50th ANNUAL MEETING
OF THE PANCREAS CLUB

May 20-21, 2016 in San Diego, CA

The Pancreas Club, Inc.
Welcome to the 50th Annual Meeting of the Pancreas Club at the Hyatt Regency Mission Bay Hotel in San Diego, CA. 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.

The Pancreas Club is pleased to continue an expanded two full-day program with the annual dinner taking place on Friday evening. We know that you will be fully engaged in both listening to the excellent presentations and in the discussions which follow. Posters of Distinctions will be presented by authors and addressed by leading faculty during the Poster Rounds with Professors. Authors will also be available posterside during the several Poster Sessions.

This meeting will offer Continuing Medical Education (CME) Credits through a joint providership 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 also available on our website [http://pancreasclub.com/annualmeeting](http://pancreasclub.com/annualmeeting).

We hope that you will also have the opportunity to bring your family and explore the San Diego/Mission Bay Region. Mission Bay is the largest aquatic preserve in the U.S. and the hotel is surrounded by the serenity of azure blue water and eight acres of lush landscaping are dotted with serene gardens. Downtown San Diego, SeaWorld, San Diego Zoo, Legoland and shopping at Fashion Valley Mall are all located just short drive of Mission Bay.

Thank you for your participation and support of the Pancreas Club. Enjoy the meeting, the city and associating with old and new friends!
Join us in celebrating The 50th Annual Meeting of The Pancreas Club. Our Golden Anniversary! With its humble first meeting of perhaps 10 dedicated Pancreatic Surgeons in a small room at Northwestern University back in 1966, we now, 50 years later, boast one of the richest, if not the richest scientific programs devoted entirely to clinical and scientific progress in Pancreatic Diseases. With the exception of one skipped meeting in 1974 we have met on a yearly basis, preserved a less formal atmosphere, valued content over regulations, openly welcomed even previously published seminal reports as well as papers presented in other fora, all to maximally benefit from perhaps the most valuable asset we possess; our membership. Over the years the membership has grown in size and breadth with members from 5 continents and countless U.S. and international centers. Over these years we have seen our junior members grow to Chairmanships and to positions of influence. We have seen the sophistication and depth of knowledge in both benign and malignant diseases of the pancreas grow exponentially. We have seen some previously unknown diagnoses, such as IPMN, first populate our programs in small case studies and then dominate our program with large, multi-institutional reports. We have seen technologies arrive and take their places in the surgical armamentarium. And we have grown to this year a record 293 submitted abstracts and a total of 64 oral presentations over a full two day schedule. So much for us to celebrate in our past and so much reason to anticipate a very bright future for the Club!
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GENERAL INFORMATION

MEETING LOCATION

Hyatt Regency Mission Bay
1441 Quivira Road
San Diego, CA  92109
PHONE: 619-224-1234

MEETING HOURS

REGISTRATION

Thursday, May 19, 2016 • 6:00 pm – 8:00 pm
Friday, May 20, 2016 • 6:30 am – 6:30 pm
Saturday, May 21, 2016 • 6:45 am – 5:00 pm

SCIENTIFIC SESSIONS

Friday, May 20, 2016 • 7:45 am – 5:35 pm
Saturday, May 21, 2016 • 8:00 am – 5:00 pm

EXHIBITS

Friday, May 20, 2016
9:30 am – 3:45 pm  Exhibits Open
9:45 am – 10:00 am  Refreshment Break in Exhibit Area
3:30 pm – 3:45 pm  Refreshment Break in Exhibit Area

Saturday, May 21, 2016
9:30 am – 3:30 pm  Exhibits Open
9:45 am – 10:00 am  Refreshment Break in Exhibit Area
3:00 pm – 3:15 pm  Refreshment Break in Exhibit Area

GENERAL BUSINESS MEETING

Saturday, May 21, 2016 • 5:00 pm – 5:30 pm

ANNUAL DINNER/RECEPTION

Friday, May 20, 2016 • 6:30 pm – 9:30 pm

WINE & CHEESE AWARDS RECEPTION

Saturday, May 21, 2016 • 5:30 pm – 6:30 pm
CONTINUING MEDICAL EDUCATION

MEETING/LEARNING OBJECTIVES

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

- Describe drain management following pancreatoduodenectomy.
- Recognize that hepaticojejunostomy stricture may occur following Whipple resection.
- Outline a contemporary treatment sequence for a patient with borderline resectable pancreatic cancer.
- Explain the impact of Ca 19-9 antigen level in resectable pancreatic cancer and impact on survival.
- Compare neoadjuvant therapy and surgery first approaches for Stage III pancreatic cancer.
- Detail the natural history of large (>3 cm) branch-duct intraductal papillary mucinous neoplasms.
- Discuss the pattern of recurrence of of invasive IPMN compared to ordinary PDAC.
- Explain the role of (18) Fluoro-deoxyglucose PET scanning as a predictor of survival in resected pancreatic cancer.
- Compare robotic and laparoscopic pancreatoduodenectomy relative to feasibility, cost, safety and outcomes.
- Describe the modified Appleby operation for carcinoms of the body of the pancreas and characteristic complications associated with the procedure.
- Explain the role of FOLFIRINOX in the management of pancreatic cancer in the neoadjuvant setting.
CONTINUING MEDICAL EDUCATION CREDIT INFORMATION

Accreditation
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership 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 13.75 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

PROGRAM COMMITTEE MEMBERS
William Nealon, MD, Chair
Horacio Asbun, MD
Marshall Baker, MD, MBA
Michael Farnell, MD
Cristina Ferrone, MD
Ernst Klar, MD
Kyoichi Takaori, MD, PhD
Matthew Weiss, MD
Christopher Wolfgang, MD, PhD
Nicholas Zyromski, MD
DISCLOSURE INFORMATION

In accordance with the 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. Therefore, it is mandatory that both the program planning committee and speakers complete disclosure forms. Members of the program committee were required to disclose all financial relationships and speakers were required to disclose any financial relationship as it pertains to the content of the presentations. The ACCME defines a ‘commercial interest’ as “any entity producing, marketing, re-selling, or distributing health care goods or services consumed by, or used on, patients”. It does not consider providers of clinical service directly to patients to be commercial interests. The ACCME considers “relevant” financial relationships as financial transactions (in any amount) that may create a conflict of interest and occur within the 12 months preceding the time that the individual is being asked to assume a role controlling content of the educational activity.

ACS is also required, through our joint providership partners, to manage any reported conflict and eliminate the potential for bias during the activity. All program committee members and speakers were contacted and the conflicts listed below have been managed to our satisfaction. However, if you perceive a bias during a session, please report the circumstances on the session evaluation form.

Please note we have advised the speakers that it is their responsibility to disclose at the start of their presentation if they will be describing the use of a device, product, or drug that is not FDA approved or the off-label use of an approved device, product, or drug or unapproved usage.

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

### SPEAKERS/MODERATORS/CHAIRS/DISCUSSIONS

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<th>Name</th>
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<tr>
<td>Marc Besselink</td>
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<td>Uggo Boggi</td>
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<td>Roberto Coppola</td>
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<td>Matthew Katz</td>
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<td>Tobias Keck</td>
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<td>Michael Kendrick</td>
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### PLANNING COMMITTEE

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<td>Horacio Asbun</td>
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### PRESENTERS

*Unless indicated below, the Oral Presenters do not have any financial relationships to disclose relating to the content of this activity.*

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<td>Ji Young Bang</td>
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* Indicates also moderator/faculty

Olympus Medical Systems Corporation – Consulting Fee Consultant
SCHEDULE-AT-A-GLANCE

MEETING ROOMS

SCIENTIFIC SESSIONS: Bayview Ballroom
POSTERS: Regatta Pavilion
REGISTRATION & EXHIBITS: Bayview Foyer

THURSDAY, MAY 19, 2016

6:00 pm – 8:00 pm  Registration  Bayview Foyer
6:00pm – 8:00pm  Poster Setup  Regatta Pavilion
6:00 pm – 8:00 pm  Advisory Committee Meeting/Dinner  Red Marlin

FRIDAY, MAY 20, 2016

6:30 am – 6:30 pm  Registration  Bayview Foyer
7:00 am – 7:45 am  Continental Breakfast  Regatta Pavilion
7:45 am – 8:00 am  Welcome and Introductory Remarks  Bayview Ballroom
8:00 am – 9:45 am  SCIENTIFIC SESSION I: Drains/ Pancreatic Fistula/Complications of Pancreatic Surgery  Bayview Ballroom
9:30 am – 3:45 pm  Exhibits Open  Bayview Foyer
9:45 am – 10:00 am  Break with Exhibitors & Poster Viewing
10:00 am – 11:00 am  SCIENTIFIC SESSION II: Surgical Techniques and Innovations  Bayview Ballroom
11:00 am – 12:00 pm  Poster Rounds with Professors  Regatta Pavilion
12:00 pm – 1:00 pm  Lunch  Regatta Pavilion
Free lunch for all attendees
1:00 pm – 3:30 pm  SCIENTIFIC SESSION III: Borderline Resectable Pancreatic Cancer/ Neo-Adjuvant Therapy  Bayview Ballroom
3:30 pm – 3:45 pm  Break with Exhibitors & Poster Viewing
3:45 pm – 5:35 pm  SCIENTIFIC SESSION IV: Basic Science Studies/ Rare Pancreatic Tumors  Bayview Ballroom
6:30 pm – 9:30 pm  Pancreas Club Annual Reception & Dinner  Reception: Banyan Court Dinner: Regatta Pavilion
### SCHEDULE-AT-A-GLANCE

#### SATURDAY, MAY 21, 2016

<table>
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<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tr>
<td>6:45 am – 5:00 pm</td>
<td>Registration</td>
<td>Bayview Foyer</td>
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<tr>
<td>7:00 am – 8:00 am</td>
<td>Continental Breakfast</td>
<td>Regatta Pavilion</td>
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<tr>
<td>8:00 am – 9:45 am</td>
<td><strong>SCIENTIFIC SESSION V:</strong> Acute and Chronic Pancreatitis/Disconnected Duct Syndrome/TPIAT</td>
<td>Bayview Ballroom</td>
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<tr>
<td>9:30 am – 3:30 pm</td>
<td>Exhibits Open</td>
<td>Bayview Foyer</td>
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<tr>
<td>9:45 am – 10:00 am</td>
<td>Break with Exhibitors &amp; Poster Viewing</td>
<td>Bayview Foyer</td>
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<td>10:00 am – 11:00 am</td>
<td>History of Pancreas Club</td>
<td>Bayview Ballroom</td>
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<tr>
<td>11:00 am – 12:00 pm</td>
<td>Poster Rounds with Professors</td>
<td>Regatta Pavilion</td>
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<tr>
<td>12:00 pm – 1:00 pm</td>
<td>Lunch</td>
<td>Regatta Pavilion</td>
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<tr>
<td>1:00 pm – 3:00 pm</td>
<td><strong>SCIENTIFIC SESSION VI:</strong> Centralization/Nomogram/Genetics of Cancer</td>
<td>Bayview Ballroom</td>
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<td>3:00 pm – 3:15 pm</td>
<td>Break with Exhibitors &amp; Poster Viewing</td>
<td>Bayview Ballroom</td>
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<td>3:15 pm – 5:00 pm</td>
<td><strong>SCIENTIFIC SESSION VII:</strong> IPMN</td>
<td>Bayview Ballroom</td>
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<td>5:00 pm – 5:30 pm</td>
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<td>5:30 pm – 6:30 pm</td>
<td>Wine &amp; Cheese Awards Reception</td>
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The Pancreas Club will recognize five outstanding presentations. They will be awarded during the closing, Saturday afternoon, reception:

**PanCan Research Award:** One $1,000 and two $500 awards for the best oral presentation of pancreatic cancer research by a resident or fellow. This award is generously funded by the Pancreatic Cancer Action Network.

**Kenneth Warren Research Award:** $1,000 for the best oral presentation of clinical or basic science pancreatitis by a resident or fellow. This award is generously funded by the Kenneth Warren Foundation.

**John Howard Research Award:** $1,000 for the best presentation from young junior faculty, who is within 5 years of their end of residency. This award is generously funded by the Arpa Foundation.
Join us on Friday evening as we celebrate the Golden Anniversary of our Annual Meeting.

WHERE: Reception @ Banyan Court starting @ 6:30 pm
Dinner @ Regatta Pavilion @ 7:30 pm

COST: $80 per person. Stop by our registration desk by 2:00 pm to get your tickets.

Johann Georg Wirsung (July 3, 1589 Augsburg – August 22, 1643 Padua) was a German anatomist who was a long-time prosector in Padua. He is remembered for the discovery of the pancreatic duct (“duct of Wirsung”) during the dissection of a man who had been recently hanged for murder. Instead of publishing the results of his discovery, he engraved a sketch of the duct on a copper plate, from which he made several imprints, and subsequently had them delivered to leading anatomists throughout Europe. (info credit Wikipedia)

PAST ANNUAL DINNER HONOREES
2015 Claudio Bassi, MD
2014 L. William Traverso, MD
2012 Howard Reber, MD
2011 Edward Bradley, III, MD
2010 Hans Beger
2009 Prof. Seiki Matsuno
2008 Andy Warshaw, MD
2007 Charles Frederick Frey, MD
2005 John M. Howard, MD
2004 John Cameron, MD and Fujio Hanyu, MD
SUPPORTERS & EXHIBITORS

EDUCATIONAL GRANTS
The Pancreas Club would like to recognize and thank the following companies for their support through educational grants:

**PLATINUM**
- AbbVie
- ChiRhoClin, Inc.
- Digestive Care, Inc.

**GOLD**
- Celgene Corporation
- Medtronic
- NewLink Genetics

**BRONZE**
- AngioDynamics, Inc.

MARKETING & EXHIBITOR SUPPORT
The Pancreas Club wishes to recognize and thank the following companies for their marketing support:

**Alcresta Pharmaceuticals**
One Newton Executive Park, Suite 100, Newton, MA 02462
PHONE: 617-431-3600 | WEB: www.alcresta.com

Alcresta Pharmaceuticals, Inc. uses novel, enzyme-based technology to address challenges faced by people living with rare diseases and gastrointestinal disorders. Alcresta Pharmaceuticals is determined to make a difference by helping people achieve their potential and experience a better quality of life through the use of novel enzyme-based products.

**AngioDynamics, Inc.**
14 Plaza Drive, Latham, NY 12110
PHONE: 518-795-1400 | WEB: www.angiodynamics.com

AngioDynamics is a leading provider of innovative, minimally invasive medical devices used by professional healthcare providers for vascular access, surgery, peripheral vascular disease and oncology. AngioDynamics’ product lines include market-leading ablation systems, fluid management systems, vascular access products, angiographic products and accessories, angioplasty products, drainage products, thrombolytic products and venous products.

**Celgene Corporation**
86 Morris Avenue, Summit, NJ 07901
PHONE: 908-673-9000 | WEB: www.celgene.com

Celgene Corporation (Nasdaq:CELG) is a global biopharmaceutical company that is helping healthcare providers turn incurable cancers into chronic, manageable diseases through innovative therapies. This dedication to medical progress goes hand-in-hand with our industry-leading patient support and access programs. Together, these aspects form the core of our commitment to patients worldwide.

Supporters & Exhibitors
ChiRhoClin, Inc.
4000 Blackburn Lane, Suite 270, Burtonsville, MD 20866
ChiRhoClin, Inc. is the manufacturer of ChiRhoStim® (Human Secretin for Injection). Our mission is to develop orphan drug products for diagnosing gastrointestinal diseases. ChiRhoStim® is approved for Pancreatic Function Testing (PFT), ERCP’s, and Gastrinoma Testing. Secretin - enhanced MRCP improves pancreatic image quality and function.

Digestive Care, Inc.
1120 Win Drive, Bethlehem, PA 18017
PHONE: 610-882-5950 | FAX: 610-882-0349 | WEB: www.pertzye.com
Digestive Care, Inc. (DCI) is dedicated to developing unique pharmaceutical products to alleviate complications and symptoms of gastrointestinal disorders. DCI’s flagship product, PERTZYE® (pancrelipase) is a unique formulation of enteric-coated microspheres containing pancreatic enzymes buffered with bicarbonate, designed to simulate normal pancreatic function and create a pH microenvironment for optimized biological activity of the enzymes at the site of release.

NewLink Genetics
2700 Via Fortuna, Suite 100, Austin, TX 78746 | WEB: www.linkp.com

RenovoRx
4546 El Camino Road, Suite 203, Los Altos, CA 94022
RenovoRx is developing innovative solutions for targeted delivery of therapeutic agents to selected sites in the peripheral vascular system. The ability to deliver these materials at high concentration to specific visceral organs, without perfusion overlap to other organs, is a central paradigm of our technology.

Vector Surgical
20975 Swenson Drive, Suite 430, Waukesha, WI 53186
Vector Surgical® offers unique medical devices that improve outcomes in cancer surgery. MarginMarker® is a sterile ink kit used by the surgeon to clearly and completely mark excised tissue margins in the OR. Using MarginMarker helps to ensure that tissue margins are interpreted consistently from surgical excision to pathology analysis.

AWARDS SUPPORT
- **Pancreatic Cancer Action Network**: In support of PanCan Research Award
- **Kenneth Warren Foundation**: In support of Kenneth Warren Research Award
- **Arpa Foundation**: In support of John Howard Research Award

Supporters & Exhibitors
SCIENTIFIC PROGRAM

FRIDAY, MAY 20, 2016

6:30 am – 6:30 pm  Registration  Bayview Foyer
7:00 am – 7:45 am  Continental Breakfast  Regatta Pavilion
7:45 am – 8:00 am  Welcome & Introductory Remarks  Bayview Ballroom
William H. Nealon, MD
Yale University Medical Center, New Haven, CT
Michael Farnell, MD
Mayo Clinic, Rochester, MN
Christopher Wolfgang, MD, PhD
John Hopkins University, Baltimore, MD
Nicholas Zyromski, MD
Indiana University School of Medicine, Indianapolis, IN

8:00 am – 9:45 am  SCIENTIFIC SESSION I: Drains / Pancreatic Fistula / Complications of Pancreatic Surgery  Bayview Ballroom

S001 INCIDENCE OF HEPATICOJEJUNOSTOMY STRUCTURE FOLLOWING HEPATICOJEJUNOSTOMY – Francesca M Dimou, MD (Long)

S002 A MULTICENTER, RISK-STRATIFIED, PROSPECTIVE TRIAL OF DRAIN MANAGEMENT FOR PANCREATODUODENECTOMY – Charles M Vollmer, MD (Long)

S003 THE EVOLUTION OF VERONA’S EXPERIENCE: LOOKING FOR A NEW DRAIN AMYLASE VALUE CUT-OFF TO PREDICT PANCREATIC FISTULA. RESULTS OF A PROSPECTIVE STUDY. – Alessandra Pulvirenti, MD (Long)

S004 GRADING OF SURGEON TECHNICAL PERFORMANCE PREDICTS POST-OPERATIVE PANCREATIC FISTULA FOR THE PANCREATICODUODENECTOMY INDEPENDENT OF PATIENT RELATED VARIABLES – Melissa Hogg (Short)

S005 USING THE PANCREATIC DEMONSTRATION PROJECT TO DERIVE A FISTULA RISK SCORE FOR PREOPERATIVE RISK STRATIFICATION IN PATIENTS UNDERGOING PANCREATICODUODENECTOMY – Olga Kantor, MD (Long)

S006 IDENTIFICATION OF RISK FACTORS OF PANCREATIC EXOCRINE INSUFFICIENCY AFTER PANCREATICODUODENECTOMY USING 13C-LABELED MIXED TRIGLYCERIDE BREATH TEST – Seiko Hirono, MD (Long)

S007 STENT ASSOCIATED INFECTIOUS COMPLICATIONS AFTER PANCREATODUODENECTOMIES CAN BE PREVENTED BY PERIOPERATIVE ANTIBIOTIC THERAPY. – Esther A Biesel (Short)

S008 MODIFIED FRAILTY INDEX PREDICTS MORBIDITY AND MORTALITY AFTER PANCREATICODUODENECTOMY – Harveshp Mogal, MD (Long)

S009 IMPACT OF SARCOPENIA ON SURGICAL OUTCOMES IN PATIENTS UNDERGOING PANCREATICODUODENECTOMY BY USING ENANCED RECOVERY AFTER SURGERY (ERAS) PROTOCOL. – Valeri Sergio, MD (Short)
SCIENTIFIC PROGRAM

9:30 am – 3:45 pm  Exhibits Open  Bayview Foyer
9:45 am – 10:00 am  Break with Exhibitors & Poster Viewing  Bayview Foyer
10:00 am – 11:00 am  SCIENTIFIC SESSION II: Surgical Techniques and Innovations  Bayview Ballroom

MODERATORS: Michael Kendrick, MD & Ugo Boggi, MD

**S010** ROBOTIC WHIPPLE BIOTISSUE CURRICULUM IMPROVES TECHNICAL PERFORMANCE FOR FELLOWS AND HAS CONSTRUCT VALIDITY – Melissa E Hogg *(Long)*

**S011** ROBOTIC VERSUS OPEN PANCREATODUODENECTOMY: A PROPENSITY SCORE-MATCHED ANALYSIS OF PANCREATIC FISTULA – Charles M Vollmer, MD *(Long)*

**S012** ROBOT-ASSISTED VERSUS OPEN PANCREATOCODUODENECTOMY: A CASE-MATCHED STUDY BASED ON CLINICAL RISK SCORE FOR PANCREATIC FISTULA – Niccolo Napoli, MD *(Long)*

**S013** MODIFIED APPLEBY PROCEDURE FOR PANCREATIC TUMORS: THE JOHNS HOPKINS EXPERIENCE – Niek A Peters, BS *(Short)*

**S014** IMPACT OF A NATIONWIDE TRAINING PROGRAM IN LAPAROSCOPIC DISTAL PANCREATECTOMY (LAELAPS) – Marc Besselink *(Short)*

**S015** LAPAROSCOPIC VERSUS OPEN DISTAL PANCREATECTOMY: THE OUTCOMES OF JAPANESE MULTICENTER COMPARATIVE STUDY USING PROPENSITY SCORE-MATCHING – Yoshihiro Miyasaka *(Short)*

11:00 am – 12:00 pm  Poster Rounds with Professors  Regatta Pavilion

PROFESSORS: Matthew Weiss, MD & Horacio Asbun, MD
See page 24 for list of posters.
First 10 Posters marked with ★: Authors will be by their posters to discuss their research poster presentations and Professor will lead short Q&A.

12:00 pm – 1:00 pm  Lunch  Regatta Pavilion

Free lunch for all attendees

1:00 pm – 3:30 pm  SCIENTIFIC SESSION III: Borderline Resectable Pancreatic Cancer / Neo-Adjuvant Therapy  Bayview Ballroom

MODERATORS: Michael Farnell, MD & Ernst Klar, MD

**S016** NEOADJUVANT CHEMOTHERAPY IS ASSOCIATED WITH A SURVIVAL ADVANTAGE IN EARLY STAGE PANCREATIC HEAD CANCER – Waseem Lutfi, BS *(Long)*

**S017** PREOPERATIVE THERAPY FOR PATIENTS WITH PANCREATIC DUCTAL ADENOCARCINOMA UNDERGOING PANCREATODUODENECTOMY: IMPACT OF RADIATION DOSE ON OUTCOMES – Jordan M Cloyd *(Long)*
SCIENTIFIC PROGRAM

S018 DOES RADIOLOGIC RESPONSE CORRELATE TO PATHOLOGIC RESPONSE IN PATIENTS UNDERGOING NEOADJUVANT THERAPY FOR PANCREATIC MALIGNANCY? – Brent T Xia, MD (Long)

S019 SURVIVAL AFTER NEOADJUVANT THERAPY AND RESECTION VERSUS RESECTION ALONE FOR EARLY STAGE PANCREATIC CANCER: A PROPENSITY SCORE MATCHED ANALYSIS IN A NATIONAL COHORT OF PATIENTS – Ali A Makdad, MD, MSc (Short)

S020 ABILITY OF TNM STAGING TO PREDICT OVERALL SURVIVAL AFTER RESECTION OF PANCREATIC ADENOCARCINOMA IN PATIENTS UNDERGOING NEOADJUVANT CHEMOTHERAPY – Katherine E Poruk, MD (Short)

S021 OVERALL SURVIVAL IS INCREASED AMONG STAGE III PANCREATIC ADENOCARCINOMA PATIENTS RECEIVING NEOADJUVANT CHEMOTHERAPY COMPARED TO SURGERY FIRST AND ADJUVANT CHEMOTHERAPY: AN INTENTION TO TREAT ANALYSIS OF THE NATIONAL CANCER DATABASE – Christopher R Shubert, MD (Short)

S022 CARBOHYDRATE ANTIGEN 19-9 IN ANATOMICALLY RESECTABLE, EARLY STAGE PANCREATIC CANCER IS INDEPENDENTLY ASSOCIATED WITH DECREASED OVERALL SURVIVAL AND AN INDICATION FOR NEOADJUVANT THERAPY: A NATIONAL CANCER DATABASE STUDY. – John R Bergquist, MD (Long)

S023 PATTERN OF CA19-9 RESPONSE TO NEOADJUVANT CHEMOTHERAPY IN LOCALLY ADVANCED, BORDERLINE RESECTABLE PANCREATIC CANCER PREDICTS PROGRESSION – J. Bart Rose, MD (Long)

S024 NEOADJUVANT TREATMENT WITH FOLFIRINOX FOR RESECTABLE AND BORDERLINE RESECTABLE PANCREATIC DUCTAL ADENOCARCINOMA: FEASIBILITY AND CLINICOPATHOLOGICAL IMPLICATIONS – Nigel B Jamieson, PhD (Long)

S025 DOWNSTAGING OF LIVER METASTASES FROM PANCREATIC CANCER FOLLOWING PRIMARY CHEMOTHERAPY: IS SURGICAL RESECTION WORTHWHILE? – Stefano Crippa (Short)

S026 PROGNOSTIC RELEVANCE OF THE TIMING OF INITIATING AND THE COMPLETION OF ADJUVANT THERAPY IN PATIENTS WITH RESECTED PANCREATIC DUCTAL ADENOCARCINOMA – Yoo-Seok Yoon (Short)

S027 ADJUVANT RADIOTHERAPY DOES NOT IMPROVE OUTCOMES FOLLOWING PANCREATICODUODENECTOMY FOR PANCREATIC ADENOCARCINOMA: A MARGIN-STRATIFIED ANALYSIS – Lee M Ocuin, MD (Short)

S028 EXTERNAL RADIATION IS ASSOCIATED WITH IMPROVED SURVIVAL IN RESECTED MARGIN-NEGATIVE STAGE IIB PANCREATIC ADENOCARCINOMA – Olga Kantor, MD (Long)

S029 EXTENDED LONG-COURSE INDUCTION SYSTEMIC CHEMOTHERAPY, CONSOLIDATIVE CHEMORADIATION, AND AGGRESSIVE RESECTION OF “AT-RISK” ANATOMY IS ASSOCIATED WITH SIGNIFICANT SURVIVAL BENEFIT IN STAGE III (BR/LA) PANCREATIC ADENOCARCINOMA: THE MAYO CLINIC EXPERIENCE. – Mark J Truty, MD, MSc (Long)
<table>
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<tr>
<td>3:30 pm – 3:45 pm</td>
<td><strong>Break with Exhibitors &amp; Poster Viewing</strong></td>
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<td>3:45 pm – 5:35 pm</td>
<td><strong>SCIENTIFIC SESSION IV:</strong> Basic Science Bayview Ballroom Studies / Rare Pancreatic Tumors</td>
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<td>MODERATORS: Marshall Baker, MD, MBA &amp; Jens Werner, MD</td>
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<th>Session</th>
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<tr>
<td>S030</td>
<td>TUMOR ESTABLISHMENT AND RATE OF GROWTH IN PATIENT-DERIVED PANCREATIC DUCTAL ADENOCARCINOMA XENOGRAFT MODELS ARE ASSOCIATED WITH ADVERSE CLINICOPATHOLOGICAL FEATURES AND POOR SURVIVAL/OUTCOMES</td>
<td>Ilaria Pergolini (Long)</td>
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<td>S031</td>
<td>HYPERGLYCEMIA IMPACTS TUMOR BIOLOGY AND CHEMOTHERAPY RESPONSE IN PRE-CLINICAL CANCER MODELS AND PATIENTS WITH PANCREATIC CANCER</td>
<td>Avinoam Nevler (Long)</td>
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<td>S032</td>
<td>AN INDEL DNA SEQUENCE EMBEDDED IN THE WEE1 REGULATORY BINDING SITE CORRELATES WITH INCREASED CANCER RISK IN FAMILY MEMBERS OF PANCREATIC CANCER PATIENTS</td>
<td>Avinoam Nevler (Long)</td>
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<td>S033</td>
<td>RESTITUTION OF TUMOR SUPPRESSOR MIR-145 USING MAGNETIC NANOPARTICLES INHIBITS PANCREATIC CANCER</td>
<td>Stephen W Behrman, MD (Short)</td>
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<td>S034</td>
<td>A PERSONALIZED APPROACH TO ADJUVANT THERAPY: CYTOPLASMIC HUR STATUS PREDICTS DISEASE FREE SURVIVAL AFTER RESECTION FOR PANCREATIC DUCTAL ADENOCARCINOMA</td>
<td>Talar Tatarian, MD (Short)</td>
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<td>S035</td>
<td>T-CELL INFILTRATE AS A SIMPLE TOOL TO PREDICT INTERMEDIATE TERM SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA</td>
<td>McKenzie E Bedra, MPH (Short)</td>
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<td>S036</td>
<td>ANALYSIS OF 337 PATIENTS WITH SOLID PSEUDOPAPILLARY TUMORS OF THE PANCREAS: ROLE FOR SURGERY IN METASTATIC DISEASE</td>
<td>Zeljka Jutric, MD (Short)</td>
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<td>S037</td>
<td>PERIAMPULLARY CANCERS: HISTOPATHOLOGIC SUBTYPE IS A STRONGER DETERMINANT OF PATIENT SURVIVAL THAN ANATOMIC LOCATION</td>
<td>Jennifer L Williams, MD (Long)</td>
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<td>S038</td>
<td>PANCREATICODUODENECTOMY FOR METASTATIC PANCREATIC NEUROENDOCRINE TUMOR</td>
<td>Richelle T Williams, MD (Long)</td>
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<td>S039</td>
<td>SURGICAL OUTCOMES OF RESECTED FUNCTIONAL PANCREATIC NEUROENDOCRINE TUMORS: A SINGLE INSTITUTION EXPERIENCE</td>
<td>Ammar A Javed, MD (Long)</td>
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6:30 pm – 9:30 pm | **Pancreas Club Annual Reception & Dinner** 50th Anniversary Celebration. See page 12 for details | Reception: Banyan Court  Dinner: Regatta Pavilion |
SATURDAY, MAY 21, 2016

6:45 am – 5:00 pm  Registration  Bayview Foyer
7:00 am – 8:00 am  Continental Breakfast  Regatta Pavilion
8:00 am – 9:45 am  SCIENTIFIC SESSION V: Acute and Chronic Pancreatitis / Disconnected Duct Syndrome / TPIAT
                    MODERATORS: Nicholas Zyromski, MD & Marc Besselink, MD, MSc, PhD

S040 VARIATIONS OF ORAL AND FECAL MICROBIOTA ARE ASSOCIATED WITH AUTOIMMUNE PANCREATITIS – Giulia M Cavestro, MD, PhD (Long)

S041 CLINICAL SIGNIFICANCE OF B CELL-ACTIVATING FACTOR IN AUTOIMMUNE PANCREATITIS – Teru Kumagi, MD, PhD (Short)

S042 OUTCOMES AFTER SALVAGE TOTAL PANCREATECTOMY FOR REFRACTORY CHRONIC PANCREATITIS – William P Lancaster, MD (Long)

S043 COMPARABLE RATE OF LONG TERM INSULIN INDEPENDENCE BETWEEN ADULT PATIENTS UNDERGOING REMOTE AND LOCAL TPIAT – Samuel J Kesseli (Long)

S044 VITAMIN AND IRON DEFICIENCIES ARE COMMON IN PATIENTS WHO UNDERGO TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION FOR CHRONIC PANCREATITIS – Stefanie Owczarski, PAC, MPAS (Short)

S045 IMPACT OF DISCONNECTED PANCREATIC DUCT SYNDROME (DPDS) ON ENDOSCOPIC TREATMENT OUTCOMES IN PANCREATIC FLUID COLLECTIONS (PFCS) – Ji Young Bang, MD, MPH (Long)

S046 EUS-BASED STEP-UP TREATMENT APPROACH IS ASSOCIATED WITH BETTER SYSTEMIC INFLAMMATORY RESPONSE IN WALLED-OFF NECROSIS (WON) – Ji Young Bang, MD, MPH (Long)

S047 TRANSGASTRIC NECROSECTOMY FOR THE MANAGEMENT OF WALLED-OFF PANCREATIC NECROSIS: LONG-TERM OUTCOMES AT A HIGH-VOLUME PANCREATIC CENTER – Andrea Jester, MD (Long)

S048 NOVEL METHOD OF DIRECT ENDOSCOPIC NECROSECTOMY IN THE TREATMENT OF WALLED-OFF PANCREATIC NECROSIS – Anand Singla, MD (Short)

9:30 am – 3:30 pm  Exhibits Open  Bayview Foyer
9:45 am – 10:00 am  Break with Exhibitors & Poster Viewing
10:00 am – 11:00 am **History of Pancreas Club**

**Bayview Ballroom**

PRESENTED BY: William Nealon, MD

The focus of this talk will be the progress in scientific and clinical advances in the evolution of care for patients with diseases of the pancreas over the past half century. We will examine the major contributions made by members of the Pancreas Club over these years and document the development of a truly international membership with major contributions to the scientific literature.

At the conclusion of this session, participants will be able to:
1. Articulate the emergence of diagnoses such as Intraductal Papillary Mucinous Neoplasms and Auto-immune Pancreatitis
2. Recognize great advances in the outcomes after pancreatic resections for Pancreatic Cancer
3. Relate greatly improved outcomes for episodes of severe necrotizing pancreatitis
4. Trace the basic science growth in understanding of the mechanisms involved in pancreatic cancer and in acute pancreatitis
5. Recognize the evolution of minimally invasive pancreatic surgery and in robotic pancreatic surgery

11:00 am – 12:00 pm **Poster Rounds with Professors**

**Regatta Pavilion**

PROFESSORS: Cristina Ferrone, MD & Stefano Crippa, MD, PhD

*See page 36 for list of posters.*

First 10 Posters marked with ★: Authors will be by their posters to discuss their research poster presentations and Professor will lead short Q&A.

12:00 pm – 1:00 pm **Lunch**

**Regatta Pavilion**

Free lunch for all attendees

1:00 pm – 3:00 pm **SCIENTIFIC SESSION VI:**

**Bayview Ballroom**

Centralization / Nomogram / Genetics of Cancer

MODERATORS: Matthew Katz, MD & Roberto Coppola, MD

**S049** CONTINUOUS WOUND INFILTRATION VERSUS EPIDURAL ANALGESIA AFTER OPEN PANCREATIC AND HEPATO-BILIARY SURGERY (POP-UP): A MULTICENTRE, RANDOMISED CONTROLLED, OPEN-LABEL, NON-INFERIORITY TRIAL – Timothy Mungroop, MD, MSc *(Long)*

**S050** PANCREATIC SURGERY AT SAFETY NET HOSPITALS: SHOULD IT BE ABANDONED? – Richard S Hoehn, MD *(Long)*

**S051** ASSESSING THE FINANCIAL TOXICITY ASSOCIATED WITH TREATMENT OPTIONS FOR RESECTABLE PANCREATIC CANCER – Marcelo Cerullo, BA *(Long)*
S052 PHYSIOLOGIC PANCREATIC CANCER IMAGING USING DYNAMIC CONTRAST-ENHANCED MAGNETIC RESONANCE IMAGING (DCE-MRI) – Laura E Fischer, MD, MS (Long)

S053 THE EFFECT OF CENTRALIZATION ON PROGNOSIS: OPERATED PANCREATIC DUCTAL ADENOCARCINOMA (PDA) PATIENTS IN FINLAND 2002-2008. – Reea Ahola (Long)

S054 EFFECT OF ANGIOTENSIN SYSTEM INHIBITORS ON OVERALL SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA PATIENTS – Hao Liu, MD (Long)

S055 A NEW NOMOGRAM BETTER STRATIFIES PATIENTS WITH RESECTED PANCREATIC DUCTAL ADENOCARCINOMA THAN DOES THE AJCC STAGING: AN ANALYSIS OF 3,473 PATIENTS FROM THE PANCREAS SURGERY CONSORTIUM – Ammar A Javed, MD (Long)

3:00 pm – 3:15 pm Break with Exhibitors & Poster Viewing

3:15 pm – 5:00 pm SCIENTIFIC SESSION VII: IPMN Bayview Ballroom
MODERATORS: Christopher Wolfgang, MD, PhD & Kyoichi “Tony” Takaori, MD, PhD

S056 BIOMARKERS FOR DETECTION OF HIGH GRADE DYSPLASIA AND CARCINOMA-IN-SITU IN IPMN – Ehab N Abdelfatah, MD (Short)

S057 LOW PROGRESSION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS WITH WORRISOME FEATURES AND HIGH-RISK STIGMATA UNDERGOING NON-OPERATIVE MANAGEMENT: A MID-TERM FOLLOW-UP ANALYSIS – Stefano Crippa (Long)

S058 CIRCULATING EPITHELIAL CELLS IN INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS AND CYSTIC PANCREATIC LESIONS – Katherine E Poruk, MD (Long)

S059 MULTI-INSTITUTIONAL STUDY ON THE NATURAL HISTORY OF LARGE-SIZED(?3CM) BRANCH-DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM – Alexandra M Roch, MD, MS (Short)

S060 PATTERNS OF RECURRENCE AND LONG-TERM OUTCOMES IN PATIENTS WHO UNDERWENT PANCREATECTOMY FOR INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS WITH HIGH GRADE DYSPLASIA: IMPLICATIONS FOR SURVEILLANCE – Aaron U Blackham, MD (Long)

S061 ELEVATED SERUM CA19-9 IN BRANCH-DUCT IPMN IS A HIGHLY-SPECIFIC PREDICTOR OF INVASIVE CANCER – Vicente Morales-Oyarvide, MD, MPH (Short)

S062 LOCAL PROGRESSION IN THE PANCREATIC REMNANT FOLLOWING RESECTION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN) OF THE PANCREAS OCCURS BY ONE OF THREE DISTINCT MECHANISMS – Antonio Pea, MD (Long)
**S063** THE PATTERN OF RECURRENCE OF INVASIVE-IPMN IS DIFFERENT THAN CONVENTIONAL PDAC – Neda Rezaee, MD (Long)

**S064** NON-OPERATIVE MANAGEMENT OF LOW-RISK BRANCH-DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS IS SAFE IN THE LONG-TERM (> 5 YEARS) FOLLOW-UP – Stefano Crippa (Long)

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5:00 pm – 5:30 pm **PC General Business Meeting & Awards Announcements**  
Presentation of three $1,000 awards and two $500 awards (4 for resident/fellow; 1 for junior faculty)  
*Bayview Ballroom*

5:30 pm – 6:30 pm **Wine & Cheese Awards Reception**  
*Sunset Terrace*
Posters are located in Regatta Pavilion

The ★ symbol indicates Poster of Distinction and they will be identified on the poster board by GOLD dot. Authors will be available for short oral presentations during the “Poster Rounds with Professors”.

Complete Poster Abstract descriptions are available online at www.pancreasclub.com

FRIDAY, MAY 20, 2016

★ P001 EARLY LESSONS FROM THE JEFFERSON PANCREAS TUMOR REGISTRY (JPTR) Theresa P Yeo, PhD, MPH, AOCNP, Harish Lavu, MD, FACS, Jennifer Brumbaugh, MA, Jordan M Winter, MD, FACS, Jonathan R Brody, PhD, Charles J Yeo, MD, FACS; Thomas Jefferson University, Philadelphia, US

★ P002 PREDICTIVE MARKERS OF MALIGNANCY IN INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS: EXPLORING THE VALUE OF NEUTROPHIL-TO-LYMPHOCYTE RATIO. Georgios Gemenetzis, MD, Fabio Bagante, MD, James F Griffin, MD, Neda Rezaee, MD, Ammar A Javed, MD, Lindsay L Manos, MPAS, Anne Marie Lennon, MD, PhD, Laura D Wood, MD, PhD, Ralph H Hruban, MD, Atif Zaheer, MD, Elliot K Fishman, MD, Nita Ahuja, MD, John L Cameron, MD, Matthew J Weiss, MD, Jin He, MD, PhD, Christopher L Wolfgang, MD, PhD; Johns Hopkins University - School of Medicine, Baltimore, US

★ P003 THE EVOLUTION AND IMPACT OF LYMPH NODE DISSECTION DURING PANCREATICODUODENECTOMY FOR PANCREATIC CANCER Mariam F Eskander, MD¹, Susanna W de Geus, BS¹, Gyulnara G Kasumova, MD¹, Sing Chau Ng, MS¹, Wadhah B Al-Refaie, MD², Gamze Ayata, MD³, Jennifer F Tseng, MD, MPH¹; ¹Department of Surgery, Beth Israel Deaconess Medical Center, ²Department of Surgery, Georgetown University Hospital, ³Department of Pathology, Beth Israel Deaconess Medical Center, Somerville, US

★ P004 PANCREATIC MUCINOUS CYSTIC NEOPLASMS (MCN) DURING PREGNANCY: SERIES OF 29 OPERATED CASES SHOWS HIGH RATE OF MALIGNANT TRANSFORMATION, SUGGESTING OPERATIVE MANAGEMENT DURING SECOND TRIMESTER Judith Millastre Bocos¹, Anne Antila², Awad Shamali³, Margaret Geri Keane⁴, Linda N Nilsson⁵, Monica Marijnissen Van Zanten⁶, Cristina Verdejo Gil⁷, Marco Del Chiaro⁸, Johanna Laukkanen²; ¹Miguel Servet University Hospital, ²Tampere University Hospital, ³Southampton University Hospital, United Kingdom, ⁴Institute for Liver and Digestive Health, University College London, United Kingdom, ⁵Department of Surgery, Karolinska University Hospital, Sweden, ⁶Nijmegen University Hospital, Netherland, ⁷Ciudad Real University Hospital, Zaragoza, ES

★ P005 RECENT-ONSET DIABETES IN INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM IS AN INDEPENDENT PREDICTOR OF INVASIVE CANCER Vicente Morales-Oyarvide, MD, MPH, Mari Mino-Kenudson, MD, Cristina R Ferrone, MD, Andrew L Warshaw, MD, Keith D Lillemoe, MD, Carlos Fernandez-del Castillo, MD; Massachusetts General Hospital, Harvard Medical School, Boston, US

★ P006 PANCREATITIS AFTER PANCREAS RESECTION PREDICTS CLINICALLY RELEVANT POSTOPERATIVE PANCREATIC FISTULA Christian Kuhlbrey, MD, Nikki Samiei, BS, Olivia Sick, BS, Frank Makowiec, MD, Ulrich T Hopt, MD; Universitätsklinikum Freiburg, Freiburg, DE
**P007** THE VALUE OF (18)FLUORO-DEOXYGLUCOSE POSITRON EMISSION TOMOGRAPHY/COMPUTED TOMOGRAPHY AS PREOPERATIVE PREDICTOR OF SURVIVAL IN RESECTED PANCREATIC CANCER. Ilaria Pergolini, MD, Stefano Crippa, Matteo Salgarello, Giacomo Ruffo, Giulio Belfiori, Antonio E Spinelli, Giuseppe Zamboni, Alessandro Pucci, Massimo Falconi; 1Department of Surgery, Massachusetts General Hospital, Boston MA, USA, 2Division of Pancreatic Surgery, San Raffaele Scientific Institute, Vita-Salute University, Milan, Italy, 3Department of Nuclear Medicine, Ospedale Sacro Cuore-Don Calabria, Negrar, Italy, 4Department of Surgery, Ospedale Sacro Cuore-Don Calabria, Negrar, Italy, 5Department of Surgery, Universita’ Politecnica delle Marche, Ancona, Italy, 6Department of Medical Physics and Experimental Imaging, San Raffaele Scientific Institute, Milan, Italy, 7Department of Pathology, Ospedale Sacro Cuore-Don Calabria, Negrar, Italy, Boston, US

**P008** LEARNING CURVE EFFECT ON ‘STEP UP APPROACH’ FOR MANAGEMENT OF SEVERE ACUTE PANCREATITIS. Rahul Gupta, Sunil Shenvi, Raghavendra Y Babu, Abhishek Chandra, Surinder Singh Rana, Mandeep Kang, Deepak Kumar Bhasin, Rajinder Singh, Rajesh Gupta; 1Surgical Gastroenterology Division, Department of General Surgery, PGIMER, Chandigarh, India, 2Transplant surgery division, Department of Surgery, Medical University of South Carolina, Charleston, SC, US, 3Medanta, the Medicity, Gurgaon, India, 4Department of Gastroenterology, PGIMER, Chandigarh, India, 5Department of Radiology, PGIMER, Chandigarh, India, Chandigarh, IN

**P009** SURGICAL TRANSGASTRIC NECROSECTOMY FOR SYMPTOMATIC PANCREATIC NECROSIS IN ACUTE NECROTIZING PANCREATITIS. Jessica L Cioffi, MD, Jose G Trevino, Steven J Hughes, MD, Kevin E Behrns, MD; University of Florida, Gainesville, US

**P010** EFFECT OF GEMCITABINE PLUS ABRAXANE ON PANCREATIC DUCTAL ADENOCARCINOMA XENOGRAFT MODELS. Eugenio Morandi, MD, Michela Monteleone, MD, Marco Castoldi, MD, Gian Andrea Vignati, MD; O.U. of General Surgery, Azienda Ospedaliera G. Salvini, Rho, Milan, Italy, Lecco, IT

**P011** ABSOLUTE QUANTITATION OF CIRCULATING TUMOR DNA IN PANCREATIC DUCTAL ADENOCARCINOMA. Naoto Hadano, MD, Yoshiaki Murakami, MD, Kenichiro Uemura, MD, Naru Kondo, MD, Naoya Nakagawa, MD, Taijiro Sueda, MD, Eiso Hiyama, MD; Hiroshima University, Hiroshima, JP

**P012** HUR DEPENDENT INHIBITION OF PARG ENHANCES PARP INHIBITOR THERAPY FOR DNA REPAIR PROFICIENT AND DEFICIENT PANCREATIC CANCER CELLS. Cinthya S Yabar, MD, Saswati N Chand, PhD, Mahsa Zarei, PhD, Talar Tatarian, MD, Akshay R Kamath, Matthew J Schiewer, PhD, Carmella Romeo, Joseph A Cozzitorto, Nicole Meisner-Kober, PhD, Eric Londo, PhD, Isidore Rigoutsos, PhD, Karen Knudsen, PhD, John M Pasic, PhD, Charles J Yeo, MD, Jordan M Winter, MD, Jonathan R Brody, PhD; 1Department of Surgery, Jefferson Pancreas, Biliary and Related Cancer Center, Thomas Jefferson University, Philadelphia, US

**P013** EFFICACY OF TUMOR-TARGETING SALMONELLA TYPHIMURIUM A1-R AGAINST A SYNGENEIC PANCREATIC CANCER MOUSE MODEL IS ASSOCIATED WITH CD8+ T CELL INFILTRATION. Takashi Murakami, Yukihiro Hiroshima, Yong Zhang, Ming Zhao, Tasuku Kiyuna, Ryusei Matsuyama, Takashi Chishima, Kuniya Tanaka, Michael Bouvet, Itaru Endo, Robert M Hoffman; 1University of California San Diego, AntiCancer, Inc., Yokohama City University, 2University of California San Diego, AntiCancer, Inc. and University of the Ryukyus, 3University of California San Diego, 4University of California San Diego and AntiCancer, Inc., La Jolla, US


**P014** TARGETED THERAPY IN PANCREATIC CANCER UTILIZING THE NOVEL SMALL MOLECULE DRUG CONJUGATE SW V-49 IN COMBINATION WITH STANDARD CHEMOTHERAPY  
Kerri A Ohman, MD, Yassar M Hashim, MD, Timothy M Nywening, MD, Darren R Cullinan, MD, Suwanna Vangveravong, PhD, Dirk Spitzer, PhD, William G Hawkins, MD; Washington University School of Medicine in St. Louis, Saint Louis, US

**P015** TARGETING PAK1 IN PANCREATIC STELLATE CELLS INCREASES PANCREATIC CANCER SURVIVAL  
Dannel Yeo, Hong He, Graham Baldwin, Mehrdad Nikfarjam; University of Melbourne, Heidelberg, AU

**P016** MORPHOLOGIC MODELLING DISCLOSES A SEQUENCE OF EMT-EVENTS AT THE INVASIVE FRONT OF PANCREATIC DUCTAL ADENOCARCINOMA  
Louisa Bolm, candmed\(^1\), Peter Bronsert, MD\(^2\), Moritz Bader, MD\(^2\), Kathrin Enderle-Ammour, MD\(^2\), Dirk Bausch, MD\(^1\), Tobias Keck, MD\(^1\), Ulrich F Wellner, MD\(^1\); 'Sugery, UKSH Campus Lubeck, \(^2\)Pathology, University of Freiburg, Lubeck, DE

**P017** ADDING TARGETED INHIBITION OF PI3K AND MAPK SIGNALING PATHWAYS TO STANDARD CHEMOTHERAPY IN EXPERIMENTAL PANCREATIC CANCER  
Niranjan Awasthi, PhD, Margaret A Schwarz, Roderich E Schwarz; IUSM South Bend, Goshen, US

**P018** CRISPR-KNOCKOUT OF HUR RENDERS PANCREATIC CANCER CELLS INCAPABLE OF GROWTH IN VITRO AND IN VIVO  
Talar Tatarian, MD, Shruti Lal, PhD, Edwin Cheung, Nicole C Mambelli-Lisboa, Mahsa Zarei, PhD, Saswati Chand, Carmella Romeo, Kevin O’Hayer, MD, PhD, Joseph A Cozziortto, Charles J Yeo, MD, PhD, Jordan M Winter, MD, Jonathan R Brody, PhD; Department of Surgery, Division of Surgical Research; Jefferson Pancreas, Biliary and Related Cancer Center; Jefferson Medical College; Thomas Jefferson University, Philadelphia, PA, Philadelphia, US

**P019** MINNELIDE SYNERGIZES WITH TRAIL TO INDUCE CELL DEATH IN CANCER ASSOCIATED FIBROBLASTS AND THUS DEPLETE STROMA.  
Shrey Modi, Nikita S Sharma, John George, Bhuwan Giri, Vikas Dudeja, Sulagna Banerjee, Ashok Saluja; University of Minnesota, Minneapolis, US

**P020** IGF-II AND NSC-631570 COMPOUNDS AFFECT PMP22 GENE EXPRESSION IN PANCREATIC DUCTAL ADENOCARCINOMA. COULD BE THE NEW TARGET FOR BOTH CHEMO-RESISTANCE AND NEURONAL INVASION?  
Niccola Funel, PhD\(^1\), Serena Pelliccioni, BSc\(^2\), Maria Denaro, PhD\(^2\), Andrea Cacciato Insilla, MD\(^2\), Luca Pollina, MD\(^2\), Daniela Campani, MD\(^2\), Ugo Boggii\(^1\); ‘Department of Translational Research and New Technologies in Medicine and Surgery, \(^2\)Division of Surgical Pathology, University of Pisa, Italy, Pisa, US

**P021** DOES ABERRANT RIGHT HEPATIC ARTERIAL ANATOMY IMPACT THE COMPLICATION RATE OR SURVIVAL FOLLOWING RESECTION OF THE PANCREATIC HEAD?  
Kimberly A Bertens, MD, MPH\(^1\), Jesse Clanton, MD\(^2\), Angelena Crown, MD\(^1\), Adnan Alseidi, MD, EdM\(^1\), Biehl Thomas, MD\(^1\), William S Helton, MD\(^1\), Flavio G Rocha, MD\(^1\); ‘Section of General, Thoracic, and Vascular Surgery, Virginia Mason Medical Center, Seattle, Washington, USA, \(^2\)Division of General Surgery, West Virginia University, Charleston, West Virginia, USA, Seattle, US

**P022** IMPROVED SURVIVAL FOLLOWING PANCREATIC CANCER AMONG PATIENTS RECEIVING METFORMIN  
Marcelo Cerullo, Faiz Gani, Joseph K Canner, Timothy M Pawlik; Johns Hopkins University School Of Medicine, Baltimore, US
P023 EXTENT OF RESECTION DOES NOT AFFECT THE NATIONAL FAILURE TO TREAT LOCALIZED PANCREAS CANCER WITH POST-RESECTION ADJUVANT CHEMOTHERAPY

John R Bergquist, MD, Christopher R Shubert, MD, Tommy Ivanics, MD, Rory L Smoot, Michael L Kendrick, MD, David M Nagorney, MD, Michael B Farnell, MD, Mark J Truty, MD; Mayo Clinic, Rochester, US

P024 CAN PREOPERATIVE ALBUMIN VALUE BE USED TO IMPROVE PREOPERATIVE TREATMENTS IN PATIENTS WITH PANCREATIC ADENOCARCINOMA? A MULTICENTER RETROSPECTIVE ANALYSIS OF PREOPERATIVE ALBUMIN VALUES.

Alessandro Coppola, MD1, John A Stauffer, MD2, Segersward Ralf, MD3, Marco Del Chiaro, MD3, Horacio J Asbun, MD3; 1HPB Unit, Department of General Surgery, Catholic University of Rome, A. Gemelli Hospital, Italy, 2Department of General Surgery, Mayo Clinic Jacksonville, FL, USA, 3Pancreatic Surgery Unit, Division of Surgery, Department of Clinical Science, Intervention and Technology (CLINTEC), Karolinska Institute, Stockholm, Sweden, Rome, IT

P025 ASSOCIATION OF RURAL RESIDENCE WITH DISPARITIES IN PANCREATIC ADENOCARCINOMA: A SEER ANALYSIS

Douglas S Swords, MD, Lisa M Pappas, MS, Sean J Mulvihill, MD, Courtney L Scaife, MD; University of Utah, Salt Lake City, US

P026 INCIDENCE AND MANAGEMENT OF PANCREATIC EXOCRINE INSUFFICIENCY AFTER NEOADJUVANT THERAPY AND RESECTION FOR BORDERLINE RESECTABLE PANCREATIC CANCER

June S Peng, MD, Trang K Nguyen, MD, R. Matthew Walsh, MD, Mohamed E Abazeed, MD, PhD, Gareth Morris-Stiff, MD, PhD; Cleveland Clinic Foundation, Cleveland, US

P027 NEOADJUVANT CHEMOTHERAPY IMPROVES SURVIVAL IN PATIENTS UNDERGOING SURGICAL RESECTION OF PANCREATIC CANCER

Keita Wada, MD, Keiji Sano, Fumihiko Miura, Tadahiro Takada; Teikyo university school of medicine, Tokyo, JP

P028 ADJUVANT INTERFERON-BASED CHEMORADIATION FOLLOWED BY GEMCITABINE FOR PANCREATIC ADENOCARCINOMA: LONG-TERM FOLLOW UP

Kerri A Ohman, MD1, Esther Lu, PhD1, David C Linehan2, Marcus C Tan, MD3, Benjamin R Tan, MD3, William G Hawkins, MD1; 1Washington University School of Medicine in St. Louis, 2University of Rochester Medical Center, 3University of South Alabama Medical Center, Saint Louis, US

P029 ONCOLOGIC VALIDATION OF YONSEI CRITERIA IN TREATING LEFT SIDED PANCREATIC CANCER

Chang Moo Kang, MD, PhD, Sung Hwan Lee, Ho Kyoung Hwang, Woo Jung Lee; Yonsei University College of Medicine, Seoul, KR

P030 YONSEI CRITERIA HARBORS MICROENVIRONMENT TO PREVENT TUMOR RECURRENT IN RESECTED LEFT-SIDED PANCREATIC CANCER; A POTENTIAL LINKAGE TO INTRATUMORAL FOXP3+/ CD8+ RATIO

Ho Kyoung Hwang, MD, Sung Hwan Lee, Hyoung-IL Kim, Se Hoon Kim, Chang Moo Kang, MD, PhD, Woo Jung Lee, MD, PhD; Yonsei University College of Medicine, Seoul, KR

P031 PANCREATIC CYSTIC NEOPLASMS: A SINGLE INSTITUTION EXPERIENCE

Alessandra Landmann, MD, T Garwe, PhD, Z Shakir, BS, Morgan M Bonds, MD, N Bhandari, MPH, J L Calisto, MD, R G Postier, MD; University of Oklahoma, Pittsburgh, US
P032 SYSTEMATIC REVIEW OF OUTCOMES INCLUDING RESECTION RATE AFTER FOLFIRINOX TREATMENT IN PATIENTS WITH LOCALLY ADVANCED Pancreatic Cancer. Steffi J Rombouts1, Marieke S Walma1, Jantien A Vogel2, Bengt L Rijssen2, Hanneke Wilmink2, Hjalmar C van Santvoort3, Nadia Haj Mohammad4, Quintus I Molenar5, Mark G Besselink2; 1University Medical Center Utrecht, 2Academic Medical Center Amsterdam, 3St Antonius hospital, Nieuwegein, Utrecht, NL

P033 PHASE II STUDY OF INDUCTION CHEMOTHERAPY WITH OxALIPLATIN AND GEMCITABINE FOLLOWED BY HIGH WEEKLY DOSE OF GEMCITABINE CONCURRENT TO RADIATION THERAPY IN PATIENTS WITH BORDERLINE RESECTABLE AND UNRESECTABLE LOCALLY ADVANCED Pancreatic Cancer. Michele Fiore, MD1, Sergio Valeri, MD2, Lucio Trodella, MD1, Barnaba Floreno, MD1, Luca Eolo Trodella, MD1, Rolando Maria D’Angelillo, MD1, Sara Ramella, MD1, Roberto Coppola, MD2; 1Radiation Oncology, Campus Bio-Medico University, 2General Surgery, Campus Bio-Medico University, Rome, IT

P034 IS INTRA-ARTERIAL WITH TARGETED DELIVERY OF GEMCITABINE SAFE IN TREATMENT OF PATIENTS WITH LOCO-REGIONAL Pancreatic Tumors? Alexander Rosemurgy1, Sharona Ross1, Paul Vitulli1, Reza Malek2, Jiali Li2, Ramtin Agah2; 1Florida Hospital Tampa, 2El Camino Hospital, Los Altos, US

P035 IMPACT OF TREATMENT SEQUENCING ON SITES OF FIRST RECURRANCE IN PATIENTS WITH LOCALIZED Pancreatic Cancer Chad Barnes, MD, Mohammed Aldakkak, MD, Kathleen Christians, MD, Paul Ritch, MD, Ben George, MD, Fabian Johnston, MD, Beth Erickson, MD, Parag Tolat, MD, William Foley, MD, Douglas Evans, Susan Tsai, MD; Medical College of Wisconsin, Milwaukee, US

P037 EARLY HYPERGLYCEMIA FOLLOWING PANCREATICODUODENECTOMY FOR Pancreatic Cancer IS A MARKER FOR TUMOR RECURRENTE Sushanth Reddy, MD, Thomas N Wang, MD, PhD, Carlo C Contreras, MD, Martin J Hesslin, MD, MSHA; University of Alabama at Birmingham, Birmingham, US

P038 ANALYSIS OF CIRCULATING MICRORNAS TO PREDICT DISEASE PROGRESSION DURING FOLFIRINOX THERAPY IN UNRESECTABLE Pancreatic Ductal Adenocarcinoma. Niccola Funel, PhD1, Laura L Meijer, MD2, Ingrid Garajova, MD3, Chiara Capparello, MD4, Tessy L Le Large5, Enrico Valse1, MD4, Gunter Kazemier, MD5, Elisa Govannetti, MD, PhD6, Ugo Boggi, MD7; 1Cancer Pharmacology Lab, AIRC-Start-Up, University Hospital of Pisa, Pisa, Italy and Department of Translational Research and The New Technologies in Medicine and Surgery, 2Department of Medical Oncology, VUmc, Amsterdam, The Netherlands and Department of Surgery, VUmc, Amsterdam, The Netherlands, 3Department of Medical Oncology, VUmc, Amsterdam, The Netherlands and Department of Medical Oncology, University Hospital of Bologna, Bologna, Italy, 4Department of Medical Oncology, University Hospital of Pisa, Pisa, Italy, 5Department of Surgery, VUmc, Amsterdam, The Netherlands, 6Cancer Pharmacology Lab, AIRC-Start-Up, University Hospital of Pisa, Pisa, Italy and Department of Medical Oncology, VUmc, Amsterdam, The Netherlands, 7Department of Translational Research and The New Technologies in Medicine and Surgery, Pisa, IT

P039 METASTATIC NON-RENAL CELL CARCINOMA TO THE PANCREAS: 20-YEAR SINGLE CENTER EXPERIENCE Waleed O Gibreel, MBBS, Rushin R Brahmbhatt, MD, Michael B Farnell, MD, FACS, Michael L Kendrick, MD, FACS, Rory L Smoot, MD, FACS, Mark J Truty, MD, FACS, David M Nagorney, MD, FACS; Department of Subspecialty General Surgery, Mayo Clinic College of Medicine, Rochester, MN, USA, Rochester, US
**P040** RESECTED PANCREATIC MCN HAVE A BETTER PROGNOSIS THAN IPMN: A LARGE SINGLE INSTITUTION SERIES
James F Griffin, MD, Andrew J Page, MD, Georges Samaha, MD, Adrienne Christopher, Feriyi Bhaijee, MD, Maryam K Pezhohou, MD, Ralph H Hruban, MD, Martin A Makary, MD, Kenzo Hirose, MD, Frederick E Eckhauser, MD, Timothy M Pawlik, MD, MPH, PhD, John L Cameron, MD, Christopher L Wolfgang, MD, PhD, Matthew J Weiss, MD; 1Department of Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, 2Department of Pathology, Johns Hopkins University School of Medicine, Baltimore, MD, Baltimore, US

**P041** PANCREATIC MUCINOUS CYSTIC NEOPLASMS (MCN) OF ANY SIZE, WITHOUT WORRISOME FEATURES OR SYMPTOMS CAN BE SAFELY SURVEYED IN WOMEN BUT SHOULD BE RESECTED IN MEN: A MULTINATIONAL COHORT STUDY INCLUDING 185 PATIENTS
Margaret G Keane, MSc, MRCP, Awad Shamali, Linda N Nilsson, Anne Antila, Judith Millastre Bocos, Monica Marijissen Van Zanten, Christina Verdejo Gil, Yrjo Vaalavuo, Toby Hoskins, Stuart M Robinson, Guralp O Ceyhan, Mohammed Abuhilal, Stephen P Pereira, Johanna Laukkarinen, Marco Del Chiaro; 1Institute of Liver and Digestive Health, University College London, UK, 2Southampton University Hospital, UK, 3Department of Surgery, Karolinska University Hospital, Sweden, 4Tampere University Hospital, Finland, 5Miguel Servet University Hospital, Zaragoza, Spain, 6Nijmegen University Hospital, Netherlands, 7Ciudad Real University Hospital, Spain, 8Freeman Hospital, Newcastle, UK, 9Chirurgische Klinik, Klinikum rechts der Isar, Technische Universitat Munchen, Germany, London, GB

**P042** FOLFIRINOX AND GEMCITABINE/NAB-PACLITAXEL DEMONSTRATE IMPROVED SURVIVAL IN LOCALLY ADVANCED UNRESECTABLE PANCREATIC ADENOCARCINOMA
Filip Bednar, Lee M Ocuin, Jennifer Steve, Mazen S Zenati, Sharon Winters, Melissa Hogg, Nathan Bahary, Herbert J Zeh, Amer H Zureikat; 1Division of Surgical Oncology, Department of Surgery, University of Pittsburgh, Pittsburgh, Pennsylvania, 2UPMC Cancer Registry, University of Pittsburgh, Pittsburgh, Pennsylvania, 3Division of Oncology, Department of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania, Pittsburgh, US

**P043** IS THE INCREASE IN PREVALENCE OF PANCREATIC DUCTAL ADENOCARCINOMA ASSOCIATED WITH A CHANGE IN THE DEMOGRAPHIC PROFILE?
Maitham A Moslim, MD, Colin O’Rourke, R Matthew Walsh, MD, FACS, Gareth Morris-Stiff, MBBCh, MCh, PhD, FRCS; Cleveland Clinic Foundation, University Heights, US

**P044** OUTCOMES OF RESECTED MUCINOUS CYSTIC NEOPLASMS OF THE PANCREAS WITH INVASIVE CANCER
Alicia Edwards, BS, Kimberly Bertens, MD, Russell Dorer, MD, PhD, Richard Kozarek, MD, Vincent J Picozzi, MD, Adnan Alseidi, MD, Thomas Biehl, MD, Scott Helton, MD, Flavio G Rocha, MD; Virginia Mason Medical Center, Seattle, US

**P045** CLINICAL SIGNIFICANCE OF PLASMA APOLIPOPROTEIN-AII ISOFORMS AS A SURROGATE OF THE EFFECT OF CHEMORADIOTHERAPY AND PANCREATIC EXOCRINE INSUFFICIENCY FOR PATIENTS WITH LOCALLY ADVANCED PANCREATIC CANCER, PAYING ATTENTION TO MORPHOLOGICAL CHANGE OF PANCREAS
Aoi Hayasaki, MD, Yasuhiro Murata, MD, Masanobu Usui, MD, Naohisa Kuriyama, MD, Yoshinori Azumi, MD, Masashi Kishiwada, MD, Shugo Mizuno, Hiroyuki Sakurai, MD, Shuji Isaji, MD; Department of Hepatobiliary Pancreatic and Transplant Surgery Mie University, Tsu, Mie, JP
P046 CONTRIBUTION OF HISTOLOGICAL EFFECT OF CHEMORADIOThERAPY TO SECURING A SURGICAL MARGIN AND IMPROVING PROGNOSIS IN PATIENTS WITH PANCREATIC ADENOCARCINOMA, CLINICAL APPLICATION OF INTRATUMORAL EXPRESSION OF TENASCIN C AS A POTENTIAL SURROGATE MARKER

Yasuhiro Murata, MD1, Aoi Hayasaki, MD1, Masanobu Usui, MD1, Naohisa Kuriyama, MD1, Masashi Kishiwada, MD1, Shugo Mizuno, MD1, Hiroyuki Sakurai, MD1, Toshimichi Yoshida, MD2, Shuji Isaji, MD1; 1Division of Hepatobiliary Pancreatic and Transplant Surgery, Mie university, 2Division of Matrix Biology, Mie university, Tsu, Mie, JP

P047 THE ROLE OF STAGING LAPAROSCOPY IN PATIENTS WITH PANCREATIC ADENOCARCINOMA: WITHSTANDING THE TEST OF TIME

Zhi Ven Fong, MD1, Winta T Mehtsun, MD, Keith D Lillemoe, Andrew L Warshaw, MD, Carlos Fernandez-del Castillo, David C Chang, PhD, MPH, MBA, Cristina R Ferrone, MD; Massachusetts General Hospital, Natick, US

P048 WHAT IS A BETTER PREDICTOR OF CLINICALLY RELEVANT PANCREATIC FISTULA (CRPF) FOLLOWING PANCREATICODUODENECTOMY (PD): POSTOPERATIVE DAY ONE DRAIN AMYLASE (POD1DA) OR THE FISTULA RISK SCORE (FRS)?

Kimberly A Bertens, MD, MPH1, Angelena Crown, MD1, Jesse Clanton, MD2, Farzad Alemi, MD, MS3, Thomas Biehl, MD1, William S Helton, MD1, Adnan Alseidi, MD, EdM1, Flavio G Rocha, MD1; 1Section of General, Thoracic, and Vascular Surgery, Virginia Mason Medical Center, Seattle, Washington, USA, 2Division of General Surgery, West Virginia University, Charleston, West Virginia, USA, 3Division of General Surgery, University of Missouri, Kansas City, Missouri, USA, Seattle, US

P049 TOTAL NEOADJUVANT THERAPY FOR PANCREATIC CANCER: AN INSTITUTIONAL EXPERIENCE

Ashlie Nadler, MD, Francis SW Zih, MD, MSc, John Hoffman, MD, Elin Sigurdson, MD, PhD, Sanjay Reddy, MD; Fox Chase Cancer Center, Philadelphia, US

P050 VOLUME MATTERS! - BETTER ONE-YEAR RESULTS IN HIGH VOLUME CENTERS

Guido Alsfasser, MD, FACS1, Hanna Leicht2, Christian Gunster2, Bettina M Rau1, Gerhard Schilling1, Ernst Klar, MD, FACS1; 1University of Rostock, 2Research Institute of the Local Health Care Funds (AOK), 3Federal Association of the AOK, Rostock, DE

P052 GERIATRIC ASSESSMENTS IMPROVE PREDICTION OF POST-OPERATIVE DELIRIUM FOLLOWING PANCREATIC RESECTION IN OLDER ADULTS

Andrew B Schneider, MD, Andrew J Benjamin, MD, Mary Buschmann, PhD, Kevin K Roggin, MD, William Dale, MD, PhD; University of Chicago Medicine, Chicago, US

P053 HEADS OR TAILS? PREDICTING SURVIVAL IN PANCREATIC ADENOCARCINOMA BASED ON LOCATION

Gyulnara G Kasumova, MD, Mariam F Eskander, MD, Susanna W de Gues, BS, Daniel Wong, MD, Jacob Rauh, Tara S Kent, MD, A J Moser, Mark P Callery, MD, Jennifer F Tseng, MD, MPH; Beth Israel Deaconess Medical Center, Boston, US

P054 IS LAPAROSCOPIC PANCREATICODUODENECTOMY ASSOCIATED WITH IMPROVED OUTCOMES FOR PANCREATIC CANCER?

John A Stauffer, MD1, Alessandro Coppola, MD2, Kabir Mody, MD1, Elizabeth Johnson, MD1, Horacio J Asbun, MD1; 1Mayo Clinic, 2Universita Cattolica del Sacro Cuore, Jacksonville, US
P055 PNEUMONIA IS ASSOCIATED WITH A HIGH RISK OF DEATH FOLLOWING PANCREATECTOMY Ramzy T Nagle1, Harish Lavu, MD2, Ernest L Rosato, MD2, Charles J Yeo, MD2, Jordan M Winter, MD2; 1Sidney Kimmel Medical College at Thomas Jefferson University, 2Department of Surgery, Thomas Jefferson University, Philadelphia, US

P056 DOES HYPERBILIRUBINEMIA CONTRIBUTE TO ADVERSE PATIENT OUTCOMES FOLLOWING PANCREATODUODENECTOMY? Ben L Zarzaur, MD, MPH1, Nicholas J Zyromski, MD1, Henry A Pitt, MD2, Taylor S Riall, MD3, Bruce L Hall, MD4, Stephen W Behrman, MD5; 1Indiana University School of Medicine, 2Temple University, 3University of Arizona, 4Washington University, 5University of Tennessee Health Science Center, Memphis, US

P057 EMPIRIC POST-OPERATIVE METOCLOPRAMIDE REDUCES DELAYED GASTRIC EMPTYING AFTER PANCREATICODUODENECTOMY Brandon C Chapman, MD, James McCullough, BS, Douglas Overbey, MD, Alessandro Paniccia, MD, Cheryl Meguid, DNP, Ana Gleisner, MD, PhD, Martin D McCarter, MD, Csaba Gajdos, MD, Richard D Schulick, MD, Barish H Edil, MD; University of Colorado School of Medicine, Denver, US

P058 MULTIDISCIPLINARY DILIGENCE DRAMATICALLY LOWERS SURGICAL SITE INFECTIONS AFTER PANCREATIC RESECTIONS Alexander Rosemurgy, MD, FACS, McWayne Weche, BS, Christian Rodriguez, BS, Darrell Downs, ATC, Jacqueline Whitaker, RN, Indraneil Mukherjee, MD, Sharona Ross, MD, FACS; Florida Hospital Tampa, Tampa, US

P059 NATIONAL EVALUATION OF PATIENT SELECTION CRITERIA FOR MINIMALLY-INVASIVE DISTAL PANCREATECTOMY S Klompmaker1, D van Zoggel1, A A Watkins1, M F Eskander2, M G Besselink3, J F Tseng4, A J Moser1; 1The Pancreas and Liver Institute at Beth Israel Deaconess Medical Center, Boston, USA, 2Surgical Outcomes Analysis & Research at Beth Israel Deaconess Medical Center, Boston, USA, 3Department of Surgery, Academic Medical Center, Amsterdam, the Netherlands, Boston, US

P060 PREOPERATIVE VOLUME-BASED PET PARAMETER, MTV2.5 , AS A POTENTIAL SURROGATE MARKER FOR TUMOR BIOLOGY AND RECURRENCE IN RESECTED PANCREATIC CANCER Sung Hwan Lee, MD, Chang Moo Kang, MD, PhD, Ho Kyoung Hwang, MD, Mi Jin Yun, MD, Woo Jung Lee, MD, PhD; Yonsei University College of Medicine, Seoul, KR

P061 LYMPHOVASCULAR INVASION: AN UNDERAPPRECIATED PROGNOSTIC FACTOR IN PANCREATIC CANCER Jeffrey D Epstein1, Geoffrey Kozak, MD2, Zhi Ven Fong, MD3, Wei Jiang, MD4, Cristina R Ferrone, MD1, Keith Lillemoe, MD3, Harish Lavu, MD2, Charles Yeo, MD2, Carlos Fernandez-del Castillo, MD3, Jordan Winter, MD2; 1Sidney Kimmel Medical College at Thomas Jefferson University, Philadelphia, PA, United States, 2Thomas Jefferson University Hospital, Department of Surgery, Philadelphia, PA, United States, 3Massachusetts General Hospital, Department of Surgery, Boston, MA, United States, 4Thomas Jefferson University Hospital, Department of Pathology, Philadelphia, PA, United States, Philadelphia, US
P062 PANCREATIC FISTULA GRADE B DOES NOT ADVERSELY AFFECT SURVIVAL AFTER PANCREATECTOMY FOR PANCREATIC CANCER: A MULTICENTER OBSERVATIONAL STUDY  Manabu Kawai, MD, PhD; Seiko Hirono, MD, PhD; Ken-ichi Okada, MD, PhD; Yoshiaki Murakami, MD, PhD; Fuyuhiko Motoi, MD, PhD; Masayuki Sho, MD, PhD; Sohei Sato, MD, PhD; Ippei Matsumoto, MD, PhD; Goro Honda, MD, PhD; Michiaki Unno, MD, PhD; Yoshiyuki Nakajima, MD, PhD; Kenichiro Uemura, MD, PhD; A-Hon Kwon, MD, PhD; Takumi Fukimoto, MD, PhD; Masanao Kurata, MD, PhD; Hiroki Yamaue, MD, PhD; Wakahama Medical University; Hiroshima University; Tohoku University Graduate School of Medicine; Nara Medical University; Kansai Medical University; Kobe University Graduate School of Medicine; Tokyo Metropolitan Cancer and Infectious diseases Center Komagome Hospital, Wakayama, JP

P063 MORPHOMETRIC ANALYSIS AND FRAILTY ASSESSMENT IMPROVES PREDICTION OF NSQIP SERIOUS COMPLICATIONS FOLLOWING PANCREATECODOUODENECTOMY IN OLDER ADULTS  Andrew J Benjamin, MD; Andrew Schneider, MD; Mary Buschmann, PhD; Brian Derstine; Jeffrey F Friedman; Stuart Wang, MD; William Dale, MD, PhD; Kevin Roggin, MD; University of Chicago; University of Michigan, Chicago, US

P064 POOR OUTCOMES ASSOCIATED WITH EMERGENCY DEPARTMENT PRESENTATION FOR PANCREATIC CANCER RESECTIONS: THINKING BEYOND HOSPITAL VOLUME  Vishes Mehta, MD, MPH; Patricia Friedmann, MS; Peter Muscarella, MD; Haejin In, MD, MBA, MPH; Montefiore Medical Center; Albert Einstein College of Medicine, Bronx, US

P065 NEOADJUVANT CHEMORADIATION THERAPY FOR PANCREATIC CANCER: AN ANSWER TO PANCREATIC FISTULA?  Luffarelli Paolo, MD; Valeri Sergio, MD; Borzomati Domenico, MD, PhD, FACS; Fiore Michele, MD; Trodella Lucio, MD; Copolla Roberto, MD, FACS; Department of General Surgery, Campus Bio-Medico University, Rome, Rome, IT

P066 COMPARISON OF PATENCY RATES BETWEEN DIFFERENT RECONSTRUCTION WAYS FOLLOWING PORTAL/SUPERIOR MESENTERIC VEIN RESECTION DURING PANCREATECTOMY  Xinglong Dai, Wentao Gao, MD, PhD; Cuncai Dai, MD, PhD; Kuirong Jiang, MD, PhD; Junli Wu, MD, PhD; Qiang Li, MD, PhD; Feng Guo, MD; Jianmin Chen, MD, PhD; Jishu Wei, MD, PhD; Zipeng Lu, MD, MRCSEd; Min Tu, MD, Yi Miao, MD, PhD, FACS, FICS Hon; Pancreas Center, The First Affiliated Hospital of Nanjing Medical University, Nanjing, CN

P067 CLINICOPATHOLOGIC AND SURVIVAL ANALYSIS OF RESECTED AMPULLARY ADENOCARCINOMA  Matthew P Doepker, MD; Zachary J Thompson, PhD; Richard D Kim, MD; Gregory M Springett, MD; Domenico Coppola, MD; Barbara A Centeno, MD; Pamela J Hodul; Moffitt Cancer Center, Tampa, US

P068 LAPAROSCOPIC TOTAL PANCREATECTOMY: SAFETY, FEASIBILITY AND OUTCOMES AT A SINGLE INSTITUTION  Toshiro Masuda, Janani S Reisenauer, Rory L Smoot, Mark J Truty, David M Nagorney, Michael B Farnell, Michael L Kendrick; Mayo Clinic, Rochester, US
P069 NONMEDICAL, NONSURGICAL PREDICTORS OF READMISSIONS AFTER PANCREATECTOMY Motahar Hosseini, Anne M Sill, Artem (Tim) Shmelev, Naeem Goussous, Gopal C Kowdley, Juan A Sanchez, Steven C Cunningham; Saint Agnes Hospital Center, Ellicott City, US

P070 PANCREATODUODENECTOMY WITH VENOUS OR ARTERIAL RESECTION: ARE THE OUTCOMES COMPARABLE? Joel D Beane, MD,1 Michael G House, MD,1 Susan C Pitt, MD, MPH5,2 Taylor S Riall, MD3, Bruce L Hall, MD4, Henry A Pitt, MD6; 1Indiana University, 2University of Wisconsin, 3University of Arizona, 4Washington University, 5Temple University, Indianapolis, US

P071 THE EFFECT OF TREATMENT FACILITY TYPE ON OVERALL SURVIVAL IN SURGICALLY TREATED PANCREAS CANCER Alessandro Paniccia, MD, Patrick W Hosokawa, MS, William Henderson, PhD, Richard D Schulick, MD, MBA, Barish Edil, MD, Martin D McCarter, MD, Csaba Gajdos, MD; University of Colorado Denver, Aurora, US

P072 PREDICTORS OF IN-HOSPITAL MORTALITY FOLLOWING PANCREATECTOMY, INCLUDING A NEW PATIENT-SAFETY INDICATOR Artem (Tim) Shmelev, Anne M Sill, Juan A Sanchez, Steven C Cunningham; Saint Agnes Hospital Center, Ellicott City, US

P073 POSTOPERATIVE CHOLANGITIS AFTER PANCREATEODUODENECTOMY: A RETROSPECTIVE STUDY Hiroki Ueda, MD,1 Daisuke Ban, MD, PhD, Shinji Tanaka, MD, PhD, Minoru Tanabe, MD, PhD; 1Department of Hepatobiliary and Pancreatic Surgery, Tokyo Medical and Dental University, 2Department of Molecular Oncology, Tokyo Medical and Dental University, Tokyo, Bunkyo-ku, JP

P074 PREOPERATIVE CHOLANGITIS DURING BILIARY DRAINAGE INCREASES THE INCIDENCE OF POSTOPERATIVE SEVERE COMPLICATIONS AFTER PANCREATEODUODENECTOMY Yuji Kitahata, MD, Manabu Kawai, MD, PhD, Seiko Hirono, MD, PhD, Ken-ichi Okada, MD, PhD, Motoki Miyazawa, MD, PhD, Atsushi Shimizu, MD, PhD, Hiroki Yamaue, MD, PhD; 2nd department of surgery, Wakayama Medical University, Wakayama, JP

P075 LYMPH NODE EVALUATION FOR TREATMENT OF ADENOCARCINOMA OF THE PANCREAS Schelomo Marmor, PhD, Erin E Burke, MD, Pamela R Portschy, MD, Beth A Virnig, PhD, Eric H Jensen, MD, Todd M Tuttle, MD; University of Minnesota, Saint Paul, US

P076 LYMPH NODE EVALUATION FOR Pancreatic ADENOCARCINOMA AND ITS VALUE AS A QUALITY METRIC Erin E Burke, MD, Schelomo Marmor, PhD, Beth A Virnig, PhD, Todd M Tuttle, MD, Eric H Jensen, MD; University of Minnesota, Saint Paul, US

P077 ROBOTIC-ASSISTED VS. OPEN DISTAL PANCREATECTOMY WITH CELIAC AXIS RESECTION (DPCAR): COMPARISON OF PERIOPERATIVE AND ONCOLOGIC OUTCOMES AT A HIGH-VOLUME COMPREHENSIVE CANCER CENTER Lee M Ocuin, MD, Jennifer L Miller, MD, David L Bartlett, MD, Melissa E Hogg, MD, Herbert J Zeh III, MD, Amer H Zureikat, MD; University of Pittsburgh Medical Center, Pittsburgh, US

P078 SYSTEMATIC USE OF DOUBLE JEJUNAL LOOP RECONSTRUCTION AFTER PANCREATEODUODENECTOMY Marcel C Machado, MD, Marcel Autran Machado, MD; University of sao Paulo, Sao Paulo, BR
P079 PROSPECTIVE RANDOMIZED STUDY COMPARING OUTCOME OF DUODENUM PRESERVING PANCREATIC HEAD CORING WITH DUODENUM PRESERVING PANCREATIC HEAD AND BODY CORING IN CHRONIC PANCREATITIS Vikash Moond1, Rajesh Gupta1, Surinder Singh Rana2, Ritambhra Nada3, Mandeep Kang4, Rajinder Singh1, Deepak Kumar Bhasin2; 1Surgical Gastroenterology Division, Department of General Surgery, PGIMER, Chandigarh, India, 2Department of Gastroenterology, PGIMER, Chandigarh, India, 3Department of Histopathology, PGIMER, Chandigarh, India, 4Department of Radiology, PGIMER, Chandigarh, India, Chandigarh, IN

P080 PRIOR PORTACAVAL SHUNT PANCREATODUODENECTOMY EN-BLOC WITH PORTAL VEIN-SUPERIOR MESENTERIC VEIN RESECTION FOR PANCREATIC CANCER Cuncai Dai, MD, PhD, Jian Liu, Kuirong Jiang, MD, PhD, Junli Wu, MD, PhD, Wentao Gao, MD, PhD, Feng Guo, MD, Jianmin Chen, MD, PhD, Jishu Wei, MD, PhD, Zipeng Lu, MD, MRCSEd, Yi Miao, MD, PhD, FACS, FICSHon; Pancreas Center, The First Affiliated Hospital of Nanjing Medical University, Nanjing, CN

P081 LAPAROSCOPIC PANCREATICO-DUODENECTOMY (LPD) WITH POSTERIOR AND UNCINATE PROCESS FIRST APPROACH Wentao Gao, MD, PhD, Kuirong Jiang, MD, PhD, Junli Wu, MD, PhD, Qiang Li, Feng Guo, MD, PhD, Jianmin Chen, MD, PhD, Jishu Wei, MD, PhD, Zipeng Lu, MD, MRCSEd, Min Tu, MD, PhD, Xinglong Dai, Cuncai Dai, MD, PhD, Yi Miao, MD, PhD, FACS, FICSHon; Pancreas Center, The First Affiliated Hospital of Nanjing Medical University, Nanjing, CN

P082 PANCREATODUODENECTOMY AFTER ROUX-EN-Y GASTRIC BYPASS June S. Peng, MD, Ricard Corcelles, MD, PhD, Matthew Poturalski, MD, Namita Gandhi, MD, R. Matthew Walsh, MD, Stacy A Brethauer, MD, Gareth Morris-Stiff, MD, PhD; Cleveland Clinic Foundation, Cleveland, US

P083 SPLEEN-PRESERVING LAPAROSCOPIC DISTAL PANCREATECTOMY: A META-ANALYSIS OF CURRENT SURGICAL TECHNIQUES Yongfei Hua, MD, Ammar A Javed, MD, Niek A Peters, BS, Fabio Bagante, MD, Matthew J Weiss, MD, Christopher L Wolfgang, MD, PhD, Jin He, MD, PhD; The Johns Hopkins Hospital, Baltimore, US

P084 TOTAL LAPAROSCOPIC VERSUS OPEN PANCREATODUODENECTOMY: A PROPENSITY SCORE MATCHED ANALYSIS Christopher R Shubert, MD, May C Tee, MD, Amy E Glasgow, MHA, Bijan J Borah, PhD, Rory L Smoot, MD, Mark J Truty, MD, David M Nagorney, MD, Michael B Farnell, MD, Michael L Kendrick, MD; Mayo Clinic, Rochester, US


P086 ANTERIOR APPROACH TO THE SUPERIOR MESENTERIC ARTERY BY USING NERVE PLEXUS HANGING MANEUVER FOR BORDERLINE RESECTABLE / LOCALLY UNRESECTABLE PANCREATIC HEAD CARCINOMA: USEFULLNESS OF PANCREATICO-DUODENECTOMY WITH SPLENIC ARTERY RESECTION (PDSAR) Shugo Mizuno, Masashi Kishiwada, Akihiro Tanemura, Yasuhiro Murata, Hiroyuki Kato, Naohisa Kuriyama, Yoshinori Azumi, Masanobu Usui, Hiroyuki Sakurai, Shuji Isaji; Mie University, Tsu, JP
P087 ANATOMICAL STUDY OF THE UNCUS OF THE PANCREAS FOR THE SURGERY IN PANCREATODUODENECTOMY  Daisuke Ban, PhD, MD, Keiichi Akita, PhD, MD, Kumiko Yamaguchi, PhD, MD, Hiroki Ueda, MD, Minoru Tanabe, Department of Hepatobiliary and Pancreatic Surgery, Tokyo Medical and Dental University, Clinical Anatomy, Tokyo Medical and Dental University, Tokyo, JP

P088 THE ACTION OF THE TRISULFATED DISACCHARIDE ON INTRACELLULAR CALCIUM OF PANCREATIC CELLS SUBMITTED TO CALCIUM OVERLOAD  Enio RV Vasques, MD, PhD, Jose Eduardo M Cunha, MD, PhD, Ana M Coelho, PhD, Sandra NSampietre, Emilio E Abdo, MD, PhD, Helena B Nader, PhD, Ivarne L Tersariol, PhD, Marcelo A Lima, PhD, Luiz Augusto C D’Albuquerque, MD, PhD, Eleazar Chaib, MD, PhD, Tiago Rodrigues, PhD, University of Sao Paulo Medical School, Federal University of Sao Paulo, Mogi Das Cruzes, BR

P089 ADAM 10 & 17 INHIBITION: A THERAPEUTIC STRATEGY TO MODULATE LOCAL AND SYSTEMIC INFLAMMATION IN ACUTE PANCREATITIS  John George, MBBS, Archana Sareen, PhD, Ajay Dixit, PhD, Zuobiao Yuan, MD, PhD, Shrey Modi, MBBS, MS, Sushil Garg, MD, Vikas Dudeja, MD, Rajinder Dawra, PhD, Ashok Saluja, PhD, University of Minnesota, Minneapolis, US

P090 GRADED MORBIDITY PROFILES FOR DUODENUM PRESERVING PANCREATIC HEAD RESECTION ARE EQUIVALENT TO THOSE FOR PANCREATODUODENECTOMY IN HEAD-DOMINANT CHRONIC PANCREATITIS  Olga Kantor, MD, Jeffrey B Matthews, MD, Mark S Talamonti, MD, Waseem Lutfi, BS, Marshall Baker, MD, Department of Surgery, University of Chicago, Department of Surgery, NorthShore University HealthSystem, Chicago, US

P091 PREOPERATIVE SPLENIC EMBOLIZATION FOR DISCONNECTED PANCREATIC DUCT SYNDROME IN THE MANAGEMENT OF ACUTE NECROTIZING PANCREATITIS  Jessica L Cioffi, MD, Robert J Feezor, MD, Jose G Trevino, MD, Kevin E Behrns, MD, Steven J Hughes, MD, University of Florida, Gainesville, US

P092 PATIENTS’ PERSPECTIVES ON FUTURE CLINICAL AND RESEARCH TOPICS IN PANCREATIC DISEASES  Janneke van Grinsven, Yama Issa, Marco J Bruno, Olivier R Busch, Arja Gerritsen, Harry van Goor, Jantien A Vogel, Hugo DG van Willigen, Marja A Boermeester, Marc GH Besselink, Academic Medical Center Amsterdam, Erasmus Medical Center, Rotterdam, Radboud University Medical Center, Nijmegen, Amsterdam, NL
SATURDAY, MAY 21, 2016

★ P093 CXCR2 INHIBITION PROFOUNDLY SUPPRESSES METASTASES AND IMPROVES IMMUNOTHERAPY IN PANCREATIC CANCER Colin W Steele, MRCS, PhD1, Saadia A Karim, PhD2, Joshua D Leach, MRCVS2, Colin J McKay, MD1, Euan J Dickson, MD1, Nigel B Jamieson, MRCS, PhD1, Owen J Sansom, PhD2, Jennifer P Morton, PhD2, Ross Carter, MD1; 1Glasgow Royal Infirmary, 2Beatson Institute for Cancer Research, Strathaven, GB

★ P094 MUC-13 - AN EARLY DIAGNOSTIC MARKER FOR PANCREATIC DUCTAL ADENOCARCINOMA Sheema Khan, PhD, Stephen W Behrman, MD, Nadeem Zafar, MD, Meena Jaggi, PhD, Subhash C Chauhan, PhD; University of Tennessee Health Science Center, Memphis, US

★ P095 THE IMPACT OF MULTI-AGENT CHEMOTHERAPY AND STEREOTACTIC BODY RADIATION THERAPY ON CLINICAL OUTCOMES IN LOCALLY ADVANCED PANCREATIC CANCER Lauren M Rosati, BS1, Amy Hacker-Prietz, MS, PAC1, Avani S Rao1, John L Cameron, MD2, Jin He, MD, PhD2, Timothy M Pawlik, MD, MPH, PhD2, Martin A Makary, MD, MPH2, Kenzo Hirose, MD2, Ana De Jesus-Acosta, MD2, Dung T Le3, Lei Zheng, MD3, Daniel A Laheru, MD3, Susannah G Ellsworth, MD1, Christopher L Wolfgang, MD, PhD2, Matthew J Weiss, MD2, Joseph M Herman, MD, MSc1; 1Department of Radiation Oncology, Johns Hopkins University School of Medicine, Baltimore, Maryland, 2Department of Surgery, Johns Hopkins University School of Medicine, Baltimore, Maryland, 3Department of Medical Oncology, Johns Hopkins University School of Medicine, Baltimore, Maryland, Baltimore, US

★ P096 COMPARISON BETWEEN PRE-OPERATIVE MRI AND THE CORRESPONDING PATHOLOGICAL FINDINGS IN 73 CONSECUTIVE PANCREATIC RESECTIONS FOR IPMN Filippo Scopelliti, Paolo Regi, Stefano Gobbo, Paolo Tinazzi Martini, Roberto Girelli, Isabella Frigerio, Alessandro Giardino, Paolo Pederzoli, Giovanni Butturini; Pederzoli Hospital, Peschiera Del Garda, IT

★ P097 LEFT-SIDED PORTAL HYPERTENSION AFTER PANCREATICODUODENECTOMY WITH RESECTION OF SUPERIOR MESENTERIC-PORTAL VEIN CONFLUENCE: IMPACT OF COMBINED RESECTION OF SPLENIC ARTERY Kazuyuki Gyoten, Shugo Mizuno, Akihiro Tanemura, Hiroyuki Kato, Yasuhiro Murata, Yoshinori Azumi, Naohisa Kuriyama, Masashi Kishiwada, Masanobu Usui, Hiroyuki Sakurai, Shuji Isaji; Department of Hepatobiliary Pancreatic and Transplant Surgery, Mie University School of Medicine, Tsu, JP

★ P098 PROPHYLACTIC PANCREATECTOMIES CARRY PROHIBITIVE MORTALITY AT LOW VOLUME CENTERS A.F Callahan, MD, P Ituarte, PhD, L Goldstein, PhD, S G Warner, MD, Y Woo, MD, G Singh, MD, Y Fong, MD, L Melstrom, MD; City of Hope Medical Center, Duarte, US

★ P100 ROBOTIC APPROACH MITIGATES RISK OF WOUND INFECTION AND PERIOPERATIVE MORBIDITY IN OBESE PATIENTS FOLLOWING PANCREATICODUODENECTOMY Mark Girgis, MD, Jennifer Steve, BS, Mazen Zenati, MD, Amer Zureikat, MD, Herbert Zeh, Melissa Hogg, MD; University of Pittsburgh Medical Center, Pittsburgh, US
Poster Listings

P101 SUPERIOR MESENTERIC ARTERY RESECTION DURING PANCREATECTOMY: POST-OPERATIVE RESULTS AND SURVIVAL  Emanuele F Kauffmann, MD¹, Niccolo Napoli, MD¹, Francesca Menonna, MD¹, Nelide De Lio, MD¹, Vittorio Perrone, MD¹, Fabio Vistoli, MD¹, Carla Cappelli, MD², Enrico Vasile, MD³, Niccola Funel, PhD⁴, Luca Pollina, MD⁴, Daniela Campani, MD⁴, Ugo Boggi, MD⁴; ¹Division of General and Transplant Surgery, University of Pisa, ²Division of Diagnostic and Interventional Radiology, University of Pisa, ³Division of Medical Oncology, University of Pisa, ⁴Division of Pathology, University of Pisa, Pisa, IT

P102 BIOBANK OF PANCREATIC DUCTAL ADENOCARCINOMA OF ORTHOTOPIC PATIENT-DERIVED TUMOR XENOGRAFTS  Michela Monteleone, MD, Eugenio Morandi, MD, David Alessio Merlini, Marco Castoldi; O.U. of General Surgery, Azienda Ospedaliera G. Salvini, Rho, Milan, Italy, Lecco, IT

P103 RACIAL INEQUALITY IN PANCREATIC CANCER HEALTHCARE: A COST ANALYSIS OF NEW YORK STATE PATIENTS  Sonia Bharel¹, Juan Carlos Bucobo¹, Jonathan M Buscaglia¹, Ellen Li¹, Purvi Parikh¹, Aaron Sasson¹, Mark Talamini¹, Rebecca Nelson², Joseph Kim³; ¹Stony Brook University Hospital, City of Hope, East Setauket, US

P104 PANCREATIC CANCER CELL AND STROMA GROWTH INHIBITION WITH A GLIANTAGONIST IN VITRO AND IN VIVO  Kim C Honselmann, MD¹, Moritz Pross, MD¹, Ulrich F Wellner, MD¹, Laura Frohneberg, MD¹, Steffen Deichmann, MD¹, Hryhoriy Lapshyn, MD¹, Jens K Habermann, MD, PhD², Tobias Keck, MD, MBA¹, Dirk Bausch, MD¹; ¹University Medical Center Luebeck, ²Section of Translational Surgical Oncology & Biobanking, University of Luebeck & University Medical Center Luebeck, Luebeck, DE

P105 MECHANO-CHEMICAL COUPLING IN PANCREATIC DUCTAL ADENOCARCINOMA  Stefano Coppola, PhD¹, Annarosa Arcangeli, MD, PhD², Thomas Schmidt, PhD³; ¹Physics of Life Processes, Huygens-Kamerlingh Onnes Laboratory, Leiden University, Leiden, The Netherlands, ²Department of Experimental and Clinical Medicine, University of Florence, Florence, Italy, Leiden, NL

P106 THE INFLUENCE OF A METAL STENT ON THE DISTRIBUTION OF THERMAL ENERGY DURING IRREVERSIBLE ELECTROPORATION  Jantien A Vogel¹, Hester J Scheffer¹, Willemien van den Bos¹, Robert E Neal II², Krijn P van Lienden³, Marc G Besselink¹, Martin J van Gemert³, Cees W van der Geld³, Martijn R Meijerink³, John H Klaessens⁴, Rudolf M Verdaasdonk⁵; ¹Department of Surgery, Academic Medical Center, Amsterdam, the Netherlands, ²Department of Radiology and Nuclear Medicine, VU University Medical Center, Amsterdam, the Netherlands, ³Department of Urology, Academic Medical Center, Amsterdam, the Netherlands, ⁴Department of Radiology, The Alfred Hospital, Melbourne, Australia, ⁵Department of Biomedical Engineering and Physics, Academic Medical Center, Amsterdam, the Netherlands, ⁶Department of Mechanical Engineering, Eindhoven University of Technology, Eindhoven, the Netherlands, ⁷Department of Physics and Medical Technology, VU University Medical Center, Amsterdam, the Netherlands, Amsterdam, NL

P107 CELL LINES REFLECT AMPULLARY CANCER SUBTYPE  Ulrich F Wellner, MD¹, Zon W Lai, PhD², Louisa Bolm¹, Oliver Schilling, PhD², Dirk Bausch, MD¹, Tobias Keck, MD¹, Peter Bronsert, MD³; ¹Sugery, UKSH Campus Lubeck, ²Molecular Medicine, University of Freiburg, ³Pathology, University of Freiburg, Lubeck, DE
P108 GENE EXPRESSION PROFILING IN LONG AND SHORT-TERM SURVIVORS AFTER RESECTION FOR PANCREATIC CANCER
Bryan Thibodeau, PhD1; Brandon Stone, MD1; Andrew Baschnagel, MD2; Laura Fortier, MS1; Amro Almrad, MD1; George Wilson, PhD1; Robert Jury, MD1; 1Beaumont Health System Royal Oak, MI, US, 2Carbone Cancer Center of Wisconsin University, Madison, WI, US, Royal Oak, US

P109 GALECTIN-1 EXPRESSED IN PANCREATIC STELLATE CELLS PROMOTES TUMOR PROGRESSION IN PANCREATIC CANCER VIA UPREGULATION OF SDF-1 AND ACTIVATION OF NF-κB
Dong Qian, Zipeng Lu, MD, MRCSEd, Qingcheng Xu, Pengfei Wu, Lei Tian, MD, MR, Liangtao Zhao, Baobao Cai, MD, Jie Yin, Dong Tang, MD, PhD, Kuirong Jiang, MD, PhD, Yi Miao, MD, PhD, FACS, FICSHon; Pancreas Center, The First Affiliated Hospital of Nanjing Medical University, Nanjing, CN

P110 ARID1A LOSS DRIVES MURINE PANCREATIC MUCINOUS CYST FORMATION
Ibrahim Nassour, MD1; Sam C.Wang, MD1; Xuxu Sun, PhD2; Jen-Chiang Chuang, PhD2; Liem H.Nguyen2; Shuyuan Zhang2; Lan Peng, MD3; Hao Zhu, MD2; 1University of Texas Southwestern, Department of surgery, 2University of Texas Southwestern, Children’s Medical Center Research Institute, 3University of Texas Southwestern, Department of Pathology, Dallas, US

P112 THE MICRORNA PROCESSING ENDORIBONUCLEASE DICER HAS ALTERED EXPRESSION IN PANCREATIC DUCTAL ADENOCARCINOMA AND PROGNOSTIC IMPLICATIONS
Niccola Funel, PhD1; Adam E Frampton2; Elisa Giovannetti3; Raida Ahmed, MD4; Jonathan Krell, MD5; Thomas Knoesel, MD6; Long R Jiao7; Ugo Boggi, MD8; Justin Stebbing2 Stebbing, MD1; 1Cancer Pharmacology Lab, AIRC-Start-Up, University Hospital of Pisa, Pisa and Dept. of Translational Research and The New Technologies in Medicine and Surgery, 2HPB Surgical Unit and Division of Cancer, Dept. of Surgery & Cancer, Imperial College, London, UK, 3Cancer Pharmacology Lab, AIRC-Start-Up, University Hospital of Pisa, Pisa Dept. of Medical Oncology, VU University Medical Center, The Netherlands, 4Dept. of Histopathology, Imperial College Healthcare NHS Trust, Hammersmith Hospital, London, UK, 5Division of Cancer, Dept. of Surgery & Cancer, Imperial College, London, UK, 6Institute of Pathology, Ludwig-Maximilians-University, Munich, Germany, 7HPB Surgical Unit, Dept. of Surgery & Cancer, Imperial College, London, UK, 8Dept. of Translational Research and The New Technologies in Medicine and Surgery, Pisa, IT

P113 DISRUPTION OF CANCER CELL-STELLATE CELL CROSS TALK WITH TRIPOLIDE SUPPRESSES PANCREATIC CANCER GROWTH
Shrey Modi, Xianda Zhao, Bhuwan Giri, John George, Vikas Dudeja, Ashok Saluja, Sulagna Banerjee; University of Minnesota, Minneapolis, US

P114 HSP 70 IN STROMA MODULATES GROWTH AND METASTASIS IN A NOVEL MURINE MODEL OF PANCREATIC CANCER
Bhuwan Giri, Shrey Modi, John George, Kaustav Majumder, Vikas Dudeja, Ashok Saluja; University of Minnesota, Minneapolis, US

P115 INHIBITION OF HISTONE ACETYLTRANSFERASE IN PANCREATIC CANCER CELL LINES RESULTS IN NF-KB DOWNREGULATION AND APOPTOTIC CELL DEATH.
Bhuwan Giri, John George, Shrey Modi, Vikas Dudeja, Ashok Saluja; University of Minnesota, Minneapolis, US
P116 A NOVEL NOMOGRAM PREDICTS SURVIVAL IN PATIENTS WITH NONFUNCTIONAL PANCREATIC NEUROENDOCRINE TUMORS Megan V Beems, MD1, Yue Ma, PhD, MA1, Robert C Martin II, MD, PhD2, David A Kooby, MD3, Shishir K Maithel, MD4, Charles R Scoggins, MD, MBA5, Alexander A Parikh, MD, MPH6, Nipun B Merchant, MD7, Hong J Kim, MD8, Emily R Winslow, MD9, Sharon M Weber, MD10, Clifford S Cho, MD11; 1University of Wisconsin, 2University of Louisville, 3Emory University, 4Vanderbilt University, 5University of North Carolina, Madison, Wi, US

P117 FEASIBLE FACTORS TO IMPROVE POSTOPERATIVE SURVIVAL AFTER PANCREATECTOMY FOR PANCREATIC CANCER IN DIAGNOSTIC AND SURGICAL PROCESSES: A RETROSPECTIVE REVIEW Yusuke Watanabe, MD1, Kazuyoshi Nishihara1, Yusuke Niina2, Yuji Abe1, Toru Nakano1, Shoshu Mitsuyama1; 1Department of Surgery, Kitakyushu Municipal Medical Center, 2Department of Gastroenterology, Kitakyushu Municipal Medical Center, Kitakyushu, JP

P118 EXTENT OF LYMPH NODE BURDEN PROVIDES ENHANCED PROGNOSTIC VALUE FOR PANCREATIC ADENOCARCINOMA. Cinthya S Yabar, MD, Jamie Metkus, Jeffrey Epstein, Geoffrey Kozak, MD, Harish Lavu, MD, Charles Yeo, MD, Jordan Winter, MD; Department of Surgery, Jefferson Pancreas, Biliary and Related Cancer Center, Thomas Jefferson University, Philadelphia, US

P119 THE SURGICAL IMPACT OF ARTERY FIRST APPROACH FOR BORDERLINE RESECTABLE PANCREATIC CANCER Hiroki Yamaue, Manabu Kawai, Seiko Hirono, Ken-ichi Okada, Motoki Miyazawa, Atsushi Shimizu, Yuji Kitahata; Second department of Surgery, Wakayama Medical University, Wakayama, JP

P120 C-REACTIVE PROTEIN AND PROCALCITONIN AS PREDICTORS OF POSTOPERATIVE INFLAMMATORY COMPLICATIONS AFTER PANCREATIC SURGERY Alessandro Giardino1, Gaya Spolverato1, Paolo Regi1, Isabella Frigerio1, Filippo Scopelliti1, Roberto Girelli1, Timothy M Pawlik2, Paolo Pederzoli1, Giovanni Butturini1; 1Surgery, Casa di Cura Pederzoli, 2Johns Hopkins - Surgery, Verona, IT

P121 SMALL CELL CARCINOMA OF THE PANCREAS: A SURGICAL DISEASE Tommy Ivanics, MD, John R Bergquist, MD, Christopher R Shubert, MD, Rory L Smoot, MD, Elizabeth B Habermann, PhD, Mark J Truty, MD; Mayo Clinic, Rochester, US

P122 MUC13 INTERACTION WITH RECEPTOR TYROSINE KINASE HER2 DRIVES PANCREATIC DUCTAL ADENOCARCINOMA PROGRESSION Stephen W Behrman, MD, Sheema Khan, PhD, Nadeem Zafar, MD, Meena Zafar, PhD, Subhash C Chauhan, PhD; University of Tennessee Health Science Center, Memphis, US

P123 RETROSPECTIVE REVIEW OF EFFICACY AND DOSE INTENSITY OF SECOND-LINE(SL) NAB-PACLITAXEL PLUS GEMCITABINE (NABG) FOR PATIENTS WITH METASTATIC PANCREATIC ADENOCARCINOMA (MPAC) AFTER FIRST-LINE (FL) FOLFRINOX (FFX). Shinoj Pattali, MD, Jayakumar Sreenivasan, MD, Naveen Premnath, MD, Shannon Schmidt, MD, Paul S Ritch, MD, James P Thomas, MD, Beth Erickson, MD, Kathleen K Christians, MD, SUsan Tsai, MD, Douglas B Evans, MD, Ben George, MD; Medical College of Wisconsin, Greenfield, US

P124 PANCREATICODUODENECTOMY WITH HEPATIC ARTERY RESECTION FOR BORDERLINE RESECTABLE PANCREATIC HEAD CARCINOMA Naru Kondo, MD, Yoshiaki Murakami, MD, Kenichiro Uemura, MD, Naoya Nakagawa, MD, Kazuhide Urabe, MD, Tajiro Sueda, MD; Department of Surgery, Institute of Biomedical and Health Sciences, Hiroshima University, Hiroshima, US
P125 MULTIMODALITY THERAPY IS ASSOCIATED WITH IMPROVED CAUSE-SPECIFIC SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA WITH VASCULAR ABUTMENT Olga Kantor, MD1, Mark S Talamonti, MD2, Waseem Lutfi, BS2, Kristine Kuchta, MS1, David J Winchester, MD2, Richard A Prinz, MD2, Marshall S Baker, MD, MBA2; 1Department of Surgery, The University of Chicago Medicine, 2Department of Surgery, NorthShore University HealthSystem, 3Center for Biomedical Research Informatics, NorthShore University HealthSystem, Chicago, US

P126 MULTIMODALITY APPROACH TO LOCALLY ADVANCED PANCREATIC CANCER WITH FOLFIRINOX, SURGICAL EXPLORATION AND IRREVERSIBLE ELECTROPORATION: PROSPECTIVE SERIES OF 132 CONSECUTIVE PATIENTS Jantien Vogel, MD1, Thijs de Rooij1, Krijn van Lienden2, Johanna Wilmink3, Hanneke van Laarhoven3, Jeanin van Hooft4, Otto van Delden2, Marcel Dijkstra3, Robert Martin4, Olivier Busch1, Marc Besselink1; 1Department of Surgery, Academic Medical Center, Amsterdam, the Netherlands, 2Department of Radiology, Academic Medical Center, Amsterdam, the Netherlands, 3Department of Medical Oncology, Academic Medical Center, Amsterdam, the Netherlands, 4Department of Gastroenterology, Academic Medical Center, Amsterdam, the Netherlands, 5Clinical Research Unit, Academic Medical Center, Amsterdam, the Netherlands, 6Department of Surgery, University of Louisville, Louisville, Kentucky, USA, Amsterdam, NL

P127 NATIONAL COMPLIANCE TO AN EVIDENCE-BASED MULTIDISCIPLINARY GUIDELINE ON PANCREATIC AND PERIAMPUTLAR CANCER Lennart B van Rijssen, MD1, Lydia G van der Geest, MSc2, Thomas L Bollen, MD3, Marco J Bruno, MD, PhD4, Ate van der Gaast, MD, PhD4, Laetitia Veerbeek, PhD5, Fibo J Ten Kate, MD, PhD5, Olivier R Busch, MD, PhD1; 1Academic Medical Center, Amsterdam, 2Netherlands Comprehensive Cancer Organisation, 3St. Antonius Hospital, Nieuwegein, 4Erasmus Medical Center, Rotterdam, 5University Medical Center Utrecht, Amsterdam, NL

P128 ONGOING CENTRALIZATION OF PANCREATIC SURGERY FURTHER IMPROVES OUTCOMES Lydia G van der Geest, MSc1, Lennart B van Rijssen, MD2, Isaac Q Molenaar, MD, PhD3, Ignace H de Hingh, MD, PhD4, Bas Groot Koerkamp, MD, PhD5, Olivier R Busch, MD, PhD2, Valery E Lemmens, PhD1, Marc G Besselink, MD, MSc, PhD2; 1Netherlands Comprehensive Cancer Organisation, 2Academic Medical Center, Amsterdam, 3University Medical Center Utrecht, 4Catharina Hospital, Eindhoven, 5Erasmus Medical Center, Rotterdam, Amsterdam, NL

P129 PHASE II STUDY OF INTRAVENOUS AND INTRAPERITONEAL PACLITAXEL WITH S-1 FOR PANCREATIC DUCTAL ADENOCARCINOMA PATIENTS WITH PERITONEAL METASTASIS Masamichi Mizuma1, Sohei Sato1, Tsutomu Fujii1, Hiroaki Yanagimoto2, Masanori Kurata3, Naminatsu Takahara4, Suguru Yamada2, Fuyuhiko Moto1, Tomohisa Yamamoto2, Goro Honda4, Hiroyuki Isayama2, Hironori Ishigami6, Michiaki Unno1, Masanori Kon1; 1Department of Surgery, Tohoku University Graduate School of Medicine, 2Department of Surgery, Kansai Medical University, 3Department of Gastroenterological Surgery (Surgery II), Nagoya University Graduate School of Medicine, 4Department of Surgery, Tokyo Metropolitan Cancer and Infectious diseases Center Komagome Hospital, 5Department of Gastroenterology, Graduate School of Medicine, the University of Tokyo, 6Department of Chemotherapy, the University of Tokyo, Hirakata, JP
P130 IMPLICATIONS OF INTRA-OPERATIVELY DETECTED LYMPH NODE METASTASIS IN PATIENTS WITH PANCREATIC AND PERI-AMPULLARY CARCINOMA: A SYSTEMATIC REVIEW AND META-ANALYSIS
Lennart B van Rijssen, MD,1, Poorvi Narwade1, Nadine C van Huijgevoort!, Dorine S Tseng, MD2, Hjalmar C van Santvoort, MD, PhD3, Isaac Q Molenaar, MD, PhD2, Hanneke W van Laarhoven, MD, PhD1, Casper H Van Eijck, MD, PhD4, Olivier R Busch, MD, PhD5, Marc G Besselink, MD, MSc, PhD1; 1Academic Medical Center, Amsterdam, 2University Medical Center Utrecht, 3St. Antonius Hospital, Nieuwegein/Utrecht, 4Erasmus Medical Center, Amsterdam, NL.

P131 TUMOR BIOLOGY TRUMPS CHEMOTHERAPY REGIMEN FOR PATIENTS WITH LOCALIZED AND UNRESECTABLE PANCREATIC CANCER.
Geoffrey M Kozak, MD, S Deshmukh, MD, B Scott, H Lavu, MD, C J Yeo, MD, J M Winter, MD; Thomas Jefferson University, Philadelphia, US.

P132 PANCREATIC ADENOCARCINOMAS ARISING ON THE BACKGROUND OF PANCREATIC CYSTIC DISEASE HAVE A BETTER OUTCOME THAN DUCTAL ADENOCARCINOMAS
A Mohan, M Moslim1, C O’Rourke, MS2, R M Walsh, MD4, G Morris-Stiff, MD4; 1Case Western Reserve University School of Medicine, 2Department of General Surgery, Cleveland Clinic Foundation, 3Department of Quantitative Health Sciences, Cleveland Clinic Foundation, 4Department of HPB Surgery, Digestive Disease Institute, Cleveland Clinic Foundation, Cleveland, US.

P133 PREOPERATIVE CARBOHYDRATE ANTIGEN 19-9 IS AN INDEPENDENT PROGNOSTIC PARAMETER AND CORRELATES WITH LYMPH NODE METASTASIS - A MULTICENTER ANALYSIS OF DISTAL CHOLANGIOCARCINOMA (CCC)
Louisa Bolm,1 Sebastian Zach, MD2, Felix Ruckert, MD2, Bettina Rau, MD3, Robert Grutzmann, MD4, Gabriel Seifert, MD5, Ulrich T Hopf, MD5, Frank Makowiec, MD3, Tobias Keck, MD1, Dirk Bausch, MD1, Ulrich F Wellner, MD1; 1UKSH Campus Lubeck, 2Universitatsmedizin Mannheim, Dept of Surgery, 3University of Rostock, Dept of Surgery, 4University of Erlangen, Dept of Surgery, 5University of Freiburg, Clinic for General and Visceral Surgery, Lubeck, DE.

P134 INTRAOPERATIVE RADIOTHERAPY (IORT) IN THE ERA OF INTENSIVE NEOADJUVANT CHEMOTHERAPY AND CHEMORADIOOTHERAPY FOR LOCALLY ADVANCED AND BORDERLINE RESECTABLE ADENOCARCINOMA OF THE PANCREAS (PDAC)
Cristina R Ferrone, MD, Jennifer Y Wo, MD, Florence K Keane, MD, Theodore S Hong, MD, Jeffrey W Clark, MD, Lawrence S Blaszkowsky, MD, Jill N Allen, MD, Eunice L Kwak, MD, PhD, David P Ryan, MD, Keith D Lillemoe, MD, Carlos Fernandez-del Castillo, MD, MGH, Boston, US.

P135 ONCOLOGICAL OUTCOME OF LAPAROSCOPICALLY ASSISTED PANCREATIC HEAD RESECTION FOR DUCTAL ADENOCARCINOMA
Uwe A Wittel, MD, Simon Kuesters, MD, Olivia Sick, BS, Tobias Keck, MD, Frank Makowiec, MD, Ulrich T Hopf, MD; Universitatsklinikum Freiburg, Freiburg, DE.

P136 CYSTIC LESIONS OF THE PANCREAS: WHY WE GET IT WRONG
Lindsey L Manos, PAC, Robert Moran, MD, Neda Rezaee, MD, Ashley M Salamone, CRNP, Ralph H Hruban, MD, Elliot K Fishman, MD, Nita Ahuja, MD, John L Cameron, MD, Jin He, MD, PhD, Matthew J Weiss, MD, Anne Marie Lennon, MD, PhD, Christopher L Wolfgang, MD, PhD; The Johns Hopkins Hospital, Baltimore, US.
P137 NUTRITION STATUS CORRELATES WITH PATIENT-REPORTED QUALITY OF LIFE PRIOR TO TREATMENT FOR PANCREATIC ADENOCARCINOMA. Maria Q Petzel, BS, RD\(^1\), Sarah Thornton, MPH, RD\(^2\), Vanessa Martinez, BS\(^1\), Hsiang-Chun Chen, PhD\(^1\), Xuemei Wang, MS\(^1\), Jason B Fleming, MD\(^1\), Jeffrey E Lee, MD\(^1\), Justin Folloder, MS, PA, MBA\(^1\), Carol Clegg, BS, MEd, MS, RN, ANP\(^1\), Rae Reynolds, MS, RN, ANP\(^1\), Matthew H Katz, MD\(^1\); 1The University of Texas MD Anderson Cancer Center, 2Sacred Heart Hospital on the Emerald Coast, Houston, US

P138 PRIMARY TUMOR RESECTION FOLLOWING NEOADJUVANT CHEMOTHERAPY IN STAGE IV PANCREATIC ADENOCARCINOMA: A BI-INSTITUTIONAL ANALYSIS. G Paul Wright, MD\(^2\), Katherine E Poruk, MD\(^1\), Mazen S Zenati, MD, PhD\(^2\), Melissa E Hogg, MD\(^2\), Amer H Zureikat, MD\(^2\), Herbert J Zeh, MD\(^2\), Christopher L Wolfgang, MD\(^1\), Matthew J Weiss\(^1\); 1University of Pittsburgh Medical Center, 2The Johns Hopkins University School of Medicine, Pittsburgh, US

P139 IMPACT OF NUMBER OF REGIONAL LYMPH NODE METASTASIS IN PATIENTS WITH LOCALIZED PANCREATIC CANCER FOLLOWING NEOADJUVANT THERAPY. Chad Barnes, MD, Mohammed Aldakkak, MD, Kathleen Christians, MD, Paul Ritch, MD, Ben George, MD, Fabian Johnston, MD, Beth Erickson, MD, Catherine Hagen, MD, Kiyoko Oshima, MD, Douglas Evans, MD, Susan Tsai, MD; Medical College of Wisconsin, Milwaukee, US

P141 EXOCRINE PANCREATIC INSUFFICIENCY (EPI) AND PANCREATIC ENZYME REPLACEMENT THERAPY (PERT) IN PATIENTS UNDERGOING PANCREATIC RESECTION: SURVEY OF PANCREAS CLUB MEMBERS. Gareth Morris-Stiff, MD, PhD; Cleveland Clinic, Cleveland, US

P142 CHANGES IN ENDOCRINE FUNCTION AFTER NEOADJUVANT THERAPY AND RESECTION FOR BORDERLINE RESECTABLE PANCREATIC CANCER. June S Peng, MD, Trang K Nguyen, MD, R. Matthew Walsh, MD, Mohamed E Abazeed, MD, PhD, Gareth Morris-Stiff, MD, PhD; Cleveland Clinic Foundation, Cleveland, US

P143 SARCOPENIA AS A PREDICTOR FOR COMPLETION OF EXTENDED NEOADJUVANT CHEMOTHERAPY IN PATIENTS WITH BORDERLINE RESECTABLE PANCREATIC ADENOCARCINOMA (BRPA). Kevin A Penn, MD, J B Rose, MD, W S Helton, MD, FACS; Virginia Mason Medical Center, Seattle, US

P144 DOES ABO BLOOD GROUP PREDISPOSE TO PANCREATIC CANCER? Maitham A Moslim, MD, Colin O’Rourke, John McMichael, Gareth Morris-Stiff, MBBCh, MD, MCh, PhD, FRCS; Cleveland Clinic Foundation, University Heights, US

P145 CHEMORADIOTHERAPY FOLLOWED BY SURGERY FOR LOCALLY ADVANCED UNRESECTABLE PANCREATIC CANCER. Masashi Kishiwada, MD, PhD, Yusuke Izawa, MD, Hiroyuki Kato, MD, PhD, Yasuhiro Murata, MD, PhD, Akihiro Tanemura, MD, PhD, Naohisa Kuriyama, MD, PhD, Yoshinori Azumi, MD, PhD, Shugo Mizuno, MD, PhD, Masanobu Usui, MD, PhD, Hiroyuki Sakurai, MD, PhD, Shuji Isaji, MD, PhD; Hepatobiliary Pancreatic and Transplant Surgery, Mie University School of Medicine, Tsu, Japan, Tsu, JP

P146 INTEGRATED MODELING STRATEGIES FOR IMPROVING LOCALIZED PANCREATIC SURGERY. Amanda J Green, MD\(^1\), Kara Rothenberg, MD\(^1\), Ck Chang, MD\(^2\), Austin Spitzer, MD\(^2\), George Kazantsev, MD\(^2\), Peter Peng, MD\(^2\), Rene Ramirez, MD\(^1\), Yan Li, MD\(^2\); 1University of California San Francisco East Bay Surgery, 2Kaiser Oakland Medical Center, 3Kaiser San Leandro Medical Center, Oakland, US
P147 UNPLANNED REOPERATION AFTER PANCREATIC RESECTION: AN ANALYSIS OF RISK FACTORS
Heather Lyu, Resident, in, Surgery1, Gaurav Sharma, Postdoctoral, Research, Fellow, Resident1, Ethan Brovman, Anesthesia, Resident2, Richard Urman, Assistant, Professor2, Jason S Gold, Associate, Professor4, Edward E Whang, Director, Center, for, Perioperative, Research2; 1Dana-Farber/Brigham and Women’s Cancer Center, 2Brigham and Women’s Hospital/Harvard Medical School, Boston, US

P148 DOES GIVING PASIREOTIDE TO PATIENTS UNDERGOING PANCREATICODUODENECTOMY PAY FOR ITSELF?
Fang Yuan, MSc, Amiram Gafni, PhD, Chu-Shu Gu, MSc, Deepak Dath, MD, Michael Marcaccio, MD, Leyo Ruo, MD, Ved Tandan, MD, Pablo E Serrano, MD; McMaster University, Hamilton, CA

P149 PANCREATIC LEAK RATES FROM PATIENTS WITH CYTOREDUCTIVE SURGERY PLUS HYPERPERMIC INTRAPERITONEAL CHEMOTHERAPY VS PATIENTS WITH DISTAL PANCREATECTOMY.
Michael Reynolds, MD1, Julie Thompson, RN1, Heather Garaghty, RN2, Harold Huss, MD, FACS2, Hatem Halabi, MD2, Komen Brown, MD2, Juan Sanabria, MD, MSc, FRCSC, FACS, FAASLD2; 1Case Western Reserve University, Pepper Pike, US

P150 PERI-OPERATIVE BUNDLE DECREASES POST-OPERATIVE SURGICAL SITE INFECTIONS IN PATIENTS UNDERGOING PANCREATECTOMY
M V Hill, MD1; K A Newhall, MD1, R Louie, MD1, M Cheung, MD2, C Angeles, MD1, T A Colacchio, MD1, R J Barth, MD1, K D Smith, MD1; Dartmouth Hithcock Medical Center, 1Yale- New Haven Hospital, Lebanon, US

P151 CLINICO-PATHOLOGICAL FEATURES AND SURVIVAL OF RESECTED PANCREATIC NEOENDOCRINE TUMORS ASSOCIATED WITH VON HIPPEL LINDAU DISEASE AND MULTIPLE ENDOCRINE NEOPLASIA TYPE 1.
Ilaria Pergolini, MD1, Othon Iliopoulos2, Cristina Ferrone1, Vikram Deshpande3, Keith Lillemoe1, Carlos Fernandez del Castillo1; 1Department of Surgery, Massachusetts General Hospital, 2Department of Oncology, Massachusetts General Hospital, 3Department of Pathology, Massachusetts General Hospital, Boston, US

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N. S McCall, C. B Chen, M. J Pucci, MD, S. Doane, MD, J. M Winter, MD, C. J Yeo, MD, H. Lavu, MD; Thomas Jefferson University, Department Of Surgery, Philadelphia, PA, USA, Philadelphia, US

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Kazi Ullah1, Rebecca Nelson, PhD2, Aaron Sasson, MD3, Joseph Kim, MD1, Purvi Parikh, MD1; 1Stony Brook School of Medicine, 2City of Hope, 3Stony Brook Medicine, Stony Brook, US

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Dong Qian1, Zipeng Lu, MD, MRCS1, Richard Jackson2, Kuirong Jiang, MD, PhD1, Yi Miao1, MD, PhD, FACS, FICS Hon1; 1Pancreas Center, The First Affiliated Hospital of Nanjing Medical University, 2Liverpool Cancer Trials Unit, University of Liverpool, Nanjing, CN
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A H Zureikat1, H Zeh1, V Thompson2, D Bentrem3, B L Hall4, H A Pitt5; 1University of Pittsburgh, 2American College of Surgeons, 3Northwestern University, 4Washington University, 5Temple University, Philadelphia, US

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Sunil Shenvi1, Pragyan Khawaunju2, Dibyajyoti Banerjee3, Ashish Bhalla4, Mandep Kang5, Surinder Singh Rana6, Deepak Bhasin7, Rajinder Singh7, Rajesh Gupta7; 1Transplant surgery division, Department of Surgery, Medical University of South Carolina, Charleston, SC, US, 2PGIMER, Chandigarh, India, 3Department of Gastroenterology, PGIMER, Chandigarh, India, 4Department of Radiology, PGIMER, Chandigarh, India, 5Department of Experimental Medicine, PGIMER, Chandigarh, India, 6Department of Internal Medicine, PGIMER, Chandigarh, India, 7Department of Gastroenterology, PGIMER, Chandigarh, India, 8Department of Gastroenterology Division, Department of General Surgery, PGIMER, Chandigarh, India, Chandigarh, IN

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Janneke van Grinsven1, Jeroen LA van Vugt2, Arvind Gharbharan2, Thomas L Bollen3, Marc GH Besselink4, Hjalmar C van Santvoort5, Casper HJ van Eijck2, Djamila Boerma2; 1Academic Medical Center, Amsterdam, 2Erasmus Medical Center, Rotterdam, 3St. Antonius Hospital, Nieuwegein, Amsterdam, NL
P191 THE VALUE OF A 24/7 ONLINE NATIONWIDE MULTIDISCIPLINARY EXPERT PANEL FOR NECROTIZING PANCREATITIS: A 5-YEARS' EXPERIENCE Janneke van Grinsven1, Sandra van Brunschot2, Nicolien J Schepers3, Bente H Doeve2, Olaf J Bakker2, Stefan AW Bouwense4, Marja A Boermeester1, Thomas L Bollen5, Marco J Bruno3, Vincent C Cappendijk6, Cornelis HC Dejong7, Casper HJ van Eijck3, Paul Fockens1, Harry van Goor4, Jan Willem Haveman8, Sijbrand H Hofker8, Johan S Lameris1, Maarten S van Leeuwen2, Krijn P van Lienden4, Vincent B Nieuwenhuijs9, Jan-Werner Poley3, Alexander FM Schaapherder10, Robin Timmer5, Hein G Gooszen4, Marc GH Besselink1, Hjalmar C van Santvoort5; 1Academic Medical Center, Amsterdam, 2University Medical Center, Utrecht, 3Erasmus Medical Center, Rotterdam, 4Radboud University Medical Center, Nijmegen, 5St. Antonius Hospital, Nieuwegein, 6Jeroen Bosch Hospital, ’s-Hertogenbosch, 7Maastricht University Medical Center+, Maastricht, 8University Medical Center, Groningen, 9Isala Clinics, Zwolle, 10Leids University Medical Center, Leiden, Amsterdam, NL

P192 RECURRENT OR PERSISTENT PAIN AFTER PRIMARY SURGERY FAILURE IN CHRONIC PANCREATITIS - RESULTS FROM 75 SALVAGE REOPERATIONS Yogesh K Vashist, Kai Bachmann, Asad Kutup, Oliver Mann, Emre Yekebas, Jakob Izbicki; University Medical Center Hamburg-Eppendorf, Hamburg, DE

P193 HIGH LEVEL OF PLASMA SOLUBLE UROKINASE PLASMINOGEN ACTIVATOR RECEPTOR (P-SUPAR) PREDICTS THE LONG-TERM MORTALITY AFTER FIRST ACUTE ALCOHOL-INDUCED PANCREATITIS. Anu Aronen, MD1; Janne Aittoniemi2, Reetta Huttunen3, Anssi Nikkola4, Jussi Nikkola4, Olli Limnell4, Isto Nordback4, Juhani Sand3, Johanna Laukkanen3; 1Tampere University Hospital, Finland, 2Fimlab Laboratories, Tampere, Finland, 3Department of Internal Medicine, Tampere University Hospital, Finland, 4University of Tampere, School of Medicine, Tampere, Finland, Tampere, FI

P194 INTRAHEPATIC PANCREATIC PSEUDOCYST: REVIEW OF THE WORLD LITERATURE Andrew Demeusy; Motahar Hosseini, Steven C Cunningham; Saint Agnes Hospital Center, Ellicott City, US
**S001 INCIDENCE OF HEPATICOJEJUNOSTOMY STRicture FOLLOWING HEPATICOJEJUNOSTOMY**

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**Introduction:** Operations requiring hepaticojejunostomy are uncommon and the true incidence of biliary stricture after hepaticojejunostomy is unknown. Our goal was to use population-based data to determine the timing, incidence, and management of stricture after hepaticojejunostomy for benign and malignant disease.

**Methods:** We used 5% Medicare claims data (1996 to 2011) to identify patients ≥66 years who underwent an operation requiring a hepaticojejunostomy (alone or as part of a larger operation). Hepaticojejunostomy stricture was identified by diagnosis codes for stricture (ICD-9 code 576.2) and/or PTC drain placement occurring >3 months after the initial operation. A cumulative incidence curve was used to describe timing of stricture diagnosis. The use of imaging and intervention were evaluated. In the cumulative incidence curve, patients were censored when they died (no longer at risk for stricture) or were lost to follow-up (no further Medicare claims). A Cox proportional hazards model was constructed to identify factors associated with stricture diagnosis.

**Results:** 3,374 patients underwent an operation requiring a hepaticojejunostomy. The 2- and 5-year survival for the cohort was 57% and 43%. The mean age at the time of surgery was 75.3±6.2 years. 1,729 (51.2%) patients had a malignant diagnosis. Overall, 403 patients developed a stricture after surgery. Taking into account death and loss to follow-up, the cumulative incidence of stricture was 12.5% at 2 years and 17.4% at 5 years. Mean time to stricture formation was 16.8±21.6 months (median=8.5 months). 51.9% (N=209) of patients who developed a stricture had a percutaneous transhepatic catheter (PTC) placed. Of the 403 patients with a stricture diagnosis, 233 (57.8%) were for complications related to stricture. The most common reason for stricture-related admission was cholangitis (N=94). Only 18 of the 403 patients (4.5%) required definitive reoperation. Based on a Cox proportional hazards model, only the presence of a preoperative endostent (HR 1.67; 95% CI 1.35, 2.07) predicted stricture formation; preoperative PTC (HR 1.29; 95% CI 0.70, 2.37) did not.

**Conclusion:** In patients who survive, strictures occur with high frequency after an operation requiring hepaticojejunostomy and should be followed with serial liver function tests. Preoperative stent placement is associated with future stricture formation in patients who undergo hepaticojejunostomy. Even though the majority can be managed non-operatively, stricture diagnosis remains burdensome requiring frequent rehospitalizations, follow-up, and procedures.

**S002 A MULTICENTER, RISK-STRATIFIED, PROSPECTIVE TRIAL OF DRAIN MANAGEMENT FOR PANCREATODUODENECTOMY**

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**Introduction:** Pancreatic fistula is the most common and morbid complication following pancreatic resection. Recent evidence suggests value for both selective drain...
placement and early drain removal for pancreatoduodenectomy (PD). We designed and prospectively evaluated a drain management protocol combining these principles.

Methods: The protocol was applied to 248 consecutive PDs at two institutions over 16 months. Risk for ISGPF clinically relevant pancreatic fistula (CR-POPF) was determined intra-operatively using the Fistula Risk Score (FRS); drains were omitted in negligible/low risk patients and drain fluid amylase (DFA) was measured on POD1 for moderate/high risk patients. Early drain removal (POD3) occurred for patients with POD1 DFA ≤5000 U/L, while patients with POD1 DFA >5000 U/L were managed by clinical discretion. Outcomes were compared with a historical cohort (N=557; 2011-2014).

Results: Fistula risk did not differ between cohorts (Median FRS: 4 vs. 4; p=0.982). No CR-POPFs developed in the 66 (26.6%) negligible/low risk patients. In the overall series, CR-POPF rates were significantly lower following protocol implementation (11.6 vs 20.6%, p=0.002). The protocol cohort also demonstrated lower rates of percutaneous drainage for complication management (3.6 vs. 8.1%, p=0.020) and fewer severe complications (Clavien ≥3; 20.2 vs. 25.9%, p=0.083); these patients also experienced reduced hospital stay (9 vs. 10 days, p=0.006). There were no differences between cohorts in bile or chyle leaks. Comparisons of postoperative outcomes were also stratified by FRS risk zone (Table).

Conclusion: Drains can be safely obviated for one-quarter of PDs. Drain amylase analysis identifies which moderate/high risk patients benefit from early drain removal. This unique, risk-stratified approach has significantly decreased the occurrence of clinically relevant pancreatic fistula.

<table>
<thead>
<tr>
<th>Variable, N (%)</th>
<th>Negligible/Low Risk (FRS 0-2)</th>
<th>Moderate/High Risk (FRS 3-10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>Pre-Protocol</td>
<td>Protocol</td>
</tr>
<tr>
<td></td>
<td>168 (30.2)</td>
<td>66 (26.6)</td>
</tr>
<tr>
<td>CR-POPF</td>
<td>6 (3.6)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Grade B</td>
<td>5 (3.0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Grade C</td>
<td>1 (0.6)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Any complication (Clavien 1-5)</td>
<td>88 (52.4)</td>
<td>35 (53.0)</td>
</tr>
<tr>
<td>Severe complication (Clavien ≥3)</td>
<td>31 (18.5)</td>
<td>7 (10.6)</td>
</tr>
<tr>
<td>Bile leak</td>
<td>8 (4.8)</td>
<td>2 (3.0)</td>
</tr>
<tr>
<td>Chyle leak</td>
<td>4 (2.4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Intensive care unit admission</td>
<td>30 (17.9)</td>
<td>5 (7.6)</td>
</tr>
<tr>
<td>Percutaneous drainage</td>
<td>49 (29.2)</td>
<td>18 (27.3)</td>
</tr>
<tr>
<td>Reoperation (90-day)</td>
<td>10 (6.0)</td>
<td>1 (1.5)</td>
</tr>
<tr>
<td>Duration of stay, days</td>
<td>8 (7-14)</td>
<td>8 (7-12)</td>
</tr>
<tr>
<td>Readmission (30-day)</td>
<td>9 (5.4)</td>
<td>3 (4.6)</td>
</tr>
<tr>
<td>Mortality (90-day)</td>
<td>2 (1.2)</td>
<td>1 (1.5)</td>
</tr>
</tbody>
</table>
**ORAL ABSTRACTS**

**S003 THE EVOLUTION OF VERONA’S EXPERIENCE: LOOKING FOR A NEW DRAIN AMYLASE VALUE CUT-OFF TO PREDICT PANCREATIC FISTULA. RESULTS OF A PROSPECTIVE STUDY.** Alessandra Pulvirenti, MD, Antonio Pea, MD, Valentina Allegrini, MD, Luca Casetti, MD, Phd, Roberto Salvia, MD, Phd, Claudio Bassi, MF, FACS; University of Verona, Verona, IT

**Background:** Post-operative pancreatic fistula (POPF) still remains the complication that mainly influence the morbity and the mortality after pancreaticoduodenectomy (PD). Currently, due to the severity of an untreated and undiagnosed pancreatic fistula, abdominal drainage is still recommended, but early drains removal is preferred in low-risk patients. Many models have been proposed to guide surgeons in drain removal decision making and drain management on the basis of Drain Amylase Value (DAV) is the most common. With this study we aim to analyze systematically the DAV in the first postoperative days in order to identify the most accurate threshold to predict the development of POPF.

**Methods:** Clinical data from patients, who underwent PD from September 2013 and December 2014 at our institution, were prospectively collected. The drain amylase value (DAV) was prospectively measured in post-operative day (POD) 1, 2, 3 and 5. Outcome was defined as the presence of POPF accordingly with the ISGPF definition and classification. DAV was evaluated using receiver operating characteristic (ROC) analysis to establish cut-offs predictive of POPF occurrence.

**Results:** 197 PD were performed at our Institution between September 2013 and December 2014. DAV in POD 1 and POD 3 were collected for 148 patients. At the ROC analysis, the POD 3 DAV was the stronger predictor of POPF (AUC 0,963). The POD 3 DAV 200 U/L was identified as cut-off showing the best accuracy (94,6%), the highest sensitivity (94,9%) and specificity (94,4%) to predict POPF occurrence. The ROC analysis for POD 1 DAV showed a AUC of 0,916, and the best DAV cut off was 600 U/L with the accuracy of 83%, sensitivity of 80,9 % and the specificity of 86,4%. In the POD 1, 8 (10%) patients with a DAV< 600 U/L developed POPF, of these were 4 (50%) A grade, 3 (37,5%) B and 1 (12,5%) C. In the group of patients with a POD 3 DAV≤200 U/L, 3 (3,4%) B grade POPFs were observed. Specific predictors of postoperative POPF were analyzed at univariate and multivariate analysis. DAV >200 U/L on POD 3 was confirmed as the strongest independent predictor for POPF development (OR 161,76, 95% CI 26,1-998,82; P < 0.0001).

**Discussion\Conclusion:** A new DAV of 600 U/L has been recently proposed as a new threshold to predict the POPF development. Our study confirms the DAV in the very first POD allows to predict POPF and that a DAV of ≥600 U/l on POD 1 has a good accuracy to predict POPF occurrence. In our cohort, 8 patients with POD 1 DAV < 600 U/L developed POPF and 50% of these were clinically relevant. The drain amylase value on POD 3 appears to have higher accuracy, sensitivity and specificity to predict the development of POPF. We therefore propose a new model for drain management that include early drain removal not before the third postoperative day using DAV>200 U/L in the POD 3 as new cut off. A prospectively validation of this model is currently in progress.
**S004 GRADING OF SURGEON TECHNICAL PERFORMANCE PREDICTS POST-OPERATIVE PANCREATIC FISTULA FOR THE PANCREATICODUODENECTOMY INDEPENDENT OF PATIENT RELATED VARIABLES** Melissa Hogg, Mazen Zenati, Stephanie Novak, Yong Chen, Yan Jun, Jennifer Steve, Stacy Kowalsky, David L Bartlett, Amer H Zureikat, Herbert J Zeh; 1UPMC, 2The first affiliated hospital-Chongqing Medical University?, 3Shanghai Jiao Tong University Affiliated Sixth People’s Hospital, Pittsburgh, US

**Background:** Pancreatic fistula (POPF) majorly contributes to pancreaticoduodenectomy morbidity. Braga and Callery scores, derived from patient variables, are validated for predicting POPF. Birkmeyer showed assessment of surgical proficiency is an important component of outcomes. We hypothesized that video grading of surgical performance would contribute to estimating risk of POPF following pancreaticoduodenectomy.

**Methods:** POPF were diagnosed using ISGPF. Technical performance of robotic pancreaticojejunostomy (Blumgart duct-to-mucosa) video was graded by two blinded surgeons using: 1) subjective prediction of POPF, 2) pancreaticojejunostomy step-by-step variables (PJV; max=115), and 3) and scoring used by Birkmeyer (OSATS).

**Results:** 133 pancreaticojejunostomies were analyzed (139.5 video hours). POPF was 18%. Higher Braga (p=0.041) and Callery (p=0.011) scores predicted POPF. Graders’ subjective prediction did not correlate with Braga/Callery scores. PJV and OSATS scores highly correlated (p<0.0001). Grader 1 scores (p=0.043), but not grader 2 (p=0.44), predicted POPF. PJV scores >105 were predictive of POPF (p=0.039). Scoring only PJV duct-to-mucosa stitches (max=50) was highly predictive of POPF (p=0.0053). Higher OSATS scores were associated with decreased rate of POPF (p=0.022). On multivariate analysis, adding technical scoring to significant patient variables (BMI, texture, and duct size) improves the model and can independently predict POPF. The strongest predictive model for POPF consisted of soft gland (Odds=18.28 [95%=2.19-152.57]) and low OSATS (Odds=0.82 [95%=0.70-0.96]). OSATS, modeled with Braga or Callery scores, independently predicted PF.

**Conclusion:** This is the first study to demonstrate that technical scoring of a surgeon’s performance independently predicts patient outcomes in pancreatic surgery. Future studies should consider how to incorporate this metric.

**S005 USING THE PANCREATIC DEMONSTRATION PROJECT TO DERIVE A FISTULA RISK SCORE FOR PREOPERATIVE RISK STRATIFICATION IN PATIENTS UNDERGOING PANCREATICODUODENECTOMY** Olga Kantor, Mark S Talamonti, Henry A Pitt, Charles M Vollmer, Taylor S Riall, Bruce L Hall, Marshall S Baker; 1University of Chicago, 2NorthShore University HealthSystem, 3Indiana University, 4University of Pennsylvania, 5University of Arizona, 6Washington University, Chicago, US

**Introduction:** The Fistula Risk Score (FRS) is a clinical tool developed from single-institutional data using intraoperative factors to characterize risk of clinically relevant pancreatic fistula (CR-POPF) following pancreaticoduodenectomy. This model employs at least one factor difficult to reliably quantitate (blood loss). We attempt to develop a FRS based on objective nationally accruing data more readily determined prior to resection.
Methods: Patients undergoing pancreaticoduodenectomy were culled from the NSQIP Pancreatic Demonstration Project. CR-POPFs were examined. A random 70% cohort sample was used for testing/model development and the remaining 30% for validation.

Results: 1731 patients underwent pancreaticoduodenectomy between 2011-2012. There were no differences in demographic/preoperative/intraoperative factors or CR-POPF rates between testing and validation cohorts. Univariate analysis was used to identify predictors of CR-POPF. Variables with p-value<0.1 were included in multivariate modeling. Five significant predictors of CR-POPF were identified and assigned points based on odds ratios: gender, BMI, ductal diameter, gland texture, and pathology [Table 1]. In the testing group, risk scores of 0 (negligible risk), 0.5-4 (low risk), 4.5-6 (low-intermediate risk), 6.5-8 (high-intermediate risk), and 8.5-10 (high risk) were associated with CR-POPF rates of 0%, 8.9%, 19.6%, 25.9%, and 45.2%. Similar values were seen using the validation cohort: 0%, 7.8%, 16.1%, 27.3%, and 38.9% respectively. On ROC curve modeling, AUC=0.688 (p<0.01).

<table>
<thead>
<tr>
<th>MODIFIED FRS MODEL</th>
<th>Odds Ratio (95% CI)</th>
<th>Risk Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female Male</td>
<td>Reference 1.64 (1.17-2.30)</td>
<td>0 1.5</td>
</tr>
<tr>
<td>Normal BMI</td>
<td>Reference 1.66 (1.12-2.47) 1.89 (1.23-2.91)</td>
<td>0 1.5 2</td>
</tr>
<tr>
<td>Overweight BMI</td>
<td>Reference 1.49 (0.75-2.97) 2.65 (1.32-5.32)</td>
<td>0 1 2.5</td>
</tr>
<tr>
<td>Obese BMI</td>
<td>Reference 0.95 (0.45-2.0) 2.47 (1.54-3.95)</td>
<td>0 0 2.5</td>
</tr>
<tr>
<td>Duct &gt;6mm</td>
<td>Reference 1.49 (0.75-2.97) 2.65 (1.32-5.32)</td>
<td>0 1 2.5</td>
</tr>
<tr>
<td>Duct 3-6mm</td>
<td>Reference 0.95 (0.45-2.0) 2.47 (1.54-3.95)</td>
<td>0 0 2.5</td>
</tr>
<tr>
<td>Duct &lt;3mm</td>
<td>Reference 1.49 (0.75-2.97) 2.65 (1.32-5.32)</td>
<td>0 1 2.5</td>
</tr>
<tr>
<td>Hard gland</td>
<td>Reference 0.95 (0.45-2.0) 2.47 (1.54-3.95)</td>
<td>0 0 2.5</td>
</tr>
<tr>
<td>Intermediate gland</td>
<td>Reference 0.95 (0.45-2.0) 2.47 (1.54-3.95)</td>
<td>0 0 2.5</td>
</tr>
<tr>
<td>Soft gland</td>
<td>Reference 0.95 (0.45-2.0) 2.47 (1.54-3.95)</td>
<td>0 0 2.5</td>
</tr>
<tr>
<td>PDAC/pancreatitis</td>
<td>Reference 1.51 (1.06-2.16)</td>
<td>0 1.5</td>
</tr>
<tr>
<td>Other pathology</td>
<td></td>
<td>0 1.5</td>
</tr>
<tr>
<td>Total</td>
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<tr>
<td>Negligible risk</td>
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<td>0 (0% risk)</td>
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<tr>
<td>Low risk</td>
<td></td>
<td>0.5-4 (&lt;10% risk)</td>
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<tr>
<td>Low-intermediate risk</td>
<td></td>
<td>4.5-6 (10-20% risk)</td>
</tr>
<tr>
<td>High-intermediate risk</td>
<td></td>
<td>6.5-8 (20-30% risk)</td>
</tr>
<tr>
<td>High risk</td>
<td></td>
<td>8.5-10 (&gt;30% risk)</td>
</tr>
</tbody>
</table>

Conclusion: This modified FRS allows for reliable estimation of CR-POPF risk using preoperative and easily determinable intraoperative risk factors. Based in NSQIP, this will allow easy comparison of institutional data to national norms and improved preoperative counseling regarding CR-POPF risk.
**ORAL ABSTRACTS**

**S006 IDENTIFICATION OF RISK FACTORS OF PANCREATIC EXOCRINE INSUFFICIENCY AFTER PANCREATICODUODENECTOMY USING 13C-LABELED MIXED TRIGLYCERIDE BREATH TEST**

Seiko Hirono, MD,1 Yoshiaki Murakami, MD,2 Manabu Kawai, MD,1 Ken-ichi Okada, MD,1 Kenichiro Uemura, MD,2 Takeshi Sudo, MD,2 Yasushi Hashimoto, MD,2 Naoya Nakagawa, MD,2 Naru Kondo, MD,2 Hiroki Yamaue, MD,1 1Wakayama Medical University, 2Hiroshima University, Wakayama, JP

**Introduction:** There are only a few reports concerning long-term pancreatic exocrine function after pancreatoduodenectomy (PD), although the number of long-term survivors has increased. We assessed pancreatic exocrine function after PD in 189 patients to identify risk factors for exocrine insufficiency.

**Methods:** The present study included 90 patients that underwent pancreaticogastrostomy (PG) at Hiroshima University Hospital and 99 patients that underwent pancreaticojejunostomy (PJ) at Wakayama Medical University Hospital, the standard reconstruction techniques during PD at the respective hospitals. We evaluated patients’ exocrine function by using the 13C-labeled mixed triglyceride breath test, a noninvasive test feasible in outpatient service units. Pancreatic exocrine insufficiency was defined as %13CO2 cumulative dose at 7 h (%CD-7 h) less than 5% of 13C-labeled mixed triglyceride breath test, as previous reported. We also analyzed long-term morphological changes of remnant pancreas by computed tomography (main pancreatic duct dilation and parenchymal atrophy), nutritional status (body weight change, serum total protein, albumin, and prognostic nutrition index (PNI) by calculating as 10 × albumin + 0.005 × total lymphocyte count), and endocrine function after PD.

**Results:** The results of the 13C-labeled mixed triglyceride breath test were significantly correlated with postoperative morphological changes (duct-parenchymal rate: r=-0.486, P<0.001), nutritional status (body weight change: r=0.243, P=0.001, prognostic nutritional index: r=0.254, P<0.001, serum albumin level: r=0.222, P=0.002, and serum total protein level: r=0.192, P<0.001), and endocrine function (glucose: r=-0.198, P=0.007, HbA1c: r=-0.180, P=0.014). The independent risk factors for the exocrine insufficiency after PD include hard pancreas (P=0.003, odds ratio: 3.157) and PG reconstruction (P=0.040, odds ratio: 2.321). When the results of the 13C-labeled mixed triglyceride breath test were compared between PG group and PJ group, the rate of postoperative pancreatic exocrine insufficiency was significantly higher in PG group (PG vs. PJ: 66 vs. 38%, P<0.001). In the patients with soft pancreas, the rate of pancreatic exocrine insufficiency was significantly higher in PG group (PG vs. PJ: 59 vs. 29%, P<0.001), although there was no significant difference of pancreatic exocrine insufficiency rate between PG and PJ group (PG vs. PJ: 75 vs. 60%, P=0.193). The atrophic changes of the remnant pancreas in the PG group were more severe than those in the PJ group (P<0.001). Furthermore, for patients with a soft pancreas, the postoperative body weight changes (P=0.022), prognostic nutritional index (P=0.018), serum total protein levels (P=0.014) as well as exocrine breath test were worse in the PG group, compared with the PJ group.

**Conclusion:** Our results showed that PJ reconstruction might be superior to PG during PD, from the viewpoint of long-term pancreatic exocrine function, although further prospective studies are needed.
S007 STENT ASSOCIATED INFECTIONOUS COMPLICATIONS AFTER PANCREATODUODENECTOMIES CAN BE PREVENTED BY PERIOPERATIVE ANTIBIOTIC THERAPY. Esther A Biesel, Olivia Sick, BS, Frank Makowiec, MD, Ulrich T Hopt, MD, Uwe A Wittel, MD; Universitatsklinikum Freiburg, Freiburg, DE

Background: Although the postoperative mortality after pancreatoduodenectomies has dramatically reduced during the last years, the perioperative morbidity still remains high and is situated between 20 and 50%. One of the factors still suspected being responsible for an increased rate of perioperative complications is the implantation of bile duct stents prior to surgery. As a consequence patients with bile duct stents receive antibiotic therapy for the first 5 postoperative days after pancreatoduodenectomy in our institution. In our retrospective single center study, we now evaluated the influence of preoperative bile duct stent and perioperative antibiotic therapy vs. primary operation of patients.

Methods: Clinical data of 716 patients undergoing a pancreatoduodenectomy at the University Hospital Freiburg between 2001 and 2015 were explored retrospectively, accessing a prospectively maintained pancreatic SPSS database. Postoperative fistula, delayed gastric emptying, and postoperative hemorrhage were graded by current international definitions. All patients with complete data concerning preoperative bile duct stenting and preoperative bilirubin were included in the analyses. After explorative analysis, statistical significance was examined by chi-square tests in case of normal distribution.

Results: 670 patients with pancreatoduodenectomy were included in the analyses of which 314 (46.9%) patients were previously treated with a bile duct stent. Histopathological diagnoses were not identical in both groups with chronic pancreatitis being overrepresented in the no stent group (p < 0.005).

No significant difference in postoperative pancreatic fistula or post pancreatectomy hemorrhage was observed in patients with stent or without bile duct stent (POPF grade B/C stent 16.1%, no stent 22.4%, p=0.362; PPH grade B/C stent 6.7%, no stent 12.2%, p=0.061). Surprisingly, infectious abdominal complications were reduced in patients with preoperative bile duct stent (stent 8.1% vs. no stent 15.9% p = 0.010) and the rate of bile duct complications such as postoperative insufficiencies of the biliodigestive anastomosis was reduced (stent 0.7% vs. no stent 5.3%, p<0.005) in patients receiving preoperative bile duct stent.

Conclusion: Perioperative antibiotic prophylaxis administered to stent-bearing patients appears to reduce the perioperative risk for abdominal infectious diseases. In our collective preoperative bile duct stents even reduce bile duct complications due to the thickened bile duct wall regularly observed in these patients.

S008 MODIFIED FRAILTY INDEX PREDICTS MORBIDITY AND MORTALITY AFTER PANCREATICODUODENECTOMY Harveshp Mogal, MD, Rebecca Dobson, MD, Nora Fino, MS, Russell Howerton, MD, Perry Shen, MD, Clancy J Clark, MD; Wake Forest Baptist Health, Winston Salem, US

Introduction: Pancreatic cancer is a disease of older adults who may present with limited physiologic reserve. We hypothesized that a frailty index can predict postoperative outcomes after pancreaticoduodenectomy.
Methods: All patients who underwent pancreaticoduodenectomy were identified in the 2005-2012 NSQIP Participant Use File. Patients undergoing emergency procedures, ASA 5, or diagnosed with preoperative sepsis were excluded. A modified Frailty Index (mFI) was defined by 11 variables within NSQIP previously used for the Canadian Study of Health and Aging-Frailty Index. mFI score of 0.27 or more was defined as high mFI. Univariate and multiple variable analyses were performed to evaluate postoperative outcomes.

Results: 9986 patients (age 65+/12, 48.8% female) underwent pancreaticoduodenectomy with 6.4% (n= 637) having a high mFI (>=0.27). Increasing mFI was associated with higher prevalence of postoperative morbidity (p < 0.001) and 30-day mortality (p <0.001). On univariate analysis, high mFI was associated with increased morbidity (OR 1.68, 1.43-1.97 95% CI, p <0.001) and 30-day mortality (OR 2.45, 1.74-3.45 95% CI, p < 0.001). After adjusting for age, sex, ASA, albumin < 3, and BMI, high mFI remained an independent preoperative predictor of postoperative morbidity (OR 1.42, 1.15-1.75 95% CI, p = 0.001) and 30-day mortality (OR 1.54, 1.05-2.25 95% CI, p 0.027).

Conclusions: High modified Frailty Index is associated with postoperative morbidity and mortality after pancreaticoduodenectomy and can aid in preoperative risk stratification.

S009 IMPACT OF SARCOPENIA ON SURGICAL OUTCOMES IN PATIENTS UNDERGOING PANCREATICODUODENECTOMY BY USING ENANCHED RECOVERY AFTER SURGERY (ERAS) PROTOCOL. Valeri Sergio, MD1, Emerenziani Sara, MD2, Borzomati Domenico, MD, PhD, FACS1, Cicala Michele, MD2, Luffarelli Paolo, MD1, Muscaritoli Maurizio, MD4, Giorgio GiovanBattista, MD1, Beomonte Zobel Bruno, MD3, Coppola Roberto, MD, FACS3, 1Department of General Surgery, Campus Bio-Medico University, Rome, 2Department of Gastroenterology, Campus Bio-Medico University, Rome, 3Radiology Unit, Campus Bio-Medico University, Rome, 4Department of Clinical Medicine, Sapienza University, Rome, Rome, IT

Background: Sarcopenia is characterized by a loss of skeletal muscle mass, leading to impaired function and physical performance. Retrospective studies have shown that preoperative sarcopenia is associated with poor clinical outcome after surgery. The effect of sarcopenia in consecutive surgical pancreatic cancer (PC) patients has not been investigated yet. Enhanced Recovery After Surgery (ERAS) is an interdisciplinary approach designed to accelerate postoperative recovery and to reduce morbidity and mortality. The aim of the present study was to assess the impact of sarcopenia on postoperative outcome in consecutive PC patients undergoing pancreaticoduodenectomy (PD) comply with ERAS protocol.

Methods: After informed consent was obtained, 25 patients (10 M, mean age 66±12) were consecutively enrolled at the Surgical Department at the Campus Bio-Medico University in Rome, Italy. All patients had been diagnosed with resectable pancreatic ductal adenocarcinoma or distal cholangiocarcinoma cancer and underwent PD. Surgical procedures were performed by the same team of surgeons in all patients. All patients followed the ERAS protocol after surgery. Muscle mass was assessed on routine preoperative computed tomography (CT) scans using image analysis by Osirix® by measuring skeletal muscle at the third lumbar vertebra (L3) level. Patients were classified as sarcopenic or non-sarcopenic according to sex-specific cut-offs based on the literature (Prado 2008). Rate of post-operative infectious complication, length of
hospital stay and overall mortality were considered as surgical outcomes. Blood samples were collected for C-Reactive Protein (CRP) as a marker of inflammation. Results were expressed as mean ± SD. Statistical analysis was performed using the Prism 5.0 ® software, with appropriate tests according to the variables analysed.

**Results:** Sarcopenia (i.e reduced muscle mass at CT scan) was present in 16 out of 25 patients (64%). Infectious complications occurred in 9 patients (36%), length of hospital stay was 17 ± 14.8 days. Overall readmission was 12%, while overall mortality was 8%. No difference in terms of age, sex and presence of comorbidities were observed among sarcopenic and non-sarcopenic patients. Rate of post-operative infections was 11% in non-sarcopenic and 50% in sarcopenic (p<0.03). Length of hospital stay (days) was 20.9 ±18 and 9.6 ±3 in sarcopenic and non-sarcopenic patients respectively (p<0.04). CRP units were significantly higher in sarcopenic than in non-sarcopenic patients (8.3 ± 6.5 vs 24.8 ± 27 respectively; p<0.04). Readmission rate was 22.2% in non-sarcopenic and 6.25% in sarcopenic group (p = 0.332). Overall mortality was 0 in non-sarcopenic and 12.5% in sarcopenic patients (p = 0.268).

**Conclusions:** Preoperative sarcopenia is highly prevalent in PC patients undergoing PD. PC patients with preoperative sarcopenia show significantly higher post-operative infections rate, higher CRP values and longer hospital stay compared to those without sarcopenia. This study strengthens the view that preoperative sarcopenia may independently predict complication rate in PC patients undergoing PD comply with ERAS protocol.

**SO10 ROBOTIC WHIPPLE BIOTISSUE CURRICULUM IMPROVES TECHNICAL PERFORMANCE FOR FELLOWS AND HAS CONSTRUCT VALIDITY**

Melissa E Hogg¹, Mazen Zenati¹, Stephanie Novak¹, Yong Chen², Amer H Zureikat¹, Herbert J Zeh¹; ¹UPMC, ²The first affiated hospital?Chongqing Medical University?, Pittsburgh, US

**Introduction:** Many view the pancreaticoduodenectomy as the most complex intra-abdominal operation. Additionally, the past five years have seen a rise in performing the procedure with minimally invasive technology. A major criticism of incorporating the robotic platform for the pancreaticoduodenectomy is that it is difficult to teach and disseminate the technique. Our group has experience with >300 robotic pancreaticoduodenectomies and has published a learning curve of 80 cases to optimize performance. We hypothesize by instituting an advanced robotic training curriculum we can decrease that learning curve for robotic whipples.

**Methods And Procedures:** A three-step curriculum: 1) simulation, 2) biotissue and 3) operative coaching was implemented. The biotissue curriculum consisted of sewing artificial organs to simulate a hepaticojejunostomy (HJ), gastrojejunostomy (GJ) and pancreaticojejunostomy (PJ). These were evaluated for time, errors and Birkmeyer Score (30 top score, 6 categories with a Likert scale of 5). Three attendings with experience of greater than 80 robotic pancreaticoduodenectomies performed each biotissue anastomosis once for validity. Two blinded graders scored all videos.

**Results:** Fourteen fellows performed 195 anastomoses during first year: 66 (HJ), 64 (GJ) and 66 (PJ). The attendings’ first attempt outperformed the fellows’ first attempt in all 9 categories. The fellows’ performances were analyzed as a group by attempt. For the HJ, time, errors and Birkmeyer all improved linearly over 7 analyzed attempts (p<0.007).
For the GJ, time, errors and Birkmeyer all improved linearly over 9 analyzed attempts (p<0.002). For the PJ, errors and Birkmeyer both improved linearly over 8 analyzed attempts (p<0.002); however, time trended down without plateauing but did not reach statistical significance (p=0.08). The attendings’ first attempts were faster than fellows’ last attempt for all anastomoses (p<0.041). For the GJ and PJ but not HJ, errors and Birkmeyer were on par between attendings’ first and fellows’ last attempt. The graders scoring correlated for errors and Birkmeyer (p<0.0001). Since incorporating the biotissue curriculum, every fellow completing a three-month rotation has successfully performed a robotic pancreaticoduodenectomy (p<0.05; compared to before the curriculum).

**Conclusion:** A pancreaticoduodenectomy biotissue curriculum has face and construct validity. It can improve fellow performance in both an inanimate and operative environment. Time is the most difficult parameter in which to reach attending mastery. The PJ takes more attempts to show improvement, and the HJ is the most difficult to obtain attending mastery performance. This curriculum is a valid tool for teaching robotic pancreaticoduodenectomies with established milestones for reaching optimum performance.

**S011 ROBOTIC VERSUS OPEN PANCREATODUODENECTOMY: A PROPENSITY SCORE-MATCHED ANALYSIS OF PANCREATIC FISTULA**

Matthew T McMillan, BA1, Amer H Zureikat, MD2, Melissa E Hogg, MD2, Stacy J Kowalsky, MD2, Jeffrey A Drebin, MD, PhD1, Herbert J Zeh, MD2, Charles M Vollmer, MD1; 1University of Pennsylvania Perelman School of Medicine, 2University of Pittsburgh Medical Center, Philadelphia, US

**Introduction:** The adoption of robotic pancreatoduodenectomy (RPD) is gaining momentum, however its impact on major outcomes, including pancreatic fistula, has yet to be adequately compared with open pancreatoduodenectomy (OPD). Propensity score-matching offers a method to minimize bias from non-randomized treatment assignment. This approach was applied in a multi-center setting to compare clinically relevant fistula (CR-POPF) outcomes between patients undergoing RPD and OPD.

**Methods:** Data were accrued from 2846 pancreatoduodenectomies (OPD, N=2661; RPD, N=185), performed by 51 surgeons at 15 institutions worldwide (2003-2015). All RPDs were conducted at a single-center — in a standardized fashion — by surgeons who had surpassed the RPD learning curve. RPD and OPD cohorts were matched by propensity scores accounting for factors significantly associated with either undergoing robotic surgery or CR-POPF occurrence on logistic regression analysis. These variables included: pancreatic gland texture, pancreatic duct diameter, intraoperative blood loss, disease pathology, intraoperative drain placement, octreotide prophylaxis, and trans-anastomotic stent placement.

**Results:** The propensity score-matched cohort comprised 185 RPD and 152 OPD; all covariate imbalances were alleviated. Patients undergoing RPD demonstrated similar CR-POPF rates compared with patients in the OPD cohort (8.6 vs. 11.2%, p=0.436). This relationship held for all levels of ISGPF fistula (Grades A, B, and C). There were also equivalent outcomes between the matched RPD and OPD cohorts in terms of the occurrence of any complication, severe complications (Clavien-Dindo ≥3), hospital stay, 30-day readmission, and 90-day mortality (Table).
**Conclusion:** This largest comparative analysis of robotic versus open pancreatoduodenectomy demonstrates equivalency in terms of pancreatic fistula development and other major postoperative outcomes.

<table>
<thead>
<tr>
<th>Variable, N (%)</th>
<th>Open PD</th>
<th>Robotic PD</th>
<th>p-value</th>
</tr>
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<tbody>
<tr>
<td>Patients</td>
<td>152</td>
<td>185</td>
<td>-</td>
</tr>
<tr>
<td>ISGPF pancreatic fistula (POPF)</td>
<td>34 (22.4)</td>
<td>31 (16.8)</td>
<td>0.194</td>
</tr>
<tr>
<td>Grade A POPF</td>
<td>17 (11.2)</td>
<td>15 (8.1)</td>
<td>0.338</td>
</tr>
<tr>
<td>Clinically relevant POPF</td>
<td>17 (11.2)</td>
<td>16 (8.6)</td>
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<tr>
<td>Grade B POPF</td>
<td>14 (9.2)</td>
<td>16 (8.6)</td>
<td>0.857</td>
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<tr>
<td>Grade C POPF</td>
<td>3 (2.0)</td>
<td>0 (0)</td>
<td>0.091</td>
</tr>
<tr>
<td>Any complication (Clavien 1-5)</td>
<td>101 (66.4)</td>
<td>136 (73.5)</td>
<td>0.158</td>
</tr>
<tr>
<td>Mild complication (Clavien 1-2)</td>
<td>65 (42.8)</td>
<td>93 (50.3)</td>
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<tr>
<td>Severe complication (Clavien ≥3)</td>
<td>36 (23.7)</td>
<td>43 (23.2)</td>
<td>0.924</td>
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<tr>
<td>Readmission (30-day)</td>
<td>33 (21.7)</td>
<td>40 (21.6)</td>
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<td>Duration of hospital stay, days</td>
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<tr>
<td>Mean ± SD</td>
<td>11.8 ± 10.6</td>
<td>10.9 ± 7.5</td>
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<tr>
<td>Median (IQR)</td>
<td>8.5 (7-12)</td>
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<td>Mortality (90-day)</td>
<td>2 (1.3)</td>
<td>5 (2.7)</td>
<td>0.464</td>
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</table>

**S012 ROBOT-ASSISTED VERSUS OPEN PANCREATICODUODENECTOMY: A CASE-MATCHED STUDY BASED ON CLINICAL RISK SCORE FOR PANCREATIC FISTULA**

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**Introduction:** Clinical risk score for pancreatic fistula (CRS-PF) predicts postoperative pancreatic fistula (POPF) after both laparoscopic and open pancreaticoduodenectomy (PD). We herein report a case-matched comparison between laparoscopic robot-assisted PD (LRA-PD) and open PD (O-PD) based on CRS-PF stratification.

**Methods:** Between 2007 and 2014, 344 PD were performed at our institution including 85 LRA-PD and 259 O-PD. For the purpose of this study we decided to analyze only the outcome of patients receiving a pancreaticojejunostomy, using either an invaginating or a duct-to-mucosa technique. The primary study end-point was incidence and severity of POPF, which was defined and classified according to definition of the international study group of pancreatic fistula. B and C grade POPF were defined as clinically relevant (CR-POPF).

**Results:** The CRS-PF was calculated for 204 PD, including 73 LRA-PD and 131 O-PD. POPF occurred in 26 LRA-PD (35.6%) and in 29 O-PD (22.1%) (p=0.04*; Pearson). CR-POPF occurred in 19 LRA-PD (26%) (B= 14; C= 5) and in 14 O-PD (10.7%) (B= 10; C= 4) (p=
There were no POPF in patients classified at the low risk (score 1-2), in both study groups (table 1). Sixty-eight LRA-PD were matched to 68 O-PD based on CRS-PF (table 2). No POPF occurred in patients classified at low risk (score 1-2), in both study groups (table 1).

Sixty-eight LRA-PD were matched to 68 O-PD based on CRS-PF (table 2). No POPF occurred in patients classified at intermediate risk (score 3-6). POPF occurred in 19 LRA-PD (45.2%) and in 8 O-PD (19.1%) (p=0.01*, Pearson). CR-POPFF occurred in 17 LRA-PD (40.5%) (B= 13; C= 4) and in 4 O-PD (9.5%) (B= 2; C= 2) (p=0.002*; Fisher). In patients classified at high risk (score 8-10), POPF occurred in 6 LRA-PD (28.6%) and in 12 O-PD (57.1%) (p=0.06; Pearson). CR-POPFF occurred in 2 LRA-PD (9.5%) (B= 1; C= 1) and in 5 O-PD (23.8%) (B= 5; C= 0) (p=0.41; Fisher) (figure 1).

In the intermediate risk category the OR for POPF in patients undergoing LRA-PD vs O-PD was 6.5 (1.67-42.5 confidence interval; p=0.004*Mid-p). In the high risk category the equivalent figure was 0.25 (0.04-1.08 confidence interval; p=0.07 Mid-p).

Discussion/Conclusion: This is the first case-matched comparison between LRA-PD and O-PD based on CRS-PF. Our analysis provides some novel information. First, the initial selection for LRA-PD excludes patients at negligible risk for POPF, unbalancing non-stratified comparison for POPF between LRA-PD and O-PD. Second, patients at low risk did not develop POPF in our experience irrespective of surgical technique. Provided that LRA-PD does not compromise oncologic radicality, this could be the ideal group of patients to which LRA-PD should be offered. Third, LRA-PD is associated with higher incidence of POPF in the intermediate risk category but with lower incidence of POPF in the high risk category. These are probably the patients that should be included in a prospective randomized comparison. Implementation of a prospective multi-institutional registry would also be of great value.
pathologically classified as pancreatic ductal adenocarcinomas (85.7% and 85.3%). Median operative time was longer for DP-CAR as compared to DP alone (405 min vs 238 min, P < 0.001). DP-CAR was associated with significantly increased blood loss (median: 1000 mL vs 450 mL, P = 0.013), and longer length of stay (median: 7.5 days vs 6 days, P = 0.034). In the DP-CAR group, 2 (14.3%) patients had a concomitant portal vein resection and reconstruction. There was no difference in margin negative resection rates between groups (76.9% vs 87.2%, P = 0.223). No difference in overall complications (42.9% vs 26.9%, P = 0.322) or postoperative pancreatic fistula (7.1% vs 22.0%, P = 0.423) was observed between DP-CAR and DP groups. There was no difference in either 30-day (28.6% vs 31.7%, P = 0.826) or 90-day (4.8% vs 14.2%, P = 0.915) readmission rates. No peri-operative mortalities occurred in either group. Long-term follow-up was available for 11 (78.6%) DP-CAR patients, and the 1- and 3-year overall survival was 79.6% and 31.8%, respectively.

**Conclusion:** The modified Appleby procedure can be performed safely and is a viable treatment option for patients with tumors involving the CA.

**SO14 IMPACT OF A NATIONWIDE TRAINING PROGRAM IN LAPAROSCOPIC DISTAL PANCREATECTOMY (LAELAPS)** Thijs de Rooij¹, Jony van Hilst¹, Djamila Boerma², Peter van den Boezem³, Bert Bonsing⁴, Koop Bosscha⁵, Peter-Paul Coene⁶, Freek Daams⁷, Ronald van Dam⁸, Cees Dejong⁹, Marcel Dijkgraaf¹, Casper van Eijck⁸, Joris Erdmann¹⁰, Sebastiaan Festen¹¹, Michael Gerhards¹², Bas Groot Koerkamp⁹, Erwin van der Harst¹, Ignace de Hingh¹², Geert Kazemier⁷, Joost Klaase¹³, Ruben de Kleine¹⁶, Kees van Laarhoven³, Daan Lips¹⁴, Misha Luyer¹², Quintus Molenaar¹⁴, Vincent Nieuwenhuijs¹⁵, Gijs Patijn¹⁵, Daphne Roos¹⁶, Hjalmar van Santvoort², Joris Scheepers¹⁶, George van der Schelling⁷, Pascal Steenvoorde¹³, Lieke Welling⁴, Jan Wijsman¹⁷, Olivier Busch¹, Dirk Gouma¹, Mohammed Abu Hilal¹⁸, Marc Besselink¹; ¹Academic Medical Center, ²St Antonius Hospital, ³Radboud Nijmegen University Medical Center, ⁴Leiden University Medical Center, ⁵Jeroen Bosch Hospital, ⁶Maasstad Hospital, ⁷VU University Medical Center, ⁸Maastricht University Medical Center, ⁹Erasmus University Medical Center, ¹⁰University Medical Center Groningen, ¹¹Onze Lieve Vrouwe Gasthuis, ¹²Catharina Hospital, ¹³Medisch Spectrum Twente, ¹⁴University Medical Center Utrecht, ¹⁵Isala Clinics, ¹⁶Reinier de Graaf Gasthuis, ¹⁷Amphia Hospital, ¹⁸Southampton University Hospital, Amsterdam, NL

**Introduction/background:** Expert centers report superior outcomes of laparoscopic distal pancreatectomy (LDP) compared with open distal pancreatectomy. In the Netherlands (2005-2013) 10% of distal pancreatectomies was LDP (36% conversion) and LDP training was welcomed by 85% of surgeons.¹ The feasibility and impact of a nationwide LDP training program was unknown. Aim of this study was to assess the impact of a nationwide training program in LDP.

**Methods:** From Jan-2014 to Jul-2015, 32 pancreatic surgeons from all 17 centers of the Dutch Pancreatic Cancer Group participated in a nationwide LDP training program, including video training, detailed technique description and on-site proctoring by two (inter)national proctors (LAELAPS). Participating surgeons had experience with laparoscopic gastrointestinal surgery and worked in a high-volume pancreatic center performing >20 pancreatoduodenectomies annually. Retrospectively collected LDP outcomes before training (Jan-2005 to Dec-2013) were compared with prospectively collected LDP outcomes after training (Jan-2014 to Oct-2015). Sensitivity analysis was performed by excluding centers that performed > 10 LDPs after training.
Results: In total, 180 patients were included, of whom 69 patients underwent LDP in 9 years before training vs. 111 patients in 21 months after training (7-fold increase; P < 0.001). Groups were comparable for baseline characteristics, operative time and blood loss. The conversion rate was lower in the period after training (36% (n = 25) vs. 8% (n = 9) patients; P < 0.001). After training, relatively more pancreatic adenocarcinomas were resected (7 (10%) vs. 27 (24%); P = 0.02), with comparable R0 resection rates (4/7 (57%) vs. 19/27 (70%); P = 0.51), ISGPF grade B/C pancreatic fistulas (20 (29%) vs. 40 (36%) patients; P = 0.33) and Clavien-Dindo ≥ 3 complications (14 (20%) vs. 19 (17%) patients; P = 0.59) for both groups. Length of stay was shorter after training (9 (7-12) vs. 7 (5-8) days; P < 0.001) and 30-day mortality was 3% vs. 0% (P = 0.07). Sensitivity analysis showed similar conversion rate, complication rate and length of stay.

Discussion/conclusion: A nationwide training program in LDP was feasible and was followed by an increase in the LDP use, including in cancer patients, and decreased conversion rates. Future studies will have to determine whether these training programs are also applicable in other settings.


S015 LAPAROSCOPIC VERSUS OPEN DISTAL PANCREATECTOMY: THE OUTCOMES OF JAPANESE MULTICENTER COMPARATIVE STUDY USING PROPENSITY SCORE-MATCHING
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Background: Laparoscopic distal pancreatectomy (LDP) has widely been accepted for the treatment of benign and low-grade malignant lesions of the body and tail of the pancreas. Many studies showed that LDP was associated with better postoperative outcomes compared to open distal pancreatectomy (ODP). However, these studies were retrospective and did not include any randomized control trials, and, therefore, confounding bias cannot be denied. Propensity score-matching analysis has been advocated to minimize confounding bias in observational studies. To establish evidence concerning perioperative outcomes of LDP, Japanese Society of Hepato-Biliary-Pancreatic Surgery and Japanese Society for Endoscopic Pancreatic Surgery conducted a multicenter study to compare perioperative outcomes of LDP and ODP using propensity score-matching analysis.

Methods: We retrospectively collected perioperative data of 2,266 patients who underwent distal pancreatectomy for benign or low-grade malignant lesion in 69 institutes of Japan from 2006 to 2013. Among the 2,266 patients, 2,010 patients were eligible to this study. The 2,010 patients were divided into 2 groups, ODP (n=1,108) and
LDP (n=902). A propensity score was estimated by a logistic regression model using the following 14 relevant variables: age, gender, body mass index, maximum tumor size, location of tumors, prior abdominal surgery, comorbidity, hemoglobin, albumin, CEA, CA19-9, pancreatitis other than obstructive pancreatitis, obstructive pancreatitis, and combined resection of other organs. One patient in LDP group was matched to one patient in ODP group according to a propensity score using a greedy nearest-neighbor algorithm. Perioperative outcomes were compared between the matched groups.

**Results:** In 58 patients, LDP was converted to ODP. These cases were included in LDP based on intention-to-treat analysis. After propensity score matching, 1,458 patients, including 729 patients in LDP group and 729 patients in ODP group, remained. LDP was associated with higher rate of preservation of spleen and splenic vessels (P < 0.001); lower rates of intraoperative transfusion (P = 0.020), clinically significant pancreatic fistula (International Study Group on Pancreatic fistula grade B and C; P < 0.001), and morbidity (P < 0.001); and shorter hospital stay (P = 0.001) and longer operative time (P < 0.001) while drain amylase levels on 1POD, days of drainage, day of meal intake and rate of intraoperative complication, any pancreatic fistula (International Study Group on Pancreatic fistula grade A-C) and mortality were not significantly different between two groups.

**Conclusions:** This multicenter comparative study of a large cohort using propensity score-matching revealed that LDP was associated with more favorable perioperative outcomes than ODP in the treatment of benign and low-grade pancreatic lesions of the pancreas.

**S016 NEOADJUVANT CHEMOTHERAPY IS ASSOCIATED WITH A SURVIVAL ADVANTAGE IN EARLY STAGE PANCREATIC HEAD CANCER**

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**Introduction:** There continues to be substantial debate regarding the efficacy of neoadjuvant chemotherapy (NCT) given prior to resection in early stage pancreatic cancer.

**Methods:** We queried the National Cancer Data Base to identify patients that underwent pancreaticoduodenectomy (PD) for clinical stage (cStage) I-II pancreatic adenocarcinoma (PDAC) between 2006 and 2012. Multivariate logistic regression was used to analyze treatment trends and outcomes. Cox-modeling was used for survival analysis.

**Results:** For the period studied, 7,881 patients underwent PD for cStage I-II PDAC. 3,083 (39.1%) were cStage I and 4,798 (60.9%) were cStage II. A total of 1,188 (15.1%) of the total received NCT. 4,523 (57.4%) received adjuvant chemotherapy (ACT), and 2,170 (27.5%) received no chemotherapy. Of the NCT patients, 319 (26.9%) received “perioperative chemotherapy” (PCT) receiving both NCT and postoperative chemotherapy. Use of NCT nearly doubled over the period evaluated (12.0% of total patients in 2006 to 20.2% in 2012, p<0.01). Patients were more likely to receive NCT if they had private insurance (19.5% vs. 8.2%, P<0.01), had vascular abutment (42.0% vs. 11.9%, p<0.01), were treated at an academic center (17.6% vs. 10.8%, p<0.01), or at a high

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volume hospital (19.2% vs. 11.6%, p<0.01). Patients receiving NCT were more likely to have margin negative resection (80.2% vs. 73.0%, p<0.01) and be lymph node negative on final pathology (58.2% vs. 41.4% p<0.01) than those that did not. Multivariate regression adjusting for age, sex, race, comorbidities, insurance, socio-economic status, hospital type, location and volume, tumor grade, and vascular abutment identified patient age ≤55 years (OR 2.44, CI: 1.81-3.30), African-American race (OR 1.36, CI: 1.06-1.75), private insurance (OR 2.60, CI: 1.45-4.63), tumors that had vascular abutment (OR 4.20, CI: 3.53-5.01), cStage II disease (OR 1.62, CI: 1.39-1.88), and treatment at a facility with high surgical volume (OR 1.65, CI: 1.27-2.15) to be factors independently associated with use of NCT. Cox survival analysis adjusted for age, sex, race, comorbidities, insurance, socio-economic status, hospital type, location and volume, tumor grade, margins, radiation therapy, pathological stage, and vascular abutment demonstrated a significant survival advantage in patients receiving PCT. The median overall survival for patients receiving PCT was 25.8 months vs. 21.2 months for NCT only, 23.1 months for ACT only, and 14.9 months for patients receiving no chemotherapy (p<0.05). The survival advantage associated with PCT was statistically significant for patients that had both cStage I and cStage II disease.

Conclusion: Patients with cStage I and II PDAC treated with NCT prior to resection demonstrate higher rates of margin negative and node negative resection than stage-matched patients receiving no chemotherapy prior to resection. Patients who receive NCT without additional postoperative chemotherapy demonstrate rates of overall survival similar to patients who receive ACT alone while PCT appears to be associated with a statistically relevant survival benefit for patients presenting with clinical stage I and II disease.

S017 PREOPERATIVE THERAPY FOR PATIENTS WITH PANCREATIC DUCTAL ADENOCARCINOMA UNDERGOING PANCREATODUODENECTOMY: IMPACT OF RADIATION DOSE ON OUTCOMES Jordan M Cloyd, Christopher Crane, Eugene Koay, Rebecca Snyder, Prajnan Das, Sunil Krishnan, Huamin Wang, Michael Kim, Jeffrey E Lee, Guari Varadhachary, Milind Javle, Rachna Shroff, Robert Wolff, David Fogelman, Matthew Katz; MD Anderson Cancer Center, Houston, US

Introduction: We have previously demonstrated that preoperative chemoradiation is associated with an improved margin negative resection rate and local tumor control among patients who undergo pancreatoduodenectomy (PD) for pancreatic ductal adenocarcinoma (PDAC). However, the optimal preoperative regimen has not been established.

Methods: Consecutive patients with PDAC who received both preoperative chemoradiation and PD between 1999-2015 were retrospectively reviewed. The effects of two external-beam radiation therapy (RT) regimens were compared: a hypofractionated course of 30Gy/10 fractions and a standard course of 50.4Gy/28 fractions. Differences in clinicopathologic characteristics, superior mesenteric artery (SMA) margin distance, local recurrence (LR), recurrence free survival (RFS) and overall survival (OS) were assessed.

Results: 443 patients received either 30Gy (n=224) or 50.4Gy (n=219) of RT with concurrent gemcitabine or capecitabine followed by PD. Patients who received 50.4Gy were more likely to have received induction chemotherapy (43.3% vs 61.6%, p<0.001),
received capecitabine as a sensitizing agent (40.6% vs 56.6%, p<0.001), required vascular resection (33.9% vs 46.1%, p<0.05) and received treatment later in the study period (17.4% vs 50.7%, p<0.0001). There was no difference in the R1 margin status (tumor cells ≤1mm from any margin: 25.4% vs 22.8%), SMA margin length (6.1 ± 5.9mm vs 6.7 ± 6.2mm), or treatment effect (complete pathologic response 3.1% vs 3.7%). 50.4Gy was associated with a lower frequency of positive lymph nodes (58.9% vs 46.6%, p<0.01) and a lower lymph node ratio (0.09 ± 0.12 vs 0.06 ± 0.1, p<0.01). There was no difference in LR, RFS or OS (Figure). On multivariate (HR 0.93, 95% CI 0.71-1.22) Cox proportional hazards analysis, 50.4Gy was not associated with improved survival.

Conclusion: Notwithstanding potential differences in pre-treatment tumor size and disease stage, preoperative hypofractionated chemoradiation for PDAC was associated with similar pathologic and survival outcomes following PD compared to standard fractionated radiation.

**S018 DOES RADIOLOGIC RESPONSE CORRELATE TO PATHOLOGIC RESPONSE IN PATIENTS UNDERGOING NEOADJUVANT THERAPY FOR PANCREATIC MALIGNANCY?**
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**Introduction:** In the modern era, neoadjuvant therapy is increasingly being utilized for patients with pancreatic malignancy, with radiographic response serving as the primary driver to determine if patients should be offered surgical intervention. We sought to determine whether there was any correlation between radiographic and pathologic response rates, and their influence on patient outcomes.

**Methods:** Between 2005 to 2015, 40 patients underwent neoadjuvant therapy followed by pancreaticoduodenectomy for pancreatic cancer. Perioperative imaging and pathology were reviewed. Radiographic response post-neoadjuvant therapy and pathologic response were graded according to RECIST and Evans’ criteria, respectively. Evans grade IIB was designated partial response.

**Results:** There were 21 male and 19 female patients, with a median age of 63 years. The proportion of patients that harbored borderline, locally advanced, and resectable diseases were 62.5% (n=25), 32.5% (n=13), and 5% (n=2), respectively. Preoperatively, 47.5% of patients underwent chemotherapy alone and 52.5% underwent chemotherapy/chemoradiotherapy. The most common chemotherapy regimens were single-agent gemcitabine (n=8), gemcitabine/nab-paclitaxel (n=14), and gemcitabine/erlotinib (n=7). Altogether, 67.5% (n=27) of patients had stable disease (SD), and 30% (n=12) demonstrated a partial response (PR) based on preoperative imaging (RECIST criteria).
The remainder (n=1) had progressive disease (PD). Of patients with SD on imaging, 25.9% (n=7) had Evans grade IIB or greater pathologic response. Among patients with vascular involvement and without radiographic response, 76.5% (n=13) achieved a R0 resection. Pathologically, 67.5% (n=27) of patients had Evans grade I-IIA response, 27.5% (n=11) had a grade IIB-III response, and 5% (n=2) had a grade IV complete response. The majority of patients who achieved a partial or complete pathologic response underwent both chemotherapy and chemoradiotherapy.

**Conclusions:** Our data indicates that (1) approximately one-fourth of patients who did not have a RECIST response on imaging had a grade IIB or greater pathologic response, (2) in the absence of distal disease, lack of down-staging following neoadjuvant therapy should not be utilized to determine resectability, and (3) pathologic response is most likely to occur when patients receive both chemotherapy and chemoradiotherapy.

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**S019 SURVIVAL AFTER NEOADJUVANT THERAPY AND RESECTION VERSUS RESECTION ALONE FOR EARLY STAGE PANCREATIC CANCER: A PROPENSITY SCORE MATCHED ANALYSIS IN A NATIONAL COHORT OF PATIENTS**

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**Background:** Neoadjuvant therapy (NAT) for early-stage resectable pancreatic adenocarcinoma remains controversial. Several single-arm phase II trials have suggested a potential benefit of NAT for resectable pancreatic cancer. No previous studies have addressed this issue using a national cohort of patients. We compared survival between pancreatic cancer resection alone and resection following neoadjuvant therapy in a large national cancer dataset.

**Methods:** Patients with resected, stage I and II, pancreatic adenocarcinoma were identified in the National Cancer Data Base from 2006 through 2012. Preoperative clinical and postoperative management-based propensity scores were used to match, in a one to one manner, patients that underwent NAT (chemotherapy or chemoradiotherapy) followed by resection to those that underwent resection only. Kaplan-Meier survival curves and absolute differences in mortality between resection alone and NAT plus resection were estimated. Relative survival effect of the addition of NAT to resection was examined using a Cox regression model.

**Results:** Over a seven-year period, we identified 30,921 patients with stage I and II pancreatic adenocarcinoma that underwent definitive resection; 27,745 underwent resection only and 3,176 had NAT followed by resection. We matched 3,153 (99.3%) patients that underwent NAT and resection to 3,153 resection-only patients. In the matched sample, the median followup time was 21 months for the entire cohort. The median survival times were 21 months in the resection only and 26 months in the NAT plus resection group (stratified log-rank p < 0.01), respectively. An early protective effect in the NAT plus resection group dissipated three and a half years after the time of diagnosis (Figure 1). The absolute reductions in mortality between NAT plus resection versus resection only were 15%, 8%, and -2% at 1, 2, and 5 years. The estimated hazard ratio for death was 0.83 (95% confidence interval = 0.78 - 0.88) for NAT plus resection to resection only.

**Conclusions:** NAT followed by resection for early stage pancreatic cancer may have a transient survival benefit, but similar 5-year survival, when compared to resection alone.
Figure 1. Kaplan-Meier survival curves (with 95% confidence intervals) for propensity score matched patients with early stage pancreatic cancer that underwent resection only versus NAT plus resection. Survival rates at 1, 2, 3, and 5 years in the NAT plus resection group were 84%, 54%, 36%, and 21%, compared to 69%, 46%, 33%, and 24% in the resection only group, respectively.

S020 ABILITY OF TNM STAGING TO PREDICT OVERALL SURVIVAL AFTER RESECTION OF PANCREATIC ADENOCARCINOMA IN PATIENTS UNDERGOING NEOADJUVANT CHEMOTHERAPY

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Introduction: Surgical resection offers the best chance for cure for pancreatic adenocarcinoma (PDAC), but only a minority of patients are resectable at diagnosis. Neoadjuvant chemotherapy improve survival through selection of favorable biology and/or control of micrometastatic disease, but it is unknown whether tumor stage by pathology after these therapies accurately reflects a patient’s survival. We assessed the ability of TNM staging to predict overall survival after PDAC resection in patients undergoing neoadjuvant chemotherapy.

Methods: Data were retrospectively collected on all patients undergoing resection of PDAC at a tertiary hospital from January 2010 to January 2015. Differences between patient cohorts were assessed by Student’s T-test and Fisher’s exact test. Overall survival (OS) was assessed by the Kaplan Meier method and univariate cox models.

Results: During the study period, 569 patients underwent PDAC resection. Neoadjuvant chemotherapy was administered to 131 patients (23%) for locally advanced or borderline tumors while 438 patients (77%) had resectable disease and went immediately to surgery. By histopathology, patients who did not receive neoadjuvant chemotherapy were more likely to have larger tumors (mean 2.7 vs. 3.2 cm, P<0.01), positive lymph nodes (44% vs. 73%, P<0.01), and higher T- and overall stage (P<0.01). However, there was no difference in rates of adjuvant chemotherapy (73% vs. 68%, P=0.33). Despite these key differences, median OS was decreased for neoadjuvant patients compared to those undergoing immediate
resection for stage 1 (18.0 vs. 36.6 months (mos.), P<0.01) and stage 2 (16.5 vs. 18.4 mos., P=0.04) disease but not stage 3 (15.1 vs. 6.4 mos., P=0.20). Survival for all stages combined was also decreased for neoadjuvant patients compared to immediate resection (16.8 vs. 19.5 mos., P=0.02) (Figure). Based upon tumor size, median OS was decreased for neoadjuvant chemotherapy patients for T1 (20.9 vs. 34.3 mos., P=0.05) and T2 tumors (16.1 vs. 26.2 mos., P=0.04), but not for T3 (16.5 vs. 16.7 mos., P=0.09) or T4 tumors (5.6 vs. 6.4 mos., P=0.34). Median OS was decreased for patients undergoing neoadjuvant chemotherapy based on N0 (18.0 vs. 30.1 mos., P<0.01) and N1 stage (13.0 vs. 17.5 mos., P=0.05).

**Conclusion:** For patients undergoing neoadjuvant chemotherapy, survival is likely based upon stage at diagnosis and not at time of resection. Pathological stage at resection does not predict equivalent overall survival for patients with or without neoadjuvant chemotherapy. Clinicians should be aware that patients with low stage disease or negative nodes after neoadjuvant chemotherapy and resection should still receive adjuvant chemotherapy given potentially worse survival.

**Figure:** Kaplan-Meier Curve for Overall Survival for all Stages split by Neoadjuvant Chemotherapy

**5021** OVERALL SURVIVAL IS INCREASED AMONG STAGE III PANCREATIC ADENOCARCINOMA PATIENTS RECEIVING NEOADJUVANT CHEMOTHERAPY COMPARED TO SURGERY FIRST AND ADJUVANT CHEMOTHERAPY: AN INTENTION TO TREAT ANALYSIS OF THE NATIONAL CANCER DATABASE

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**Introduction:** Outcomes of neoadjuvant systemic therapy versus upfront surgery for clinical stage III pancreatic adenocarcinoma remains poorly defined. Our aim was to compare overall survival among clinical stage III pancreatic adenocarcinoma patients receiving neoadjuvant chemotherapy first versus surgery first and adjuvant chemotherapy with an intention to treat(ITT) analysis.

**Methods:** National Cancer Data Base(NCDB) was reviewed from 2002-2011 for patients with clinical stage III pancreatic adenocarcinoma of the head or body. Patients were categorized as neoadjuvant (with/without preoperative radiation and with/without...
adjuvant chemotherapy) or surgery first (with adjuvant chemotherapy). Clinical Stage III patients without neoadjuvant systemic chemotherapy or curative intent pancreatectomy as first course of therapy were not included. ITT was performed by including all neoadjuvant therapy patients who were intended to receive curative intent surgery and all curative resection first patients who were intended to receive adjuvant therapy, regardless of receipt. ITT overall survival was compared by Kaplan-Meier and Cox Proportional Hazards multivariable regression. Survival was also assessed among those that did not progress (Pathological Stage III) versus those who down-staged (Pathological Stage 0, I or II) to neoadjuvant therapy.

Results: 593 patients were identified, 377(63.6%) neoadjuvant (of which 104(27.6%) experienced pre-surgical attrition) and 216(36.4%) surgery first (of which 30(13.9%) did not receive intended adjuvant chemotherapy). Neoadjuvant patients were pathological stage O 3(1.3%), I 58(25.9%), II 111(49.6%), III 51(22.8%) and IV 1(0.4%) vs the surgery first cohort O 0(0%), I 2(1.2%), II 56(34.6%), III 99(60.5%) and IV 6(3.7%), respectively. ITT Kaplan-Meier analysis demonstrated significantly superior 3 year survival in the neoadjuvant cohort compared to the surgery first cohort (median survival 20.7 months vs. 13.7 months). Figure Multivariable adjusted survival analysis revealed a hazard ratio of 0.68 (95% CI 0.53-0.86, p=0.0012) for neoadjuvant compared to surgery first.

Neoadjuvant patients were more likely R0 status (79.2% vs 53.3%,p<0.0001) and node negative (63.6% vs 25.1%,p<0.0001). R0 neoadjuvant patients have improved survival compared to surgery first patients (median 23.5 months vs. 16.3 months, p=0.0114). Node negative neoadjuvant patients have improved survival compared to surgery first patients (median survival 22.6 months vs. 14.2 months, p=0.004). Among neoadjuvant patients the use of multiagent (vs single agent) chemotherapy was associated with improved survival (median survival 21.9 months vs. 19.7 months, p=0.0241). The addition of postsurgical adjuvant chemotherapy to neoadjuvant patients was associated with improved survival (median survival 31.6 months vs 22.6 months, p=0.0273). Among the neoadjuvant cohort who proceeded to surgery, patients down-staged 76.8% (n=172) compared to 22.8% (n=51) of those who did not but had no difference in survival (p=0.0959).

Conclusion: Despite pre-surgical attrition, neoadjuvant therapy in clinical stage III pancreatic adenocarcinoma patients is associated with improved overall survival when compared to those who received curative intent surgery first and planned adjuvant chemotherapy. Node negativity and R0 rates are increased among neoadjuvant chemotherapy patients. RO and node negative neoadjuvant patients have improved survival compared to surgery first. Among neoadjuvant patients multiagent therapy and additional postsurgical therapy are both associated with improved survival while down-staging was not.

S022 CARBOHYDRATE ANTIGEN 19-9 IN ANATOMICALLY RESECTABLE, EARLY STAGE PANCREATIC CANCER IS INDEPENDENTLY ASSOCIATED WITH DECREASED OVERALL SURVIVAL AND AN INDICATION FOR NEOADJUVANT THERAPY: A NATIONAL CANCER DATABASE STUDY. 

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Background: Patient triage in anatomically resectable, early stage pancreatic ductal adenocarcinoma (PDAC) with elevated Carbohydrate Antigen 19-9 (CA 19-9) remains
unclear. We hypothesized that any CA 19-9 elevation above normal indicates biologically borderline resectability.

**Study Design:** The National Cancer Data Base (2009-2011) was reviewed for PDAC patients with measured CA 19-9. Non-secretors were analyzed separately. Stage I patients were stratified by CA 19-9 above/below normal (37 U/mL). Unadjusted Kaplan-Meier and multivariable Cox proportional hazards survival modeling were performed.

**Results:** 18,266 patients with measured CA 19-9 were identified. Among Stage I patients (N=1242), 133 (11%) were non-secretors, 327 (26%) had normal levels, and 782 (63%) had elevated levels. Demographics and peri-operative outcomes were otherwise similar between groups. Stage-specific survival was better in all stages for non-secretors compared secretors as a whole but similar to those with normal levels. Stage I patients with elevated CA 19-9 had decreased overall survival at 1, 2, and 3 years (47% vs. 65%, 27% vs. 47%, 18% vs. 34%, all p < 0.001) relative to normal level patients. Elevated CA 19-9 independently conferred increased mortality hazard (HR 1.47, p<0.001). Repeat modeling in neoadjuvant cohort demonstrated complete abrogation of this increased mortality hazard (p=0.72).

**Conclusions:** CA 19-9 non-secretors and those with normal levels achieve equivalent survival. CA 19-9 elevation independently predicts increased mortality hazard in Stage I patients. Neoadjuvant systemic therapy followed by curative-intent surgery best mitigates this mortality hazard among all potential treatment sequences. PDAC patients with any CA 19-9 levels above normal despite anatomic resectability are biologically borderline resectable and neoadjuvant systemic therapy is suggested.
S023 PATTERN OF CA19-9 RESPONSE TO NEOADJUVANT CHEMOTHERAPY IN LOCALLY ADVANCED, BORDERLINE RESECTABLE PANCREATIC CANCER PREDICTS PROGRESSION

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Introduction: As neoadjuvant therapy of locally advanced, borderline resectable pancreatic cancer (BRPC) is becoming more widely utilized; better indicators of progression are needed to help guide therapeutic decisions. The aim of this study is to determine if CA19-9 response during treatment predicts disease progression.

Methods: A retrospective review was performed on all patients with BRPC (by AHPBA/SSO consensus criteria) between 2008-2015 who received 24 weeks of neoadjuvant gemcitabine and docetaxel. Patients with medical comorbidities limiting treatment completion were excluded. Serum CA19-9 levels were checked at baseline and every 3 weeks while on therapy. A normal CA19-9 level was defined as < 37.5 units/mL and levels with concomitant biliary obstruction were censored. CA19-9 response was analyzed as a predictor of disease progression, recurrence, and survival.

Results: Eighty patients were included with a mean of 11 CA19-9 levels checked per patient during treatment. Thirty-two (40%) progressed on treatment (18 local and 14 distant) and 48 (60%) were resected (79% R0). CA19-9 responses were categorized into 5 groups (fig 1): 1) Always normal [n=13]; 2) Increasing [n=3]; 3) Slow decline [n=7]; 4) Rapid decline with plateau [n=41]; and 5) Rapid decline with late rise [n=16]. Univariate logistic regression analysis found that a final CA19-9 decline >50% of baseline (OR 0.06, p=<.0001), a normal final CA19-9 (OR 0.08, p=<.0001), pattern group 1 (OR 0.16, p=.0001), and group 4 (OR 0.10, p=.0001) were predictive of non-progression. Baseline or maximum CA19-9 levels were not predictive of progression. All patients in group 2 progressed; none were resected. Patients in pattern group 5 that underwent resection had an increased risk of recurrence (HR 12.5, p=.0005). Median overall survival for groups 1-5 were 20.4, 9.3, 20.8, 31.4, and 16.4 months respectively.

Conclusion: Patients with measurable CA19-9 levels who do not have rapid decline with sustained low or normal levels should be considered high risk for progression or recurrence and alternative treatment strategies should be entertained prior to curative resection.

Figure 1: Representative graphs of each pattern group. Always normal (Group 1 not shown), Increasing (Group 2), Slow decline (Group 3), Rapid decline with plateau (Group 4), and Rapid decline with late rise (Group 5).
Oral Abstracts

ORAL ABSTRACTS

S024 NEOADJUVANT TREATMENT WITH FOLFIRINOX FOR RESECTABLE AND BORDERLINE RESECTABLE PANCREATIC DUCTAL ADENOCARCINOMA: FEASIBILITY AND CLINICOPATHOLOGICAL IMPLICATIONS

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Introduction/Background: Neoadjuvant therapy has been reported to increase the rate of negative resection margins following resection for locally advanced and borderline resectable pancreatic ductal adenocarcinoma (PDAC). On the basis of the ACCORD trial, FOLFIRINOX is the rational choice for a neoadjuvant therapeutic agent. We have recently adopted a neoadjuvant approach for all patients with cytologically proven PDAC that have resectable and borderline resectable disease. In this study we report our initial experience using induction FOLFIRINOX focussing on tumour response, resection rates along with outcomes.

Methods: From August 2012 until September 2015 all patients diagnosed with PDAC were discussed at the regional West of Scotland Pancreatic Unit multidisciplinary meeting. Patients with CT scan visible disease that were considered resectable or borderline resectable according to Endoscopic Ultrasound and MRI imaging and considered objectively fit for resection according to cardiopulmonary exercise testing assessment were included. All study patients were prospectively followed-up. Response and resectability following induction chemotherapy (FOLFIRINOX or Gemcitabine/Capecitabine) along with clinicopathological outcomes were reported. Results were compared to 51 patients undergoing upfront resection for PDAC during the prior 36 month period (June 2009 to July 2012).

Results: 92 patients (41 female: 51 male) with potentially resectable cytology proven PDAC were identified. The median age was 65 years (range 39 – 79), and the median follow-up period was 13 months (range 3.4-35.6 months). The patients received a median of six cycles of Induction FOLFIRINOX (median 1-12). Episodes of grade III or IV toxicity were unusual. At initial restaging 18 patients (19.6%) had evidence of metastatic disease. 33 patients (35.9%) had evidence of locally progressive disease while 41 patients (44.6%) did not progress and underwent surgical exploration of which 3 (3.2%) patients had metastatic disease evident and underwent palliative bypass. 38 (41.3%) successfully underwent surgical resection. Of these 19 (50%) had chemoradiation prior to resection. A partial radiographic response was evident in 14 patients (36.8%). For the 92 patient cohort the median overall survival survival was 20.1 months, while for the group that progressed following treatment with FOLFIRINOX overall survival was 12.8 months and 28.9 months for the group that did not progress and underwent resection. On final pathological assessment, 5 patients treated with induction chemotherapy had a complete pathological response. Furthermore, there was a significant decrease (P<0.001) in the frequency of T3 stage tumours (63% vs 98%), lymph node positivity (44% vs 87%) and resection margin involvement (<1mm) (41% vs 80%) when neoadjuvantly treated cohort were compared.
to the 51 patients undergoing primary resection during the prior 36 month period. Notably, radiological response failed to correlate with partial or complete pathological response following resection.

**Discussion/Conclusion:** We have provided evidence that the use of induction FOLFIRINOX in patients with resectable or borderline resectable PDAC is safe, well tolerated, and achieved a complete pathological response in 13% of patients who underwent resection. However, further evaluation will be required to determine whether the improvement in traditional pathological prognostic features will translate into a prolonged long-term overall survival.

**S025 DOWNSTAGING OF LIVER METASTASES FROM PANCREATIC CANCER FOLLOWING PRIMARY CHEMOTHERAPY: IS SURGICAL RESECTION WORTHWHILE?**
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**Introduction/Background:** New chemotherapeutic regimens have improved survival for stage IV pancreatic ductal adenocarcinoma (PDAC) and occasionally downstaging of liver metastases can be observed. Aim of this work is to analyze the outcomes of patients undergoing primary chemotherapy for liver metastases from PDAC and to evaluate the results of surgical resection.

**Methods:** Retrospective analysis of patients with liver metastases from PDAC who underwent primary chemotherapy. Exclusion criteria were: patients with extra-hepatic metastases, patients with ECOG ≥ 3, patients undergoing supportive care alone.

**Results:** 127 patients (76 males, 51 females, median age 65 years) were identified. Liver metastases were unilobar in 28.5% of patients and only 10% had a single liver lesion. Chemotherapy regimens included gemcitabine alone or in association with other agents (44%) and multi-agents chemotherapy with FOLFIRINOX (8%) or PEXG/PDFG/PEFG (48%). 56 patients (44%) had a complete (7%) or partial response (37%). Median CA 19.9 values at diagnosis and at re-staging were 1483 and 63 U/mL in patients with partial/complete response (P=0.036). Compared with CA 19.9 values at diagnosis, 55 patients (43.5%) had CA19.9 reduction < 50%, 35 patients (27.5%) had reduction of 50-89%, and 37 (29%) had reduction ≥ 90% (major biochemical response). Surgical resection was carried out in 11 patients (8.5%) after a median of 12 months from initial diagnosis. 77% of these patients underwent multi-agents chemotherapy. Median disease-specific survival (DSS) was 11 months for the entire cohort and 15 months for those with partial/complete response. In this sub-group median DSS was significantly longer (39 versus 11 months) for patients undergoing resection (P<0.0001). Independent predictors of DSS were chemotherapy with multiple agents (HR: 0.512), surgical resection (HR: 0.360), > 5 metastases at diagnosis (HR: 3.515) and CA 19.9 reduction < 50% of baseline value (HR: 2.708).

**Conclusions:** Although complete response is a rare, partial radiological response can be accomplished in 4 out of 10 patients with liver metastases from PDAC mainly due to chemotherapeutic regimens with multiple agents. Surgical resection can be considered in selected cases and it is associated with improved survival.
**S026 PROGNOSTIC RELEVANCE OF THE TIMING OF INITIATING AND THE COMPLETION OF ADJUVANT THERAPY IN PATIENTS WITH RESECTED PANCREATIC DUCTAL ADENOCARCINOMA**

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**Background:** Although the role of adjuvant therapy in patients with resected pancreatic ductal adenocarcinoma (PDAC) is well established, its optimal timing and duration is still controversial. In this study, we evaluated the effects of the time of initiating and the completion of adjuvant therapy on the oncological outcomes of patients with resected PDAC.

**Methods:** The study population included 311 patients with PDAC who underwent potentially curative resection followed by adjuvant therapy in two tertiary university hospitals. We respectively analysed survival data according to the time of initiation and the completion of adjuvant therapy after pancreatectomy.

**Results:** There were no significant differences in the 5-year overall survival (OS) (32.8% versus 35.4%, P = 0.539) and disease-free survival (DFS) rates (26.2% versus 23.3%, P = 0.865) between patients with early (≤ 6 weeks after surgery) and late (> 6 weeks after surgery) initiation of adjuvant therapy. However, the 5-year OS (42.6% versus 22.2%, P < 0.001) and disease-free survival (29.2% versus 18.4%, P = 0.042) rates were significantly greater in patients with complete (≥ 6 cycles of chemotherapy) adjuvant therapy than in patients with incomplete (< 6 cycles) adjuvant therapy. Multivariable analysis revealed that incomplete adjuvant therapy was an independent prognostic factor for decreased OS (P = 0.001; odds ratio 1.811; 95% confidence interval 1.257–2.609).

**Conclusion:** The results show that completion of adjuvant therapy is a more important prognostic factor in terms of OS than early initiation of adjuvant therapy for improving the survival of patients with resected PDAC.

**S027 ADJUVANT RADIOTHERAPY DOES NOT IMPROVE OUTCOMES FOLLOWING PANCREATICODUODENECTOMY FOR PANCREATIC ADENOCARCINOMA: A MARGIN-STRATIFIED ANALYSIS**

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**Introduction:** The role of radiotherapy (RT) following pancreaticoduodenectomy (PD) for pancreatic adenocarcinoma (PDA) remains controversial due to ambiguity in the definition of R0/R1 margin status in existing clinical trials. Recent data suggest that increased margin clearance (MC) is associated with improved survival after PD for PDA, however the role of adjuvant radiotherapy (ADRT) in patients with known MC is undefined. We sought to analyze the influence of ADRT on outcomes of PD for PDA based on MC data.

**Methods:** We retrospectively identified 326 patients with MC data (in mm) who underwent PD between 2002-2014. Recurrence-free (RFS) and overall survival (OS) was determined by Kaplan-Meier analysis. Hazard ratios (HR) were calculated by Cox multivariate regression analysis on significant variables.
**Results:** Mean age was 68 yrs and 55% were male. Median follow-up was 21 mos (IQR 12-34 mos). ADRT was administered to 87 patients (27%). Median RFS and OS for the entire cohort was 14 mos and 25 mos. On univariate analysis, ADRT was not associated with improved median RFS (13 vs. 14 mos; p=NS) or OS (23 vs. 27 mos; p=NS), but increasing MC was associated with prolonged median RFS [10 (0mm) vs. 13 (0-1mm) vs. 23 mos (>1mm); p<0.02 for all pairs] and OS [16 (0mm) vs. 23 (0-1mm) vs. 40 mos (>1mm); p<0.01 for all pairs]. After controlling for sex, BMI, neoadjuvant therapy, LVI, PNI, lymph node ratio>0.2, tumor size>2.5cm, and adjuvant chemotherapy, increasing MC was independently associated with improved OS [HR 0.680; p=0.034 (0-1mm); HR 0.451; p<0.001 (>1mm), compared to 0mm]. Patients were subsequently stratified into 3 groups based on MC [0mm (n=73); 0-1mm (n=118); >1mm (n=135)]. ADRT was administered less frequently to patients with greater MC [0mm (n=29; 41%); 0-1mm (n=36; 31%); >1mm (n=22; 16%); p<0.001]. Even when stratified by MC, ADRT was not associated with improved RFS [10 vs. 9 mos (0mm); 13 vs. 12 mos (0-1mm); 21 vs. 23 mos (>1mm); p=NS for all pairs] or OS [16 vs. 18 mos (0mm); 24 vs. 23 mos (0-1mm); 33 vs. 42 mos (>1mm); p=NS for all pairs].

**Conclusions:** Adjuvant radiotherapy is not associated with improved recurrence-free or overall survival following PD for PDA regardless of margin clearance. The use of radiotherapy following PD for PDA should be re-examined.

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**S028 EXTERNAL RADIATION IS ASSOCIATED WITH IMPROVED SURVIVAL IN RESECTED MARGIN-NEGATIVE STAGE IIB PANCREATIC ADENOCARCINOMA**

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**Background:** The absolute benefit of adjuvant external beam radiation following a margin negative resection in early stage pancreatic cancer has not been determined.

**Methods:** We queried the National Cancer Data Base for patients with pathologic stage I-II pancreatic adenocarcinoma who underwent surgical resection between 2004 and 2012. Multivariate Cox-regression modeling was used to analyze stage-specific survival.

**Results:** 21,724 patients with stage I-II pancreatic adenocarcinoma underwent surgical resection during the period studied. 908 (4.3%) were pathologic stage IA, 1,720 (8.2%) stage IB, 4,278 (20.5%) stage IIA, and 14,003 (67.0%) stage IIB. 5437 (25.6%) patients were treated with adjuvant chemotherapy, 8,107 (38.2%) with adjuvant chemoradiation (chemoRT), and 7,696 (36.2%) with no adjuvant therapy. ChemoRT utilization increased with increasing stage (24.6% in stage IA vs 40.7% in stage IIB, p<0.001). ChemoRT was more common at community centers (51.9% vs 34.0% at academic centers, p<0.001), low volume centers (40.9% vs 33.1% at high volume centers, p<0.001), and in patients with private insurance (46.4% vs 31.7% in patients with Medicare, p<0.001).

Multivariate Cox-regression adjusted for age, race, comorbidities, facility type, location, and volume, type of pancreatcetomy, and grade was used to estimate stage-specific survival. Treatment at a high volume center was associated with decreased mortality (HR 0.78-0.89, p<0.04) across all stages. Age ≥70 (HR 1.16-1.37, p<0.01) and higher grade (HR
1.66-2.47, p<0.01) were associated with higher risk of mortality at all stages. ChemoRT was associated with a benefit in median overall survival over chemotherapy alone for stage IIB disease (20.2 vs 18.2 months, p<0.001). ChemoRT was not associated with a significant benefit in median overall survival for stage IA, IB, or IIA disease (p>0.30).

Conclusions: Addition of radiation to adjuvant chemotherapy after margin negative resection of pancreatic adenocarcinoma is associated with a survival benefit in patients with pathologic stage IIB disease and should be considered as adjuvant therapy in this patient group.

SO29 EXTENDED LONG-COURSE INDUCTION SYSTEMIC CHEMOTHERAPY, CONSOLIDATIVE CHEMORADIATION, AND AGGRESSIVE RESECTION OF "AT-RISK" ANATOMY IS ASSOCIATED WITH SIGNIFICANT SURVIVAL BENEFIT IN STAGE III (BR/ LA) PANCREATIC ADENOCARCINOMA: THE MAYO CLINIC EXPERIENCE. Mark J Truty, MD, MSc, Rory L Smoot, MD, David M Nagorney, MD, Michael L Kendrick, MD, Michael B Farnell, MD; Mayo Clinic College of Medicine, Rochester, US

Objective: Optimal preoperative sequencing for Stage III pancreatic adenocarcinoma (PDAC) is unknown. This study reviews outcomes of surgeon-directed preoperative sequencing strategy and curative-intent resection to identify factors predictive of optimal oncologic outcomes.

Methods: Retrospective review of consecutively resected Stage III PDAC after induction systemic chemotherapy/consolidative chemoradiation. Resectability classifications per Alliance consensus criteria (BR/LA). Pathologic response per CAP grading system (0-3). Patients underwent resection of “at-risk” anatomy with en-bloc vascular/multivisceral resections as necessary to accomplish R0. Outcomes were assessed.

Results: 57 patients comprised the cohort with BR/LA anatomy at diagnosis in 28/29(49%/51%) pts. respectively. Median no. induction cycles was 7(2-21) with 43(75%) completing >4 cycles of FOLFIRINOX (89%) or Gemcitabine/Nab-Paclitaxel (11%). All patients completed chemoradiation (50.4Gy). 45 pts. had CA19-9 elevation (median 353), 98% had biomarker response (median 10-fold), and 70% had normalization. Only 40% had objective CT/MR response and <10% had BR/LA classification downstaging. Pancreatic resections included: 36(63%) pancreaticoduodenectomies, 13(23%) subtotal distal pancreatectomies, and 8(14%) total. En-bloc vascular resection/reconstruction performed in 43(77%) patients with 39(70%) venous resections (PV/SMV), 22(39%) arterial resections (celiac/HA/SMA), and 18(32%) combined. 11/(20%) required en-bloc multivisceral resections. Median operative time was 490 min with 23% >10 hrs. Median EBL was 700cc with 35% >1000cc. Median LOS was 10 days with 23% >14 days. There were 3(6%) perioperative deaths (90-day). 55(96%) pts. acheived R0 resection. On pathologic evaluation 9(16%) had grade 0 (complete pathologic response), 17(30%) grade 1 (minimum residual viable tumor), 22(38%) grade 2 (moderate response), and 9(16%) grade 3 (minimum treatment effect). Radiographic response did not correlate with pathologic response. There was trend in grade 0/1 pathologic responses with >4 induction cycles (p=0.06). Median no. of LN’s was 18(3-48) and 6(11%) pts were LN+ on final pathology despite 13(23%) having biopsy-proven nodes pretherapy. At last follow-up (within 60 days) 42(74%) pts were alive with 37(88%) and 5(12%) pts without/with recurrent disease respectively. Median overall/recurrence-free survival for the cohort was 40.7/32.4 months respectively. Factors predictive of improved survival were >4
induction chemotherapy cycles (p=0.02), Grade 0/1 pathologic response (p=0.03), and normalization of CA19-9 (p=0.02). Extent of resection (arterial/multivisceral) was not associated with worse survival outcomes.

Conclusions: Our results support the combination of long-course systemic induction chemotherapy and chemoradiation prior to resection in highly selected patients with Stage III PDAC. Radiographic response and anatomical downstaging is uncommon and rare respectively, however this does not correlate with pathologic response or preclude an R0 resection with the utilization of aggressive resection of all “at risk” anatomy. Extended longer-course induction chemotherapy (>4 cycles), normalization of CA19-9, and pathologic response are predictive of excellent longterm oncologic outcomes. These results merit further validation.

S030 TUMOR ESTABLISHMENT AND RATE OF GROWTH IN PATIENT-DERIVED PANCREATIC DUCTAL ADENOCARCINOMA XENOGRAFT MODELS ARE ASSOCIATED WITH ADVERSE CLINICOPATHOLOGICAL FEATURES AND POOR SURVIVAL/OUTCOMES
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Introduction: Patient-derived xenograft tumors are powerful tools to study the biology of cancer. However, only a subpopulation of cancers grows in mice. The factors influencing the success of a tumor implant in pancreatic cancer patient-derived xenograft models are not well known. The aims of this study were to identify the clinicopathological features of human pancreatic ductal adenocarcinoma (PDAC) associated with the growth of xenograft tumors, and to evaluate the correlation between successful xenograft formation and survival outcomes of patients.
**Methods:** Primary or metastatic PDAC were subcutaneously implanted into nu/nu mice. Xenograft tumors were harvested and reimplanted into new mice once they reached 1 cm in diameter. Failed xenograft engraftment was defined as the absence of a palpable tumor after six months. Demographic, clinicopathological, and survival characteristics of these patients were compared in relation to success and growth rate of the engraftments. Categorical variables were compared using a Chi square test or Fisher exact test, as appropriate; continuous variables were analyzed using a Wilcoxon rank-sum test. Survival analysis was performed using the Kaplan-Meier method and the log-rank test.

**Results:** Between April 2009 and July 2012, 135 PDACs were implanted into immunodeficient mice. Of those, 62 (46%) successfully formed xenografts while 73 (54%) did not grow. Patients in both groups showed similar demographic characteristics. Successful xenograft tumor growth was significantly associated with lymphovascular invasion (OR 2.83, 95% CI 1.22, 6.58; p = 0.011), and marginally correlated with lymph node metastasis (p = 0.091) and advanced tumor stage of the parental tumor (p = 0.061). There were no differences in tumor size, tumor grade, perineural invasion, or history of neoadjuvant therapy between tumors that grew and those that failed to form xenografts. Moreover, patients with tumors that successfully engrafted in mice had significantly shorter overall survival (OS) (median OS 14 vs. 24 months, log-rank p = 0.024). Among successful engraftments, the median time required to reach sufficient size for reimplantation was 147 days (range 39-329). 29 xenografts were reimplanted into new mice in less than 147 days and this was considered a rapid growth rate. These xenografts, compared with engraftments with slower growth, were significantly associated with male gender (OR 3.0, 95% CI 0.97, 9.33; p = 0.044), lymph node metastasis (OR 8.47, 95% CI 0.81, 88.58; p = 0.044), and poor OS of patients (median OS 12 vs. 18 months, log-rank p = 0.067).

**Conclusions:** Successful PDAC engraftment in mice and rapid xenograft growth are associated with adverse clinicopathological features of the original tumor and poor patient survival. The positive association between xenograft formation and the presence of lymphovascular invasion may reflect an underlying biological mechanism that allows these tumors to adapt and grow in a new environment.

S031 HYPERGLYCEMIA IMPACTS TUMOR BIOLOGY AND CHEMOTHERAPY RESPONSE IN PRE-CLINICAL CANCER MODELS AND PATIENTS WITH Pancreatic CANCER
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**Background:** Diabetes mellitus (DM) has been associated with pancreatic ductal adenocarcinoma (PDA), as both a risk factor and a presenting symptom. However, hyperglycemia has not been studied extensively as a driver for tumor progression, but may affect tumor growth due to enhanced nutrient availability in the tumor microenvironment. Moreover, enhanced tumor growth in the context of nutrient abundance may render PDA more susceptible to cytotoxic chemotherapy which targets dividing cells.

**Materials and Methods:** PDA cell lines were treated with chemotherapy in low (5 mM) and high (25 mM) glucose conditions, and interrogated using multiple biologic assays that assess cell viability and DNA synthesis. Additionally, we analyzed records from 730 consecutive patients with PDA resected at TJU between 2002 and 2014. A total of 470 patients were included in the study based on criteria related to glucose control. Patients
were categorized as having high glucose (HG) if they carried an existing diagnosis of DM (n=229, 49%) or had a pre-operative HgbA1C > 6.0% (n=106, 22%; total n=335, 72%). Patients without a diagnosis of diabetes and a HgbA1C levels ≤6.0% were included in the low glucose (LG) group (135= 29%). Histopathological features and disease free survival (DFS) were analyzed.

**Results:** MiaPaCa2 cells cultured in high glucose media were more sensitive to chemotherapy (IC50 to gemcitabine of 0.8 µM) than cells cultured in low glucose (3.7µM, p<0.00001). Cell cycle analysis revealed that PDA cells at low glucose had a diminished S-phase (decreased DNA synthesis) population, and an increased number of cells in G2-M (reduced cell cycling). Accordingly, a gene expression array study revealed that DNA synthesis enzymes were globally downregulated in cells cultured in low glucose.

An analysis of clinical data limited to patients with preoperative HgbA1C levels (N=349) showed that elevated HgbA1C directly correlated with increased tumor size, increased number of regional lymph node metastases, elevated preoperative serum CA19-9 and elevated preoperative serum CEA (P=0.04,P=0.00002,P=0.04,P=0.009, respectively). In the total cohort (N=470), those in the HG group had larger tumors than those in the LG group (>3 cm, OR 2.43, 95% CI 1.59-3.73). In patients with regional lymph node metastases (N=324), the HG group had increased perineural invasion (OR 2.95, 95% CI 1.20-7.21) and a higher rate of positive resection margins (OR 1.80, 95% CI 1.06-3.01). Despite these aggressive features, a multivariate Cox hazard-model paradoxically indicated that patients in the HG group had a more favorable outcome with gemcitabine treatment (see figure, DFS, HR=0.31, p=0.00006).

**Conclusions:** Pre-clinical studies demonstrated that PDA cells have increased proliferation and DNA synthesis under high glucose, with a paradoxically increased susceptibility to DNA targeting agents under high glucose conditions. Concordantly, clinical data reveal that patients with poor glucose control have larger more aggressive tumors, yet these tumors may actually be more sensitive to DNA targeting chemotherapy. This underappreciated concept has substantial implications for patients with PDA. If validated, glucose levels may represent a novel prognostic and predictive marker, and this line of research may even affect strategies of glucose control in patients undergoing chemotherapy.

![Cox Adjusted Survival Function](image)
**ORAL ABSTRACTS**

**S032 AN INDEL DNA SEQUENCE EMBEDDED IN THE WEE1 REGULATORY BINDING SITE CORRELATES WITH INCREASED CANCER RISK IN FAMILY MEMBERS OF PANCREATIC CANCER PATIENTS**

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**Background:** WEE1 is a mitotic kinase inhibitor that regulates cell cycle and DNA repair in pancreatic ductal adenocarcinoma (PDA) cells and other cancer types. Recently, we showed that WEE1 is post-transcriptionally upregulated by the RNA binding protein HuR upon treatment with DNA damaging agents. Detailed sequencing of the 56 base-pair HuR regulatory binding site within a non-coding region of the WEE1 gene revealed an INDEL (i.e., insertion of TT) in a T-enriched region containing 10 thymidine repeats (rs141541085). In this study, we aimed to evaluate the clinical relevance of this finding.

**Materials and Methods:** The WEE1 HuR binding site was sequenced in 117 pancreatic tumor samples from patients operated between 2006 and 2013. Sanger sequencing and Capillary Electrophoresis (CE) techniques were used to detect the presence of 10T (most common), 11T, or 12Ts. Inconclusive sequencing or cases in which only Sanger sequencing was available were excluded. Genotypes were considered germline (in light of the contribution from the stromal compartment), and for the purposes of the study, a wild type genotype referred to tumors with 10T/10T sequence. All other genotypes were classified in the INDEL group. Patient familial cancer history and histopathological features were recorded.

**Results:** A total of 99 pancreatic tumor patients were analyzed. Fifty-nine patients (59.6%) were wild-type (WT) homozygous (10T/10T), 3 patients (3%) were 10T/11T heterozygous, 39 patients (39.4%) were 10T/12T heterozygous and 6 patients (6.1%) were 12T/12T homozygous. Patients who harbored the INDEL displayed higher rates of microscopic pancreatitis (OR 2.4, P=0.042). Additionally, various familial cancer patterns (in first degree relatives) were associated with the presence of an INDEL in the index patient germline DNA, with the following odds ratios: colorectal cancer (OR 6.32, P=0.012), familial cluster-1 [colorectal, gastric, ovarian, endometrial] (OR=4.30, P=0.016), and familial cluster-2 [colorectal, gastric, ovarian, endometrial, Breast, Hepatobiliary, Pancreas, Endometrial, Ovary, Melanoma, Brain, Urinary] (OR=2.38, P=0.057). These findings did not change when adjusted to patients’ smoking status (see table) and in a stratification according to race and ethnicity. Transfection studies in PDA cell lines suggest that the presence of the INDEL in the regulatory region of the WEE1 mRNA transcript impaired the ability of HuR to regulate WEE1.

**Conclusions:** Analysis of this small cohort of patients with pancreatic cancer reveal an association with a polymorphism in the noncoding region of the WEE1 mRNA transcript, and familial cancer risk. In vitro studies indicate that INDELS in this region affect the stabilization of WEE1 by the regulatory protein, HuR. This germline abnormality may be detrimental to DNA repair, and increase long-term risk for pancreatitis and cancer in family members with the abnormal genotype. Additional studies in larger patient cohorts, along with linkage analysis in affected family members, are needed to validate these findings. Validation of these results could have important implications for genetic counseling and cancer screening for individuals harboring the INDEL.
Background: Pancreatic cancer (PC) is the fourth leading cause of cancer related death in the United States, with a 5-year survival rate of less than 5%. MicroRNAs have been identified as attractive targets for therapeutic intervention. The functional significance of lost microRNAs have been reported in several human malignancies, including PC. Therefore, restoring lost miRNA function can provide a potential therapeutic benefit. Prior work has identified microRNA-145 (miR-145) as a tumor suppressor miRNA in pancreatic cancer. The restoration of miR-145 downregulates a number of oncogenes including mucin MUC13 – a glycoprotein that is aberrantly expressed in PC, and efficiently inhibits tumor growth in mice. The main challenge for successful translation of microRNAs into clinical practice remains an effective in vivo delivery system. The focus of this study was to develop and assess the efficacy of a miRNA delivery method for PC treatment.

Methods: Magnetic nanoparticle (MNP) based nanoformulation of miR-145 (miR-145-MNPF) was developed for the intracellular delivery and sustained release of miR-145. The positively charged polyethyleneimine molecules were used to increase the loading efficiency of miR-145. MUC13 expressing PC cell lines (HPAF-II and Capan-1) were utilized. Following transient transfection of miR-145-MNPF, Western blotting and immunofluorescence techniques were used to investigate the effects of miR-145 restoration on a number of oncoproteins including MUC13. Additionally, functional studies of the effects of miR-145 restitution using miR-145-MNPF including cell proliferation, colony formation, cell migration, and cell invasion assays were analyzed.

Results: miR-145 expression was progressively suppressed over the course of development from PanIN I-III to late stage poorly differentiated PDAC. Treatment of cells with miR-145-MNPF led to efficient intracellular delivery and upregulation of...
miR-145 as observed through prussian blue staining and qRT-PCR respectively. miR-145 restitution resulted in significant downregulation of target oncogenes including MUC13, HER2, P-AKT and p53 as observed through Western blotting and immunofluorescence techniques. miR-145-MNPF inhibited cell proliferation, clonogenicity, migration, and invasion of PC cells. MNPF mediated restitution of miR-145 effectively sensitized PC cells to paclitaxel and TRAIL therapy.

**Conclusions:** 1) MNP based delivery systems can be efficiently used for microRNA replacement therapy in order to restore lost microRNAs in cancer. 2) miR-145-MNPF efficiently restores miR-145 in pancreatic cancer cells and inhibits growth and invasion of PC. 3) miR-145 restitution using miR-145-MNPF may offer a potential therapeutic strategy for pancreatic cancer treatment alone or in combination with other therapies.

**S034 A PERSONALIZED APPROACH TO ADJUVANT THERAPY: CYTOPLASMIC HUR STATUS PREDICTS DISEASE FREE SURVIVAL AFTER RESECTION FOR PANCREATIC DUCTAL ADENOCARCINOMA**

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**Objective:** Previous studies have validated the RNA binding protein, HuR, as a predictive marker for pancreatic ductal adenocarcinoma (PDA) in the adjuvant setting. These studies, however, were based on small cohorts of patients outside of a clinical trial, where patients received combination therapies. To better evaluate cytoplasmic HuR (cHuR) as a predictive marker, we examined tumor samples from an international, randomized trial (ESPAC-3) in which resected PDA patients received either gemcitabine [GEM] vs. 5-fluoruracil [5-FU] adjuvant monotherapy.

**Methods:** Tissue samples from 379 PDA patients enrolled in the ESPAC-3 trial were stained with an anti-HuR antibody. cHuR expression was dichotomized to high versus low histological scores.

**Results:** Median disease free survival (DFS; shown in figure) for patients treated with GEM with tumors exhibiting low cHuR was 12.9 months (95% CI=11.2-15.4), vs. 10.9 months (95% CI=7.5-14.2) for high cHuR. The median DFS for patients treated with 5-FU with tumors with low cHuR was 12.8 months (95% CI=10.6-14.6) vs. 20.1 months (95% CI=8.3-36.4) for high cHuR. Unadjusted Cox regression of DFS supports high cHuR as a predictive marker for patients receiving 5-FU treatment (p=0.012).

**Conclusion:** High cHuR is a positive predictive marker for patients who receive 5-FU adjuvant therapy. If validated, this personalized approach may improve outcomes after resection. Future studies will determine if cHuR is also predictive for FOLFIRINOX therapy in the adjuvant setting.
**S035 T-CELL INFILTRATE AS A SIMPLE TOOL TO PREDICT INTERMEDIATE TERM SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA** Sabrina C Sopha, MD¹, McKenzie E Bedra, MPH², Jennifer Emel, MA², Cherif N Boutros, MD²; ¹Department of Pathology, University of Maryland Baltimore Washington Medical Center, ²Department of Surgery, University of Maryland Baltimore Washington Medical Center, Glen Burnie, US

**Introduction:** Pancreatic ductal adenocarcinoma (PDAC) remains one of the most lethal carcinomas. There is a growing body of evidence of the role of immunology in cancer. In this study, we assessed the relationship of T-cell infiltrate with PDAC TNM stage and overall survival.

**Methods:** Under IRB approval, we accessed a prospectively maintained PDAC database and identified 51 cases with available tumor blocks and clinical follow-up. 5-µm sections were immunostained with CD3 and correlated with the H&E tumor sections. CD3+ tumor-infiltrating lymphocytes (TILs) were quantified in 5 hot spots (or maximum number of fields available) at 200X using image-capture assistance by a single blinded pathologist (SCS) and normalized to 10 HPF. Cancer registry data were accessed to analyze the overall pathologic stage at diagnosis, and overall survival. CD3+ T-cell quantification was graded as follows: Grade 0, < 10 cells/10HPF; Grade 1, < 1%, or 10-20 cells/10 HPF; Grade 2, 1-5%, or 21-50 cells/10 HPF; and Grade 3, > 5%, or > 50 cells/10 HPF (Table 1). Statistical analyses were performed using SPSS.

**Results:** 51 patients were identified (54.9% female), average age at diagnosis was 69±11.4 years. 86.3% of tissue samples were from primary tumor specimens and 13.7% from distant metastatic specimens. Frequencies of CD3+ TILs grades are listed in Table 1. CD3+ TILs inversely correlated with TNM stage at diagnosis shown in Table 2 Pearson correlation coefficient (-.637; p<0.01). CD3+ TILs grade III have better overall survival than grades 0-II (69.5% vs. 58.3% at 1 year and 47.2% vs. 33.3 at year 2; p=.036), overall median survival was 625 days.

**Conclusion:** T-cell infiltration in PDAC inversely correlates with TNM stage at diagnosis. T-cell infiltration can predict intermediate survival of patients with pancreatic adenocarcinoma. Further studies with a higher sample size are needed to validate results of overall survival and addition of T-cell subsets analysis.

### Table 1: Frequency of CD3+ TILs grades in PDAC

<table>
<thead>
<tr>
<th>Grade</th>
<th>Number of Cells/10HPF</th>
<th>Percentage Cells</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>&lt;10</td>
<td></td>
<td>2% (1/51)</td>
</tr>
<tr>
<td>1</td>
<td>10-20</td>
<td>&lt;1%</td>
<td>14% (7/51)</td>
</tr>
<tr>
<td>2</td>
<td>21-50</td>
<td>1-5%</td>
<td>12% (6/51)</td>
</tr>
<tr>
<td>3</td>
<td>&gt;50</td>
<td>&gt;5%</td>
<td>73% (37/51)</td>
</tr>
</tbody>
</table>

### Table 2: Correlation between CD3+ TILs and TNM Stage at Diagnosis (Pearson Correlation -.637)

<table>
<thead>
<tr>
<th>Stage I</th>
<th>Stage II</th>
<th>Stage III</th>
<th>Stage IV</th>
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<tbody>
<tr>
<td>Grade 0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Grade 1</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Grade 2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Grade 3</td>
<td>7</td>
<td>20</td>
<td>0</td>
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</table>
ANALYSIS OF 337 PATIENTS WITH SOLID PSEUDOPAPILLARY TUMORS OF THE PANCREAS - ROLE FOR SURGERY IN METASTATIC DISEASE

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Introduction: Current literature addressing the treatment of solid pseudopapillary tumors of the pancreas is based on case series and small single institutional reviews. Due to the rarity of these tumors, the natural history is not well defined. We aim to define predictive indicators of survival in a large series of patients.

Methods: The National Cancer Data Base (NCDB) was queried for patients diagnosed with solid pseudopapillary tumors of the pancreas between 1998 and 2011. Single predictor univariate analyses were performed on variables including demographics, tumor characteristics, and surgery outcomes. Multivariate Cox proportional hazards survival analysis was then completed with backward elimination.

Results: 337 patients were identified; 82% were female, median age was 39 years. 84% of the patients had no co-morbidities. Patients undergoing surgical resection (n = 296) had superior survival (p < 0.0001), as did patients treated at academic centers and those with private insurance (p = 0.009, p = 0.007 respectively). Sex, age, tumor size, presence of lymph node metastases, positive surgical margins and presence of distant metastases were not significant predictors of survival in multivariate analysis. Of 24 patients with distant metastases, seven patients were treated surgically and experienced longer survival (HR = 8.6 for no surgery group).

Conclusion: Solid pseudopapillary tumors of the pancreas are rare neoplasms with excellent overall survival. However, in a low number of patients they do metastasize. Patients with and without metastatic disease that underwent resection survived longer. Although retrospective, these data support consideration of resection, even in patients with metastatic disease.
**S037 PERIAMPULLARY CANCERS: HISTOPATHOLOGIC SUBTYPE IS A STRONGER DETERMINANT OF PATIENT SURVIVAL THAN ANATOMIC LOCATION** Jennifer L Williams, MD1, Carmen Chan, BS2, Paul A Toste, MD1, Irmina A Gawlas, MD1, Charles R Vasquez, MD2, Dharma B Sunjaya, MD2, Eric Swanson, MD3, Jamie Koo, MD3, O. Joe Hines, MD1, Howard A Reber, MD1, David W Dawson, MD, PhD3, Timothy R Donahue, MD1;
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**Background:** Patients with periampullary adenocarcinomas have highly variable survival. These cancers can be subdivided into pancreaticobiliary (PB)- or intestinal (IN)-types based on histopathologic criteria which may represent distinct diseases. The aims of this study were to identify factors predictive of survival in patients with periampullary cancers (PACs) and compare survival between those with IN- or PB-type cancers originating from the ampulla (A), common bile duct (CBD), or duodenum (D) to those with pancreatic ductal adenocarcinoma (PDAC).

**Methods:** Records from patients who underwent pancreaticoduodenectomy for adenocarcinoma from 1995–2014 at a single-institution were reviewed. Three pathologists separately evaluated histopathologic type.

**Results:** We identified 510 patients: 13 D, 110 A, 43 CBD, and 344 PDACs. Median overall survival (OS) was 61.1, 51.3, 36.3, and 31.1 months for patients with cancers of the D, A, CBD, or pancreas, respectively (P=0.035). Most D (61.5%) and A cancers (51.8%) were IN-type, and most CBD tumors were PB-type (86.0%). Those with IN-type D, A, or CBD adenocarcinomas had longer median OS survival than PB-type (71.7 vs 33.3 months, P=0.017) or PDAC (31.1 months, P=0.003). There was no difference between all non-PDAC PB-type and PDAC (P=0.658). On multivariate analysis, grade (hazard ratio (HR) 1.94, P=<0.001), histopathologic phenotype (HR 1.75, P=0.008), and nodal status (HR 1.37, P=0.020) were significant predictors of survival.

**Conclusions:** Histologic phenotype is a better predictor of survival in patients with PACs than tumor location. Those with PB-type D, A, or CBD adenocarcinomas have similar survival to those with PDAC.

**S038 PANCREATICODUODENECTOMY FOR METASTATIC PANCREATIC NEUROENDOCRINE TUMOR** Richelle T Williams, MD1, Nicholas Sich, MD2, Thomas Clancy, MD1, Jiping Wang, MD, PhD1, Christopher Pezzi, MD2, Stanley Ashley, MD1, Richard Swanson, MD1;
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**Background:** The optimal management of metastatic pancreatic neuroendocrine tumor (PNET) is controversial, with some advocating aggressive surgical resection and others a more conservative approach. The objective of this study was to define the role of pancreaticoduodenectomy (PD) in the treatment of patients with metastatic PNET utilizing a large, multi-institutional patient cohort.

**Methods:** We analyzed 4,667 patients diagnosed with metastatic PNET in the National Cancer Data Base from 1998 to 2012. Thirty- and 90-day mortality was calculated for patients undergoing PD compared to patients managed without resection of the primary tumor. Kaplan-Meier and Cox regression models were used to assess the effect of relevant clinicopathologic and treatment factors on survival.
**Results:** Approximately 1/5 of patients with metastatic PNET (970, 20.8%) underwent surgical resection of the primary tumor, with 351 (7.5%) undergoing PD and almost 1/2 of these (184, 52.4%) combined with resection of extrapancreatic disease (PD+). In the no primary resection (NPR) group, 185 (5%) had resection of extrapancreatic tumor (NPR+). Compared to NPR, PD patients were more likely to be younger (45% age <55 vs. 32.9%), privately insured (64.4% vs. 48.9%), treated in an academic medical center (71.2% vs. 47.2%), and less likely to have received chemotherapy (25.9% vs. 48.6%), p<0.001 for all. Thirty- and 90-day mortality rates, respectively, were 1.4% and 4.3% after PD and 7.6% and 19.2% for NPR. Median survival times in months were: NPR only 14.5, NPR+ 24.9, PD only 67.9, and PD+ 93.2. Overall 5- and 10-year survival rates, respectively, were 57% and 37% with PD, and 19% and 8% with NPR. On multivariable analysis, PD remained an independent predictor of improved survival (HR 0.41, 95% CI 0.33-0.50).

**Conclusion:** PD is associated with a significant long-term survival advantage and minimal postoperative mortality in patients with metastatic PNET. Resection of the primary and resection of extrapancreatic disease each improved survival but seemed to have an additive effect when combined. PD, preferably with resection of metastatic disease, should be considered in patients with metastatic PNET when possible.

**Table 1. Survival in Patients with Metastatic PNET Treated with Pancreaticoduodenectomy Versus No Resection of the Primary Tumor**

<table>
<thead>
<tr>
<th></th>
<th>NPR (n = 3687)</th>
<th>PD (n = 351)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number of patients (%)</strong></td>
<td>3502 (95%)</td>
<td>185 (5%)</td>
</tr>
<tr>
<td><strong>30-day Mortality (%)</strong></td>
<td>7.6</td>
<td>1.4</td>
</tr>
<tr>
<td><strong>90-day Mortality (%)</strong></td>
<td>19.2</td>
<td>4.3</td>
</tr>
<tr>
<td><strong>Median survival (months)</strong></td>
<td>14.5</td>
<td>24.9</td>
</tr>
<tr>
<td><strong>OS at 5 Years (%)</strong></td>
<td>19</td>
<td>57</td>
</tr>
<tr>
<td><strong>OS at 10 Years (%)</strong></td>
<td>8</td>
<td>37</td>
</tr>
</tbody>
</table>

NPR = No primary resection, NPR+ = No primary resection but resection of extrapancreatic disease, PD = Pancreaticoduodenectomy, PD+ = Pancreaticoduodenectomy with resection of extrapancreatic disease, OS = Overall survival

**S039 SURGICAL OUTCOMES OF RESECTED FUNCTIONAL PANCREATIC NEUROENDOCRINE TUMORS: A SINGLE INSTITUTION EXPERIENCE** Ammar A Jayed, MD, Fabio Bagante, MD, David O’Neill, BS, Kevin Soares, MD, Ralph H Hruban, MD, John L Cameron, MD, Matthew J Weiss, MD, Jin He, MD, PhD, Christopher L Wolfgang, MD, PhD; 1Johns Hopkins Hospital, 2University of Verona, Baltimore, US

**Introduction:** Functional pancreatic neuroendocrine tumors (f-PNETs) are rare tumors of the pancreas. Literature available on f-PNETs is limited. We here report a large single institute series of patients with f-PNETs. Having a large series allowed us to better analyze their clinicopathological characteristics.
Method: A prospectively maintained database on patients undergoing pancreatectomies was used to identify patients who underwent resection for f-PNETs at Johns Hopkins Hospital between 1995 and 2015. The clinicopathological information of these patients were collected and compared between different types of f-PNETs.

Results: The 69 patients who underwent pancreatectomy for f-PNETs were found to have tumors that secreted insulin (N=48, 69.6%), gastrin (N=10, 14.5%), VIP (N=7, 10.1%), glucagon (N=1, 1.5%), pancreatic polypeptide (N=1, 1.5%) and somatostatin (N=2, 2.9%). The median age of patients was 52.2 years (39.2-61.4) and a majority was females (N=43, 62.3%). A majority of patient underwent distal pancreatectomy (N=38, 55.1%), the most common postoperative complication being wound infection (N=23, 33.3%). Eight (11.6%) patients had an associated MEN1 syndrome, 1 (1.5%) a neurofibromatosis while 60 (86.9%) patients had non-syndromic tumors. The median size of the tumor was 1.9cm, a majority had no nodal disease (N=53, 76.8%) and negative margins (N=58, 85.3%). Sixty-one (88.4%) patients had well differentiated tumors and 8 (11.6%) were found to have metastatic disease. During a median follow-up of 32.3 months, 10 (14.5%) patients died as a result of f-PNETs while 3 (4.3%) died of other causes.

When types of f-PNETs were compared, VIPomas were found to be common in the elderly patients (age >65 years) (N=6, 85.7%) while other types were more common in patients with age<65 years (P=0.002). Not surprisingly, there was a wide range of presenting complaints depending on the type of f-PNETs. Insulinoma was associated with hypoglycemia (N=40, 83.3%), gastrinoma with epigastric pain, nausea, and emesis (N=2, 20%), while VIPoma with diarrhea (N=7, 100%). VIPomas were found to be the largest in size at the time of resection (median=6cm) and insulinomas were found to be the smallest (median=1.5cm) (P<0.001). VIPomas (N=7, 100%) and insulinomas (N=44, 61.7%) were predominantly unifocal while gastrinomas and other f-PNETs had an equal distribution of unifocal and multifocal disease. Patients with VIPomas had the highest rate of metastatic disease (N=4, 57.1%) A majority of f-PNETs were well-differentiated(N=67, 97.1%) and presented with a low Ki-67 of 0-5%(N=56, 81.2%). The survival curves demonstrated a better survival in patients with gastrinomas and insulinomas as compared to VIPomas, and glucogonoma (P=0.003). There was a trend towards better survival in patients with syndrome associated f-PNETs however this was not statistically significant (P=0.118).

Conclusion: F-PNETs have a wide range of clinicopathological characteristics based on their type. With appropriate surgical management the 5-year survival is high. Improved survival is associated with gastrinomas, and insulinomas.

S040 VARIATIONS OF ORAL AND FECAL MICROBIOTA ARE ASSOCIATED WITH AUTOIMMUNE PANCREATITIS Giulia M Cavestro, MD, PhD, Roberto Ferrarese, MD, Maria C Petrone, MD, Milena Di Leo, MD, Elisa R Ceresola, MD, Laura Visconti, Massimo Clementi, Pier Alberto Testoni, Filippo Canducci; 1UO Gastroenterologia, Universita Vita Salute San Raffele, IRCCS Ospedale San Raffaele, 2Laboratory of Microbiology, Ospedale San Raffaele, Milan, Milan, IT

An increasing number of studies, have demonstrated that in human inflammatory and autoimmune diseases such as irritable bowel diseases, diabetes, rheumatoid arthritis and multiple sclerosis, both a dysregulated immune response and an altered microbiota composition are present, suggesting a direct involvement of gut microbes in disease
pathogenesis. In this pilot prospective trial, we measured variations of both salivary and fecal microbiome composition in treatment-naive patients at their first diagnosis of autoimmune pancreatitis.

**Methods:** Stool and salivary samples were collected from 14 control subjects (7 males and 7 females) and 8 patients (6 males and 2 females) with autoimmune pancreatitis from the same geographical region. The average age was 50 years for control subjects, 56 years for patients. All patients were naive and sequentially enrolled at diagnosis. Biological samples were obtained before medical treatment. Exclusion criteria included antibiotic usage within the last month and other autoimmune diseases. Total DNA was extracted from each sample and purified, the V3-V5 region of the 16s rRNA gene was amplified and massive ultra-deep pyrosequencing was performed by 454-GS Junior. Sequences with high quality score and length >250bp were analyzed with QIIME (v1.6.0). Chimeric sequences were removed by ChimeraSlayer tool and good coverage index was evaluated for every sample.

**Results:** Comprehensive comparison of the salivary and fecal microbiota between patients with treatment naive autoimmune pancreatitis and healthy control subjects revealed a significant variation of salivary/fecal microflora. NGS analysis showed an increase of Proteobacteria phylum (p=0.008) in fecal samples of patients with autoimmune pancreatitis compared to control subjects: in particular, we observed an increase in Enterobacteriaceae family (p=0.005). Moreover in patients with autoimmune pancreatitis we found at genera level an increase of Lactobacillus (p=0.05) and Oscillospira (p=0.05) and a decrease of Faecalibacterium (p=0.01) compared to control subjects. In salivary samples, we observed a decrease of Haemophilus parainfluenzae (p=0.02) in patients with autoimmune pancreatitis but no significant differences at any other taxonomic level of analyses.

**Conclusions:** We observed a significant association between variations of patients’ salivary/fecal microbiome with autoimmune pancreatitis when compared with normal controls. Interestingly Proteobacteria that are known to increase in local and systemic inflammatory diseases or can be causative agents of immune proliferative diseases such as MALT lymphomas are also significantly increased in autoimmune pancreatitis patients. These results open the way to larger trials and to novel therapeutic approaches with the aim of re-establish a beneficial intestinal environment and microbiome composition in patients with autoimmune pancreatitis.

**S041 CLINICAL SIGNIFICANCE OF B CELL-ACTIVATING FACTOR IN AUTOIMMUNE PANCREATITIS** Teru Kumagi, MD, PhD, Hirofumi Yamanishi, Tomoyuki Yokota, Nobuaki Azemoto, Mitsuhito Koizumi, Taira Kuroda, Yoshinori Ohno, Morikazu Onji, Yoichi Hiasa; Ehime University Graduate School of Medicine, To-on, JP

**Objectives:** Overexpression of B Cell-activating factor (BAFF) is involved in autoimmunity, but little is known about its role in autoimmune pancreatitis (AIP). The aim of this study was to investigate the role of BAFF in the diagnosis and pathogenesis of AIP.

**Methods:** Patients with AIP (n = 19) were compared with 2 disease control groups (chronic pancreatitis [n = 17] and pancreatic cancer [n = 15]) and a healthy subject group (n = 19). Serum BAFF levels were assessed using an enzyme-linked immunosorbent
assay. The expressions of BAFF and BAFF receptor in the pancreatic tissue of patients with AIP were estimated using immunohistochemistry.

**Results:** Mean serum BAFF levels were higher in the patients with AIP than in the patients with chronic pancreatitis, the patients with pancreatic cancer, and the healthy subjects (P < 0.0001 for all groups). Using the cutoff value of 1389 pg/mL, the sensitivity and specificity to differentiate AIP from disease and healthy controls were 89.5% and 92.2%, respectively. Glucocorticoid therapy decreased serum BAFF levels below 1389 pg/mL in all patients with AIP (P < 0.0001). B Cell-activating factor and BAFF receptor were expressed on cells infiltrating the pancreas of patients with AIP.

**Conclusions:** B Cell-activating factor could be a novel marker for diagnosis and treatment response in AIP and may contribute to its pathogenesis.

**OUTCOMES AFTER SALVAGE TOTAL PANCREATECTOMY FOR REFRACTORY CHRONIC PANCREATITIS**

William P Lancaster, MD, Stefanie M Owczarski, PAC, David B Adams, MD, Katherine A Morgan, MD; Division of Gastrointestinal and Laparoscopic Surgery, Medical University of South Carolina, Charleston, South Carolina, Charleston, US

**Introduction:** Total pancreatectomy with islet cell auto-transplantation (TPIAT) is a treatment for refractory chronic pancreatitis. Many patients undergoing TPIAT have had previous pancreatic surgery. We sought to determine the long term outcomes of TPIAT as a salvage procedure.

**Methods:** Retrospective review of a prospectively maintained database identifying patients with previous pancreatic surgery who underwent TPIAT. Chi-square and Fisher’s exact test were used where appropriate.

**Results:** A total of 109 patients with previous pancreatic surgery and at least one year of follow-up were identified. Mean age was 41 years and the majority of patients were female (79%). The most common etiology of pancreatitis was sphincter of Oddi dysfunction (43%) followed by pancreas divisum (28%). The most common prior operation was Whipple (17%). There were no postoperative deaths. Preoperative physical and psychological quality of life scores were measured and compared with postoperative scores. Compared with preoperative assessment, physical quality of life improved at 1 year postoperative (28.4 vs. 35.3, p=0.001) and this effect persisted until 3 years postoperative (28.4 vs. 33.5, p=0.002). Psychological quality of life improved at 1 year postoperative (39.4 vs. 43.2, p=0.01) and this change was still seen at 5 years postoperative (39.4 vs. 44.8, p=0.05). The average daily insulin requirement at 1 year postoperative was 24.8 units/day and this was unchanged at 5 years postoperative (28.6 units/day, p=0.5).

**Conclusions:** TPIAT is an effective salvage operation for refractory chronic pancreatitis. It is safe and offers improved quality of life in patients with previous pancreatic surgery. This effect is durable and can persist for at least five years. Patients can expect to require insulin after salvage total pancreatectomy.
**S043 COMPARABLE RATE OF LONG TERM INSULIN INDEPENDENCE BETWEEN ADULT PATIENTS UNDERGOING REMOTE AND LOCAL TPIAT**

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**Objectives:** Total pancreatectomy with islet autotransplantation (TPIAT) is increasingly being performed remotely – i.e. removing the pancreas from the patient in one location, transporting the islets to and isolating them in another location, then returning the islets to the original location for re-implantation into the patient. However, the effect of the “remote” isolation on outcomes, especially long term insulin independence, is unknown. We compared the clinical outcomes of adult TPIAT patients who underwent “remote” isolation compared with those who underwent standard “local” isolation.

**Methods:** We evaluated adult patients who underwent remote TPIAT at three centers from 2010 – 2014; two centers (Cleveland Clinic Foundation and Dartmouth-Hitchcock Medical Center) performed “remote” isolation and one (University of Minnesota) performed “local” isolation. The primary clinic outcome was the percentage of patients not using insulin at one year.

**Results:** 33 patients underwent “remote” and 143 underwent “local” isolation. There was no difference between groups in terms of age, gender, BMI, type of pancreatitis, pre-operative fasting c-peptide or pre-operative morphine equivalents, although the “remote” group had a slightly higher baseline HgA1c (5.43 vs 5.25, p=0.02). There was no difference in the islet equivalents/kg, islet mass transplanted, maximum portal infusion pressures, or risk of portal vein thrombosis between groups. At one year 24% of “remote” and 34% of “local” patients were insulin independent (p=0.41). However, “remote” patients had a greater drop in fasting c-peptide level (-1.6 vs -0.6, p<0.01) and a greater rise in HgA1c (1.7 vs 1.1, p=0.04) at one year compared to “local” patients. One year narcotic use (44% vs 43%, p=1.00) and morphine equivalents (49 vs 65, p=0.48) was also similar between the “remote” and “local” groups. Having islet equivalents/kg >3,000 was the only characteristic associated with one year insulin independence (OR 12.4, 95% CI 3.6-42.7) in both groups.

**Conclusions:** Although patients undergoing TPIAT remotely have comparable rates of long term insulin independence compared with patients undergoing TPIAT locally, overall metabolic control is superior with local isolation.

**S044 VITAMIN AND IRON DEFICIENCIES ARE COMMON IN PATIENTS WHO UNDERGO TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION FOR CHRONIC PANCREATITIS**

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**Background:** Total pancreatectomy with islet autotransplantation (TPIAT) is a modern surgical option for patients with debilitating pain and poor quality of life (QOL) from chronic pancreatitis. All patients have exocrine pancreas exocrine insufficiency after TPIAT, and the incidence of iron and fat soluble vitamin deficiencies in this population is poorly defined. Symptoms associated with vitamin deficiencies include impaired wound healing, weakness, fatigue, irritability, depression, and confusion.
Methods: A prospectively collected database of 156 patients who underwent TPIAT over 6 years was retrospectively reviewed and analyzed. Patients with greater than 6 month post-operative data were included in the IRB approved study. Nutrition laboratory values were examined at pre-op, 6 months, and annually to 3 years post-op. Vitamin deficiencies were defined as Vitamin D <25 ng/ ml; Vitamin A <0.3 mg/L; Vitamin E (alpha tocopherol) <5.5 mg/L; Vitamin B12 <211 pg/mL; Ferritin: <20 ng/ml.

Results: The study group was comprised of 104 patients. Vitamin D was deficient in 37% of patients before surgery. After surgery, 52% were Vitamin D deficient at six months, 61% at one year, 64% at two years, and 69% at 3 years. Vitamin A was low in 5% of patients before surgery, 37% at six months after surgery, 28% at one year, 41% at two years, and 34% at three years after surgery. Vitamin E was low in 27% before surgery, 3% at six months after surgery, 6% at one year, 21% at two years, and 30% at three years after surgery. Twenty-one percent were low in Ferritin before surgery, 53% were low at six months and one year after surgery, 43% at two years, and 34% at three years after surgery. No patients were Vitamin B-12 deficient before or after surgery.

Conclusion: Iron and fat soluble vitamin deficiencies are common after TPIAT despite active monitoring and treatment with pancreas enzyme replacement therapy, IV iron, and prescription fat-soluble vitamins. Symptoms associated with these vitamin deficiencies can affect post-operative quality of life.

S045 IMPACT OF DISCONNECTED PANCREATIC DUCT SYNDROME (DPDS) ON ENDOSCOPIC TREATMENT OUTCOMES IN PANCREATIC FLUID COLLECTIONS (PFCs) Ji Young Bang, MD, MPH1, Muhammad Hasan, MD1, Udayakumar Navaneethan, MD1, Pablo Arnoletti, MD2, Robert Hawes1, Shajarn Peter, MD2, John Christein, MD4, C. Mel Wilcox, MD3, Shyam Varadarajulu, MD1; 1Center for Interventional Endoscopy, Florida Hospital, 2Department of Surgery, Florida Hospital, 3Division of Gastroenterology & Hepatology, University of Alabama at Birmingham, 4Department of Surgery, University of Alabama at Birmingham, Indianapolis, US

Background: Disconnected pancreatic duct syndrome (DPDS) encountered in severe pancreatitis is characterized by complete transection of the main pancreatic duct (MPD) resulting in a variable portion of the upstream pancreatic gland becoming isolated from the MPD downstream. The upstream gland secretes pancreatic juice that results in a non-resolving percutaneous fistula or pancreatic fluid collection (PFC). Data on the clinical outcomes after endoscopic management of PFCs in DPDS are limited.

Aim: Evaluate the frequency of occurrence and impact of DPDS on the endoscopic treatment outcomes of patients with PFCs.

Methods: This is a retrospective study of patients who underwent endoscopic transmural drainage of PFCs over a 12-year (2003-2015) period. Treatment consisted of endoscopic or EUS-guided transmural stent placement in all patients. If treatment response was suboptimal, additional therapy consisted of further drainage by EUS-guided multi-gate technique, percutaneous large-bore drain placement and/or endoscopic/sinus tract necrosectomy. The transmural stents were routinely removed after PFC resolution in all patients during the initial 5 years of this study. In the latter years of the study, the stents were left permanently in situ in patients recognized to have DPDS. Treatment success was defined as resolution of PFC and clinical symptoms.
without the need for rescue surgery. The primary outcome measures were: 1) the frequency of occurrence of DPDS and 2) endoscopic treatment outcomes.

**Results:** Of 354 patients who underwent endoscopic drainage of PFCs, 176 (49.7%) were pseudocysts, 144 (40.7%) WON and 34 (9.6%) acute collections. DPDS was diagnosed in 162 of 354 (45.8%) patients and was most frequent in WON patients compared to other PFC types (83.8% vs. 34.4%; p<0.001). DPDS was found more frequently in the neck/body-tail region compared to the head of pancreas (78.4 vs. 21.6%). While there was no significant difference in overall treatment success between patients with and without DPDS (84.6 vs. 91.8%; p=0.07), more patients with DPDS underwent endoscopic re-interventions (29.6 vs. 18.9%; p=0.04) that were complex (multi-gate and/or percutaneous drainage and/or necrosectomy: 30.2 vs. 4.9%; p<0.001) and required rescue surgery (13.0 vs. 4.9%; p=0.02). Additionally, the rate of PFC recurrence was significantly higher in DPDS patients without permanent indwelling transmural stents (17.4 vs. 1.7%; p=0.001).

**Conclusions:** DPDS is seen in nearly 50% of patients undergoing endoscopic transmural drainage of PFCs, particularly in cases with WON. A multidisciplinary approach is imperative for managing PFCs in the setting of DPDS due to the need for frequent re-interventions and rescue surgery. PFC recurrence rates in DPDS can be minimized by the placement of permanent indwelling transmural stents.

**S046 EUS-BASED STEP-UP TREATMENT APPROACH IS ASSOCIATED WITH BETTER SYSTEMIC INFLAMMATORY RESPONSE IN WALLED-OFF NECROSIS (WON)** Ji Young Bang, MD, MPH, Bronte Holt, MD, Udayakumar Navaneethan, MD, Muhammad Hasan, MD, Robert Hawes, MD, Shyam Varadarajulu, MD; Center for Inteventional Endoscopy, Florida Hospital, Indianapolis, US

**Background:** Patients with symptomatic walled-off necrosis (WON) require decompression and debridement by surgical or endoscopic techniques. Prior data have shown that the clinical outcomes of treatment are directly related to the resolution of systemic inflammatory response.

**Aim:** To compare the systemic inflammatory response following minimally-invasive surgery or EUS-based step-up treatment for WON.

**Methods:** Patients with infected or symptomatic WON unresponsive to conservative treatment were randomly allocated to minimally-invasive surgery or EUS-based treatment in this ongoing clinical trial (NCT02084537). The surgical arm involved laparoscopic cystogastrostomy with debridement or video-assisted retroperitoneal or percutaneous anterior abdominal debridement. The endoscopic arm consisted initially of EUS-guided transluminal drainage via single or multi-gate technique (for WON size ≥ 80mm) followed by step-up transgastric or percutaneous endoscopic necrosectomy at 72 hours if clinically and radiologically indicated. The outcome measure was resolution of preexisting systemic inflammatory response syndrome (SIRS) at 48 hours post-treatment. SIRS was assessed using clinical criteria developed by the American College of Chest Physicians and the Society of Critical Care Medicine.

**Results:** 34 patients were randomized; 16 to surgery and 18 to EUS-based step-up treatment. All patients were ASA class III or IV. There was no significant difference in patient demographics or disease characteristics between the two cohorts. Resolution
of pre-existing SIRS at 48 hours was encountered more frequently in the EUS-based step-up treatment cohort than surgery (60 vs. 0%; p=0.04). Also, the median number of SIRS criteria at 48 hours post-treatment was significantly lower for EUS compared with surgery (0.5 vs. 2; p=0.02).

**Conclusions:** In patients with infected or symptomatic WON, EUS-based step-up treatment is associated with early resolution of systemic inflammatory response, which is in turn a surrogate marker for better clinical outcomes.

**S047 TRANSGASTRIC NECROSECTOMY FOR THE MANAGEMENT OF WALLED-OFF PANCREATIC NECROSIS: LONG-TERM OUTCOMES AT A HIGH-VOLUME PANCREATIC CENTER** Andrea Jester, MD, Eugene Ceppa, MD, Attila Nakeeb, C. Max Schmidt, MD, PhD, Michael G House, Nicholas Zyromski, MD; Indiana University School of Medicine, Indianapolis, US

**Introduction:** Surgical transgastric cyst gastrostomy (TG CG) has emerged as a treatment for both sterile and infected walled-off pancreatic necrosis (WOPN). To date, there are no published data regarding the long-term outcomes of patients who have undergone TG CG for the management of WOPN. We sought to review the outcomes of surgical TC CG for the management of WOPN at a high-volume pancreas center.

**Method:** This is a retrospective review of patients who underwent surgical TC CG and necrosectomy for WOPN between 2008 and 2015.

**Results:** Fifty-three patients underwent surgical intervention. The median age was 53 years (range: 21-77 years). Five (9%) patients failed endoscopic management prior to surgical referral. Mean time to operative intervention was 86 days. Median post-operative length of stay was 4 days (range: 3-28 days). There was no 90-day mortality. Median follow up was 17 months (range: 1.5-72 months). Thirty-day morbidity was 7.5 %. Long-term follow up demonstrated that 4 patients (7.5 %) developed recurrent WOPN requiring endoscopic management. The median time to re-intervention was 12 months (range: 6-17 months). Eight patients (15 %) required re-operation. Two were within 30 days, one for ischemic colitis and the other for persistent infected WOPN. Re-operations included 2 distal pancreatectomy and splenectomy, 3 Whipples, and 1 roux-en-y pancreaticojejunostomy. Median time for re-operation was 17 months (range: 8-48 months). Other long-term sequelae included incisional hernia (8 %), GERD (10 %), pancreatic exocrine insufficiency (7 %) and chronic pancreatitis (10 %).

**Conclusions:** Transgastric cystgastrostomy is an effective treatment for walled-off pancreatic necrosis. However, there are long-term sequelae that present outside of the traditional surgical follow-up period, and these patients should be followed on a long-term basis.

**S048 NOVEL METHOD OF DIRECT ENDOSCOPIC NECROSECTOMY IN THE TREATMENT OF WALLED-OFF PANCREATIC NECROSIS** Anand Singla, MD, Adam Templeton, MD, Michael D Saunders, MD; University of Washington, Seattle, US

**Introduction/Background:** Walled-off pancreatic necrosis (WOPN) as sequelae of severe acute, necrotizing pancreatitis has been associated with high morbidity and mortality. The management traditionally has been surgical, though direct endoscopic necrosectomy has recently been studied and shown to be similarly effective and
potentially safer. Endoscopic necrosectomy is usually performed using a standard midsize upper endoscope with a 2.8mm working channel for irrigation and suction, and sometimes with a larger therapeutic endoscope with a 3.7mm working channel. With the ability to offer optimal irrigation and suction for debridement, the “jumbo-channel” gastroscope is an ideal instrument for direct endoscopic necrosectomy in patients requiring treatment for WOPN.

Methods: We present a retrospective case series of patients identified at our institution from 2010 to 2014 with WOPN treated with direct endoscopic necrosectomy using the “jumbo-channel” gastroscope (GIF-XTQ160, Olympus Corporation, Tokyo, Japan). This endoscope boasts an extra-large, 6mm working channel for debridement, with overall outer diameter of 12.9mm. Patient characteristics, details of original clinical presentation, radiologic findings, and procedural details / outcomes were recorded via chart review.

Results: A total of 11 patient records were identified and examined. Complete resolution of WOPN was achieved in 9 out of 11 patients, with the remaining 2 patients having significantly reduced necrotic collections and associated clinical improvement. The mean age of the patients was 46, with 6 out of 11 patients being male. The most common etiology of pancreatitis was gallstones (5), followed by hypertriglyceridemia (2). All patients underwent endoscopic ultrasound (EUS) guided cystgastrostomy prior to necrosectomy. Median time to cystgastrostomy following initial presentation of necrotizing pancreatitis was 69 days (range 28-280), and median time to subsequent endoscopic necrosectomy was 21 days (range 4-44). The mean procedure time for initial necrosectomy was 65 minutes (range 21-122). Overall, most patients required only one necrosectomy session (mean 1.6, range 1-4). Median time to complete resolution from first necrosectomy was 105.5 days (range 67-153). There were no immediate procedural complications and no deaths. Two patients experienced fistulization from cyst to the duodenum; one patient experienced gastrointestinal bleeding secondary to ulcer around the cystgastrostomy stent; one patient experienced migration of the cystgastrostomy stent into the necrotic cavity.

Discussion/Conclusion: The superior irrigation and suction capabilities of the “jumbo-channel” gastroscope allow for aggressive and effective direct endoscopic debridement of WOPN. The procedure time, number of necrosectomy sessions, and time to complete resolution of necrotic collection observed using the “jumbo-channel” gastroscope appear to be lower than with conventional methods reported in literature, with comparably low complication rates.

S049 CONTINUOUS WOUND INFILTRATION VERSUS EPIDURAL ANALGESIA AFTER OPEN PANCREATIC AND HEPATO-BILIARY SURGERY (POP-UP): A MULTICENTRE, RANDOMISED CONTROLLED, OPEN-LABEL, NON-INFERIORITY TRIAL

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Introduction/Background: Epidural analgesia is the international standard for pain treatment after Pancreatic and Hepato-biliary Surgery. Although some studies have suggested that continuous wound infiltration (CWI) with local anaesthetic could be equally effective, robust evidence is lacking, especially on opioid adverse effects and patient reported outcomes. We aimed to determine the effectiveness of continuous wound infiltration in the field of Pancreatic and Hepato-Biliary Surgery performed by laparotomy.
**Methods:** In this multicentre, open-label, randomised controlled non-inferiority trial (POP-UP), we enrolled adult patients undergoing open hepato-pancreato-biliary surgery in two Dutch hospitals in an enhanced recovery setting. Patients were centrally randomized (1:1) to either pain treatment by continuous (subfascial) wound infiltration (CWI) with bupivacaine plus patient controlled analgesia (PCA) with morphine or epidural analgesia with bupivacaine and sufentanil. Randomization was stratified by centre and type of incision (subcostal/midline). The primary outcome was the mean Overall Benefit of Analgesic Score (OBAS) from day 1-5, a validated composite endpoint of pain scores, opioid side effects and patient satisfaction. Analysis was by per-protocol. To establish non-inferiority, the upper bound of a one-sided 90% confidence interval for the mean OBAS from day 1-5 had to be less than +3·0. The trial is registered with the Netherlands Trial Registry, number NTR4948.

**Results:** Between Jan 20, 2015 and Sept 16, 2015, we randomly assigned 102 eligible patients. In the per-protocol analysis, the median OBAS was 3·0 [IQR 2·0-4·8] vs 4·0 [IQR 2·4-5·8] in favour of the intervention group (n=55 vs n=47), with the upper bound of the one-sided 90% CI +0·13 (95% CI: -1·54-+0·30), meeting the criteria for non-inferiority (p<0·0001). One serious adverse event, temporary local anaesthetic toxicity, occurred in the intervention group. There was less vasopressor use ((median; IQR) 0·3[0-0·8] vs 0·7 [0·3-1·5], p=0·008) and the urinary catheter could be removed earlier ((median; IQR) 4[3-5] vs 5[4-6] days, p=0·02) in the intervention group.

**Discussion/Conclusion:** Continuous wound infiltration is non-inferior to epidural analgesia after open hepato-pancreato-biliary surgery. Because of its simpler and less invasive nature it has the potential to become the analgesic method of preference in this field.

**Funding:** Solely funded by departmental sources of the Academic Medical Center, Amsterdam

**S050 PANCREATIC SURGERY AT SAFETY NET HOSPITALS: SHOULD IT BE ABANDONED?** Derek E Go, MS, Daniel E Abbott, MD, Koffi Wima, MS, Audrey E Ertel, MD, MS, Alex L Chang, MD, Jeffrey J Sussman, MD, Shimul A Shah, MD, MHCM, Richard S Hoehn, MD; University of Cincinnati, Cincinnati, US

**Introduction:** Previous work has demonstrated significant outcome and cost disparities between hospitals with different safety-net burdens, though the implications of policy changes to limit operative interventions at specific hospital types are unclear. We aimed to understand how performance of pancreaticoduodenectomy (PD) at different safety-net burden hospitals affects cost and outcomes, and how redistribution of patients from low performing hospitals to high performing hospitals could improve cost-effectiveness.

**Methods:** Hospitals performing PD were queried from the University HealthSystem Consortium database (UHC; 2009-2013) and grouped according to safety-net burden (proportion of Medicaid and uninsured patient charges, as previously described). A decision analytic model was then constructed, populated with UHC clinical and DRG-based cost data. Primary endpoints were perioperative mortality, readmission, and cost (index hospitalization plus readmission, when applicable). Sensitivity analyses were conducted to determine how primary endpoints were affected by alternative distribution of patients between hospital types and clinical outcomes.
Results: 15,090 patients populated the final dataset. Low (LBH), medium (MBH) and high (HBH) burden hospitals were comprised of 4,220 (28%), 9,505 (63%) and 1,365 (9%) patients, respectively. Perioperative mortality was twice as high at HBH (3.7%) than at LBH (1.6%) and MBH (1.7%) (p<0.001). In the base case, when all clinical and cost data were considered, PD at HBH hospitals cost $35,628/patient, 35% and 55% higher than MBH ($26,357) and LBH ($22988) hospitals, respectively. This increased cost at HBH hospitals was associated with a significantly higher readmission rate (23%) than at MBH (18%) and LBH (15%) hospitals (p<0.001). Patients at HBH hospitals were more likely to have extreme severity of illness (SOI) (18.8%) than at MBH (12.8%) and LBH (11.4%) but less likely to have moderate SOI (74.2% vs. 78% and 79.4%, respectively) (p<0.001). After patient SOI-adjusted equal redistribution of all HBH to LBH and MBH hospitals, per patient cost remained significantly lower at LBH ($25,594; 39.2% less) and MBH ($27,860; 27.9% less), with a readmission rate of 18% (vs. 23% at HBH in the base case).

Conclusion: HBH perform a minority of PDs, but have higher readmission and perioperative mortality rates at significantly higher costs. Redistribution of patients from HBH to LBH/MBH—adjusted for patient-specific risk—demonstrates significant improvement in clinical outcomes, with a potential for over $2M in annual cost savings. While patient-specific factors partially contribute to sub-optimal clinical outcomes, these data show that inherent hospital characteristics are important in optimizing cost-effective PD care, and that certain hospital types may not be optimally equipped for complex pancreatic surgery.

S051 Assessing the financial toxicity associated with treatment Options for resectable pancreatic cancer

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Background: Patients often carry the rising burden of out-of-pocket healthcare costs associated with cancer care. The high costs of different therapies, sometimes referred to as “financial toxicity,” and the relative benefit associated with these treatments have been poorly studied. We therefore sought to characterize and compare the overall costs relative to survival benefit of the treatment options most commonly employed for resectable pancreatic cancer.

Methods: The Truven Health MarketScan database was used to identify commercially insured patients who underwent a pancreatic resection (pancreatoduodenectomy, partial/distal pancreatectomy, or total pancreatectomy) between January 01, 2010 and December 31, 2012. Total costs for inpatient surgery, outpatient chemotherapy, and outpatient radiation therapy were calculated for each patient and adjusted to 2012 dollars. Generalized linear models were constructed to predict the mean costs for each treatment group. Overall survival (OS) was estimated using the Kaplan-Meier method and multivariable Cox proportional hazards regression analysis was performed to calculate the survival benefit for each treatment modality.

Results: Among the 3,395 patients identified, 1,788 (52.7%) underwent surgery only, 655 (19.3%) underwent surgery+chemotherapy, 140 (4.1%) underwent surgery+radiation therapy and 812 (23.9%) surgery+chemotherapy+radiation therapy. The average total cost for treatment was $86,201 (95%CI: $82,936-$89,416). Patients who underwent surgery alone incurred a mean cost of $64,785 (95%CI: $61,846 –
$68,084) while the costs for treatment modalities involving surgery+chemotherapy were higher (surgery+chemotherapy: $97,237 [95%CI: $89,295-$105,178]; surgery+chemotherapy+radiation therapy: $125,663 [95%CI: $116,366-$134,959]; both p<0.05, Figure 1). Median OS was 20.1 months (95%CI: 19.1–21.4 months). Upon stratification by nodal status, patients with nodal metastasis who were treated with surgery+chemotherapy+radiation therapy had a longer OS compared with surgery alone (HR=0.76, 95%CI: 0.61–0.95, p=0.01). In contrast, despite a mean increased cost of up $19,668, administration of adjuvant chemotherapy or radiation did not translate into a survival benefit among patients without nodal disease (p>0.05).

Conclusions: Total costs of care relative to improvement in survival vary significantly by treatment modality and tumor characteristics. Future studies should seek to investigate not only the efficacy of different treatment modalities, but also target areas of excess spending to reduce the cost of care for pancreatic cancer.

S052 PHYSIOLOGIC PANCREATIC CANCER IMAGING USING DYNAMIC CONTRAST-ENHANCED MAGNETIC RESONANCE IMAGING (DCE-MRI) Laura E Fischer, MD, MS, Matthias Schabel, PhD, Bryan Foster, MD, Charles R Thomas, Jr., MD, William Rooney, PhD, Alexander Guimaraes, MD, PhD, Brett C Sheppard, MD, Erin W Gilbert, MD, MCR; Oregon Health and Science University, Portland, US

Importance: Accurately identifying and monitoring pancreatic ductal adenocarcinoma (PDA) remains a significant challenge. Whereas conventional imaging (CT and MRI) report only anatomic information, dynamic contrast-enhanced MRI (DCE-MRI) has the ability to show physiologic differences in tissue perfusion based on the kinetics of IV-contrast.

Objective: We coupled DCE-MRI with data-modeling methods to obtain reproducible, high-resolution physiologic images in a series of patients with and without PDA in order to more accurately identify PDA.
Design: Prospective observational study.

Setting: The Advanced Imaging Research Center at a single academic center.

Participants: Patients with borderline-resectable PDA (BR-PDA) or at high-risk for PDA (HR), but without cancer.

Main Outcome Measures: T1-weighted images were collected during IV contrast injections [Figure 1]. Both tumor and the pancreatic body or non-tumor pancreas were identified and using a dual-compartment pharmacokinetic model, DCE-MRI parameters were quantified and compared between groups.

Results: Between 6/2014 and 2/2015, 12 DCE-MRIs were completed: 9 HR and 3 BR-PDA. Significant differences in the DCE-MRI parameters blood flow (F), first-pass extraction (E), mean capillary transit-time (tc), transfer constant (Ktrans), volume of extravascular-extracellular space (Ve) and blood volume (Vb) were found in normal[eg1] -appearing HR pancreases vs. the tumors in BR-PDA patients [Table 1A]. When comparing non-tumoral pancreases in both cohorts, only [eg2] F, Ktrans, and Vb differed [Table 1B].

Conclusions: DCE-MRI of the pancreas is feasible and provides imaging biomarkers of vascular structure and function in both tumoral and non-tumoral pancreases which may allow for earlier and more accurate identification of PDA in these high risk populations.
THE EFFECT OF CENTRALIZATION ON PROGNOSIS: OPERATED PANCREATIC DUCTAL ADENOCARCINOMA (PDA) PATIENTS IN FINLAND 2002-2008. Reea Ahola¹, Antti Siiki¹, Kaja Vasama², Martinen Vornanen², Juhani Sand¹, Johanna Laukkarinen¹; ¹Dept of Gastroenterology and alimentary tract surgery, Tampere University Hospital, Finland, ²Dept. of Pathology, Finlab Laboratories², Tampere University Hospital, Tampere, Finland, Tampere, FI

Introduction: The prognosis of operated pancreatic ductal adenocarcinoma (PDA) may be associated with hospital volume. During the past decades, centralization of pancreatic surgery has been promoted and slowly proceeded in Finland, which has a sparse population of 5.5 million people. Our aim was to analyse the characteristics of all PDA patients operated during 2002-2008 in Finland and to evaluate the effect of centralization on survival.

Patients and methods: Combining the information from the Finnish national cancer register, operation register and patient archives, all PDA patients operated in 2002-2008 in Finland were selected. Age, sex, medical history, tumor histopathology, TNM and R status, operating hospital, oncological treatment and survival were recorded. The cut point of survival was 31.12.2013. Patients with a survival of more than 4 years were re-evaluated for histopathologic diagnosis by an expert pancreatic pathologist. Operation volume per hospital was defined by the rate of operated PDA cases per year and scaled as high (>15 PDA cases) medium (5-15 PDA cases) and low (<5 PDA cases) volume centers.

Results: Nationwide, a total of 462 patients were operated for PDA during the study period (median age 67, range 40-86 years, 52% male), 43% had no previous medical history. Pancreaticoduodenectomy (PD) was performed in 86%, distal resection in 7.1% and total pancreatectomy or other resection in 6.5%. National centralization proceeded during the study period: the rate of PDA operated in high volume centers raised from 36% in 2002 to 52% in 2008. Together with this, the proportion of operated staged III or IV tumours tended to increase (6.8% vs 15.6%, ns), the proportion of resections classified
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as R1 increased (17% vs 43%; p<0.01) and the proportion of patients receiving adjuvant therapy increased (42% vs 62%; p>0.01), whereas the median timespan for the onset of adjuvant therapy remained unchanged (median 8 weeks). Significantly more lymph nodes were analysed in high volume centers compared to low volume centers (77% vs 27% over 14 nodes analysed; p<0.01). 30 day mortality was 0% in high volume centers and 4.9% in low volume centers (p<0.01). Median overall survival for RO/R1/R2 resections was 1.6/1.2/0.9 years. Survival for R0 resections in high volume centers tended to be longer compared to low volume centers (1.9 vs. 1.4 years, ns). R0 resection and adjuvant therapy were independent prognostic factors for better survival in multivariable analysis.

Conclusions: Centralization proceeded during the study period in Finland: the rate of PDA operated in high volume centers raised from 36% in 2002 to 52% in 2008. If the PDA was operated in a high-volume center, 30-day mortality was lower, survival for R0 resection tended to be longer and histopathological analysis was more precise compared to a low-volume hospital. These results encourage further centralization of pancreatic surgery in Finland.

S054 EFFECT OF ANGIOTENSIN SYSTEM INHIBITORS ON OVERALL SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA PATIENTS Hao Liu, MD, Hang Lee, PhD, Carlos Fernandez-Del Castillo, MD, Alex Jacobson, Daniela Dias Santos, MD, Andrea Zancotano, MD, James Michaelson, PhD, Yves Boucher, PhD, Keith Lillimoe, MD, Cristina Ferrone, MD, Rakesh Jain, PhD; MGH, Boston, US

Introduction: Angiotensin system inhibitors (ASI) are frequently used to manage hypertension. Laboratory data suggests that ASIs can improve cancer prognosis. The aim of this study is to investigate the effect of ASIs on overall survival in PDAC patients in a single high volume institute.

Methods: Retrospective review of the clinicopathologic records of patients with PDAC seen at the Massachusetts General Hospital (MGH) between 1/2006 – 12/2010 was performed. Patients on angiotensin converting enzyme inhibitors (ACEi) or angiotensin receptor blockers (ARB) were included as ASI users. Statistical analysis was performed using Kaplan-Meier estimator and Cox proportional hazards ratio model.

Results: A total of 826 consecutive PDAC patients were included, of whom 307 (37.2%) were on ASIs and 204 (24.7%) were on non-ASI antihypertensive drug therapy. Patients on ASIs were older (69 vs 65 years old, p=0.001). A significantly higher fraction of patients with ASI use had their primary tumors resected (44.3% vs 34.9%, p=0.005). Univariate analysis revealed that for patients who underwent tumor resection, ASI users had a significantly longer overall survival (median OS: 36.8 vs 19.3 months, p=0.006). The hazards ratio (HR) estimated from multivariate Cox’s model for death with ASI use was 0.73 (0.60-0.89, p=0.002), with adjustment for age, gender, site of tumor, surgical resection and different radiation and chemotherapy regimens. Specifically, in patients with primary tumor resection, the survival advantage of ASI therapy was still highly significant (HR=0.51, 0.33-0.77 p=0.001), while the survival benefit was smaller in patients with un-resected primary tumors (HR=0.77, 0.61-0.97, p=0.025).

Conclusion: In patients with PDAC, ASI use may improve overall survival. These findings strongly suggest that a prospective study should be performed to determine the efficacy of ASI in PDAC patients.
A NEW NOMOGRAM BETTER STRATIFIES PATIENTS WITH RESECTED PANCREATIC DUCTAL ADENOCARCINOMA THAN DOES THE AJCC STAGING: AN ANALYSIS OF 3,473 PATIENTS FROM THE PANCREAS SURGERY CONSORTIUM

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Introduction: We developed and evaluated a nomogram for patients who underwent pancreatectomy for pancreatic ductal adenocarcinoma (PDAC) and compared it with the 7th edition of the American Joint Committee on Cancer (AJCC) staging system.

Methods: Five cohorts of patients from high-volume institutions of the Pancreas Surgery Consortium (Johns Hopkins Hospital [JHH], Massachusetts General Hospital [MGH], Memorial Sloan-Kettering Cancer Center [MSKCC], University of Verona Hepatobiliary [Verona-HB], and Pancreatic Surgery [Verona-P] Units) were included in the analysis: JHH cohort was used to develop the nomogram while other cohorts were used to externally validate the resultant nomogram. The discriminative abilities of the nomogram and AJCC system were compared using the Harrell’s concordance index (c-index).

Results: A total of 3,743 patients were analyzed. Disease specific survival (DSS) was available for the 1,373 patients of the JHH cohort, which formed the training set used to develop the predictive model. In the multivariable analyses of this cohort, the most important predictor of DSS was lymphnode ratio (LNR=11%-20%, HR=1.52, p<0.001; LNR=21%-40%, HR=2.00, p<0.001; LNR>40%, HR=2.48, p<0.001). Strong predictors also included tumor grade (moderately, HR=1.57, p=0.029; poorly, HR=2.25, p<0.001), and perineural invasion (present, HR=1.93, p<0.001). Other independent prognostic factors were tumor size (>4 cm, HR=1.44, p<0.001), age (>65 years, HR=1.37, p<0.001), margin status (positive, HR=1.53, p<0.001), adjuvant therapy (performed, HR=0.61, p<0.001), and CA19-9 serum level (>100 U/mL, HR=1.15, p=0.001). All these eight variables were included in the final nomogram representing the prognostic model. In the bootstrapped internal validation, c-index was 0.68 while in the external validation, the best predictive ability was observed in Verona-P cohort with a c-index 0.66. C-indexes for MGH, MSKCC, and Verona-HB cohorts were 0.65, 0.64, and 0.63, respectively. The pooled institution-level estimate of c-index for our nomogram was 0.66 as compared to 0.55 for AJCC system. We classified the study population into different Classes of Risk according to the 5-year survival predicted by our nomogram with 20%, 40%, and 60% as cutoffs. There was a poor correlation between our Classes of Risk and the AJCC stages.
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for Class 4), Ila (from 59.2% for Class 1 to 15.3% for Class 4), and IIb (from 50.1% for Class 1 to 6.8% for Class 4).

**Conclusion:** We developed a nomogram that both predicts prognosis and stages patients with resected PDAC better than does the current AJCC system.

**S056 BIOMARKERS FOR DETECTION OF HIGH GRADE DYSPLASIA AND CARCINOMA-IN-SITU IN IPMN**

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**Background:** The classification and management of Intraductal Papillary Mucinous Neoplasms (IPMN) has undergone many modifications over the past 30 years, and the optimal approach to managing these lesions is still being elucidated. Criteria such as the Sendai Consensus Guidelines have been developed which attempt to guide management based on clinical, cytological and radiological criteria. However these guidelines are less helpful for certain lesions, such as branch-duct IPMNs. Epigenetic biomarkers could serve as a valuable addition to these guidelines to further elucidate lesions at highest risk of malignancy. 2 genes, ADAMTS1 and BNC1 have been developed as DNA methylation-based biomarkers in pancreatic cancer tissue and further validated in circulating, tumor-free DNA in the serum. We have applied these genes to IPMN tissue with varying degrees of dysplasia to determine if they can predict higher-risk lesions.

**Methods:** Paraffin embedded tissue from 102 patients who had undergone resection for IPMN was collected. Cores from regions of IPMN were taken and degree of dysplasia reported was confirmed by a pathologist. DNA was extracted and probe-based quantitative Methylation Specific PCR (qMSP) was performed using fluorescent probes. Primers and probes were designed for each gene. Statistical analysis was performed using STATA13 software.

**Results:** 18 samples with low grade dysplasia in IPMN, 43 samples with moderate dysplasia, and 41 samples with high grade dysplasia/ carcinoma-in-situ (CIS) were assessed. For ADAMTS1 alone, methylation frequency in low grade dysplasia was 11.1%, moderate was 44.2%, and high/CIS was 61.0% (p = 0.002). The difference between moderate and high was not significant, however (p = 0.124). For BNC1 alone, frequency was 27.8%, 46.5%, and 65.9% for low, moderate and high grade, respectively (p = 0.007). Again, difference between moderate and high grade was not significant (p = 0.074). For a panel combining the 2 genes, the rates for low, moderate, and high grade, respectively are 33.3%, 62.8%, and 85.4%, respectively (p<0.001) and the difference between rates of moderate and high grade is significant (p = 0.019).

**Discussion/ Conclusion:** There is a significantly increased risk of ADAMTS1 and BNC1 methylation in high grade/ carcinoma-in-situ IPMN tissue compared to moderate and low grade lesions. To further investigate this result, further experiments evaluating invasive cancer in IPMN must be performed, as well as extension of this testing to circulating tumor DNA from patients with IPMN with the aim of developing non-invasive biomarkers. A combination of an epigenetic biomarker panel using these genes with clinical and radiologic guidelines already established for IPMN can improve our management of this disease.
**S057 LOW PROGRESSION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS WITH WORRISOME FEATURES AND HIGH-RISK STIGMATA UNDERGOING NON-OPERATIVE MANAGEMENT: A MID-TERM FOLLOW-UP ANALYSIS**

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**Introduction/Background:** There is very little information regarding the fate of patients with pancreatic intraductal papillary mucinous neoplasms (IPMNs) with worrisome features or high-risk stigmata undergoing surveillance. This subgroup of patients is of particular interest since they can give insight on the real risk of IPMN-related mortality. Aim of the study is to evaluate mid-term outcomes and predictors of survival in non-operated patients with IPMNs with worrisome features or high-risk stigmata as defined by International Consensus Guidelines for IPMN.

**Methods:** In this retrospective, multicenter analysis IPMNs were classified as branch-duct (BD) and main-duct (MD), the latter including mixed IPMNs. Reasons for non-surgical options were physician’s recommendation, patient personal choice or comorbidities precluding surgery. ACE 27 score was used for comorbidities evaluation. Univariate and multivariate analysis for overall (OS) and disease-specific (DSS) survival were obtained.

**Results:** Of 281 patients identified, 159 (57%) had BD-IPMNs and 122 (43%) had MD-IPMNs; 50 (18%) had high-risk stigmata and 231 (82%) had worrisome features. Median follow-up was 51 months. The 5-year OS and DSS for the entire cohort were 81% and 89.9%. An invasive pancreatic malignancy developed in 34 patients (12%); 31 had invasive IPMNs (11%) and 3 had IPMN-distinct pancreatic ductal adenocarcinoma (1%). Independent predictors of poor DSS in the entire cohort were age > 70 years, atypical/malignant cyst fluid cytology, jaundice and MD >15 mm. Compared to MD-IPMNs, BD-IPMNs had significantly better 5-year OS (86% versus 74.1%, P=0.002) and DSS (97% versus 81.2%, P<0.0001). Patients with worrisome features had better 5-year DSS compared to those with high-risk stig mata (96.2% versus 60.2%, P<0.0001). Independent predictors of poor DSS were age > 70 years and ACE27 score > 3 for patients with worrisome features and age > 70 years, malignant cytology, jaundice and MPD > 15 mm for those with high-risk stigmata.

**Conclusions:** In patients with IPMNs that have worrisome features, the 5-year DSS is 96%, suggesting that conservative management instead of upfront surgery may be appropriate. By contrast, presence of high-risk stigmata is associated with a 40% risk of IPMN-related death, reinforcing that surgical resection should be offered to fit patients.
**S058 CIRCULATING EPITHELIAL CELLS IN INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS AND CYSTIC PANCREATIC LESIONS** Katherine E Poruk, MD¹, Ralph H Hruban, MD², John L Cameron, MD¹, Matthew J Weiss, MD¹, Nita Ahuja, MD¹, Anne Marie Lennon, MD³, Laura D Wood, MD, PhD², Christopher L Wolfgang, MD, PhD¹; ¹The Johns Hopkins Hospital Department of Surgery, ²The Johns Hopkins Hospital Department of Pathology, ³The Johns Hopkins Hospital Department of Gastroenterology, Baltimore, US

**Background:** Intraductal papillary mucinous neoplasms (IPMN) are benign mucin-producing cystic lesions of the pancreatic ducts that may be precursors to pancreatic adenocarcinoma. Prior research has suggested that circulating epithelial cells (CECs) are present in the blood of patients with IPMNs despite the absence of an associated malignancy. We assessed the blood of patients undergoing resection for IPMN or other benign pancreatic lesions for CECs with comparison to tumor histopathology.

**Methods:** Peripheral blood was collected from 26 patients without malignancy prior to pancreatic resection and filtered using the Isolation by Size of Epithelial Tumor (ISET) cell method. CECs were identified with commercially available antibodies to cytokeratin and pdx-1, a pancreas marker.

**Results:** Nineteen patients underwent resection of an IPMN without an associated malignancy. Eleven patients (58%) had cytokeratin-positive CECs. CECs were significantly more likely to be found in patients with IPMNs with high or moderate grade dysplasia (P=0.04), but there was no significant difference in IPMN size, number, or location. Additionally, 10 of the 11 patients with cytokeratin-positive CECs also had separate populations of cytokeratin-positive, pdx-1-positive CECs, suggesting a pancreatic source. Similarly, patients with cytokeratin and pdx-1 positive CECs were found in patients with higher grade dysplasia (P=0.04). No CECs were found in the blood of five patients undergoing resection for solid pseudopapillary neoplasms (SPN), one with a mucinous cystic neoplasm (MCN), and one with benign lobular proliferation. Patients with IPMNs were significantly more likely to have pan-cytokeratin CECs in the blood than those without IPMNs (P=0.01).

**Conclusions:** CECs staining positive for cytokeratin and pancreas-specific markers have been found in patients with IPMNs, even without associated malignancy. CECs may help to differentiate patients with high-grade IPMN from lower grades of dysplasia and those with other pancreatic cysts.

**S059 MULTI-INSTITUTIONAL STUDY ON THE NATURAL HISTORY OF LARGE-SIZED (?3CM) BRANCH-DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM** Alexandra M Roch, MD, MS¹, Eugene P Ceppa, MD¹, May C Tee, MD², George Marshall, MD³, Gareth Morris-Stiff, MD, PhD⁴, Adnan Alseidi, MD³, Matthew Walsh, MD⁴, Michael B Farnell, MD², C. Max Schmidt, MD, PhD, MBA¹; ¹Department of Surgery, Indiana University School of Medicine, Indianapolis, IN, USA, ²Section of Hepato-Pancreatic-Biliary Surgery, Division of Subspecialty General Surgery, Department of Surgery, Mayo Clinic, Rochester, MN, USA, ³Department of Surgery, Virginia Mason Medical Center, Seattle, WA, USA, ⁴Digestive Disease Institute, Cleveland Clinic, Cleveland, OH, USA, Indianapolis, US

**Background:** The association between size and malignant potential for branch-duct intraductal papillary mucinous neoplasm (BD-IPMN) is controversial.
Hypothesis: We hypothesized that the natural history of large-sized BD-IPMN (≥3cm) had similar outcomes than that of small BD-IPMN (<3cm).

Setting: From 2006 to 2014 at 4 academic high-volume medical centers.

Design: Prospectively followed patients who underwent primary surveillance of BD-IPMN ≥3cm.

Patients and Methods: Diagnosis of BD-IPMN was based on imaging [multifocality, main duct (MD) connection], cyst fluid analysis, and/or cytology. Patients with MD≥5mm or <6months surveillance were excluded. Indications for surveillance included comorbidities, patient preference or absence of additional International Consensus Guidelines worrisome features/high-risk stigmata (ICG-WF/HR).

Results: Sixty-three patients with BD-IPMN ≥3cm were followed for a median of 38months (8-103). Median maximal diameter was 3.3cm (3-11) at diagnosis, and 3.9cm (3-10) at last follow-up. During surveillance, BD-IPMN size increased in 37 patients (59%). Forty-one patients were managed with primary surveillance alone, whereas 22 underwent resection during follow-up [after median of 13 months (6-75)] for size increase (n=3), symptoms (n=15), cytology (n=3) and radiological features change (n=1). Surgical pathology revealed low/moderate-grade dysplasia in 18 and high-grade dysplasia in 4 patients (29% and 6%, respectively). No significant difference in malignancy was observed in patients with symptoms, size increase or additional ICG-WF/HR. No patient developed invasive carcinoma. Twelve (19%) deaths occurred, none of them related to IPMN.

Conclusion: This multi-institutional prospective series reports a better disease-specific prognosis of large-sized BD-IPMN than previously published in retrospective surgical series. Increase in BD-IPMN size during surveillance does not predict primary surveillance failure. Primary surveillance may be a reasonable option in select patients with BD-IPMN ≥3cm.

S060 PATTERNS OF RECURRENCE AND LONG-TERM OUTCOMES IN PATIENTS WHO UNDERWENT PANCREATECTOMY FOR INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS WITH HIGH GRADE DYSPLASIA: IMPLICATIONS FOR SURVEILLANCE

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Introduction: Intraductal papillary mucinous neoplasms (IPMN) are considered non-invasive, pre-malignant lesions of the pancreatic duct. While IPMNs with high-grade dysplasia (HGD) are thought to represent high-risk lesions, the natural history of this disease following pancreatic resection is unknown.

Methods: Patients with IPMN treated with pancreatectomy were identified from a single institution database from January 1999 to May 2015. Pathology reports were reviewed to classify the degree of dysplasia and the presence of invasive disease. Patterns of recurrence and long-term outcomes were analyzed retrospectively.

Results: HGD was diagnosed in 93 out of 276 patients (33.7%) who underwent pancreatectomy for IPMN. Seventeen patients had predominately moderate dysplasia but had foci of HGD. Four patients had HGD with microinvasion. IPMN was classified as main duct, branch duct, or mixed in 23 (27.4%), 31 (36.9%) and 30 patients (35.7%), respectively.
Total pancreatectomy was performed on 14 patients (15.1%) due to diffuse or multifocal disease. IPMN involved the pancreatic resection margin in 16 patients (17.2%) but the margin was positive for HGD in only 2 patients (2.2%). With a median follow-up of 32.0 months (range 1-170 months), 85 patients (91.4%) had stable residual disease or no evidence of recurrence in the remnant pancreas. No recurrence was seen in the 4 patients with microinvasion at the time of last follow-up (8.1, 32.0, 34.3 and 39.3 months). Four patients developed new or enlarging IPMN without high-risk features in the remnant pancreas and continued active surveillance. One patient who developed a second IPMN underwent completion pancreatectomy for HGD and subsequently presented with metastatic disease 31.7 months later. Three additional patients (3.2%) developed invasive adenocarcinoma in the remnant pancreas not associated with IPMN; one patient was treated with resection and the other two were treated with chemotherapy due to the presence of synchronous metastases in one and the other being medically unfit for surgery. Median time to recurrence of HGD or development of invasive adenocarcinoma was 24.0 months (range 6.7 to 72.0 months).

**Conclusion:** The prognosis of IPMN with HGD following resection is very good for most patients and the majority of patients with residual or recurrent IPMN have stable, low-risk disease. However, HGD IPMN may be a marker for developing IPMN recurrence or non-IPMN associated adenocarcinoma in a small subset of patients. As such, more intensive initial and long-term surveillance should be considered.

**S061 ELEVATED SERUM CA19-9 IN BRANCH-DUCT IPMN IS A HIGHLY-SPECIFIC PREDICTOR OF INVASIVE CANCER** Vicente Morales-Oyarvide, MD, MPH, Mari Mino-Kenudson, MD, Cristina R Ferrone, MD, Andrew L Warshaw, MD, Keith D Lillemoe, MD, Carlos Fernandez-del Castillo, MD; Massachusetts General Hospital, Harvard Medical School, Boston, US

**Background:** The utility of serum CA19-9 in detecting malignancy in intraductal papillary mucinous neoplasm (IPMN) remains controversial. We used a large cohort of resected IPMNs to evaluate its role in detecting invasive carcinoma.

**Methods:** Cohort study of 460 resected IPMNs from the Massachusetts General Hospital. We assessed the diagnostic value of preoperative serum CA19-9 using univariate and multivariate analysis, and created ROC curves.

**Results:** The median age was 68 years; there were 222 (48%) men, 200 (43%) branch-duct (BD) IPMN, and 101 (22%) patients with invasive carcinoma. The mean and median serum CA19-9 were 117.7 U/mL (SD 753 U/ml) and 11 U/mL, respectively. In univariate analysis, elevated CA19-9 was significantly associated with clinical symptoms, diabetes, main-duct/mixed IPMN, and invasive cancer. Using a cut-off value of ≥100 U/ml, the ROC area under the curve (AUC) for detection of invasive cancer in the entire cohort was 0.69 (95% CI 0.62, 0.75), with a sensitivity of 30%, specificity of 97%, positive predictive value (PPV) of 71% and negative predictive value (NPV) of 83%. Among BD-IPMN, the AUC for invasive cancer was 0.68 (95% CI 0.52, 0.84), with a sensitivity of 35%, specificity of 98%, PPV of 60%, and NPV of 94%. In multivariate analysis CA19-9 ≥100 U/mL was an independent predictor of invasive cancer in BD-IPMN (OR 16.6, 95% CI 3.2, 86.7, p=0.001) after adjusting for sex, age, symptoms, and diabetes.

**Conclusion:** Serum CA19-9 is a useful marker for detecting invasive carcinoma in BD-IPMN. Using a cut-off value of ≥100 U/mL, it is a strong predictor of invasive cancer and
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has a very high specificity. Due to its low sensitivity, non-elevated values do not rule the presence of invasive cancer.

**S062 LOCAL PROGRESSION IN THE PANCREATIC REMNANT FOLLOWING RESECTION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM (IPMN) OF THE PANCREAS OCCURS BY ONE OF THREE DISTINCT MECHANISMS**

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**Objective:** Patients that undergo surgical resection of pancreatic intraductal papillary mucinous neoplasms (IPMNs) are at risk to develop another neoplasm in the remnant pancreas.

Progression to invasive cancer in the remnant pancreas can manifest as either a conventional ductal adenocarcinoma (PDAC) that appears to have arisen from a premalignant IPMN, or as a de novo solid mass with no identifiable associated or antecedent cyst. In both IPMN and conventional PDAC, it is not clear whether these multifocal neoplasms represent disease progression of the primary resected neoplasm or independent neoplasms. This study aims to define the patterns of local progression following resection for IPMN or ductal adenocarcinoma using targeted DNA sequencing.

**Design:** Patients who developed a new neoplasm in the remnant pancreas after resection of an IPMN or a conventional PDAC and patients who underwent a resection of a PDAC and a concomitant IPMN in their resected pancreas were identified for genetic analysis.

A targeted genetic analysis was performed to identify somatic mutations of genes known to be targeted in pancreatic ductal neoplasms. The clonal relationship between metachronous or synchronous but morphologically separated pancreatic neoplasm was assessed. Subsequently, clinical and pathological characteristics of 381 patients that underwent resection of an IPMN were reviewed to identify risk factors associated with local disease progression and specifically with the development of another high-risk lesion (IPMN with HGD or Invasive carcinoma) in the remnant pancreas.

**Results:** Based on the genetic relatedness metachronous or synchronous but morphologically separated, we identified three different mechanisms underlying the development of neoplastic lesions in the remnant pancreas: 1) progressing residual microscopic disease at the resection margin 2) intraductal spread of neoplastic cell 3) multifocal disease with clonally distinct lesions. Invasive carcinoma was the main risk factor to develop a local progression in the remnant pancreas (p<0.001). In patients with non-invasive IPMNs, family history for pancreatic cancer (OR=5.82; 95% CI, 1.49–22.78; p=0.011) and high-grade dysplasia (OR=9.22; 95% CI, 2.76–20.71; p<0.001) were independent risk factors to develop another independent high-risk lesion.

**Conclusions:** We identified three distinct mechanisms for development of metachronous disease in the pancreas affected by IPMN. Our genetic analyses identified a subset of patients with multiple independent pancreatic neoplasms, confirming the presence of
truly multifocal disease with a tendency to develop high-grade lesions. Patients with a family history of pancreatic cancer or with an IPMN with high-grade dysplasia are at increased risk to develop independent pancreatic neoplasms. Taken together, these results highlight the clinical importance of multifocal neoplasms, in particular in patients with a family history of pancreatic cancer.
**S063 THE PATTERN OF RECURRENCE OF INVASIVE-IPMN IS DIFFERENT THAN CONVENTIONAL PDAC**

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**Background:** Factors associated with favorable biology of invasive intraductal papillary mucinous neoplasm (invasive IPMN) and patterns of recurrence in this disease in comparison with conventional pancreatic ductal adenocarcinoma (PDAC) are not well-studied.

**Methods:** Patients who underwent pancreatectomy for IPMN-associated invasive carcinoma or PDAC from 1995-2012 were identified. Patients with < 6-months follow-up or unknown recurrence status were excluded.

**Results:** A total of 1158 (86%) conventional PDAC and 183 (14%) invasive IPMN (colloid=44[24%], tubular=139[76%]) were identified. Compared to invasive IPMN, PDAC was associated with higher rates of advanced T-stage (T3-4: 29% vs. 78%, p<0.001), lymph-node metastasis (53% vs. 77%, p<0.001), perineural (59% vs. 89%, p<0.001), vascular invasion (33% vs. 59%, p<0.001) and positive margins (34% vs. 52%, p<0.001). Recurrence was observed in 920 (80%) conventional PDAC and 57 (31%) invasive IPMN (p<0.001). Distant metastasis was identified in 479 (41%) PDAC, and 31 (17%) invasive IPMN patients (p<0.001). Locoregional recurrence was observed in 128 (11%) conventional PDAC and 14 (8%) invasive IPMN (p=0.164). Twelve (7%) patients with invasive IPMN and 313 (27%) patients with PDAC developed both local and metastatic recurrence (p<0.001). The pattern of metastasis in patients with conventional PDAC was liver-only in 207 (35%), lung-only in 101 (17%), carcinomatosis in 53 (9%), multiple sites in 46 (8%) and other locations in 19 (3%). In patients with invasive IPMN pattern of recurrence was liver-only in 11 (38%), lung-only in 12 (41%), carcinomatosis in 2 (7%), multiple locations in 3(10%) and other locations in 1 (4%) patients.

The 5-year disease-specific survival (DSS) of tubular IPMN was 33%, compared to 59% in colloid IPMN (p=0.012), and 22% in conventional PDAC (p<0.001), (figure).

**Conclusion:** Invasive IPMN demonstrates less adverse pathologic characteristics, lower rates of recurrence and distant-metastasis, and superior DSS compared to conventional PDAC following surgical resection, indicating indolent behavior and favorable survival outcomes.
S064 NON-OPERATIVE MANAGEMENT OF LOW-RISK BRANCH-DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS IS SAFE IN THE LONG-TERM (> 5 YEARS) FOLLOW-UP

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Introduction/Background: International Consensus Guidelines (ICG) recommend non-operative management for branch-duct intraductal papillary mucinous neoplasms without worrisome features (WF) or high-risk stigmata (HRS) (“low-risk” BD-IPMNs). While some Authors suggest the effectiveness of this management, others advocate surgery for most if not all BD-IPMNs. Moreover there are few data on the long-term follow-up of low-risk BD-IPMNs. Aim of the study is to evaluate long-term outcomes of non-operated patients with low-risk BD-IPMNs (no symptoms, cyst < 30 mm, main pancreatic duct < 5 mm, no nodules, no atypical cytology).

Methods: In this retrospective, multicenter study we considered patients with a minimum follow-up of 5 years who underwent surveillance with magnetic resonance imaging. Changes during follow-up were defined as clinically-relevant (new onset of WF/HRS) or minor changes; cyst growth rate as well as predictors of clinically-relevant changes were evaluated. Surgery during follow-up was recorded. Long-term survival was analyzed.

Results: 144 patients (74 males, 70 females, mean age 62.5 years) were followed for a median of 84 months (range 68-227). At diagnosis multifocal BD-IPMNs were found in 53% of cases and mean size of the largest cyst was 15.5 ± 6.6 mm. Changes during follow-up were observed in 69 patients (48%), including increase in number of lesions (29 pts, 20%), change from unifocal to multifocal lesions in 19 (13%), increase in main pancreatic duct size (21 pts, 15%), development of nodules (5 pts, 4%), increase in cyst size in 58 patients (40%). In these patients, cyst growth rate was 0.98 ± 1.19 mm/year. Nine patients (6%) developed some symptoms, including vague abdominal pain in six, severe abdominal pain/pancreatitis in two and jaundice in one. Clinically significant changes (WF/ HRS) were observed in 24 patients (17%) but WF in only four cases (3%). WF and HRS developed after a median follow-up of 67 and 77.5 months from diagnosis, respectively. Independent predictors of WF/HRS development were size at diagnosis > 15 mm, increase in number of lesions, increase in main pancreatic duct size, cyst growth rate > 1 mm/year. Overall 7 patients (5%) underwent surgical resection for histologically-confirmed BD-IPMNs with low-grade (n=1), borderline (n=4), high-grade dysplasia (n=1) and invasive carcinoma (n=2). Overall, five patients (3.5%) developed a pancreatic malignancy and pancreatic malignancy-related mortality was 1.4%.

Conclusions: In patients with “low-risk” BD-IPMNs changes during long-term follow-up are observed in one out of two patients. However clinically-relevant changes are less common and HRS are found in only 3%. Clinically-relevant changes occur late in the follow-up, beyond 5 years. Non-operative management of “low-risk” BD-IPMNs defined by ICG is safe with a pancreatic malignancy-related mortality of less than 2%.
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EMAIL: nzyromsk@iupui.edu
# Past & Future Meetings

**SAVE THE DATE**  
51st Annual Pancreas Club Meeting  
May 5-6, 2017  
Chicago, IL

<table>
<thead>
<tr>
<th>DATE</th>
<th>LOCATION</th>
<th>HOST</th>
</tr>
</thead>
<tbody>
<tr>
<td>2015</td>
<td>Washington Court Hotel, Washington, DC</td>
<td>Christopher Wolfgang</td>
</tr>
<tr>
<td>2014</td>
<td>Westin Lombard, Chicago, IL</td>
<td>Gerard Aranha</td>
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<tr>
<td>2013</td>
<td>WDW Swan &amp; Dolphin Hotel, Orlando, FL</td>
<td>Pablo Arnoletti</td>
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<tr>
<td>2012</td>
<td>Hyatt Mission Bay, San Diego, CA</td>
<td>Mark Talamini</td>
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<tr>
<td>2011</td>
<td>Chicago, IL</td>
<td>Gerard Aranha, Mark Talamonti, David Bentrem</td>
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<tr>
<td>2010</td>
<td>New Orleans, LA</td>
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<tr>
<td>2009</td>
<td>Chicago, IL</td>
<td>Gerard Aranha, Mark Talamonti, David Bentrem</td>
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<tr>
<td>2008</td>
<td>San Diego, CA</td>
<td>Mark Talamini, Mike Bouvet</td>
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<tr>
<td>2007</td>
<td>Children’s Medical Center, Washington, DC</td>
<td>Dana Anderson</td>
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<tr>
<td>2006</td>
<td>Los Angeles, CA</td>
<td>Howard A. Reber</td>
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<tr>
<td>2005</td>
<td>Chicago, IL</td>
<td>Gerard V. Aranha, Richard Bell</td>
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<tr>
<td>2004</td>
<td>New Orleans, LA</td>
<td>Alton Ochsner</td>
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<td>2003</td>
<td>Orlando, FL</td>
<td>Michael Murr</td>
</tr>
<tr>
<td>2002</td>
<td>San Francisco, CA</td>
<td>Kimberly Kirkwood</td>
</tr>
<tr>
<td>2001</td>
<td>Hilton Atlanta, Atlanta, GA</td>
<td>Aaron Fink</td>
</tr>
<tr>
<td>2000</td>
<td>University of California, SD, San Diego, CA</td>
<td>A.R. Moosa</td>
</tr>
<tr>
<td>1999</td>
<td>Peabody, Orlando, FL</td>
<td>Michael M. Murr, James G. Norman</td>
</tr>
<tr>
<td>1997</td>
<td>University Health Sciences, Bethesda, MD</td>
<td>John W. Harmon</td>
</tr>
<tr>
<td>1996</td>
<td>Laurel Heights, UCSF, San Francisco, CA</td>
<td>Sean Mulvihill</td>
</tr>
<tr>
<td>1995</td>
<td>University of California, SD, San Diego, CA</td>
<td>A.R. Moosa</td>
</tr>
<tr>
<td>1994</td>
<td>Tulane University, New Orleans, LA</td>
<td>Elmo Cerise, J. Patrick O’Leary</td>
</tr>
<tr>
<td>1993</td>
<td>Massachusetts General Hospital, Boston, MA</td>
<td>Andrew Warshaw</td>
</tr>
<tr>
<td>1992</td>
<td>University of California, SF, San Francisco, CA</td>
<td>Carlos Pellegrini</td>
</tr>
</tbody>
</table>
1991  LSU, Tulane, New Orleans, LA  Elmo Cerise, J. Patrick O’Leary
1990  University of Texas, San Antonio, TX  Bradley Aust
1989  Washington Hilton  Gregory Bulkley, Frances Milligan, John Cameron
1988  Tulane University, New Orleans, LA  Elmo Cerise
1987  University of Illinois, Chicago, IL  Phillip Donahue
1986  Ft. Miley VA, San Francisco, CA  Carlos Pellegrini
1985  Mt. Sinai Hospital, New York, NY  David Pellegrini
1984  LSU Medical Center, New Orleans, LA  Francis Nance
1983  Washington Hilton, Washington, DC  Francis Milligan
1982  University of Chicago, Chicago, IL  A.R. Moosa
1981  Alumni Hall, NYU, New York, NY  John Ranson
1980  Salt Lake City, UT  Frank Moody
1979  LSU Medical Center, New Orleans, LA  Isadore Cohn
1978  Jockey Club, Las Vegas, NV  Charles Frey
1977  Toronto, Canada  Roger Keith
1976  Doral on the Ocean, Miami, FL  Robert Zeppa
1975  University of Texas, San Antonio, TX  Bradley Aust
1974  No Meeting
1973  Mt. Sinai Hospital, New York, NY  David Dreiling
1972  University of California, SF, San Francisco, CA  Englebert Dunphy
1971  Sheraton Hotel, Philadelphia, PA  John Howard
1970  University of Chicago, Chicago, IL  Edward Paloyan
1969  Mt. Sinai Hospital, New York, NY  David Dreiling
1968  University of California, SF, San Francisco, CA  Leon Goldman
1967  Philadelphia, PA  John Howard
1966  Northwestern, Evanston, IL  Marion Anderson
51st ANNUAL MEETING OF THE PANCREAS CLUB

May 5-6, 2017 in Chicago, IL