTAL LOW PAPILLARY CARCINOMA (ILPC) WITHOUT MASS FORMING

Yoshihiro Nakashima, MD, Koji Yoshida, MD, Yamato Tada, MD, Toshiyasu Iwao, MD; Division of biliopancreatology, Kawasaki medical school
P056 EFFICACY OF AN ABSORBABLE FIBRIN SEALANT PATCH APPLICATION AFTER ENUCLEATION OF PANCREATIC HEAD TUMORS, SELECTIVELY COMBINED WITH PRE-OPERATIVE WIRSU NG STENTING
Alessandro Zerbi, MD, Francesca Gavazzi, MD, Cristina Ridolfi, MD, M. Rachele Angiolini, MD, Barbara Fiore, MD, Barbara Fiore, MD, M. Carla Tinti, MD, Marco Montorsi, MD; Section of Pancreatic Surgery, General Surgery Department Istituto Clinico Humanitas, Rozzano (Milan)

P057 ELECTROPORATION THERAPY IN THE MANAGEMENT OF LOCALLY ADVANCED PANCREATIC ADENOCARCINOMA AS BRIDGE TO SURGICAL RESECTION. THE FIRST CASE IN ITALY. CASE REPORT Nicola Guglielmo, Fabio Melandro, Giovanni Battista Levi Sandri, Gioacchino Maria Montalto, Pasquale Bartolomeo Berloco; sapienza university of rome

P058 DIABETES BY PATIENTS AFTER PANCREATODUODENECTOMY AND BY-PASS SURGERY DUE TO PANCREATIC ADENOCARCINOMA *Aleksandra Kolarczyk, *Mariusz Seweryn, *Weronika Bulska, ^Katarzyna Kusnierz, PhD; *Medical student of Medical University of Silesia, Katowice, Poland; ^Department of Gastrointestinal Surgery, Medical University of Silesia, Katowice, Poland

P059 COMPLETE AND NEAR COMPLETE PATHOLOGICAL RESPONSES IN PATIENTS WITH ADVANCED PANCREATIC ADENOCARCINOMA FOLLOWING CHEMOTHERAPY AND RADIATION IN SIX YOUNG, NON OBESE PATIENTS Daniel E Kleiner, MD, David C Linehan, MD, William G Hawkins, MD, Ryan C Fields, MD, Steven Strasberg, MD; Washington University in St. Louis

P060 THE ADDITION OF METFORMIN TO CHEMOTHERAPY IN PATIENTS WITH PANCREATIC CANCER AND OTHER MALIGNANCIES: A PHASE I CLINICAL TRIAL Marvin Duque, MD, Wasif Saif, MD, Robin Millis, RN BSN CCRP, John Nystrom, MD, Pamela Smith, MD PhD, Furha Cossor, MD MS, Philip Tsichlis, MD, Madhumita Das, PhD, Robert Martell, MD PhD; Division of Hematology Oncology and the Molecular Oncology Research Institute, Tufts Medical Center, Boston, MA

P061 DELAYED ARTERIAL HEMORRHAGE AFTER PANCREATICOUDUODENECTOMY Kazuhiro Suzumura, Nobukazu Kuroda, Hisashi Kosaka, Yuji Iimuro, Tadamichi Hirano, Toshihiro Okada, Yasukane Asano, Ikuo Nakamura, Yuichi Kondo, Shogo Tanaka, Seikan Hai, Yugo Uda, Hideaki Sueoka, Akito Yada, Koichiro Ohashi, Tomohiro Okamoto; Department of Surgery, Hyogo College of Medicine
P062 RIGHT TO LEFT APPROACH FOR DISTAL LAPAROSCOPIC PANCREATECTOMY. EXPERIENCE IN 70 PATIENTS
**Ricardo Jureidini, MD PhD, Telesforo Bacchella, MD PhD, Guilherme Naccache Namur, MD, Thiago Costa Ribeiro, MD, Mauricio Sorbello, MD, Ulysses Ribeiro Jr, MD PhD, Vagner Birk Jeismann, MD, Jose Eduardo Monteiro da Cunha, MD PhD, Ivan Ceconell; University of São Paulo - Instituto do Câncer do Estado de São Paulo**

P063 TOTAL LAPAROSCOPIC CENTRAL PANCREATECTOMY
**Ricardo Jureidini, MD PhD, Telesforo Bacchella, Md PhD, guilherme Naccache Namur, MD, Thiago Costa Ribeiro, MD, Mauricio Sorbello, MD, Vagner Birk Jeismann, MD, Estela Regina Ramos Figueira, MD PhD, Ulysses Ribeiro Jr, MD PhD, Jose Eduardo Mon; University of São Paulo - Instituto do Câncer do Estado de São Paulo**

P064 HIGH SELECTIVE CRITERIA FOR IMPROVMENT RESULTS IN LAPAROSCOPIC PANCREATODUODENECTOMY
**Ricardo Jureidini, MD PhD, Telesforo Bacchella, Md PhD, guilherme Naccache Namur, MD PhD, Thiago Costa Ribeiro, MD, Vagner Birk Jeismann, MD, Estela Regina Ramos Figueira, MD PhD, Ulysses Ribeiro Jr, MD PhD, Jose Eduardo Monteiro da Cunha, M; University of São Paulo - Instituto do Câncer do Estado de São Paulo**

P065 INTRAOPERATIVE ISLET ISOLATION FOR PANCREATEAS AUTOTRANSPANTATION: A NOVEL TECHNIQUE
**Michol Cooper, MD PhD, Niraj Desai, MD, Kenzo Hirose, MD, Zhao Sun, PhD, Daniel Warren, PhD, Vikes Singh, MD MSc, Rita Kalyani, MD MHS, Erica Hall, CRNP, Kate Knott, CRNP, Latif Asad, MD, Michael Shamblott, PhD, Martin Makary, MD MPH; Department of Surgery, Johns Hopkins Hospital**

P066 CENTRAL PANCREATIC RESECTION
**Vichin C Puri, MD, Vijay G Menon, MD, Alagappan A Annamalai, MD, Nicholas N Nissen, MD; Hepatobiliary and Pancreatic Surgery, Cedars-Sinai Medical Center**

P067 ADJUVANT CHEMOTHERAPY IN RESECTED DUCTAL PANCREATIC CANCER: DO GUIDELINE RECOMMENDATIONS REACH CLINICAL APPLICATION?
**Guido Alsfasser, MD, J Bochow, MS, Ernst Klar, MD, Bettina M Rau, MD; Dept of General Surgery, University of Rostock, Rostock, Germany**

P068 PRE-OPERATIVE LOVENOX DOES NOT INCREASE BLOOD LOSS DURING PANCREATICODUODENECTOMY COMPARED TO HEPARIN
**Tanaka, MD MPH, W C Conway, MD, S Jhamb, BA, A Dornelles, J S Bolton, MD; Ochsner Medical Center, New Orleans, LA**

P069 EPIDEMIOLOGY AND MANAGEMENT OF PANCREATIC CYSTIC NEOPLASMS IN CHINA: 16-YEAR DATA FROM A SINGLE CENTRE
**Xueli Bai, PhD, Longyun Ye, Qi Zhang, MD, Tingbo Liang, MD PhD; Department of Hepatobiliary and Pancreatic Surgery, The Second Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou 310009, China**
THE OPTIMAL RANGE OF DISSECTION OF THE SUPERIOR MESENTERIC ARTERY (SMA) PLEXUS FOR PANCREATIC CANCER: TAKING INTO ACCOUNT THE BRANCHING SITE OF THE INFERIOR PANCREATICODUODENAL ARTERY (IPDA) FROM THE SMA
Hiroshi Nitta, MD, Hiroshi Itoh, MD; Fukaya Red Cross Hospital, Department of Surgery

ASSESSING THE IMPACT OF FISTULAS AFTER PANCREATICODUODENECTOMY USING QUANTITATIVE SEVERITY WEIGHTING
Benjamin C Miller, BA, John D Christein, MD, Mark P Callery, MD, Stephen W Behrman, MD, Jeffrey A Drebin, MD PhD, Tara S Kent, MD, Wande B Pratt, MD MPH, Charles M Vollmer, MD; Hospital Of The University Of Pennsylvania; University Of Alabama At Birmingham School Of Medicine; Beth Israel Deaconess Medical Center; University of Tennessee Health Science Center

CLINICAL EFFICACY OF ADJUVANT SURGERY FOLLOWING SYSTEMIC TREATMENTS IN PATIENTS WITH INITIALLY UNEFFECTABLE PANCREATIC CANCER: RESULTS OF A PROJECT STUDY FOR PANCREATIC SURGERY BY THE JAPANESE SOCIETY OF HEPATO-BILIARY-PANCREATIC SURGERY?
Hiroti Yamaue, MD, Sohei Sato, MD, Kentaro Kato, MD, Shinichiro Takahashi, MD, Seiko Hirono, MD, Shin Takeda, MD, Hidetoshi Eguchi, MD, Masayuki Sho, MD, Keita Wada, MD, Hiroyuki Shinchi, MD, Satoshi Hirano, MD, A-Hon Kwon, MD, Taira Kinoshit; Second Department of Surgery, Wakayama Medical University School of Medicine, The Japanese Society of Hepato-Biliary-Pancreatic Surgery

SHORT-TERM BUT NOT LONG-TERM PATENCY OF VENOUS RECONSTRUCTION DURING PANCREATIC RESECTION PREDICTS SURVIVAL
I Gawlas, I Epelboym, M Winner, J DiNorcia, Y Woo, J L Lee, B A Schrope, J A Chabot, J D Allendorf; Department of Surgery, Columbia University Medical Center

MORBIDITY AND MORTALITY AFTER PANCREATICODUODENECTOMY IN PATIENTS WITH BORDERLINE RESECTABLE TYPE C CLINICAL CLASSIFICATION
Ching-Wei D Tzeng, MD, Matthew H Katz, MD, Jason B Fleming, MD, Holly M Holmes, MD, Jeffrey E Lee, MD, Peter W Pisters, MD, Jean-Nicolas Vauthey, MD, Gauri Varadhachary, MD, Robert A Wolff, MD, James Abbruzzese, MD, Thomas A Aloia, MD; The University of Texas MD Anderson Cancer Center

NEOADJUVANT FOLFIRINOX FOR PANCREATIC CANCER: IS THE CLINICAL REALITY WORTH THE HYPE?
Brian A Boone, MD, Jennifer Steve, MD, Nathan Bahary, MD, Amer Zureikat, MD, Herbert J Zeh, MD; University of Pittsburgh Medical Center, Pittsburgh, Pa

THE LACTATE RECEPTOR, GPR81, IS CRITICAL FOR PANCREATIC CANCER CELL SURVIVAL
Christina L Roland, MD, Thiru Arugumam, PhD, Defeng Deng, MD, Vijaya Ramachandran, PhD, Shi He Liu, MD, Zobeida Cruz-Monserrate, PhD, Craig D Logsdon, PhD; University of Texas MD Anderson Cancer Center, Houston, TX
★ P077 ACTIVATION OF Pancreatic enZYME PLUS BACTERIAL INFECTION PLAYS AN IMPORTANT ROLE IN THE PATHOGENIC MECHANISM OF CLINICALLY RELEVANT POPF AFTER Pancreaticoduodenectomy Kenichiro Uemura, MD, Yoshiaki Murakami, MD, Takashi Sudo, MD, Yasushi Hashimoto, MD, Naru Kondo, MD, Naoya Nakagawa, MD, Hayato Sasaki, MD, Kenjiro Okada, MD, Hiroki Ohge, MD, Taijiro Sueda, MD; Hiroshima University Hospital

★ P078 IMPACT OF CARDIAC COMORBIDITY ON EARLY OUTCOMES AFTER Pancreatic RESECTION Sean M Ronneklev-Kelly, MD, David Y Greenblatt, MD, Chee Paul Lin, Kaitlyn J Kelly, MD, Clifford S Cho, MD, Emily R Winslow, MD, Sharon M Weber, MD; University of Wisconsin School of Medicine and Public Health

★ P079 UNDERSTANDING HOSPITAL READMISSIONS AFTER Pancreaticoduodenectomy: CAN WE PREVENT THEM? A 10-YEAR CONTEMPORARY EXPERIENCE WITH 1173 PATIENTS AT THE MASSACHUSETTS GENERAL HOSPITAL Zhi Ven Fong, MD, Klaus Sahora, MD, Kimberly J Seefeld, Cristina R Ferrone, MD, Sarah P Thayer, MD, Andrew L Warshaw, MD, Keith D Lillemoe, MD, Matthew M Hutter, MD, Carlos Fernandez-del Castillo, MD; Massachusetts General Hospital

★ P080 INCIDENT DIAGNOSIS AS PROGNOSTIC FACTOR IN DIFFERENT TUMOR-STAGES OF NON-FUNCTIONING Pancreatic ENDOCRINE TUMORS Letizia Boninsegna, MD, Stefano Crippa, MD, Stefano Partelli, MD, Claudio Bassi, MD, Scarpa Aldo, MD, Zamboni Giuseppe, MD, Massimo Falconi, MD; Departments of Surgery, University of Verona, Ospedale Sacro Cuore Negrar and Universita’ Politecnica delle Marche and Departments of Pathology University of Verona and Ospedale Sacro Cuore Negrar, ITALY

★ P081 Radiosurgery VS Pancreaticoduodenectomy for octogenarians with Pancreatic cancer Melissa E Hogg, MD, Carolyn H Kim, MD, Brian A Boone, MD, Kenneth K Lee, MD, A J Moser, MD, David L Bartlett, MD, Dwight E Heron, MD, Steve A Burton, MD, Herbert J Zeh, MD, Amer H Zureikat; UPMC

★ P082 JAK-2 Inhibition Sensitizes Pancreatic cancer cells to Trail Induced Cell Death Vikas Dudeja, MD, Steven J Skube, BS, Amanda Oliveira, MS, Rohit Chugh, MD, Sulagna Banerjee, PhD, Veena Sangwan, PhD, Rajinder Dawra, PhD, Selwyn M Vickers, MD, Ashok K Saluja, PhD; Division of Basic and Translational Research, Department of Surgery, University of Minnesota, Minneapolis, MN, USA.
**P083** CAN THE REMNANT PANCREAS VOLUME PREDICT THE DEVELOPMENT OF NONALCOHOLIC FATTY LIVER DISEASE AFTER PANCREATICODUODENECTOMY?  
Rie Sato, Masashi Kishiwada, Takehiro Fujii, Akihiro Tanemura, Naohisa Kuriyama, Yoshinori Azumi, Ichiro Osawa, Shugo Mizuno, Masanobu Usui, Hiroyuki Sakurai, Masami Tabata, Shuji Isaji; Hepatobiliary Pancreatic and Transplant Surgery, Mie University School of Medicine, Tsu, Japan

**P084** DOES HYPERTRIGLYCERIDEMIA CAUSE MORE VIRULENT ACUTE PANCREATITIS?  
Rosalie A Fillenwarth, MS, Benjamin J Rejowski, MS, Henry A Pitt, MD, Gregory A Cote, MD, Nicholas J Zyromski, MD; Indiana University Department of Surgery

**P085** MRI-GUIDED FOCUSED ULTRASOUND MEDIATED DRUG DELIVERY AS A TREATMENT FOR PANCREATIC ADENOCARCINOMA  
Shea, PhD, A Payne, PhD, C Dillon, R Gupta, PhD, N Rapoport, PhD, C Scaife, MD; University of Utah

**P086** APTAMER-MEDIATED DELIVERY OF CHEMOTHERAPY TO Pancreatic Cancer Cells  
Partha Ray, PhD, Marcus A Cheek, PhD, Mariam L Sharaf, PhD, Bruce A Sullenger, PhD, Barbara R Shaw, PhD, Rebekah R White, MD; Duke University

**P087** IS ACS-NSQIP ORGAN SPACE INFECTION A SURROGATE FOR Pancreatic Fistula?  
Janak A Parikh, MD, Joal D Bean, MD, E Molly Kilbane, RN, Daniel P Milgrom, BS, Henry A Pitt, MD; Indiana University

**P088** TREATMENT PATTERNS AND SURVIVAL IN PATIENTS 70 AND OLDER WITH RESECTABLE Pancreatic Cancer  
Holly M Holmes, Jude K Des Bordes, David R Fogelman, Shana Palla, Nathan Parker, Jason B Fleming, Jeffrey E Lee, Peter W Pisters, Douglas B Evans, Christopher Crane, Robert A Wolff, Gauri R Varadhachary, Matthew H Katz; UT MD Anderson Cancer Center

**P089** REAPPRAISAL OF CENTRAL Pancreaectomy: A 12 YEARS SINGLE CENTER EXPERIENCE  
Sébastien Gaujoux, Yvain Goudard, Safi Dokmak, Anne Couvelard, Philippe Ruzniewski, Jacques Belghiti, Alain Sauvanet; Department of HPB Surgery – PMAD - Hopital Beaujon - AP-HP - Clichy, France

**P090** A NOVEL P21-ACTIVATED KINASE 1 INHIBITOR, GLAUCARUBINONE, COMBINED WITH GEMCITABINE SYNERGISTICALLY INHIBITS THE GROWTH OF Pancreatic Ductal Adenocarcinoma  
Dannel Yeo, BScHons, Hong He, PhD, Christopher Christophi, MD FRACS FACS, Graham Baldwin, PhD, Arthur Schulkes, PhD, Mehrdad Nikfarjam, MD PhD FRACS; University of Melbourne Department of Surgery, Austin Health, Heidelberg, Melbourne, Victoria, Australia.
★ P091 NEOADJUVANT THERAPY INCRESSES SECRETED PROTEIN ACIDIC AND RICH IN CYSTEINE (SPARC) EXPRESSION IN RESECTED PANCREATIC ADENOCARCINOMA: PROGNOSTIC IMPLICATIONS Charles Pilgrim, MD PhD, Anna Mahmoud, BS, Xiuwu Chen, PhD, Luisa Gonzalez, MD, Anna West, MD, T. C Gamblin, MD MS, Kiran Turaga, MD MPH, Kathleen Christians, MD, Edward J Quebbeman, MD, Douglas B Evans, MD, A C Mackinnon, MD PhD, Susan Tsai,; Medical College of Wisconsin

★ P092 DETECTION OF LOW-PREVALENCE MUTATIONS IN A STROMAL-RICH BACKGROUND USING NEXT GENERATION AMPLICON-SEQUENCING: EARLY STEPS TOWARD PERSONALIZED TREATMENT FOR PANCREATIC CANCER Vicente Valero III, MD, Tyler Saunders, BS, Christine A Iacobuzio-Donahue, MD PhD, Christopher L Wolfgang, MD PhD; The Johns Hopkins University School of Medicine

★ P093 LONG TERM FOLLOW-UP OF PATIENTS TREATED BY WALL STENT-ENHANCED LATERAL PANCREATICOJEJUNOSTOMY FOR SMALL DUCT CHRONIC PANCREATITIS B J Rejowski, BS, R A Fillenwarth, BS, J A Madura, MD, C Gonzales, MD, E L Fogel, MD, G A Lehman, MD, N J Zyromski; Department of Surgery and Division of Gastroenterology, Indiana University School of Medicine, Indianapolis, IN USA

★ P094 QUALITY OF LIFE IN PATIENTS WITH PANCREATIC ADENOCARCINOMA UNDERGOING NEOADJUVANT THERAPY Danielle E Green *, Charles H C Pilgrim *, MD, Kathleen K Christians *, MD, Kiran K Turaga *, MD, Susan Tsai *, MD, Lauren A Wiebe ^, MD, Douglas B Evans *, MD, Sam G Pappas *, MD; *Department of Surgery at Medical College of Wisconsin, Milwaukee, WI, USA. ^Department of Hematology and Oncology at Medical College of Wisconsin, Milwaukee, WI, USA.

★ P095 CLINICOPATHOLOGIC AND MOLECULAR CHARACTERISTICS AND BIOLOGIC BEHAVIOR OF CONCOMITANT PANCREATIC DUCTAL ADENOCARCINOMA L. Fazlollahi, MD MPH, M. Lew, MD, D. Dias-Santagata, PhD, K. Sahora, MD, V. Morales-Oyarvide, L.a. Bernardo, BS, M.b. Pitman, MD, C. Fernandez-del Castillo, MD, M. Mino-Kenudson, MD; Departments of Pathology and Surgery, Massachusetts General Hospital, Boston, USA

P096 RELATIONSHIP BETWEEN STENT CHARACTERISTICS AND TREATMENT OUTCOMES IN ENDOSCOPIC TRANSMURAL DRAINAGE OF PANCREATIC PSEUDOCYSTS Ji young Bang^, MBBS MPH, C. Mel Wilcox^, MD, Jessica Trevino^, MD, Jayapal Ramesh^, MD, Shyam Varadarajulu*, MD; Center for Interventional Endoscopy, Florida Hospital, Orlando, FL, USA*; Division of Gastroenterology-Hepatology, University of Alabama at Birmingham, Birmingham, Alabama, USA^
P097 THE ROLE OF GENETIC POLYMORPHISMS IN PATIENTS WITH SEVERE ACUTE PANCREATITIS IN THE VIEW OF BETA DEFENSINS
Gyula Farkas Jr., PhD MD, Zoltan Tiszlavicz, MD, Tamas Takacs, DSc MD, Gyorgy Lazar, PhD MD; 1 Department of Surgery, 2 Department of Medical Microbiology and Immunology, 3First Department of Internal Medicine, Faculty of Medicine, University of Szeged, Hungary

P098 THE EFFECT OF PREOPERATIVE RENAL INSUFFICIENCY ON POSTOPERATIVE OUTCOMES FOLLOWING PANCREATIC RESECTION: A SINGLE INSTITUTION EXPERIENCE OF 1061 CONSECUTIVE PATIENTS
Malcolm H Squires, MD, Vishes V Mehta, BA, Sarah B Fisher, MD, Neha L Lad, MD, David A Kooby, MD, Juan M Sarmiento, MD, Kenneth Cardona, MD, Maria C Russell, MD, Charles A Staley, MD, Shishir K Maithel, MD; Department of Surgery, Division of Surgical Oncology, Emory University

P099 DOES THE SITE OF PANCREATIC TRANSECTION INFLUENCE PANCREATIC FISTULA? A REVIEW OF 294 DISTAL PANCREATECTOMIES
Naomi M Sell, MHS, Salil Gabale, MD, Michael J Pucci, MD, Patricia K Sauter, RN MSN, Jordan M Winter, MD, Ernest L Rosato, MD, Charles J Yeo, MD, Harish Lavu, MD; Department of Surgery, Thomas Jefferson University, Philadelphia, PA.

P100 EVOLUTION OF ROBOTIC SURGICAL OUTCOMES: OVERCOMING THE LEARNING CURVE OF ROBOTIC ASSISTED PANCREATECODUODENECTOMY
Brian A Boone, MD, Mustapha Daouadi, MD, Mazen Zenati, A. James Moser, MD, Herbert J Zeh, MD, Amer Zureikat, MD; University of Pittsburgh Medical Center, Pittsburgh, PA

P101 HISTOLOGICAL DEGREE OF ISLET CELLS AT CUT MARGIN INDICATES POSTOPERATIVE GLUCOSE METABOLISM INSUFFICIENCY AFTER DISTAL PANCREATECTOMY
Masahiko Morifuji, Yoshiaki Murakami, Kenichirou Uemura, Takeshi Sudo, Yasushi Hashimoto, Taijirou Sueda, Akio Sakamoto; Internal Medicine, Sanmu Medical Center, Chiba, Japan.

P102 FINNISH BINDING PANCREATECOCOJEJUNOSTOMY AFTER PANCREATECODUODENECTOMY: A PROSPECTIVE STUDY OF 161 CONSECUTIVE PANCREATECODUODENECTOMIES
Johanna Laukkarinen, MD PhD, Isto Nordback, MD PhD, Sari Räty, MD PhD, Vilma Jormanainen, BM, Juhani Sand, MD PhD; Department of Gastroenterology and Alimentary Tract Surgery, Tampere University Hospital, Tampere, Finland.

P103 PTK6 REGULATES MIGRATION AND INVASION OF PANCREATIC CANCER CELLS WITH ERK1/2 DEPENDENT PATHWAY
Hiroaki Ono, MD PhD, Marc D Basson, MD PhD MBA, Hiromichi Ito, MD; Department of Surgery, Michigan State University
P104 SURGICAL GASTROSTOMY TO ACCESS THE BYPASSED STOMACH: SAME DAY OR DELAYED ERCP? Carlos R Gonzalez*, MD, James L Watkins^, MD, Lee McHenry^, MD, Evan L Fogel^, MD, Glen A Lehman^, MD, Nicholas J Zyromski*, MD; * Hepatobiliary Surgery, Indiana University Hospital, Indianapolis, IN, United States. ^ Gastroenterology, Indiana University Hospital, Indianapolis, IN, United States.

P105 GNAS/KRAS MUTATIONAL ANALYSES ARE USEFUL FOR DISTINCTION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM OF THE PANCREAS FROM CO-EXISTING PANCREATIC DUCTAL ADENOCARCINOMA Noboru Ideno, MD, Takao Ohtsuka, MD PhD, Koji Tamura, MD, Tepppei Aso, MD, Hiroshi Kono, MD, Yosuke Nagayoshi, MD, Yasunori Oda, MD, Shinichi Aishima, MD PhD, Tetsuhide Ito, MD PhD, Kenoki Ohuchida, Junji Ueda, MD PhD, Shunichi Takahata, Kazuhiro; Department of Surgery and Oncology, Graduate School of Medical Science, Kyushu University, Fukuoka, Japan

P106 CLINICAL SIGNIFICANCE OF PORTOMESENTERIC VEIN ABUTMENT AMONG PATIENTS WITH PANCREATIC DUCTAL ADENOCARCINOMA Victor Zaydfudim, MD MPH, Kengo Asai, MD PhD, Clancy J Clark, MD, Christina M Wood-Wentz, MS, Heather J Wiste, BA, David M Nagorney, MD, Michael B Farnell, MD, Michael L Kendrick, MD; Mayo Clinic, Rochester, MN

P107 VARIATION IN DEFINITION AND METHOD OF RETRIEVAL OF COMPLICATIONS INFLUENCE OUTCOME STATISTICS AFTER PANCREATICODUODENECTOMY Dominic E Sanford, MD, Cheryl A Woolsey, PAC, David C Linehan, MD, William G Hawkins, MD, Ryan C Fields, MD, Bruce L Hall, MD PhD MBA, Steven M Strasberg, MD; Washington University in St. Louis

P108 IMPACT OF A SINGLE-DAY MULTIDISCIPLINARY CLINIC ON THE MANAGEMENT OF PANCREATIC CANCER: 3-YEAR UPDATE Katherine Y Fan, Aaron T Wild, Avani S Dholakia, Rachit Kumar, Amol K Narang, Susannah Ellsworth, Amy Hacker-Prietz, Mary Hodgin, Dung T Le, Ana De Jesus-Acosta, Daniel A Laheru, Ralph H Hruban, Syed Ali, Lei Zheng, Elliot K Fishman, Timothy M; Johns Hopkins Hospital

P109 ELEVATED PERIOPERATIVE SERUM CA 19-9 LEVEL IS AN INDEPENDENT PREDICTOR OF POOR OUTCOME IN PATIENTS WITH RESECTABLE CHOLANGIOCARCINOMA Naru Kondo, MD, Yoshiaki Murakami, MD, Kenichiro Uemura, MD, Takeshi Sudo, MD, Yasushi Hashimoto, MD, Hayato Sasaki, MD, Kenjiro Okada, MD, Taijiro Sueda, MD; Institute of Biomedical and Health Sciences Applied Life Sciences Surgery, Hiroshima University
P110 THE VOLUME OF REMNANT PANCREAS AFTER PANCREATECTOMY IS MORE CLOSELY ASSOCIATED WITH POSTOPERATIVE PANCREATIC EXOCRINE INSUFFICIENCY Naoya Nakagawa, MD, Yoshiaki Murakami, MD, Kenichiro Uemura, MD, Takeshi Sudo, MD, Yasushi Hashimoto, MD, Masahiko Morifuji, MD, Naru Kondo, MD, Yuto Sasaki, MD, Kenjiro Okada, MD, Hiroki Ohge, MD, Taijiro Sueda, MD; Hiroshima University Hospital

P111 ADJUVANT INTRA-ARTERIAL CHEMOTHERAPY AND RADIOTHERAPY VERSUS SURGERY ALONE IN RESECTABLE PANCREATIC AND NON-PANCREATIC PERIAMPULLARY CANCER, A RANDOMISED CONTROLLED TRIAL Joris Erdmann, Marjolein Morak, Niels Kok, Casper van Eijck; Erasmus MC

P112 TRENDS IN PANCREATIC SURGERY: INDICATIONS, OPERATIVE TECHNIQUES AND POSTOPERATIVE OUTCOME OF 1120 PANCREATIC RESECTIONS Frank Makowiec, MD, Tobias Keck, MD, Ulrich Wellner, MD, Hartwig Riediger, MD, Ulrich Adam, MD, Uwe Wittel, MD, Ulrich T Hopt, MD; Department of Surgery, University of Freiburg

P113 THE DECREASING INCIDENCE OF NEGATIVE EXPLORATION IN PERIAMPULLARY AND PANCREATIC CANCER: 13-YEAR EXPERIENCE Marc G Mesleh, MD, John A Stauffer, MD, S.p. Bowers, MD, Horacio J Asbun, MD; Mayo Clinic Florida

P114 DOES TIME INTERVAL BETWEEN CHEMORADIATION AND SURGERY AFFECT OUTCOMES IN PANCREATIC CANCER? Kathryn T Chen, MD, Karthik Devarajan, PhD, John P Hoffman, MD; Fox Chase Cancer Center

P115 CYSTIC LESIONS OF THE PANCREAS – IS RADICAL SURGERY REALLY WARRANTED? U F Wellner, MD, S Geserick, D Tittelbach-Helmrich, MD, U T Hopt, MD, T Keck, MD, W K Karcz, MD, D Bausch, MD; Department of Surgery, University Hospital Schleswig-Holstein, Campus Luebeck, Luebeck, Germany; Department of General

P116 PATIENTS WITH FAMILIAL PANCREATITIS HAVE A BETTER QUALITY OF LIFE AFTER TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION Stefanie Owczarski, PAC MPAS, Katherine Morgan, MD FACS, David Adams, MD FACS, Alok Madan, PHD, Jeffrey Borckardt, PHD, Hongjun Wang, PHD; Medical University of South Carolina

P117 HOW DANGEROUS ARE SMALL MUCINOUS PANCREATIC CYSTS? Amanda B Cooper, MD, Jason B Fleming, MD, Jeffrey E Lee, MD, Wang Wei-Lien, MD, Jeffrey H Lee, MD, Brian R Weston, MD, Manoop S Bhutani, MD, William A Ross, MD MBA, Matthew H Katz, MD; The University of Texas, MD Anderson Cancer Center
P118 RE-IRRADIATION WITH STEREOTACTIC BODY RADIATION THERAPY AS A NOVEL TREATMENT OPTION FOR ISOLATED LOCAL RECURRENT OF PANCREATIC CANCER AFTER MULTIMODALITY THERAPY: EXPERIENCE FROM TWO INSTITUTIONS Aaron T Wild, BA, Susan M Hiniker, MD, Daniel T Chang, MD, Phuoc T Tran, MD PhD, Mouen A Khashab, MD, Maneesha R Limaye, BA, Daniel A Laheru, MD, Dung T Le, MD, Rachit Kumar, MD, Jonathan S Pai, BS, Blaire Hargens, MS RTT CMD, Andrew B Sharabi; Johns Hopkins University School of Medicine; Stanford University School of Medicine

P119 VOLUME/OUTCOME RELATIONSHIP IN PANCREATIC SURGERY – THE SITUATION IN GERMANY Guido Alsfasser, MD, Hanna Leicht, MD, Gerhard Schillinger, MD, Ernst Klar, MD; Dept of General Surgery, University of Rostock, Rostock, Germany and Federal Association of the AOK, Berlin, Germany

P120 CLINICAL APPLICATIONS OF CONTRAST-ENHANCED ENDOSCOPIC ULTRASOUND (CE-EUS) IN SUSPECTED PANCREATIC NEUROENDOCRINE TUMORS: A SINGLE TERTIARY MEDICAL CENTER EXPERIENCE Sabrina G Testoni, MD, Silvia Carrara, MD, Maria Chiara Petrone, MD, Giulia Martina Cavestro, MD, Alberto Mariani, MD, Pier Alberto Testoni, Prof, Paolo G Arcidiacono, MD; Division of Gastroenterology and Gastrointestinal Endoscopy, Vita-Salute San Raffaele University - Scientific Institute San Raffaele, Milan, Italy

P121 CLINICAL APPLICATIONS OF CONTRAST-ENHANCED ENDOSCOPIC ULTRASOUND (CE-EUS) IN PANCREATIC CYSTIC LESIONS: A SINGLE TERTIARY MEDICAL CENTER EXPERIENCE Sabrina Gloria G Testoni, MD, Silvia Carrara, MD, Maria Chiara Petrone, MD, Giulia Martina Cavestro, MD, Alberto Mariani, MD, Pier Alberto Testoni, Prof, Paolo G Arcidiacono, MD; Division of Gastroenterology and Gastrointestinal Endoscopy, Vita-Salute San Raffaele University - Scientific Institute San Raffaele, Milan, Italy

P122 MAB DAS-1 IS SPECIFIC FOR HIGH-RISK AND MALIGNANT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN) K.k. Das, MD, H.d. Xiao, MD PhD, X. Geng, PhD, C. Fernandez-del-Castillo, MD, V. Morales-Oyarvide, MD, D.g. Forcione, MD, B.c. Bounds, MD, W.r. Brugge, MD, M.b. Pitman, MD, K.m. Das, MD PhD, M. Mino-Kenudson, MD; Massachusetts General Hospital, Boston, MA; UMDNJ-Robert Wood Johnson Medical School, New Brunswick, NJ
P123 PATHOHISTOLOGICAL SUBTYPE PREDICTS SURVIVAL IN PATIENTS WITH INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN) OF THE PANCREAS Marius Distler, MD, Stephan Kersting, MD, Marco Niedergethmann, MD, Daniela Aust, MD, Felix Rückert, MD, Florian Ehehalt, MD, Christian Pilarsky, PhD, Stefan Post, MD, Hans-D. Saeger, MD, Robert Grützmann, MD; 1 Department of General-, Thoracic- and Vascular Surgery, University hospital Carl Gustav Carus, TU Dresden, Germany 2 Department of Surgery, University hospital Mannheim, Germany 3 Institute for Pathology, University hospital Carl Gustav Carus, TU Dresden

P124 EVALUATION OF POSSUM FOR PATIENTS UNDERGOING PANCREATODUODENECTOMY IN THREE GERMAN HIGH VOLUME CENTRES Robert Grützmann, PhD, Marius Distler, Marcus Bahra, Marco Niedergethmann, Stefan Post, Hans Detlev Saeger, Felix Rückert; 1 Department of Surgery, University Hospital Mannheim, University Heidelberg, Germany 2 Department of General, Thoracic and Vascular Surgery, University Hospital Carl Gustav Carus, Technical University Dresden, Germany 2 Department of Medical Informati

P125 NATURAL HISTORY FOLLOWING A SINGLE EPISODE OF ACUTE PANCREATITIS Giulia Martina Cavestro, MD PhD, Raffaella Alessia Zuppardo, MD PhD, Elisabetta Goni, MD, Paolo Giorgio Arcidiacono, MD, Silvia Carrara, MD, Alberto Mariani, MD, Maria Chiara Petrone, MD, Gioacchino Leandro, MD, Pier Alberto Testoni, MD; Vita-Salute San Raffaele University, San Raffaele Scientific Institute, Milan

P126 A UNIFYING CONCEPT FOR PERIAMPULLARY CARCINOMA FROM CLINICO-PATHOLOGIC ANALYSIS OF 198 PATIENTS Peter Bronsert, MD, Ilona Kohler, MD, Dirk Bausch, MD, Frank Makowiec, Prof MD, Martin Werner, Prof MD, Ulrich T Hopt, Prof MD, Tobias Keck, Prof MD, Ulrich F Wellner, MD; University of Schleswig-Holstein and University of Freiburg

P127 PROGNOSTIC SIGNIFICANCE OF INCIDENTALLY DIAGNOSED NON-FUNCTIONING PANCREATIC NEUROENDOCRINE TUMORS David Birnbaum, Sébastien Gaujoux, Rim Cherif, Anne Couvelard, Safi Dokmak, David Fuks, Beatrice Aussillou, Marie-Pierre Vuillerme, Philippe Ruszniewski, Jacques Belghiti, Alain Sauvanet; Department of HPB Surgery – PMAD - Hopital Beaujon - AP-HP - Clichy, France

P128 PARENCHYMA-SPARING PANCREATIC RESECTIONS FOR PRESUMED NON-INVASIVE INTRADUCTAL AND PAPILLARY MUCINOUS TUMORS OF THE PANCREAS Alain Sauvanet, Sebastien Gaujoux, Safi Dokmak, Benjamin Blanc, Anne Couvelard, Marie-Pierre Vullierme, Philippe Ruszniewski, Philippe Lévy, Jacques Belghiti; Department of HPB Surgery – PMAD - Hopital Beaujon - AP-HP - Clichy, France
P129 TYPE I INTERFERON RECEPTOR EXPRESSION IN PANCREATIC AND PERIAMPUTARY CANCER TISSUE (1,2) Stephanie Booy, Msc, (1) Leo Hofland, PhD, (1) Marlijn Waaijers, (1) Peter van Koetsveld, (4) Ed Croze, PhD, (3) Lisette de Vogel, (3) Katharina Biermann, PhD, (1) Casper van Eijck, PhD; Departments of Surgery, Internal Medicine, and Pathology Erasmus MC, Rotterdam, the Netherlands and International review of investigational science, Lafayette, CA, USA

P130 BACTERIAL CONTAMINATION IN ASCITIC FLUID IS ASSOCIATED WITH THE DEVELOPMENT OF CLINICALLY RELEVANT PANCREATIC FISTULA FOLLOWING PANCREATODUODENECTOMY Yuichi Nagakawa, MD, Yuichi Hosokawa, MD, Takaaki Matsudo, MD, Yosuke Hijikata, Satoru Kikuchi, Yoshiaki Suzuki, Kazuhiko Kasuya, Akihiko Tsuchida; Department of Surgery, Tokyo Medical University

P131 GNAS AND KRAS MUTATIONS IN MULTIFOCAL INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS (IPMN) OF THE PANCREAS K. Sahora, MD, D. Dias-Santagata, PhD, L. Fazlollahi, MD MPH, V. Morales-Oyarvide, MD, L.a. Bernardo, BS, A.j. Iafrate, MD PhD, M.b. Pitman, MD, C. Fernandez-del Castillo, MD, M. Mino-Kenudson, MD; Departments of Surgery and Pathology, Massachusetts General Hospital, Harvard Medical School, Boston, USA

P132 OSTEOPONTIN (OPN) ISOFORMS, DIABETES, OBESITY, AND CANCER; WHAT’S ONE GOT TO DO WITH THE OTHER? A NEW ROLE FOR OPN Konrad Sarosiek, MD, Elizabeth Jones, BS, Galina Chipitsyna, PhD, David Tichansky, MD, Charles J Yeo, MD, Hywdya A Arafat, MD PhD; Thomas Jefferson University

P133 SIGNIFICANCE OF RADIOGRAPHIC SPLENIC VESSEL INVOLVEMENT IN PANCREATIC DUCTAL ADENOCARCINOMA (PDAC) OF THE BODY AND TAIL Nathaniel Paul, MD, Geraldine Chen, MD, Adnan Alseidi, MD, Thomas Biehl, MD, Ravi Moonka, MD, Scott Helton, MD, David Coy, MD, Flavio G Rocha, MD; Virginia Mason Medical Center

P134 NONTHERAPEUTIC CELIOTOMY INCIDENCE IS NOT AFFECTED BY VOLUME OF PANCREATICODUODENECTOMY FOR PANCREATIC ADENOCARCINOMA Paul G Toomey, MD, Sharona B Ross, MD, Chris Childs, BHS, Krishen D Patel, Kenneth Lubercice, BS, Alexander S Rosemurgy, MD; Florida Hospital Tampa

P135 TARGETING THE PP2A TUMOR SUPPRESSOR FOR THE TREATMENT OF HUMAN PANCREATIC CANCER Brett Sheppard, MD, Amy Farrell, PhD, Brittany Allen-Peterson, PhD, Colin Daniel, Ping Wang, PhD, Dale Christensen, PhD, Charles Lopez, MD PhD, Rosalie Sears, PhD; Oregon Health and Science University and Cognosci Inc
P136 MANAGEMENT OF DELAYED POST-PANCREATECTOMY HEMORRHAGE BY ENDOVASCULAR TECHNIQUES Kengo Asai, MD PhD, Victor Zaydfudim, MD MPH, James C Andrews, MD, Kaye Reid Lombardo, MD, Michael L Kendrick, MD, Florencia G Que, MD, David M Nagorney, MD, Michael B Farnell, MD; Division of Gastroenterologic and General Surgery and Division of Vascular and Interventional Radiology, Mayo Clinic, Rochester, MN, USA

P137 THE EFFECT OF OPERATIVE APPROACH ON SPLENIC VESSEL PATENCY AFTER SPLEEN AND SPLENIC VESSEL-PRESERVING DISTAL PANCREATECTOMY IN MULTI-INSTITUTIONAL STUDY: LAPAROSCOPIC VERSUS OPEN APPROACH Yoo-Seok Yoon1, Kyoung Ho Lee2, Ho-Seong Han1, Jai Young Cho, Mee Joo Kang3, Jin Young Jang3, Sun-Whee Kim3, Sung-Sik Han4, Sang-Jae Park, Young Joon Ahn5; Departments of Surgery1 and Radiology2, Seoul National University Bundang Hospital, Departments of Surgery3, Seoul National University Hospital, Center for liver center, National Cancer Center4, Boramae Medical Center5, Korea

P138 DECREASED PANCREATIC FISTULA RATE FOLLOWING PANCREATICODUODENECTOMY USING A NOVEL TECHNIQUE OF PANCREATICOJEJUNOSTOMY: FULL THICKNESS PANCREATIC STAY SUTURES AND VIDEO MICROSCOPY Nicholas N Nissen, MD, Vijay G Menon, MD, George Berci, MD; Hepatobiliary and Pancreatic Surgery, Cedars-Sinai Medical Center

P139 IMPACT OF LAPAROSCOPIC APPROACH ON POSTOPERATIVE PAIN AND OPIOID CONSUMPTION AFTER PANCREATEDUODENECTOMY Naru Kondo, MD, Florencia G Que, MD, Michael B Farnell, MD, Kaye M Reid-Lombardo, MD, David M Nagorney, MD, Michael L Kendrick, MD; Mayo Clinic Rochester

P140 SAFETY OF PERIOPERATIVE ASPIRIN THERAPY IN PANCREATIC SURGERY Andrea M Wolf, MD, Jordan M Winter, MD, Salil D Gabale, MD, Eugene P Kennedy, MD, Ernest L Rosato, MD, Harish Lavu, MD, Charles J Yeo, MD; Spectrum Health, Grand Rapids MI and Thomas Jefferson University, Philadelphia PA
**S001 COMPARING EARLY AND DELAYED READMISSION AFTER SURGERY FOR PANCREAS CANCER: A SEER-MEDICARE STUDY**

Marquita R Decker, MD, MPH, David Y Greenblatt, MD, MSPH, Chee P Lin, MS, Jeffery A Havlena, MS, Sharon M Weber, MD, Noelle Loconte, MD, Maureen A Smith, MD, MPH, PhD, Caprice C Greenberg, MD, MPH, Emily Winslow, MD

University of Wisconsin: Department of Surgery - Wisconsin Surgical Outcomes Research (WISOR), Department of Medicine - Division of Hematology-Oncology, UW Comprehensive Cancer Center, UW Health Innovation Program

**BACKGROUND & OBJECTIVE:** Of all major general surgical operations, pancreatic resection is associated with the highest rate of hospital readmission. Given that both early and delayed complications are common after surgery for pancreas cancer, it is unclear whether 30-day or 90-day readmission is the optimal quality measure. The objective of this study was to compare predictors and outcomes of early and delayed readmission in order to determine the utility of these metrics.

**METHODS:** Medicare beneficiaries who underwent pancreatic cancer resection from 2000 to 2008 were identified from the Surveillance, Epidemiology, and End Results-Medicare database. Early and delayed readmissions were defined as first-time readmissions within 1-30 days and 31-90 days after discharge, respectively. Subsequent readmissions were not analyzed. Demographics, comorbidities, cancer- and treatment-related variables, as well as hospital characteristics were examined. Using multivariable logistic regression analysis, predictors of early and delayed readmission were determined and then compared. Outcomes after early and delayed readmission were then examined.

**RESULTS:** Of 2,469 patients who underwent pancreatic cancer resection, 512 (21%) were readmitted within 30 days and an additional 332 (13%) were readmitted within 31-90 days after discharge. Predictors of early readmission included Charlson comorbidity score ≥ 3, discharge to skilled nursing facility (SNF), and initial length of stay greater than 10 days. Predictors of delayed readmission included Charlson comorbidity score ≥ 2, discharge to SNF, and advanced cancer stage. Seventy six percent of early readmissions and 54% of delayed readmissions returned to the index surgical facility (p<0.001). The most common primary diagnosis for early readmission was infection, whereas hypovolemia/dehydration was the leading primary diagnosis for delayed readmissions. Mean length of stay was 8.8 days for early readmission and 6.4 days for delayed readmission (p<0.001). While there was no significant difference in inpatient mortality (6% vs. 5%, p=0.59), the rate of mortality within 30 days of readmission was markedly higher after delayed readmission (27%) versus early readmission (7%, p<0.001).

**CONCLUSIONS:** Early and delayed readmissions after surgery for pancreas cancer are distinct clinical entities that result from different processes. Quality improvement programs should measure both and address them with targeted interventions.

Predictors of Early and Delayed Readmission After Surgery for Pancreas Cancer.
Odds ratios (ORs) are adjusted for age, time from diagnosis until surgery, number of hospitalizations in the year before surgery, receipt of neoadjuvant therapy, perioperative blood transfusion, and index hospital medical school affiliation, NCI cancer center designation, and hospice availability, in addition to the factors listed in the table.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>OR for Early Readmission</th>
<th>95%CI</th>
<th>P</th>
<th>OR for Late Readmission</th>
<th>95%CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Charlson Comorbidity Score</td>
<td></td>
<td></td>
<td>0.019</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0</td>
<td>Reference</td>
<td>Reference</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0.91 (0.70-1.18)</td>
<td>1.25 (0.92-1.70)</td>
<td></td>
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<td></td>
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<tr>
<td>2</td>
<td>1.19 (0.85-1.66)</td>
<td>1.85 (1.44-3.45)</td>
<td></td>
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<tr>
<td>3+</td>
<td>1.59 (1.11-2.26)</td>
<td>2.24 (1.25-2.73)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer Stage At Diagnosis</td>
<td></td>
<td>0.559</td>
<td>0.049</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local or In Situ</td>
<td>Reference</td>
<td>Reference</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regional</td>
<td>0.95 (0.73-1.25)</td>
<td>1.49 (1.06-2.09)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unstaged</td>
<td>1.48 (0.62-3.51)</td>
<td>0.74 (0.16-3.35)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial Length of Stay</td>
<td>&lt;0.001</td>
<td></td>
<td>0.137</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10</td>
<td>Reference</td>
<td>Reference</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;10</td>
<td>1.69 (1.32-2.16)</td>
<td>1.24 (0.93-1.64)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Discharge to SNF</td>
<td>1.48 (1.14-1.93)</td>
<td>0.003</td>
<td>1.44 (1.05-1.99)</td>
<td>0.026</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**S002 REDUCING READMISSIONS FOLLOWING PANCREATECTOMY: COORDINATION OF THE CARE CONTINUUM**

Eugene P Ceppa, MD, Henry A Pitt, MD, Attila Nakeeb, MD, C Max Schmidt, MD, Nicholas J Zyromski, MD, Michael G House, MD, Alisha George-Minkner, RN, Elizabeth W Brand, BSN, Denise J Weidert, MSN, Keith D Lillemoe, MD Indiana University; Indiana University Health

**BACKGROUND:** In 2012 the Centers for Medicine and Medicaid initiated a hospital pay-for-performance program for all-cause readmissions. Recent analyses of major gastrointestinal operations document that postoperative complications are a key driver of readmissions. However, efforts to reduce readmissions require coordination of many aspects of the care continuum, and successful programs to reduce readmissions after major operations have not been reported. Therefore, this analysis documents a series of steps that were implemented to reduce complications and readmissions following pancreatectomy.

**METHODS:** From July, 2007 through June, 2012, the 30-day all-cause readmission rates for patients undergoing pancreatectomy were measured. Patients undergoing proximal, distal and total pancreatectomy were monitored. Length of stay and mortality indices were risk-adjusted by the University Health
Consortium. In 2008, a multifactorial effort was undertaken by all pancreatic surgeons which resulted in fewer surgical site infections by 2009. Subsequently, some of the surgeons altered their drain management. In 2010, a team of surgeons, nurses, advanced providers, pharmacists, social workers and care managers was formed to focus on reducing readmissions. During that year, discharges with home care were increased from 20 to 50%, and relationships with skilled nursing homes, rehabilitation centers and long-term acute care hospitals were strengthened. In 2011, the Readmissions Quality Improvement Team adopted “Project RED” (ReEngineering Discharges) and employed a “discharge coach” to assure that patients were ready for discharge and had proper instructions as well as coordination of follow-up care. Statistical analysis included control charts and Fischer’s Exact tests.

RESULTS: Over five years, 1,147 patients underwent proximal (69%), distal (26%) or total pancreatectomy (5%). The mean age was 58 years; 50% were female; and 39% had pancreatic cancer. The mortality index (observed rate/expected rate) was 0.73 and did not vary significantly over time. The length of stay (LOS) index (observed days/expected days) and 30-day all-cause readmission rates are presented in the table.

<table>
<thead>
<tr>
<th></th>
<th>2007-08</th>
<th>2008-09</th>
<th>2009-10</th>
<th>2010-11</th>
<th>2011-12</th>
</tr>
</thead>
<tbody>
<tr>
<td>PATIENTS</td>
<td>192</td>
<td>241</td>
<td>224</td>
<td>228</td>
<td>262</td>
</tr>
<tr>
<td>LOS Index</td>
<td>1.09</td>
<td>1.04</td>
<td>1.00</td>
<td>1.10</td>
<td>1.01</td>
</tr>
<tr>
<td>READMISSIONS (%)</td>
<td>23.1</td>
<td>19.1</td>
<td>15.4</td>
<td>17.1</td>
<td>11.5*</td>
</tr>
</tbody>
</table>

*P<0.05 vs 2007-11

CONCLUSIONS: All-cause readmissions following pancreatectomy can be reduced without altering length of stay. Efforts by surgeons to reduce complications as well as care coordination by nurses, advanced providers, pharmacists, social workers, care managers and a “discharge coach” are required to reduce readmissions.

1:30pm – S003 READMISSIONS AFTER PANCREAS SURGERY: COMPLEX AND COSTLY

Zeling Chau, MD, Sing Chau Ng, BA, MS, Elan R Witkowski, MD, Tara S Kent, MD, Arthur J Moser, MD, Mark P Callery, MD, Jennifer F Tseng, MD, MPH Beth Israel Medical Center- Harvard Medical School; University of Massachusetts Medical School

BACKGROUND: Hospital readmission after surgical procedures has been advocated as a quality metric. The extent to which readmissions can be reduced and the cost of readmission is still unclear after pancreatectomy. This study aims to identify factors and costs for readmission.

METHODS: The Florida State Inpatient Database with supplemental files for revisit analysis were linked using unique identifiers to allow analysis of longitudinal in-hospital admissions and readmissions. All pancreas resections performed for cancer during 2007-2009 were identified using ICD-9 codes. Demographic data collection included patient characteristics, medical
comorbidities, and hospital volume. Readmission was defined as inpatient admission within 30 days of discharge. Costs were determined by linking the core database to the Healthcare Cost and Utilization Project Cost-to-Charge Files. Univariate and multivariate analysis performed by chi-square and logistic regression. For all, p-values <0.05 were considered statistically significant.

RESULTS: 1,203 patients underwent pancreas resection 2007-2009. 839 (69.7%) Whipple, 247 (20.3%) distal, 45 (3.7%) total, and 72 (6.0%) were proximal/other. The overall readmission rate was 15.0% with a median LOS of 5 days (Quartile 1 Quartile 3 range, 3-8 days), and a median cost of $7,508 (Q1-Q3 $4,870-$12,866). The total calculated cost over the study period was $2.3 million, or $762,770/year. Most common reasons for readmission were GI (n=66), infectious (n=54), malnutrition (n=15), cardiopulmonary (n=14), vascular (n=11). The most costly readmission type was infectious with a total cost of $867,875, median per-admission cost of $9,518 (Q1-Q3 $6,342-14,709) and median LOS of 5.5 (Q1-Q3 3-8) days. Of all readmissions, 25% underwent interventional radiology procedures 15% GI endoscopy, and 6.6% underwent surgical procedure. Of note, high-volume hospitals had higher unadjusted rates of readmission 17.3% vs 12.3% for low-volume hospitals, but after multivariable analysis, this volume effect lost significance.

CONCLUSION: Early readmission after pancreatectomy is common with substantial costs. Our results demonstrate that infectious causes for readmissions are prevalent and expensive. High-volume hospitals are not immune, and due to case mix, may be particularly vulnerable. In the current cost- and outcomes-driven era, interventions aimed at reducing preventable readmissions after pancreatectomy warrants further study.

TABLE: Readmission type and cost

<table>
<thead>
<tr>
<th>Readmit Type</th>
<th>N</th>
<th>% 30 day readmission</th>
<th>Median Days to readmit (Q1-Q3)</th>
<th>Median LOS (Q1-Q3)</th>
<th>Median Cost $ (Q1-Q3)</th>
<th>Total Cost $</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>66</td>
<td>36.7</td>
<td>18 (12-21)</td>
<td>5 (3-8)</td>
<td>6,223 (4,347-10,866)</td>
<td>817,725</td>
</tr>
<tr>
<td>ID</td>
<td>54</td>
<td>30</td>
<td>18 (14-24)</td>
<td>5.5 (3-8)</td>
<td>9,518 (6,342-14,709)</td>
<td>867,875</td>
</tr>
<tr>
<td>Malnutrition/FTT</td>
<td>15</td>
<td>8.3</td>
<td>21 (17-25)</td>
<td>5 (4-9)</td>
<td>9,043 (5,478-13,630)</td>
<td>149,422</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>14</td>
<td>7.8</td>
<td>17.5 (14-19)</td>
<td>2 (1.7)</td>
<td>5,269 (2,535-11,902)</td>
<td>113,282</td>
</tr>
<tr>
<td>PE/DVT</td>
<td>11</td>
<td>6.1</td>
<td>19 (12-25)</td>
<td>5 (4-7)</td>
<td>6,212 (4,892-13,728)</td>
<td>136,329</td>
</tr>
<tr>
<td>Other</td>
<td>20</td>
<td>11.1</td>
<td>18.5 (15-27.5)</td>
<td>4.5 (2-10)</td>
<td>6,010 (3,647-11,194)</td>
<td>203,676</td>
</tr>
</tbody>
</table>

**Oral Abstracts**
LIMITATIONS OF NSQIP IN REPORTING COMPLICATIONS FOR PATIENTS UNDERGOING PANCREATECTOMY: UNDERSCORING THE NEED FOR A PANCREAS-SPECIFIC MODULE

Irene Epelboym, MD, Irmina Gawlas, BA, James A Lee, MD, Beth A Schroepe, MD, PHD, John A Chabot, MD, John D Allendorf, MD
Columbia University Medical Center, Department of Surgery

BACKGROUND: Administrative databases are used with increased frequency for reporting hospital-specific and nationwide trends and outcomes after various surgical procedures in order to improve quality of surgical care. NSQIP is a risk-adjusted case-weighted complication tracking initiative that reports 30-day outcomes from more than 400 academic and community institutions in the United States alone. However, the accuracy of reported events specific to pancreatic surgery has never been reported in depth.

METHODS: We retrospectively reviewed a randomly selected subset of patients, the information on whose postoperative course was originally reported through NSQIP. Preoperative characteristics, operative data, and postoperative events were recorded after review of electronic medical records including physician and nursing notes, operative room records and anesthesiologist reports. We compared categorical variables using chi-square or Fischer’s exact test and continuous variables using Student’s t-test.

RESULTS: Between 2006 and 2010, 316 pancreatectomy cases were reported to NSQIP by our institution. Two hundred and forty-nine were reviewed in detail, among them 145 (58.2%) Whipples, 19 (7.6%) total pancreatectomies, 65 (26.1%) distal pancreatectomies, and 15 (6.0%) central or partial resections. Median age was 65.7, males comprised 41.5% of the group, and 74.3% of patients were Caucasian. Overall rate of complications reported by NSQIP was 44.0%, compared with 55.0% in our review, however discordance was observed in 73 (29.3%) cases (p<0.001), including 24 cases of reporting a complication where there was not one, and 49 cases of missed complication. Most frequently reported event was postoperative bleeding requiring transfusion (22.7%), however true incidence of postoperative bleeding was actually 19.0%, with NSQIP missing 27 (57.5%) and incorrectly reporting 36 (64.3%), p<0.001. Four procedures unrelated to the index operation were recorded as reoperation events. While a pancreas-specific module does not yet exist, NSQIP reports a 7.6% rate of organ-space surgical site infections; when compared with our institutional rate of grade B and C postoperative fistula (8.8%), we observed discordance 6% of the time, p<0.001. Delayed gastric emptying, a common post-pancreatectomy morbidity, was not captured at all. Additionally, there were significant inaccuracies in reporting urinary tract infections, postoperative pneumonia, wound complications, and postoperative sepsis, with discordance rates of 4.4%, 3.2%, 3.6%, and 6.8%, respectively.

CONCLUSIONS: NSQIP data is an important and valuable tool for evaluating quality of surgical care, however pancreatectomy-specific postoperative events are often misclassified, underscoring the need for a hepatopancreatobiliary-specific module to better capture key outcomes in this complex and unique
INTRODUCTION: Multiple prospective, randomized trials have demonstrated that the addition of adjuvant therapy after surgical resection of pancreatic cancer improves survival compared to surgery alone. However, the optimal type of adjuvant therapy, chemotherapy alone or chemotherapy combined with chemoradiation therapy, remains controversial. Our aim was to determine whether the type of adjuvant therapy for pancreatic cancer given in the United States has changed by examining treatment trends using the National Cancer Data Base.

METHODS: The National Cancer Data Base (NCDB) is a national oncology outcomes database for over 1,500 Commission on Cancer-accredited cancer programs. Patients diagnosed with stage 1-2 pancreatic adenocarcinoma between 2003-2009 were selected from the NCDB Hospital Comparison
BENCHMARK REPORTS. Attention was paid to the initial treatment regimen, such as surgery alone, surgery plus chemotherapy, or surgery plus chemoradiation. In addition, data on hospital setting (teaching-research hospitals vs. community hospitals) were collected and analyzed. The Cochran-Armitage test for trend was used to assess changes in treatment over time.

RESULTS: 47,086 patients with stage 1-2 pancreatic adenocarcinoma were included in the analysis. Between 2003-2009, the use of surgery alone as first course treatment of stage 2 disease decreased significantly at both teaching-research hospitals and community hospitals by nearly 25% (p<0.0001 for both cases). In the same period, the use of chemotherapy in addition to surgery as treatment of stage 1 and 2 disease increased two-fold at both types of hospitals (p<0.0001 for all cases). Treatment with surgery plus chemoradiation decreased significantly for both stages in both hospital settings by approximately 30% (p<0.05 for all cases). Non-surgical treatment for stage 2 disease was surprisingly high and significantly increased over time (p<0.0001 for both), ranging from approximately 30-37% at teaching-research hospitals and 40-49% at community hospitals.

CONCLUSION: Data from the NCDB from 2003-2009 illustrate changes in the adjuvant treatment of pancreatic cancer. There is an alarmingly high rate of non-surgical therapy for stage 1 and 2 disease. The use of chemotherapy alone as adjuvant therapy increased whereas the use of multimodality therapy decreased.

S006 NINETY-DAY MORTALITY RATE AFTER RESECTION OF CANCER OF THE PANCREAS IS NEARLY DOUBLE THIRTY-DAY MORTALITY: ANALYSIS OF 20,000 PANCREATECTOMIES IN THE NATIONAL CANCER DATA (NCDB)

Richard S Swanson*, MD, Kathy Mallin^, PhD, Christopher M Pezzi**, MD, Andrew Stewart^, MA, Bryan Palis^, MA, David P Winchester^, MD

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INTRODUCTION/BACKGROUND: Operative Mortality has traditionally been defined as death occurring within 30 days or during the hospitalization for the surgery, and most of the studies that established the volume/outcome relationship for pancreatectomy have used a similar definition. We hypothesized that 90-day mortality after pancreatectomy would be significantly higher than 30-day operative mortality, but that the volume/outcome relationship would persist.

METHODS: All pancreatectomies reported to the NCDB in 2004-2008 performed in adults at more than 1,500 hospitals were examined for 30 and 90 day mortality rates. Unadjusted mortality rates were compared by type of resection, stage of disease, co-morbidities and average annual hospital surgical volume. Hierarchical logistic regression models generated risk-adjusted odds ratios for 30- and 90-day mortality.

RESULTS: Data on 30-day mortality was available for 19,965 pancreatectomies, and for 90-day operative mortality from 19,780 of those cases during the
study period. Unadjusted operative mortality for all cases was 4.4% (3.9-4.8), 95%CI) at 30 days and 8.7% (8.0-9.4) at 90 days. Unadjusted and risk-adjusted mortality was higher with increasing age, increasing stage of disease, male gender, lower income, low hospital volume, non-endocrine tumors, resections other than distal pancreatectomy, and multiple different co-morbidities at 30 and 90 days. The lowest volume (< 5/year) hospitals performed 5,233 pancreatic resections with a risk-adjusted odds ratio for mortality that was 2.8 times higher (2.2-3.5, 95%CI) at 30 days and remained 2.6 times higher (2.1-3.1, 95%CI) at 90 days, compared to hospitals with high volumes (>=20 year).

DISCUSSION/CONCLUSION: Mortality rates within 90 days after pancreatic resection are nearly double mortality rates at 30 days after surgery. The volume/outcome relationship persists. While the reasons for this ongoing mortality after pancreatectomy are not fully known, the examination of mortality rates 90 days after pancreatectomy may be important when examining hospital mortality rates.

S007 PATTERNS OF FAILURE FOLLOWING WHIPPLE PROCEDURE FOR RESECTABLE PancreATIC DuCTAL ADENOCARCINOMA Avani S Dholakia, BS, Rachit Kumar, MD, Aaron T Wild, BA, Amy Hacker-Price, MS, PAC, Susannah Ellsworth, MD, Siva P Raman, MD, Dung T Le, MD, Ana De Jesus-Acosta, MD, Le Zheng, MD, PhD, Elliot K Fishman, MD, Ralph H Hruban, MD, Matthew J Weiss, MD, Johns Hopkins School of Medicine

INTRODUCTION/BACKGROUND: Studies demonstrate a ~20-60% local failure rate for resectable pancreatic ductal adenocarcinoma (R-PDA) following adjuvant therapy (AT). Given the significant morbidity and mortality associated with local failure, it is important to characterize their precise location to guide approaches to local therapies. This is the first detailed illustration of the relationship of local failures to key anatomical landmarks.

METHODS: Databases of patients with PDA treated from 2000-2010 were queried revealing 873 patients with R-PDA. Patients were included if they underwent pancreaticoduodenectomy (PD) with available operative records and at least one computed tomography scan >60 days following PD. The final cohort of 211 patients was divided based on AT in the following groups: no adjuvant (NA), chemotherapy alone (CA), chemotherapy and/or chemoradiation (CRT). Radiology images were reviewed for local and distant failure. Corresponding images were reviewed. Local failures were categorized as at or near superior mesenteric artery or vein (SMA/V), celiac artery, portal vein/confluence, loco-regional nodes, or other locations. Failures were plotted to scale with respect to celiac artery, SMA, and renal veins on one CT scan of a post-PD patient using Pinnacle Software creating a three-dimensional illustration of local failure following PD.
RESULTS: Of the 211 patients studied, 41 (19.4%) received NA, 35 (16.6%) CA, and 135 (64.0%) CRT AT. Local failure rates were 19/41 (46.3%), 25/35 (68.6%), and 53/135 (39.3%), with statistically significant differences between NA vs. CA (p=0.043) and CA vs. CRT (p=0.002). Overall, median local progression free survival (LPFS) was 25.00 months (95% CI 16.55-33.45). We observed progressively increasing LPFS based on AT category at 9.50 months (95% CI 5.01-14.00), 14.00 months (95% CI 8.34-19.66) and 41.00 months (95% CI 21.66-60.34) for NA, CA, and CRT groups respectively (p=<0.001). Local failure frequencies in margin positive patients were 5/13 (38.5%), 5/5 (100%), and 26/57 (45.6%), with statistically significant difference between NA vs. CA (p=0.03) and CRT vs. CA (p=0.03). Local failure frequencies in node positive patients were 16/33 (48.5%), 18/27 (66.7%), and 40/106 (37.7%), with statistically significant difference between CRT vs. CA (p=0.01). There was again a trend of progressively increasing LPFS based on category in both node (p=<0.001) and margin positive patients (p=<0.001).

Local failure occurred within the surgical bed along the SMA/V or celiac artery in 60/96 (62.5%) of total cases, distributing as 14/19 (73.8%), 13/24 (54.1%), and 33/53 (62.3%) cases in NA, CA, and CRT groups respectively. Loco-regional nodal failures were uncommon, occurring in only 15/96 (15.6%) of all cases, specifically 2/19 (10.5), 5/24 (20.8), and 8/53 (15.1) in NA, CA, and CRT groups respectively. Local failures are plotted to scale in the included figure.

DISCUSSION/CONCLUSION: This reconstitution illustrates the precise points of local failure in the experience of our institution. This map can be used to guide improvements in adjuvant treatment using increased radiation does to higher risk areas while minimizing dose to areas where local failure is unlikely.
S008 PERIOPERATIVE BLOOD TRANSFUSION REDUCES SURVIVAL IN PATIENTS WITH PANCREATIC ADENOCARCINOMA: A MULTIPLE-INSTITUTIONAL STUDY OF 698 PATIENTS
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INTRODUCTION: In this multi-institutional study of patients undergoing pancreaticoduodenectomy (PD) for pancreatic adenocarcinoma, we sought to identify factors associated with a perioperative blood transfusion requirement. In addition, we investigated the hypothesis that undergoing peri-operative blood transfusion reduces long-term survival in this patient population.

METHODS: A retrospective chart review was performed across six high-volume institutions to identify patients who underwent PD between 2005 and 2010. Data collection included patient demographics, perioperative factors, transfusion status, and survival data. For statistical analysis, patients were then grouped according to whether they received 0, 1-2, or >2 units of packed red blood cells (pRBCs).

RESULTS: Among 698 patients identified, 168 (24%) required blood transfusion. 105 (15%) patients received 1-2 units and 63 (9%) patients received >2 units (range 0-25 units). Patient demographics associated with an increased transfusion requirement included age, smoking status, and heart disease (all p < 0.03). Operative variables associated with an increased transfusion requirement included operative time, estimated blood loss, tumor size, and R1/R2 margin status (all p < 0.03). Postoperative complications were not associated with transfusion requirement. However, those patients who received transfusions experienced a longer length of stay (p = 0.0009) as well as increased rate of readmission within 90 days (p = 0.002). The median survival of patients who received >2 units of pRBCs was significantly less than those who received either 0 or 1-2 units (10.2 months vs. 18.4 or 18.9 months, p = 0.0002). A multivariate model including margin status, nodal involvement, tumor size, and transfusion status identified the transfusion of >2 units of pRBCs as an independent predictor of reduced survival (HR 1.56, p = 0.03).

CONCLUSIONS: This multi-institutional study represents the largest series to date analyzing the effects of pRBC transfusion in patients undergoing PD for pancreatic adenocarcinoma. The transfusion rate in this series is less than what has been previously reported. Our data confirm that blood transfusion confers a negative impact on long-term survival in this patient population. These results can be utilized as a benchmark for future studies.
S009 COMPARING THE IMPACT OF COMPLICATIONS AFTER MAJOR PANCREATECTOMIES USING THE POSTOPERATIVE MORBIDITY INDEX

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OBJECTIVE: Postoperative complications are traditionally reported by incidence, but not quantified. The postoperative morbidity index (PMI) is derived using two validated systems, ACS-NSQIP and the Accordion Severity Grading System, for which quantitative complication severity weights were previously derived by expert opinion. This study compares the burden of complications and presents the morbidity spectrum of common pancreatic operations.

METHODS: Nine American centers contributed ACS-NSQIP complication data for 2308 pancreatic resections from 2005-2011. Each complication was assigned one of six previously established Accordion severity weights ranging from 0.11 for the least severe complication (grade 1) to 1.00 for postoperative death (grade 6). The PMI reflects the sum of complication weights for all complications divided by the total number of patients. PMI can range from 0 (no complication in any patient) to 1.00 (all patients died of complications). Contribution to total burden of complications by each complication grade was also derived and displayed in a severity “spectrogram”. The PMI and specific morbidity profile were compared between proximal (PD), distal (DP), and total (TP) pancreatectomies.

RESULTS: ACS-NSQIP complications occurred in 712 cases (30.9%). The frequency of complications differed significantly (P=0.002) among the three types of procedures (Table) and Grade 2 complications were highest in frequency. There were 30 deaths for a mortality rate of 1.3%. The PMI for all pancreatectomies in the series was 0.109. The most common complication contributing to the PMI for each operation was Organ Space Infection (7.8% overall). The spectrogram for major pancreatectomy (Figure) illustrates the difference between frequency and burden of different complication grades. DPs were significantly less morbid than were either PDs (p=0.001) or TPs (p=0.028). This was due to a higher occurrence of grade 5/6 (multiorgan failure/death) complications contributing to more burden in PD and TP. Conversely, Grade 2/3 complications provided a greater contribution in DPs. PD did not differ from TP (p=0.214). There was significant variation in PMI by institution for PD (p<0.001), but not for other procedures. Variation also existed among individual surgeons for PD and DP. Subgroup analysis reveals the PMI of DP did not vary based on laparoscopic vs. open approach or the performance of splenectomy, but did increase with the addition of colon resection. The PMI of TP increases with age. The complication burden in complication-
bearing patients only was also examined; there was no difference among the procedures.

CONCLUSION: This study establishes quantitative benchmarks for morbidity of common pancreatic operations. It illustrates the fact that frequency of complications does not equate to their burden. The PMI provides an objective means of comparing the impact of types and grades of complications across various operations.

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% Contribution to overall PMI

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OBJECTIVE: Pancreatic ductal adenocarcinoma (PDAC) appears to arise from pancreatic intraepithelial neoplasms (PanINs). Cancer cells break through the basement membrane from PanIN-3, they evolve into infiltrating adenocarcinoma. The invasion process is the crucial step in PDAC, because cancer cells that invade the vasculature, or lymphatic or neural vessels, can progress further to metastasis only after obtaining infiltrating status, however, the genes related to invasion remain unclear. In this study, we identified specific molecular markers, MUC16 and mesothelin, that were associated with invasion in PDAC by gene expression profiling.

METHODS: The microarray data of the infiltrating cancer and PanIN-3, which were harvested from an individual PDAC patient by laser microdissection, were compared to identify the specific genes for invasion process in PDAC. Coimmunoprecipitation assay was performed using pancreatic cancer cell lines and surgical specimens to address binding between MUC16 and mesothelin. To investigate the effect of MUC16 and mesothelin expression on invasion and migration of pancreatic cancer cells, in vitro invasion and migration assays were performed in the membrane culture system. We analyzed the relationship between MUC16/mesothelin expression and PDAC clinicopathological factors by immunohistochemistry in 106 patients with PDAC.

RESULTS: We focused on MUC16 and mesothelin among 87 genes that were significant up-regulated in infiltrating components compared to PanIN-3 in all PDAC patients by gene expression profiling, because MUC16 was the most differently expressed between two regions, and mesothelin was reported as MUC16 ligand in ovarian cancer. Immunohistochemical analysis revealed that MUC16 and mesothelin were expressed simultaneously only in infiltrating components and not expressed in both all PanIN lesions and normal pancreatic tissues, furthermore, the expression of these genes increased at the invasion front in PDAC. The immunoprecipitation assay showed binding of MUC16 and mesothelin in both cell lines and surgical tissues of PDAC. The down-regulation of MUC16 by shRNA and the blockage of MUC16 binding to mesothelin by antibody inhibited both invasion and migration of pancreatic cancer cell line. Immunohistochemical analysis for 106 patients with PDAC showed that a tumor size >4.0 cm, serosal invasion, invasion of other organs, and lymphatic permeation occurred significantly more often in the MUC16 high/mesothelin high expression group than in the other groups (P<0.01, P= 0.01, P=0.03, and P=0.02, respectively). MUC16 high/ mesothelin high expression was an independent prognostic factor for poor survival in PDAC patients (P=0.01, HR, 1.99, 95%CI, 1.15-3.41).

CONCLUSION: MUC16 and mesothelin are involved in pancreatic cancer cell invasion and migration, and MUC16 and mesothelin clinically represent
new prognostic biomarkers for PDAC and might be new therapeutic targets for patients with PDAC, including immunotherapy using a peptide vaccine or monoclonal antibody therapy.

**S011 DCK IS A PROGNOSTIC MARKER AND CORRELATES WITH 5-FLUOROURACIL RESPONSE AND HUR STATUS IN PANCREATIC CANCER: ANALYSIS FROM THE RTOG 9704 TRIAL**

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**INTRODUCTION/BACKGROUND:** There is an urgent need to identify potential biomarkers for the treatment of pancreatic ductal adenocarcinoma (PDA). Previous studies have shown that both deoxycytidine kinase (dCK), the rate-limiting enzyme involved in the metabolism of gemcitabine, and the RNA binding protein, HuR, have predictive value for sensitivity to gemcitabine, the standard of care treatment for advanced pancreatic cancer since 1997. This study investigates the prognostic value of dCK and HuR status in the context of a previously published randomized clinical trial on patients with resected pancreatic cancer and the predictive value to determine sensitivity to gemcitabine and 5-fluorouracil (5-Fu).

**METHODS:** The RT0G 9704 was a randomized phase III trial in which patients with pancreatic cancer were randomized, after surgical resection, to receive 5-Fu-based chemoradiation preceded and followed by either 5-Fu or gemcitabine. The dCK and HuR subcellular status expression levels in tissue harvested in this study were determined by immunohistochemistry. Their association with overall survival (OS) and disease-free survival (DFS) status was analyzed using the log-rank test and the Cox proportional hazards model. Furthermore, the association between dCK levels and HuR status and their joint predictive value was also examined. In a cell culture model, 5-Fu’s ability to translocate HuR from the nucleus to the cytoplasm (i.e., HuR activation) was detected.

**RESULTS:** Of 538 randomized patients, dCK levels were determined for 165 patients, of which 116 patients also had HuR status data. dCK expression was associated with improved OS for all patients analyzed from RTOG 9704 (HR: 0.66, 95% CI [0.47-0.93], p = 0.015). Surprisingly, dCK expression was not associated with OS in the gemcitabine arm. dCK expression was instead associated with OS (HR: 0.53, 95% CI [0.33, 0.85], p = 0.0078) and DFS (HR 0.60, 95% CI [0.38, 0.95], p=0.027) in multivariate analysis for patients that received 5-Fu only. dCK and HuR scores were strongly associated (chi-square p=0.015) and had an additive predictive value in the 5-Fu arm (p=0.0018). Additionally, we detected that 5-Fu exposure to PDA cells translocates HuR from the nucleus to the cytoplasm, most likely regulating target transcripts important for 5-Fu efficacy.

**DISCUSSION/CONCLUSION:** In a large prospective randomized trial, dCK expression levels in combination with HuR cytoplasmic status have a predictive
value for DFS in patients who received 5-FU, but not in the patients that received gemcitabine. One explanation for this is the deleterious effect radiation has on the HuR stress response (including the regulation of dCK) and the fact that cycling, proliferative cells (i.e., high dCK and HuR expressing cells) are better targets for 5-FU therapy. Additionally, it is demonstrated for the first time that HuR cytoplasmic levels correlate with protein expression of dCK, an established HuR target, in untreated, resected PDA specimens. Once validated, these findings may have important clinical utility since dCK and HuR may be reliable predictive markers in different trial settings for both 5-FU and gemcitabine (with no radiation)-based therapies for PDA.

S012 PROGNOSTIC AND BIOLOGICAL ROLE OF MIR-101, MIR-155 AND MIR-21 IN PANCREATIC INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS

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PURPOSES: The goal of this multicenter study was to identify microRNAs (miRNAs) as potential prognostic biomarkers in patients affected by intraductal papillary mucinous neoplasms (IPMNs) of the pancreas.

EXPERIMENTAL DESIGN: The expression of three candidate miRNAs (miR-21, miR-155 and miR-101) was quantified by quantitative RT-PCR in 86 laser-microdissected (LMD) formalin-fixed paraffin embedded (FFPE) specimens, including 65 invasive IPMNs, 16 non-invasive IPMNs and 5 normal pancreatic ductal tissues. Univariate and multivariate analyses compared miRNAs and clinical parameters with overall and disease-free-survival (OS, DFS) using log-rank test and Cox’s proportional hazards model.

RESULTS: MiR-21 and miR-155 were significantly upregulated in invasive IPMN compared to non-invasive IPMN, as well as in non-invasive IPMN compared to normal ductal tissues. Conversely, miR-101 levels were significantly higher in non-invasive IPMN and normal tissues versus invasive IPMN. Kaplan-Meier survival analysis revealed that high levels of miR-21 expression were closely associated with worse OS (hazard ratio [HR] 2.47, P=0.0047). Patients with high miR-21 expression also had a significantly shorter median DFS (10.9 vs. 29.9 months, log-rank P=0.01). Multivariate analysis confirmed miR-21 as independently prognostic for both mortality and disease progression (death-risk, HR=3.3, P=0.02; progression-risk, HR=2.3, P=0.02), as well as positive lymph-node status (death-risk, HR=2.6, P=0.03; progression-risk, HR=2.2, P=0.04).

CONCLUSIONS: The miRNAs evaluated in the present study showed significant differences in invasive versus non-invasive IPMN, and miR-21 expression emerged as an independent prognostic biomarker in patients affected by invasive IPMN, offering innovative tools for the optimal management of these tumors.
OBJECTIVE: Main pancreatic duct (MPD) involvement is a well-demonstrated risk factor for malignancy in intraductal papillary mucinous neoplasm (IPMN). Preoperative radiographic determination of IPMN type (main, mixed, or branch) is relied upon heavily in preoperative oncologic risk stratification. We hypothesize that preoperative radiographic assessment of MPD involvement in IPMN is an accurate predictor of pathologic MPD involvement.

METHODS: Data regarding all patients undergoing resection for IPMN at a single, academic institution between 1992 and 2012 were gathered prospectively. Retrospective analysis of imaging, clinical, and pathologic data was undertaken. Preoperative classification of IPMN type was based on cross-sectional imaging (CT or MRI).

RESULTS: Three-hundred and sixty four patients underwent resection for IPMN. Of these, 335 had adequate data on both radiographic and pathologic parameters for comparison. Of 184 suspected branch duct (BD) IPMN, 35 (19%) demonstrated MPD involvement on final pathology. Of 84 mixed-type (MT) IPMN 16 (19%) demonstrated no MPD involvement. Of 68 suspected main duct (MD) IPMN 13 (19%) demonstrated no MPD involvement. Of 35 of 184 (19%) that had a suspected BD IPMN but were found to have MPD involvement on pathology, 12 (34%) had invasive carcinoma. Alternatively, in patients with suspected MD or MT IPMN who ultimately were found to have no main duct involvement on pathology 2 (7%) demonstrated invasive carcinoma.

CONCLUSION: In resected IPMN, MPD involvement has been demonstrated as an independent risk factor for invasive cancer. Preoperative radiographic IPMN type correlates with final pathology in 81% of patients. In addition, risk of invasive carcinoma correlates with pathologic presence (or absence) of main duct involvement. Consequently, preoperative imaging for oncologic risk stratification may over or under weigh risk in up to one in five patients.

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<td>16 (19%)</td>
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* = P < 0.05  ** = P < 0.0001
CONSERVATIVE MANAGEMENT OF BRANCH DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM OF THE PANCREAS WITH WORRISOME FEATURES

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INTRODUCTION: The natural history of branch duct intraductal papillary mucinous neoplasm (BD-iPMN) is not well-established, and the proper management and follow-up strategy of BD-iPMN is controversial. International consensus guidelines 2012 for the management of BD-iPMN has documented “worrisome features” including clinical pancreatitis at presentation and cyst of ≥3 cm, thickened enhanced cyst walls, main pancreatic duct (MPD) size of 5-9 mm, non-enhanced mural nodules (MNs), abrupt changes in the MPD caliber with distal pancreatic atrophy and lymphadenopathy on imaging studies. The aim of this study was to assess our single-institutional follow-up data of BD-iPMN with worrisome features.

METHODS: Between January 1997 and December 2012, we experienced 416 pts with presumed BD-iPMN, confirmed by surgical resection and typical findings of various imaging modalities including CT, EUS, MRCP and/or ERCP. Of 416 pts, 85 pts with worrisome features (46 men, 39 women; mean age at diagnosis, 73.7 years, range 50-92) were followed conservatively for more than 1 year because of unfit and/or refusal for surgery. The median follow-up period was 67 months (range 12-149 months) and 8 pts were lost to follow-up. CT scan with EUS/MRCP was alternated with an interval of maximum 6 months as possible. Cyst size increase was considered significant ≥5 mm.

RESULTS: Acute pancreatitis was present in 4 pts (4.7%) during the follow-up period. The mean value of the initial cyst size in all pts was 25.3 mm (range, 9-50 mm). On follow-up, 27 pts (31.8%) showed an increase in size (median increase: 8 (5-25) mm), 53 pts (62.3%) remained unchanged in size, and 5 pts (5.9%) decreased in size. MNs were present in 72 pts (84.7%) at the time of diagnosis and a MN appeared in 4 pts (4.7%). Diameter of MPD ≥5 mm was present in 22 pts (25.9%) and 2 pts showed an increase in diameter more than 10 mm. One underwent surgical resection after a follow-up of 77 months, and histological findings revealed adenoma. Invasive pancreatic ductal adenocarcinoma occurred in 3 pts, after 20, 24, and 52 months follow-up, respectively. During the follow-up period, there were 21 nonpancreatic cancer deaths and 3 pancreatic cancer deaths. Of the nonpancreatic cancer deaths, 8 were due to extrapancreatic malignancy (EPM), 10 were due to nonmalignant disease, and 3 were from unknown causes. Among patients who died of EPM, 2, 3 and 3 EPMs had been diagnosed before, during, and after the diagnosis of BD-iPMN.

CONCLUSION: For pts at high risk for nonpancreatic cancer mortality, a follow-up management is more reasonable than immediate surgical resection in pts with BD-iPMN with worrisome features. However, particular attention
of the entire pancreas should be paid to the development of pancreatic ductal adenocarcinoma in patients with BD-IPMN.

**S015 SMALL NON-FUNCTIONAL PANCREATIC NEUROENDOCRINE TUMORS ARE ASSOCIATED WITH A LOW INCIDENCE OF NODAL METASTASIS AND AN EXCELLENT OVERALL SURVIVAL**

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INTRODUCTION/BACKGROUND: Pancreatic neuroendocrine tumors (PNET) are rare neoplasms comprising 3-5% of all pancreatic tumors and are associated with an excellent long-term survival. PNETs are further classified into functional or non-functional (NF) subgroups. Surgery is the primary treatment modality for early-stage resectable tumors. However, the need for resection and the appropriate operation remain controversial for small NF-PNETs.

METHODS: The records of 156 patients who underwent resection for PNETs between 1989 and 2012 were retrospectively reviewed. Functional tumors were defined by patients with preoperative symptoms or elevated serum hormones, and histopathologic confirmation after resection. There were 40 patients with functional tumors and 116 with NF tumors, the latter being the focus of our study. The parameters analyzed included patient demographics, surgical data, pathologic diagnosis, postoperative morbidity and mortality, and overall survival. Survival probability was calculated by the Kaplan-Meier method, and univariate Cox regression was used to assess the correlation of various clinical and pathologic factors with survival.

RESULTS: The median age of the 116 patients with NF-PNETs was 57.5 years (range: 18-84). Operations performed included 65 distal pancreatectomies (56%), 43 pancreaticoduodenectomies (37.1%), 4 enucleations (3.4%) and 4 middle pancreatectomies (3.4%). The majority of tumors (n=105, 90.5%) were WHO 2010 G1 or G2 and well-differentiated. 32 patients (27.6%) had positive lymph nodes (LN), and 17 patients (14.7%) had metastases at the time of resection. 37 patients (31.9%) had tumors smaller than 2cm, and 79 patients (68.1%) had tumors 2cm or larger. The median follow-up was 49.2 months (range: 3.4 – 247.1), and there were no perioperative deaths. The overall 5- and 10-year survivals were 85.8% and 77.9%, respectively. The most significant predictor of improved survival was negative LNs (p=0.01); smaller tumor size (p=0.02) and lack of metastases at the time of resection (p=0.04) also significantly correlated with survival. The 10-year survival was 89.8% for patients with negative LNs and 53.5% for patients with positive LNs. Tumor size was positively correlated with LN status (p<0.001). ROC analysis demonstrated that tumor size is a reasonable predictor of LN status (AUC 0.75) and that a size cutoff of 2cm is associated with a 97.3% sensitivity for nodal disease. Moreover, 2.7% of tumors smaller than 2cm were associated with positive LNs, while the rate was 39.2% for tumors 2cm or larger.

DISCUSSION/CONCLUSION: It remains unclear if small NF-PNETs must be
resected and, if so, if a limited resection is sufficient. In this study, LN status, a marker of systemic disease, was the most significant predictor of survival. Furthermore, tumor size, a measure that can be assessed noninvasively, was correlated with nodal disease. Tumors less than 2cm in size were unlikely to have LN metastases. Therefore, it would be reasonable to consider close observation or limited resection (e.g. enucleation, middle pancreatectomy or spleen-preserving distal pancreatectomy) for these small NF-PNETs. Treatment decisions should be based on an assessment of the risk-benefit ratio for each patient, with nonoperative management most suitable for those with high risk.

S016 PREDICTORS OF LYMPH NODE METASTASES AND IMPACT ON SURVIVAL IN RESECTED PANCREATIC NEUROENDOCRINE TUMORS, A SINGLE CENTER EXPERIENCE Joyce Wong, MD, William Fulp, PhD, Jonathan R Strosberg, MD, Larry K Kvols, MD, Pamela J Hodul, MD Moffitt Cancer Center

BACKGROUND: Currently, staging for pancreatic neuroendocrine tumors (PNET) considers tumor size, lymph node status, and histologic differentiation. However, the predictive value of these factors as it relates to overall survival (OS) remains unclear. This study reviews predictors of lymph node (LN) metastases and the impact on survival for resected PNET.

METHODS: A prospectively maintained database of patients treated for PNET was reviewed. Patients undergoing surgical resection without evidence of metastatic disease at time of resection were included in this analysis. Chi-Square Test was used to compare categorical variables and LN metastases, and Wilcoxon Rank Sum Test was used for continuous variables, both with the exact method using Monte Carlo estimation. Univariate and multivariate analysis was performed with Cox proportional hazard models and survival calculated with Kaplan Meier curves.

RESULTS: From 1999-2012, 150 patients underwent surgical resection for PNET. The majority (53%) were male, with a median age of 56 years (range 17-82). Incidentally discovered PNET was the most common presentation (42%), followed by abdominal pain (32%). Tumors were uncommonly functional (7%). Distal pancreatectomy was performed in 58%; pancreaticoduodenectomy in 29%, and enucleation in 7%.

Of 113 (75%) patients with LN data available for review, 32 (28%) had positive LN (LN+). Both age and lymph node retrieval differed in the LN negative (LN 0) vs. LN+ group, with younger median age (53 years) and higher median LN count (9 vs. 6) in the LN+ group, p=0.05 and p=0.04, respectively. Univariate analysis showed gender, race, clinical presentation, surgery type, and tumor size was not predictive of LN+. Presence of perineural (p=0.016) and lymphovascular (p<0.001) invasion, however, was more common in LN+. With multivariate analysis, only poor/moderate differentiation predicted LN+, with an odds ratio of 7.3 (95% CI: 1.9, 27.6).

Median follow-up for the cohort was 52 months; estimated median OS was 225 months with 5 year OS of 90%. Univariate and multivariate analysis identified
older age at diagnosis and poor/moderate differentiation as factors that negatively impacted OS. 52 (35%) patients developed recurrent disease; the majority recurred with distant metastases (N=46, 88%), with liver being the most common site. Of those who recurred, 25 (48%) had received adjuvant therapy following resection. Estimated median disease free survival (DFS) was 74 months. Only poor/moderate differentiation affected DFS. Tumor size and LN+ did not significantly impact survival.

RESULTS: PNET is an uncommon entity with an unclear prognosis based on variables commonly factored into the staging criteria. In this study, tumor size did not predict LN+; furthermore, LN+ did not predict a worse OS or DFS. Tumor differentiation appears to be more important in determining prognosis for resected PNET.

S017 STAT3 INHIBITION ATTENUATES CHEMoresistance AND ENHANCES Drug Delivery in Pancreatic Cancer Jason Castellanos, MD, Nagaraj Nagathihalli, PhD, Nagaraj Nagathihalli, PhD, Yughander Beesetty, MS, Michelle Reyzer, PhD, Chanjuan Shi, MD, Richard Caprioli, PhD, Nipun Merchant, MD Vanderbilt University Medical Center

INTRODUCTION: The failure of conventional and targeted chemotherapy regimes to produce meaningful impact on survival in patients with pancreatic cancer (PDAC) highlights a desperate need for novel treatment strategies. A hallmark in PDAC is the presence of a dense desmoplastic stroma within the tumor microenvironment (TME) which is characterized by proliferation of fibrotic tissue in which the vasculature functions poorly and impedes the delivery of chemotherapeutic drugs to the tumor cells. We have previously established a mechanistic rationale for activated STAT3 as a biomarker of resistance to cytotoxic and molecularly targeted therapy in PDAC. The purpose of this study was to determine the mechanism of regulation of STAT3 on the TME and to test whether STAT3 inhibition improves drug delivery and therapeutic efficacy.

METHODS: Total and activated STAT3 expression were determined in human PDAC tissues (n=106) and human and mouse cell lines generated from PanIn,PDAC and liver metastases. IC50 values for AZD1480, a JAK2/STAT3 inhibitor, were determined against nine human PDAC cell lines to determine sensitive and resistant lines. STAT3 knockdown and overexpressed cells were developed and assayed for tumorigenicity and multiple downstream effectors. PDAC cells treated with AZD1480 or STAT3 siRNA were evaluated for mRNA and protein expression of HIF-1α, MMP9, MMP7, VEGF, SPARC, cyclin-D1, c-Myc, c-Fos and survivin, and VEGF release by ELISA. ChiP assay was performed to study STAT3 binding to c-Myc, cyclin-D1 and iNOS promoters. In vivo effects of AZD1480, gemcitabine or the combination were determined on orthotopic xenografts of BxPC3 and PANC1 cells. In vivo expression of pSTAT3, cleaved caspase3, Ki67 SPARC, fibronectin, VEGF and CD31 were studied by immunohistochemistry and drug delivery was analyzed by imaging MALDI-mass-spectrometry.
RESULTS: STAT3 activation is necessary for malignant phenotype and affects survival in PDAC. Nuclear STAT3 forms a transcriptional complex with the c-Myc promoter. Treatment with AZD1480 or STAT3 siRNA inhibits stromal, angiogenesis, hypoxia and proliferation markers. The combination of AZD1480 and gemcitabine resulted in cooperative inhibition of cell migration and invasion of both sensitive and resistant PDAC cells at concentrations that were ineffective as individual agents. The combined treatment of AZD1480 and gemcitabine increased drug delivery to the resistant pancreas tumor tissue in vivo and enhanced growth inhibition of orthotopic tumor xenografts.

CONCLUSIONS: Targeting STAT3 overcomes drug resistance by regulating the tumor stroma, angiogenesis and hypoxia. These results provide evidence that targeted STAT3 inhibition combined with gemcitabine affects the TME by inhibiting tumor stroma, normalizing vasculature and enhancing drug delivery to the tumor in vivo, thereby resulting in improved therapeutic response of PDAC.

S018 TUMOR ASSOCIATED FIBROBLASTS PROMOTE PANCREATIC TUMOR PROGRESSION AND CHEMoresISTANCE THROUGH A POTENTIAL C-MET DEPENDENT-ID1 SIGNALING AXIS

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INTRODUCTION: Previously, we demonstrated that inhibitor of differentiation-1 (Id1) promotes tumorigenesis and chemoresistance in pancreatic cancer through a Src-dependent signaling axis. Further, exposure to tobacco components, which are a significant risk factor for pancreatic cancer, promote these tumorigenic properties through similar mechanisms. Recent studies have determined that the c-Met tyrosine kinase receptor is a marker of human pancreatic cancer stem cell function and c-Met mediated activation of Src support tumor growth and metastatic potential in a variety of solid organ tumors. Unfortunately, most in vivo models of pancreatic cancer utilize murine hepatocyte growth factor (HGF), the ligand of c-Met, which does not bind to or activate human c-Met. This limitation of the current models of pancreatic adenocarcinoma (PC) may underestimate the HGF/c-Met signaling pathway on PC biology, or the potential therapeutic effect of inhibiting this signaling route. Therefore, we sought to examine how pancreatic tumor associated fibroblasts (TAFs) might activate the c-Met receptor and downstream signaling mechanisms to promote tumor progression and chemoresistance.

METHODS: Under IRB approval, pancreatic tumor samples were collected from human patients at time of surgical resection; these tumors were harvested and cultured for isolation of TAFs. Protein expression of c-Met, Id1, phospho-Akt, human HGF and mRNA expression for alpha5, alpha7, alpha9 and beta3 nAchR were determined by Western and RT-PCR analyses respectively. Co-culture experiments of TAFs and PC cells were performed with cell-culture inserts. Viability was assessed by MTT. Metastatic pancreatic cancer cells were stably transfected with a luciferase-expressing vector, and utilized in an orthotopic
xenograft model, monitoring for in vivo growth and metastasis by luminescent signal concentration. Confirmation of primary tumor and metastases was achieved by H&E and IHC techniques.

RESULTS: HGF production is significantly higher in TAFs than human PC cells. Cultured media from TAFs induce c-Met and Id1 protein expression as well as activation of Src tyrosine activity and Akt at higher levels than recombinant human HGF in PC cell lines. Cultured media from TAFs also induced a more gemcitabine resistant phenotype in an otherwise chemosensitive pancreatic cancer cell line. Co-inoculation of TAFs and PC cells in an orthotopic xenograft model resulted in a more aggressive phenotype in vivo based on tumor growth and luciferase bioluminescence. Additionally, nicotine, the addictive component of tobacco, induced c-Met and Id1 protein expression, and also activated the PI3k/Akt pathway in pancreatic cancer cells. Interestingly, TAFs express alpha5-nAchR nicotinic receptors while only PC cells express the alpha7-nAchR.

CONCLUSION: Taken together, expression of HGF and other cytokines from human derived TAFs are activating a variety of signaling mechanisms which result in a more aggressive and chemoresistant pancreatic cancer phenotype. Nicotine might be supporting the role of the tumor microenvironment on the aggressive phenotype of pancreatic cancer by activation of the alpha5-nAchR on TAFs. Our models of harvesting tumor-associated fibroblasts from a bio-diverse sample of resected human pancreatic tumors allow us to adequately study the tumor promoting effects of TAFs’ HGF production on c-Met activation in PC cells. These studies will allow for future delineation of this signaling cascade.

S019 BIOPHYSICAL MARKERS DERIVED FROM STANDARD PRE-TREATMENT IMAGING QUANTITATIVELY DESCRIBE GEMCITABINE DELIVERY AND CHEMORADIATION RESPONSE IN HUMAN PANCREATIC ADENOCARCINOMA

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BACKGROUND: The therapeutic resistance of pancreatic ductal adenocarcinoma (PDAC) is partly ascribed to ineffective chemotherapy delivery to cancer cells owing to physical barriers of the tumor, including disorganized vasculature, characteristically dense stroma, and deregulated cellular transport proteins. We hypothesized multi-scale transport phenomena would influence delivery of and response to gemcitabine-based therapy in human PDAC.

METHODS: We used the measurements obtained during the pancreatic protocol CT in a mathematical model to yield phenomenological parameters of mass transport that describe qualities of the tissue and its surrounding vasculature. Using these methods, we analyzed pancreatic protocol CTs from 171 patients with localized primary PDAC who had received pancreatic protocol CTs prior to initiation of therapy. To study the transport-related factors that influence the delivery of systemic gemcitabine into pancreatic tumors, we conducted a first-in-kind, prospective “phase 0” clinical trial in which gemcitabine was intravenously
infused during resection for 12 patients with localized primary PDAC. After the resection, specimens were collected for pathological analysis. We then tested the hypothesis that the variability of gemcitabine incorporation in tumors within individual patients could be explained by mass transport phenomena.

RESULTS: Here we show how mathematical modeling of tumor density changes during contrast-enhanced computed tomography (CT) scans can describe gemcitabine incorporation and pathological response to therapy. Transport modeling of 171 CT scans illustrated striking differences between normal pancreas and tumor (2-10 fold differences in transport parameters). Moreover, PDAC fibrosis score directly correlated with CT-derived parameters in accord with their mathematical definitions. A prospective “phase 0” trial of gemcitabine infusion during PDAC resection for 12 patients demonstrated 6-fold differences in tumor gemcitabine incorporation, despite similar intravascular pharmacokinetics. Gemcitabine incorporation by tumor cells was inversely related to CT-derived parameters and to fibrosis score, after accounting for human equilibrative nucleoside transporter (hENT1) levels. Among 105 patients who received preoperative gemcitabine-based chemoradiotherapy, CT-derived parameters correlated with pathological response and survival.

CONCLUSION: CT-derived transport parameters represent quantitative biophysical markers that reflect underlying histopathology and provide clinically-relevant prognostic information to guide cancer management.

S020 IMPLICATIONS FOR PANCREATIC CANCER CELL RESISTANCE AND SURVIVAL: CRITICAL CANCER-RELATED GENES ARE SELECTIVELY REGULATED BY HUR WHEN EXPOSED TO CHEMOTHERAPEUTICS AND NUTRIENT DEPRIVATION Richard A Burkhart, MD, Danielle Pineda, MD, Joseph A Cozzitorto, Charles J Yeo, MD, Jordan M Winter, MD, Judith C Keen, PhD, Jonathan R Brody, PhD Department of Surgery and the Jefferson Pancreas, Biliary and Related Cancer Center, Thomas Jefferson University, Philadelphia, PA

INTRODUCTION/BACKGROUND: Chemotherapeutic stress and nutrient deprivation in the tumor microenvironment can exert ‘selective pressures’ and drive tumorigenesis to develop drug resistant pancreatic cancer cells (e.g., cancer stem cells). Post-transcriptional gene regulation of available mRNA transcripts is a major determinant of the acute stress response and can drastically alter the proteome within a cell. The RNA binding protein HuR (ELAVL1) plays a central role in post-transcriptional regulation of core pancreatic cancer-associated genes. Upon stress, HuR carries mRNA cargo to the cytoplasm where it facilitates processing and protein translation (‘HuR activation’) of pro-survival transcripts. HuR activation occurs in response to various stressors from glucose deprivation to chemotherapeutics. To date, little has been done to determine whether the genes regulated by HuR are conserved across varied stressors. Identifying HuR’s unique mRNA cargo under different stressors may unravel how lethal cancer cells develop chemotherapeutic resistance and thrive.

METHODS: Dose and time under each of four stressors (gemcitabine, tamoxifen,
Death Receptor 5 agonist: DR5A, and glucose deprivation) was optimized for maximal HuR activation as defined by immunoblots detecting cytoplasmic HuR. Ribonucleotide-immunoprecipitation (RIP) assays were performed with an anti-HuR antibody to isolate mRNA transcripts bound to HuR under each stress and in a control non-stressed condition. RNA transcripts for approximately 240 cancer-specific gene targets were quantified using nCounter® technology (Nanostring, Inc.), allowing for resolution at the level of a single transcript. For each condition, an HuR target profile was created based on the number of unique mRNA transcripts and the relative quantity of shared mRNA transcripts.

RESULTS: Of the 240 gene targets evaluated with Nanostring technology, approximately 25% of the targets bound to HuR with high affinity when compared to the background expression profile of the cell (IgG immunoprecipitation). As a positive control, known HuR targets p53 and HiF1α were enriched in all HuR-RIP samples. Novel targets identified in this screen include the cell-surface glycoprotein CD44 (a marker of cancer stem cell biology) and the anti-apoptotic regulator BIRC5 (highly expressed in pancreatic cancer). There were three distinct HuR-bound mRNA target profiles identified. One occurred under control (non-stressed) conditions. Comprising a second profile, HuR-bound mRNA under tamoxifen, DR5A, and glucose deprivation were virtually identical. Interestingly, a third unique HuR-regulated mRNA profile was identified under gemcitabine stress.

DISCUSSION/CONCLUSION: An efficient response to acute cellular stress is imperative for pancreatic cancer cell survival in harsh tumor microenvironments. Here we show that the HuR survival response is nuanced and determined by the stressor that activates the protein. The network of regulated gene targets can be similar between specific stressors (as in DR5, tamoxifen, and glucose deprivation) or unique (as in gemcitabine). We are the first to report that HuR regulates several critical pancreatic cancer-related genes (CD44, BIRC5). Taken together, these data define target genes that are the backbone for pancreatic cancer cell viability under the clinically relevant stress present in the tumor microenvironment. Ongoing work continues toward development of a novel drug-discovery pipeline targeting this unique pro-survival network in pancreatic cancer.

S021 CXCR2 INHIBITION PROVIDES PROTECTION AGAINST METASTASES IN PANCREATIC DUCTAL ADENOCARCINOMA Colin Steele, MD, Jennifer Morton, PhD, Colin McKay, MD, Jeffry Evans, MD, Ross Carter, MD, Owen Sansom, PhD Beatson Institute for Cancer Research, Glasgow, UK. West of Scotland Pancreatic Unit, Glasgow Royal Infirmary, Glasgow, UK

INTRODUCTION: Current therapies for pancreatic ductal adenocarcinoma (PDAC) are hindered by advanced stage at presentation, and recurrence following surgery and adjuvant chemotherapy in earlier stage tumors. Recent studies have shown metastases may occur in PDAC even before a primary tumor has formed, highlighting the need for targeted therapy against metastases in addition to surgery to obtain better outcomes for PDAC patients. Metastasis is accelerated
in the presence of pancreatic inflammation, while the most invasive areas of tumour are seen at foci of inflammation. Molecules mediating tumor-stromal crosstalk, including those involved in tumor-associated inflammation, therefore have considerable potential as therapeutic targets in PDAC. CXCR2, a G-protein coupled chemokine receptor, has recently been shown to have a key role in the survival of metastases in breast cancer and is significantly upregulated in PDAC, therefore represents a potential target for treatment of metastases in PDAC.

METHODS: We generated cohorts of the Pdx1-Cre; KrasG12D/+, Trp53R172H/+ (KPC) murine model of PDAC which phenotypically and histologically recapitulates the human disease. We randomized mice to treatment with a CXCR2 targeting peptide 'pepducin', to vehicle treatment, to pepducin in combination with gemcitabine, or gemcitabine alone. Mice were treated from 70 days of age, when histological evidence of PDAC becomes apparent. Additionally we bred Pdx1-Cre; KrasG12D/+, Trp53R172H/+, CXCR2/- and aged the cohort. Mice were culled upon signs of tumor burden. Post-mortem analysis was performed and organs sampled. 3D Organotypic assays were formulated using rat tail collagen and human telomerase immortalized fibroblasts to form discs and then 7 day invasion assays were performed with primary KPC tumor cells.

RESULTS: Quantitative PCR data generated from tumor tissue from KPC mice showed significant upregulation of CXCR2 ligands. Cytokine array confirmed secretion of CXCR2 ligands by KPC cells in culture. When KPC mice were treated with a CXCR2 targeting peptide, pepducin, tumor-free survival was significantly enhanced compared with controls. And while 85% of KPC mice developed metastatic PDAC, CXCR2-targeted pepducin treatment reduced the rate of metastasis to 22%. Treatment had little effect on tumor cell proliferation or differentiation status, however, microvessel density was reduced in pepducin-treated mice, suggestive of lower rates of intratumoral angiogenesis. Examination of immune cell infiltrate demonstrated a decrease in polymorphonuclear leukocytes within treated tumors. Combination of the CXCR2-targeting pepducin with chemotherapeutic gemcitabine provided no additional survival benefit but further inhibited the incidence of metastasis in KPC mice. Furthermore when KPC mice were crossed onto a CXCR2 knockout background (Pdx1-Cre; KrasG12D/+ , Trp53R172H/+ CXCR2/-) metastases were significantly reduced compared to KPC controls. Additionally, 3D organotypic invasion assays demonstrated a significant reduction on the capacity of KPC cells to invade through a collagen matrix upon CXCR2 inhibition.

CONCLUSION: CXCR2 inhibition prolonged survival and decreased number of metastases in KPC mice in our study. When combined, CXCR2 inhibition and gemcitabine treatment abrogated metastases. CXCR2 inhibition in combination with gemcitabine holds promise as a novel chemotherapeutic approach in the treatment of metastases in PDAC.
MULTI-TARGETED APPROACHES IN THE TREATMENT OF PANCREATIC DUCTAL ADENOCARCINOMA (PDAC) Brett L Broussard*, MD, Juan P Arnoletti^, MD, Alevtina Mikhayлина*, MS, Martin J Heslin*, MD, Andrey Frolov*, MD, PhD *Department of Surgery, University of Alabama at Birmingham, Birmingham, Alabama; ^Department of Surgery, Florida Hospital, Orlando, Florida

BACKGROUND: PDAC has a complex tumor biology that involves close interaction between carcinoma cells and stroma under the influence of multiple signaling pathways. We have previously demonstrated the involvement of the ErbB pathway in this tumor-stroma interaction via neuregulin ligand and its ErbB3 receptor, which undergoes heterodimerization with the Epidermal Growth Factor Receptor (EGFR). Additional pathways such as Hedgehog (HH) have also been proposed as key mediators in the PDAC microenvironment signaling mechanisms. We hypothesize that simultaneous inhibition of multiple pathways including both pan-ErbB and HH targeting results in more effective abrogation of PDAC tumorigenesis.

METHODS: AsPC-1, BxPC-3, C3, Panc-1 and Panc-1+ErbB3 (stable ErbB3 transfected cell line) PDAC cells were treated with PF-299 (a pan-ErbB inhibitor), cyclopamine (HH pathway inhibitor), and vismodegib (HH pathway inhibitor). In vitro, proliferation assays were performed and ErbB signaling was analyzed via immunoblot. In vivo, AsPC-1 and BxPC-3 PDAC murine xenografts were developed and treated with PF-299. The PDAC microenvironment was further recreated by establishing AsPC-1/BxPC-3 xenografts in combination with cancer-associated fibroblast (CAFs) and subsequently treating them with PF-299.

RESULTS: In vitro, PF-299, cyclopamine and vismodegib effectively inhibited PDAC cell proliferation alone in a dose dependent manner (1 µM-50 µM). The combination of PF-299 and vismodegib had an additive effect on PDAC cell inhibition of proliferation (p=0.05). Immunoblot analysis showed marked inhibition of EGFR and ErbB3 receptor activation by PF-299 and the addition of vismodegib corroborated an additive effect on inhibition of EGFR, ErbB3, AKT and ERK1/2 targets. In vivo, PF-299 was an effective inhibitor of AsPC-1 and BxPC-3 tumor growth (p=0.04 and p=0.002, respectively). The combination of AsPC-1 and BxPC-3 with CAFs increased tumor volume in vivo and decreased the effectiveness of PF-299 treatment suggesting a role for CAFs in resistance mechanisms to targeted therapy strategies.

CONCLUSIONS: We demonstrated that pan-ErbB inhibition is effective at inhibiting PDAC tumor progression and the inhibition of additional pathways, such as the HH pathway, may provide an additive therapeutic effect. A multi-targeted approach in the treatment of PDAC deserves serious consideration in future studies and may ultimately improve outcomes in PDAC.
S023 DEVELOPMENT OF A MUC1-DRIVEN DIPHTHERIA TOXIN-A NANTHERAPY FOR THE SELECTIVE KILLING OF AGGRESSIVE PANCREATIC CANCER CELLS Renée M Tholey, MD, Richard A Burkhart, MD, Joseph A Cozzitorto, Charles J Yeo, MD, Janet A Sawicki, PhD, Jonathan R Brody, PhD, Jordan M Winter, MD Department of Surgery and the Jefferson Pancreas, Biliary, and Related Cancer Center, Thomas Jefferson University, Philadelphia. Lankenau Institute for Medical Research; Wynnewood

BACKGROUND: A transmembrane protein overexpressed in several cancers, MUC1 has been associated with facilitating the epithelial-to-mesenchymal transition which enhances the invasiveness of tumor cells. In pancreatic ductal adenocarcinoma (PDA), we recently validated MUC1 as a biomarker predictive of early patient death (PLoS One. 2012;7,8) observing that MUC1 status was found to be superior to pathologic features in a series of patient samples. With little to no expression in adjacent normal tissues, MUC1 represents an ideal potential therapeutic target. We have also designed an innovative approach of administering a suicide gene (diphtheria toxin-A) via a nanoparticle based delivery to mesothelin-positive PDA cells (Cancer Biol Ther. 10:1584-90). In this study, we plan to optimize this approach by using a systematic delivery system and the MUC1 promoter that is active in the most aggressive forms of PDA cells.

METHODS: Endogenous MUC1 levels were surveyed in seven human pancreatic cancer cell lines (BxPC3, Capan2, Hs766T, MiaPaCa2, PL45, PL5, and Su.86.86). Availability of the MUC1 RNA transcript was quantified by quantitative polymerase chain reaction (q-PCR) of whole cell RNA isolates. Protein levels were evaluated by immunoblot of protein lysates. Subsequently, the human MUC1 promoter was subcloned from the pDRiVE vector. In brief, following PCR amplification and purification, the promoter region was spliced into a PgL4 luciferase vector. Sequencing analysis was performed to ensure accuracy of the transcript. Using lipofectamine, pMUC1-luciferase was transfected to target MUC1-positive and negative (control) expressing pancreatic cancer cell lines. As expected based on our q-PCR results, we found that Capan2 displayed high luciferase activity, while Su.86.86 displayed low activity. In addition, plasmids have been created incorporating the suicide gene diphtheria toxin-A (DT-A), as well as luciferase for detection, downstream of the MUC1 promoter. These will be transfected using lipofectamine and then using a novel, biodegradable nanoparticle.

RESULTS: Similar to results found in patient tumor samples, MUC1 expression levels varied widely across pancreatic cancer cell lines. Capan2 cells (the highest expression line) had approximately 18 times the amount of MUC1 as Su.86.86 cells (the lowest expression line). As expected, upon lipofectamine transfection of pMUC1-luciferase, high MUC1 expressing pancreatic cancer cell lines (Capan2) produced more luciferase than cell lines without MUC1 (Su86.86). The addition of diphtheria toxin-A to our constructs downstream of the MUC1 promoter should act as a potent suicide gene only in those cell lines that express MUC1, and thus will provide the rationale for personalized therapy (for MUC1-expressing tumors only).
DISCUSSION: MUC1 is an available target in human pancreatic cancer as evident by the above described cell line work and patient PDA immunohistochemical studies. Further, the promoter region can be accurately transformed into a plasmid vector. Pairing a cancer-specific promoter (such as MUC-1) with a diphtheria toxin-A transcript into a nanoparticle delivery system can effectively and selectively inhibit the growth of MUC1-positive PDA cells for a novel personalized therapy. Development of a systematic delivery dendrimer is underway to enhance the targeted delivery of these constructs to metastatic PDA cells.

S024 INHIBITION OF CENTROSOME DUPLICATION AS A THERAPEUTIC APPROACH TO PANCREATIC CANCER WITH POTENTIALLY FEW SIDE EFFECTS

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BACKGROUND: The majority of chemotherapeutic drugs target DNA replication, effectively killing rapidly dividing cancer cells, as well as rapidly dividing normal cells. Damage to the normal tissues with rapidly dividing cells, such as hair follicles, intestinal epithelium, and bone marrow, leads to common side effects such as alopecia, diarrhea, and hematologic disorders including anemia. Centrosomes duplicate semi-conservatively: once per cell cycle, as chromosomes do. In normal cells, centrosome duplication is always coupled with DNA duplication. However, in most cancer cells, centrosome duplication is uncoupled from DNA duplication, leading to centrosome overamplification and aneuploidy. Recently, we found that the inhibition of centrosome duplication in cancer cells by depleting a critical centrosomal protein, centrobin, lead to cells with three, one or no centrioles, and eventually to cell death; while in normal cells, inhibition of centrosome duplication leads to cells with unduplicated centrioles and cell cycle arrest, but not to cell death. Therefore, we hypothesize that the inhibition of centrosome duplication can be a novel therapeutic approach for cancers with potentially few side effects, since normal cells would still be functional while in G1 arrest and would resume their proliferation when centrosome duplication inhibitors decay. Additionally, the inhibition of centrosome duplication would not cause heritable DNA mutations leading to further tumorigenesis transmittable to progeny.

METHODS: We first examined the effects of inhibiting centrosome duplication on pancreatic cancer cells using a centrobin RNAi. A panel of pancreatic cancer cell lines and an untransformed pancreatic epithelial cell line (HPDE6C7) were transfected with control or centrobin RNAi to inhibit the centrosome duplication. The cell viability was then assessed using MTT assay. We recently found that centrobin-tubulin interaction is required for centriole elongation and stability and that disruption of this interaction can inhibit centrosome duplication. We have localized the tubulin binding domain of centrobin to a region of 129 amino acids. To be able to examine the therapeutic effect of inhibiting centrosome duplication in vivo, we developed a set of six peptides fused with protein transduction
domain based on the tubulin binding domain. These peptides can potentially disrupt the centrobin/tubulin interaction and inhibit centrosome duplication. We tested the effect this set of peptides on the viability of the pancreatic cell lines.

RESULTS: We found that inhibition of centrosome duplication by centrobin depletion using RNAi induced extensive cell death of pancreatic cancer cells while inhibition of centrosome duplication had a significantly less effect on the viability of untransformed pancreatic epithelial cells. Treatment of pancreatic cancer cells with a set of six peptides fused with protein transduction domain revealed three of these peptides can induce cell death of pancreatic cancer cells but not that of the untransformed pancreatic epithelial cells.

CONCLUSION: Inhibiting centrosome duplication can be a potential therapeutic approach for pancreatic cancer. We are in the process of developing reagents to perform animal studies to further evaluate this therapeutic approach for pancreatic cancer.

S025 PINCH EXPRESSION IN PANCREATIC NEUROENDOCRINE TUMORS
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INTRODUCTION: PINCH is an integrin-associated protein that has been found to be upregulated in multiple malignancies, including pancreatic cancer. PINCH has been shown to be highly expressed at the boundary between tumor and stromal cells and higher PINCH expression correlates with poorer survival. There is no published data regarding PINCH expression in pancreatic neuroendocrine tumors.

METHODS: PINCH expression was assessed immunohistochemically in the tumor specimens of 20 patients with pancreatic neuroendocrine tumors. PINCH expression was examined in the tumor cells as well as the tumor-associated stroma and quantified in 2 ways: as a percentage of cells staining positive and by the intensity of staining. Percent cell staining was graded on a 1-4 scale with 1 indicating 0-25% staining, 2 26-50% staining and so on. Staining intensity was graded on a 0-3 scale with 0 indicating no staining, 1 mild, 2 moderate and 3 strong intensity staining. PINCH expression was correlated to clinicopathological characteristics.

RESULTS: PINCH staining and intensity was statistically higher in the tumor-associated stroma in specimens from patients with stage II or higher disease compared to those with stage IA or IB disease (p<.05). Patients with stage IA and IB tumors had mean staining of 2.53 ± 1.13, correlating to approximately 50% of cells staining, with a mean intensity of staining of 1.53 ± 0.66 while those with stage II or higher tumors had mean PINCH staining of 3.57 ± 0.78, correlating to approximately 75% of cells staining, with a mean intensity of 2.43 ± 0.5. In contrast, PINCH expression in the tumor cells was minimal and of lower intensity with a mean percent staining score of 1.27 ± 0.88 and a mean intensity staining of 0.22 ± 0.4.

CONCLUSIONS: PINCH is more highly expressed in the tumor-associated stroma of patients with higher stage pancreatic neuroendocrine tumors. It is worth investigating PINCH expression in larger patient populations.
INTRODUCTION: The first successful local resection of a periampullary tumor was performed by Halsted in 1898. Kausch performed the first regional resection in 1909, and the operation was popularized by Whipple in 1935.

METHODS: 2000 consecutive pancreaticoduodenectomies performed by one surgeon from the 1960s to the 2000s were retrospectively reviewed from a prospectively maintained database. Changes in indications and outcomes were assessed.

RESULTS: The most common indication throughout was adenocarcinoma of the head of the pancreas (PDAC) - (46%). Benign IPMN increased from 1% (1990s) to 8% (2000s)(p=0.002). Age increased from 59 years (1980s) to 66 (2000s) (p=0.001), as did those over 80 (3% to 12%, p=0.002). 30 day mortality was 1.4%; hospital mortality was 1.7%. Median number of blood transfusions decreased from 2 (1980s) to 0 (1990s and 2000s) (p=0.004). Length of stay decreased from 21 days (1980s) to 13 (1990s) days to 10 days (2000s) (p=0.002). 5-years survival for PDAC increased from 19% (1990s) to 24% (2000s) (p=0.02). 5-year survival for node-negative, margin negative PDAC patients was 39%.

CONCLUSION: The volume of pancreatic pathology has attracted 22 basic and clinical scientists to Hopkins, who have 28.5 million dollars of direct support and over 30 million dollars in endowments, to support research in pancreatic cancer. The volume of clinical material has also supported the training of many young surgeons, 15 of whom have become Department Chairman, and over 20 have become Division Chiefs.

S027 A MULTI-INSTITUTIONAL EXTERNAL VALIDATION OF THE FISTULA RISK SCORE FOR PANCREATICODUODENECTOMY Benjamin C Miller, BA, John D Christein, MD, Stephen W Behrman, MD, Jeffrey A Drebin, MD, PhD, Wande B Pratt, MD, MPH, Mark P Callery, MD, Charles M Vollmer, MD Hospital of the University of Pennsylvania, University of Alabama, Birmingham Medical Center, University of Tennessee Health Science Center, Beth Israel Deaconess Medical Center

BACKGROUND: Accurate prediction of postoperative pancreatic fistula (POPF) after pancreaticoduodenectomy (PD) would help tailor optimal intra- and postoperative management of this morbid complication. Distinct risk factors for ISGPF clinically relevant fistulas (CR-POPF), previously identified as small duct size, soft gland texture, high-risk pathology, and increased blood loss, are best discerned intraoperatively. The Fistula Risk Score (FRS), a 10 point scale derived at a single institution, relies on weighted influence of these four variables and has been shown to effectively predict (area under the curve of 0.942) CR-POPF development and its consequences. External validation of this tool would confirm its universal applicability.
METHODS: From 2001-2012, 594 PDs with pancreatojejunostomy reconstruction were performed by four pancreatic surgical specialists at three institutions. POPFs, when they occurred, were graded by ISGPF standards as biochemical (Grade A) or clinically relevant (Grades B and C). The FRS was calculated for each patient and clinical outcomes were evaluated across four discrete categories (Negligible Risk, 0 points; Low Risk, 1-2 points; Moderate Risk, 3-6 points; High Risk, 7-10 points). Receiver operator curve analysis was performed to judge model validity.

RESULTS: 142 patients developed any sort of POPF, of which 68 were CR-POPF (11.4% overall: 8.9% Grade B, 2.5% Grade C). There were 21 overall deaths, six of which were directly attributable to pancreatic fistula. Increasing FRS scores (0-10) correlated well with CR-POPF development (p<0.001), with an area under the curve of 0.716. When segregated by FRS risk groups, CR-POPFs occurred in Low, Moderate and High Risk patients 6.6%, 12.9% and 28.6% of the time respectively (figure). Clinical outcomes including complications, length of stay, and readmission rates, also increased across risk groups (table).

CONCLUSION: This multi-institutional experience confirms the Fistula Risk Score as a valid tool for predicting the development of CR-POPF in patients undergoing pancreaticoduodenectomy. Patients devoid of any risk factors did not develop a CR-POPF, and the rate of CR-POPF approximately doubles with each subsequent risk zone. The lower value of the area under the curve in this analysis is attributable to the decreased rate of CR-POPF observed in the high risk group (29% vs. 89% originally). This difference might be ascribed to variations in operative technique, postoperative management styles, patient characteristics, and a larger sample size in the current study. Despite this, the FRS is validated as an accurate prediction tool, with widespread applicability, which can be readily translated into common practice.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Negligible Risk (0 points)</th>
<th>Low Risk (1-2 points)</th>
<th>Moderate Risk (3-6 points)</th>
<th>High Risk (7-10 points)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, n (% total)</td>
<td>63 (10.6)</td>
<td>166 (27.9)</td>
<td>302 (50.9)</td>
<td>63 (10.6)</td>
<td>-</td>
</tr>
<tr>
<td>CR-POPF, n (%)</td>
<td></td>
<td>11 (6.6)</td>
<td>39 (12.9)</td>
<td>18 (28.6)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Grade B</td>
<td></td>
<td>9 (5.4)</td>
<td>29 (9.6)</td>
<td>15 (23.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Grade C</td>
<td></td>
<td>2 (1.2)</td>
<td>10 (3.3)</td>
<td>3 (4.8)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Any complication, n (%)</td>
<td>32 (50.8)</td>
<td>97 (58.4)</td>
<td>216 (71.5)</td>
<td>54 (85.6)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Length of stay, median</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Readmission, n (%)</td>
<td>10 (15.9)</td>
<td>24 (14.5)</td>
<td>51 (16.9)</td>
<td>21 (33.3)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
OBJECTIVES: Robotic-assisted minimally invasive surgery allows complex resections and anastomotic reconstructions to be performed with identical standards to open surgery. For the last four years we have applied this technology to a wide variety of major pancreatic resections in order to assess the safety, utility and efficacy of this platform.

METHODS: A retrospective review of a prospectively maintained database of robotic assisted pancreatic resections at a single institution between August 2008 and November 2012 was performed. Preoperative, operative, pathologic, and post-operative outcomes were analyzed.

RESULTS: 250 consecutive robotic assisted major pancreatic resections were analyzed; pancreaticoduodenectomy (PD=132), distal pancreatectomy (DP=83), central pancreatectomy (CP=13), pancreatic enucleation (10), total pancreatectomy (TP=5), Appleby resection (4), and Frey procedure (3). Median age was 65 and 51% of patients were female. Median BMI was 27.5. 52% of patients had prior abdominal surgery. Indication for resection included pancreatic adenocarcinoma (n=77, 31%), periampullary carcinoma (43, 17%), neuroendocrine tumor (58, 23%), premalignant (52, 21%), and benign conditions (20, 8%). Mean operative time for the two most common procedures was 529 ± 103 mins for PD and 257 ± 93 mins for DP (last 50 PD: 444 +/- 76 mins; DP: 222 +/- 73 mins). Conversion to an open procedure was required in 16 patients (6%; 11 PD, 2 DP, 2 CP, 1 TP) for failure to progress (14) and bleeding (2). 90 day mortality was 1.8% (4) for the 225 subjects with at least 90 day follow up.

CONCLUSIONS: This represents to our knowledge the largest known series of robotic assisted pancreatic resections. The safety metric outcomes, including the low incidence of conversion, support the robustness of this platform when applied to a broad range of pancreatic procedures.
A STANDARDIZED RADIOGRAPHIC ASSESSMENT OF THE TUMOR-VEIN INTERFACE PREDICTS THE NEED FOR VENOUS RESECTION AND THE PRESENCE OF HISTOLOGIC VENOUS INVASION IN BORDERLINE RESECTABLE PANCREATIC CANCER

Hop S Tran Cao, MD, Aparna Balachandran, MD, Huamin Wang, MD, PhD, Jason B Fleming, MD, Jeffrey E Lee, MD, Peter W Pisters, MD, Matthew H Katz, MD

Department of Surgical Oncology, U.T. M.D. Anderson Cancer Center; Department of Diagnostic Radiology, U.T. M.D. Anderson Cancer Center; Department of Pathology, U.T. M.D. Anderson Cancer Center

BACKGROUND: Venous resection may be required to achieve complete resection of pancreatic cancers (PC). We have previously shown that histologic invasion of the superior mesenteric vein-portal vein (SMV-PV) is associated with poor prognosis following resection. Using high-definition multidetector computed tomography (CT), we sought to evaluate the ability of two commonly-used sets of radiographic criteria to predict the need for SMV-PV resection at pancreatectomy and the histologic presence of SMV-PV invasion.

METHODS: All patients who underwent pancreaticoduodenectomy for PC between 2004 and 2011 at the authors’ institution were identified. Preoperative pancreatic protocol CT images were re-reviewed to characterize the interface between the tumor and SMV-PV (no interface, abutment [≤180 degrees], encasement [>180 degrees], occlusion) and the appearance of the SMV-PV using Ishikawa criteria (normal, smooth shift, unilateral narrowing, bilateral narrowing without collaterals, bilateral narrowing with collaterals). Findings were correlated to the need for venous resection at pancreatectomy and to the presence of histologic venous invasion.

RESULTS: 266 patients underwent pancreaticoduodenectomy and met inclusion criteria, of whom 99 required concomitant resection of the SMV-PV. The greatest sensitivity for predicting SMV-PV resection was achieved by an interface threshold of abutment (sensitivity 91.9%, negative predictive value 87.9%), whereas excellent specificity was reached with a threshold of encasement (97.6%, positive predictive value 89.7%). Among patients who underwent venous resection, vessel encasement was associated with a 78.3% rate of histologic SMV-PV invasion; this rate increased to 90% when the vein was occluded. The Ishikawa system, while more detailed, offered no advantage in predicting the need for SMV-PV resection and was less accurate in predicting histologic venous invasion. Subset analyses performed for patients who received neoadjuvant chemoradiation and for those who did not yielded similar findings.

CONCLUSIONS: A simple radiographic classification system that categorizes the extent of the tumor-SMV-PV interface accurately predicts the need for SMV-PV resection at pancreatectomy, and correlates with the pathologic involvement of the resected vein. To assist in treatment planning, a standardized description of this anatomic relationship should be routinely performed for patients with borderline resectable tumors.
INTRODUCTION: Negative margins are the goal with pancreaticoduodenectomy for pancreatic adenocarcinoma. Thereby, margins are assessed intraoperatively with frozen-section analysis and negative margins are chased. This study was undertaken to determine the impact of margin status with pancreaticoduodenectomy for pancreatic adenocarcinoma and the value of additional resections to achieve negative margins.

METHODS: The intraoperative frozen-section analysis and final margins for 448 patients undergoing pancreaticoduodenectomy for pancreatic adenocarcinoma were assessed and their impact on survival was determined. Median data are presented.

RESULTS: 298 (67%) patients had negative margins (R0), an additional 110 (25%) patients had microscopically positive and macroscopically negative margins (R1), and an additional 40 (9%) patients had initially positive microscopic margins which became negative with further resection (R1→R0). R0 resections were more likely to have smaller tumors, earlier T stage, earlier N stage, lower AJCC stage, and less frequent extrapancreatic extension (p≤0.03 for each). Survival was better with R0 resections than R1 resections (20 months vs. 12 months, p<0.001); extending resections to achieve negative margins (i.e., R1→R0) did not improve survival beyond R1 resections (14 months vs. 12 months, p=0.19) (Figure 1). For patients undergoing R1 or R1→R0 resections, local recurrence did not generally presage metastatic disease.

CONCLUSIONS: Survival after pancreaticoduodenectomy is disappointing. Patients with initial negative margins do best. Positive microscopic margins reflect more aggressive tumor-specific factors and lead to abbreviated survival because of metastatic disease, even with additional extended resections to achieve negative margins (i.e., R1→R0). With an initial positive margin, pursuing negative margins does not improve survival and, thereby, negative margins should not be “chased.”
A SINGLE CENTER EXPERIENCE OF 129 PANCREATIC ENUCLEATIONS: INDICATIONS, SHORT AND LONG-TERM OUTCOME

Sebastien Gaujoux, Francois Faitot, Safi Dokmak, Benjamin Blanc, David Fuks, Philippe Ruszniewski, Jacques Belghiti, Alain Sauvanet

Department of HPB Surgery – PMAD - Hopital Beaujon - AP-HP - Clichy, France

BACKGROUND: The widespread use of cross-sectional imaging has led to an increased diagnosis of benign or low-grade pancreatic neoplasms. Their standard resection (pancreaticoduodenectomy or distal pancreatectomy) is associated with significant postoperative morbidity and disappointing functional results. This drawback could be overcome by parenchyma-sparing pancreatectomies, including enucleation, but results of this procedure have been poorly evaluated. This study was undertaken to assess short and long-term outcome of a large unicentric series of pancreatic enucleations for benign and low-grade neoplasms, with a specific attention to the incidence and risk factors of pancreatic fistula (PF).

METHODS: Were included all 126 patients undergoing pancreatic enucleation in our department between 1996 and 2010. Demographic, radiologic, operative and pathologic data were obtained from a prospective database with additional retrospective medical record review.

RESULTS: Patients were mainly women (65%), with a median age of 50 years, with incidentally diagnosed lesion (71%). Lesions were most often located in the head and uncinate process (46%), with a median size of 20 mm. Enucleation were mainly performed for branch-duct IPMN (30%), non-functioning pancreatic neuroendocrine tumors (27%) and mucinous cystadenoma (9%). Overall mortality was 0.8% and morbidity was 63%, mainly due to minor complications, i.e. Dindo-Clavien grade 1 or 2 (48%). Reoperation rate was 3%, mainly due to hemorrhage after PF. PF rate was 57%, and the main cause of postoperative morbidity. Most of PF (72%) were clinically significant i.e. grade B and C, but managed conservatively in 85% of cases. The only independent risk factor of PF was a BMI over 30kg/m2. After a median follow-up of 23 (8-48) months, de novo diabetes was appeared in 2% of patients, whereas optimization of preoperative anti-diabetic therapy was needed in 3% patients. Exocrine insufficiency was observed in 1%. One, 3 and 5-year disease-free survival were 100%, 98% (89-99) and 93% (80-98) respectively.

CONCLUSION: Pancreatic function both exocrine and endocrine following enucleation is excellent at the expense of a high morbidity, especially PF, underestimated by the published literature. Enucleation, as an alternative to standard resection, are best indicated for benign or low-grade lesions in young and fit patients able to tolerate postoperative morbidity and who could benefit from the excellent long-term results.
The Puestow procedure is a well-established intervention in the management of chronic pancreatitis. Little has been written about results of pancreatico-jejunostomy limited to the body and tail of the pancreas. We postulate that two

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trends in pancreatic diseases over the past two decades have led to a rise in the number of patients who are candidates for this procedure. The first of these is the rising survival rates in patients who have had an episode of moderate/severe acute necrotizing pancreatitis with accompanying pancreatic duct disruption and the other is the high number of pancreaticoduodenectomies performed for IPMN in patients with normal (non-dilated) pancreatic ducts leading to postoperative anastomotic stricture. Patients present with imaging evidence of a dilated pancreatic duct in the body/tail of the pancreas or in the pancreatic remnant often with associated pseudocyst. The typical patient has had many hospitalizations, imaging studies and visits to additional physicians, including surgeons before the diagnosis is established. Nearly all records made note only of the pseudocyst.

Methods: Between 1992 and 2012 all patients undergoing pancreatico-jejunostomy to the body/tail were prospectively followed. Patients were categorized for the cause of ductal dilatation. Note was taken of the predominant symptom as well as number of clinical encounters prior to diagnosis and intervention.

CONCLUSION: We conclude that physician and surgeon awareness of this emerging clinical syndrome is required. Operative drainage is effective in resolving pain, pseudocyst, inability to eat, and associated episodes of pancreatitis.

RESULTS

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Post-Whipple</th>
<th>Post-Pancreatitis</th>
<th>Post-Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>90 Patients</td>
<td>34/90 (38%)</td>
<td>47/90 (52%)</td>
<td>9/90 (10%)</td>
</tr>
<tr>
<td>Recurrent Pancreatitis</td>
<td>46/90 (51%)</td>
<td>11/34 (38%)</td>
<td>27/47 (57%)</td>
<td>8/9 (89%)</td>
</tr>
<tr>
<td>Pain</td>
<td>86/90 (96%)</td>
<td>31/34 (91%)</td>
<td>46/47 (98%)</td>
<td>9/9 (100%)</td>
</tr>
<tr>
<td>Pseudocyst/Fluid Collection</td>
<td>71/90 (79%)</td>
<td>21/34 (62%)</td>
<td>44/47 (94%)</td>
<td>6/9 (100%)</td>
</tr>
<tr>
<td>Compromised Nutritional Intake</td>
<td>43/90 (48%)</td>
<td>15/34 (44%)</td>
<td>26/47 (55%)</td>
<td>2/9 (22%)</td>
</tr>
<tr>
<td>Time from First Imaging to Surgery</td>
<td>3.1 +/- 1.1 years</td>
<td>4.2 +/- 1.3 years</td>
<td>2.9 +/- 1.4 years</td>
<td>1.1 +/- 0.7 years</td>
</tr>
<tr>
<td>Physicians seen from Index Imaging to Surgery</td>
<td>8.6 +/- 2.6 years</td>
<td>7.2 +/- 3.1 years</td>
<td>8.9 +/- 3.6 years</td>
<td>2.7 +/- 0.9 years</td>
</tr>
<tr>
<td>Resolution of Symptoms after Pancreatico-jejunostomy</td>
<td>86/90 (96%)</td>
<td>32/34 (94%)</td>
<td>45/47 (96%)</td>
<td>9/9 (100%)</td>
</tr>
</tbody>
</table>

S034 PERCUTANEOUS DRAINAGE OF PANCREATIC NECROSIS- BEYOND THE PANTER TRIAL Greggory S Flint, Cody J Boyce, MD, John C Kirkham, MD, Sean M Carr, MD, Brent D Nelson, MD, Don A Bell, MD, R. Taylor Handley, MD, Stephen M Schutz, MD, Joshua G Barton, MD, L. William Traverso, MD St. Luke’s Health System

INTRODUCTION: In 2010, the PANTER trial (NEJM 2010; 362: 1491-1502) showed a reduction in deaths and major complications by using a “step-up” approach among patients with necrotizing pancreatitis. The first “step” was percutaneous drainage (PCD) which occurred in almost all patients (93%); one-third of these patients required only PCD. What is the outcome if only PCD is utilized?
METHODS: Between August, 2010 and November, 2012 we used PCD as the primary treatment in 32 consecutive cases of severe acute pancreatitis, 16 (50%) of which had pancreatic necrosis and were treated solely with an aggressive PCD protocol. We compared our outcomes to that of the PANTER trial using their definitions and methods.

RESULTS:

<table>
<thead>
<tr>
<th>PATIENTS WITH NECROSIS</th>
<th>SLHS (N=16)</th>
<th>PANTER (N=43)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>52 ± 15</td>
<td>58 ± 2</td>
</tr>
<tr>
<td>Max modified CTSI (median) [range]</td>
<td>10 [6-10]</td>
<td>8 [4-10]</td>
</tr>
<tr>
<td>Max CRP (normalized to mg/L)</td>
<td>224 ± 134</td>
<td>213 ± 106</td>
</tr>
<tr>
<td>MOF before treatment</td>
<td>38%</td>
<td>35%</td>
</tr>
<tr>
<td>Days in ICU (median) [range]</td>
<td>2 [0-105]</td>
<td>9 [0-281]</td>
</tr>
<tr>
<td>Days in Hospital (median) [range]</td>
<td>36 [5-206]</td>
<td>50 [1-287]</td>
</tr>
<tr>
<td># of CT scans (mean)</td>
<td>13</td>
<td>Not Stated</td>
</tr>
<tr>
<td>Mean # of percutaneous drain sites per patient</td>
<td>2.6</td>
<td>1.9</td>
</tr>
<tr>
<td>Operations (% of patients) -VARD or necrosectomy</td>
<td>0%</td>
<td>60%</td>
</tr>
<tr>
<td>Death</td>
<td>0%</td>
<td>19%</td>
</tr>
</tbody>
</table>

MOF= multiple organ failure of ≥ 2 organ systems

DISCUSSION: We were able to avoid VARD (video-assisted retroperitoneal debridement) or open necrosectomy while using only percutaneous drainage in 16 consecutive cases of pancreatic necrosis that had similar severity scores as the PANTER trial. With this treatment protocol we achieved a zero death rate and a shorter length of ICU and hospital stay.

CONCLUSION: A dedicated multispecialty team using aggressive percutaneous drainage can provide primary treatment of pancreatic necrosis.

2:30pm – S035 EVOLVING TREATMENT STRATEGIES IN THE ENDOSCOPIC MANAGEMENT OF WALLED-OFF PANCREATIC NECROSIS (WOPN) Ji Young Bang^, MBBS, MPH, Muhammad Hasan*, MD, Jayapal Ramesh^, MD, Jessica Trevino^, MD, C. Mel Wilcox^, MD, Robert Hawes*, MD, Shyam Varadarajulu*, MD Center for Interventional Endoscopy, Florida Hospital, Orlando, FL, USA*; Division of Gastroenterology-Hepatology, University of Alabama at Birmingham, Birmingham, AL, USA^  

BACKGROUND: Although endoscopic techniques are increasingly used, the treatment outcomes are suboptimal for patients with WOPN. AIM: Identify factors that improve treatment outcomes in patients undergoing endoscopic drainage of WOPN.

METHODS: This is a retrospective study of patients with WOPN treated endoscopically over 9 yrs. Patients underwent placement of double pigtail stents and nasocystic catheters within the necrotic cavity. In select patients, multiple transluminal gateway technique (MTGT) was adopted to create several openings in the stomach or duodenum to facilitate better drainage. Prior to transmural drainage, an MRCP or ERCP was attempted to evaluate for pancreatic duct leak.
In patients with a disconnected pancreatic duct syndrome (DPDS), the transmural stents were left in place indefinitely to minimize chances of PFC recurrence. A CT of the abdomen was obtained at 8-weeks to assess treatment response. Treatment success was defined as resolution of WOPN with symptom relief at 8-week follow-up. Main outcome measures: Identify factors that (a) predict treatment success and (b) decrease rates of PFC recurrence.

RESULTS: Of 74 patients who underwent endoscopic drainage of WOPN, treatment was successful in 51 (68.9%) patients. Patients treated by MTGT (n=16) had higher treatment success than conventional drainage (n=58) techniques (93.8% vs. 62.1%, p=0.02). On multivariate analysis, only MTGT (adjusted OR= 14.8; 95% CI=1.62-134; p =0.02) and lack of need for multiple endoscopic interventions (adjusted OR=3.97; 95%CI=1.14-13.8; p=0.03) were predictive of treatment success when adjusted for the size of PFC, duration of illness (< or > 4 weeks), WOPN location (head vs. body/tail), CT severity index, white cell count and serum albumin. Of the 51 patients with treatment success, on long-term follow-up, PFC recurrence was encountered in 0 of 27 patients with permanent indwelling transmural stents compared to 5/24 patients who had their stents removed (0% vs. 20.8%, p=0.02). At a mean follow-up of 314 days (SD=124), complications were encountered in 5 of 40 (12.5%) patients with DPDS who had initial treatment success: 2 required total/distal pancreatectomy for persistent symptoms or PFC recurrence and transmural stent migration was encountered in 3 patients.

CONCLUSIONS: While the multiple transluminal gateway technique improves treatment success, the concept of placing permanent indwelling transmural stents decreases the rates of PFC recurrence. Endoscopic techniques designed to provide durable treatment success is required in patients with DPDS given the suboptimal long-term clinical outcomes in these patients.

S036 SALVAGE DUAL MODALITY DRAINAGE FOR PERSISTENT WALLED OFF PANCREATIC NECROSIS ELIMINATES EXTERNAL PANCREATIC FISTULAE BUT DOES NOT REDUCE LENGTH OF HOSPITALIZATION NOR USE OF RADIOLOGIC RESOURCES

Michael Gluck, MD, Flavio G Rocha, MD, Andrew R Ross, MD, Shayan Irani, MD, Seng I Gan, MD, Richard A Kozarek, MD
Virginia Mason Medical Center

BACKGROUND: Walled off pancreatic necrosis (WOPN), a complication of severe acute pancreatitis, has evolved into a disease managed preferentially by minimally invasive techniques. Our group has previously described a hybrid technique of combining percutaneous and endoscopic drainage (DMD) at the onset of treatment that has resulted in avoidance of surgical necrosectomy, elimination of external pancreatic fistulae (EPF), reduction in length of hospitalization, and decreased use of radiological resources. For patients who had standard percutaneous drainage (SPD), a salvage technique (SDMD) was developed for those with persistent fluid collections, large volume pancreatic fistula output, and residual infection. We sought to determine if patients with SDMD differed from those with DMD and if they had equivalent results.
METHODS: We reviewed an IRB-approved, prospective database of all treated WOPN between 1/1/2006 and 11/01/2012. SDMD were defined as those with initial percutaneous drains placed at our institution or with persistent WOPN after surgical necrosectomy at outside facilities. Outcomes between DMD and SDMD groups were analyzed.

RESULTS: Of 152 patients with WOPN, 98 initially had DMD while 44 had SPD. Of those 44, 14 required SDMD for persistent EPF including two who had a previous surgical necrosectomy at an outside facility. Age, gender, choledocholithiasis etiology, and computed tomographic severity index were not different between groups. 72% of SDMD were infected on initial aspirate in contrast to 48% of DMD (p<0.02). SDMD patients had a longer length of stay (48 v 24 days, p<0.02), required more drains (2.5 v 1.4, p<0.01), and had a longer interval of external drainage (162 v 84 days, p<0.03) than those with DMD. SDMD patients had a shorter interval to first drainage than those with DMD (27 v 58 days, p<0.02). There were 4 deaths prior to completion of therapy in the DMD and none in SDMD group. There was a single pseudoaneurysm bleed in the DMD, 5 in the SPD, and none in the SDMD group. No patient in either the DMD or SDMD group needed an operation for EPF, recurrent fluid collections, or persistent necrosis.

CONCLUSIONS: SDMD provides a safe, non-operative alternative for resolving persistently infected, symptomatic WOPN and EPF in patients initially treated with SPD or surgical necrosectomy. DMD provides the greatest reduction in length of stay and radiological resources; however SDMD can be useful early in the course of WOPN that has failed previous treatment.

S037 DOES ACUTE PANCREATITIS CHANGE THE NATURAL HISTORY OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN)? Sejoon Lee*, MD, Joshua A Waters, MD, C M Schmidt, MD, Henry A Pitt, MD, Nicholas J Zyromski, MD Department of Gastroenterology, Yonsei University College of Medicine, Seoul, Korea* and Department of Surgery, Indiana University School of Medicine, Indianapolis, IN

BACKGROUND: Intraductal papillary mucinous neoplasms (IPMN) of the pancreas may cause acute pancreatitis (AP), though the influence of AP on IPMN biology is poorly understood. We therefore compared IPMN patients with and without AP, hypothesizing that AP may change the natural history of IPMN.

METHODS: Clinical, imaging, and histologic data of 278 patients with surgically resected IPMN (March 2002 to March 2010) were collected. Outcomes of IPMN patients with and without AP were compared. Descriptive statistics were applied; p<0.05 was considered significant.

RESULTS: 92 (33%) IPMN patients with AP and 186 (67%) IPMN patients without AP were compared. No differences in sex, diabetes prevalence, or alcohol use were observed between the two groups. Abdominal pain (93% vs. 61%) and smoking (48% vs. 33%) were significantly greater in the IPMN/AP group, (p<0.05), as were the radiologic findings of chronic pancreatitis (82% vs. 2%, p<0.01). Cyst
type (main duct, branch duct, mixed), location (head, body), and number were similar between the two groups. Interestingly, the cyst size of branch duct (BD)-IPMN with AP was smaller than BD-IPMN without AP, (mean, 21 mm vs. 27 mm, p<0.05). IPMN with dysplasia (borderline) were significantly more common in IPMN/AP, (51% vs. 25%, p<0.01), but invasive carcinoma (10% vs. 27%, p<0.01) was significantly more common in IPMN without AP.

CONCLUSIONS: Acute pancreatitis is common (33%) in IPMN. IPMN/AP patients more commonly have abdominal pain, smoking history, and radiologic changes of chronic pancreatitis than those without AP. No differences in cyst type, location or number were seen in IPMN patients with or without AP, though IPMN/AP patients had smaller branch duct cysts. IPMN/AP patients more commonly harbored dysplasia, but had significantly less invasive carcinoma, suggesting that early resection of symptomatic IPMN may interrupt the adenoma to carcinoma progression.

**S038 DOES WEIGHT AFFECT OUTCOMES FOLLOWING TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION?** Stefanie Owczarski, PAC, MPAS, Katherine Morgan, MD, FACS, David Adams, MD, FACS, Kelley Martin, MPH, RD, LD, Hongjun Wang, PhD, Jeffrey Borckardt, PHD, Alok Madan, PHD, Joseph Romagnuolo, MD, MSC, FRCP Medical University of South Carolina

**INTRODUCTION:** The incidence of weight loss following total pancreatectomy with islet autotransplantation for chronic pancreatitis and its effect on insulin requirements and Quality of life (QoL) post-operatively is poorly understood.

**METHOD:** A prospectively collected, IRB approved database at a single institution was reviewed. Patients with a BMI greater than or equal to 25 were compared to those with a BMI less than 25 prior to surgery. Data pertaining islet yield, insulin requirements, laboratory results, and quality of life (QoL) were reviewed at 6 months and 1 year following surgery. The SF12 survey was used to assess QOL (normal population 50, SD 10). QoL is improved if the result increases by at least 3 points or is ≥ 35.

**RESULTS:** 100 consecutive patients were reviewed (78 females, average age 42) from March 2009 to present. 19 patients were omitted due to lack of at least 6 month post-op data and 12 patients were omitted who were insulin diabetics pre-op. 42/69 patients (60%) were overweight or obese prior to surgery (BMI > 25), with an average BMI pre-op of 30, prealbumin 23, A1C 5.5, pQOL 25, mhQOL 36, and took no insulin prior to surgery. Their median islet yield was 244,781 IEQ and 3,316 IEQ/kG (range 14,312-1,168,725 IEQ, 234-16,009 IEQ/Kg). At 1 year post-op, their BMI decreased to 25, and prealbumin was 17, A1C 7.6, pQOL 34, mhQOL 41, mean daily insulin 21 u. 34/42 (80%) of these overweight patients had a sustained post-operative weight loss of at least 10% of their pre-op weight. Comparatively, the patients who had a BMI < 25 prior to surgery (n=27) were found to have an average BMI 20 pre-op, prealbumin 21, A1C 5.6, pQOL 26, mhQOL 36, and took no insulin prior to surgery. Their
median islet yield was 150,168 IEQ and 2,370 IEQ/Kg (range 16,266 – 816,425 IEQ, 312 – 15404 IEQ/Kg). At 1 year post-op, their BMI was 19, Pre-albumin 15, A1C 7.1, pQOL 35, mhQOL 41, and they averaged 10 u/D insulin. The difference in insulin requirements between the two groups is statistically significant (p=0.042).

CONCLUSION: Patients who are overweight or obese prior to TPIAT require more insulin following surgery even though they have higher islet yield and experience significant weight loss compared to those who are not overweight prior to surgery. Both groups experienced an improved physical and mental health QOL following TPIAT.

S039 MULTIVARIABLE LOGISTIC REGRESSION ANALYSIS OF ALCOHOL CONSUMPTION, CIGARETTE SMOKING AND PANCREAS DIVISUM IN THE RISK OF RECURRENT ACUTE AND CHRONIC PANCREATITIS Giulia Martina Cavestro, MD, PhD, Elisabetta Goni, MD, Raffaella Alessia Zuppardo, MD, PhD, Paolo Giorgio Arcidiacono, MD, Silvia Carrara, MD, Alberto Mariani, MD, Maria Chiara Petrone, MD, Gioacchino Leandro, MD, Pier Alberto Testoni, MD Vita-Salute San Raffaele University, San Raffaele Scientific Institute, Milan

BACKGROUND AND AIM: Pathophysiology of acute recurrent pancreatitis (ARP) and its progression through chronic pancreatitis (CP) is still debate and not completely elucidated. Moreover, clinical differences between CP and ARP are controversial too. The aim of the present study was to evaluate the association of alcohol intake, smoking habits and pancreas divisum with ARP and CP.

MATERIAL AND METHODS: ARP patients were classified on the basis of recurrence of acute pancreatitis in the absence of radiological findings of CP (ductal dilation/alteration and/or pancreatic calcifications). Pancreas divisum was diagnosed by means of secretin-enhanced magnetic resonance (sMR) and/or endoscopic retrograde cholangiopancreatography (ERCP). 174 patients with CP and 77 patients with ARP were evaluated. Patients were classified by drinking status: abstainers (≤2 Alcoholic Unit per day), moderate drinkers (2 Alcoholic Unit per day), heavy drinkers (>2 Alcohol Unit per day). Similarly, smoking status was classified as abstainers (≤3 packs/years), moderate smokers (from 3 to 10 packs/years), heavy smokers (>10 packs/years). Statistical analysis was performed by means of multivariable logistic regression.

RESULTS: a) When compared with abstainers, cigarette smoking is a risk factor for CP (respectively moderate smoker p=0.000; O.R=0.98 and heavy smoker p=0.023; O.R=0.209) b) When compared with abstainers, heavy drinker intake is a risk factor to develop CP (p=0.038; O.R=2.583) c) Pancreas divisum is a risk factor to develop ARP (p=0.000; O.R=10.533) but not CP. c) Moderate alcohol intake is not statistically significant in both ARP and CP.

CONCLUSIONS: Heavy alcohol consumption and smoking habits are independent risks for CP. Pancreas divisum is a risk factor to develop ARP.
S040 DOES RESIDENT EXPERIENCE AFFECT OUTCOMES IN COMPLEX ABDOMINAL SURGERY? Daniel Relles, MD, Richard A Burkhart, MD, Jocelyn Sendecki, MS, Michael Pucci, MD, Renee Tholey, MD, Ross Drueing, BS, Patricia K Sauter, CRNP, Eugene P Kennedy, MD, Jordan M Winter, MD, Harish Lavu, MD, Charles J Yeo, MD Thomas Jefferson University

BACKGROUND: Understanding the factors contributing to improved postoperative patient outcomes remains paramount. For complex abdominal operations, the influence of provider and hospital volume on surgical outcomes has been described. The impact of resident experience is less well understood.

METHODS: We reviewed perioperative outcomes after pancreaticoduodenectomy (PD) at a single high-volume center between 2006 and 2012. Resident participation and outcomes were collected in a prospectively maintained database. Resident experience was defined as post-graduate year (PGY) and number of PDs performed.

RESULTS: Twenty-nine residents and four attending surgeons completed 681 PDs. The overall complication rate was 44%; PD-specific complications (defined as pancreatic fistula, delayed gastric emptying, bile leak, abscess, and wound infection) occurred in 28% and were significantly more common when the first assistant was a PGY 4 rather than a PGY 5 or 6 (44% vs. 27%, p=0.016). Logistic regression demonstrated that as residents perform more cases, PD-specific complications decrease (OR=0.97, p<0.01). For a resident’s first case, the predicted probability of a PD-specific complication is 27%; this rate decreases to 19% by case 15.

CONCLUSIONS: Complex cases provide unparalleled learning opportunities and remain an important component of surgical training. We highlight the impact of resident involvement in complex abdominal operations, demonstrating that as residents build experience with PD, patient outcomes improve. This is consistent with volume-outcome relationships for attending physicians and high-volume hospitals. Maximizing resident repetitive exposure to complex surgical procedures benefits both the patient and the trainee.

S041 POSITRON EMISSION TOMOGRAPHY (PET) HAS LIMITED UTILITY IN PREOPERATIVE STAGING OF PANCREATIC ADENOCARCINOMA Peter Einersen, BA, Irene Epelboym, MD, Megan Winner, MD, David Leung, MD, John A Chabot, MD, John D Allendorf, MD Columbia University Medical Center

BACKGROUND: Utility of positron emission tomography (PET) as an adjunctive imaging modality to CT or MRI in evaluating resectability of pancreatic cancer is a subject of controversy. In this study, we seek to assess the utility of PET in identifying occult metastatic disease, as well as evaluate predictive value of maximum standard uptake value (SUV) with respect to tumor resectability and patient survival.

METHODS: Cross sectional imaging, clinical course, operative outcomes, and overall survival of all patients who presented with pancreatic adenocarcinoma and had PET scan in workup were reviewed retrospectively. Resectability was
assessed based on established criteria. Continuous variables were compared using Student’s t-test or ANOVA. Categorical variables were compared using chi-square or Fisher’s exact test. Prediction models were constructed using linear or logistic regression where appropriate.

RESULTS: Complete imaging and follow-up data was available for 123 patients evaluated from 2005 to 2011. Of this cohort, 36 patients (29%) were thought to be free of extrapancreatic disease and offered resection, 21 (17%) had metastatic disease, and 66 (53%) were deemed locally advanced and referred for neoadjuvant therapy. PET and CT/MRI were concordant in 108 (88%) cases, however metastatic lesions were identified in 7 (5.6%) patients deemed resectable by CT or MRI. Among those offered immediate resection, 5 (14%) patients had occult metastatic disease identified at diagnostic laparoscopy, including 3 previously identified by nonconcordant PETs and 2 missed by false negative PETs. False positive PETs led to unnecessary procedures delaying surgery for 3 (8.3%) patients who went on to resection. In a cohort of patients thought to be free of metastatic disease, in terms of detecting metastases, overall sensitivity and specificity of PET were 89.3% and 85.1%, respectively, compared with 62.5% and 93.5% for CT and 61.5% and 100.0% for MRI. Positive predictive value and negative predictive value of PET were 64.1% and 96.4% respectively, compared with 75.0% and 88.9% for CT and 100.0% and 91.9% for MRI. Average difference in maximum SUV of resectable and unresectable lesions was not statistically significant (5.65 vs. 6.5, p=0.224), nor was maximum SUV a statistically significant predictor of survival (p=0.18).

CONCLUSION: PET is a more sensitive modality for identifying metastatic disease than CT or MRI, however, it has a lower specificity and lower positive predictive value. While PET identified an additional 5.6% of patients with occult metastatic disease, it is likely that unresectability would have been established at diagnostic laparoscopy, thus not saving an unnecessary resection. We therefore conclude that PET has limited utility in workup of patients who already undergo CT or MRI as part of initial staging of pancreatic adenocarcinoma.

S042 THE VALUE OF (18)FDG-PET/CT IN PATIENTS WITH RESECTABLE PANCREATIC CANCER: A PROSPECTIVE STUDY Stefano Crippa, MD, Matteo Salgarello, MD, Silvia Laiti, MD, Stefano Partelli, MD, Giuliano Barugola, MD, Paola Castelli, MD, Giuseppe Zamboni, MD, Massimo Falconi, MD Departments of Surgery, Universita’ Politecnica delle Marche, Ancona and Ospedale Sacro Cuore Negrar, Italy and Departments of Nuclear Medicine and of Pathology, Ospedale Sacro Cuore Negrar, Italy

INTRODUCTION/BACKGROUND: Whole-body (18)fluor-deoxyglucose positron emission tomography/computed tomography (PET/CT) has emerged as a promising diagnostic modality in different tumors. The role and the utility of (18)FDG-PET/CT in resectable pancreatic cancer is debated. Aim of the present work was to assess prospectively the value of (18)FDG-PET/CT in addition to conventional imaging as a staging modality in candidates for resection of resectable pancreatic cancer.
METHODS: Whole-body (18)FDG-PET/CT was performed in 72 patients with pancreatic ductal adenocarcinoma who were judged resectable at high-resolution imaging. Neoadjuvant therapy was performed in the 20% of cases. Maximum standardized uptake value (SUVmax) was evaluated 60 minutes after FDG injection. PET/TC was considered “positive” for pancreatic cancer when SUV > 3.

RESULTS: 8/72 (11%) patients were spared unwarranted resection since (18)FDG-PET/CT detected synchronous advanced lung cancer (n=1) or metastatic disease (n=7). Median CA 19.9 was 48.8 U/mL for the entire cohort and 292 U/mL for seven patients with metastases (p=0.112). In other two patients (18)FDG-PET/CT identified one colon carcinoma and a thoracic neurinoma. 15/72 (21%) patients had low metabolic activity (SUVmax<3), and 60% of these patients had undergone neoadjuvant treatment (p=0.0001). At laparotomy 3/64 (5%) patients did not undergo resection because of locally-advanced (n=1) or metastatic disease (n=2). All these patients had SUVmax<3. 61 patients underwent pancreatic resections with curative intent. N1 rate was 77%, with a median of 33 resected nodes. In 8/61 (13%) patients (18)FDG-PET/CT identified metastatic lymph nodes that required an extension of lymphadenectomy outside the usual lymphadenectomy field (i.e. para-aortic nodes). Sensitivity and specificity of (18)FDG-PET/CT for the detection of metastatic disease were 78% and 100%, respectively.

DISCUSSION/CONCLUSION: (18)FDG-PET/CT findings resulted in changes of therapeutic management-operative procedures in one third of patients with resectable pancreatic cancer, thus improving the preoperative staging of these patients. Considering these results, (18)FDG-PET/CT should be considered in the preoperative evaluation of these patients, especially in those with high CA 19.9 levels. Neoadjuvant treatment is significantly associated with low metabolic activity limiting the value of (18)FDG-PET/CT in this setting.

S043 A STANDARDIZED REPORTING SYSTEM FOR EUS/FNA CYTOPATHOLOGY OF SOLID PANCREATIC MASSES Giuseppe Perrone*, MD, PhD, Domenico Borzomati**, MD, PhD, Francesco Di Matteo^, MD, Chiara Brunelli^^, MD, Francesco Panzera*, MD, Gennaro Nappo**, MD, Andrea Onetti Muda*, MD, Roberto Coppola**, MD Units of *Pathology, **General Surgery and ^Digestive Endoscopy, Campus Bio-Medico University of Rome, Italy; ^^Department of Pathology, Catholic University of Rome, Italy

BACKGROUND: Over the past 2 decades, endoscopic ultrasonography (EUS) coupled with fine-needle aspiration biopsy (FNA) has become an effective tool to define suspected pancreatic lesions. Also, the advent of neoadjuvant therapies for pancreatic cancer has increased the need for unequivocal histological diagnosis. However, the reported diagnostic accuracy of EUS-FNA in solid pancreatic lesions ranges between 62% and 96%, with significant variability in terms of sensitivity and specificity. In contrast to breast and thyroid pathology, a standardized reporting system for cytological examination of solid pancreatic lesions is lacking, and this absence could account for such discrepancy. Here we
report the results of a standardized, five-class diagnostic system evaluated in 216 pancreatic FNAC samples, based on literature data as well as on personal experience.

METHODS: EUS/FNAC samples of solid pancreatic mass performed at our Institution from 2008 to 2011 were retrospectively reassessed using 5 clinically relevant pathologic categories:

- PANC1: non diagnostic/inadequate (scant cellularity, smearing artifacts, obscuring blood)
- PANC2: negative for neoplasm (normal acinar and/or ductal epithelium)
- PANC3: atypical/inconclusive (mild to moderate cell atypia, with a low suspicion of malignancy, often in an inflammatory background)
- PANC4: suspicious for carcinoma (strongly suggesting malignancy but cytological features are not sufficient in terms of quantity and/or quality for a definitive diagnosis)
- PANC5: diagnostic of carcinoma (adenocarcinoma, metastatic disease, neuroendocrine tumors)

Sensibility and specificity were calculated according to histology or clinical follow up (at least 6 months).

RESULTS: A total of 216 EUS-FNA were considered. 109 (50.5%) were classified as PANC5, 35 (14.2%) as PANC4, 20 (9.3%) as PANC3, 31 (14.3%) as PANC2 and 21 (9.7%) as PANC1. Clinical follow up or histology was available for all cases. A final diagnosis of carcinoma was obtained in 100% of PANC5, 100% of PANC4, 75.0% of PANC3, 45.2% of PANC2, 66.7% of PANC1 cases. Overall false-positive and false-negative rate was 0% and 14.9%. Sensitivity, specificity, positive predictive value, negative predictive value, and accuracy were 83.2%, 100%, 100%, 43.1%, 85.1% respectively.

CONCLUSIONS. EUS-FNA-based cytological examination of solid pancreatic lesions is an accurate procedure for diagnosis and planning of therapy. Atypical/inconclusive diagnosis (PANC3) may have a detrimental effect on clinical management of patients. In such cases, supplementary diagnostic techniques, such as tumor marker analysis, could be employed on the same samples in order to enhance the EUS-FNA diagnostic yield. Of note, benign FNA findings (PANC2) do not necessarily exclude the presence of pancreatic malignancy, and repeated sampling should be considered whenever the presence of malignancy be suspected clinically. In summary, a uniform reporting system for pancreatic FNA would facilitate communication among pathologists, surgeons, radiologists and other health care providers, allowing easy and reliable sharing of data from different laboratories for collaborative studies.
S044 A LOW LYMPH NODE RATIO IS ASSOCIATED WITH IMPROVED SURVIVAL, DECREASED RECURRENCE AND POSTOPERATIVE CHEMOTHERAPY BENEFIT AFTER NEOADJUVANT CHEMORADIATION FOR PANCREATIC DUCTAL ADENOCARCINOMA Christina L Roland, MD, Ching-Wei D Tzeng, MD, Matthew H Katz, MD, Anthony D Yang, MD, Heather Lin, PhD, Jean-Nicolas Vauthey, MD, Peter W Pisters, MD, Robert A Wolff, MD, Christopher H Crane, MD, Jeffrey E Lee, MD, Jason B Fleming, MD The University of Texas MD Anderson Cancer Center, Houston, Texas

BACKGROUND: Even after completion of multimodality therapy including resection for pancreatic ductal adenocarcinoma (PDAC), recurrence remains a problem, with up to 80% of patients developing locoregional disease. The lymph node ratio (LNR) has been proposed as a prognostic marker of oncologic outcomes after resection. However, its utility in patients who have undergone neoadjuvant chemoradiation (NAC) remains unknown. In this study, we sought to evaluate the effectiveness of the LNR in stratifying outcomes in patients treated with NAC and resection for PDAC.

METHODS: A prospective tumor registry database was queried to identify patients who underwent resection for PDAC between 1990 and 2008 following NAC. Clinical and pathologic factors including LNR were compared to identify associations with overall survival (OS) and time to recurrence (TTR).

RESULTS: One hundred thirty-two (49%) of the 270 patients with PDAC treated with NAC and resection had at least one lymph node metastasis (stage N1), with a median number of 19 lymph nodes harvested (range, 2-55). Median LNR was 0.12 (range, 0.02 – 0.54) for patients with N1 disease. Median OS for the entire cohort was 32 months (95% confidence interval, CI, 27-36). At a median follow-up of 26 months (66 months for survivors), 72% of patients developed recurrent disease. LNR was stratified into the following groups: 0, 0.1-0.14, ≥0.15, centered around the median LNR for N1 patients. There was no difference in median OS or TTR for node-negative patients versus those with LNR >0 and <0.15 (36 vs. 33 months, p=0.86 and 19 vs. 15 months, p=0.59, respectively; Fig. 1A). Patients with a high LNR (LNR ≥ 0.15) had worse OS and TTR compared to low LNR patients (21 vs. 34 months, p<0.001 and 9 vs. 18, p=0.02; Fig. 1A). Furthermore, patients treated with NAC and having a low LNR had better OS and TTR following the administration of additional postoperative chemotherapy (Fig. 1B), whereas patients with LNR ≥ 0.15 received no appreciable benefit from further postoperative chemotherapy (Fig. 1C). On multivariate analysis, patients with a low LNR who received additional postoperative chemotherapy had a reduced risk of death (hazard ratio, HR 0.49; p=0.02) and recurrence (HR 0.58; p=0.04).

CONCLUSION: A low LNR (<0.15) is associated with improved OS and disease-free survival in patients treated with NAC for PDAC. Moreover, additional postoperative chemotherapy for patients with a low LNR is associated with further improved oncologic outcomes. LNR may serve as a clinical marker of tumor biology and help guide surveillance strategies and further therapy after resection in patients with PDAC treated with NAC.
S045 LOCALLY ADVANCED PANCREATIC CANCER: PROLONGED PREOPERATIVE TREATMENT IS ASSOCIATED WITH LYMPH NODE NEGATIVITY AND EXCELLENT OVERALL SURVIVAL

Brian E Kadera, MD, Dharma Sunjaya, BS, William Isacoff, MD, Luyi Li, MS, Oscar J Hines, MD, James Tomlinson, MD, PhD, David Dawson, MD, PhD, Matthew Rochefort, MD, Graham Donald, MD, James Farrell, MD, Barbara Clerkin, RN, MPH, Howard Reber, MD, T University of California, Los Angeles

BACKGROUND: Treatment of patients with locally advanced pancreatic cancer (LAPC) is not standardized. The objectives of the study are to 1) review our institution’s experience with 49 LAPC patients who were downstaged and underwent surgical resection, and 2) identify prognostic biomarkers that could guide adjuvant therapy in this patient subgroup.

METHODS: Retrospective analysis of patients from a single institution during 1992-2011 with AJCC stage III LAPC, who were initially unresectable as determined by staging CT and/or surgical exploration, that were treated and then surgically resected. Clinicopathologic variables and prognostic biomarkers SMAD4, S100A2, and microRNA-21 were correlated with survival by univariate and multivariate Cox proportional hazard modeling (MVA).

RESULTS: All 49 patients were deemed initially unresectable due to vascular involvement. After completing preoperative chemotherapy for a median of 7.1 months (1.8 – 29.8), a majority (75.5%) underwent a pylorus-preserving...
Whipple operation; 3 patients (6.1%) had a vascular resection. Strikingly, 37/49 patients were lymph node (LN) negative (75.5%), 40 (85.1%) had negative margins and 54.2% of evaluable patients achieved a complete/near complete histopathologic (HP) response. The overall median survival (OS) was 40.1 months (11.8 – 213.1). A comprehensive univariate analysis of HP prognostic biomarkers (Table) revealed that perineural invasion (PNI, HR 5.53, p=0.007) and grade (HR 3.68, p=0.02) were most significant. LN involvement, as a marker of systemic disease, was also significant on univariate analysis (p=0.04). Patients with no LN involvement had longer OS (44.4 vs. 23.2 months, p=0.001) than LN positive patients and were more likely to have received 5-FU (24 of 34 (70.6%) vs. 4 of 10 (40%), p=0.07). The molecular biomarkers, SMAD4 loss (Figure, p=0.01) in tumor cells and microRNA-21 expression in the stroma (p=0.04) also correlated with OS. On MVA of HP and molecular markers, only SMAD4 loss was significant (HR 5.44, p=0.04). To our knowledge, this is the first study to show a correlation of SMAD4 and microRNA-21 with survival in this patient subgroup.

CONCLUSION: Prolonged preoperative chemotherapy for patients with LAPC is associated with a high incidence of LN negative disease and excellent OS. After surgical resection, PNI, grade and SMAD4 status should guide adjuvant treatment decisions in this select subset of patients.

Cox proportional hazard models for significant prognostic factors and biomarkers

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**S046 FREQUENCY AND INTENSITY OF POSTOPERATIVE SURVEILLANCE AFTER CURATIVE TREATMENT OF PanCREATIC CANCER: A COST-EFFECTIVENESS ANALYSIS** Daniel E Abbott, MD, Ching-Wei D Tzeng, MD, Scott B Cantor, PhD, Jason B Fleming, MD, Jeffrey E Lee, MD, Peter W Pisters, MD, Gauri R Varadhachary, MD, James L Abbruzzese, MD, Robert A Wolff, MD, Syed A Ahmad, MD, Matthew H Katz, MD University of Cincinnati, The University of Texas MD Anderson Cancer Center

**INTRODUCTION/BACKGROUND:** Few data exist to guide oncologic surveillance following curative treatment of pancreatic cancer. We sought to identify a rational, cost-effective postoperative surveillance strategy.

**METHODS:** We constructed a decision-analytic (Markov) model to compare the cost-effectiveness of five postoperative surveillance strategies. No scheduled surveillance served as the baseline strategy. Clinical evaluation (comprehensive symptom assessment and physical exam) and serum carbohydrate antigen (CA) 19-9 testing without or with routine computed tomography of the abdomen/pelvis and chest x-ray at either 6- or 3-month intervals served as the four
comparison strategies of increasing intensity. We populated the model with symptom, recurrence, treatment, and survival data from patients who received intensive surveillance following multimodality treatment at our institution between 1998-2008. Costs were based on Medicare payment (2011 U.S. dollars).

RESULTS: No scheduled surveillance was associated with a 24.6-month postoperative overall survival (OS) duration and a cost of $3,837 per patient. The four scheduled surveillance strategies each cost between $7,496 and $24,775 per patient and were associated with 32.8-34.1-month postoperative OS. Clinical evaluation with CA 19-9 scheduled every 6 months was associated with a 32.8-month postoperative OS and a cost of $7,496 per patient, with an incremental cost effectiveness ratio (ICER) of $5,364 per life-year (LY). The addition of routine imaging every 6 months incrementally increased cost by $3,465 without increasing OS. ICERs associated with more frequent and intensive 3-month clinical evaluations and CA 19-9 without or with routine imaging were $127,680 and $294,696 per LY, respectively. Sensitivity analyses changed the strategies’ absolute costs without changing the relative ranks of their ICERs.

DISCUSSION/CONCLUSION: In our model, increasing the frequency and intensity of postoperative surveillance of pancreatic cancer beyond a limited strategy of clinical evaluation and CA 19-9 scheduled every 6 months was associated with increased cost but no clinically significant survival benefit.

S047 TREATMENT SEQUENCING FOR RESECTABLE PANCREATIC CANCER: INFLUENCE OF EARLY METASTASES AND SURGICAL COMPLICATIONS ON MULTIMODALITY THERAPY COMPLETION RATES AND SURVIVAL

Ching-Wei D Tzeng, MD, Daniel E Abbott, MD, Jeffrey D Lee, MD, Peter W Pisters, MD, Jason B Fleming, MD, Jean-Nicolas Vauthey, MD, Matthew H Katz, MD

The University of Texas MD Anderson Cancer Center; University of Cincinnati

INTRODUCTION: Multimodality therapy (MMT) is important to the long-term survival of patients with resectable pancreatic adenocarcinoma (PDAC), but its completion can be hindered by early cancer progression or by treatment complications. We sought to compare the influence of each of these factors on the MMT completion rates of operable patients with resectable PDAC treated with either a neoadjuvant (NT) or surgery-first (SF) sequencing strategy.

METHODS: We retrospectively evaluated all patients with PDAC at our institution from 2002-2007, who had 1) a radiographically resectable pancreatic head tumor, 2) a performance status (PS) and comorbidities suitable for immediate surgery, and 3) a carbohydrate antigen (CA) 19-9 <1000 U/ml. MMT was defined as resection before or after completion of planned pre- or post-operative therapy. Postoperative major complications (PMC) were defined as Clavien Grade ≥3. Disease progression was considered early when it developed within 3 months in SF patients or prior to planned resection in NT patients. Reasons for and rates of failure to complete MMT, 90-day PMC, and overall survival (OS) were compared between the two cohorts.

RESULTS: 112 NT and 58 SF patients met inclusion criteria. 92/112 (82%) NT and
33/56 (59%) SF patients with complete follow-up completed MMT (p<0.001). NT patients did not complete MMT due to early progression (n=13, including 8 nontherapeutic laparotomies) and PS (n=7). SF patients did not complete MMT due to early progression (n=10), PMC (n=6), and PS (n=3); 4 SF patients also underwent nontherapeutic laparotomy due to metastases.

AMONG all patients, those who completed MMT lived longer than those who did not (36 vs. 11 mo, p<0.001). The median OS durations of all NT and SF patients (NT 28 vs. SF 21 mo, p=0.082), the subset in each cohort who completed MMT (NT 36 vs. SF 36 mo, p=0.565), and the subset in each cohort who did not complete MMT (NT 11 vs SF 13 mo, p=0.325) were not statistically different.

THE rate of PMC did not differ between NT and SF groups (19% vs. 17%, p=0.782). SF patients with no PMC had a 71% (31/44) MMT completion rate vs. 25% (2/8) after PMC (p=0.014). When resected NT patients suffered PMC, there was no significant decrease in OS (36 vs. 30 mo, p=0.934), in contrast to the negative effect of PMC in SF patients (26 vs. 10 mo, p<0.001).

CONCLUSIONS: Completion of multimodality therapy is strongly associated with improved survival of operable patients with resectable PDAC. Even in the highly selected cohort evaluated in this study, early cancer progression and PMC negatively impacted MMT completion rates and OS, particularly among SF patients. Thus, NT sequencing remains a valuable alternative to SF sequencing for tumor biology evaluation and patient selection.
S048 A COMPARATIVE ANALYSIS OF PLASTIC VERSUS METAL ENDOSCOPIC BILIARY STENTS IN BORDERLINE RESECABLE PANCREATIC CANCER PATIENTS UNDERGOING EXTENDED NEOADJUVANT CHEMOTHERAPY

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BACKGROUND: Endoscopic biliary stenting is widely accepted as a treatment for malignant biliary obstruction from pancreatic cancer. While it is well-established that patency with metal stents is superior to plastic stents in patients with malignant biliary obstruction, their relative clinical efficacy in patients with borderline resectable pancreatic cancer undergoing extended neoadjuvant chemotherapy (>6 months) is unknown. We hypothesized that in this patient population, initial metal stent placement for malignant biliary obstruction is associated with a decreased incidence of biliary complications compared to plastic stents.

METHODS: All patients with biopsy-proven borderline resectable pancreatic cancer by AHPBA/SSO consensus criteria were identified prospectively over a 4-year period (2008-2012). Patients who did not require biliary stenting were excluded from analysis. A retrospective review of all stented patients was performed. Patient demographics, stent history, complications, need for exchange, and time to operation were analyzed. Continuous variables were compared between groups using a Mann-Whitney U test, while proportions were compared utilizing a chi-square test. Multivariate logistic regression analysis was used to investigate variables leading to complications. Statistical analysis was performed using MedCalc 12.3 statistical software (Mariakerke, Belgium).

RESULTS: Of the 62 patients with borderline resectable pancreatic cancer, 40 (65%) required preoperative endoscopic biliary stenting for malignant obstruction. Twenty-five of the 40 patients (63%) had plastic stents placed initially. Twenty-one of the 40 patients (53%) were initially stented at an outside hospital. Complications requiring stent exchange (cholangitis, pancreatitis, abscess, cholecystitis, biliary obstruction) occurred significantly more often in patients with initial plastic stents (76% vs. 13.5%; p=.001). Mean functional stent time (defined as time from placement to exchange, resection, or death) was significantly longer in the metal stent cohort (363 vs. 176 days; p=0.015). There was no statistical difference in patient age, sex, tumor size, time to resection, resectability between metal and plastic stent cohorts. The occurrence of stent-related complications did not impact resectability.

CONCLUSIONS: Metal stents should be considered initially in patients with malignant biliary obstruction from borderline resectable pancreatic cancer undergoing extended neoadjuvant chemotherapy due to a decreased rate of complications and increase in patency.
S049 EXTENDED NEOADJUVANT CHEMOTHERAPY FOR LOCALLY ADVANCED, RESECTABLE PANCREATIC CANCER DEMONSTRATES PROMISING POSTOPERATIVE OUTCOMES AND SURVIVAL J B Rose, F Rocha, A Alseidi, T Biehl, R Moonka, J Ryan, B Lin, V Picozzi, S Helton Virginia Mason Medical Center

INTRODUCTION: The optimal duration of neoadjuvant chemotherapy for patients with locally advanced, resectable pancreatic cancer has yet to be established. Herein we report the surgical outcomes of a 6 month regimen of gemcitabine and docetaxel prior to attempted resection.

METHODS: Included in this study were all patients between 2008-2012 with locally advanced, resectable pancreatic cancer by AHPBA/SSO criteria, who had pancreatic head/neck/body lesions, and who were offered extended neoadjuvant chemotherapy at our institution. Medically fit patients who completed 6 months of chemotherapy and had no radiographic signs of progression were offered an operation for curative intent.

RESULTS: 59 patients with locally advanced, resectable pancreatic cancer started neoadjuvant therapy. 23 (39%) patients had progression of disease, chemotherapy toxicity, or medical comorbidities that precluded operation. 36 (61%) patients completed neoadjuvant therapy and underwent an attempt at resection. 10 (17%) were found to have incurable disease intraoperatively while 26 (44%) patients had a subsequent pancreatectomy. All resections were R0, 14 (54%) had positive lymph nodes, 10 (38%) required en bloc venous resection, and 4 (15%) had Tis or T0 lesions on final review. There were no postoperative deaths at 90 days, 26% of patients had Clavien-Dindo Grade 3 or greater complications, with a 30 day readmission rate of 8%. The median overall survival of all 59 patients was 31 months, with a median survival for unresectable patients of 19 months. 25 of 29 (86%) of the resected patients are still alive with a median follow-up of 24 months.

CONCLUSION: Extended neoadjuvant chemotherapy with gemcitabine and docetaxel is well tolerated, selects a subset of patients for curative surgery with low postoperative morbidity, and is associated with favorable survival.

S050 GEMCITABINE-BASED CHEMORADIOThERAPY FOLLOWED BY SURGERY FOR RESECTABLE, BORDERLINE RESECTABLE AND LOCALLY UNRESECTABLE PANCREATIC ADENOCARCINOMA Masashi Kishiwada, MD, PhD, Motoyuki Kobayashi, MD, Akihiro Tanemura, MD, PhD, Naohisa Kuriyama, MD, PhD, Yoshinori Azumi, MD, PhD, Ichiro Osawa, MD, PhD, Shugo Mizuno, MD, PhD, Masanobu Usui, MD, PhD, Hiroyuki Sakurai, MD, PhD, Masami Tabata, MD, Hepatobiliary Pancreatic and Transplant Surgery, Mie University School of Medicine

Chemoradiotherapy (CRT) prior to surgery for pancreatic ductal adenocarcinoma (PDAC) may provide for early treatment of micrometastatic disease, allows for the identification of the patients with metastatic disease at the
time of reassessment, and may increase R0 resection rate, resulting in reduced risk of local tumor recurrence. Our institution has introduced gemcitabine-based chemoradiotherapy followed by surgery (gem-CRTS) for PDAC. The aim of this study was to evaluate the efficacy of gem-CRTS for PDAC regarding the three resectability groups (resectable: R, borderline resectable: BR and locally unresectable: UR) defined by NCCN pancreatic cancer guidelines (2012). These resectability groups were defined by artery related factors (eg. celiac axis, superior mesenteric artery, and peripancreatic arteries) and venous structure related factors (eg. superior mesenteric vein and portal vein). BR PDAC consists of three groups: artery-related BR(A-BR), vein-related BR(V-BR), and both factors-related BR(A+V-BR).

PATIENTS & METHODS: From February 2005 to December 2011, 124 patients with PDAC had been enrolled for our gem-CRTS protocol. Gem-CRT regimen were 45 to 50.4 Gy radiation in 25 to 28 fractions with weekly intravenous 800 mg/m² gemcitabine for 5 weeks including one-week break. These patients were retrospectively classified into three respectability groups based on four-phase dynamic CT. The 124 patients were classified as R in 16 patients, BR in 57 and UR in 51. The patients underwent curative-intent resection after reassessment by response of gem-CRT completion. We evaluated survival rates and R0 resection respectability. Especially in BR, we compared the survival rates among the A-BR, V-BR and A+V-BR groups.

RESULTS: The overall 3-year survival rate was 43.8% in R, 28.1% in BR and 6.1% in UR, respectively. Interestingly, in BR, the 3-year survival rate was significantly higher in 29 patients with CA19-9 reduction rate of more than 50% than in 24 with less than 50%: 42.8% vs. 9.7% (p=0.0018). In the three groups of BR, the 3-years survival rate was 16.7% in A-BR (n=8), 38.5% in V-BR (n=33) and 20.2% in A+V-BR (n=16). At the time of reassessment, distant metastases had become apparent in 15% in R, 11% in BR and 22% in UR, respectively. Among re-evaluated patients, tumor resection rate was 71.4% in R, 77.8% in BR and 43.4% in UR. In the 75 patients with curative-intent resection, R0 resection rate 100% in R (n=9), 80% in BR (n=36) and 43% in UR (n=9), and the 3-year survival rate was 77.8%, 34.0% and 21.1%, respectively.

CONCLUSION: Our gem-CRTS protocol is effective in R and BR but not in UR. Among BR PDAC, artery-related BR had much poorer prognosis than vein-related BR, and thus this type of PDAC should be treated separately.

S051 RESECTION OF LOCALLY ADVANCED PANCREATIC CANCER AFTER NEOADJUVANT CHEMOTHERAPY WITH MODIFIED FOLFIRINOX: A PROSPECTIVE PHASE II STUDY Enrico Vasile, MD, Nelide De Lio, MD, Mario Antonio Belluomini, MD, Francesca Costa, MD, Carla Cappelli, MD, Daniela Campani, Alfredo Falcone, Ugo Boggi, FACS Division of General and Transplant Surgery, University of Pisa, Pisa, Italy 1. Division of Oncology, University of Pisa, Pisa, Italy 2. Division of Radiology, University of Pisa, Pisa, Italy 3. Division of Pathology, University of Pisa, Pisa, Italy
INTRODUCTION: At the time of diagnosis pancreatic ductal adenocarcinoma (PDAC) is deemed resectable only in 20% of the patients. In approximately 30-40% of the patients surgery is denied because of local tumor growth only, in the absence of obvious distant metastatic spread. These patients could be still be considered for resection, if responsive to neoadjuvant chemotherapy (NACT) or chemoradiation. We herein report the results of a phase II clinical trial, coupling high-dose multi-drug NACT with aggressive surgery.

METHODS: All patients enrolled in this study were selected by a multidisciplinary workgroup, including surgeons, oncologists and radiologists. Selection criteria included stage III locally advanced PDAC (suspected arterial involvement), ECOG PS 0-1, age 18-75 years. All patients underwent a phase II NACT protocol, employing a modified FOLFIRINOX regimen. Tumor response was evaluated according to RECIST criteria by comparing pre-treatment contrast-enhanced computed tomography (CT) scan with follow-up imaging obtained at 4-week intervals. The opportunity to add a local treatment, either surgery or radiation therapy, was evaluated by the multidisciplinary team after every CT follow-up.

RESULTS: Between November 2010 and November 2012, 26 patients were enrolled in this study. Mean age was 59 years (range 44-75). All patients had a stage III tumor because of CT diagnosis of celiac axis (n=9; 34.6%) superior mesenteric artery (n=11; 42.3%), or celiac axis and superior mesenteric artery (n=6; 22.2%) involvement. Nine patients had a partial response (34%), 15 had a stable disease (57%), and 2 progressed (7.6%). While 3 patients are still awaiting a final decision after NACT, 14 out of 23 patients were selected for surgery (60.8%) and 11 underwent resection with curative intent (47.8%). Two patients underwent pylorus-preserving pancreaticoduodenectomy (2/11) and nine total pancreatectomy with en-bloc splectomy (9/11). Multivisceral resection was necessary in 6 patients.

Mean operative time was 618 minutes (480-900). One patient died because of sepsis due a multidrug-resistant bacteria. Overall postoperative morbidity was 62%. In particular, the surgical morbidity was 12%, and medical morbidity 50%. Mean hospital-stay was 26 days (17-42). All operations were R0 resections. The mean number of resected lymph nodes was 67 (22-90), and the mean number of nodal metastasis was 4 (1-6). Twelve percent of resected venous segments and 33% of resected arterial segments were not involved on histology. Progression-free survival of the entire population of 26 patients was 17.6 months. Progression-free survival of resected patients was 17.8 months as compared to 10.3 months for patients who never became surgical candidates. Median overall survival was 24 months.

CONCLUSIONS: Our interim analysis confirms the activity of modified FOLFIRINOX protocol in PDAC, allowing extended resection in a relevant percentage of stage III PDAC with results comparable to those achieved in primary resectable patients. New data from further studies and from larger cohorts are needed before any final conclusion may be drawn.
S052 IMPACT OF MARGIN CLEARANCE ON SURVIVAL AFTER PANCREATICODUODENECTOMY FOR PANCREATIC DUCTAL ADENOCARCINOMA Yasushi Hashimoto, MD, Yoshiaki Murakami, MD, Kenichiro Uemura, MD, Takeshi Sudo, MD, Naru Kondo, MD, Hayato Sasaki, MD, Taijiro Sueda, MD Department of Surgery, Applied Life Sciences Institute of Biomedical

BACKGROUND: Microscopic involvement of a resection margin by tumor is associated with a poor prognosis. It is unclear whether a proximity to resection margins by tumor confers a survival benefit over margin involved R1 resection of their pancreatic ductal adenocarcinoma (PDAC) after pancreticoduodenectomy (PD). The aim is to better understand the impact of resection status on clinical and pathologic staging, and long-term survival after PD for PDAC, and to explore the prognostic significance of a proximity to surgical margins.

METHODS: We assessed the relationships between margin involvement (R1), the proximity to resection margins (R0-close) and outcome in a cohort of 124 consecutive patients who underwent PD for PDAC between 2002 and 2012. Resected specimens were analyzed according to the improved standardized pathology protocol which included permanent section analysis of the surgical margins. R0-close margin was defined as tumor within 1-mm of the resection margins and a patient with a margin of greater than 1-mm was defined as R0-wide margin. Follow-up data on overall and disease-free survival, presence and site of tumor recurrence were examined.

RESULTS: Of the 124 patients, the resection margins were positive (R1) in 30 (24%) and negative (R0) in 94 patients (76%) including 38 patients (31%) with an R0-close resection. Patients with R1 resections had an unfavorable survival compared with those with R0 resections (median, 18 vs 35 months; P<0.01), but survival with R0-close margin were comparable to R1 resections: but both groups had a significantly shorter survival than patients with R0-wide margins (18 vs 32 vs 44 months, respectively; P=0.02). Disease-free survival was shorter in R1/R0-close margins comparing to R0-wide group (median, 12 vs 19 months; P=0.04). By multivariate analysis, predictors of R1/R0-close margins were patients underwent portal vein resection and larger tumor size of greater than 20-mm. The pattern of tumor recurrence had a greater rate of regional metastases in the R1/R0-close margins group comparing to patients with R0-wide margins (48% vs 14%; P=0.01).

CONCLUSION: These data demonstrate that a margin clearance of more than 1-mm is important for long-term survival in a subgroup of patients. Complete histologic evaluation of the resected PD specimens is important for prognosis in patients with PDAC who underwent PD. More aggressive therapeutic approaches that target locoregional disease such as neoadjuvant radiation therapy may be beneficial in patients with close surgical margins.
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<th>DATE &amp; LOCATION</th>
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<td>1966 – Northwestern</td>
<td>Marion Anderson</td>
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<td>1967 – Philadelphia</td>
<td>John Howard</td>
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<td>1968 – University of California, San Francisco</td>
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<td>1969 – Mt. Sinai Hospital</td>
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<td>1970 – University of Chicago</td>
<td>Edward Paloyan</td>
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<td>1971 – Sheraton Hotel, Philadelphia, PA</td>
<td>John Howard</td>
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<td>1972 – University of California, San Francisco</td>
<td>Englebert Dunphy</td>
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<td>David Dreiling</td>
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<td>1976 – Doral on the Ocean, Miami, FL</td>
<td>Robert Zeppa</td>
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<td>1977 – Toronto, Canada</td>
<td>Roger Keith</td>
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<td>1978 – Jockey Club, Las Vegas, NV</td>
<td>Charles Frey</td>
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<td>1979 – LSU Medical Center, New Orleans</td>
<td>Isadore Cohn</td>
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<td>1980 – Salt Lake City</td>
<td>Frank Moody</td>
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<td>1981 – Alumni Hall, NYU</td>
<td>John Ranson</td>
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<td>1982 – University of Chicago</td>
<td>A.R. Moosa</td>
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<td>1983 – Washington Hilton</td>
<td>Francis Milligan</td>
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<td>1984 – LSU Medical Center, New Orleans</td>
<td>Francis Nance</td>
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<td>David Dreiling</td>
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<td>1986 – Ft. Miley VA, San Francisco, CA</td>
<td>Carlos Pellegrini</td>
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<td>1987 – University of Illinois, Chicago</td>
<td>Phillip Donahue</td>
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<td>1988 – Tulane University, New Orleans, LA</td>
<td>Elmo Cerise</td>
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<td>1989 – Washington Hilton</td>
<td>Gregory Bulkley, Frances Milligan, John Cameron</td>
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<tr>
<td>1990 – University of Texas, San Antonio</td>
<td>Bradley Aust</td>
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</table>
1991 – Tulane University, LSU  Elmo Cerise, J. Patrick O’Leary
1992 – University of California, San Francisco  Carlos Pellegrini
1993 – Massachusetts General Hospital, Boston, MA  Andrew Warshaw
1994 – Tulane University, New Orleans, LA  Elmo Cerise, J. Patrick O’Leary
1995 – University of California, San Diego  A.R. Moosa
1996 – Laurel Heights, UCSF  Sean Mulvihill
1997 – University Health Sciences, Bethesda, MD  John W. Harmon
1998 – LSU, Tulane  J. Patrick O’Leary, Elmo Cerise
1999 – Peabody, Orlando, FL  Michael M. Murr, James G. Norman
2000 – University of California, SD  A.R. Moosa
2001 – Hilton Atlanta, GA  Aaron Fink
2002 – San Francisco, CA  Kimberly Kirkwood
2003 – Orlando, FL  Michael Murr
2004 – New Orleans, LA  Alton Ochsner
2005 – Chicao, IL  Gerard V. Aranha, Richard Bell
2006 – Los Angeles, CA  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
2010 – New Orleans, LA
2011 – Chicago, IL  Gerry Aranha, Mark Tamamonit, David Bentrem
2012 – San Diego, CA
2013 – Orlando, FL

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48th Annual Meeting of the Pancreas Club
May 2-3, 2014
Chicago, Illinois

Past Meetings of the Pancreas Club
SAVE THE DATE

48th Annual Meeting of the Pancreas Club

MAY 2-3, 2014

Chicago, Illinois