53rd Annual Meeting
of the
Pancreas Club

#PanClub19

Final Program

May 17 - 18, 2019
San Diego, CA
Hyatt Regency Mission Bay
THE PANCREAS CLUB THANKS OUR INDUSTRY PARTNERS

The Pancreas Club would like to thank the following organizations for their marketing support of the 2019 Annual Meeting:

IntraOp | Silver Sponsor
AngioDynamics | Bronze Sponsor

The Pancreas Club would like to thank the following companies for their generous support as Exhibitors:

Abbvie
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Medi Lazer
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MEETING HIGHLIGHTS

DIRECTORS

William H. Nealon, MD
Christopher L. Wolfgang, MD, PhD
Nicholas J. Zyromski, MD

65 Oral Presentations
20 Posters of Distinction
200+ Posters

What is the Definition of Unresectable?

NEW GRANT!
Nikki Mitchell Foundation & Pancreas Club
$20,000 Seed Grant
View Poster Abstracts and Member Directory in Meeting App or Online in the Members Only Section!
PANCREAS CLUB AWARDS

The Pancreas Club will recognize five outstanding presentations. They will be awarded during the closing, Saturday afternoon, reception:

**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.

**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.

**Pancreas Club 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 Nikki Mitchell Foundation.

**Nikki Mitchell Award:** $1,500 award for the most promising research project by a young investigator (Junior faculty within 5 years of practice, post training).

**New Award Announcement!** The Pancreas Club is very pleased to unveil the Nikki Mitchell Foundation & Pancreas Club Seed Grant, generously donated by the Nikki Mitchell Foundation. The seed grant is worth up to $20,000 and will fund promising Pancreas Cancer research, with a focus on Basic Science.
The Pancreas Club thanks our generous partners for their contributions which allow us to fund awards for promising research presented at the Annual Meeting.

**ARPA Foundation (funds John Howard Research Award)**

The ARPA Foundation “Fondazione Arpa” is based in Pisa, Italy. The Foundation aims to promote study, research, documentation and updating activities in the field of medical-surgical sciences. The Foundation intends to promote and support those research projects which, although born out of surgical interest, project their questions and their results on multiple fields of interest, among which, by way of example, we indicate oncology, new technologies and the study of organizational models and health economics. In this sense, the Foundation operates through the direct funding of scientific research, through the establishment of scholarships for Italy and abroad, as well as with scientific collaboration contracts in order to allow students and young researchers, acquisition of new knowledge and specific skills in this field.

ARPA also promotes exchanges between students and graduates with university institutions of foreign countries, creating the basis for the creation and development of common research programs. Acts of volunteering, sharing, participation, solidarity: these are the values that, since 1992, embodies the Foundation. Over the years ARPA has chosen to invest resources to make an important contribution to the development of clinical and basic research, with attention to the field of oncology and transplants. The ARPA Foundation’s story tells of a growing commitment to increase the training of the multiple professionals who work in the health field, both in Italy and in developing countries.

For more information, please visit http://fondazionearpa.it/
The Pancreas Club thanks our generous partners for their contributions which allow us to fund awards for promising research presented at the Annual Meeting.

Pancreatic Cancer Action Network (funds PanCAN Research Award) Founded in 1999, the Pancreatic Cancer Action Network (PanCAN) is dedicated to fighting the world’s toughest cancer. Their mission is to save lives, and attack pancreatic cancer on all fronts: research, clinical initiatives, patient services and advocacy. PanCAN’s effort is amplified by a nationwide network of grassroots support. They are determined to improve patient outcomes today and to double survival by 2020.

The Pancreatic Cancer Action Network is changing the future of pancreatic cancer. PanCAN believes we need to give people more than just time. They want patients to have access to more powerful and effective treatments. As well as offer ALL patients and their families hope and health. PanCAN invests in patients and relentlessly works to realize them. They partner with forward-thinking friends who dare to change the world of medical science — for patients here and around the world. With 58 affiliates and a volunteer corps of more than 8,000 people, PanCAN raises funds and national awareness of the disease through their PurpleStride run/walk events in communities nationwide. Their grassroots efforts have also resulted in a monumental global movement — PanCAN plays a lead role in the World Pancreatic Cancer Coalition, composed of more than 65 pancreatic cancer patient groups from around the world.

For more information, please visit https://www.pancan.org/
The Pancreas Club thanks our generous partners for their contributions which allow us to fund awards for promising research presented at the Annual Meeting.

Nikki Mitchell Foundation (funds Pancreas Club Award, Nikki Mitchell and Nikki Mitchell Foundation & Pancreas Club Seed Grant)

Nikki Mitchell saw only great possibilities in life and in others. When Nikki landed in Nashville, she ran the enterprises of Waylon Jennings and Jessi Colter and served as President of their company for 22 years. During that time, she made history of her own with the Bridge of Wings flight, circumnavigating the northern hemisphere as co-captain with NMF President Rhonda Miles in a single engine aircraft. This commemorative flight served to spotlight the heroic Soviet female pilots whom time had forgotten. In December 2010, Nikki was diagnosed with pancreatic cancer and battled the disease for 31 months. Her last dream was to save others from the devastating disease of pancreatic cancer. Caring about early detection research led her to participate, upon her death, in a floating tumor cell research program facilitated by Johns Hopkins Hospital. Nikki’s donation was the first to contribute a vast amount of data to the scientists.

The Nikki Mitchell Foundation is dedicated to providing comfort and relief for those affected by pancreatic cancer, while raising awareness and searching for the cure.

For more information, please visit http://www.nikkimitchellfoundation.org/
PANCREAS CLUB

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Laura Fitzgerald  Finance
Corinne Hornsey  Meetings & Events
Deborah East  Industry Relations
Mary Kawulok  Registrar
Shayla Concannon  Communications
Nora Barrett  Onsite Registration

Pancreas Club Headquarters
t: 913.402.7102
pancreas@lp-etc.com
www.pancreasclub.com

Please mail any registration or dues payments to:

Pancreas Club
PO Box 219191
Kansas City, MO 64121-9191
EDUCATIONAL OBJECTIVES

LEARNING OBJECTIVES
This program has been constructed by the Program Committee of the Pancreas Club and has been selected from abstracts submitted by the membership of the Club. The subject matter selected is a cross-section of the cutting edge of surgical practice today. At the conclusion of this activity participants will:

1. Define the definitions and grading systems for managing pancreatic fistula.

2. Understand the principles of immunotherapy and their application in the management of pancreatic cancer

3. Recognize the principles in managing PNETs between 1 and 2 cm in diameter

4. Recognize the importance of non-progression in patients managed with borderline respectable principles and locally advanced PDAC

5. Understand the impact of different treatment on circulating tumor cells and circulating DNA (precision medicine).

6. Explain the role of gene expression profiling in the progression of IPMN to cancer.

7. Comment on the role of imaging in the clinical staging of pancreatic cancer after multi-drug modalities

DISCLOSURE INFORMATION
In compliance 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. All reported conflicts are managed by a designated official to ensure a bias-free presentation. Please see the insert to this program for the complete disclosure list.
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 **15 AMA PRA Category 1 Credits™**. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Of the **15** listed above, a maximum of **15** credits meet the requirements for Self-Assessment.
Thank you to the 2019 Scientific Program Committee:

William Nealon
Christopher Wolfgang
Nicholas Zyromski
Marc Besselink
Rae Brana
Melissa Hogg
Nigel Jamieson
Giuseppe Malleo
Kyoichi Takaori
Yoo-Seok Yoon
PANCREAS CLUB ANNUAL MEETING LOCATIONS

2018 Willard InterContinental, Washington DC
2017 Drake Hotel Chicago, IL
2016 Hyatt Regency Mission Bay, San Diego, CA
2015 Washington Court Hotel, Washington, DC, Christopher Wolfgang
2014 Westin Lombard, Chicago, IL, Gerard Aranha
2013 WDW Swan & Dolphin Hotel, Orlando, FL, Pablo Arnoletti
2012 Hyatt Mission Bay, San Diego, CA, Mark Talamini
2011 Chicago, IL, Gerard Aranha, Mark Talamonti, David Bentrem
2010 New Orleans, LA
2009 Chicago, IL, Gerard Aranha, Mark Talamonti, David Bentrem
2008 San Diego, CA, Mark Talamini, Mike Bouvet
2007 Children’s Medical Center, Washington, DC, Dana Anderson
2006 Los Angeles, CA, Howard A. Reber
2005 Chicago, IL, Gerard V. Aranha, Richard Bell
2004 New Orleans, LA, Alton Ochsner
2003 Orlando, FL, Michael Murr
2002 San Francisco, CA, Kimberly Kirkwood
2001 Hilton Atlanta, Atlanta, GA, Aaron Fink
2000 University of California, SD, San Diego, CA, A.R. Moosa
1999 Peabody, Orlando, FL, Michael M. Murr, James G. Norman
1998 LSU, Tulane, New Orleans, LA, J. Patrick O’Leary, Elmo Cerise
1997 University Health Sciences, Bethesda, MD, John W. Harmon
1996 Laurel Heights, UCSF, San Francisco, CA, Sean Mulvihill
1995 University of California, SD, San Diego, CA, A.R. Moosa
1994 Tulane University, New Orleans, LA, Elmo Cerise, J. Patrick O’Leary
1993 Massachusetts General Hospital, Boston, MA, Andrew Warshaw
1992 University of California, SF, San Francisco, CA, Carlos Pellegrini
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
PANCREAS CLUB ANNUAL MEETING LOCATIONS (continued)

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 Dreiling
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
SCHEDULE AT A GLANCE
## SCHEDULE AT A GLANCE

### Friday, May 17

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<td>Registration</td>
<td>Bayview Foyer</td>
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<td>7:00am - 8:00am</td>
<td>Breakfast in Exhibit Hall</td>
<td>Regatta Pavilion</td>
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<tr>
<td>7:45am - 8:00am</td>
<td>Welcome &amp; Introductory Remarks</td>
<td>Bayview Ballroom</td>
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<td>8:00am - 9:45am</td>
<td>Scientific Session 1</td>
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<td>10:00am - 11:00am</td>
<td>Scientific Session 2</td>
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<td>Poster Rounds with Professors</td>
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<td>Lunch</td>
<td>Regatta Pavilion</td>
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<td>1:00pm - 3:30pm</td>
<td>Scientific Session 3</td>
<td>Bayview Ballroom</td>
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<td>3:30pm - 3:45pm</td>
<td>Afternoon Break</td>
<td>Regatta Pavilion</td>
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<td>3:45pm - 5:30pm</td>
<td>Scientific Session 4</td>
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<td>5:30pm - 8:30pm</td>
<td>Pancreas Club Annual Reception and Dinner</td>
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**SCHEDULE AT A GLANCE (continued)**

**Saturday, May 18**

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<td><strong>Registration</strong></td>
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<td><strong>Breakfast in the Exhibit Hall</strong></td>
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<td><strong>Definition of Unresectable</strong></td>
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<td><strong>Scientific Session 6</strong></td>
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<td><strong>Afternoon Beverage</strong></td>
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<td>3:15pm - 5:00pm</td>
<td><strong>Scientific Session 7</strong></td>
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<td>5:00pm - 6:00pm</td>
<td><strong>Awards Reception</strong></td>
<td>Sunset Terrace</td>
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SCIENTIFIC PROGRAM

- **John Howard Research Award**
  Best presentation from young junior faculty, who is within 5 years of their end of residency

* **Nikki Mitchell Award**
  Most promising research project by a young investigator, who is junior faculty within the first 5 years of practice

- **PanCan Research Award**
  Best presentation of pancreatic cancer research by a resident or fellow.

† **Pancreas Club Award**
  Best presentation of clinical or basic science by a resident or fellow.
Friday, May 17, 2019

7:00am - 5:30pm | Bayview Foyer
Registration

7:00am – 8:00am | Regatta Pavilion
Breakfast in Exhibit Hall

7:45am - 8:00am | Bayview Ballroom
Welcome & Introductory Remarks
William Nealon MD | Northwell Health System

8:00am - 9:45am | Bayview Ballroom
Scientific Session 1: Cancer Basic/Clinical
Moderators:
Michael Bouvet MD | University of California San Diego
Nigel Jamieson MD, PhD | Glasgow Royal Infirmary

8:00am - 8:15am
• † 1. GUT MICROBIOTA FACILITATE TOLL-LIKE RECEPTOR 2-MEDIATED PANCREATIC CANCER METASTASES
Saba Kurtom MD | University of Miami

8:15am - 8:30am
• 2. INTERNATIONAL VALIDATION OF THE AMSTERDAM MODEL FOR SURVIVAL PREDICTION AFTER RESECTED PANCREATIC CANCER
Stijn van Roessel MD, MSc | Amsterdam UMC

+ John Howard Research Award  • Nikki Mitchell Award  • PanCan Research Award  † Pancreas Club Award
8:30am - 8:45am
• † 3. CELL-INTRINSIC PD-1 PROMOTES PROLIFERATION IN PANCREATIC CANCER TARGETING CYR61/CTGF VIA HIPPO PATHWAY
Ning Pu MD | Johns Hopkins University School of Medicine

8:45am - 9:00am
• † 4. SEX AFFECTS RESPONSE TO TYROSINE KINASE INHIBITION IN PANCREATIC ADENOCARCINOMA
Betzaira G. Childers MD | University of Cincinnati

9:00am - 9:15am
• † 5. A BLOOD-TEST TO MEASURE OUTCOME AND RESPONSE TO THERAPY: DEVELOPING THE NECESSARY TOOLS FOR PRECISION TREATMENT OF PANCREATIC DUCTAL ADENOCARCINOMA
Ammar Javed MD | Johns Hopkins University School of Medicine

9:15am - 9:20am
† 6. PLATFORMS FOR DELIVERY OF TUMOR-SPECIFIC FLUORESCENCE IMAGING OF HUMAN PANCREATIC CANCER
Michael Bouvet MD | University of California, San Diego

9:20am - 9:25am
• 7. WNT11 DRIVES PDAC CELL MIGRATION AND INVASION AND IS ASSOCIATED WITH Β-INTEGRIN SIGNALING PATHWAYS
Tara Hughes MD | University of Texas MD Anderson Cancer Center
9:25am - 9:30am
• † 8. DIVERSITY OF GERMLINE VARIANTS AMONG PATIENTS WITH LOCALIZED PANCREATIC CANCER
Ashley Krepline MD | Medical College of Wisconsin

9:30am - 9:45am
9. ANTI-CTGF HUMAN RECOMBINANT MONOCLONAL ANTIBODY PAMREVLUMAB (FG-3019) INCREASED RESECTABILITY AND RESECTION RATES WHEN COMBINED WITH GEMCITABINE/NAB-PAACLITAXEL IN PHASE 1/2 CLINICAL STUDY FOR THE TREATMENT OF LOCALLY ADVANCED PANCREATIC CANCER PATIENTS
Flavio Rocha MD | Virginia Mason Medical Center

9:45am – 10:00am | Regatta Pavilion
Morning Break

10:00am - 11:00am | Bayview Ballroom
Scientific Session 2: Technique/Outcomes
Moderators:
Katy Morgan MD | Medical University of South Carolina
William Burns MD | Johns Hopkins Medicine

10:00am - 10:15am
10. IMPACT OF REINFORCED STAPLER DURING DISTAL PANCREATECTOMY FOR PANCREATIC FISTULA, A MULTICENTER RANDOMIZED CONTROLLED TRIAL
Kenichiro Uemura MD | Hiroshima University
10:15am - 10:30am  
**11. MENTORSHIP AND FORMAL ROBOTIC PROFICIENCY SKILLS CURRICULUM IMPROVE SUBSEQUENT GENERATIONS’ LEARNING CURVE FOR THE ROBOTIC PANCREATODUODENECTOMY**  
Melissa Hogg MD, MS | NorthShore HealthSystem

10:30am - 10:45am  
• † **12. VARIABLE LIFE ADJUSTED DISPLAY (VLAD): NOVEL UTILITY OF A METRIC FOR QUANTIFICATION OF PERIOPERATIVE PATIENT LIVES GAINED FOLLOWING PANCREATIC SURGERY**  
Naomi Sell MD, MHS | Massachusetts General Hospital

10:45am - 10:50am  
**13. VIDEO REVIEW OF SURGEON TECHNICAL FACTORS OF THE HEPATICOJEJUNOSTOMY INDEPENDENTLY PREDICTS POSTOPERATIVE BILIARY COMPLICATIONS AFTER PANCREATICODUODENECTOMY**  
James Brown | University of Pittsburgh Medical Center

10:50am - 10:55am  
• **14. IMPLEMENTING ROBOT-ASSISTED DISTAL PANCREATECTOMY THROUGH A PROCEDURE-SPECIFIC TRAINING PROGRAM: AN ACADEMIC CENTER’S EXPERIENCE**  
Sjors Klompmaker MD | Beth Israel Deaconess Medical Center
10:55am - 11:00am

† 15. ARE THERE ANY DIFFERENCES IN OUTCOMES WITH THE VARIOUS SURGICAL APPROACHES TO MANAGEMENT OF CHRONIC PANCREATITIS; AN AMERICAN COLLEGE OF SURGERY (ACS) NATIONAL SURGICAL QUALITY IMPROVEMENT PROGRAM (NSQIP) SURVEY
George Baison MD | Virginia Mason Medical Center

11:00am - 12:00pm | Regatta Pavilion
Poster Rounds with Professors | P 1 - P 10 Presentations
Moderators:
Marshall Baker MD | Northshore University Health Systems
Flavio Rocha MD | Virginia Mason Medical Center

12:00pm - 1:00pm | Regatta Pavilion
Lunch

1:00pm – 3:30pm | Bayview Ballroom
Scientific Session 3: Clinical Cancer/Outcomes
Moderators:
Charles M Vollmer MD | University of Pennsylvania
Giovanni Marchegiani MD | Verona University - Pancreas Institute

1:00pm - 1:15pm

16. A PHASE III STUDY OF CHEMOTHERAPY WITH OR WITHOUT ALGENPANTUCEL-L (HYPERACUTE®-PANCREAS) IMMUNOTHERAPY IN SUBJECTS WITH BORDERLINE RESECTABLE OR LOCALLY ADVANCED UNRESECTABLE PANCREATIC CANCER
D. Brock Hewitt MD, MPH, MS | Thomas Jefferson University

+ John Howard Research Award  • Nikki Mitchell Award  • PanCan Research Award  † Pancreas Club Award
1:15pm - 1:30pm

17. GASTRIC CANDIDIASIS PREOPERATIVELY EXAMINED IS A CRUCIAL PREDICTOR OF POSTOPERATIVE INFECTION-RELATED COMPLICATIONS AFTER PANCREATICODUODENECTOMY: THE RESULTS OF PROSPECTIVE STUDY
Kazuyuki Gyoten MD | Mie University School of Medicine

1:30pm - 1:45pm

• 18. MOLECULAR STAGING IN PANCREATIC ADENOCARCINOMA UTILIZING KRAS MUTANT CELL-FREE DNA ANALYSES IN PERIPHERAL BLOOD AND PERITONEAL FLUID: ARE WE THERE YET?
Jennifer Leiting MD | Mayo Clinic Rochester

1:45pm - 2:00pm

• 19. MINIMALLY INVASIVE DISTAL PANCREATECTOMY REDUCES MAJOR MORBIDITY AND LENGTH OF STAY COMPARED TO OPEN: MULTINATIONAL VALIDATION OF A NEW GOLD STANDARD
James Moser MD | Beth Israel Deaconess Medical Center

2:00pm - 2:15pm

† 20. RISK FACTORS FOR MULTI-DRUG RESISTANT BACTERIA INFECTION AMONG RECTAL CARRIERS SUBMITTED TO PANCREATICODUODENECTOMY: A PROSPECTIVE OBSERVATIONAL STUDY
Salvatore Paiella MD | University of Verona
2:15pm - 2:30pm
+ * 21. VARIATION IN THE SURGICAL MANAGEMENT OF LOCALLY ADVANCED PANCREATIC CANCER
Bradley Reames MD, MS | University of Nebraska Medical Center

2:30pm - 2:45pm
+ * 22. THE EFFECTS OF HIGH DOSE PANCREATIC ENZYME REPLACEMENT THERAPY ON BODY WEIGHT, NUTRITIONAL ASSESSMENT AND QUALITY OF LIFE AFTER PANCREATODUODENECTOMY
Hongbeom Kim MD | Seoul National University Hospital

2:45pm - 3:00pm
* 23. SOMATIC MUTATIONS IN RESECTED SPECIMENS OF PANCREATIC CANCER WITH PATHOLOGICAL COMPLETE RESPONSE TO NEOADJUVANT THERAPY
Lingdi Yin MD | Johns Hopkins University School of Medicine

3:00pm - 3:05pm
• 24. TEXTBOOK OUTCOME AS A NOVEL COMPOSITE QUALITY MEASURE IN PANCREATIC SURGERY: A NATIONWIDE ANALYSIS
Stijn van Roessel MD, MSc | Amsterdam UMC

3:05pm - 3:10pm
25. SOCIAL AND EMOTIONAL WELL-BEING IN AN HPB SURGERY PATIENT POPULATION
Theresa Yeo PhD | Thomas Jefferson University
3:10pm - 3:15pm

26. SYSTEMATIC REVIEW OF SURGICAL RESECTION OF PANCREATIC CANCER WITH SYNCHRONOUS LIVER METASTASES IN THE ERA OF MULTIAGENT CHEMOTHERAPY
Stefano Crippa MD, PhD | San Raffaele Scientific Institute

3:15pm - 3:30pm

• 27. THE LAPAROSCOPIC APPROACH TO PANCREATICODUODENECTOMY IS COST NEUTRAL IN VERY HIGH-VOLUME CENTERS
Emanuel Eguia MD, MHA | Loyola University Medical Center

3:30pm – 3:45pm | Regatta Pavilion
Afternoon Break

3:45pm – 5:30pm | Bayview Ballroom
Scientific Session 4: Clinical Cancer/Outcomes II
Moderators:
Miao Yi MD, PhD | Nanjing Medical University
William Nealon MD | Northwell Health

3:45pm - 4:00pm

+ * 28. NATIONWIDE ASSESSMENT OF A RISK-STRATIFIED DRAIN PLACEMENT STRATEGY DURING PANCREATICODUODENECTOMY USING THE MODIFIED FISTULA RISK SCORE
Jordan Cloyd MD | The Ohio State University
4:15pm - 4:30pm
• † 29. ROLE OF SURGICAL RESECTION IN THE ERA OF FOLFIRINOX FOR ADVANCED PANCREATIC CANCER
Yoonhyeong Byun MD | Seoul National University Hospital

4:30pm - 4:45pm
30. DEFINING THE SAFETY PROFILE FOR PERFORMING PANCREATODUODENECTOMY IN THE SETTING OF HYPERBILIRUBINEMIA
Bofeng Chen BA | University of Pennsylvania

4:45pm - 5:00pm
• 31. MULTI-INSTITUTIONAL DEVELOPMENT AND EXTERNAL VALIDATION OF A NOMOGRAM TO PREDICT RECURRENCE AFTER CURATIVE RESECTION OF PANCREATIC NEUROENDOCRINE TUMORS
Alessandra Pulvirenti MD | University of Verona

5:00pm - 5:15pm
• 32. IMMUNE MODULATION BY LIPOPOLYSACCHARIDE SUPPRESSES PANCREATIC CANCER PROGRESSION
Anthony Ferrantella MD | University of Miami

5:15pm - 5:30pm
• 33. IRREVERSIBLE ELECTROPORATION ACTS AS AN IN SITU VACCINE IN A MURINE PANCREATIC CANCER MODEL
Jayanth Shankara Narayanan PhD | University of California San Diego

+ John Howard Research Award  • Nikki Mitchell Award  • PanCan Research Award  † Pancreas Club Award
SCIENTIFIC PROGRAM  
Friday, May 17

5:30pm - 8:30pm | Banyan Court & Lawn
Pancreas Club Annual Reception and Dinner
Saturday, May 18, 2019

7:00am – 5:00pm | Bayview Foyer
Registration

7:00am – 8:00am | Regatta Pavilion
Breakfast in the Exhibit Hall

8:00am – 9:45am | Bayview Ballroom
Scientific Session 5: Cancer Basic/Clinical II
Moderators:
Matthew Katz MD | University of Texas MD Anderson Cancer Center
Keita Wada MD, PhD | Teikyo University

7:40am - 7:55am
• 34. CIRCULATING TUMOR CELL DYNAMICS ARE ASSOCIATED WITH OUTCOMES IN PANCREATIC DUCTAL ADENOCARCINOMA: UPDATES ON THE PROSPECTIVE CLUSTER TRIAL
Alina Hasanain MD | Johns Hopkins University School of Medicine

7:55am - 8:10am
• 35. THE PRESENCE OF STEM CELL PHENOTYPE CIRCULATING TUMOR CELLS IN PANCREATIC CANCER IS ASSOCIATED WITH AGGRESSIVE TUMOR BIOLOGY
Floortje van Oosten MD | Johns Hopkins University School of Medicine
8:10am - 8:25am
• † 36. OUTCOME OF PATIENTS WITH BORDERLINE RESECTABLE PANCREATIC CANCER IN THE CONTEMPORARY ERA OF NEOADJUVANT CHEMOTHERAPY
Ammar Javed MD | Johns Hopkins University School of Medicine

8:25am - 8:40am
• † 37. PREDICTIVE VALUE OF CYTOKINE PROFILES FROM FINE NEEDLE ASPIRATES FOR THE DIAGNOSIS OF PANCREATIC DUCTAL ADENOCARCINOMA
Patrick Underwood MD | University of Florida

8:40am - 8:45am
• 38. PANCREATIC FLUID INTERLEUKIN-1B COMPLEMENTS PROSTAGLANDIN E2 AND SERUM CA19-9 IN PREDICTION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM DYSPLASIA
Rachel Simpson MD | Indiana University School of Medicine

8:45am - 9:00am
39. QUALITY OF LIFE AND GLOBAL HEALTH AFTER PANCREATIC SURGERY IS CONSISTENT WITH THE GENERAL POPULATION: THE LONG-TERM OUTLOOK FROM 9 YEARS
Michael Kluger MD, MPH | Columbia University
9:00am - 9:15am

• † 40. COMPREHENSIVE GENOMIC PROFILING OF PANCREATIC CANCER TUMOR SPECIMENS: IS MORE BETTER?
Lindsay Bliss MD, MPH | Medical College of Wisconsin

9:15am - 9:20am

• † 41. THE IMPACT OF PATHOLOGIC COMPLETE RESPONSE ON SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA
Naomi Sell MD, MHS | Massachusetts General Hospital

9:20am - 9:35am

+ 42. ENDOSCOPIC ULTRASOUND-GUIDED CELIAC GANGLION RADIOFREQUENCY ABLATION VERSUS CELIAC PLEXUS NEUROLYSIS FOR PALLIATION OF PAIN IN PANCREATIC CANCER: A RANDOMIZED CONTROLLED TRIAL
Ji Young Bang MD, MPH | Florida Hospital Orlando

9:35am - 9:40am

• † 43. SIMULATED VOLUME-BASED REGIONALIZATION OF PANCREATECTOMY PROCEDURES: IMPACT ON SPATIAL ACCESS TO CARE
Zhi Ven Fong MD, MPH | Massachusetts General Hospital

+ John Howard Research Award  • Nikki Mitchell Award  • PanCan Research Award  † Pancreas Club Award
9:40am - 9:45am
† 44. PATHOLOGY HAS THE LAST WORD - PANCREATIC FIBROSIS IS A BETTER PARAMETER FOR PREDICTION OF PANCREATIC FISTULA THAN TEXTURE - A RETROSPECTIVE ANALYSIS FROM THE RECO-PANC TRIAL
Ekaterina Petrova MD | University Clinic Schleswig-Holstein (UKSH) Campus Lübeck, Germany

9:45am – 10:00am | Regatta Pavilion
Morning Break

10:00am - 11:00am | Bayview Ballroom
Definition of Unresectable
Moderator: Nicholas J Zyromski MD | Indiana University School of Medicine
Borderline, History and Current State
Matthew Katz MD | University of Texas MD Anderson Cancer Center
Options for Locally Advanced Rx
Motaz Qadan MD, PhD | Massachusetts General Hospital
Metastatic PDAC – Is There Any Logic for Resection?
Matthew Weiss MD | Northwell Health
SCIENTIFIC PROGRAM

Saturday, May 18

11:00am - 12:00pm | Regatta Pavilion
Poster Rounds with Professors | P 11 - P 20 Presentations
Moderators:
William Lancaster MD | Medical University of South Carolina
Stjin van Roessel MD, MSc | Amsterdam UMC

12:00pm - 1:00pm | Regatta Pavilion
Lunch

1:00pm - 3:00pm | Bayview Ballroom
Scientific Session 6: Basic/Clinical Pancreatitis
Moderators:
Rajesh Gupta MBBS, MS, MCh | Postgraduate Institute of Medical Education and Research
Nicholas Zyromski MD | Indiana University Hospital

1:00pm - 1:15pm
45. PREDICTORS OF SAME-ADMISSION CHOLECYSTECTOMY IN MILD, ACUTE, BILIARY PANCREATITIS
Steven Cunningham MD | Saint Agnes Hospital

1:15pm - 1:20pm
46. INTERLEUKIN 4 RECEPTOR SIGNALING MEDIATES A REGENERATIVE RESPONSE IN THE DUCTAL EPITHELIUM IN RESPONSE TO PANCREATITIS
Kate Von Alt BS | Massachusetts General Hospital

+ John Howard Research Award  • Nikki Mitchell Award  • PanCan Research Award  † Pancreas Club Award
1:20pm - 1:25pm

47. LOCAL AND SYSTEMIC EFFECTS OF AGING ON ACUTE PANCREATITIS
Marcel Machado MD | University of Sao Paulo School of Medicine

1:25pm - 1:30pm

† 48. THERAPEUTIC USE OF ADIPOSE-DERIVED STROMAL CELLS IN A MURINE MODEL OF ACUTE PANCREATITIS
Alexandra Roch MD | Indiana University School of Medicine

1:30pm - 1:45pm

49. THE CLINICAL COURSE AND DIAGNOSTIC WORK-UP OF IDIOPATHIC ACUTE PANCREATITIS, A POST-HOC ANALYSIS OF A PROSPECTIVE MULTICENTER OBSERVATIONAL COHORT
Devica Umans | Erasmus Medical Center

1:45pm - 2:00pm

† 50. NATURAL HISTORY OF DISCONNECTED PANCREATIC DUCT SYNDROME: WHICH OPERATION AND WHEN?
Thomas Maatman MD | Indiana University School of Medicine

2:00pm - 2:15pm

51. INTERNATIONAL STUDY GROUP FOR PANCREAS SURGERY: STANDARDS FOR REPORTING OF SURGERY FOR CHRONIC PANCREATITIS
Ajith Siriwardena MD | Manchester Royal Infirmary
2:15pm - 2:30pm
52. COMPLICATIONS OF PERCUTANEOUS DRAINAGE IN STEP-UP APPROACH FOR MANAGEMENT OF PANCREATIC NECROSIS: EXPERIENCE OF TEN YEARS FROM A TERTIARY CARE CENTRE
Rajesh Gupta MBBS, MCh | Postgraduate Institute of Medical Education and Research Chandigarh

2:30pm - 2:45pm
† 53. EARLY ENDOSCOPIC RETROGRADE CHOLANGIOGRAPHY WITH BILIARY SPHINCTEROTOMY OR CONSERVATIVE TREATMENT IN PREDICTED SEVERE ACUTE BILIARY PANCREATITIS (APEC): A MULTICENTER RANDOMIZED CONTROLLED TRIAL
Nicolien Schepers MD | Erasmus Medical Center

2:45pm - 2:50pm
† 54. DISCONNECTED PANCREATIC DUCT SYNDROME: SPECTRUM OF OPERATIVE MANAGEMENT
Thomas Maatman MD | Indiana University School of Medicine

2:50pm - 2:55pm
+ 55. SUPERIORITY OF ENDOSCOPIC INTERVENTIONS OVER MINIMALLY INVASIVE SURGERY FOR INFECTED NECROTIZING PANCREATITIS: A META-ANALYSIS OF RANDOMIZED TRIALS
Ji Young Bang MD, MPH | Florida Hospital, Center for Interventional Endoscopy
2:55pm - 3:00pm

56. ROLE OF INFLAMMATORY MARKERS IN STEP-UP APPROACH IN ACUTE NECROTIZING PANCREATITIS: RESULTS OF A PROSPECTIVE STUDY
Aditya Kulkarni MS, DNB | Postgraduate Institute of Medical Education and Research Chandigarh

3:00pm - 3:15pm | Regatta Pavilion
Afternoon Beverage

3:15pm – 5:00pm | Bayview Ballroom
Scientific Session 7: Clinical Cancer/Outcomes
Moderators:
Christopher Wolfgang MD, PhD | Johns Hopkins Medicine
Kyoichi Takaori MD | Kyoto University

3:15pm - 3:30pm
+ * 57. NUMBER OF HARVESTED LYMPH NODES AND STAGE MIGRATION EFFECT IN PANCREATODUODENECTOMY FOR PANCREATIC DUCTAL ADENOCARCINOMA: REAPPRAISING THE STANDARDS BASED ON THE EIGHTH EDITION OF THE AMERICAN JOINT COMMITTEE ON CANCER (AJCC) STAGING SYSTEM
Giuseppe Malleo MD, PhD | University of Verona

3:30pm - 3:45pm
58. A NOVEL VALIDATED RECURRENCE RISK SCORE TO GUIDE A PRAGMATIC SURVEILLANCE STRATEGY AFTER RESECTION OF PANCREATIC NEUROENDOCRINE TUMORS: AN INTERNATIONAL STUDY OF 1006 PATIENTS
Valentina Andreasi MD | Emory University

+ John Howard Research Award • Nikki Mitchell Award • PanCan Research Award † Pancreas Club Award
3:45pm - 3:50pm
• † 59. CYSTIC PANCREATIC NEUROENDOCRINE NEOPLASMS: A MULTICENTER INTERNATIONAL COHORT STUDY
Laura Maggino MD | University of Verona

3:50pm - 4:05pm
60. LOCALIZED INTRA-ARTERIAL GEMCITABINE: IMPACT ON SURVIVAL IN PATIENTS WITH LAPC—A NEW TREATMENT PARADIGM
Peter Muscarella MD | RenovoRx

4:05pm - 4:20pm
+ * 61. GROWTH RATE AND CYST STABILITY REDEFINE THE RISK OF CANCER IN BRANCH DUCT IPMN OF THE PANCREAS: INTRODUCING “TRIVIAL IPMNS”
Giovanni Marchegiani MD, PhD | University of Verona

4:20pm - 4:25pm
• † 62. POSTOPERATIVE DEEP VEIN THROMBOSIS AFTER PANCREATECTOMY FOR CANCER: A PERSISTENT PROBLEM LACKING A MECHANISTIC SOLUTION
Hunter Moore MD, PhD | University of Colorado

4:25pm - 4:40pm
• † 63. NEGATIVE PRESSURE WOUND THERAPY FOR SURGICAL-SITE INFECTIONS: A RANDOMIZED TRIAL
Ammar Javed MD | Johns Hopkins University School of Medicine

† John Howard Research Award  • Nikki Mitchell Award  • PanCan Research Award  † Pancreas Club Award
4:40pm - 4:55pm
**64. CLINICAL OUTCOME OF ENDOSCOPIC TREATMENT OF SYMPTOMATIC STERILE WALLED-OFF NECROSIS**
Rogier Voermans MD | Academic Medical Center

4:55pm - 5:10pm
**65. LONG AND SHORT-TERM OUTCOMES AFTER DISTAL PANCREATECTOMY WITH CELIAC AXIS RESECTION FOR PANCREATIC CANCER IN 582 PATIENTS: RETROSPECTIVE COHORT STUDY IN JAPAN**
Toru Nakamura MD, PhD | Hokkaido University Faculty of Medicine

5:00pm – 6:00pm | Sunset Terrace
**Awards Reception**
POSTER LIST

Posters of Distinction
P 1. MEDICAL CABINET ONCOLOGY: DISULFIRAM AND COPPER GLUCONATE SHOW SIMILAR EFFICACY IN PANCREATIC CANCER CELL LINES AND PATIENT-DERIVED XENOGRAFT TISSUE
Presenter: Jennifer Leiting MD
JL Leiting, MC Hernandez, L Yang, MJ Truty
Mayo Clinic Rochester

P 2. IMPACT OF THE INTRODUCTION OF FOLFIRINOX ON VARIATION IN THE USE OF CHEMOTHERAPY IN PATIENTS WITH METASTATIC PANCREATIC CANCER: A POPULATION-BASED ANALYSIS
Presenter: Stijn van Roessel MD, MSc
AEJ Latenstein, TM Mackay, LG van der Geest, HWM van Laarhoven, AJ Ten Tije, GJ Creemers, J de Vos-Geeelen, JW de Groot, MY Homs, CJH van Eijck, IQ Molenaar, N Haj Mohammad, MG Besselink, JW Wilmink
Amsterdam UMC

P 3. FACTORS PREDICTING FAILURE OF PERCUTANEOUS DRAINAGE IN THE “STEP-UP APPROACH” FOR ACUTE NECROTIZING PANCREATITIS: A 10-YEAR RETROSPECTIVE OBSERVATIONAL STUDY WITH A “PROACTIVE” PERCUTANEOUS DRAINAGE STRATEGY
Presenter: Rajesh Gupta MS, MCh
RG Gupta, A Kulkarni, RY Baby, S Shenvi, RA Gupta, R Nimje, GS Sharma, PV Vaswani, RD Sriram, HS Singh, VS Sharma, SS Rana, M Kang
Postgraduate Institute of Medical Education and Research Chandigarh

Presenter: Stefano Crippa MD, PhD
SCrippa, R Valente, AFogliati, U Arnello, A Halimi, Z Ateeb, ELongo, F Aleotti, PArcidiacono, M Lohr, MFalconi, MDel Chiaro
San Raffaele Scientific Institute

**POSTER LIST**

**Posters of Distinction**
P 5. AN EPIGENETIC CLASSIFICATION OF PANCREATIC DUCTAL ADENOCARCINOMA IDENTIFIES UNIQUE SUBTYPES ASSOCIATED WITH CLINICAL OUTCOME
Presenter: Victoria Aveson MD
VG Aveson, R Chandwani
New York Presbyterian - Weill Cornell Medical Center

P 6. DOES THE MICROBIOLOGY OF BACTIBILIA DRIVE POSTOPERATIVE COMPLICATIONS AFTER PANCREATODUODENECTOMY?
Presenter: Thomas Maatman MD
TK Maatman, DJ Weber, B Qureshi, EP Ceppa, A Nakeeb,
CM Schmidt, NJ Zyromski, MG House
Indiana University School of Medicine

P 7. PREOPERATIVE BILIARY STENTING IS ASSOCIATED WITH HIGHER POSTOPERATIVE MORBIDITY AND EQUIVALENT MORTALITY IN PATIENTS WITH MODERATE AND SEVERE HYPERBILIRUBINEMIA - AN ANALYSIS OF THE GERMAN STUDIOQLPANCREAS REGISTRY
Presenter: Louisa Bolm MD
L Bolm, E Petrova, L Woehrmann, J Werner, W Uhl, N Nuessler,
M Ghadimi, D Bausch, H Lapshyn, J Geadcke, O Belyaev, JG D’Haese,
T Klier, T Keck, UF Wellner
University Medical Center Schleswig-Holstein

P 8. PHASE II CLINICAL TRIAL USING NOVEL PEPTIDE VACCINE COCKTAIL COMBINED WITH GEMCITABINE FOR SURGICALLY RESECTED PANCREATIC CANCER PATIENTS
Presenter: Motoki Miyazawa MD, PhD
M Miyazawa, M Katsuda, M Kawai, S Hirono, K Okada, Y Kitahata,
R Kobayashi, M Ueno, S Hayami, H Yamaue
Wakayama Medical University
P 9. NOVEL PREOPERATIVE PATIENT-CENTERED SURGICAL WELLNESS PROGRAM IMPACTS LENGTH OF STAY FOLLOWING PANCREATECTOMY
Presenter: Danielle DePeralta MD
Indiana University School of Medicine

P 10. DRAIN MANAGEMENT FOLLOWING DISTAL PANCREATECTOMY: CHARACTERIZATION OF CONTEMPORARY PRACTICE AND IMPACT OF EARLY REMOVAL
Presenter: Laura Maggino MD
LMaggino, T Seykora, JB Liu, HA Pitt, CM Vollmer Jr
University of Pennsylvania

P 11. THE EFFICACY OF THE CDK 4/6 INHIBITOR (ABEMACICLIB) IN PANCREATIC CANCER CELLS
Presenter: Teena Dhir MD
TDhir, C Schultz, A Jain, CJ Yeo, T Golan, JR Brody
Thomas Jefferson University

P 12. SHOULD DIFFUSE MAIN-DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPASMS BE TREATED WITH TOTAL PANCREATECTOMY?
Presenter: Alex Blair MD
AB Blair, RM Beckman, JF Griffin, VP Groot, J Yu, MA Makary, RA Burkhart, MJ Weiss, JL Cameron, CL Wolfgang, J He
Johns Hopkins University School of Medicine

P 13. PERCEPTION VS. REALITY: A NATIONAL ANALYSIS OF THE SURGERY-FIRST APPROACH FOR PANCREATIC CANCER
Presenter: John Bergquist MD
JR Bergquist, CL Shubert, CA Thiels, EB Habermann, RL Smoot, ML Kendrick, DM Nagorney, MJ Truty
Mayo Clinic Rochester

Posters of Distinction
P 14. VISCERAL ARTERY PSEUDOANEURYSM IN NECROTIZING PANCREATITIS: INCIDENCE AND OUTCOMES
**Presenter:** Thomas Maatman MD
TK Maatman, MA Heimberger, KA Lewellen, AM Roch, CL Colgate, EP Ceppa, MG House, A Nakeeb, CM Schmidt, NJ Zyromski
Indiana University School of Medicine

P 15. UPDATED ALTERNATIVE FISTULA RISK SCORE TO INCLUDE MINIMALLY-INVASIVE PANCREATODUODENECTOMY: PAN-EUROPEAN VALIDATION
**Presenter:** Timothy Mungroop MD
Academic Medical Center

P 16. SERUM CA19-9 RESPONSE TO NEOADJUVANT CHEMOTHERAPY IS PREDICTIVE OF TUMOR SIZE REDUCTION AND SURVIVAL IN PANCREATIC ADENOCARCINOMA
**Presenter:** Melissa Hogg MD
AI Al Abbas, MS Zenati, CJ Reiser, AB Hamad, JP Jung, AH Zureikat, HJ Zeh III, ME Hogg
University of Pittsburgh Medical Center

P 17. SMAD4 LOSS IS A PREDICTOR OF NEOADJUVANT TREATMENT RESPONSE IN PANCREATIC DUCTAL ADENOCARCINOMA
**Presenter:** Rajesh Ramanathan MD
R Ramanathan, J Hodges, AI Al-Abbass, A Singhi, H Zeh, ME Hogg, AH Zureikat
University of Pittsburgh Medical Center
P 18. STAGING OF PANCREATIC DUCTAL ADENOCARCINOMA AFTER NEOADJUVANT THERAPY: REAPPRAISAL OF THE PROGNOSTIC ACCURACY OF THE 8TH EDITION OF AMERICAN JOINT COMMISSION ON CANCER STAGING SYSTEM

Presenter: Elisa Bannone MD
E Bannone, G Malleo, L Maggino, F Casciani, S Paiella, G Marchegiani, C Bassi, R Salvia
University of Verona

P 19. PANCREATIC FISTULA IN THE ERA OF NEOADJUVANT CHEMOTHERAPY IS AN UNCOMMON COMPLICATION BUT MAY HAVE MAJOR IMPACT ON LONG-TERM SURVIVAL

Presenter: Thomas Hank MD
T Hank, M Sandini, CR Ferrone, C Rodrigues, M Weniger, M Qadan, AL Warshaw, KD Lillemoe, C Fernández-del Castillo
Massachusetts General Hospital

P 20. DOES PREOPERATIVE PHARMACOLOGIC PROPHYLAXIS REDUCE THE RATE OF VENOUS THROMBOEMBOLISM IN PANCREATECTOMY PATIENTS?

Presenter: Zhi Ven Fong MD, MPH
ZV Fong, G del Carmen, DC Chang, CR Ferrone, C Fernandez-del Castillo, KD Lillemoe, M Qadan
Massachusetts General Hospital

P 22. DEVELOPMENT OF RISK PREDICTION PLATFORM FOR PANCREATIC FISTULA AFTER PANCREATECTOMY USING ARTIFICIAL INTELLIGENCE

Presenter: In Woong Han MD, PhD
IW Han, N Kim, Y Ryu, DJ Park, SH Shin, JS Heo, DW Choi, BH Cho
Samsung Medical Center
P 25. ZEBRAFISH EMBRYO AS AVATAR OF PATIENTS WITH PANCREATIC CANCER: PRELIMINARY EXPERIENCE TOWARD A PERSONALIZED MEDICINE
Presenter: Matteo Palmeri MD
G Di Franco, A Usai, M Palmeri, N Furbetta, D Gianardi, S Guadagni, M Bianchini, E Vasile, S Latteri, A Falcone, V Raffa, L Morelli
University of Pisa

P 26. CONDITIONAL SURVIVAL IN PATIENTS WITH RESECTABLE PANCREATIC CANCER: A DUTCH POPULATION-BASED STUDY
Presenter: Stijn van Roessel MD, MSc
AEJ Latenstein, LG van der Geest, RM van Dam, B Groot Koerkamp, IHJT de Hingh, MY Homs, JM Klaase, L Kwakkenbos, V Lemmens, IQ Molenaar, MJW Stommel, JW Wilmink, MG Besselink
Amsterdam UMC

P 29. MAIN DUCT DILATATION IS THE BEST PREDICTOR OF HIGH GRADE DYSPLASIA OR INVASION IN INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS
Presenter: Ross Beckman MD
M Del Chiaro, RM Beckman, Z Ateeb, N Orsini, N Rezaee, L Manos, R Valente, C Yuan, D Ding, GA Margonis, L Yin, JL Cameron, MA Makary, RA Burkhart, MJ Weiss, J He, U Arnelo, J Yu, CL Wolfgang
Johns Hopkins University School of Medicine

P 30. LAPAROSCOPIC DISTAL PANCREATECTOMY IS ASSOCIATED WITH A COST SAVINGS IN HIGH VOLUME CENTERS
Presenter: Emanuel Eguia MD, MHA
E Eguia, PC Kuo, P Sweigert, MC Nelson, GV Aranha, G Abood, CV Godellas, MS Baker
Loyola University Medical Center
P 32. RECEIPT OF ADJUVANT THERAPY AMONG PANCREATIC CANCER PATIENTS WITH A HIGH AREA OF DEPRIVATION INDEX  
**Presenter:** Susan Tsai MD  
J Mora, AN Krepline, I Akinola, KK Christians, CN Clarke, B George, PS Ritch, WA Hall, BA Erickson, K Dua, M Griffin, M Holt, DB Evans, S Tsai  
Medical College of Wisconsin

P 34. IMPACT OF PANCREATIC ENZYME REPLACEMENT THERAPY IN THE OUTCOME OF PATIENTS WITH CARCINOMA OF PANCREAS: PROSPECTIVE RANDOMISED STUDY  
**Presenter:** Shantanu Shantanu MS  
S Shantanu, R Gupta, S Rana, R Nada, H Singh, S Rana  
Postgraduate Institute of Medical Education and Research Chandigarh

P 35. ASSOCIATION BETWEEN PREOPERATIVE VASOSTATIN-1 AND PATHOLOGICAL FEATURES OF AGGRESSIVENESS IN LOCALIZED NONFUNCTIONING PANCREATIC NEUROENDOCRINE TUMORS (NF-PANNET)  
**Presenter:** Valentina Andreasi MD  
V Andreasi, S Partelli, M Manzoni, F Muffatti, M Mazza, B Colombo, A Corti, M Falconi  
San Raffaele Scientific Institute

P 36. ELUCIDATING THE CAUSES OF IMPROVED SURVIVAL IN RECENT RANDOMIZED ADJUVANT PANCREATIC DUCTAL ADENOCARCINOMA (PDAC) CLINICAL TRIALS  
**Presenter:** Andre Alabd BS  
A Alabd, O Bolaji, AG Alabd, J Ammori, J Hardacre, J Winter  
Thomas Jefferson University
P 37. IMPACT OF R0 RESECTIONS AFTER NEOADJUVANT TREATMENT AND PANCREATODUODENECTOMY IN PATIENTS WITH PANCREATIC CANCER
Presenter: Shimpei Maeda MD, PhD
Tohoku University Graduate School of Medicine

P 38. COMPLETION OF ADJUVANT CHEMOTHERAPY FOLLOWING UPFRONT SURGICAL RESECTION FOR PANCREATIC CANCER MAY BE TOO LOFTY A GOAL
Presenter: Keith Wirth MD
AM Altman, K Wirth, SMarmor, K Chang, E Lou, JYC Hui, TM Tuttle, EH Jensen, JW Denbo
University of Minnesota

P 39. PANCREATODUODENECTOMY FOLLOWING ROUX-EN-Y GASTRIC BYPASS: OPERATIVE CONSIDERATIONS AND OUTCOMES
Presenter: Maxwell Trudeau BS
MT Trudeau, L Maggino, BL Ecker, CM Vollmer
University of Pennsylvania

P 40. OPTIMAL PANCREATIC SURGERY: ARE WE MAKING PROGRESS?
Presenter: Joal Beane MD
JD Beane, JD Borrebach, AH Zureikat, EM Kilbane, VJ TThompson, HA Pitt
University of Pittsburgh Medical Center

P 42. COLLAGEN XVII AND VII SIGNALING COMPLEX IN NEOPLASTIC AND STROMAL CROSS-TALK IN PANCREATIC CANCER
Presenter: Annie Li
A Li, TP Hank, KC Honselmann, DJ Birnbaum, SKS Begg, KD Lillemoe, AL Warshaw, C Fernández-del Castillo, AS Liss
Massachusetts General Hospital
P 43. MEK INHIBITOR TRAMETINIB IN COMBINATION WITH GEMCITABINE REGRESSES A PATIENT-DERIVED ORTHOTOPIC XENOGRAFT (PDOX) PANCREATIC CANCER NUDE MOUSE MODEL
Presenter: Kei Kawaguchi MD, PhD
K Kawaguchi, M Bouvet, M Unno, RM Hoffman
Tohoku University Graduate School of Medicine

P 44. KRAS-MUTATED CIRCULATING TUMOR DNA PREDICTS EARLY RECURRENT IN PATIENTS WITH PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Takuro Yamaguchi MD
T Yamaguchi, K Uemura, Y Murakami, N Kondo, N Nakagawa,
K Okada, S Seo, S Takahashi, T Sueda
Hiroshima University

P 45. LONG-TERM SEQUELAE OF NECROTIZING PANCREATITIS: A LIFE-LONG DISEASE
Presenter: Thomas Maatman MD
TK Maatman, AM Roch, EP Ceppa, MG House, A Nakeeb,
CM Schmidt, NJ Zyromski
Indiana University School of Medicine

P 46. NONSELECTIVE B-ADRENERGIC BLOCKADE IMPACTS PANCREATIC CANCER TUMOR BIOLOGY, DECREASES PERINEURAL INVASION AND IMPROVES PATIENT SURVIVAL
Presenter: Alex Blair MD
AB Blair, N Jurcak, A Javed, J Teinor, VP Groot, N Rozich, JL Cameron,
MJ Weiss, J He, CL Wolfgang, L Zheng, RA Burkhart
Johns Hopkins University School of Medicine

P 47. EVALUATION OF CIRCUMFERENTIAL RESECTION MARGINS FOR PANCREATIC HEAD CANCER: A PROSPECTIVE STUDY WITH COMPARISON OF 0MM VERSUS 1MM RULES FOR R1 RESECTION
Presenter: Yoo-Seok Yoon MD, PhD
Y-S Yoon, H Kim, Y Lee, H-S Han, S Ahn, J-Y Choe, Y Lee
Seoul National University Bundang Hospital
P 48. TUMOR BUDDING MORPHOMIC ANALYSIS REVEALS EMT AND PROGNOSTIC INFORMATION IN PERIAMPULLARY CANCER
Presenter: Ulrich F. Wellner MD
UF Wellner, E Kocsmar, A Kiss, G Lotz, M Hoerner, E Petrova, S Timme, A Csanadi, M Werner, T Keck, P Bronsert
University Medical Center Schleswig-Holstein

P 49. FACTORS ASSOCIATED WITH DISEASE PROGRESSION OR PERFORMANCE STATUS DECLINE IN PATIENTS UNDERGOING NEOADJUVANT CHEMOTHERAPY FOR LOCALIZED PANCREATIC HEAD ADENOCARCINOMA
Presenter: Alessandro Paniccia MD
A Paniccia, AL Gleisner, MS Zenati, AI Al Abbas, R Schlabach, KKW Lee, ME Hogg, H Zeh, AH Zureikat
University of Pittsburgh Medical Center

P 51. EXTREMES OF BMI ARE ASSOCIATED WITH A HIGHER RISK OF PANCREATIC FISTULA FOLLOWING PANCREATICODUODENECTOMY: AN ANALYSIS USING THE NSQIP DATABASE
Presenter: Shravan Leonard-Murali MD
S Leonard-Murali, T Ivanics, A Tang, CP Steffes, RA Shah, DS Kwon
Henry Ford Hospital

P 52. TREATMENT SEQUENCING STRATEGIES AND SURVIVAL IN OCTOGENARIANS WITH EARLY STAGE PANCREATIC HEAD ADENOCARCINOMA: A NATIONAL CANCER DATABASE ANALYSIS
Presenter: Tommy Ivanics MD
T Ivanics, S Leonard-Murali, X Han, C Steffes, R Shah, D Kwon
Henry Ford Hospital
P 53. NEOADJUVANT THERAPY FOR BODY AND TAIL PANCREATIC ADENOCARCINOMA: PROPENSITY SCORE MATCHED ANALYSIS USING THE NATIONAL CANCER DATABASE
Presenter: Tommy Ivanics MD
T Ivanics, S Leonard-Murali, X Han, C Steffes, D Kwon, R Shah
Henry Ford Hospital

P 55. SURGICAL RESECTION IS ASSOCIATED WITH IMPROVED SURVIVAL FOR SMALL, MODERATE AND WELL DIFFERENTIATED PANCREATIC NEUROENDOCRINE TUMORS
Presenter: Waseem Lutfi BS
W Lutfi, E Eguia, S Sharpe, G Abood, G Aranha, CV Godellas, PC Kuo, MS Baker
Loyola University Medical Center

P 57. MANAGEMENT OF SMALL ASYMPTOMATIC NONFUNCTIONING PANCREATIC NEUROENDOCRINE TUMORS: FROM GUIDELINES TO REAL LIFE
Presenter: Michele Mazza MD
M Mazza, S Partelli, M Falconi
San Raffaele Scientific Institute

P 58. IS PANCREATIC CANCER DIFFERENT ACCORDING TO THE LOCATION?; A SURVIVAL AND PROGNOSTIC FACTOR ANALYSIS
Presenter: Mirang Lee MD
M Lee, W Kwon, Y Byun, J Kim, H Kim, JY Jang, SW Kim
Seoul National University Hospital

P 59. IS THE NEW T1 CATEGORY DEFINED BY THE 8TH EDITION AJCC PANCREATIC CANCER STAGING SYSTEM AN IMPROVEMENT? AN EVALUATION BY MULTINATIONAL DATA
Presenter: Wooil Kwon MD, PhD
W Kwon, J He, R Higuchi, D Son, SY Lee, M Lee, J Kim, SW Kim, CL Wolfgang, JL Cameron, M Yamamoto, JY Jang
Seoul National University Hospital
P 60. WHERE IS THE APPROPRIATE BORDERLINE? -THE POOR PROGNOSIS IN RESECTABLE PANCREATIC CANCER WITH HAZY DENSITY
Presenter: Tomotaka Kato MD
T Kato, D Ban, T Ogura, K Ogawa, H Ono, Y Mistunori, A Kudo, M Tanabe
Tokyo Medical and Dental University

P 61. FUNCTIONAL OUTCOMES AFTER EXTRAHEPATIC TRANSPLANTATION OF ISLETS IN A PILOT COHORT OF TOTAL PANCREATECTOMY WITH ISLET AUTOTRANSPLANTATION (TPIAT) PATIENTS
Presenter: Kendall McEachron MD
KR McEachron, GJ Beilman, TL Pruett, TB Dunn, S Chinnakotla, MD Bellin
University of Minnesota

P 62. PREOPERATIVE SERUM LEVELS OF PHOSPHORUS PREDICTS OCCURRENCE AND SEVERITY OF POST-OPERATIVE PANCREATIC FISTULA AFTER PANCREATEDUDENECTOMY
Presenter: Niccolò Napoli MD
S Iacopi, N Napoli, C Lombardo, F Menonna, EF Kauffmann, C Cacace, A Natali, M Miccoli, U Boggi
University of Pisa

P 63. ASSESSING THE IMPACT OF UNDERLYING LIVER DISEASE ON 30-DAY POSTOPERATIVE OUTCOMES AFTER PANCREATECTOMY: AN ACS NSQIP® ANALYSIS
Presenter: Amer Zureikat MD
AI Al Abbas, JD Borrebach, J Bellon, AH Zureikat
University of Pittsburgh Medical Center

P 64. EFFECT OF SILENCING RAB23 GENE WITH SIRNA ON PROLIFERATION AND APOPTOSIS OF PANC-1 CELLS
Presenter: Yunjian Liu PhD
Y Liu, H Zhou, T Yu, J Zhang, X Zeng
Indiana University School of Medicine
P 65. REPURPOSING THE FDA APPROVED DRUG PYRVINIUM PAMOATE TO TARGET HUR IN PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Teena Dhir MD
CW Schultz, T Dhir, AO Haber, W Jiang, S Chand, SZ Brown, EF Petricoin, CJ Yeo, JR Brody
Thomas Jefferson University

P 66. FATTY ACID BINDING PROTEIN 4 PROMOTES PANCREATIC CANCER GROWTH VIA ALTERED REDOX SIGNALING
Presenter: Keith Wirth MD
K Wirth, S Kizy, A Sheka, S Ikraumuddin, D Bernlohr, M Yamamoto
University of Minnesota

P 67. SYSTEMIC TREATMENT WITH MESOTHELIN-TARGETED ONCOclytic ADENOVIRUS SHOWS EFFICACY PATIENT-DERIVED XENOGRAFT OF PANCREATIC CANCER
Presenter: Masato Yamamoto MD, PhD
M Sato-Dahlman, Y Miura, P Hajeri, H Yoshida, K Jacobsen, C Yanagiba, M Yamamoto
University of Minnesota

P 68. CAN MORTALITY OF OPERATIVE PANCREATIC DEBRIDEMENT BE PREDICTED ACCURATELY?
Presenter: Alexandra Roch MD
AM Roch, TK Maatman, EM Kilbane, EP Ceppa, MG House, A Nakeeb, CM Schmidt, NJ Zyromski
Indiana University School of Medicine

P 69. INSULIN PROMOTES THE INVASION AND MIGRATION OF PANCREATIC CANCER THROUGH THE EMT PATHWAY
Presenter: Zipeng Lu MD, PhD, MRCS
Y Gao, H Gao, G Wang, B Cai, Z Lu, Y Miao
Pancreas Center, The First Affiliated Hospital with Nanjing Medical University
P 70. WNT/BETA-CATENIN SIGNALING PLAYS MULTIPLE ROLES DURING THE PROCESS OF FIBROTIC REMODELING IN THE EXOCRINE PANCREAS
Presenter: Anu Aronen MD
M Bläuer, M Laaninen, J Sand, J Laukkaninen
Tampere University Hospital

P 71. PERSONALIZED MEDICINE FOR PANCREATIC FISTULA AFTER PANCREATEODUODENECTOMY: TARGETING IMPROVEMENT THROUGH RISK PROFILING
Presenter: Maxwell Trudeau BS
MT Trudeau, BL Ecker, L Maggino, MT Mcmillan, CM Vollmer Jr
University of Pennsylvania

P 72. PATIENT PERCEPTIONS OF THE POST-PANCREATECTOMY DISCHARGE PROCESS
Presenter: Rodrigo Calvillo-Ortiz MD
R Calvillo-Ortiz, AA Watkins, M Castillo-Angeles, L Anguiano-Landa, MP Callery, JA Moser, TS Kent
Beth Israel Deaconess Medical Center

P 73. PERIOPERATIVE PANCREATIC ENZYME REPLACEMENT THERAPY REDUCES MORBIDITY IN PATIENTS UNDERGOING PANCREATEODUODENECTOMY
Presenter: Amit Khithani MD
AS Khithani, TB Cengiz, K El-Hayek, T Augustin, RA Simon, RM Walsh, GJ Morris-Stiff
Cleveland Clinic Foundation

P 74. IMPACT OF BLOOD TRANSFUSIONS ON SHORT-TERM OUTCOMES AND OVERALL SURVIVAL IN PATIENTS UNDERGOING PANCREATIC SURGERY FOR MALIGNANCY
Presenter: Essa M. Aleassa MD, MSc
M Chumakova, EA Aleassa, G Morris-Stiff
Cleveland Clinic Foundation
**P 75. PROGNOSTIC SIGNIFICANCE OF TUMOR BUDDING IN PANCREATIC CANCER**
**Presenter:** Ekaterina Petrova MD
E Petrova, V Zielinski, L Bolm, J Knief, D Bausch, C Thorns, T Keck, U Wellner
University Clinic Schleswig-Holstein (UKSH) Campus Lübeck, Germany

**P 77. NEOADJUVANT THERAPY VERSUS UPFRONT SURGERY FOR RESECTABLE PANCREATIC CANCER: A SYSTEMATIC REVIEW AND META-ANALYSIS**
**Presenter:** Gennaro Nappo MD
G Nappo, S Bozzarelli, T Comito, F Gavazzi, C Ridolfi, G Capretti, J Galvanin, G Donisi, L Rimassa, A Zerbi
Humanitas University

**P 78. SOLID PSEUDOPAPILLARY NEOPLASMS OF THE PANCREAS: AN EXTENDED ANALYSIS OF A SINGLE INSTITUTION SERIES AFTER PANCREATIC RESECTION**
**Presenter:** Yayun Zhu MD
Y Zhu, T Saunders, L Yin, J Griffin, N Pu, H Hu, RA Burkhart, MA Makary, JL Cameron, MJ Weiss, CL Wolfgang, J He, J Yu
Johns Hopkins University School of Medicine

**P 79. SEPSIS IS THE STRONGEST DRIVER OF AVOIDABLE IN-HOSPITAL COST IN PATIENTS UNDERGOING PANCREATICODUODENECTOMY**
**Presenter:** Emanuel Eguia MD, MS, MHA
E Eguia, PJ Sweigert, M Nelson, F Luchette, M Afshar, MS Baker
Loyola University Medical Center
P 80. LYMPH NODE ASSESSMENT INFORMS PROGNOSIS BUT EXTENT OF LYMPHADENECTOMY IS NOT ASSOCIATED WITH SURVIVAL IN PATIENTS UNDERGOING RESECTION FOR PANCREATIC NEUROENDOCRINE TUMORS

Presenter: Emanuel Eguia MD, MS, MHA
E Eguia, P Sweigert, M Afshar, GV Aranha, G Abood, C Godellas, PC Kuo, MS Baker
Loyola University Medical Center

P 82. IMPACT OF ADJUVANT TREATMENT FOLLOWING SURGERY FOR INVASIVE INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS OF THE PANCREAS

Presenter: Clifton Rodrigues MD
C Rodrigues, D Ciprani, T Hank, M Weniger, M Qadan, C Ferrone, AL Warshaw, KD Lillemoe, C Fernandez-Del Castillo
Massachusetts General Hospital

P 83. DOES PRACTICE MAKE PERFECT FOR PANCREATIC ADENOCARCINOMA? HIGHER VOLUME CENTERS DELIVERING CHEMORADIATION ARE ASSOCIATED WITH IMPROVED SURGICAL AND SURVIVAL OUTCOMES

Presenter: Lindsay Bliss MD, MPH
LA Bliss, AN Krepline, CA Barnes, KK Christians, B George, PS Ritch, BA Erickson, DB Evans, WA Hall, S Tsai
Medical College of Wisconsin

P 84. IMPLICATIONS OF PERINEURAL INVASION ON DISEASE RECURRENCE AND SURVIVAL FOLLOWING PANCREATECTOMY FOR PANCREATIC HEAD DUCTAL ADENOCARCINOMA

Presenter: Stefano Crippa MD, PhD
S Crippa, I Pergolini, AA Javed, KC Honselmann, MJ Weiss, F Di Salvo, R Burkhart, CR Ferrone, G Zamboni, J Yu, G Belfiori, M Qadan, C Rubini, J He, KD Lillemoe, C Fernandez-del Castillo, CL Wolfgang, M Falconi
San Raffaele Scientific Institute
P 85. PREVALENCE OF PANCREATIC SECRETORY TRYPsin INHIBITOR GENE MUTATION IN PATIENTS WITH ACUTE ALCOHOL PANCREATITIS COMPARED TO ALCOHOLICS AND HEALTHY CONTROLS
Presenter: Anssi Nikkola MD
A Nikkola, KH Herzig, K Mäkelä, SJ Mutt, T Lehtimäki, M Kähönen, O Raitakari, I Seppälä, P Paakkanen, H Seppänen, I Nordback, J Sand, J Laukkarinen
Tampere University Hospital

P 86. LONG-TERM OUTCOME OF CONVERSION SURGERY FOR INITIALLY UNRESECTABLE PANCREATIC CANCER: A SINGLE INSTITUTIONAL EXPERIENCE
Presenter: Keita Wada MD, PhD
K Wada, K Sano, F Miura, M Shibuya, M Kainuma, K Takahashi, S Kawamura
Teikyo University School of Medicine

P 87. UNIQUE STEATOSIS PATTERNS ON CONTRAST ENHANCED COMPUTED TOMOGRAPHY AFTER TOTAL PANCREATECTOMY AND ISLET AUTO-TRANSPLANTATION
Presenter: Alexandria Coughlan MD
AJ Coughlan, R Schat, B Spilseth, ME Skube, M Bellin, M Stice, GJ Beilman
University of Minnesota

P 88. IDENTIFYING PRETREATMENT PREDICTORS OF NEOADJUVANT CHEMOTHERAPY BENEFIT IN LOCALIZED PANCREATIC HEAD CANCER: A 15-YEAR EXPERIENCE AT A HIGH VOLUME PANCREATIC CANCER CENTER
Presenter: Amer Zureikat MD
AI Al Abbas, J Hodges, P Varley, C Bucholz, J Bellon, N Bahary, A Singhi, ME Hogg, HJ Zeh III, AH Zureikat
University of Pittsburgh Medical Center
P 90. HEMOGLOBIN A1C AS A MARKER TO STRATIFY DIABETES RISK FOLLOWING PANCREATICODUODENECTOMY
Presenter: Josh Bleicher MD, MS
J Bleicher, HM Shepherd, CL Scaife
University of Utah

P 91. DIAGNOSTIC POTENTIAL OF CT TEXTURE ANALYSIS FOR PANCREATIC CYSTIC LESIONS
Presenter: Victoria Rendell MD
VR Rendell, AM Awe, BM Hanlon, N Marka, PJ Pickhardt, MG Lubner, ER Winslow
University of Wisconsin

P 92. INTENSIVE PERIOPERATIVE REHABILITATION IMPROVES SURGICAL OUTCOMES AFTER PANCREATICODUODENECTOMY
Presenter: Yuji Kitahata MD, PhD
Y Kitahata, M Kawai, S Hirono, K Okada, M Miyazawa, R Kobayashi, H Yamaue
Wakayama Medical University

P 95. IMPACT OF PERIOPERATIVE HYPERGLYCEMIA ON POST-OPERATIVE MORBIDITY AND OVERALL SURVIVAL IN PATIENTS UNDERGOING ONCOLOGIC PANCREATOBILIARY SURGERY
Presenter: Essa M. Aleassa MD
M Chumakova, EA Aleassa, G Morris-Stiff
Cleveland Clinic Foundation

P 98. COMPARISON OF PANCREATIC ADENOCARCINOMA LIVER METASTASIS DETECTION BETWEEN COMPUTER TOMOGRAPHY AND MAGNETIC RESONANCE IMAGING
Presenter: Armando Rosales MD
A Rosales, A Stoylen, A Wallace, H Asbun, L Tsamalaidze, JA Stauffer
Mayo Clinic Florida
P 99. PORTOMESENTERIC VENOUS COMPLICATIONS FOLLOWING PANCREATIC RESECTION: TOWARD DEVELOPMENT OF A CLINICAL SCORING SYSTEM TO PREDICT INCIDENCE, SEQUELAE AND MANAGEMENT

Presenter: Jennifer Leiting MD
L Yohanathan, SM Thompson, WS Harmsen, RL Smoot, MJ Truty, TE Grotz, SP Cleary, DM Nagorney, JC Andrews, ML Kendrick
Mayo Clinic Rochester

P 100. ACINAR CELL CARCINOMA OF THE PANCREAS: A HISTOLOGICALLY AND EPIDEMIOLOGICALLY DISTINCT ENTITY

Presenter: Jennifer Yonkus MD
JA Yonkus, JR Bergquist, T Ivanics, TR Halfdanarson, RL Smoot, SP Cleary, DM Nagorney, ML Kendrick, MJ Truty
Mayo Clinic Rochester

P 101. IMPACT OF AREA OF DEPRIVATION INDEX ON HOSPITAL READMISSIONS AFTER SURGERY FOR PANCREAS CANCER

Presenter: Ashley Krepline MD
AN Krepline, J Mora, M Aldakkak, S Misustin, KK Christians, CN Clarke, B George, PS Ritch, WA Hall, BA Erickson, N Kulkarni, AH Khan, DB Evans, S Tsai
Medical College of Wisconsin

P 102. THE IMPACT OF SURGICAL RESECTION AFTER INITIAL CHEMORADIOThERAPy FOLLOWED BY SYSTEMIC CHEMOTHERAPy FOR LOCALLy ADVANCED PANCREATIC ADENOCARCINOMA

Presenter: Masashi Kishiwada MD, PhD
M Kishiwada, A Hayasaki, T Fujii, Y Iizawa, H Kato, A Tanemura, Y Murata, Y Azumi, N Kuriyama, S Mizuno, M Usui, H Sakurai, S Isaji
Mie University School of Medicine
P 103. A RETROSPECTIVE COHORT STUDY OF 4,739 PATIENTS WITH PANCREATIC NEUROENDOCRINE TUMORS IDENTIFIES PROLONGED SURVIVAL AFTER SURGICAL RESECTION EVEN IN ADVANCED STAGES
Presenter: Benjamin Powers MD
BD Powers, A Kumar, JB Fleming MD, JR Strosberg, DA Anaya
Moffitt Cancer Center

P 104. SUB-ADVENTITIAL DIVESMENT TECHNIQUE FOR ARTERY-INVOLVED PANCREATIC CANCER: SAFETY AND ONCOLOGICAL EFFICACY
Presenter: Kuirong Jiang MD, PhD
Y Miao, B Cai, Z Lu, K Zhang, L Yin, Y Gao, K Jiang
Pancreas Center, The First Affiliated Hospital with Nanjing Medical University

P 105. CLINICAL IMPLICATIONS OF AN UNPLANNED CONVERSION (UC) DURING MINIMALLY INVASIVE PANCREATICODUODENECTOMY (MIPD) – REVIEW OF AMERICAN COLLEGE OF SURGEONS NATIONAL SURGICAL QUALITY IMPROVEMENT PROGRAM (NSQIP) DATA FROM 2014 - 2016
Presenter: Andreas Schneider MD
AM Schneider, E Alonso, ES Tang, ST Chiu, ML Babicky, PH Newell, PD Hansen
Providence Portland Medical Center

P 108. SMALL PANCREATIC NEUROENDOCRINE TUMORS: RESECT OR ENUCLEATE
Presenter: Joal Beane MD
JD Beane, JD Borrebach, AH Zureikat, HA Pitt
University of Pittsburgh Medical Center
P 109. FACTORS ASSOCIATED WITH DELAYED GASTRIC EMPTYING AFTER PANCREATICODUODENECTOMY IN THE NATIONAL SURGICAL QUALITY IMPROVEMENT DATABASE
Presenter: Rebecca Snyder MD, MPH
RA Snyder, JA Ewing, AA Parikh
University of South Carolina School of Medicine- Greenville

P 110. COMPARISON OF SHORT- AND LONG-TERM OUTCOMES BETWEEN ANATOMICAL SUBTYPES OF RESECTED BILIARY TRACT CANCER IN A WESTERN HIGH-VOLUME CENTER
Presenter: Marin Strijker MD
E Roos, M Strijker, LC Franken, OR Busch, JE van Hooft, HJ Klümpen, HW van Laarhoven, JW Wilmink, J Verheij, TM van Gulik, MG Besselink
Amsterdam UMC

P 113. TKS5-POSITIVE INVADOPODIA-LIKE STRUCTURES IN Pancreatic Surgical Specimens AND Clinical Outcomes
Presenter: Kathryn Chen MD
KT Chen, YC Chen, M Baik, J Byers, SW French, B Diaz
Harbor-UCLA Medical Center

P 114. DRUG RESISTANCE IN INFECTED Pancreatic Necrosis: Cause for Alarm?
Presenter: Alexandra Roch MD
AM Roch, TK Maatman, S Jain, EP Ceppa, MG House, A Nakeeb, CM Schmidt, NJ Zyromski
Indiana University School of Medicine
P 116. DIFFICULTY SCORING SYSTEM IN LAPAROSCOPIC DISTAL PANCREATECTOMY
Presenter: Takao Ohtsuka MD, PhD
T Ohtsuka, D Ban, Y Nakamura, Y Nagakawa, M Tanabe, Y Gotoh, VVDM Velasquez, K Nakata, Y Sahara, K Takaori, G Honda, T Misawa, M Kawai, H Yamaue, T Morikawa, T Kuroki, Y Mou, WJ Lee, SV Shrikhande, CN Tang, C Conrad, HS Han, C Palanivelu, HJ Asbun, DA Koo
Kyushu University

P 121. ROLE OF NEOADJUVANT RADIATION IN DOWNSTAGING PATIENTS WITH LOCALIZEDPancreatic CanCER - ANALYSIS OF THE NCDB DATABASE
Presenter: Ashley Krepline MD
AN Krepline, CA Barnes, M Aldakkak, I Akinola, KK Christians, CN Clarke, B George, PS Ritch, WA Hall, BA Erickson, DB Evans, S Tsai
Medical College of Wisconsin

P 122. MULTIMODALITY NEOADJUVANT THERAPY INCREASES NORMALIZATION OF CARBOHYDRATE ANTIGEN 19-9 (CA19-9) IN PATIENTS WITH PANCREATIC CANCER
Presenter: Ashley Krepline MD
AN Krepline, CN Clarke, KK Christians, PS Ritch, B George, AH Khan, N Kulkarni, C Hagen, BA Erickson, WA Hall, M Aldakkak, DB Evans, S Tsai
Medical College of Wisconsin

P 125. THE EVOLUTION OF POSTOPERATIVE PANCREATIC FISTULA (POPF) SEVERITY ACCORDING TO GRADE B SUBCLASSIFICATION REFLECTS A STEP-UP MANAGEMENT
Presenter: Valentina Andreasi MD
V Andreasi, S Partelli, R Ariotti, G Guarneri, M Pagnanelli, G Balzano, S Crippa, M Falconi
San Raffaele Scientific Institute
P 126. RAPID GROWTH SPEED OF CYSTS CAN PREDICT MALIGNANT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS

Presenter: Keiichi Akahoshi MD, PhD
K Akahoshi, H Ono, M Akasu, D Ban, A Kudo, A Konta, S Tanaka, M Tanabe
Tokyo Medical and Dental University

P 127. IS THIS THE TIME FOR THE “GOLDEN AGE” OF EARLY PANCREATIC CANCER DETECTION? A NANOPARTICLE-BASED BLOOD TEST

Presenter: Damiano Caputo MD
D Caputo, L Di Giacomo, D Pozzi, S Palchetti, C Cascone, M Cartillone, R Coppola, G Caracciolo
University Campus Bio-Medico di Roma

P 128. PREOPERATIVE RADIOGRAPHIC MEASUREMENTS CAN PREDICT POSTOPERATIVE PANCREATIC FISTULA FORMATION FOLLOWING PANCREATEODUODENECTOMY

Presenter: Edmond Box BS
EW Box, L Deng, DE Morgan, R Xie, JK Kirklin, S Reddy, TN Wang, MJ Heslin, JB Rose
University of Alabama at Birmingham

P 129. PRELIMINARY TOXICITY FOR A PHASE II TRIAL OF ESCALATED DOSE PROTON RADIOTHERAPY WITH ELECTIVE NODAL IRRADIATION AND CONCOMITANT CHEMOTHERAPY FOR PATIENTS WITH UNRESECTABLE, BORDERLINE RESECTABLE OR MEDICALLY INOPERABLE PANCREATIC ADENOCARCINOMA

Presenter: Romaine Nichols MD
RC Nichols, CG Morris, D Pham, ME Shaikh, MS Rutenberg
University of Florida
P 131. A PROGNOSTIC SCORE FOR THE SELECTION OF PATIENTS WHO COULD BE CURED BY PANCREATECTOMY WITH SIMULTANEOUS ARTERIAL RESECTION DESPITE LOCALLY ADVANCED DUCTAL PANCREATIC ADENOCARCINOMA
Presenter: Niccolò Napoli MD
N Napoli, EF Kauffmann, S Iacopi, F Menonna, C Cacace, G Taddei, A Cacciato Insilla, C Cappelli, D Campani, D Caramella, F Vistoli, U Boggi
University of Pisa

P 133. IS THE ROUTINE USE OF SOMATOSTATIN AFTER PANCREATODUODENECTOMY JUSTIFIED? A CASE-MATCHED COMPARISON ACCORDING TO FISTULA RISK SCORE, ASA SCORE AND SURGICAL TECHNIQUE
Presenter: Matteo Palmeri MD
L Morelli, N Furbetta, S Guadagni, G Di Franco, M Palmeri, D Gianardi, M Bianchini, C D’isidoro, G Caprili, G Di Candio, F Mosca
University of Pisa

P 134. PANCREATIC RESECTIONS IN THE ELDERLY: A TERTIARY CARE CENTER ANALYSIS WITH PARTICULAR VIEW ON THE HIGH AMERICAN SOCIETY OF ANESTHESIOLOGISTS’ RISK SCORE PATIENTS
Presenter: Matteo Palmeri MD
L Morelli, G Di Franco, M Palmeri, S Guadagni, N Furbetta, D Gianardi, J Bronzoni, M Bianchini, G Stefanini, G Caprili, G Di Candio, F Mosca
University of Pisa

P 135. MANAGEMENT OF LIVER METASTASIS IN PATIENTS WITH PANCREATIC NEUROENDOCRINE TUMOR: SURGICAL OR NON-SURGICAL?
Presenter: Eiman Ghaffarpasand MD
E Ghaffarpasand, ASO Carranza, MP Callery, AJ Moser, TS Kent
Beth Israel Deaconess Medical Center, Harvard Medical School
P 136. TRENDS AND PREDICTORS OF TIME TO INITIATION OF TREATMENT FOR NON-METASTATIC PANCREATIC CANCER: A NATIONAL CANCER DATABASE ANALYSIS
Presenter: Essa M. Aleassa MD, MSc
EM Aleassa, E Brinza, G Morris-Stiff
Cleveland Clinic Foundation

P 137. ASSESSING THE ROLE AND DANGERS OF TOTAL PARENTERAL NUTRITION AFTER PANCREATODUODENECTOMY
Presenter: Alexandra Roch MD
AM Roch, KA Lewellen, TK Maatman, DK DePeralta, EP Ceppa, NJ Zyromski, CM Schmidt, A Nakeeb, MG House
Indiana University School of Medicine

P 138. SURVIVAL OUTCOMES OF PANCREATICODUODENECTOMY VERSUS EXTENDED PANCREATICODUODENECTOMY PROCEDURE FOR PANCREATIC HEAD CARCINOMA: A PROPENSITY SCORE MATCHING STUDY
Presenter: Ning Pu MD
N Pu, W Lou, J Yu
Johns Hopkins University School of Medicine

P 139. RISK FACTOR OF ARTERIAL PSEUDOANEURYSM AFTER PANCREATICODUODENECTOMY AND ITS OPTIMAL MANAGEMENT
Presenter: Hiroyuki Kato MD
H Kato, J Chipaila, D Noguchi, A Hayasaki, Y Iizawa, A Tanemura, Y Murata, Y Azumi, N Kuriyama, M Kishiwada, S Mizuno, M Usui, H Sakurai, S Isaji
Mie University School of Medicine
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P 140. NEUTROPHIL TO LYMPHOCYTE RATIO IS PREDICTIVE OF PATIENT SURVIVAL AFTER RESECTION OF PANCREATIC ADENOCARCINOMA
Presenter: David Pointer MD
DT Pointer, DJ Roife, BD Powers, G Murimwa, S Elessawy, PJ Hodul, J Pimiento, JB Fleming, MP Malafa
Moffitt Cancer Center

P 142. TEXTURE ANALYSIS OF CONTRAST ENHANCED CT IMAGING OF PANCREATIC NEUROENDOCRINE NEOPLASMS AND ITS CORRELATION WITH TUMOR GRADING
Presenter: Zipeng Lu MD, PhD, MRCS
JL Wu, WB Xu, ZP Lu, K Zhang, HY Shi, Q Xu, WT Gao, CC Dai, KR Jiang, Y Miao
Pancreas Center, The First Affiliated Hospital with Nanjing Medical University

P 143. PLASMA SUPAR IN DISTINGUISHING A MALIGNANT AND BENIGN PANCREATIC MASS
Presenter: Anu Aronen MD
A Aronen, J Aittoniemi, R Huttunen, A Nikkola, J Nikkola, O Limnell, J Sand, J Laukkanen
Tampere University Hospital

P 144. QUALITY OF LIFE AND PANCREATIC EXOCRINE AND ENDOCRINE FUNCTION IN THE LONG-TERM FOLLOW-UP AFTER PANCREATICODUODENECTOMY
Presenter: Ismo Laitinen MD
I Laitinen, I Rinta-Kiikka, Y Vaalavuo, A Antila, A Siiki, J Sand, J Laukkanen
Tampere University Hospital
P 145. PROGNOSTIC FACTORS IN PATIENTS UNDERGOING PANCREATODUODENECTOMY FOR AMPULLARY CANCER - A LARGE MULTICENTER COHORT STUDY OF MORE THAN 200 PATIENTS
Presenter: Louisa Bolm MD
L Bolm, K Ohrner, F Rückert, BM Rau, E Petrova, D Bausch, J Weitz, T Keck, UF Wellner, M Distler
University Medical Center Schleswig-Holstein

P 146. RISK STRATIFICATION FOR INTENSIVE CARE UNIT FOLLOWING PANCREATODUODENECTOMY
Presenter: Louisa Bolm MD
S Deichmann, UE Ballies, L Frohneberg, E Petrova, L Bolm, KC Honselmann, T Keck, UF Wellner, D Bausch
University Medical Center Schleswig-Holstein

P 147. PROGNOSTIC VALUE OF TP53, CDKN2A/P16 AND SMAD4/DPC4 IN LOCALLY ADVANCED, BUT RESECTED, PANCREATIC CANCER
Presenter: Niccolò Napoli MD
EF Kauffmann, N Napoli, A Cacciato Insilla, S Iacopi, F Menonna, C Cappelli, D Caramella, F Vistoli, D Campani, U Boggi
University of Pisa

P 148. ELEVATED BILIRUBIN LEVELS SHOULD NOT INFLUENCE TIMING OF PANCREATIC RESECTION
Presenter: Guido Alsfasser MD
G Alsfasser, I Kamaleddine, E Klar
University of Rostock

P 149. PANCREATIC DUCT CALIBER SHOULD NOT BE USED AS THE SOLE JUSTIFICATION FOR SURGICAL SELECTION IN MAIN DUCT IPMNS
Presenter: Tara Hughes MD
University of Texas MD Anderson Cancer Center
P 151. ISCHEMIC GASTROPATHY AFTER DISTAL PANCREATECTOMY WITH EN BLOC CELIAC AXIS RESECTION FOR PANCREATIC BODY CANCER
Presenter: Hiroki Yamaue MD
H Yamaue, K Okada, M Kawai, S Hirono, M Miyazawa, Y Kitahata, M Ueno, S Hayami
Wakayama Medical University

P 152. RNA-SCOPE TECHNOLOGY AND IMMUNOHISTOCHEMISTRY REVEAL LOW EXPRESSION OF MUC5AC IN METASTATIC PANCREATIC PATIENTS: EPIGENETIC PITFALLS OR A BETTER DISEASE STATUS?
Presenter: Niccola Funel PhD
N Funel, M Crisci, G Tasso, LE Pollina, M Palmeri, M Gentiluomo, G Di Franco, N Furbetta, D Gianardi, S Guadagni, M Bianchini, G Stefanini, C Santucci, V Nardini, L Morelli Luca
University of Pisa

P 154. DOES CHOICE OF APPROACH TO PANCREATECTOMY AFFECT POSTOPERATIVE OUTCOMES IN PATIENTS UNDERGOING RESECTION FOR CHRONIC PANCREATITIS: A NSQIP STUDY
Presenter: Essa M. Aleassa MD, MSc
EA Aleassa, G Morris-Stiff
Cleveland Clinic Foundation

P 157. CAN REGIONAL LYMPH NODE METASTASES FROM PANCREATIC CANCER BE PREDICTED WITH CROSS-SECTIONAL IMAGING?
Presenter: Katelyn Flick MD
KF Flick, TA Seltman, X Zhong, EP Ceppa, NJ Zyromski, A Nakeeb, CM Schmidt, M Tann, MG House
Indiana University School of Medicine
P 159. THE EFFICACY AND SAFETY OF DUODENAL STENT FOR PATIENTS TREATED WITH CHEMORADIOOTHERAPY FOLLOWED BY SURGERY FOR PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Takehiro Fujii MD, PhD
T Fujii, A Hayasaki, Y Iizawa, H Kato, A Tanemura, Y Murata, N Kuriyama, Y Azumi, M Kishiwada, S Mizuno, M Usui, H Sakurai, S Isaji
Mie University School of Medicine

P 160. IMPACT OF POSTOPERATIVE PANCREATIC FISTULA ON THE PROGNOSIS OF PATIENTS UNDERGOING RESECTION FOR PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Motokazu Sugimoto MD
M Sugimoto, S Kobayashi, S Takahashi, M Konishi, N Gotohda
National Cancer Center Hospital East

P 162. LONG-TERM ONCOLOGICAL OUTCOMES AFTER DISTAL PANCREATECTOMY FOR NEUROENDOCRINE NEOPLASMS: A COMPARISON BETWEEN MINIMALLY INVASIVE AND OPEN APPROACH USING PROPENSITY SCORE
Presenter: Valentina Andreasi MD
V Andreasi, S Partelli, P Rancoita, E Perez-Sanchez, F Muffatti, M Mazza, G Balzano, R Castoldi, S Crippa, D Tamburrino, M Falconi
San Raffaele Scientific Institute

P 163. IMPACT OF POST-OPERATIVE PANCREATIC FISTULA ON LONG-TERM OUTCOMES AFTER PANCREATIC RESECTION FOR PERIAMPUTLLARY ADENOCARCINOMA
Presenter: Jillian Bonaroti MD
JW Bonaroti, MS Zenati, A Al-abbas, C Reiser, AH Zureikat, BA Boone
University of Pittsburgh Medical Center
P 164. SIGNIFICANCE OF RADIOGRAPHIC SPLENIC VESSEL INVOLVEMENT IN PANCREATIC DUCTAL ADENOCARCINOMA OF THE BODY AND TAIL OF THE GLAND
Presenter: Flavio Rocha MD
JJ Hyun, JB Rose, AA Alseidi, TR Biehl, WS Helton, DL Coy, RA Kozarek, FG Rocha
Virginia Mason Medical Center

P 165. THE ASSOCIATION BETWEEN SURVIVAL AND LYMPHOCYTE MONOCYTE RATIO AFTER NEOADJUVANT THERAPY FOLLOWED BY PANCREATECTOMY IN PATIENTS WITH BORDERLINE RESECTABLE PANCREATIC CANCER
Presenter: Manabu Kawai MD, PhD
M Kawai, S Hirono, K Okada, M Miyazawa, Y Kitahata, R Kobayashi, A Shimizu, M Ueno, S Hayami, H Yamaue
Wakayama Medical University

P 166. SIGNIFICANCE OF COMPLETE RESECTION OF NERVE PLEXUS AROUND COMMON HEPATIC ARTERY IN PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Kenjiro Okada MD
K Okada, Y Murakami, K Uemura, N Kondo, N Nakagawa, S Seo
Hiroshima University

P 167. SINGLE INSTITUTION OUTCOMES OF ENHANCED RECOVERY AFTER SURGERY PROTOCOL IMPLEMENTATION IN PANCREATODUODENECTOMY
Presenter: Essa M. Aleassa MD, MSc
EM Aleassa, N Anzlovar, G Morris-Stiff
Cleveland Clinic Foundation
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P 168. TRANSDUODENAL AMPULLECTOMY FOR AMPULLARY NEOPLASMS: INITIAL RESULTS FROM A HIGH-VOLUME CENTER
Presenter: Hana Fayazzadeh MD
H Fayazzadeh, AT Strong, A Khithani, R Simon, RM Walsh, G Morris-Stiff, T Augustin, KM El-Hayek
Cleveland Clinic Foundation

P 170. BARRIERS TO IMPLEMENTATION OF AN ENHANCED RECOVERY PROGRAMME FOLLOWING UNEVENTFUL PANCREATICODUODENECTOMY. A RETROSPECTIVE COHORT STUDY
Presenter: Michele Mazza MD
S Partelli, M Michele, G Guarneri, G Maggi, N Pecorelli, D Tamburrino, S Crippa, R Meani, L Beretta, M Falconi
San Raffaele Scientific Institute

P 171. PREOPERATIVE FACTORS FOR EARLY RECURRENCE OF RESECTED PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Toshitaka Sugawara MD
T Sugawara, D Ban, S Watanabe, T Ogura, K Ogawa, H Ono, Y Mitsunori, A Kudo, M Tanabe
Tokyo Medical and Dental University

P 172. PREOPERATIVE RADIOLOGICAL PARAMETERS PREDICT POSTOPERATIVE PANCREATIC FISTULA IN PANCREATODUODENECTOMY
Presenter: Hryhoriy Lapshyn MD
H Lapshyn, E Petrova, L Bolm, D Bausch, T Keck, UF Wellner
University Medical Center Schleswig-Holstein
P 173. “I WILL TAKE GLUE WITH STITCHES”: A COMPARISON OF SKIN CLOSURE TECHNIQUES IN PANCREATICODUODENECTOMY
**Presenter:** Katelyn Flick MD
KF Flick, RE Simpson, MG House, EP Ceppa, N Zyromski, A Nakeeb, CL Colgate, ML Fennerty, MC Schmidt
Indiana University School of Medicine

P 174. EX-VIVO HUMAN MODEL OF THERANOSTIC NANODELIVERY SYSTEM FOR PANCREATIC NEOPLASMS - A FEASIBILITY TRIAL
**Presenter:** Konstantinos Chouliaras MD
K Chouliaras, L McNally, A Samykutty, E McWilliams, RB D’Agostino, CJ Clark, P Shen
Wake Forest School of Medicine

P 175. INCIDENTAL PANCREATIC TUMORS
**Presenter:** Claudia Medina MD
CR Medina, E Murrieta, MA Teliz, JA Mier, S Valanci
American British Cowdray Medical Center

P 177. IMPACT OF PREOPERATIVE BILIARY DRAINAGE AND EXTERNAL PANCREATIC STENTS ON CLINICAL OUTCOME AFTER PANCREATICO-DUODENECTOMY
**Presenter:** Joseph Reza MD
JA Reza, C Canavan, K Wissinger, M Uwah, P Veldhuis, Y Du, JP Arnoletti
Florida Hospital Orlando

P 178. EARLY NODE-NEGATIVE INVASIVE INTRADUCTAL PAPILLARY MUCINOUS TUMORS SEEM TO HAVE A BETTER PROGNOSIS THAN NODE-NEGATIVE PANCREATIC DUCTAL ADENOCARCINOMAS
**Presenter:** Francesca Gavazzi MD
F Gavazzi, G Capretti, C Ridolfi, G Nappo, S Carrara, M Sollai, M Angrisani, A Zerbi
Humanitas University
P 179. PAIN AT DIAGNOSIS AND HIGH ASA ARE NEGATIVE PROGNOSTIC FACTORS IN PATIENTS WITH PANCREATIC CANCER OR INVASIVE IPMN
**Presenter:** Alessandro Zerbi MD
FGavazzi, GCapretti, CRidolfi, GNappo, SCarrara, PSpaggiari, PRiva, AZerbi
Humanitas University

P 180. INTERNAL PANCREATICOJEJUNOSTOMY STENTING DECREASES RATE OF PANCREATIC FISTULAS AFTER PANCREATICODUODENECTOMY
**Presenter:** John Stauffer MD
AW Elias, JA Stauffer
Mayo Clinic Rochester

P 181. INFLAMMATORY MARKERS PREDICT SURVIVAL IN LOCALLY ADVANCED PANCREATIC CANCER PATIENTS AFTER CHEMOTHERAPY
**Presenter:** Sushanth Reddy MD
SReddy, JBRose, SMVickers, GRWilliams, AAAhmed, KRYaman, MJHeslin, OGbolahan, AJGunn, DEMorgan, SJGalgano,
AMcDonald, RJacob, RKPaluri
University of Alabama at Birmingham

P 182. A NOMOGRAM TO PREOPERATIVELY PREDICT EARLY CANCER-RELATED MORTALITY IN RESECTED PANCREATIC CANCER FOLLOWING NEOADJUVANT CHEMORADIATION THERAPY
**Presenter:** Ho Kyoung Hwang MD, PhD
HKHwang, KWada, MD, HYKim,YNagakawa, YHijikata,
YKawasaki,YNakamura,LSLee,DSYoon, WJLee, CMKang
Yonsei University
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P 183. PANCREATIC ADENOCARCINOMA CAUSING NECROTIZING PANCREATITIS: NOT AS RARE AS YOU THINK?
Presenter: Kyle Lewellen BS
KA Lewellen, TK Maatman, MA Heimberger, EP Ceppa, MG House, A Nakeeb, CM Schmidt, NJ Zyromski
Indiana University School of Medicine

P 184. PANCREATIC METASTASIS FROM RENAL CELL CARCINOMA: ANALYSIS OF 16 CASES
Presenter: Armando Rosales MD
A Rosales, DL Cardoso, H Asbun, JA Stauffer
Mayo Clinic Rochester

P 185. THE ASSOCIATION BETWEEN ELEVATED CA 19-9 AND RECURRENCE, SITE OF RECURRENCE, AND OVERALL SURVIVAL IN UPFRONT RESECTABLE PANCREATIC CANCER
Presenter: Benjamin Powers MD
BD Powers, KJ Kim, DK Deperalta, T Ogami, J Pimiento, PJ Hodul, MP Malafa, JB Fleming
Moffitt Cancer Center

P 186. THE STRATEGY OF COMPLETE CLEARANCE AROUND SUPERIOR MESENTERIC ARTERY, CELIAC ARTERY AND ITS TRIANGLE IN RADICAL PANCREATICODUODENECTOMY FOR PANCREATIC CANCER
Presenter: Kuirong Jiang MD, PhD
K Jiang, P Wu, Z Lu, J Chen, J Wei, F Guo, B Cai, J Yin, D Xu, Y Miao
Pancreas Center, The First Affiliated Hospital with Nanjing Medical University
P 187. DOES PLACEMENT OF EXTERNAL PANCREATIC DUCT STENT (EPDS) DURING Pancreaticoduodenectomy (PD) REDUCE THE RATE OF CLINICALLY RELEVANT POSTOPERATIVE PANCREATIC FISTULA (CR-POPF) IN HIGH RISK GLANDS?

Presenter: Andreas Schneider MD
AM Schneider, E Alonso, ES Tang, ST Chiu, ML Babicky, PH Newell, PD Hansen
Providence Portland Medical Center

P 189. PREDICTION OF PANCREATIC FISTULA AFTER DISTAL PANCREATECTOMY: IS IT NECESSARY TO PLACE PROPHYLACTIC DRAIN?

Presenter: Kazuhiro Suzumura MD, PhD
Hyogo College of Medicine

P 190. CENTRAL PANCREATECTOMY FOR EARLY-STAGE Pancreatic Ductal Adenocarcinoma: A Single-Center Case-Control Study

Presenter: Zipeng Lu MD, PhD, MRCS
JS Wei, H Gao, TT Liu, GF Wang, Y Gao, LD Yin, YP Peng, N Lyu, K Zhang, WT Gao, JL Wu, KR Jiang, Yi Miao
Nanjing Medical University

P 191. SELECTIVE USE OF A BLUMGART-TYPE TECHNIQUE FOR Panreaticojejunostomy Following Pancreaticoduodenectomy DOES NOT REDUCE CLINICALLY SIGNIFICANT FISTULA RATES

Presenter: Catherine Wang
CY Wang, J Lyons, JM Hardacre
Case Western Reserve University School of Medicine
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P 193. PREDICTING SURVIVAL OF PDAC PATIENTS BEFORE SURGERY: A MULTI-OMICS APPROACH
Presenter: Zipeng Lu MD, PhD, MRCS
Z Lu, K Zhang, H Shi, H Gao, X Huang, D Xu, Q Xu, K Jiang, Y Miao
Pancreas Center, The First Affiliated Hospital with Nanjing Medical University

P 196. THE IMPLICATION OF INDUCTION CHEMOTHERAPY FOLLOWED BY CHEMORADIOThERAPY FOR LOCALLY ADVANCED PANCREATIC CANCER, INTENDED CONVERSION SURGERY
Presenter: Daisuke Ban MD, PhD
D Ban, T Sugawara, T Kato, A Kudo, M Tanabe
Toyko Medical and Dental University

P 197. ROBOT-ASSISTED TRANS-GASTRIC TREATMENT OF WALLED-OFF PANCREATIC NECROSIS WITH THE DA VINCI XI
Presenter: Matteo Palmeri MD
L Morelli, N Furbetta, M Palmeri, G Di Franco, S Guadagni, D Gianardi, M Bianchini, G Caprili, C D’Isidoro, F Mosca, G Di Candio
University of Pisa

P 198. PANCREATICODUODENECTOMY FOR PERIAMPULLARY ADENOCARCINOMA IN A PATIENT WITH A PREPANCREATIC PORTAL VEIN
Presenter: Morgan Bonds MD
M Bonds, J Rekman, G Baison, F Rocha
Virginia Mason Medical Center

P 199. KIDNEY TRANSPLANTATION AFTER EXTENDED MULTI-VISCERAL RESECTION FOR PANCREATIC DUCTAL ADENOCARCINOMA
Presenter: Louisa Bolm MD
H Lapshyn, L Bolm, E Petrova, D Bausch, T Keck, UF Wellner
University Medical Center Schleswig-Holstein
P 200. CLINICOPATHOLOGIC FEATURES OF INTRADUCTAL TUBULOPAPILLARY NEOPLASMS OF THE PANCREAS
Presenter: Janelle Rekman MD, MAEd
J Rekman, RDorer, MBonds, GBaison, FRocha
Virginia Mason Medical Center

P 201. PANCREATICODUODENECTOMY IN PATIENT WITH JEJUNOILEAL BYPASS
Presenter: Flavio Rocha MD
NHandy, LHarmont, FGRocha
Virginia Mason Medical Center

P 202. AN UNKNOWN PRIMARY—A CASE OF A MERKEL CELL CARCINOMA IN THE INGUINAL NODES AND PANCREAS
Presenter: Shilpa Murthy MD, MPH
SSMurthy, SReddy, MHill, EO’Halloran, JHoffman, JFarma
Fox Chase Cancer Center/Temple University

P 203. LUMEN-APPOSING METAL STENTS FOR DRAINAGE OF WALLED-OFF NECROSIS—A CASE REPORT
Presenter: Nelson Coelho MSc
NHVCoelho, CRFRodrigues, ABOsvaldt
Hospital Moinhos de Vento

P 204. NEEDLE TRACT SEEDING FOLLOWING ENDOSCOPIC ULTRASOUND-GUIDED FINE-NEEDLE ASPIRATION FOR PREOPERATIVE DIAGNOSIS OF STAGE IA PANCREAS HEAD CANCER
Presenter: HIROYUKI HISAI MD, PhD
HHisai, TSakurai, YKoshiba, KWatanabe, SAmada, MSato,
RKawasaki, H Gyobu, NYoshida
Japanese Red Cross Date Hospital
P 206. ANTITUMOR ROLE AND MOLECULAR REGULATION OF HEAT SHOCK PROTEIN 70 AND 90: ACUTE PANCREATITIS AND PANCREATIC DUCTAL ADENOCARCINOMA

Presenter: Aiste Gulla MD, MHCM
A Gulla, K Strupas, GH Su
Vilnius University
ORAL ABSTRACTS
1. GUT MICROBIOTA FACILITATE TOLL-LIKE RECEPTOR 2-MEDIATED PANCREATIC CANCER METASTASES

S Kurtom, V Sethi, A Ferrantella, Jun Yi Tao, HKC Jacob, S Ramakrishnan, S Roy, A Saluja, V Dudeja

Presenter: Saba Kurtom MD | University of Miami

**Background:** The gut is composed of trillions of bacteria that play an imperative role in intestinal homeostasis. Recent evidence demonstrates that the gut microbiome plays a role in modulating the immune system. Our study aimed to evaluate microbiome-driven immunological mechanisms, specifically toll-like receptor (TLR) activation, in metastatic murine pancreatic ductal adenocarcinoma (PDAC). TLR2 and TLR4 are important mediators of the inflammatory response in cancer. We hypothesized that gut microbiome depletion would decrease liver metastases, via abrogation of the TLR-induced inflammatory response.

**Methods:** C57Bl/6, TLR 2-/-, and TLR 4-/- mice were randomized to receive daily oral saline or a gut sterilizing cocktail of poorly absorbable broad spectrum antibiotics (Vancomycin, Ampicillin, Amphotericin B, Metronidazole, and Neomycin). To induce liver metastasis, the mice underwent splenic injection of pancreatic cancer cells derived from KPC (Kras LSL.G12D/+; p53 R172H/+;Pdx::Cre) mice. Mice were sacrificed 21 days after splenic injections. Livers from each group were harvested and immunophenotyped via flow cytometry.

**Results:** Depletion of the gut microbiome decreased hepatic metastases in KPC-injected wild type and TLR4-/- mice. This tumor inhibitory effect of gut microbial depletion disappeared in the TLR2-/- KPC group, indicating a potential mechanism of action. Flow cytometry analysis revealed increased in IFN-gamma secretion from T-lymphocytes in the antibiotic-treated group. Antibiotics also decreased intrametastatic bacterial burden within liver specimens.

**Conclusion:** The gut microbiome promotes liver metastases in pancreatic cancer via modulating IFN-gamma secretion by T-lymphocytes, mediated through the TLR-2 pathway.
Figure 1: Depletion of the gut microbiome decreases tumor burden. Mice were randomized to receive oral saline or antibiotics. After 2 weeks of antibiotics or saline treatment, splenic injections were performed with KPC cells. Treatment with antibiotics or saline continued until sacrifice. Mice were sacrificed 21 days after splenic injection. Livers were harvested and weighed as shown. A) C57BL6 mice, p value 0.0013. B) TLR4-/- mice, p value 0.03. C) TLR2-/- mice, no significance.
2. INTERNATIONAL VALIDATION OF THE AMSTERDAM MODEL FOR SURVIVAL PREDICTION AFTER RESECTED PANCREATIC CANCER

S van Roessel, M Strijker, EW Steyerberg, JV Groen, JS Mieog, OR Busch, A Halimi, L Zarantonello, B Groot Koerkamp, U Wellner, JY Jang, CH van Eijck, M Abu Hilal, M del Chiaro, T Keck, A Alseidi, C Bassi, MG Besselink

Presenter: Stijn van Roessel MD, MSc | Amsterdam UMC

Background: A recent systematic review identified only two prediction models for survival in resectable pancreatic cancer of high methodological quality. One of these two, the Amsterdam prediction model (including differentiation grade, positive lymph node ratio, margin status and adjuvant therapy) have not been validated. The objective of this study was to validate the Amsterdam prediction model in a large international cohort.

Methods: All consecutive patients who underwent pancreatoduodenectomy for pancreatic cancer between 2000-2015 at 9 tertiary hospitals from 6 countries were included for analysis. Calibration and model performance for prediction of 3-year and 5-year survival were evaluated and compared to the 8th edition of the TNM staging system, using Uno’s C-statistic, the Receiver Operating Characteristics (ROC) curves and area under the curves (AUC). Validation was performed according to the TRIPOD statement.

Results: A total of 1,943 patients were included (mean age 65 years, 52% male), with a median overall survival of 23.6 months. The validation cohort was demographically comparable to the design cohort, but had a higher lymph node yield (median of 19 vs. 8) and more N2 disease (22.0% vs. 40.3%), and a higher use of adjuvant therapy (74.0% vs. 26.0%). Calibration analysis demonstrated that the predictions of 3-year survival did not systematically deviate from the observed survival rates with a slope of 0.85 and an intercept of 0.04. Uno’s C-statistic was 0.65 in the validation cohort, compared to 0.67 in the derivation cohort. ROC-curves showed an AUC of 0.70 and 0.72 for 3- and 5-year survival, compared to 0.65 and 0.68 for the TNM 8, respectively. Analysis of model performance among the different countries demonstrated a robust C-statistic, ranging from 0.63 to 0.67. The web-based calculator www.pancreascalculator.com was updated with these data.

Conclusion: This international multicenter study validated the previous predictions of the Amsterdam model for 3- and 5-year survival after pancreatoduodenectomy for pancreatic cancer. Model performance was
fairly accurate in terms of calibration and discrimination, and the model demonstrated robustness within the different countries. The model incorporates easily available variables (differentiation grade, lymph node ratio, margin status and adjuvant therapy) and the web-based calculator facilitate easy use in daily practice.
3. CELL-INTRINSIC PD-1 PROMOTES PROLIFERATION IN PANCREATIC CANCER TARGETING CYR61/CTGF VIA HIPPO PATHWAY

N Pu, S Gao, H Yin, J Li, W Wu, Y Fang, L Zhang, Y Rong, X Xu, D Wang, T Kuang, D Jin, J Yu, W Lou

Presenter: Ning Pu MD | Johns Hopkins University School of Medicine

**Background:** Pancreatic ductal adenocarcinoma (PDAC) remains as a refractory disease. Immunotherapy, such as PD-1 monotherapy, has shown robust performance in targeting several malignancies depending on the inhibitive immune checkpoint on T lymphocytes, however, the effect and mechanism of intrinsic PD-1 in pancreatic cancer cells was still unknown.

**Methods:** Associations between clinicopathological characteristics and stained tissue microarrays of PDAC specimens were analyzed. Function assays, RNA sequencing, mass spectrometric analysis, western blot, qRT-PCR and immunohistochemistry were administrated in function and mechanism analysis.

**Results:** Cell-intrinsic PD-1 was significantly correlated with OS in PDAC patients undergoing radical resection. Independently of adaptive immunity, intrinsic PD-1 in PDAC promoted tumor growth. Concomitantly, overexpression of intrinsic PD-1 enhanced cancer proliferation and inhibited cell apoptosis in vitro and vivo, whereas PD-1 knock down showed the opposite role. Mechanistically, PD-1 bound the downstream MOB1, thus inhibiting its phosphorylation. Moreover, more synergistic tumor suppression resulted from combining Hippo inhibitors with anti-PD-1 treatment than either single agent alone in vitro. Additionally, Hippo downstream targets, CYR61 (CCN1) and CTGF (CCN2), were directly affected by PD-1 mediated Hippo signaling activation in concert with survival outcomes. Finally, the formulated nomogram showed superior predictive accuracy for OS in comparison with the TNM stage alone.

**Conclusion:** Cell-intrinsic PD-1 is potentially expressed in pancreatic cancer cells, which significantly promotes tumorigenesis and progression. PD-1 immunotherapy in combination with Hippo pathway inhibitors may optimize anti-tumor efficacy in PDAC patients. Besides, a strong rational strategy is provided for use of formulated nomogram in prognostic prediction in patients with resected PDAC.
Percent M1 Macrophages and Primary Tumor Weights in Female and Male Mice
KPC484

Treatment Groups
4. SEX AFFECTS RESPONSE TO TYROSINE KINASE INHIBITION IN PANCREATIC ADENOCARCINOMA

_BG Childers, BG Childers, J Jaquish, AM Lowy_

**Presenter:** Betzaira G. Childers MD | University of Cincinnati

**Background:** Emerging data suggests that biologic differences between the sexes influence disease prevalence and therapeutic response. RON/MST1R is a receptor tyrosine kinase expressed in epithelial cells and macrophages and activates downstream signaling pathways that regulate oncogenic phenotypes in pancreatic cancer. RON and several other kinases are the primary targets of LY2801653/Merestinib (Eli Lilly), a small molecule inhibitor now in Phase 2 trials. LY2801653 reduced tumor growth and modulated macrophage phenotypes in male mice bearing orthotopic, organoid-derived pancreatic cancer. We sought to determine if similar responses would be observed in female mice, given differential immune responses have been identified between the sexes.

**Methods:** All animal procedures were conducted with approval of the Institutional Animal Care and Use Committee at UCSD. Pancreatic adenocarcinoma organoid cell lines KPC515 and KPC484 were derived from spontaneous growth in KRAS/p53 mice of B6.129S background. F1 hybrid male and female mice underwent orthotopic injection of the above mentioned organoid lines at 6-8 weeks of life. Daily dosing with RON inhibitor LY2801653 was initiated one week after surgery for a total treatment time of two weeks. Control groups received 10% Acacia Gum via oral gavage for the same duration. Mice were then euthanized and the primary tumor was excised, weighed and processed for flow cytometric analysis, histology, and immunohistochemistry.

**Results:** RON inhibition with LY2801653 in F1 hybrid male mice resulted in a significant reduction in primary tumor weight when compared to male control group (p-value < .001). However, no significant difference was observed in between F1 female treatment and control groups. Flow cytometry demonstrated significant differences in macrophage cell populations within the tumor microenvironment of male mice. Macrophages function within a phenotypic continuum for which opposite extremes are referred to classical (M1) and alternative (M2) states. Along this spectrum, macrophages may promote acute inflammation (M1), tissue rebuilding (M2), or some combination thereof. RON inhibition in male mice led to a significant increase in the M1 macrophage phenotype and a significant decrease in the M2 macrophage phenotype with p-values of (.004) and (.008) respectively.
Again, no such differences between treatment and control groups were identified between the female treatment groups.

**Conclusion:** Increasingly, research in numerous fields suggests that biologic differences between males and females influence disease pathophysiology and therapeutic response. As such, sex must be now be evaluated as a biologic variable in all NCI funded research. We found that treatment with the kinase inhibitor LY2801653 reduced tumor growth, and modulated macrophage polarization in male mice, findings that were not observed in female mice. The mechanism(s) underlying these differences are under active investigation in our lab. These findings support the idea that biologic differences between males and females may significantly influence therapeutic response in pancreatic adenocarcinoma. Additional research is imperative to better understand these biologic differences and their implications for disease onset, progression, and response to treatment.
ORAL ABSTRACTS  
(continued)

5. A BLOOD-TEST TO MEASURE OUTCOME AND RESPONSE TO THERAPY: DEVELOPING THE NECESSARY TOOLS FOR PRECISION TREATMENT OF PANCREATIC DUCTAL ADENOCARCINOMA


Presenter: Ammar Javed MD | Johns Hopkins University School of Medicine

Background: Historically, there has been a lack of effective biomarkers for pancreatic ductal adenocarcinoma (PDAC), CA19-9 is the only clinically utilized marker. Recently, circulating tumor cells (CTCs) and circulating tumor DNA (ctDNA) have been shown to be good tumor-specific biomarkers for PDAC. The aim of this study was to develop and evaluate a composite marker based on CA19-9, CTCs and ctDNA.

Methods: Preoperative peripheral blood samples were collected from patients with PDAC undergoing resection at our institution. CTCs were isolated and characterized by immunofluorescence, and Digital-droplet PCR (ddPCR) was used to detect the major PDAC-associated somatic KRAS mutations (G12D, G12V, G12R, and Q61H). A multivariable analysis was performed to identify factors associated with disease-free survival (DFS). The resulting hazard ratios were used to assign a score to each variable selected in the final model. A cut off was assigned to the combined score to stratify patients into composite marker positive and negative cohorts. DFS was evaluated between the two cohorts.

Results: The mean age of 34 patients included in the study was 64.2±10.2 years, and a majority were female (N=21, 61.8%). Neoadjuvant therapy was administered in 23 patients (67.6%), and a majority underwent a pancreaticoduodenectomy (N=22, 64.7%). Upon histopathological examination, the median tumor size was 3.4 (interquartile range(IQR): 2–5) cm and a majority had moderately differentiated tumors (N=22, 66.7%). Nodal disease, perineural invasion, or lymphovascular invasion was present in 21 (61.7%), 28 (82.4%), and 16 (47.1%) patients respectively. CTCs were identified in 32 patients (94.1%). All of these patients had epithelial CTCs (eCTCs) and 23 patients (67.6%) had epithelial/mesenchymal CTCs(mCTCs). ctDNA was detectable in 7 patients (20.6%). The median CA19-9 was 65 (IQR: 20.7 – 228.4) Units/mL, and 11 patients (35.5%) had a CA19-9 of >100 Units/mL. In the multivariable analysis, the factors that were significantly associated with DFS included presence of mCTCs (Hazard ratio(HR): 4.63,
95%CI: 1.78-12.07, p=0.002), elevated CA19-9 (HR: 3.12, 95%CI: 1.29-7.49, p=0.011), and detectable ctDNA (HR: 2.82, 95%CI: 1.22-6.54, p=0.016). The composite score ranged from 0 to 11, and 12 patients (35.3%) were found to have a score higher than 6 i.e. positive composite marker. When evaluated in a multivariable model with other clinicopathological factors, the composite marker remained significantly associated with DFS (HR: 3.33, 95%CI: 1.42-7.84, p=0.006). The median DFS for patients with a negative composite marker was 12.35 (IQR: 8.64-14.85) months, compared to 4.86 (IQR: 2.69 - 8.97) months for patients with a positive composite marker. Individually, CA19-9 under classified three patients as low-risk and overclassified three patients as high-risk, while mCTCs overclassified 11 patients as high-risk and ctDNA under classified five patients as low-risk and overclassified four patients as high-risk.

Conclusion: A composite score based on multiple biomarkers was used to develop a composite marker. Patients found to be positive for the composite marker had a significantly shorter DFS. Blood based biomarkers will be important to guide precision therapies. This test holds promise and is currently undergoing prospective validation and evaluation for longitudinal use.
6. PLATFORMS FOR DELIVERY OF TUMOR-SPECIFIC FLUORESCENCE IMAGING OF HUMAN PANCREATIC CANCER

TM Lwin, H Hollandsworth, S Amirfakhri, F Filemoni, RM Hoffman, M Bouvet

Presenter: Michael Bouvet MD | University of California, San Diego

**Background:** Pancreatic cancer is a malignancy with poor prognosis. When the lesion is local, the only chance for cure is complete surgical resection to negative margins. A high number of curative-intent pancreatic surgeries are seen with early recurrence at the surgical site or at distant sites, indicating the weakness in detection of peritoneal disease and the challenge in obtaining truly negative oncologic margins. Real-time intra-operative contrast enhancement using tumor-specific fluorescent anti-CEA probes can assist surgeons in visualizing radiographically occult localized lesions, determining resection margins, surveying the resection bed for residual disease, and targeting lymph nodes for removal.

**Methods:** Anti-CEA antibodies or nanobodies were conjugated with near-infrared fluorophores. Tumor fragments of BxPC3 human pancreatic cancer were implanted into pancreatic tail of nude mice to establish orthotopic models. After tumors reached 7-10 mm in size, 25-30 μg of fluorophore-conjugated probe was delivered intravenously. Mice were imaged 3-48 hours post-injection using the following imaging systems: the CRI Maestro small animal imaging system with spectral separation (Perkin Elmer, Waltham, MA), the da Vinci Firefly robotic laparoscope (Intuitive Surgical, Sunnyvale, CA), and the Stryker Aim laparoscope (Stryker Corp, Kalamazoo, MI). Fluorescence intensity was measured at the tumor, surrounding background tissue, and tumor-to-background-ratio was calculated.

**Results:** Pancreatic orthotopic tumors were fluorescently labeled with anti-CEA fluorescent-probes. All 3 constructs specifically co-localized with the GFP tagged orthotopic tumor. Using the CRI Maestro small animal imaging system, tumor to background ratio (TBR) using aanti-CEA-antibodies was 6.3 (imaged at 48 hours), PEG-ylated aCEA-antibodies was 14.7 (imaged at 48 hours), and anti-CEA-nanobodies was 2.96 (imaged at 3 hours). The PEG-ylated fluorescent antibody accumulated at the liver while the nanobody had renal accumulation. The fluorescence signal was detectable in these tumors using the da Vinci Firefly and the Stryker Aim clinical imaging devices.

**Conclusion:** Antibodies and nanobodies bound to near-infrared fluorophores
were able to clearly and specifically label orthotopic pancreatic xenografts. The probe co-localized with the GFP tagged tumors. There was improved contrast compared to GFP. There was adequate TBR using these probes with the best contrast using the PEG-ylated probe. However the nanobody probe has kinetics that allow same day administration and imaging. These probes are promising molecules for FGS of pancreatic cancer.

7. WNT11 DRIVES PDAC CELL MIGRATION AND INVASION AND IS ASSOCIATED WITH B-INTTEGRIN SIGNALING PATHWAYS

MP Kim, TG Hughes, B Dai, X Li, K Das, C Siangco, NE Navin, S Bai, E Sei, M Hu, TS Kumar

Presenter: Tara Hughes MD | University of Texas MD Anderson Cancer Center

Background: Wnt/β-catenin signaling is strongly associated with cancer initiation and metastasis, but non-canonical Wnt signaling and its role in pancreatic ductal adenocarcinoma (PDAC) metastasis is not well understood. Wnt11 is a ligand capable of activating canonical and non-canonical Wnt signaling and has been previously implicated in cancer cell motility through mechanisms that are diverse and tissue specific. We investigated the role of Wnt11 in PDAC cell migration/invasion and identified associated gene expression signatures through single-cell transcriptomic analysis of 14 different tumors directly isolated from PDAC patients.

Methods: Single cell suspensions were generated from patient PDAC tumors and 10x GemCode microfluidics technology was used to perform single-cell RNA sequencing (scRNA-seq). Cell subpopulations with >4-fold increased levels of Wnt11 expression relative to all sequenced tumor cells were compared to develop differentially expressed gene profiles. Pathways analysis of derived gene sets was performed using Enrichr. Expression of Wnt11 was confirmed by RT-qPCR in PDAC cell lines derived from genetically engineered mouse models and patient derived xenografts. Wnt11 was overexpressed in PDAC cell lines following transfection with Wnt11 cDNA cloned into a pcDNA3.1 backbone and was silenced using two unique siRNAs along with a non-targeting control. Transwell migration/invasion assays were performed with three cell lines overexpressing Wnt11 and following siRNA-mediated
Wnt11 knockdown.

**Results:** Single cell RNA-sequencing data derived from 14 different PDAC patients revealed strong Wnt11 expression in subpopulations of PDAC cells. RT-qPCR confirmed expression of Wnt11 in all tested KPC and human PDAC cell lines. Stable overexpression of Wnt11 resulted in a 3.2-fold increase (3.2 ±1.46; p 4-fold), Enrichr analysis using the NCI pathway interaction database revealed significant enrichment in β-integrin signaling pathways (p<0.001).

**Conclusion:** Wnt11 is strongly expressed in subpopulations of human PDAC cells and significantly increases PDAC cell motility and invasion. β-integrin signaling is associated with Wnt11 expression in human PDAC tumors and may be involved in PDAC cell motility and invasion. Given its roles in PDAC cell migration and invasion, Wnt11 may represent a therapeutic target and merits additional, ongoing in vivo validation.

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**8. DIVERSITY OF GERMLINE VARIANTS AMONG PATIENTS WITH LOCALIZED PANCREATIC CANCER**

**Authors:** AN Krepline, J Geurts, I Akinola, KK Christians, B George, PS Ritch, W Hall, BA Erickson, DB Evans, S Tsai

**Presenter:** Ashley Krepline MD | Medical College of Wisconsin

**Background:** Current guidelines for the management of patients with pancreatic cancer (PC) recommend genetic counseling in all patients and genetic testing for patients in whom there is a clinical suspicion for an inherited susceptibility.

**Methods:** Patients with localized PC treated with neoadjuvant therapy between 2009-2018 were identified. We reviewed all information regarding genetic consultation, type of genetic testing (targeted gene vs. multi-gene testing), and pathogenic or likely pathogenic variants. Personal and family history for cancer, excluding non-melanoma skin cancer, was abstracted.
Results: Of 511 patients with localized PC, 165 (32%) were seen by a genetic counselor and genetic testing was performed in 125 (24%). Patients who underwent genetic testing were younger (median age: 63 vs. 67, p=0.01) and 8 (7%) were of Ashkenazi Jewish descent. Multi-gene testing was performed in 112 (90%) of the 125 patients, target gene testing was performed in 8 (6%), and unknown in 5 (4%). Of the 125 patients with genetic testing results, 15 (12%) had pathogenic variants and 5 (4%) had likely pathogenic variants. The pathogenic or likely pathogenic variants observed were ATM (n=7, 6%), CHEK2 (n=3, 2%), BRCA1 (n=2, 2%), BRCA2 (n=2, 2%), PALB2 (n=1, 1%), MUTYH (n=1, 1%), CDKN2A (n=1, 1%), STK11 (n=1, 1%), NBN (n=1, 1%), and MSH6 (n=1, 1%). Of the 20 patients with a pathogenic/likely pathogenic variant, 9 (45%) had a personal history of cancer, 16 (80%) had a first degree relative with cancer, and 19 (95%) had any degree relative with cancer. No differences were observed in ability to undergo surgical resection or median overall survival among patients with or without pathogenic/likely pathogenic variants.

Conclusion: Pathogenic or likely pathogenic variants were identified in 16% of patients with localized PC. Variants in the homologous recombination pathway accounted for 60% of the identified mutations, highlighting a potential role for targeted therapies, such as PARPi, in these patients.
9. ANTI-CTGF HUMAN RECOMBINANT MONOCLONAL ANTIBODY PAMREVLUMAB (FG-3019) INCREASED RESECTABILITY AND RESECTION RATES WHEN COMBINED WITH GEMCITABINE/NAB-PACLITAXEL IN PHASE 1/2 CLINICAL STUDY FOR THE TREATMENT OF LOCALLY ADVANCED PANCREATIC CANCER PATIENTS

V Picozzi, FG Rocha, MJ Pishvaian, K Mody, J Winter, J Glaspy, T Larson, WC Conway, M Zhong, M Carney, TB Neff, E Kouchakji, P Yu, E Carrier

Presenter: Flavio Rocha MD | Virginia Mason Medical Center

Background: Pancreatic ductal adenocarcinomas (PDAC) exhibit a high degree of desmoplasia with extensive connective tissue growth factor (CTGF) expression and extracellular matrix production (1, 2). CTGF overexpression was associated with aberrant fibrous tissue in mouse models, in which progression of tissue adhesion was inhibited by pamrevlumab (3). We hypothesize that pamrevlumab (FG-3019), an anti-CTGF antibody, may quantitatively influence resectability of locally advanced pancreatic cancer (LAPC) by inhibiting effects of CTGF overexpression on tissue adhesion. This abstract presents resectability and resection results of Phase 1/2 study in locally advanced pancreatic cancer treated in neoadjuvant setting with gemcitabine/nab-paclitaxel +/- pamrevlumab.

Methods: In a Phase 1/2 randomized clinical study, pamrevlumab + gemcitabine/nab-paclitaxel (G/N) (Arm A) vs. standard of care G/N (Arm B) was administered to treatment-naïve locally advanced pancreatic cancer (LAPC) patients to compare resection outcomes and overall survival (OS). Thirty-seven patients were randomized 2:1 in Arm A vs. Arm B. Patients who completed 6 cycles of treatment underwent tumor resectability assessment per protocol defined criteria (NCCN conversion from unresectable to resectable or borderline resectable, CA 19-9 decrease by 50%, PET decrease by 30% and RECIST response (CR, PR)). If the patient met one of these criteria and had no medical or surgical contraindication to surgery, he/she underwent surgical exploration. Radiation was not allowed pre-surgery. Adjuvant therapy, including post-operative chemoradiation, was administered according to the investigator’s discretion.

Results: A higher percentage of patients discontinued treatment in Arm B (46.2%) vs. Arm A (25%), due to disease progression or adverse events. More patients experienced a normalized PET in Arm A vs. Arm B; 35% vs. 23%, respectively. Thirty percent of patients had a best objective (CR + PR) RECIST response. In the ITT population, a greater number of patients were eligible for surgery and resected in Arm A vs. Arm B; 70.8 % vs. 15.4% and 33.3% vs.
7.7%, respectively. Reasons for no resection: metastatic disease, encasement of arteries and patient/physician decision. Best predictors of resectability were PET and CA 19-9. RECIST and NCCN did not correlate with resectability or resection: 8/9 resected patients following 6 cycles of neoadjuvant therapy were unresectable at EOT by NCCN criteria. Improvement in OS was noted in patients who were eligible for surgery vs. patients who were not (27.73 months vs. 18.40 months, p-value=0.0766), and in resected vs. non-resected patients (NE vs. 18.56 months, p-value=0.0141). No increase in serious adverse events was observed in Arm A vs. Arm B. Thirty and 90-day mortality rates were similar in both arms. No delay in wound healing or other serious surgical complications were observed post-surgery. No intra-abdominal abscess, surgical site infection, perioperative leaks or surgical site infection were noted during post-surgical hospitalization; one case of intra-abdominal abscess was observed in each arm (8.3% and 50% in Arm A and B, respectively) during the surgical follow-up period. Out of 9 resected patients in both arms (8 in Arm A and 1 in Arm B), there were 4 R0 and 4 R1 resections in Arm A, and 1 R0 resection in Arm B.

**Conclusion:** These findings indicate that pamrevlumab may be a valuable addition to neoadjuvant therapy in LAPC without added toxicity or need for preoperative radiation. The most predictive factors for resectability were PET and CA 19-9. These results warrant a follow-on study in a larger patient population.
10. IMPACT OF REINFORCED STAPLER DURING DISTAL PANCREATECTOMY FOR PANCREATIC FISTULA, A MULTICENTER RANDOMIZED CONTROLLED TRIAL

K Uemura, T Sudo, N Kondo, N Nakagawa, K Okada, S Kuroda, N Hadano, H Matsukawa, D Satoh, M Sasaki, T Abe, S Fukuda, A Ohshita, A Nakashima, Y Hashimoto, H Ohdan, Y Murakami

Presenter: Kenichiro Uemura MD | Hiroshima University

Background: Although distal pancreatectomy (DP) using reinforced stapler is expected to reduce PF, no multicenter RCT has been performed. The aim of this study was to investigate in the multicenter randomized controlled trial (RCT) whether reinforced stapler can reduce the incidence of clinically relevant pancreatic fistula (PF) after DP compared with stapler without reinforcement.

Methods: Patients scheduled for DP were enrolled in the current study between July 2016 and December 2017 at nine hospitals in Hiroshima Japan (Hiroshima Surgical Study Group of Clinical Oncology: HiSCO-07 Trial). Patients were randomized to either reinforced stapler or stapler without reinforcement. The primary endpoint was the incidence of clinically relevant PF. This RCT was registered with UMIN Clinical Trial Registry (UMIN000022341).

Results: A total of 122 patients were assigned to reinforced stapler (n=61) and stapler (n=61), and of whom 119 (61 reinforced stapler and 59 stapler) patients were analyzed. There was no significant difference between reinforced stapler and stapler for incidence of clinically relevant PF (16.3% vs. 27.1%, p=0.15). In addition, rate of major complication (16.3% vs. 18.6%, p=0.74), postpancreatectomy hemorrhage (0% vs. 3.4%, p=0.08) and median postoperative in-hospital days (19 days vs. 20 days, p=0.78) did not differ between two groups. Within a subset of 82 patients whose thickness of pancreatic transection line was less than 14mm, significant difference was found in the incidence of clinically relevant PF with 4.5% in reinforced stapler group and 21.0% in stapler group (p=0.01).

Conclusion: In conclusion, pancreatic transection during DP with reinforced stapler does not reduce the incidence of clinically relevant PF compared with stapler without reinforcement.
11. MENTORSHIP AND FORMAL ROBOTIC PROFICIENCY SKILLS CURRICULUM IMPROVE SUBSEQUENT GENERATIONS’ LEARNING CURVE FOR THE ROBOTIC PANCREATODUODENECTOMY


Presenter: Melissa Hogg MD, MS | NorthShore HealthSystem, affiliate of University of Chicago

Background: Incorporating new surgical technologies necessitates overcoming a learning curve (LC). Studies show the robotic pancreateoduodenectomy LC to be 80 with operating time (ORT) hardest to improve. This study evaluates how formal mentorship and a robotic technical skills curriculum impact the LC and complications for second and third generation adopters.

Methods: Consecutive pancreateoduodenectomies from 2008-2017 were evaluated. First Generation was two surgeons who started program without training. Second Generation was second two subsequent surgeons with mentorship. Third Generation was fellows who completed the resection after completing the curriculum. Multivariate models were performed for ORT, pancreatic fistula (CR-POPF), and major complications (Clavien>=3).

Results: 514 cases were evaluated: average ORT=413.1±106.4min, CR-POPF=7.6%, and Clavien>=3=23.9%. Patient factors predictive of increase ORT were blood loss, age, vascular resections, concomitant procedure, and adenocarcinoma. Surgeon factors predictive of increased ORT were earlier generation and earlier case number per generation (Figure). Surgical generation and percent of case performed by fellow were not predictive of CR-POPF nor Clavien>=3. Patient factors predictive of CR-POPF were increased ORT, soft gland, no neoadjuvant, and smaller duct. Patient factors predictive of increased Clavien>=3 were increased ORT, age, BMI and ASA, vascular resection, males, soft glands, and no neoadjuvant. Factors predictive of fellows performing resection were only one attending present, decreased BMI, and increased fellow surgeries.

Conclusion: In a high volume center of excellence formal mentorship and a skills curriculum decreased the starting point and steepness of learning curve for operating room time in robotic pancreateoduodenectomy. Complications were largely dependent on patient factors and not affected by introduction...
of next generation surgeons. Health care delivery systems should seek to explicitly incorporate these strategies to maximize highest value and quality patient outcomes.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate Odds Ratio (95% CI)</th>
<th>p-value</th>
<th>Multivariate Odds Ratio (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 65 years</td>
<td>0.623 (0.286, 1.361)</td>
<td>0.235</td>
<td>0.407 (0.165, 1.000)</td>
<td>0.050*</td>
</tr>
<tr>
<td>Sex (female)</td>
<td>0.587 (0.255, 1.349)</td>
<td>0.210</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI ≥ 30 kg/m²</td>
<td>1.585 (0.706, 3.555)</td>
<td>0.264</td>
<td>2.493 (0.999, 6.221)</td>
<td>0.050*</td>
</tr>
<tr>
<td>Prior abdominal surgery</td>
<td>0.624 (0.284, 1.371)</td>
<td>0.240</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-op album</td>
<td>2.073 (0.918, 4.682)</td>
<td>0.080</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preoperative radiation</td>
<td>5.160 (0.825, 32.284)</td>
<td>0.079</td>
<td>9.341 (1.283, 68.000)</td>
<td>0.027*</td>
</tr>
<tr>
<td>Tumor size ≥ 3.4 cm</td>
<td>2.348 (1.050, 5.252)</td>
<td>0.038*</td>
<td>2.937 (1.182, 7.296)</td>
<td>0.020*</td>
</tr>
<tr>
<td>Classic vs PPPD</td>
<td>0.529 (0.107, 2.624)</td>
<td>0.436</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vascular resection</td>
<td>1.994 (0.816, 4.872)</td>
<td>0.130</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total operative time</td>
<td>1.005 (1.000, 1.010)</td>
<td>0.052</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time to complete HJ</td>
<td>1.014 (0.976, 1.053)</td>
<td>0.473</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preoperative biliary stent</td>
<td>0.526 (0.234, 1.185)</td>
<td>0.121</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cystic duct incorporated into HJ</td>
<td>1.490 (0.523, 4.247)</td>
<td>0.455</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duct diameter, mm</td>
<td>0.797 (0.679, 0.937)</td>
<td>0.066*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thickness of bile duct, mm</td>
<td>1.080 (0.495, 2.359)</td>
<td>0.846</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intra-operative biliary stent, yes</td>
<td>2.035 (0.926, 4.476)</td>
<td>0.077</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length of HJ from hilar plate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10mm</td>
<td>Ref</td>
<td>-</td>
<td>Ref</td>
<td>-</td>
</tr>
<tr>
<td>10 – 20 mm</td>
<td>2.367 (0.776, 7.222)</td>
<td>0.130</td>
<td>3.569 (1.062, 11.996)</td>
<td>0.040*</td>
</tr>
<tr>
<td>&gt;20 mm</td>
<td>1.571 (0.369, 6.694)</td>
<td>0.541</td>
<td>1.453 (0.303, 6.982)</td>
<td>0.641</td>
</tr>
<tr>
<td>Mobilization of bile duct stump HJ technique</td>
<td>1.032 (0.464, 2.296)</td>
<td>0.938</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Running</td>
<td>Ref</td>
<td>-</td>
<td>Ref</td>
<td>-</td>
</tr>
<tr>
<td>Interrupted</td>
<td>0.955 (0.365, 2.494)</td>
<td>0.924</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hybrid</td>
<td>1.464 (0.163, 13.114)</td>
<td>0.733</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of posterior row stitches</td>
<td>0.732 (0.561, 0.956)</td>
<td>0.022*</td>
<td>0.720 (0.546, 0.949)</td>
<td>0.020*</td>
</tr>
<tr>
<td>Space between stitches, mm</td>
<td>0.443 (0.185, 1.059)</td>
<td>0.067</td>
<td></td>
<td></td>
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</tbody>
</table>
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ORAL ABSTRACTS  (continued)

12. VARIABLE LIFE ADJUSTED DISPLAY (VLAD): NOVEL UTILITY OF A METRIC FOR QUANTIFICATION OF PERIOPERATIVE PATIENT LIVES GAINED FOLLOWING PANCREATIC SURGERY

NM Sell, ZV Fong, SF Hohmann, CR Ferrone, C Fernandez del Castillo, AL Warshaw, KD Lillemoe, JP Bloom, M Qadan

Presenter: Naomi Sell MD, MHS | Massachusetts General Hospital

**Background:** Variable Life Adjusted Display (VLAD) is a metric that examines lives gained, based on predicted mortality. If patients survive, their risk-adjusted mortality is equivalent to the proportion of a life gained. The model utilizes the cumulative sum of risk taken to calculate lives accrued over time, and thereby rewards risk.

**Methods:** The Vizient® risk-adjusted database was used to calculate VLAD for patients undergoing pancreatectomy at 241 hospitals in the United States between FY2016-2018. Hospitals that performed fewer than 5 pancreatectomies per year were excluded. Pancreatectomies from the remaining 169 hospitals were included.

**Results:** A total of 39,289 pancreatectomies were performed within the study period. VLAD illustrates substantial variability of accrued lives gained or lost at each hospital (1a). Excellent performers were hospitals that operated on high-risk patients who survived, and thus accumulated lives gained. Conversely, poorer performing hospitals experienced worse outcomes, including with low-risk patients. Figure 1b depicts the upper, middle, and lower third curves of hospital VLAD performance. In examining VLAD stratified by hospital volume over time using a cutoff of 20 (“The Pledge”), high-volume hospitals demonstrated substantial accumulation of lives gained, compared with low-volume hospitals (1c). Similarly, teaching hospitals demonstrated an enhanced ability to save lives when compared with non-teaching hospitals (1d).

**Conclusion:** VLAD is a metric suitable for use in pancreatic surgery and has the potential to quantify perioperative aggregate lives gained based on risk-adjusted patient mortality. This metric demonstrates substantial variability in performance following pancreatic surgery in the United States.
## Median Overall and Progression Free Survival in 232 Patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>SOC FOLFOX</th>
<th>Gemcitabine/Nab-Paclitaxel</th>
<th>SOC + HAT</th>
<th>Gemcitabine/Nab-Paclitaxel</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N=95</td>
<td>N=45</td>
<td>N=97</td>
<td>N=45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall Survival (months)</td>
<td>14.4</td>
<td>13.9</td>
<td>15.3</td>
<td>12.7</td>
<td>0.36</td>
</tr>
<tr>
<td>Progression Free Survival</td>
<td>14.1</td>
<td>11.2</td>
<td>13.7</td>
<td>12.3</td>
<td>0.55</td>
</tr>
<tr>
<td>(months)</td>
<td></td>
<td></td>
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</tbody>
</table>

*p-log-rank test stratified for study site and resectability/CA19-9*
13. VIDEO REVIEW OF SURGEON TECHNICAL FACTORS OF THE HEPATICOJEJUNOSTOMY INDEPENDENTLY PREDICTS POSTOPERATIVE BILIARY COMPLICATIONS AFTER PANCREATICODUODENECTOMY

JA Brown, JP Jung, M Zenati, Al Al-Abbas, ME Hogg, HJ Zeh, AH Zureikat

Presenter: James Brown | University of Pittsburgh Medical Center

**Background:** Biliary complications arising from the hepaticojejunostomy (HJ) constitute an infrequent but significant morbidity after pancreaticoduodenectomy (PD), with a reported incidence ranging from 2.6% to 11.9%. While prior studies have sought to identify patient-related predictors, technical risk factors associated with these biliary complications have not been elucidated. Since video review has been shown to predict postoperative complications in both bariatric and hepato-pancreato-biliary (HPB) surgery independent of patient-related risk factors, we sought to utilize video review to identify technical factors predictive of bile leak, cholangitis and anastomotic stricture at the HJ following robotic pancreaticoduodenectomy (RPD).

**Methods:** An HPB surgeon blinded to patient outcomes reviewed videos of post-learning-curve HJs performed during RPD and documented 20 technical intraoperative variables including: time to complete HJ anastomosis, duct diameter, duct thickness, stent use, distance between HJ and hilar plate, length of bile duct stump mobilization, suturing technique (running vs. interrupted), suture type (4-0 V-loc vs. 5-0 PDS). Demographics and post-operative outcomes were identified through retrospective chart review. Stricture cases were identified by radiographic evidence of anastomotic narrowing at the HJ in the context of elevated LFTs or symptoms of cholangitis, while cholangitis was defined as abdominal pain, fever and jaundice, along with elevated alkaline phosphatase. Predictive models for bile leak, cholangitis alone and in the setting of HJ stricture were created utilizing logistic regression.

**Results:** Two hundred and forty-one HJs were analyzed. Mean age was 66.7 years and 42% were female. Incidence of biliary complications were: bile leak = 9 (3.7%), cholangitis only = 13 (5.4%), stricture and/or cholangitis = 29 (12%). Median time to bile leak, cholangitis and stricture were 6 days (IQR 3-8), 189 days (IQR 78-399), and 226 days (IQR 106-286), respectively. On multivariate analysis, predictors of biliary leak were intraoperative HJ stent use (OR 8.15, P=0.037), increased distance between HJ and hilar plate (OR 0.06, P=0.027) and mobilization of bile duct stump (OR 11.8, P=0.011). On multivariate analysis, increasing stitch count (OR 0.68, P=0.033), preoperative radiation use (OR 81.32, P=0.002), increasing common bile
duct diameter (OR 0.74, P=0.049), and increased distance between HJ and hilar plate (OR 4.79, P=0.05) were predictive of cholangitis alone. For the composite outcome of stricture and/or cholangitis, multivariate analysis identified age > 65 (OR 0.41, P=0.050), BMI > 30 (OR 2.49, P=0.050), preoperative radiation (OR 9.34, P=0.027), increasing tumor size (OR 2.94, P=0.020), vascular resection (OR 3.13, P=0.029), increased distance between HJ and hilar plate (OR 3.57, P=0.04), and increasing number of posterior row HJ sutures (OR 0.72, P=0.02) as predictors of the composite outcome.

**Conclusion:** Video review identified several technical variables predictive of bile leak, cholangitis and HJ stricture following PD. This analysis suggests that diametrically opposed technical factors are involved in predicting biliary leak on one end and cholangitis/stricture at the other, and it contributes to a growing body of evidence that video review can reliably identify technical factors predictive of surgical complications.
14. IMPLEMENTING ROBOT-ASSISTED DISTAL PANCREATECTOMY THROUGH A PROCEDURE-SPECIFIC TRAINING PROGRAM: AN ACADEMIC CENTER’S EXPERIENCE


Presenter: Sjors Klompmaker MD | Beth Israel Deaconess Medical Center

**Background:** RADP may reduce the treatment burden compared to open distal pancreatectomy (ODP), but studies on institutional training and implementation programs are scarce. We aimed to implement a procedure-specific training program for robot-assisted distal pancreatectomy (RADP) and assess its effects on the institutional learning curve and outcomes.

**Methods:** A retrospective single-center cohort study of all elective DPs, with interrupted time-series (01/2006-09/2017). Exclusion criteria were neoadjuvant therapy, vascular and unrelated organ resection. Baselines and unadjusted outcomes were compared between “before training” (ODP only; 2012) groups. We used run-charts to evaluate index length of stay (LOS) and 90-day comprehensive complication index (CCI) over time. We used CUSUM-charts of the operating time (OT) to evaluate the institutional learning curve. Here, we included all RADP cases regardless of the study inclusion criteria. In a secondary propensity-score-matched (1:1) analysis, we compared adjusted outcomes for RADP vs ODP.

**Results:** After screening, 237 patients were included in the before-training (133 ODP) and after-training (24 ODP, 80 RADP) groups. After initiation of training, mean perioperative blood loss decreased (-255 mL, P 0.05). Over time, there were non-random (P 65; Fig.1). Propensity-score matched analysis confirmed prior results and demonstrated that RADP was associated with reduced blood loss (92 vs 418 mL, P<0.001) and transfusion risk (1% vs 7%, P=0.063), while morbidity and oncological efficacy were unaffected. Increased mean operating time (306 vs 236 min, P<0.001) was offset by a one-day reduction in median 90-day cumulative length of hospital stay (6 vs 7 days, P<0.001).

**Conclusion:** This study shows how RADP can be safely introduced at an institution without reportable prior experience in minimally invasive HPB surgery, using a procedure-specific training program supervised by an experienced surgeon-coach. A steady-state in operating time was achieved.
after 65 cases.
ORAL ABSTRACTS  (continued)
15. ARE THERE ANY DIFFERENCES IN OUTCOMES WITH THE VARIOUS SURGICAL APPROACHES TO MANAGEMENT OF CHRONIC PANCREATITIS; AN AMERICAN COLLEGE OF SURGERY (ACS) NATIONAL SURGICAL QUALITY IMPROVEMENT PROGRAM (NSQIP) SURVEY

GB Baison, JF Rekman, MM Bonds, WS Helton

Presenter: George Baison MD | Virginia Mason Medical Center

Background: There are several options for surgical management of chronic pancreatitis refractory to medical management including total pancreatectomy, distal pancreatectomy, pancreaticoduodenectomy (Whipple) and drainage procedures (Frey, Berger, Bern, Puestow). The choice of procedure is often in the hands of the surgeon and the best approach remains debatable. Often these procedures have significant morbidity. This study seeks to evaluate the scope of surgical practice and outcomes among American College of Surgery (ACS) National Surgical Quality Improvement Program (NSQIP) participating hospitals and delineate if there are any differences between the outcomes of the various operations for chronic pancreatitis.

Methods: We interrogated the ACS NSQIP database for all patients that underwent surgery for chronic pancreatitis from 2014, the first year with a pancreatectomy-specific participant user file (PUF) to 2017, the most recent NSQIP PUF available. Procedure performed, demographics and surgical outcomes were identified. Univariate analysis was performed to compare the different surgical procedures. Logistic regression analysis was then performed to evaluate factors associated with poor outcome.

Results: A total of 24,321 pancreatectomies were performed by 106, 120, 137 and 142 participating hospitals in 2014, 2015, 2016 and 2017, respectively. Of these operations 1315 (5%) were for chronic pancreatitis. The total cohort had a mean age of 53 (18 – 88) years, mean BMI of 26.37 (10.54 – 51.32), 44% of patients smoked, 30% were diabetic, 57% were male, 79% were Caucasian and 71% had ASA class 3 or higher. Most (99%) of the pancreatectomies were elective operations, with an average operative time of 302 minutes and a length of stay of 10 days. The most common procedure was distal pancreatectomy (DP) (44%) followed by pancreaticoduodenectomy (26%) and then drainage procedures (Frey, Berger, Bern, Puestow) making up 24%. Total pancreatectomy (TP) was performed in 6% of patients. Combined complication rate (major and minor) for all procedures was 39.62%, while mortality was 1.75%. Significant differences between procedures were noted in operative time, length of stay, overall complication rate and mortality (TP>Drainage>Whipple>DP for all these measures). DPs had higher fistula rates (18% vs 15% for drainage procedures vs 12% for Whipple, p-value < 0.05). Logistic regression showed that overall complications were associated with higher ASA class (OR1.76, CI: 1.41 – 2.19) and increased operative time (OR 1.003, CI: 1.002 – 1.004) and but not directly with type of procedure.
ORAL ABSTRACTS  (continued)

**Conclusion:** Pancreatectomy for chronic pancreatitis remains a morbid operation with a significant complication rate regardless of the type of procedure performed. These findings are consistent with prior literature. However, the type of operation itself is not an independent predictor of poor outcome. Additional investigation is needed to elucidate other factors predictive of poor patient outcomes such as alcohol consumption, smoking, gland and bile duct pathology, and response or lack thereof to medical and endoscopic management.

16. **A PHASE III STUDY OF CHEMOTHERAPY WITH OR WITHOUT ALGENPANTUCEL-L (HYPERACUTE*-PANCREAS) IMMUNOTHERAPY IN SUBJECTS WITH BORDERLINE RESECTABLE OR LOCALLY ADVANCED UNRESECTABLE PANCREATIC CANCER**

*H Lavu, N Nissen, DB Hewitt, H Hatoum, B Musher, B Leiby, J Banks, A Coveler, R Al-Rajabi, S Shahda, L Balducci, G Vaccaro, T George, W Brenner, E Elquza, CJ Yeo, E Kennedy, N Vahanian, C Link*

**Presenter:** D. Brock Hewitt MD, MPH, MS | Thomas Jefferson University

**Background:** Data are lacking on the safety and efficacy of a combined chemotherapy and immunotherapy regimen as first-line therapy in patients with borderline resectable or locally advanced unresectable pancreatic adenocarcinoma.

**Methods:** We performed a multicenter, phase III randomized controlled trial in patients with pancreatic adenocarcinoma deemed borderline resectable or locally advanced unresectable by NCCN Guidelines (NCT01836432). Patients were randomized to standard of care (SOC) FOLFIRINOX or gemcitabine/nab-paclitaxel chemotherapy alone or in combination with algenpantucel-L immunotherapy (HAPa) (IND# 12311), which consists of allogeneic pancreatic cancer cells engineered to express the murine (1,3)GT gene. The primary outcome was overall survival.

**Results:** Between May 2013 and December 2015, 282 patients were treated from 32 participating sites. Median overall survival was 14.5 months in the SOC group (N=140) compared with 14.3 months in the HAPa group (N=142) (stratified adjusted hazard ratio (HR) 0.99; 95% CI 0.63-1.55). Median progression-free survival was 13.4 months in the SOC group and 12.4 months in the HAPa group (stratified adjusted HR 0.89; 95% CI 0.56-1.42). Serious
adverse events occurred in 61.7% of all patients, but only 2.3% of these were HAPa related.

**Conclusion:** In one of the largest pancreas cancer immunotherapy trials yet reported, the addition of algenpantucel-L immunotherapy was not associated with a survival advantage in patients with borderline resectable or locally advanced unresectable pancreatic adenocarcinoma compared to SOC chemotherapy.
17. GASTRIC CANDIDIASIS PREOPERATIVELY EXAMINED IS A CRUCIAL PREDICTOR OF POSTOPERATIVE INFECTION-RELATED COMPLICATIONS AFTER PANCREATICODUODENECTOMY: THE RESULTS OF PROSPECTIVE STUDY

K Gyoten, H Kato, D Noguchi, A Hayasaki, Y Iizawa, T Fujii, Y Murata, A Tanemura, Y Azumi, N Kuriyama, M Kishiwada, S Mizuno, M Usui, H Sakurai, S Isaji

Presenter: Kazuyuki Gyoten MD | Mie University School of Medicine

Background: Candida colonization in the human gastrointestinal tract is generally recognized as inapparent infection but is sometimes associated with invasive candidiasis in critically ill patients. We clarified that biliary candidiasis could be the independent cause of surgical site infections (SSIs) after pancreaticoduodenectomy (PD) (Pancreas 2016, Biomed Res Int. 2018), but there has been no reports regarding the impact of gastric candidiasis preoperatively examined on postoperative infectious complications. In the present study, we aimed to clarify whether gastric candidiasis had a significant impact on the clinical outcomes after PD.

Methods: Between October 2015 and February 2017, the consecutive 69 patients who underwent PD were enrolled for our study. The gastric juice was prospectively collected through the nasogastric tube immediately after the induction of general anesthesia and directly incubated onto the CHROMagar Candida plate for the cultivation of various Candida species. According to the presence or absence of gastric candidiasis, firstly, we compared the incidence of postoperative complications such as SSIs, early postoperative complications of Clavien-Dindo (C-D) classification of grade IIIa or more (no mortality in these patients) and late complications demanding rehospitalization after discharge. Secondly, we evaluated the clinical factors contributing to the occurrence of postoperative complications by multivariate analyses.

Results: Preoperative diagnosis was pancreatic ductal adenocarcinoma in 38 patients, intraductal papillary mucinous neoplasm in 13, biliary duct cancer in 12 and the others in 6. Gastric candidiasis was identified in the 23 patients (33%). Between the patients with (n=23) and without (n=46) gastric candidiasis, there were no significant differences in pre- and intra-operative clinical factors, such as age, patients’ conditions, primary disease and the rates of portal vein resection, except for operative time. As compared to the patients without gastric candidiasis, those with gastric candidiasis had significantly higher rates in SSIs (47.8% vs. 15.2%, p = 0.01), in early postoperative complications (C-D IIIa or more) (39.1% vs. 17.4%, p=0.048) and in late complications (52.2% vs. 21.7%, p=0.011). Multivariate analyses
identified gastric candidiasis as a common risk factor for SSIs, early and late complications: Odd’s ratio: 7.4 (p<0.01), 4.8 (p<0.05) and 3.7 (p<0.05), respectively.

**Conclusion:** The presence of gastric candidiasis, which is not rare in the patients undergoing PD, is a significant risk factor for postoperative infection-related complications. Meticulous postoperative cares including antifungal prophylaxis might reduce these complications.

**18. MOLECULAR STAGING IN PANCREATIC ADENOCARCINOMA UTILIZING KRAS MUTANT CELL-FREE DNA ANALYSES IN PERIPHERAL BLOOD AND PERITONEAL FLUID: ARE WE THERE YET?**

**JL Leiting, AM Abdelrahmen, BR Kipp, MJ Truty**

**Presenter:** Jennifer Leiting MD | Mayo Clinic Rochester

**Background:** Pancreatic ductal adenocarcinoma (PDAC) is a deadly malignancy with poor survival due to widespread dissemination in most patients at diagnosis, even for those with seemingly localized disease by standard imaging. The detection of KRAS mutations in cell-free DNA (cfDNA) from blood or peritoneal fluid is a novel potential method to increase detection of occult metastatic disease as most (>90%) PDACs harbor KRAS mutations. Our aim was to determine the frequency of cfDNA KRAS mutations in newly-diagnosed patients and assess the diagnostic accuracy to detect metastatic disease compared to standard staging methods.

**Methods:** With IRB approval, patients with newly diagnosed and untreated PDAC from October 2017-December 2018 underwent cfDNA mutant KRAS testing in peripheral blood. Patient plasma underwent digital droplet PCR to detect 4 common (12,13,61,146) KRAS mutations. Results were negative=no mutation, positive=mutation, or indeterminate=suspicious but not definitive. Positive/indeterminate KRAS (+KRAS) were considered significant for occult metastatic disease. Radiologic staging for metastatic disease was concurrently performed with cross section imaging (CT and/or MRI) as well as adjunctive procedures (PET or laparoscopy). Diagnostic accuracy of detecting metastatic disease was calculated using CA19-9 and/or +KRAS cfDNA status. A secondary initial pilot study was performed assessing feasibility of identifying KRAS cfDNA mutations in peritoneal fluid from patients with either positive laparoscopy (gross peritoneal metastases or
Results: Seventy-six patients underwent peripheral blood KRAS cfDNA testing. Of these, 57 (75%) were negative, and 19 (25%) were +KRAS. Patients with +KRAS had higher CA19-9 levels (891 vs. 61, p = 0.024) than patients with a negative KRAS. Of these patients with a +KRAS, 10 (52%) had findings of metastatic disease on complete staging (CT/MR, PET, or laparoscopy). In patients with +KRAS status but without radiologic metastasis that underwent neoadjuvant chemotherapy, 4 patients developed metastatic disease during therapy and 1 patient developed rapid recurrence postoperatively, all of whom were CA19-9 non-secretors. An additional 2 patients that were +KRAS had their KRAS cfDNA status turn negative following neoadjuvant systemic chemotherapy and continue to have no evidence of metastatic disease with short follow-up. Sensitivity for metastatic disease using CA19-9 elevation alone was 86%, but after including patients with +KRAS, sensitivity increased to 100% (p<0.05) (Figure 1A). Concurrently, NPV increased from 86% to 100% (p<0.05) after taking into account CA19-9 levels and KRAS status. The specificity of +KRAS as a single diagnostic test for metastatic disease was 83% and after a median follow-up of 3.5 months, specificity increased to over 90% (Figure 1B). In this small sample, no patient with a normal CA19-9 and a negative KRAS had evidence of metastatic disease at diagnosis or during follow-up. In a small peritoneal fluid pilot study, this assay was able to detect mutant KRAS cfDNA in peritoneal fluid with higher accuracy than cytological analysis alone, being able to identify atypical cells as +KRAS or -KRAS with the potential for better characterization of indeterminate peritoneal cytology samples.

Conclusion: The ability to accurately stage patients with newly diagnosed PDAC is vital as it drives treatment recommendations. KRAS cfDNA status, along with CA19-9, was able to increase the sensitivity for metastatic disease to 100%, allowing us to better identify patients that are unlikely to harbor occult metastatic disease. Additionally, +KRAS status alone had a high specificity for metastatic disease above and beyond traditional diagnostic imaging. This assay may have particular utility in CA19-9 non-secretors. Our preliminary peritoneal fluid pilot study suggests further utility in using KRAS cfDNA in determining the status of peritoneal fluid in the absence of gross carcinomatosis. Longer follow-up is needed to confirm these findings and to determine whether KRAS cfDNA status can predict outcomes and treatment responses in patients undergoing neoadjuvant therapy.
19. MINIMALLY INVASIVE DISTAL PANCREATECTOMY REDUCES MAJOR MORBIDITY AND LENGTH OF STAY COMPARED TO OPEN: MULTINATIONAL VALIDATION OF A NEW GOLD STANDARD

S Klompmaker, T De Rooij, B Groot Koerkamp, A Shankar, U Siebert, MG Besselink, AJ Moser

Presenter: James Moser MD | Beth Israel Deaconess Medical Center

Background: A recent RCT on MIDP reported significant reduction in time to functional recovery and a nonsignificant 13% absolute risk reduction for major morbidity compared to ODP. We aimed to predict the population impact of minimally invasive distal pancreatectomy (MIDP) on major morbidity compared to open distal pancreatectomy (ODP).

Methods: Nationwide observational cohort study evaluating the association between surgical approach and composite major morbidity (CMM; death or severe complications) after elective distal pancreatectomy for tumors and cysts using ACS-NSQIP® (2014-2016) and external validation using Dutch Pancreatic Cancer Group (DPCG) data (2005-2016). Multivariable logistic regression assessed the impact of MIDP on CMM at varying implementation rates between (0%-100%), including conversion (0%-25%), using marginal effects.

Results: Of 2921 ACS-NSQIP® patients, 1359 (47%) underwent ODP, and 1562 (53%) underwent MIDP with 18% conversion. MIDP was independently associated with reduced 30-day major morbidity (OR 0.50, 95CI 0.42-0.60; p<0.001), confirmed upon subgroup analysis and external validation (n=637; p<0.003). Conversion mitigated this association in ACS-NSQIP® (OR 2.74, 95CI 2.05-3.65, P<0.001). The predicted absolute population risk reduction in ACS-NSQIP® was 12% (95CI 7.8-15) at 18% conversion and 15% (95CI 11-19%) at 0% conversion (Fig. 1).

Conclusion: MIDP was associated with a 12% absolute reduction in major morbidity compared to open in two nationwide datasets, confirming findings of a recent RCT. Lower conversion rates may further reduce major morbidity, especially at higher MIPD implementation rates.
20. RISK FACTORS FOR MULTI-DRUG RESISTANT BACTERIA INFECTION AMONG RECTAL CARRIERS SUBMITTED TO PANCREATODUODENECTOMY: A PROSPECTIVE OBSERVATIONAL STUDY

S Paiella, M De Pastena, AM Azzini, G Montagnini, M Maruccio, C Filippini, A Mazzariol, G Lo Cascio, E Tacconelli, C Bassi, R Salvia

Presenter: Salvatore Paiella MD | University of Verona

Background: With the worldwide diffusion of multi-drug resistant (MDR)-bacteria, prevention strategies and surveillance programs have become crucial. Here we aimed to identify risk factors for the development of infectious complication (IC) sustained by MDR-bacteria in a cohort of colonized patients who had undergone a pancreaticoduodenectomy (PD).

Methods: An observational study was conducted analyzing all consecutive PDs performed from January 2015 to December 2017. The study population was initially divided by the presence of MDR bacteria at the preoperative rectal swab (RS), then by the development of IC related to the MDR bacteria of whom the patient was a rectal carrier. The RS was typically performed two weeks before surgery. As regards perioperative antibiotic therapy, patients with a positive RS for a Gram negative Extended-spectrum beta-lactamase (ESBL)-producing bug received a single shot of 1 gr of Ertapenem (Invanz). Whereas, when a carbapenemase-producing bacterium was detected, a tailored antibiotic prophylaxis was performed based on the RS antibiogram. Patients with a negative RS received the standard treatment. An intraoperative bile culture was always performed, and then the RS and bile culture isolation were compared.

Results: During the study period a total of 742 PDs were performed. A total of 113 (18%) patients, representing the study population, had a positive RS for MDR-bacteria, of whom 42 patients (31.5%) developed an IC sustained by the same microorganisms. Male gender (OR = 1.6, 95%CI 1.5 - 16.6), number of biliary procedures (OR = 1.2, 95%CI 1.1 - 9.1), positive correlation between RS and intraoperative bile culture (OR = 1.4, 95% CI 1.3 - 12.1), and ICU admission (OR = 2.3, 95%CI 2.3 - 61.1) were independent predictors of ICs development in RS positive patients. The postoperative course of patients that experienced an MDR-bacteria sustained IC was dramatically worse than the one of rectal carriers only. In particular, the rates of abdominal collections (64.3% vs. 19.8%, p < 0.001), CR-POPF (35.7% vs. 8.8%, p < 0.001), post-pancreatectomy hemorrhage (28.6% vs. 5.5%, p < 0.001), major complications (33.3% vs. 9.9%, p = 0.001), reoperation (31% vs. 3.3%, p < 0.001), and mortality (23.8% vs. 3.3%, p = 0.001) were significantly higher in the subgroup of patients that developed an IC sustained by the MDR-bacteria found at the RS.
Conclusion: We identified some risk factors for the development of ICs sustained by MDR-bacteria after PD in rectal colonized patients. If further confirmed by other studies, this could allow recognizing a subgroup of high-risk patients, on whom to adopt preventive strategies. A prediction model might help to estimate and stratify the risk of ICs, to develop tailored perioperative treatment or mitigation measures, to ultimately improve the surgical outcomes and reduce the diffusion of MDR bacteria.

21. VARIATION IN THE SURGICAL MANAGEMENT OF LOCALLY ADVANCED PANCREATIC CANCER

BN Reames, AB Blair, RW Krell, JC Padussis, SP Thayer, M Falconi, CL Wolfgang, MJ Weiss, C Are, J He

**Presenter:** Bradley Reames MD, MS | University of Nebraska Medical Center

**Background:** Recent reports suggest patients with locally advanced pancreatic cancer (LAPC) may become candidates for curative resection following neoadjuvant therapy, with encouraging survival outcomes. Yet the optimal management approach for LAPC remains unclear. We sought to investigate surgeon preferences for the management of patients with LAPC.

**Methods:** An extensive electronic survey was systematically distributed by email to an international cohort of pancreas surgeons. Data collected included surgeon practice characteristics, preferences for staging and management, and 6 clinical vignettes (with detailed videos of post-neoadjuvant arterial and venous imaging) to assess attitudes regarding eligibility for surgical exploration.

**Results:** A total of 150 eligible responses were received from 4 continents. Median duration in practice was 12 years (IQR 6-20) and 75% respondents work in a university setting. Most (84%) are considered high volume, 33% offer a minimally-invasive approach, and 48% offer arterial resection in selected patients. Staging preferences varied widely, as 95% typically use a pancreas protocol CT, 59% use endoscopic ultrasound, 35% use MRI, and 17% use PET/CT. AHPBA/SSO/SSAT, NCCN, and the MD Anderson resectability criteria were most commonly used to assess

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*Adjusted for age, male gender, comorbidities, insurance, malignancy, and length of stay
vascular involvement. A majority (70%) always recommend neoadjuvant chemotherapy, and 62% prefer FOLFIRINOX. Preferences for duration of neoadjuvant chemotherapy varied widely: 39% prefer at least 2 months, 41% prefer at least 4 months, and 11% prefer 6 months or more. While 41% percent frequently recommend neoadjuvant radiotherapy, preferences for radiation type were mixed: 51% prefer external beam and 28% prefer stereotactic body radiotherapy. Age above 80 years and CA 19-9 greater than 1000 U/mL were commonly considered contraindications to exploration. In 5 clinical vignettes of LAPC, the proportion of respondents that would offer exploration following neoadjuvant therapy varied extensively, from 15% to 54%. In a vignette of oligometastatic pancreatic liver metastases, 32% would offer exploration if a favorable biochemical and imaging response to neoadjuvant therapy is observed.

**Conclusion:** In an international cohort of high volume pancreatic surgeons, there is substantial variation in attitudes regarding staging preferences and surgical management of LAPC. These results underscore the importance of coordinated multi-disciplinary care, and suggest an evolving concept of “resectability.” As a result, patients and providers should have a low threshold to consider a second opinion for the surgical management of LAPC, if desired.

**22. THE EFFECTS OF HIGH DOSE PANCREATIC ENZYME REPLACEMENT THERAPY ON BODY WEIGHT, NUTRITIONAL ASSESSMENT AND QUALITY OF LIFE AFTER PANCREATEODOUODENECTOMY**

_H Kim, YS Yoon, Y Han, W Kwon, SW Kim, E Kim, JR Kim, YJ Choi, JS Kang, M Lee, HS Han, DS Yoon, JS Park, SJ Park, SS Han, SE Lee, SH Choi, IW Han, JY Jang_

**Presenter:** Hongbeom Kim MD | Seoul National University Hospital

**Background:** Many patients with pancreatectomy are facing discomforts from malnutrition or deteriorating quality of life (QOL). Pancreatic exocrine insufficiency (PEI) can occur after pancreatectomy, leading to nutritional imbalance and weight loss. In addition to nutritional deficits, there are also changes in the QOL, such as steatorrhea, changes in bowel habit or flatulence. Pancreatic enzyme replacement therapy (PERT) are helpful for PEI patients. However, there was no consensus of PERT for EPI patients after pancreatectomy. Therefore, we aimed to find out the effect of PERT on body weight, bowel habit, nutritional status and QOL, in patients with PEI after
pancreaticoduodenectomy.

Methods: This randomized controlled, placebo-using, double-blind and multicenter trial compared effect of PERT to placebo. Patients were enrolled in 7 tertiary referral hospitals in South Korea. Among the patients who underwent pancreaticoduodenectomy regardless of benign or malignant diseases, the patients who were a fecal elastase level was 200 or less in preoperative or postoperative test were included in this study. The PERT group took single capsule of pancreatin (Norzyme) 40000 IU three times a day.

Results: Between October 2014, and September 2017, 304 patients were randomly assigned. The protocol-completed patients were 71 and 93, in PERT group and in control group, respectively. In per-protocol set, 1.09kg weight increased in PERT group for 3 months, on the other hands, 2.28kg weight reduction in placebo group. There was significant weight difference of 3.37kg (p < 0.001). However there was no difference in intention-to-treat set. (figure 1) Of the change of nutritional parameters, prealbumin showed significant difference (PERT: +10.9 mg/dL vs placebo: +7.8 mg/dL, p=0.002). And transferrin also show difference between PERT group and placebo group, however there was no statistical significance (+84 mg/dL in PERT vs +76.1 mg/dL placebo, p=0.063). Pre-operative high BMI and poor compliance of PERT were weight loss risk factors in univariate analysis. However most powerful weight loss risk factors was poor PERT compliance (HR: 4.018, p value < 0.001). There was no PERT effect in sub-category QOL scores as well as total score.

Conclusion: PERT as a nutritional support increase weight and nutritional parameters in post-operative PEI patients. Active education and monitoring are important to maximize effectiveness.
ORAL ABSTRACTS (continued)

23. SOMATIC MUTATIONS IN RESECTED SPECIMENS OF PANCREATIC CANCER WITH PATHOLOGICAL COMPLETE RESPONSE TO NEOADJUVANT THERAPY

L Yin, N Pu, A Hasanain, AB Blair, D Ding, VP Groot, JA Teinor, Y Wu, AA Javed, RA Burkhart, MJ Weiss, Y Miao, JL Cameron, J He, CL Wolfgang, J Yu

Presenter: Lingdi Yin MD | Johns Hopkins University School of Medicine

Background: A pathological complete response (pCR) in resected pancreatic ductal adenocarcinoma (PDAC) after neoadjuvant chemoradiation therapy is rather rare, but when present, it has been reported to be an indicator for improved survival after resection. We previously demonstrated that despite the improved survival some of the patients with pCR still suffer from early recurrence and disease specific mortality. These findings raise the possibility that pCR may not be equal to true complete response. This goal of this study was to determine whether there is the presence of cancer cells in pCR as defined by somatic mutations in the surgical resected specimen.

Methods: A retrospective review of a prospectively maintained database was performed at a single institution. pCR was defined as no viable cancer cells identified in the resected pancreas or lymph nodes by clinical pathology. Demographics and clinical data on neoadjuvant treatment and surgical resection were documented. Macro-dissection was performed on FFPE resected specimens to isolate DNA from regions of interest (ROIs) including fibrosis, normal duct, normal parenchyma, and undefined ductal cells. Targeted next-generation sequencing was used to detect mutations of KRAS, TP53, GNAS, and SMAD4. Overall survival (OS) and disease-free survival (DFS) were reported.

Results: Twenty-six pCR cases with available tumor specimens were identified between 2008 and 2017. One to seven blocks were investigated for ROIs for each case. 332 DNA samples were isolated from ROIs including 192 of fibrosis, 9 of the normal duct samples, 27 of normal parenchyma, and 104 of undefined ductal cells. Mutations were detected in some but not all samples. Neither normal parenchyma nor normal duct harbored any mutations. Eleven of 26 (42.3%) pCR cases were positive for a mutation of at least one cancer driver gene. One patient died from postoperative hemorrhage was excluded from the survival analysis. The median OS and DFS of the 25 pCR patients were 27 months and 26 months. Univariable logistic regression analysis showed associations between adjuvant therapy (p=0.02), GNAS mutation (p=0.04) and recurrence of pCR patients within three years from resection. Multivariable analysis showed that adjuvant therapy is an independent risk factor for recurrence of pCR patients (p=0.04). Univariable and multivariable Cox regression analysis of overall survival post surgery indicated that age ≥ 60 years (p=0.04) and TP53 mutation (p=0.02) are independent adverse
prognostic factors for pCR patients.

**Conclusion:** This is the first report so far suggesting that somatic mutations existed even in PDAC patients with pCR to neoadjuvant therapy, which could also be used to predict early recurrence and reduced survival. The current regression evaluation system of PDAC to neoadjuvant therapy needs to be re-assessed at a molecular level.

**24. TEXTBOOK OUTCOME AS A NOVEL COMPOSITE QUALITY MEASURE IN PANCREATIC SURGERY: A NATIONWIDE ANALYSIS**

*S van Roessel, TM Mackay, S van Dieren, GP van der Schelling, VB Nieuwenhuijs, K Bosscha, E van der Harst, RM van Dam, MS Liem, S Festen, MW Stommel, D Roos, F Wit, IQ Molenaar, VE de Meijer, IH de Hingh, HC van Santvoort, BA Bonsing, OR Busch, B Groot Koerkamp, MG Besselink*

**Presenter:** Stijn van Roessel MD, MSc | Amsterdam UMC

**Background:** Quality assurance programs are becoming increasingly popular in surgery but require objective assessment of surgical outcome. Textbook Outcome (TO) is a multidimensional measure, reflecting the ‘ideal’ surgical outcome. TO has shown to be a valuable indicator in other surgical fields, but has so far never been used in pancreatic surgery.

**Methods:** Patients who underwent pancreatoduodenectomy (PD) or distal pancreaticectomy (DP) for all indications between 2014-2017 were evaluated. Data were obtained from the Dutch Pancreatic Cancer Audit (DPCA), a mandatory nationwide registry. An international survey (24 experts, 10 countries, 4 continents) was conducted to reach consensus on the definition of TO in pancreatic surgery. Univariable and multivariable logistic regression was performed to identify predictors of TO. Between-hospital variation in TO rates were compared using observed-versus-expected rates, based on casemix-adjustment.

**Results:** Overall, 3341 patients were included, of whom 2633 (79%) underwent PD and 708 (21%) underwent DP. Based on the survey (92% response rate), TO was defined by the absence of postoperative pancreatic fistula, bile leak, postpancreatectomy hemorrhage (all ISGPS grade B/C), severe complications (Clavien-Dindo grade III or higher), readmission and
in-hospital mortality. The overall proportion of patients that achieved TO was 60.3%; 58.3% for PD and 67.4% for DP. On multivariable analysis, only class ASA 3 and 4 predicted a worse TO rate after PD (OR 0.59 [0.44-0.80] and OR 0.19 [0.04-1.02]), whereas a dilated pancreatic duct (>3mm) was associated with an improved TO rate (OR 2.70 [2.05-3.57]). For DP, a benign/premalignant diagnosis and the absence of neoadjuvant therapy was associated with a better TO rate (OR 1.48 [1.02 - 2.14] and OR 2.17 [1.03 - 4.59], respectively). When comparing institutions, the observed-versus-

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**Fig. 1** Generalized additive model of 90-day mortality vs preoperative serum bilirubin. Blue line is the regression line. Black dotted lines represent 95% confidence interval. Green dotted lines represent visual aid for determination of cutoff point at bilirubin 13 mg/dL. Red dotted lines represent user-selected points of serum bilirubin for odds ratio comparison. As shown on the graph, the odds ratio of death at bilirubin 25 mg/dL is 17.08 times greater than that at 1.2 mg/dL.
25. SOCIAL AND EMOTIONAL WELL-BEING IN AN HPB SURGERY PATIENT POPULATION

TP Yeo, RFogg, EP Kennedy, HLavu, JM Winter, SCannaday, CJ Yeo

Presenter: Theresa Yeo PhD | Thomas Jefferson University

Background: According to American Cancer Society statistics, 55,440 Americans will be diagnosed with pancreatic cancer; 12,190 will have gallbladder cancer or other biliary cancers in 2018. Incidentally-identified pancreatic cysts are present at rates that vary from 0.7% to 36.7%. Few studies have examined the relationship between baseline health related quality of life (HRQoL) measures and post-operative complications, and survival in pancreas, related cancers and benign HPB disorders. This investigation sought to document baseline quality of life parameters in a cohort of patients with HPB conditions and to assess for associations with social and emotional support, tumor biomarkers and treatment complications.

Methods: This IRB approved, non-interventional quality of life investigation was conducted using convenience sampling of individuals presenting for a new patient encounter at a large urban NIH-designated cancer center specializing in HPB surgery. HRQoL data were collected prospectively using the Brief Pain Inventory, Fact-Hepatobiliary and Facit-Fatigue scales. The questionnaires were completed between January 2013 and March 2018. These tools measure physical, social, emotional, functional and social well-being parameters, as well as pain and fatigue severity and intensity. Data on symptoms, clinical parameters and outcomes were collected from the electronic medical record. Patients who presented for an evaluation, but who did not have a cancer (i.e. benign HPB condition) and those who were not deemed eligible for surgery also completed these questionnaires and constitute comparison groups.

Results: Approximately 900 persons with a presumptive diagnosis of malignant or benign HPB conditions (pancreas, bile duct, gallbladder, duodenal cancers, or pancreatic neuroendocrine tumors, solid pseudopapillary neoplasms, IPMNs, pancreatic cysts, chronic pancreatitis and other unusual conditions), completed the questionnaires. Of these questionnaires, more than 400 had sufficient data for inclusion in the analysis. The sample was 84% Caucasian and 51% men with average age of 64.5 years. Seventy-six percent of the respondents presented with a new diagnosis; the most frequent being pancreatic cancer (55%), followed by pancreas cysts & IPMNs (17%), PNETS (7%), other HPB cancers (6%) and chronic pancreatitis (5%). Overall baseline HRQoL of the cohort was high as reported on the Fact-Hep, and fatigue bother scores and pain severity and intensity levels were
mild. However, social and emotional well-being were significantly poorer in the HPB cancer group ($p=.005$) as compared to the benign disease group, which also correlated with more baseline comorbid conditions. Pancreas cancer patients with prior unrelated cancers had the poorest physical and emotional well-being scores. Mild pain (48%), weight loss (38%), GI issues (38%), jaundice (33%), loss of appetite (27%) and fatigue (18%) were the leading presenting symptoms. Twelve percent reported pre-existing depression, anxiety, and addiction disorders. Eighty-four percent of the respondents were subsequently scheduled for surgery.

**Conclusion:** Patients with HPB cancers suffer from poorer HRQoL, in particular with regard to sadness, worry about death, family support, ability to enjoy life and nervousness, at the time of their first visit to a HPB specialty clinic. Quality of life concerns continue to be an important but often overlooked component of decision making regarding treatment options, potential risk of complications and positive outcomes. Previous research indicates that social and emotional well-being parameters improve in response to even small clinical improvements. Health care providers should be attuned to assessing the appropriate HRQoL parameters in determining which patients harbor unsuspected stress and need supportive intervention.
26. SYSTEMATIC REVIEW OF SURGICAL RESECTION OF PANCREATIC CANCER WITH SYNCHRONOUS LIVER METASTASES IN THE ERA OF MULTIAGENT CHEMOTHERAPY

S Crippa, R. Cirocchi, MJ Weiss, CW Michalski, M Reni, CL Wolfgang, T Hackert, M Falconi

Presenter: Stefano Crippa MD, PhD | San Raffaele Scientific Institute

**Background:** Recent studies considered surgery as a treatment option for patients with pancreatic ductal adenocarcinoma (PDAC) and synchronous liver metastases. The aim of this study was to evaluate systematically the literature on the role of surgical resection in this setting as an upfront procedure or following preoperative chemotherapy.

**Methods:** A systematic search was performed of PubMed, Embase and the Cochrane Library in accordance with PRISMA guidelines. Only studies with patients with synchronous liver metastases published in the era of multi-agent chemotherapy (after 2011) were considered, excluding those with lung/peritoneal metastases or methacronous liver metastases. Median overall survival (OS) was the primary outcome.

**Results:** Six studies with 204 patients were analyzed. The number of liver metastases was reported in three studies: 52% of patients had one metastases, 17.5% two liver metastases and the remaining 30.5% had three or more metastases. 63% of patients underwent upfront pancreatic and liver resection, 35% had surgery after preoperative chemotherapy and 2% had an inverse approach (liver surgery first). 54.5% of patients undergoing surgery following neoadjuvant therapy, underwent FOLFIRINOX as chemotherapy regimen. The radiological and biochemical evaluation before and after chemotherapy was reported in three studies and a significant decrease of CA 19.9 was required as a criteria for surgical exploration. Two studies defined the radiological response to preoperative chemotherapy based on the RECIST criteria. 38 patients (18.5%) did not undergo any liver resection since metastases disappeared after chemotherapy. In the three studies considering a strategy based only on neoadjuvant treatment, the median time from initial diagnosis to surgery ranged from 9.7 to 12 months. Postoperative mortality was low (<2%). Median OS ranged from 7.6 to 14.5 months after upfront pancreatic/liver resection, and from 39 to 56 months in those undergoing preoperative treatment.
Conclusion: In the era of multi-agent chemotherapy, upfront surgery does not offer a significant survival advantage and PDAC patients with oligo metastases to the liver should undergo primary chemotherapy. Surgical resection can be considered in selected patients with a clear and prolonged biochemical and radiological response to neoadjuvant chemotherapy, as they might benefit from subsequent resection. Prospective and multicenter studies are needed to confirm this trend, and to better identify the criteria to properly select patients for this innovative treatment, that currently still should not be considered as common clinical practice.

27. THE LAPAROSCOPIC APPROACH TO PANCREATICODUODENECTOMY IS COST NEUTRAL IN VERY HIGH-VOLUME CENTERS

E Eguia, PC Kuo, P Sweigert, MC Nelson, GV Aranha, G Abood, C Godellas, MS Baker

Presenter: Emanuel Eguia MD, MHA | Loyola University Medical Center

Background: Little is known regarding the impact of minimally invasive approaches to pancreaticoduodenectomy (PD) on the aggregate costs of care.

Methods: We queried the Healthcare Cost and Utilization Project State Inpatient Database to identify patients undergoing elective laparoscopic (LPD) or open (OPD) pancreaticoduodenectomy in FL, MD, NY, and WA between 2014 and 2016. Multivariable regression (MVR) was used to evaluate the association between surgical approach and rates of postoperative complication, overall lengths of stay (LOS) and aggregate costs of care including readmissions to 90 days following PD. Candidate variables were determined a priori using best variable subsets and included: age, gender, insurance, race/ethnicity, Charlson comorbidity index (CCI), pathology (benign vs. malignant), hospital PD volume broken to terciles (low: 127 PD/year) and overall LOS.

Results: 513 (10.8%) patients underwent LPD; 4,746 (89.2%) underwent OPD. On univariate analysis, patients undergoing LPD had higher CCI (5 vs. 3, p<0.001) and rates of readmission (35% vs. 30%, p=0.04), malignant pathology (89% vs. 76%; p<0.001), and aggregate costs of care ($41,669 vs. $37,580, p<0.02) than those undergoing OPD. On MVR adjusted for age, CCI, pathologic diagnosis, and hospital volume, LPD was associated with a lower risk of prolonged LOS (OR 0.77; 95% CI [0.61, 0.97]) but higher risk of readmission (OR 1.24; 95% CI [1.02, 1.51]) compared to OPD. Rates of perioperative morbidity and overall LOS for patients undergoing LPD were identical to those for patients undergoing OPD. On MVR adjusted for age,
pathology, CCI, LOS, and volume, factors associated with being in the highest quartile for aggregate costs of care included: male gender (OR 1.19; 95% CI [1.04, 1.37]), CCI (OR 1.07; 95% CI [1.03, 1.11]), black race (OR 1.41; 95% CI [1.12, 1.78]), Hispanic ethnicity (OR 1.28; 95% CI [1.05, 1.55]), Medicare insurance (OR 1.28; 95% CI [1.05, 1.55]), readmission (OR 4.44; 95% CI [3.87, 5.09]) and low hospital volume (OR 2.46; 95% CI [1.97, 3.06]) compared to patients in lower quartiles of cost. Patients undergoing LPD in low (+$9,390; 95% CI [$2,948, $15,831]) and moderate to high (+$5,579; 95% CI [$1,783, $9,376]) volume centers had higher costs than those undergoing OPD in the same centers. In very high-volume centers, aggregate costs of care for patients undergoing LPD were identical to those undergoing OPD in the same centers (+$616; 95% CI [-$1,703, $2,936]). (Table 1)

Conclusion: Rates of morbidity and overall LOS for patients undergoing LPD are statistically identical to those undergoing OPD. At low to moderate and high-volume centers, LPD is associated with higher aggregate costs of care relative to OPD whereas at very high-volume centers LPD is cost neutral. This finding suggests that very high-volume centers develop efficiencies of scale that mitigate costs inherent in the minimally invasive approach to PD.
ORAL ABSTRACTS (continued)

8. NATIONWIDE ASSESSMENT OF A RISK-STRATIFIED DRAIN PLACEMENT STRATEGY DURING PANCREATODUODENECTOMY USING THE MODIFIED FISTULA RISK SCORE

JM Cloyd, M Dillhoff, A Ejaz, A Tsung, TM Pawlik, D Xourafas

Presenter: Jordan Cloyd MD | The Ohio State University

Background: Recent studies on postoperative pancreatic fistula (POPF) prevention following pancreateoduodenectomy (PD) have proposed omission of perioperative drains for negligible/low-risk patients and early drain removal (≤POD3) for moderate/high-risk patients with POD1 drain amylase levels ≤5000 U/L. We sought to validate this algorithm using a nationwide cohort.

Methods: The ACS-NSQIP targeted pancreatectomy database from 2014-2016 was queried to identify patients who underwent PD. Patients were initially stratified as negligible/low- or intermediate/high-risk based on a previously validated modified fistula risk score (mFRS). First, the impact of drain placement on relevant postoperative outcomes was assessed. Second, the impact of early (≤POD3) versus late (≥POD4) drain removal was assessed among patients with POD1 drain amylase levels ≤5000 U/L.

Results: Among 6730 patients undergoing PD, 3375 (50%) were high-risk and 3355 (50%) were low-risk (Figure). Among high-risk patients, drain placement (n=3093, 92%) was associated with a higher rate of POPF (26%vs16%, p=0.0003), clinically relevant (CR)-POPF (20%vs12%, p=0.0015), and extended length of stay (LOS, 9vs7 days, p<0.0001), but less serious morbidity (27%vs33%, p=0.0325). Among 719 high-risk patients with POD1 drain amylase ≤5000 U/L, early drain removal (n=205, 29%) was associated with lower rates of POPF (3%vs18%, p<0.0001), CR-POPF (2%vs15%, p<0.0001), serious morbidity (12%vs20%, p=0.0059) and hospital LOS (7vs8 days, p<0.0001). Among low-risk patients, drain placement (n=2785, 83%) was associated with a higher rate of POPF (11%vs6%, p=0.0006) and extended LOS (8vs7 days, p<0.0001), yet lower overall (25%vs31%, p=0.0033) and serious (15%vs20%, p=0.0076) morbidity. Among 821 low-risk patients with POD1 drain amylase ≤5000 U/L, early drain removal (n=273, 33%) was associated with decreased rates of POPF (1%vs6%, p=0.0030), CR-POPF (1%vs5%, p=0.0142), serious morbidity (7%vs12%, p=0.0513) and LOS (6vs8 days, p<0.0001). On multivariate logistic regression, drain placement was independently associated with an increased odds of CR-POPF (High: OR 1.72, 95% CI 1.18-2.51; Low: OR 1.553, 95% CI 1.02-2.29) but a reduced incidence of
serious morbidity (High: OR 0.71, 95% CI 0.54-0.93; Low: OR 0.71, 95% CI 0.56-0.90) among both high- and low-risk patients. Similarly, early drain removal was independently associated with decreased odds of CR-POPF (High: OR 0.17, 95% CI 0.06-0.45; Low: OR 0.33, 95% CI: 0.11-0.99) and serious morbidity (High: OR 0.55, 95% CI 0.33-0.90; Low: OR 0.60, 95% CI 0.35-1.04) among both high- and low-risk patients with POD1 drain amylase ≤5000 U/L.

Conclusion: In this national cohort, the mFRS was unable to stratify patients relative to the need for selective drain placement during PD. For both high- and low-risk patients, perioperative drain placement was associated with increased rates of POPF, CR-POPF, and extended LOS but decreased incidence of serious morbidity, while early drain removal among patients with POD1 drain amylase ≤5,000 U/L was associated with reduced POPF, CR-POPF, serious morbidity, and extended LOS. These findings suggest that routine drain placement with early drain removal based on POD1 drain amylase levels...
29. ROLE OF SURGICAL RESECTION IN THE ERA OF FOLFIRINOX FOR ADVANCED PANCREATIC CANCER

Y Byun, H Kim, M Lee, YJ Choi, JS Kang, JR Kim, W Kwon, SW Kim, JY Jang

Presenter: Yoonhyeong Byun MD | Seoul National University Hospital

Background: One of the reasons for the dismal outcome of the treatment for pancreatic cancer is that only few patients are diagnosed early enough for it to be resectable. Conroy et al. reported the results of a highly effective fluorouracil plus leucovorin, irinotecan, oxaliplatin (FOLFIRINOX) treatment in patients with metastatic pancreatic cancer that led to a major change in the therapeutic paradigm. Recently, the number of attempts for active surgical resection after downstaging of advanced lesions is increasing; however, the effects of FOLFIRINOX, the role and timing of surgery after the FOLFIRINOX treatment, and the analysis on prognostic factors is limited.

Methods: Patients pathologically diagnosed with advanced pancreatic cancer who received the FOLFIRINOX chemotherapy at Seoul National University Hospital (SNUH) from January 2011 to December 2017 were reviewed retrospectively. Among them, 342 patients were finally appropriate for this study. All radiologic images were re-reviewed, the clinical stages was reclassified as resectable, borderline resectable, locally advanced, and metastatic. According to a regular multidisciplinary conference, each patients received FOLFIRINOX chemotherapy, and after 4-6 cycles of treatment, they were performed CT. Each cases were classified according to the RECIST criteria 1.1 for the assessment of a response. The eligibility criteria for surgical exploration are as follows: patient’s willingness for surgery, response to neoadjuvant treatment with the possibility of R0 resection, and radiologic control of distant metastasis with normalized tumor marker.

Results: A total of 68 (19.9%) patients had borderline resectable pancreatic cancer (BRPC), 126 (36.8%) locally advanced pancreatic cancer (LAPC), and 148 (43.3%) metastatic pancreatic cancer. Regarding the responsiveness to FOLFIRINOX treatment, PR was 18 (26.5%) in BRPC, 41 (32.5%) in LAPC, and 48 (32.4%) in metastatic stage according to the clinical stage. Those who achieved SD was 45 (66.2%) in BRPC, 69 (54.8%) in LAPC, and 73 (49.3%) in metastatic stage. The median survival period was significantly longer in the surgical group than in the nonsurgical group in each clinical stage: BRPC, 33 vs. 14 (p=.011); LAPC, 27 vs. 15 (p=.002), and metastatic pancreatic cancer, 34 vs. 13 months (p=.010). According to the response after
Conclusion: This study clearly showed that the surgical treatment greatly affects survival outcomes in advanced pancreatic cancer treated with FOLFIRINOX, even for the metastatic ones. Therefore, if the disease progression is controlled after FOLFIRINOX treatment in patients with advanced pancreatic cancer, including metastatic stage, surgical resection should be actively attempted to improve the survival outcome. As the tumor markers in our multivariate analysis were marginally significant, it could be considered to evaluate the resectability. In order to attempt curative resection, a factor that exerts great influence on the survival outcome, other treatment modalities that were proven to be more effective, such as radiotherapy, should be introduced. Future clinical studies are required to identify the optimal neoadjuvant regimens and indications of surgical resection in advanced pancreatic cancer.
30. DEFINING THE SAFETY PROFILE FOR PERFORMING PANCREATODUODENECTOMY IN THE SETTING OF HYPERBILIRUBINEMIA

B Chen, MT Trudeau, L Maggino, BL Ecker, LJ Keele, CM Vollmer

Presenter: Bofeng Chen BA | University of Pennsylvania

Background: Hyperbilirubinemia is a putative risk factor commonly observed in patients requiring pancreatoduodenectomy (PD). Thus far, the literature regarding the danger of operating in the setting of hyperbilirubinemia is equivocal. While relief of jaundice can be, and often is, achieved through biliary stenting, this process is now well recognized to be associated with worse outcomes – particularly infections. What remains undefined is at what specific level of bilirubin there is an adverse safety profile for undergoing PD.

Methods: From 2004-2018, 803 PDs performed at a single institution were studied from a prospectively collected database. Outcomes and resource utilization were compared across the full spectrum of serum bilirubin as measured as a continuous variable using multivariable generalized additive models with splice fitting. An optimal cutoff determination was based on a nonnegative odds ratio and increasing curve slope. Subset analysis of groups below and above the evident cutoff was conducted. Normal total bilirubin is defined as \( \leq 1.2 \text{ mg/dL} \) and mortality is defined at 90-days. The Postoperative Morbidity Index (PMI) was calculated using the Modified Accordion Severity system.

Results: Median bilirubin was 0.90 mg/dL, with normal total bilirubin present in 501 patients (62.4%). Altogether, 379 patients (47.2%) had a diagnosis of pancreatic cancer, 368 (45.8%) presented with jaundice, and 317 (39.5%) received a biliary stent. Outcomes included 90-day mortality (20 patients, 2.5%), overall complications (Accordion grade 1-6, 541 patients, 67.4%), severe complications (Accordion grade 3-5, 125 patients, 15.6%), pancreatic fistula (94 patients, 11.7%), and LOS (median = 8 days). Bilirubin was significantly associated with an increase in 90-day mortality (\( p=0.043 \)), cumulative number of complications (\( p<0.001 \)), reoperation (\( p=0.010 \)), and LOS (\( p<0.001 \)). The cutoff for mortality was discerned to be 13 mg/dL (Figure 1), with 44 patients (5.48%) above this threshold. Mortality below and above 13 mg/dL were 1.7% and 15.9%, respectively (\( p<0.001 \)). PMI below and above the threshold were 0.188 and 0.298, respectively, and PMI per complication were 0.294 and 0.438, respectively. Patients above the threshold had significantly lower albumin (3.15 vs 3.85, \( p<0.001 \)) and higher
INRs (1.22 vs 1.09, p=0.018). Multivariate analysis including these and other variables suggestive of mortality confirmed that bilirubin $\geq 13$ mg/dL, age, smoking status, estimated blood loss (EBL), and pancreatic cancer histology were significant predictors of increased mortality. There was also a positive relationship between increasing bilirubin and use of antibiotics and total parenteral nutrition (TPN). Distinct bilirubin cutoffs for overall complication, severe complication, and readmission were not evident, but thresholds for greater LOS and cumulative number of complications occurred at 18 mg/dL.

**Conclusion:** A preoperative serum bilirubin value of 13 mg/dL demonstrates a threshold level that increases mortality after PD. In the rare, but high-stakes, case of a patient with deep jaundice above this level, normalization of bilirubin should be considered before proceeding to surgery. These results define a point when direct operative intervention should be delayed in order to optimize patients with hyperbilirubinemia who require PD.

**Figure 1.** Recursive partitioning using 4 of the 41 analytes can help distinguish between pancreatic ductal adenocarcinoma (PD), pancreatitis (Pa), and other benign conditions (Be).
31. MULTI-INSTITUTIONAL DEVELOPMENT AND EXTERNAL VALIDATION OF A NOMOGRAM TO PREDICT RECURRENTRESCURENCE AFTER CURATIVE RESSECTION OF PANCREATIC NEUROENDOCRINE TUMORS


Presenter: Alessandra Pulvirenti MD | University of Verona

**Background:** Among patients undergoing resection of pancreatic neuroendocrine tumors (PanNETs), approximately 17% of experience disease recurrence. It is not established which patients are at risk of recurrence, with no consensus on the optimal follow-up. Aim of this study was to develop a predicting nomogram to estimate the risk of recurrence at 5 years after curative surgery for localized G1/G2 PanNETs.

**Methods:** A multi-institutional database of patients with G1/G2 treated at Verona University Hospital and Memorial Sloan Kettering Cancer Center was used to develop a nomogram to estimate the rate of freedom from recurrence at 5-years after curative surgery. A second cohort including patients treated at Johns Hopkins Hospital, Glasgow Royal Infirmary, and Royal North Shore Hospital was used for nomogram validation. Prognostic factors were assessed by univariate analysis using Cox regression model. The nomogram was internally validated with bootstrapping and on an external cohort. The performance was assessed by concordance index (c-index) and a calibration curve.

**Results:** The nomogram was constructed using a cohort of 632 patients. The median age was 56 years old, 68% tumor was G1, and the median follow-up was 51 months. Among survivors, we observed 74 recurrences at the time of the study. Variables included in the nomogram were the number of positive nodes, tumor size, Ki-67 and presence of vascular/perineural invasion. The model had a c-index of 0.85 on the internal cohort improving the stratification provided by AJCC/ENETS staging scheme (c-index 0.76 and 0.79 respectively). On the external The performance of 328 patients, the c-index was 0.84.

**Conclusion:** We presented an externally validated nomogram that predicts
the probability of recurrence-free at five years after curative resection of PanNETs. This model improves risk estimation by the current TNM staging systems. It may help physicians, and patients better to understand the risk of recurrence and to develop personalized surveillance program.
Background: Despite preclinical studies demonstrating that an immune response can be generated against pancreatic cancer, current immunotherapeutic strategies have not been successful at changing the course of the disease. Thus, there is need to evaluate novel strategies to elicit an anti-tumor response. In the current study, we evaluate the ability of bacterial lipopolysaccharide (LPS) to provoke an immune response against pancreatic cancer in mice.

Methods: KPC pancreatic cancer cells were injected into the pancreata of C57BL/6 mice to induce tumors that were subsequently resected. Following resection, the mice were randomized to receive LPS (5mg/kg) or vehicle twice weekly by intraperitoneal injection and then followed for cancer recurrence. In a separate experiment, KPC cancer cells were injected into the spleens of C57BL/6 and Rag1-knockout mice to induce liver metastases. Following intra-splenic injection, the mice were randomized to receive LPS (1mg/kg) or vehicle twice weekly. Liver metastases were measured at the endpoint, and immunophenotyping was performed by flow cytometry. Finally, subcutaneous tumors were induced using MC38 colon cancer and Braf-Pten melanoma cell lines. The mice were randomized to receive either LPS or vehicle twice weekly, and tumor volumes were serially measured.

Results: Treatment with LPS significantly reduced cancer recurrence following resection of pancreatic tumors, and the median survival for the LPS-treated mice was more than double that of the vehicle-treated mice. LPS treatment drastically suppressed liver metastasis in immunocompetent C57BL/6 mice, but the effect of LPS was abrogated in the absence of the adaptive immune system in Rag1-knockout mice that lack mature T and B cells. We observed that, in addition to promoting the classically activated (M1) macrophage phenotype, there was a significant reduction in the pro-tumorigenic myeloid-derived suppressor cell (MDSC) populations, which are known to suppress T cell activity. LPS treatment decreased the growth of colon cancer and melanoma, suggesting that this strategy can be effective in other cancers as well.
Conclusion: Our findings demonstrate that LPS can stimulate the adaptive immune system to suppress the progression of pancreatic cancer. Elucidating the mechanism by which this anti-tumor response is triggered by LPS, and possibly even other pathogen-associated molecular patterns (PAMPs), could lead to identification of novel targets for activating the immune system against cancer, either alone or in combination with contemporary immunotherapeutic strategies.

33. IRREVERSIBLE ELECTROPORATION ACTS AS AN IN SITU VACCINE IN A MURINE PANCREATIC CANCER MODEL

JS Shankara Narayanan, P Ray, A Miller, T Hayashi, SP Schoenberger, RR White

Presenter: Jayanth Shankara Narayanan PhD | University of California San Diego

Background: Most pancreatic cancer (PC) patients either present with metastatic disease or develop distant metastatic disease despite treatment of localized disease. PC has only a moderate mutational burden, which along with the notoriously immunosuppressive PC microenvironment, contributes to its poor response to checkpoint inhibitor therapy. Irreversible electroporation (IRE) is a non-thermal method of inducing cell death that is currently being used clinically for selected patients with locally advanced PC. We hypothesize that IRE can induce an in situ vaccination effect against PC which can then be augmented by combination immunotherapy to achieve systemic anti-tumor immunity.

Methods: An immunocompetent mouse model of PC was established in male C57BL/6 mice using the KPC-luc-4580 cell line, derived from a spontaneous tumor that developed in a male LSL-KrasG12D/+; LSL- Trp53R172H/+; PDX1Cre/+; LSL-ROSA26 Luc/+ mouse. We utilized the ECM 830 square wave pulse generator to deliver IRE (100 msec pulses of electricity at 1500 V/cm using a two-needle array probe, separated by 5 mm) to subcutaneous (SQ) tumors measuring 5-7 mm in diameter. In addition to effects on local tumor growth, tumor microenvironmental changes were analyzed using immunohistochemistry and flow cytometry. Tumor-specific mutations were identified using exome and RNA sequencing and used to study tumor-specific T cell responses in IRE-treated mice.
**Results:** IRE alone consistently produced complete regression of SQ KPC tumors in approximately 20%-30% of mice while 50% – 60% of mice remained tumor-free after surgical tumor resection. However, when tumor-free mice from both groups were rechallenged with tumor cells injected into the contralateral flank, 5/5 mice from IRE group prevented growth of the secondary tumors in contrast to only 1/5 mice in the resection group (Figure). This confirmed that an adaptive anti-tumor immune response is induced after IRE, characterized by a significant increase in the tumor infiltrating CD8+ cytotoxic T cells. Furthermore, these post-IRE CD8+ T cells were more reactive against KPC tumor specific mutant neoantigen peptides than non-IRE CD8+ T cells, and conferred protection against tumor challenge when adoptively transferred into immunocompromised, tumor-naïve mice. Combining IRE with systemic anti-PD-1 antibody therapy improved immune infiltration within the local tumor microenvironment significantly compared to anti-PD1 alone (P<0.01). However, the best outcome was achieved by combining IRE with anti-PD1 and TLR7 agonist (1V270) which induced complete tumor regression in 4/7 mice with all of them demonstrating protective immunity against secondary tumor rechallenge. Most significantly, this combination resulted in the complete elimination of the untreated concomitant distant tumors in the group (abscopal effect), which IRE as a monotherapy failed to achieve. We observed a significant decrease in immunosuppressive Myeloid Derived Supressor Cells MDSCs (CD11b+, CD11c-, GR1+) at one week in the combination group. We also saw a profound shift in the ratio of immunostimulatory M1 (MHC II+, CD206lo):immunosuppressive M2 (MHC II-, CD206hi) macrophages in the combination group and a modest increase in CD8+ T cells with IRE alone that was almost doubled in the combination.

**Conclusion:** Using an immunocompetent mouse model, we have shown that IRE, with appropriate combination strategies, can achieve successful therapeutic systemic anti-tumor immunity. IRE was more effective than surgical resection in producing systemic immunity. Local delivery of 1V270 or other innate immune stimulators at the time of IRE would be highly feasible and warrants clinical investigation (alone or in combination with checkpoint inhibition) as a way to address micrometastatic disease. Ultimately, a clinical strategy combining IRE with agents that target both the adaptive and innate immune systems will likely be necessary to prevent recurrence in this recalcitrant disease.

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**Table 1: Pathogenic and likely pathogenic variants with available targeted therapy.**

<table>
<thead>
<tr>
<th>Gene</th>
<th>Number of patients with variant (%)</th>
<th>Agent Available</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATM</td>
<td>1 (2%)</td>
<td>Carboplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cisplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oxaliplatin</td>
</tr>
<tr>
<td>FH</td>
<td>1 (2%)</td>
<td>No agent available</td>
</tr>
<tr>
<td>TP53</td>
<td>6 (12%)</td>
<td>No agent available</td>
</tr>
<tr>
<td>ARID1A</td>
<td>3 (6%)</td>
<td>No agent available</td>
</tr>
<tr>
<td>ATM</td>
<td>8 (16%)</td>
<td>Carboplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cisplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oxaliplatin</td>
</tr>
<tr>
<td>BRCA1</td>
<td>2 (4%)</td>
<td>Carboplatin</td>
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<tr>
<td></td>
<td></td>
<td>Cisplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oxaliplatin</td>
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<tr>
<td></td>
<td></td>
<td>Mitomycin C</td>
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<tr>
<td></td>
<td></td>
<td>Olaparib</td>
</tr>
<tr>
<td>BRCA2</td>
<td>1 (2%)</td>
<td>Carboplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cisplatin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oxaliplatin</td>
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<td></td>
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<tr>
<td></td>
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<td>Olaparib</td>
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<tr>
<td>CDKN2A</td>
<td>9 (18%)</td>
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<tr>
<td>GNAS</td>
<td>1 (2%)</td>
<td>No agent available</td>
</tr>
<tr>
<td>KRAS</td>
<td>32 (65%)</td>
<td>No agent available</td>
</tr>
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</table>
CIRCULATING TUMOR CELL DYNAMICS ARE ASSOCIATED WITH OUTCOMES IN PANCREATIC DUCTAL ADENOCARCINOMA: UPDATES ON THE PROSPECTIVE CLUSTER TRIAL

A Hasanain, AA Javed, VP Groot, AF van Oosten, G Gemenetzis, D Ding, JA Teinor, MJ Wright, J Yu, LD Wood, RA Burkhart, JL Cameron, MJ Weiss, J He, CL Wolfgang

Presenter: Alina Hasanain MD | Johns Hopkins University School of Medicine

Background: Given the consistently poor long-term survival in patients with localized pancreatic ductal adenocarcinoma (PDAC), there is a pressing need for better treatment strategies for this disease. Precision medicine approaches have the potential to improve outcomes. However, non-invasive biomarkers capable of providing longitudinal, real-time assessment of treatment response will be required. Recently, circulating tumor cells (CTCs) have emerged as a promising biomarker for PDAC. A prospective observational study on CTC dynamics, called the CLUSTER trial, is underway at our institution, and the short-term results were reported at the 2018 American Surgical Association. Herein, we present the updated results from this cohort.

Methods: The CLUSTER study is a prospective observational study of CTC dynamics in PDAC (NCT02974764). The study enrolled a total of 200 patients, and 64 were excluded for various reasons. A total of 136 with a confirmed diagnosis of PDAC underwent resection and were included. Serial peripheral blood samples were collected; CTCs were isolated with the Isolation by Size of Epithelial Tumor Cells (ISET; Rarecells) assay and identified and characterized using immunofluorescence.

Results: The median age was 62 (interquartile range (IQR): 59-74) years and 65 (48%) patients were female. Fifty-seven patients (41.9%) received chemotherapy prior to resection. The median tumor size was 3.0 (IQR: 2.2-4.2) cm and 82 (60%) patients had nodal disease. A total of 565 samples were collected and analyzed. Two CTC subtypes were identified in this cohort: epithelial CTCs (eCTCs) and epithelial/mesenchymal CTCs (mCTCs). At the time of preoperative sampling, 132 patients (97.1%) had eCTCs and 92 patients (67.7%) had mCTCs. Total CTCs (tCTCs) (11[6-15] vs. 7[3-10], p=0.007), eCTCs (9[5-12] vs. 6[5-9], p=0.007), and mCTCs (2[1-3] vs. 1[0-2], p=0.034), were lower in patients who received neoadjuvant chemotherapy than in those who were chemo-naïve at resection. Resection of the primary tumor led to a reduction of total CTCs and subtypes (all p<0.001). The median length of follow-up of the patients was 15.5 (IQR: 9.4-20.9) months, and the median overall survival (OS) was 19.7 (IQR: 11.7-27.1) months. At the time of last follow-up, 80 (58.8%) had recurrence of disease, and the median disease free survival (DFS) was 13.3 (7.5- not yet reached) months. Presence of mCTCs was found to be significantly associated with DFS (hazard ratio...
(HR): 1.88, 95%CI: 1.13-3.13, p=0.015). Longitudinal changes in CTC number and proportion of subtypes were observed before recurrence. Interestingly, a subset of patients had persistent CTCs 1 year beyond surgical resection in the absence of clinical relapse.

**Conclusion:** The results of this follow-up of a large prospective trial of PDAC patients demonstrates that CTC characteristics and dynamics are associated with patient outcomes. This further reinforces the role of CTCs as a biomarker that can potentially be integrated into clinical practice to allow for precision medicine approaches to treat patients with PDAC.

**35. THE PRESENCE OF STEM CELL PHENOTYPE CIRCULATING TUMOR CELLS IN PANCREATIC CANCER IS ASSOCIATED WITH AGGRESSIVE TUMOR BIOLOGY**

*AF van Oosten, AA Javed, A Hasanain, K Poruk, VP Groot, G Gemenetzis, D Ding, LD Wood, JA Teinor, RA Burkhart, JL Cameron, MJ Weiss, J He, J Yu, CL Wolfgang*

**Presenter:** Floortje van Oosten MD | Johns Hopkins University School of Medicine

**Background:** Recently, circulating tumor cells (CTCs) have emerged as a potential biomarker in patients with pancreatic ductal adenocarcinoma (PDAC). It has been demonstrated that CTC characteristics, including expression of epithelial, mesenchymal (transitional) and stem cell markers (e.g. pan-CK, vimentin, aldehyde-dehydrogenase (ALDH)), correlate with patient outcomes. It has been hypothesized that CTCs are potential seeds for metastasis; however, the underlying mechanisms are poorly understood. The aim of this study was to stratify CTCs into subpopulations based on the presence of a stem cell phenotype and compare patient outcomes between populations.

**Methods:** Preoperative peripheral blood samples were collected from 56 patients undergoing surgical resection for PDAC. CTCs were isolated through the Isolation by Size and Epithelial Tumor (ISET; Rarecells) system. Immunofluorescence was used to characterize cells; epithelial CTCs (eCTCs) expressing cytokeratin alone, transitional CTCs (trCTCs) co-expressing cytokeratin and ALDH, stem cell CTCs (stem-CTCs) expressing ALDH alone, and patients without CTCs. Based on the CTC-categories present, patients were stratified into four phenotypes. The transitional phenotype included patients with either eCTCs and trCTCs, or trCTCs alone. Lastly, patients with trCTCs alone, trCTCs and stem-CTCs, or those with eCTCs, trCTCs and stem-CTCs were included in the stem cell phenotype. Differences in overall survival
ORAL ABSTRACTS (continued)

(OS) and disease free survival (DFS) for each population were evaluated using Kaplan-Meier curves.

**Results:** A total of 56 patients were included in the study, of whom 13 (23%) did not have any CTCs. Of the 23 patients (41%) included in the transitional phenotype cohort, 19 were found to have trCTCs alone, and 4 had a combination of eCTCs and trCTCs. The remaining 20 (36%) were included in the stem cell phenotype. This comprised five patients who had all three CTC phenotypes, and 15 patients who were found to have both trCTCs and mCTCs. No patients in the study population had eCTCs alone or stem-CTCs alone in their blood samples. There was no difference in clinicopathological features between the groups (all p>0.05). Patients included in the stem cell phenotype cohort were found to have a shorter OS compared to patients with no CTCs (Median OS: 32.8 (interquartile range (IQR): 22.8-34.2) months vs. 14.0 (IQR: 8.8-20.2) months, p=0.017). No significant difference was observed in the OS between the no CTC population and transitional phenotype population (Median OS: 32.8 (IQR: 22.8-34.2) months vs. 23.5 (IQR: 13.8-39.9) months). The 2yr-OS of the no CTCs, transitional and stem cell cohorts was 66%, 47%, and 25%, respectively. Similar trends were observed for DFS. Patients in the stem cell phenotype cohort were found to have a significantly shorter DFS compared to the no CTCs cohort (Median OS: 29.2 (IQR: 12.6-30.1) months vs. 9.7 (IQR: 1.7-13.4) months, p=0.017). No significant difference was observed in the DFS between the no CTCs population and the transitional population (Median OS: 29.2 (IQR: 12.6-30.1) months vs. 14.4 (IQR: 8.1-24.7) months). The 2 yr-DFS of the no CTCs, transitional and stem cell cohorts was 62%, 35%, and 9% respectively.

**Conclusion:** CTCs expressing a stem cell phenotype show a more aggressive tumor biology and are correlated with worse OS and DFS. These results are consistent with the hypothesis that these stem-CTCs are important for establishing distant metastases. The majority of failures in patients with PDAC are due to metastatic relapse. The ability to characterize stem-CTCs may provide an opportunity to develop systemic therapies targeting cells that drive systemic recurrence. Work is underway to test this hypothesis.
**36. OUTCOME OF PATIENTS WITH BORDERLINE RESECTABLE PANCREATIC CANCER IN THE CONTEMPORARY ERA OF NEOADJUVANT CHEMOTHERAPY**

**AA Javed, MJ Wright, A Siddique, AB Blair, D Ding, RA Burkhart, MA Makary, JL Cameron, A Narang, J Herman, L Zheng, D Laheru, MJ Weiss, CL Wolfgang, J He**

**Presenter**: Ammar Javed MD | Johns Hopkins University School of Medicine

**Background:** Approximately, 20% of patients with pancreatic ductal adenocarcinoma have resectable disease at diagnosis. Given improvements in locoregional and systemic therapies, some patients with borderline resectable pancreatic cancer (BRPC) can now undergo successful resection. The outcomes of patients with BRPC after neoadjuvant therapy remain unclear.

**Methods:** A prospectively maintained single-institution database was utilized to identify patients with BRPC who were managed at the Johns Hopkins Pancreas Multidisciplinary Clinic (PMDC) between 2013 and 2016. BRPC was defined as any tumor that presented with radiographic evidence of the involvement of the portal vein (PV) or superior mesenteric vein (SMV) that was deemed to be technically resectable (with or without the need for reconstruction), or the abutment (<180° involvement) of the common hepatic artery (CHA) or superior mesenteric artery (SMA), in the absence of involvement of the celiac axis (CA). We collected data on treatment, the course of the disease, resection rate, and survival.

**Results:** Of the 866 patients evaluated at the PMDC during the study period, 151 (17.5%) were staged as BRPC. Ninety-six patients (63.6%) underwent resection. Neoadjuvant chemotherapy was administered to 142 patients (94.0%), while 78 patients (51.7%) received radiation therapy in the neoadjuvant setting. The median overall survival from the date of diagnosis, of resected BRPC patients, was 28.8 months compared to 14.5 months in those who did not (p < 0.001). Factors associated with increased chance of surgical resection included lower ECOG performance status (p = 0.011) and neck location of the tumor (p = 0.001). Forty-seven patients with BRPC (31.1%) demonstrated progression of disease; surgical resection was attempted and aborted in 12 patients (7.9%). Eight patients (5.3%) were unable to tolerate chemotherapy; six had disease progression and two did not want to pursue surgery. Lastly, four patients (3.3%) were conditionally unresectable due to medical comorbidities at the time of diagnosis due to comorbidities and failed to improve their status and subsequently had progression of the disease.
Conclusion: After initial management, 31.1% of patients with BRPC have progression of disease, while 63.6% of all patients successfully undergo resection, which was associated with improved survival. Factors associated with increased likelihood of surgical resection include lower ECOG performance status and tumor location in the neck.
37. PREDICTIVE VALUE OF CYTOKINE PROFILES FROM FINE NEEDLE ASPIRATES FOR THE DIAGNOSIS OF PANCREATIC DUCTAL ADENOCARCINOMA

PW Underwood, MH Gerber, D Delitto, K Nguyen, S Han, JG Trevino, RM Thomas, WE Gooding, SJ Hughes

Presenter: Patrick Underwood MD | University of Florida

Background: Endoscopic ultrasound with fine needle aspiration (FNA) is a primary modality for tissue acquisition and diagnosis in patients with pancreatic ductal adenocarcinoma (PDAC) but fails to provide adequate tissue for a diagnosis in ~25% of patients. Thus, many patients may require additional biopsy attempts, undergo resection without definitive diagnosis, and/or face exclusion from clinical trials. We hypothesized that soluble protein concentrations from a FNA biopsy can differentiate PDAC from benign conditions.

Methods: Tissues were collected from resected surgical specimens from 82 patients with PDAC, 27 patients with pancreatitis, and 50 patients with other benign pancreatic pathology. FNA was performed on resected specimens in a subset of these patients (41 PDAC, 6 pancreatitis, and 6 other benign pancreatic pathology). Homogenates were made from both whole tissue and FNA samples and subsequently were analyzed for soluble cytokines using a 41-plex protein assay and normalized to total protein. Data were then logged and standardized for a mean of 0 and standard deviation of 1. Statistical analyses were performed to assess for cytokines that discern PDAC.

Results: The mean protein concentration obtained by FNA was 10.4 mg/ml and 30.6 mg/ml for 22-gauge and 19-gauge needles (p = 0.03), respectively. The mean protein concentration in tissue homogenates was 1.49 mg/ml in PDAC, 1.46 mg/ml in pancreatitis, and 2.65 mg/ml in other benign tissue. To distinguish PDAC from both pancreatitis and benign tissue, variable importance was evaluated by area under the ROC curve (AUC) analysis for each of 31 analytes with complete data. Of these 31 proteins, 8 had an AUC of >0.80 with the AUCs for IL-1RA, MIP-1B, MDC and IP-10 exceeding 0.90. A recursive partitioning model based on four of the analytes IL-1RA, IL-12 p70, EGF, and IL-12 p40, classified the three tissue types (PDAC, pancreatitis, or benign pathology) with 84.5% accuracy (Figure 1). An IL-1RA level >0.076 was seen in 70 of the PDAC samples and only 3 benign samples. On the other hand, low IL-1RA along with IL-12 p70 <0.404, was observed predominantly in benign tissue. When distinguishing between PDAC and non-cancer tissues, recursive partitioning based on splitting only IL-1RA provided an accuracy of 89.4% and specificity of 96%; correctly predicting 70/84 PDAC cases and 74/77 benign cases. A penalized logistic regression model had a re-substitution classification accuracy of 95% for distinguishing PDAC from...
Conclusion: Taken together, these data demonstrate that analysis of soluble proteins obtained by FNA has the potential to differentiate PDAC from non-malignant pancreatic tissue. A clinical trial designed to prospectively validate these data and assess value in the setting of a non-diagnostic FNA is warranted.

Rate of cholecystectomy, performed during hospitalizations for mild acute biliary pancreatitis (NIS 2000-2012; n=511,468)
ORAL ABSTRACTS  (continued)

38. PANCREATIC FLUID INTERLEUKIN-1B COMPLEMENTS PROSTAGLANDIN E2 AND SERUM CA19-9 IN PREDICTION OF INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM DYSPLASIA

RE Simpson, MT Yip-Schneider, KF Flick, H Wu, CL Colgate, CM Schmidt

Presenter: Rachel Simpson MD | Indiana University School of Medicine

Background: Distinguishing between high- and low-risk intraductal papillary mucinous neoplasms (IPMN) is an important but challenging task. Two inflammatory mediators in pancreatic cyst fluid, interleukin-1β (IL-1β) and prostaglandin E2 (PGE2), have been individually suggested as indicators of IPMN dysplasia. We sought to determine if IL-1β and PGE2 together with serum CA19-9 could better predict high-grade (HGD) and invasive IPMN.

Methods: Pancreatic cyst fluid (n=92) collected at the time of endoscopy or surgery (2003-2016) was analyzed for PGE2 and IL-1β by ELISA. Patients underwent surgical resection with pathology-proven IPMN. Threshold values of PGE2 (>1100pg/mL), IL-1β (>20pg/mL), and serum CA 19-9 (>36U/mL) were used to calculate predictive metrics. Biomarker levels were compared using Wilcoxon rank-sum test and receiver operating characteristic curve analysis.

Results: Levels of IL-1β were higher in HGD/Invasive-IPMN (n=42) compared to Low/Moderate-IPMN (n=37) (median:range 54.6: 0-2671 vs. 5.9: 0-797pg/mL; P<0.001; AUC 0.766). Similarly, PGE2 was higher in HGD/Invasive-IPMN (n=45) compared to Low/Moderate-IPMN (n=47) (median:range 1790: 20-15180 vs. 140: 10-14630pg/mL; P<0.001; AUC 0.748). Presence of elevated PGE2 AND IL-1β (AUC 0.79) provided greater Specificity (89%) and equal PPV (82%) compared to IL-1β alone for HGD/Invasive-IPMN. Elevated serum CA19-9 AND PGE2 AND IL-1β provided 100% Specificity and PPV for HGD/Invasive-IPMN.

Conclusion: Cyst fluid PGE2, IL-1β, and serum CA19-9 are complementary in optimizing Specificity and PPV for detection of HGD/Invasive-IPMN, allowing clinicians to be more certain of the presence of a high-risk lesion requiring resection. These biomarkers may serve as the beginning of a panel of markers to predict IPMN dysplasia.
39. QUALITY OF LIFE AND GLOBAL HEALTH AFTER Pancreatic Surgery Is Consistent With the General Population: The Long-Term Outlook from 9 Years

MD Kluger, I Rossmer, K Shaw, V Rosario, BA Schrope, JA Chabot

Presenter: Michael Kluger MD, MPH | Columbia University

Background: Pancreatic surgery is increasingly performed for prophylaxis in otherwise healthy individuals. Informed consent requires that patients understand the possible and probable long-term consequences of surgery. There is a scarcity of such information, as most quality of life and health surveys are completed soon after recovery.

Methods: Only patients surviving greater than 5 years from pancreatic surgery as of January 2014 for benign or malignant indications were administered validated instruments (EORTC-QLQ-C30, EORTC-QLQ-PAN26, ADDQoL) and one designed to query specific information about hospitalizations, operations, nutrition and diabetes management following surgery. Patients were accrued from a prospectively maintained institutional database. Instruments were scored according to their manuals, and scores compared with demographic, peri-operative, health and behavioral variables. Patients operated for pancreatitis were excluded. 64 consented individuals completed all surveys. 55% were female, 80% were Caucasian, and the median age at surgery was 65.7 years (IQR:56.4,70.1).

Results: The median follow-up from surgery was 9.3 years (IQR:8.7,9.9). 44% underwent a Whipple, 28% a distal, 17% a total and 11% a central. 48% had cystic neoplasms, 39% adenocarcinoma and 13% neuroendocrine tumors. 84% had greater than a high school education, 89% were married/widowed, and only 6% were on disability. 19% of patients not having a total pancreatectomy developed diabetes following surgery, of which 40% were insulin controlled. 9% developed DM-related complications. QLQ scores range from 0-100, with 100 indicating best quality of life, health or function. For symptom scales, 0 indicates the best score. The median QLQ-C30 global quality of life/health score was 75 (IQR:67,92) and was not significantly different by operation (p=0.84), pathology (p=0.89), difficulty maintaining weight (p=0.98), nutritional supplement requirements (p=0.12), enzyme replacement therapy (p=0.35), or diabetes (p=0.79). The median QLQ-C30 functional scale was 88 (IQR:82,93), and symptom scale score was 13 (IQR:6,23). On the QLQ-PAN-26, the median abdominal/back pain symptom score was 8 (IQR:0,17), median score combining flatulence, BM frequency and urgency symptoms was 33 (IQR:11,44), the median score combining food and...
drink restrictions was 8 (IQR:0,17). None of the QLQ scores were significantly affected by Clavien III/IV complications. For patients with diabetes, the ADDQoL quality of life score was very good (IQR:excellent,good) on a 7 point scale (excellent to extremely-bad), yet they felt their life would be much better (IQR:very-much-better, little-better) on 5 point scale (very-much-better to worse). The average weighted impact score of diabetes on patients’ lives was -1(IQR:-3,-1), scaled -9(maximum-negative-impact-of-diabetes) to +3(maximum-positive-impact-of-diabetes). There were no significant differences in these scores whether diabetes developed before or after surgery (p=0.72), or whether a total pancreatectomy was performed (p=0.5).

**Conclusion:** At a median of 9 years from pancreatic surgery for benign or malignant conditions, the global health and quality of life according to the widely used EORTC-QLQ-C30 was 75. This is similar to reference values pooled across six European studies used as general population references (Hinz et al. Acta Oncologica 2014;53(7): 958-965). For diabetics, ADDQoL scores were also similar to cohorts of patients with diabetes. Quality of life and health scores were not significantly affected by demographics, peri-operative complications or other social variables. This relatively large, long-term outlook of patients undergoing pancreatic surgery demonstrates quality of life and health to be similar to that reported in the general population. This is reassuring, given the changes in exocrine, endocrine and digestive function attributed to pancreatic surgery.
40. COMPREHENSIVE GENOMIC PROFILING OF PANCREATIC CANCER TUMOR SPECIMENS: IS MORE BETTER?

AN Krepline, M Aldakkak, KK Christians, B George, PS Ritch, WA Hall, BA Erickson, DB Evans, S Tsai

Presenter: Lindsay Bliss MD, MPH | Medical College of Wisconsin

Background: Comprehensive Genomic Profiling (CGP) is increasingly used to identify somatic alternations that have prognostic and/or predictive relevance. The predictive value of CGP in pancreatic cancer is unknown.

Methods: Surgical specimens from patients treated with neoadjuvant therapy for pancreatic cancer sent to Caris Life Science for CGP were identified from 2016-2017. Genetic variants identified by Next Generation Sequencing (NGS) were classified as benign (B), likely benign (LB), variant of unknown significance (VUS), likely pathogenic (LP), or pathogenic (P). Somatic variants with a potential therapeutic target were identified from the company’s molecular profiling report.

Results: The commercial testing consisted of either a 472-gene or 46-gene NGS panel. Of the 472 genes only 9 (1.9%) genes had actionable targeted agents: ATM, BRAF, BRCA1, BRCA2, c-KIT, Her2/Neu, PDGFRA, PIK3CA, and RET. Genomic testing using the 472-gene panel was completed in 49 patients. Of the 472 genes tested, 80 (16.0%) genes had at least one variant identified: 3 (0.6%) B, 20 (4.2%) LB, 65 (13.8%) VUS, 3 (0.6%) LP, 13 (2.8%) P. LP or P variants were identified in 14 (3.0%) genes (Table 1). Available targeted therapy was identified for 4 (0.8%) genes involving the homologous recombination DNA repair pathway (BRCA1, BRCA2, ATM) and 1 (0.2%) gene involving the mTOR (PIK3CA) pathway. Of the patients identified with an actionable variant, no patient had more than one actionable variant. Based on NGS results, the most commonly recommended therapy was a platinum agent (n=12, 24.4%). Off-label treatments recommended by the NGS profiling included everolimus in 1 (2.0%) patient, mitomycin-C in 3 (6.1%), olaparib in 3 (6.1%), and temsirolimus in 1 (2.0%). One patient had both the 472-gene and 46-gene panel was completed from the same surgical specimen. Discordant results were identified in 3 genes: KRAS and TP53 P variants were identified on the 46-gene panel but not on the 472-gene panel and a JAK2 PB variant was identified on the 472-gene panel but not the 46-gene panel.

Conclusion: Using a 472-gene panel, only 3.0% of genes had a LP or P
variant in patients with localized pancreatic cancer. Among the 49 patients, actionable variants were identified in 13 (26.5%). Platinum agents were the most common targeted agent identified. Further studies are needed to evaluate the cost-effectiveness of using NGS for pancreatic cancer, as the molecular profiling reports are unlikely to change therapy.
41. THE IMPACT OF PATHOLOGIC COMPLETE RESPONSE ON SURVIVAL IN PANCREATIC DUCTAL ADENOCARCINOMA

NM Sell, GC Lee, CR Ferrone, CF del Castillo, AL Warshaw, LS Blaszkowsky, KD Lillemoe, M Qadan

Presenter: Naomi Sell MD, MHS | Massachusetts General Hospital

The impact of pathologic complete response on survival in pancreatic ductal adenocarcinoma

Background: Multimodal treatment, including total neoadjuvant therapy, has resulted in prolonged survival in the treatment of pancreatic ductal adenocarcinoma (PDAC). Small studies have shown improved outcomes among those who have a pathologic complete response (pCR) after neoadjuvant therapy followed by surgical resection. We sought to evaluate the impact of pCR on overall survival (OS) of these patients through use of a large national database.

Methods: The National Cancer Database (NCDB) was utilized to retrospectively study patients diagnosed with PDAC from 2004-2014. A pCR was defined as no tumor identified in the pancreas or associated lymph nodes by final pathology following surgical resection. A near complete response (nCR) was defined pathologically as a primary tumor less than 1cm without lymph node metastases. The primary outcome measured was OS.

Results: A total of 5,364 patients with PDAC underwent neoadjuvant chemotherapy and/or radiation followed by pancreatectomy. Forty-one patients had a pCR (0.8%), 54 (1%) had a nCR, and the remaining 5266 (98.2%) had an incomplete response (iCR; Table 1). Patients with a pCR had a median OS of 43 months compared with 24 months for nCR and 23 months for iCR (p<0.0001). A pCR was the only variable associated with an improved OS on adjusted Cox regression. While there were no significant differences in the median time from diagnosis to either chemotherapy or radiation among the groups, the pCR group had a significantly longer interval from diagnosis to surgery (195 days pCR vs. 157 days nCR vs. 139 days iCR; p=0.0001).

Conclusion: For patients who are diagnosed with PDAC and undergo neoadjuvant treatment followed by surgical resection, achieving a pCR is associated with improved OS when compared to those with residual tumor within the specimen. Interestingly, an association between nCR and improved survival was not observed.

<table>
<thead>
<tr>
<th>Initial treatment strategy</th>
<th>n (%)</th>
<th>Success as definitive therapy, n (%)</th>
<th>Median time to definitive treatment (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Debridement and external drainage</td>
<td>91 (33%)</td>
<td>25 (27%)</td>
<td>237 days (10-3482)</td>
</tr>
<tr>
<td>Cystogastrostomy Endoscopic Surgical</td>
<td>78 (28%)</td>
<td>53 (68%)</td>
<td>163 days (13-2439)</td>
</tr>
<tr>
<td>Percutaneous drain</td>
<td>51 (19%)</td>
<td>17 (33%)</td>
<td>192 (22-1352)</td>
</tr>
<tr>
<td>Distal pancreatectomy (+/- splenectomy)</td>
<td>26 (9%)</td>
<td>23 (88%)</td>
<td>112 days (30-2482)</td>
</tr>
<tr>
<td>Internal drainage (pancreatico-/cysto-jejunostomy)</td>
<td>20 (7%)</td>
<td>15 (75%)</td>
<td>144 days (23-2481)</td>
</tr>
<tr>
<td>None</td>
<td>8 (3%)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Died before treatment</td>
<td>15</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

p-value 0.27

(TCR) was defined pathologically as a primary tumor less than 1cm without lymph node metastases. The primary outcome measured was OS.
42. ENDOSCOPIC ULTRASOUND-GUIDED CELIAC GANGLION RADIOFREQUENCY ABLATION VERSUS CELIAC PLEXUS NEUROLYSIS FOR PALLIATION OF PAIN IN PANCREATIC CANCER: A RANDOMIZED CONTROLLED TRIAL

JY Bang, B Sutton, RH Hawes, S Varadarajulu

Presenter: Ji Young Bang MD, MPH | Florida Hospital Orlando

Background: Although frequently performed, the efficacy of endoscopic ultrasound-guided celiac plexus neurolysis (EUS-CPN) for palliation of pain in pancreatic cancer is suboptimal. Recently, endoscopic ultrasound-guided radiofrequency ablation (EUS-RFA) has been proposed as a palliative treatment option for pancreatic neoplasms. We performed a single-blind, randomized trial to compare the effectiveness of EUS-CPN and EUS-RFA for palliation of pain in pancreatic cancer.

Methods: Patients with abdominal pain due to locally advanced or metastatic pancreatic cancer underwent EUS-CPN (n=14) or EUS-RFA (n=12). EUS-RFA was performed using a 1Fr monopolar probe passed via a 19G FNA needle, by targeting the area of celiac plexus or visualized ganglia. Primary outcome was pain severity as measured by the European Organization for Research and Treatment of Cancer (EORTC) pancreatic cancer-specific (QLQ-PAN26) questionnaire administered pre-treatment and at 2 and 4-weeks post-treatment. Secondary outcome measures were comparison of quality of life as determined by PAN26 and C30 (core cancer) questionnaires and opioid analgesia use between the two groups.

Results: Both the pancreatic cancer-specific (49.0 vs. 57.0, p<0.001) and core cancer (51.9 vs. 64.4, p=0.032) questionnaires revealed less pain for EUS-RFA over EUS-CPN. Also, EUS-RFA cohort experienced significantly less severe gastrointestinal symptoms, were able to plan more for the future (50.1 vs. 68.5, p=0.003), and had better emotional functioning (75.8 vs. 54.3, p<0.001) compared to the EUS-CPN group.

Conclusion: Compared to EUS-CPN, EUS-RFA provided more pain relief and improved the quality of life for patients with pancreatic cancer.
ORAL ABSTRACTS (continued)
43. SIMULATED VOLUME-BASED REGIONALIZATION OF PANCREATECTOMY PROCEDURES: IMPACT ON SPATIAL ACCESS TO CARE

ZV Fong, G Jin, DA Hashimoto, AB Haynes, N Perez, C Fernandez-del Castillo, M Qadan, CR Ferrone, AL Warshaw, KD Lillemoe, LN Traeger, DC Chang

**Presenter**: Zhi Ven Fong MD, MPH | Massachusetts General Hospital

**Background**: Policies to regionalize complex procedures to high-volume centers are being considered. While this may improve outcomes, the impact on patient access is unknown. This study simulates the regionalization of pancreatectomies to assess impact on spatial access to care in terms of patient and family driving times.

**Methods**: Patients undergoing pancreatectomies from 2005 to 2014 were identified from California’s statewide administrative database. Round-trip driving times between patients’ home ZIP code and hospital addresses were calculated via Google Maps. Simulated regionalization was performed by eliminating hospitals performing <20 pancreatectomies/year, and reassigning patients to the next closest hospital that satisfied the volume threshold. Subset analyses were performed for New York and Medicare patients to assess for influence of geography and insurance coverage, respectively.

**Results**: Of 13,317 pancreatectomies performed, 6335 (47.6%) were done by hospitals with <20 cases/year. Patients traveled a median of 49.8 minutes (IQR 30.8-96.2) at baseline. An access-restriction policy would increase median round-trip driving time by 24.1 minutes (IQR 4.5-53.5, Figure). Population mortality rates were estimated to decrease from 4.3% to 2.8% (p<0.001). Affected patients were more likely to be racial minorities (44.6% vs 36.5% of unaffected patients, p<0.001) and uninsured (16.7% vs 10.2% of unaffected patients, p<0.001). Sensitivity analyses revealed a 17.8 minutes increment for patients in NY (IQR 0.8-47.4), and 27.0 minutes increment for Medicare patients (IQR 6.2-57.1).

**Conclusion**: A policy that limits access to low-volume pancreatectomy hospitals will increase round-trip driving time by 24 minutes for patients and their caregivers; while population mortality rates may improve by 1.5%. Racial minorities and the uninsured would be most affected.
Table 1. Morbidity - 264 total events in 111 patients (53%)

<table>
<thead>
<tr>
<th>Infection</th>
<th>n (%)</th>
<th>POPF n (%)</th>
<th>Pancreatic Insufficiency n (%)</th>
<th>Other n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intra-abdominal</td>
<td>28* (13%)</td>
<td>Grade A 10 (5%)</td>
<td>Endocrine 18 (9%)</td>
<td>Venous Thromboembolism 12 (6%)</td>
</tr>
<tr>
<td>Surgical Site</td>
<td>9 (4%)</td>
<td>Grade B 31 (15%)</td>
<td>Exocrine 4 (2%)</td>
<td>Ileus 11 (5%)</td>
</tr>
<tr>
<td><em>C. difficile</em></td>
<td>8 (4%)</td>
<td>Grade C 1** (0.5%)</td>
<td>Failure to Thrive 10 (5%)</td>
<td>Entero-cutaneous Fistula 8 (4%)</td>
</tr>
<tr>
<td>CLBSI</td>
<td>6 (3%)</td>
<td></td>
<td></td>
<td>Bleeding 6 (3%)</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>6 (3%)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*26/28 required percutaneous drainage
**required reoperation
Table. Summary of outcome measures in meta-analysis comparing endoscopy and minimally invasive surgery for infected necrotizing pancreatitis

<table>
<thead>
<tr>
<th>Outcome measure</th>
<th>Number of patients (n)</th>
<th>Pooled Estimate: mean % (95% CI)</th>
<th>Pooled risk ratio (95% CI)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Endoscopy</td>
<td>Surgery</td>
<td>Endoscopy</td>
<td>Surgery</td>
</tr>
<tr>
<td>Death</td>
<td>95</td>
<td>89</td>
<td>14.1 (9.3 - 22.1)</td>
<td>16.1 (9.0 - 27.8)</td>
</tr>
<tr>
<td>New onset multiple organ failure</td>
<td>95</td>
<td>89</td>
<td>5.2 (1.7 - 10.4)</td>
<td>19.7 (5.1 - 68.7)</td>
</tr>
<tr>
<td>Enterocutaneous fistula/perforation</td>
<td>95</td>
<td>89</td>
<td>3.6 (0.2 - 11.2)</td>
<td>17.9 (10.8 - 26.3)</td>
</tr>
<tr>
<td>Pancreatic fistula</td>
<td>86</td>
<td>83</td>
<td>4.2 (1.4 - 11.8)</td>
<td>38.2 (19.9 - 54.5)</td>
</tr>
<tr>
<td>Intraabdominal bleeding</td>
<td>95</td>
<td>89</td>
<td>6.2 (2.3 - 17.0)</td>
<td>12.3 (4.5 - 25.8)</td>
</tr>
<tr>
<td>New onset diabetes mellitus</td>
<td>85</td>
<td>79</td>
<td>22.1 (14.1 - 33.9)</td>
<td>27.3 (18.0 - 37.9)</td>
</tr>
<tr>
<td>Pancreatic exocrine insufficiency</td>
<td>85</td>
<td>79</td>
<td>48.6 (7.5 - 85.9)</td>
<td>63.3 (10.9 - 90.1)</td>
</tr>
<tr>
<td>Length of hospital stay (days)</td>
<td>85</td>
<td>79</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Background: Postoperative pancreatic fistula (POPF) is the Achilles heel of pancreatic surgery. Pancreatic texture, as assessed by the surgeon, has been identified as the strongest predictor of POPF in many studies. However, texture is a subjective parameter with no proven reliability or internal or external validity. Therefore a more objective parameter is needed for exact risk stratification in pancreatic surgery. The aim was to evaluate fibrosis at the pancreatic cut margin as an alternative parameter.

Methods: The RECOPANC trial was conducted as a monitored multicenter prospective trial. Pancreatic fibrosis was assessed retrospectively from H&E stained tissue slides of the pancreatic cut margin collected centrally during conduct of the RECOPANC trial. Fibrosis was graded from 0 (no fibrosis) to III (severe fibrosis). Predictive value of fibrosis grade and pancreatic texture with regard to POPF of grade B/C was assessed by univariable and multivariable statistical modeling in R software.

Results: Fibrosis grading showed strong interrater reliability (kappa=0.74) and correlated positively with hard pancreatic texture (p<0.05). In univariable analysis, area under the curve (AUC) for the prediction of POPF B/C was higher for fibrosis grade than for pancreatic texture (0.71 vs 0.59). In multivariable analysis, the following predictors were selected by elastic net regression: sex, surgeon volume, main pancreatic duct diameter and fibrosis. The final multivariable model reached an AUC of 0.78 with PPV and NPV of 0.38 and 0.92.

Conclusion: Pancreatic fibrosis grade at pancreatic cut margin can substitute assessment of pancreatic texture and is a more objective and reliable parameter. Future studies might use fibrosis grade for risk stratification in pancreatic surgery.
45. PREDICTORS OF SAME-ADMISSION CHOLECYSTECTOMY IN MILD, ACUTE, BILIARY PANCREATITIS

A Shmelev, SC Cunningham

Presenter: Steven Cunningham MD | Saint Agnes Hospital

**Background:** Acute pancreatitis (AP) carries a 20-30% early readmission rate, and delay in cholecystectomy has been recognized as an important risk factor for readmissions. Therefore, current guidelines recommend same-admission CCY (SA CCY) for mild biliary pancreatitis. We aimed to determine factors affecting the decision to perform or not perform SA CCY before discharge.

**Methods:**
All nonpediatric, nontrauma, primary admissions for mild, acute, biliary pancreatitis were pooled from NIS 2000-2012 (n=511,467). We excluded cases with ICD codes for chronic pancreatitis, pancreatic masses, alcohol-related disorders and hypertriglyceridemia, acute cholecystitis, as well as codes indicative of AP-related organ dysfunction or complications. A logistic regression model was built based on selected demographic and hospital-level predictors, as well as ICD codes associated with performing SA CCY. Additionally, seasonal and trend analysis of SA CCY rates was conducted.

**Results:**
The rate of SA CCY in the U.S. varied from 20% (Vermont) to 55% (Alaska), with a nationwide mean of 40.5%. Major predictors of SA CCY were the presence of codes for chronic cholecystitis (odds ratio [OR], 26.9; 95% confidence interval [CI], 26.4–27.4), and the presence of ventral or umbilical hernias (OR, 5.69; CI 5.0–6.4). Increased age (OR, 0.986; CI 0.985–0.986) and Charlson comorbidity score (OR, 0.921; CI 0.916–0.927), as well as male gender (OR, 0.87; CI 0.85–0.88), presence of dementia (OR, 0.72; CI 0.68–0.76), chronic comorbidities (OR, 0.55; CI 0.52–0.58) or ostomies (OR, 0.50; CI 0.44–0.57) decreased likelihood of SA CCY. Compared to African-Americans, patients who were Caucasian, Asian, and Hispanic were all significantly more likely to have SA CCY (ORs, 1.22, 1.20, 1.29; CIs 1.16–1.26, 1.15–1.26, and 1.25–1.34), respectively. Medicare beneficiaries, compared to self-pay patients, had the least likelihood of undergoing SA CCY. Urban teaching hospitals were 1.92 times, and urban non-teaching 1.51 times, more likely to perform SA CCY, compared to rural hospitals. This effect was independent of location (state) of the hospital: South region hospitals were 1.69 times more likely to perform SA CCY, compared to Northeast hospitals. Large hospital size also was an independent predictor of SA CCY. Nationwide rates of SA CCY increased from 36% in 2000 to 43% in 2006 and remained in the 41-42% range since then. No well-defined seasonal variation in SA CCY rates was detected.

**ORAL ABSTRACTS (continued)**
Conclusion: Current adherence to guidelines in performing SA CCY for mild acute biliary pancreatitis remains inadequate. Observed strong association of codes for chronic biliary pathology and abdominal wall hernias could represent intraoperative or pathology findings, but can also be interpreted as desire to operate for both AP prevention and to take care of chronic cholecystitis or hernias. Older patients with chronic comorbid conditions are less often selected for SA CCY. With higher volume and experience, large teaching hospitals perform more SA CCYs than small rural facilities. Interestingly, hospital region was significantly and independently associated with rates of SA CCY, a finding which needs further study.
Background: The response of the intrapancreatic ductal system to inflammatory injury is poorly understood. IL-4 and IL-13 have been shown to promote epithelial cell proliferation in the intestine and bronchi, respectively. The signaling of these two cytokines is mediated by interleukin 4 receptor alpha (IL-4Rα). In this study we investigate the role of IL-4Rα in the regeneration of the ductal epithelium of the intrapancreatic biliary ducts after injury.

Methods: An acute model of pancreatitis was induced in 6-7 week-old B6129 (WT) and IL-4Rα-/- mice by eight hourly injections of cerulein (50 mg/kg) every other day for 7 days. Pancreata were harvested 1, 4, 8, and 15 days after injury. Immunohistochemical analyses were performed employing antibodies specific to ductal cells (cytokeratin 19) and a marker of proliferation (Ki67). Proliferative index quantified as the proportion of ductal cells that stained for Ki67. A two-tailed student t-test was used to determine significance (p = 0.05).

Results: Non-injured mice exhibited a relatively low proliferative index of 16.1% in the common channel, and 19.3% in the intrapancreatic biliary duct. In response to pancreatitis, ductal epithelial proliferation in the common channel increased to 23.8% by day 1, and 33.2% (p = 0.018) by day 4 post-injury. Proliferation remained elevated until day 15. Similarly, proliferation within the intrapancreatic biliary duct increased over time, although with delayed kinetics compared to the common channel. Proliferation remained unchanged at 18.5% on day 1 post-injury but increased to 30.4% by day 4 and 35.4% (p = 0.007) by day 15. To investigate whether IL-4Rα contributes to proliferation in the intrapancreatic biliary ductal system, we employed mice deficient in this receptor (IL-4Rα-/-). Similar to WT mice, non-injured IL-4Rα-/- mice exhibited a proliferation index of 10.6% in the common channel and 11.8% in the intrapancreatic biliary duct. However, in contrast, a dramatic reduction in epithelial proliferation was observed upon injury. The proliferative index of the common channel decreased by day 1 post-injury (6.8%, p = 0.041), continued to decrease until day 4 (3.5%, p = 0.002), and maintained a significant reduction in proliferation through day 15. The proliferative index of the intrapancreatic biliary duct exhibited a stepwise decrease as well, decreasing to 4.3% by day 4 (p = 0.017) which was maintained through day
Conclusion: Increased epithelial proliferation of the intrapancreatic ductal system of WT mice reveals the regenerative response of the epithelium after injury. Significantly decreased proliferation in mice lacking the IL-4Rα suggests an important role for cytokine signaling in maintaining the protective epithelial barrier of the intrapancreatic ductal system after pancreatitis.

47. LOCAL AND SYSTEMIC EFFECTS OF AGING ON ACUTE PANCREATITIS

AMM Coelho, MCC Machado, SN Sampietre, F Pinheiro-Silva, JEM Cunha, LAC D’Albuquerque

Presenter: Marcel Machado MD | University of Sao Paulo School of Medicine

Background: Acute pancreatitis (AP) in elderly patients in spite of similar occurrence of local complications is followed by a substantial increase in morbidity and mortality rates. Aging process has been found to influence the course and outcome of AP. The mechanisms underlying this age related vulnerability remain unknown. The aim of this study was to evaluate the local and systemic effects of aging on severity of AP in an experimental rat model in elderly animals

Methods: AP was induced in Wistar rats by intraductal 2.5% taurocholate injection and divided into 2 experimental groups: Young (3 month old) and Aged (18 month old). Two and 24 hours after AP induction blood samples were collected for determinations of amylase, AST, ALT, urea, creatinine, glucose, and of plasma ileal fatty acid binding protein (I-FABP). TNF-α, IL-6 and IL-10 levels were determined in serum and ascitic fluid. Liver mitochondrial function and malondialdehyde (MDA) contents, pancreas histological analysis, and pulmonar myeloperoxidase (MPO) activity were performed. Bacterial translocation was evaluated by bacterial cultures of pancreas expressed in colony-forming units (CFU) per gram.

Results: A significant increase in serum amylase, AST, ALT, urea, creatinine, glucose, I-FABP, and IL-6 levels, and a reduction in serum and ascitic fluid TNF-α levels were observed in the aged group compared to the young group.
Serum IL-10 levels were similar in both groups. However, a marked increase in the ratio of IL-10/I-L6 levels was observed in animals of AP young group when compared with animals of AP aged group (p<0.05). Liver mitochondrial dysfunction, MDA contents, and pulmonary MPO activity were increased in the aged AP group compared to the young AP group (p<0.05). A significant increase in positive bacterial cultures and histological analysis obtained from pancreas tissue in aged group was significantly increased compared to the young group (p<0.05).

Conclusion: This study demonstrated that aging influences the course of acute pancreatitis evidenced by increased local and systemic lesions and the increased in bacterial translocation. These findings may have significant therapeutic implication in the clinical setting.

Table. Preoperative factors associated with malignancy in the cohort of asymptomatic patients (n=174, symptomatic patients and patients who were metastatic at diagnosis excluded)

<table>
<thead>
<tr>
<th>Variable, n (%)</th>
<th>Univariable analysis</th>
<th>Multivariable analysis*</th>
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<tr>
<td></td>
<td>Yes 19 (10.9)</td>
<td>No 155 (89.1)</td>
</tr>
<tr>
<td>Type of Institution</td>
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<td></td>
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<tr>
<td>Low-volume</td>
<td>15 (13.5)</td>
<td>95 (86.5)</td>
</tr>
<tr>
<td>High-volume</td>
<td>4 (6.3)</td>
<td>59 (93.7)</td>
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<td>Age ≤ 65</td>
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<td>110 (92.4)</td>
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<td>&gt; 65</td>
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</tr>
<tr>
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<td>10 (11.2)</td>
<td>79 (88.8)</td>
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<tr>
<td>Female</td>
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<td>76 (89.4)</td>
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<tr>
<td>Number of Tumors</td>
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<td></td>
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<tr>
<td>1</td>
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<td>145 (90.1)</td>
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<tr>
<td>≥2</td>
<td>23 (11.1)</td>
<td>107 (88.9)</td>
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<tr>
<td>Tumor Location</td>
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<tr>
<td>Body-Tail</td>
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<td>Diffuse</td>
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<td>5 (62.5)</td>
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<td>Tumor size</td>
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<td>60 (98.4)</td>
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<tr>
<td>&gt; 2cm</td>
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<td>95 (84.1)</td>
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<td>63 (84.0)</td>
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<td>Calcifications</td>
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<tr>
<td>Thick</td>
<td>7 (8.9)</td>
<td>72 (91.1)</td>
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<tr>
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<td>19 (90.5)</td>
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<tr>
<td>No</td>
<td>18 (10.6)</td>
<td>152 (89.4)</td>
</tr>
<tr>
<td>Yes</td>
<td>1 (25.0)</td>
<td>3 (75.0)</td>
</tr>
<tr>
<td>Initial management</td>
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<td>upfront surgery</td>
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<td>Surveillance &gt; 6 months before surgery</td>
<td>1 (4.0)</td>
<td>24 (96.0)</td>
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</tbody>
</table>

*Also adjusted for the date of diagnosis
Background: Although 80% of patients with acute pancreatitis will have a mild self-limited course, up to 20% will develop severe pancreatitis with an associated 20% mortality rate. No specific therapy exists for acute pancreatitis and current treatment remains entirely supportive. Adipose stem cells (ASCs) have significant immunomodulatory activity as well as regenerative potential. We hypothesized that systemic administration of ASCs would mitigate local and systemic inflammation in AP.

Methods: All experiments were approved by institutional IACUC. Twenty male brown mice (20-24 weeks old, mean weight 44g) had acute pancreatitis induced by 6 hourly intra peritoneal injections of cerulein. Treatment was initiated at 24 hours via tail vein IV. Four treatment groups included: sham control group (no acute pancreatitis), vehicle injection, human ASC injection, and human ASC conditioned media injection. Mice were sacrificed at 48 hours; blood, pancreas, lungs, and kidneys were analyzed. Acinar cell injury was quantified histologically by a dedicated experienced pathologist and 2 pancreatic surgeons using a published scoring system (edema, inflammation and necrosis). Pancreatic inflammation, engraftment and end-organ failure were studied by immunohistochemistry and PCR technique.

Results: Mice treated with ASCs had less severe AP, as shown by a statistically significantly decreased histopathology score (edema, inflammation and necrosis) (p=0.001). ASCs infusion polarized pancreatic macrophages toward an anti-inflammatory M2 phenotype (determined by immunohistochemistry staining for M2 marker CD206). ASC treatment increased expression of the M2-related markers (resistin like alpha protein) (p<0.01), while suppressing expression of M1-related iNOS and TNFα (p<0.05). When using IV infusion of Hoechst-labeled ASCs, ASCs were found to localize to inflamed tissues: lungs and pancreas. ASC conditioned media IV infusion reduced pancreatic inflammation similarly to ASCs only, highlighting the importance of ASCs secreted factors’ paracrine mechanism in modulating inflammation.

Conclusion: Intravenous delivery of human ASC markedly reduces pancreatic
inflammation and end organ injury in a murine model of acute pancreatitis. ASCs represent an efficient and attractive therapy for acute pancreatitis.
Background: After standard diagnostic work-up, the etiology of acute pancreatitis remains unknown in up to 25% of cases, a condition referred to as idiopathic acute pancreatitis (IAP). Determining the etiology of pancreatitis is essential, as it may direct treatment in the acute phase of the disease and guide interventions to prevent recurrent pancreatitis. We explored the use and yield of additional diagnostic tests (i.e. endosonography, MRI/MRCP, CT, diagnostic ERCP and IgG4). Furthermore, we analyzed the recurrence rate of acute pancreatitis after a first episode of IAP and assessed the impact of establishing an etiological diagnosis treatment on recurrence rates.

Methods: Between 2008 and 2015, patients presenting with acute pancreatitis were registered prospectively in fifteen Dutch hospitals. Patients who initially had a negative diagnostic work-up with regard to the etiology of their first episode of pancreatitis were labelled IAP. This initial work-up included: personal history (signs of a biliary cause, alcohol use, medication use, metabolic disorders, prior abdominal trauma, surgery, or ERCP); family history (chronic or hereditary pancreatitis); trans abdominal ultrasound; and laboratory tests (i.e. liver enzymes, calcium, triglycerides) We performed a post-hoc analysis including the type and number of all diagnostic tests performed, the yield of these test to establish an etiological diagnosis, and recurrence rates after treatment.

Results: Out of the 1632 patients that were registered, 191 patients were diagnosed with a first episode of IAP. Out of these 191 patients, 176 (92%) underwent one or more additional diagnostic test: CT (n=124, yield 8%), EUS (n=62 patients, yield 35%), MRI/MRCP (n=56, yield 33%), repeat ultrasound (n=97, yield 21%), IgG4 (n=54, yield 9%), and ERCP (n=15, yield 47%). In 64 patients (36%) these tests disclosed an etiological diagnosis. Forty-one patients (22%) have had all additional diagnostic tests recommended by current guidelines. During a median follow-up of 4 years (IQR 3-6), 50 out of 191 patients (26%) had at least one recurrence, 26 of whom had more than one recurrent episode. There were 101 recurrences in total with a median of 2 per patient (IQR 1-2). Out of 141 patients with only one single episode of
idiopathic pancreatitis, 128 patients underwent additional diagnostic testing. In 35 cases (27%) an etiology was found: biliary (n=22; 1 combined with pancreas divisum), autoimmune (n=3), pancreatic carcinoma (n=3), chronic pancreatitis (n=3), ampullary carcinoma (n=2), pancreas divisum (n=1), and a neuroendocrine tumor (n=1). Of the 50 patients with recurrent episodes of acute pancreatitis, an etiological cause was identified after additional testing in 29 patients (58%): biliary etiology (n=17; 1 combined with pancreas divisum), pancreatic carcinoma (n=6; 1 combined with biliary stones and chronic pancreatitis), autoimmune pancreatitis (n=3), chronic pancreatitis (n=2) and an IPMN (n=1). In 13 out of 176 of patients (7%) additional testing showed an ampullary or pancreatic neoplasm. EUS and MRI/MRCP had a high diagnostic etiological yield, both in the single episode patients (EUS 35%; MRI/MRCP 30%) and those with recurrent episodes (EUS 35%; MRI/MRCP 35%).
ORAL ABSTRACTS (continued)

50. NATURAL HISTORY OF DISCONNECTED PANCREATIC DUCT SYNDROME: WHICH OPERATION AND WHEN?

TK Maatman, AM Roch, KA Lewellen, MA Heimberger, EP Ceppa, MG House, A Nakeeb, CM Schmidt, NJ Zyromski

Presenter: Thomas Maatman MD | Indiana University School of Medicine

Background: Disconnected pancreatic duct syndrome (DPDS) compounds management complexity in necrotizing pancreatitis (NP), an already clinically demanding disease. Remarkably few data exist to guide decision making in the management of this heterogeneous population. A variety of initial treatments exist: percutaneous drainage, pancreatic debridement with external drainage, transgastric drainage (surgical, endoscopic), internal drainage (pancreatocysto- or cysto-jejunostomy), or distal pancreatectomy. We hypothesized NP patients with DPDS will require multiple interventions and have increased disease duration and mortality relative to NP patients without DPDS. Therefore, the aim of this study is to evaluate DPDS incidence, treatment strategy, and outcomes at a high-volume referral center.

Methods: Review of 647 NP patients treated at our institution between 2005 and 2017 identified those with DPDS. Clinical factors, diagnosis method, treatment strategy, and outcomes were analyzed. Procedures included diagnostic endoscopy, endoscopic intervention, percutaneous intervention, or operative intervention. Failure of the initial treatment strategy was defined as the need for additional unique procedure or recurrence of symptoms requiring repeat intervention beyond 90 days. Where applicable, independent groups t-tests and Pearson’s correlation or Fisher’s exact tests were performed. One-way analysis of variance (ANOVA) or Kruskal-Wallis tests were performed to compare median values between groups. P-values of <0.05 were accepted as statistically significant.

Results: DPDS was diagnosed in 289/647 patients (44.7%) a median of 53 days (0-1583) following diagnosis of NP. Fifteen patients died prior to treatment for DPDS. Median age, comorbidities, and organ failure were similar between DPDS and non-DPDS patients. Patients with biliary etiology had increased risk of DPDS (OR 1.47, 95% CI 1.1-2.0, p=0.02) and post-ERCP pancreatitis patients had decreased risk of DPDS (OR 0.36, 95% CI 0.16-0.79, p=0.01). Diagnosis was most often made by contrast enhanced computer tomography (CT) imaging (69.9%); ERCP was confirmative in 28.4% of all patients. Patients with DPDS underwent a median of 3 total procedures (0-10) and 2 unique procedure types (0-4) before definitive therapy was achieved.
Treatment strategy is shown in Table 1. Median time to definitive therapy was 5.8 months (1-114); there was no difference in time to definitive therapy between treatment strategies. One hundred and forty-one patients (51.5%) required a combination of therapies. There was no difference in disease duration or overall mortality rates between DPDS and non-DPDS patients (duration: 5.8 vs 4.7 months, p=0.13; mortality: 8.0% vs 9.8%, p=0.67). Median follow-up was 25 months (1-160).

**Conclusion:** Disconnected pancreatic duct syndrome is extremely common following an episode of necrotizing pancreatitis. Diagnosis is often made several weeks into the disease course and can be made with routine contrast enhanced cross-sectional imaging. Disconnected pancreatic duct syndrome does not increase the duration of disease or mortality rates. Ideal treatment strategy should be tailored to individual anatomy and may require multiple interventions and multiple unique intervention strategies. Resolution without treatment is rare.
51. INTERNATIONAL STUDY GROUP FOR PANCREAS SURGERY: STANDARDS FOR REPORTING OF SURGERY FOR CHRONIC PANCREATITIS

AK Siriwardena, J Windsor, N Zyromski, KC Conlon, M Smith, D Radenkovic, O Busch, C Bassi, Baltatzis, C Dervenis

Presenter: Ajith Siriwardena MD | Manchester Royal Infirmary

Background: There is evidence that standards of reporting of surgery for chronic pancreatitis vary widely and as a result comparison of outcomes between procedures and between centers is problematic. The International Study Group for Pancreas Surgery (ISGPS) provides the globally-accepted definitions for reporting of post-pancreatectomy leak, post-pancreatectomy haemorrhage bleeding, chyle leak and delayed gastric emptying. The present ISGPS study is the first attempt to standardize the reporting of elective, planned surgery undertaken in chronic pancreatitis and seeks to provide comprehensive yet clinically utilizable definitions.

Methods: The ISGPS sessions were held during international congresses of the International Hepato-Pancreato-Biliary Association (IHPBA) and other similar global organisations. Multidisciplinary and multi-national input was sought using a Delphi process and a questionnaire survey.

Results: The ISGPS reporting for chronic pancreatitis can be divided into four domains: 1) patient’s clinical profile (aetiology, opiate use, prior intervention) 2) gland morphology (antero-posterior head diameter and main duct size) 3) a structured operative descriptor of the procedure (lateral pancreaticojejunostomy ± head coring, head resection, total gland resection and 4) minimal clinical outcome dataset.

Conclusion: The 2019 ISGPS reporting standards for surgery for chronic pancreatitis provide the first structured framework for the description of surgery in CP. The document is comprehensive but is sufficiently practical for routine clinical use. Implementation of this document will help standardization of reporting of surgery for chronic pancreatitis.
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Please make the following change to my contact information:

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