

# 53<sup>RD</sup> 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

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

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# MEETING HIGHLIGHTS

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## DIRECTORS



William H.  
Nealon, MD



Christopher L.  
Wolfgang, MD, PhD



Nicholas J.  
Zyromski, MD

## MEETING HIGHLIGHTS

**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

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*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.

# PANCREAS CLUB AWARDS

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*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/>

# PANCREAS CLUB AWARDS

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*The Pancreas Club thanks our generous partners for their contributions which allow us to fund awards for promising research presented at the Annual Meeting.*



**Pancreas 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/>



# PANCREAS CLUB AWARDS

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*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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## Staff

<b>Marjorie Malia &amp; Jill Willhite</b>	Management Directors
<b>Tracy Brown</b>	Meeting Coordinator
<b>Laura Fitzgerald</b>	Finance
<b>Corinne Hornsey</b>	Meetings & Events
<b>Deborah East</b>	Industry Relations
<b>Mary Kawulok</b>	Registrar
<b>Shayla Concannon</b>	Communications
<b>Nora Barrett</b>	Onsite Registration

## Pancreas Club Headquarters

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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

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## 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 AMA PRA Category 1 Credits™ listed above, a maximum of **15** credits meet the requirements for Self-Assessment.



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Accredited with Commendation by the  
Accreditation Council for Continuing Medical Education

**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

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- 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)*

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- 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

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## Friday, May 17

- 7:00am - 5:30pm **Registration** | Bayview Foyer
- 7:00am - 8:00am **Breakfast in Exhibit Hall** | Regatta Pavilion
- 7:45am - 8:00am **Welcome & Introductory Remarks** | Bayview Ballroom
- 8:00am - 9:45am **Scientific Session 1** | Bayview Ballroom
- 9:45am - 10:00am **Morning Break** | Regatta Pavilion
- 10:00am - 11:00am **Scientific Session 2** | Bayview Ballroom
- 11:00am - 12:00pm **Poster Rounds with Professors** | Regatta Pavilion
- 12:00pm - 1:00pm **Lunch** | Regatta Pavilion
- 1:00pm - 3:30pm **Scientific Session 3** | Bayview Ballroom
- 3:30pm - 3:45pm **Afternoon Break** | Regatta Pavilion
- 3:45pm - 5:30pm **Scientific Session 4** | Bayview Ballroom
- 5:30pm - 8:30pm **Pancreas Club Annual Reception and Dinner** | Banyan Court & Lawn

# SCHEDULE AT A GLANCE *(continued)*

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## Saturday, May 18

- 7:00am - 5:00pm **Registration** | Bayview Foyer
- 7:00am - 8:00am **Breakfast in the Exhibit Hall** | Regatta Pavilion
- 8:00am - 9:45am **Scientific Session 5** | Bayview Ballroom
- 9:45am - 10:00am **Morning Break** | Regatta Pavilion
- 10:00am - 11:00am **Definition of Unresectable** | Bayview Ballroom
- 11:00am - 12:00pm **Poster Rounds with Professors** | Regatta Pavilion
- 12:00pm - 1:00pm **Lunch** | Regatta Pavilion
- 1:00pm - 3:00pm **Scientific Session 6** | Bayview Ballroom
- 3:00pm - 3:15pm **Afternoon Beverage** | Regatta Pavilion
- 3:15pm - 5:00pm **Scientific Session 7** | Bayview Ballroom
- 5:00pm - 6:00pm **Awards Reception** | Sunset Terrace



# 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

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 B-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-PACLITAXEL 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 PANCREATODUODENECTOMY**

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 Klomp maker 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 Baisou 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

1:15pm - 1:30pm

**17. GASTRIC CANDIDIASIS PREOPERATIVELY EXAMINED IS A CRUCIAL PREDICTOR OF POSTOPERATIVE INFECTION-RELATED COMPLICATIONS AFTER PANCREATODUODENECTOMY: 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 PANCREATODUODENECTOMY: 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 PANCREATODUODENECTOMY 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 PANCREATODUODENECTOMY 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

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

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  
RECOFAN 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

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

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

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



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*

# POSTER LIST

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## **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-Geelen, 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

*R Gupta, A Kulkarni, RY Babu, S Shenvi, RA Gupta, R Nimje,*

*G Sharma, P Vaswani, RD Sriram, H Singh, V Sharma, SS Rana,*

*M Kang*

Postgraduate Institute of Medical Education and Research

Chandigarh



## **P 4. A TUG-OF-WAR IN IPMNS MANAGEMENT: A COMPARISON BETWEEN 2017 INTERNATIONAL AND 2018 EUROPEAN GUIDELINES**

**Presenter:** Stefano Crippa MD, PhD

*S Crippa, R Valente, A Fogliati, U Arnelo, A Halimi, Z Ateeb, E Longo,*

*F Aleotti, PG Arcidiacono, M Lohr, M Falconi, M Del Chiaro*

San Raffaele Scientific Institute



*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 STUODOQLPANCREAS 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

*DK DePeralta, M Soufi, K Flick, R Simpson, C Colgate, J Sadowski, M Kilbane, EP Ceppa, MG House, NJ Zyromski, A Nakeeb, N Strange, W Wooden, K Kelley, CM Schmidt*

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

*L Maggino, 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

*T Dhir, C Schultz, A Jain, CJ Yeo, T Golan, JR Brody*  
Thomas Jefferson University



**P 12. SHOULD DIFFUSE MAIN-DUCT INTRADUCTAL PAPILLARY MUCINOUS NEOPLASMS 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



# POSTER LIST *(continued)*

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## **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

*TH Mungroop, S Klompmaker, UF Wellner, EW Steyerberg, A Coratti, M D'Hondt, M de Pastena, S Dokmak, I Khatov, O Saint-Marc, U Wittel, M Abu Hilal, D Fuks, I Poves, T Keck, U Boggi, MG Besselink*  
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

*Al 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, Al Al-Abbass, A Singhi, H Zeh, ME Hogg, AH Zureikat*  
University of Pittsburgh Medical Center



*Posters of Distinction*

 **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 PANCREATODUODENECTOMY 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

*S Maeda, A Moore, L Yohanathan, T Hata, MJ Truty, RL Smoot, SP Clearly, DM Nagorney, TE Grotz, EJ Park, MD Girgis, HA Reber, F Motoi, T Masuda, M Unno, ML Kendrick, TR Donahue*  
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 Thompson, 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 XENOGRFT (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 RECURRENCE 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 OMM 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, Al 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

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**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

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**Presenter:** Kendall McEachron MD

*KR McEachron, GJ Beilman, TL Pruett, TB Dunn, S Chinnakotla, MD Bellin*

University of Minnesota

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**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

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**Presenter:** Amer Zureikat MD

*Al Al Abbas, JD Borreback, 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

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**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

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**Presenter:** Keith Wirth MD

*K Wirth, S Kizy, A Sheka, S Ikraumuddin, D Bernlohr, M Yamamoto*  
University of Minnesota

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**Presenter:** Masato Yamamoto MD, PhD

*M Sato-Dahlman, Y Miura, P Hajeri, H Yoshida, K Jacobsen, C Yanagiba, M Yamamoto*  
University of Minnesota

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**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

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**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



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**Presenter:** Anu Aronen MD

*M Bläuer, M Laaninen, J Sand, J Laukkarinen*

Tampere University Hospital

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**Presenter:** Maxwell Trudeau BS

*MT Trudeau, BL Ecker, L Maggino, MT Mcmillan, CM Vollmer Jr*

University of Pennsylvania

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**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

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**Presenter:** Amit Khithani MD

*AS Khithani, TB Cengiz, K El-Hayek, T Augustin, RA Simon,*

*RM Walsh, GJ Morris-Stiff*

Cleveland Clinic Foundation

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**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

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**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

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**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

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**Presenter:** Emanuel Eguia MD, MS, MHA

*E Eguia, PJ Sweigert, M Nelson, F Luchette, M Afshar, MS Baker*

Loyola University Medical Center

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**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

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**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

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**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

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**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

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**Presenter:** Amer Zureikat MD

*Al 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

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**Presenter:** Josh Bleicher MD, MS  
*J Bleicher, HM Shepherd, CL Scaife*  
University of Utah

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**Presenter:** Victoria Rendell MD  
*VR Rendell, AM Awe, BM Hanlon, N Marka, PJ Pickhardt, MG Lubner, ER Winslow*  
University of Wisconsin

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**Presenter:** Yuji Kitahata MD, PhD  
*Y Kitahata, M Kawai, S Hirono, K Okada, M Miyazawa, R Kobayashi, H Yamaue*  
Wakayama Medical University

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**Presenter:** Essa M. Aleassa MD  
*M Chumakova, EA Aleassa, G Morris-Stiff*  
Cleveland Clinic Foundation

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**Presenter:** Armando Rosales MD  
*A Rosales, A Stoylen, A Wallace, H Asbun, L Tsamalaidze, JA Stauffer*  
Mayo Clinic Florida

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**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

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**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

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**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

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**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

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**Presenter:** Benjamin Powers MD

*BD Powers, A Kumar, JB Fleming MD, JR Strosberg, DA Anaya*  
Moffitt Cancer Center

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**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

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**Presenter:** Andreas Schneider MD

*AM Schneider, E Alonso, ES Tang, ST Chiu, ML Babicky, PH Newell, PD Hansen*  
Providence Portland Medical Center

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**Presenter:** Joal Beane MD

*JD Beane, JD Borreback, AH Zureikat, HA Pitt*  
University of Pittsburgh Medical Center

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**Presenter:** Rebecca Snyder MD, MPH

*RA Snyder, JA Ewing, AA Parikh*

University of South Carolina School of Medicine- Greenville

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**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

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**Presenter:** Kathryn Chen MD

*KT Chen, YC Chen, M Baik, J Byers, SW French, B Diaz*

Harbor-UCLA Medical Center

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**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

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**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

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**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

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**Presenter:** Valentina Andreasi MD

*V Andreasi, S Partelli, R Ariotti, G Guarneri, M Pagnanelli, G Balzano, S Crippa, M Falconi*  
San Raffaele Scientific Institute

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**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 Digiaco, D Pozzi, S Palchetti, C Cascone, M Cartillone, R Coppola, G Caracciolo*

University Campus Bio-Medico di Roma

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**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

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**Presenter:** Romaine Nichols MD

*RC Nichols, CG Morris, D Pham, ME Shaikh, MS Rutenberg*

University of Florida

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**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

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**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

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**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

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**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

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**Presenter:** Ning Pu MD

*N Pu, W Lou, J Yu*

Johns Hopkins University School of Medicine

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**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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**Presenter:** David Pointer MD

*DT Pointer, DJ Roife, BD Powers, G Murimwa, S Elessawy, PJ Hodul, J Pimiento, JB Fleming, MP Malafa*

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**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

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**Presenter:** Anu Aronen MD

*A Aronen, J Aittoniemi, R Huttunen, A Nikkola, J Nikkola, O Limnell, J Sand, J Laukkarinen*

Tampere University Hospital

### **P 144. QUALITY OF LIFE AND PANCREATIC EXOCRINE AND ENDOCRINE FUNCTION IN THE LONG-TERM FOLLOW-UP AFTER PANCREATODUODENECTOMY**

**Presenter:** Ismo Laitinen MD

*I Laitinen, I Rinta-Kiikka, Y Vaalavuo, A Antila, A Siiki, J Sand, J Laukkarinen*

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

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**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 Kamaledine, 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

*MP Kim, TG Hughes, LR Prakash, JE Lee, H Wang, MH Katz, CD Tzeng, JN Vauthey, TA Aloia, JH Lee, EJ Koay, WA Ross, BR Weston, A Maitra, F McAllister, JB Fleming*

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 CHEMORADIOTHERAPY 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 Pérez-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 PERIAMPULLARY 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

**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 PANCREATODUODENECTOMY. 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*  
Toyko 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

## POSTER LIST *(continued)*

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### **P 173. "I WILL TAKE GLUE WITH STITCHES": A COMPARISON OF SKIN CLOSURE TECHNIQUES IN PANCREATODUODENECTOMY**

**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 PAPANILARY 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

*F Gavazzi, G Capretti, C Ridolfi, G Nappo, S Carrara, P Spaggiari, P Riva, A Zerbi*

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

*S Reddy, JB Rose, SM Vickers, GR Williams, AA Ahmed, KR Kyanam, MJ Heslin, O Gbolahan, AJ Gunn, DE Morgan, SJ Galgano, A McDonald, R Jacob, RK Paluri*

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

*HK Hwang, K Wada, MD, HY Kim, Y Nagakawa, Y Hijikata, Y Kawasaki, Y Nakamura, LS Lee, DS Yoon, WJ Lee, CM Kang*

Yonsei University

**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 PANCREATODUODENECTOMY 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 PANCREATICOUDENECTOMY (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

*K Suzumura, E Hatano, K Iida, H Iwama, Y Kawabata, T Okada, N Uyama, I Nakamura, M Tada, S Hai, H Sueoka, K Toriguchi, A Kurimoto, S Tamagawa, J Fujimoto*

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 PANCREATICOJEJUNOSTOMY FOLLOWING PANCREATICOUDENECTOMY DOES NOT REDUCE CLINICALLY SIGNIFICANT FISTULA RATES**

**Presenter:** Catherine Wang

*CY Wang, J Lyons, JM Hardacre*

Case Western Reserve University School of Medicine

## **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. PANCREATODUODENECTOMY 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, R Dorer, M Bonds, G Baison, F Rocha*

Virginia Mason Medical Center

## **P 201. PANCREATODUODENECTOMY IN PATIENT WITH JEJUNOILEAL BYPASS**

**Presenter:** Flavio Rocha MD

*N Handy, L Harmon, FG Rocha*

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

*SS Murthy, S Reddy, M Hill, E O'Halloran, J Hoffman, J Farma*

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

*NHV Coelho, CRF Rodrigues, AB Osvaldt*

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

*H Hisai, T Sakurai, Y Koshiba, K Watanabe, S Ameda, M Sato,*

*R Kawasaki, H Gyobu, N Yoshida*

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<sup>-/-</sup>, and TLR 4<sup>-/-</sup> 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<sup>-/-</sup> mice. This tumor inhibitory effect of gut microbial depletion disappeared in the TLR2<sup>-/-</sup> 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.

# ORAL ABSTRACTS *(continued)*

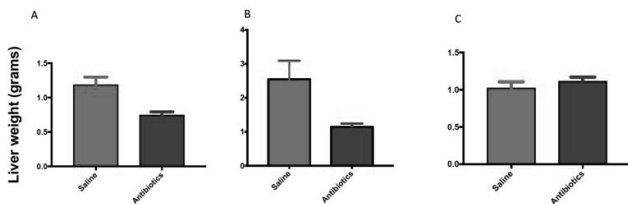
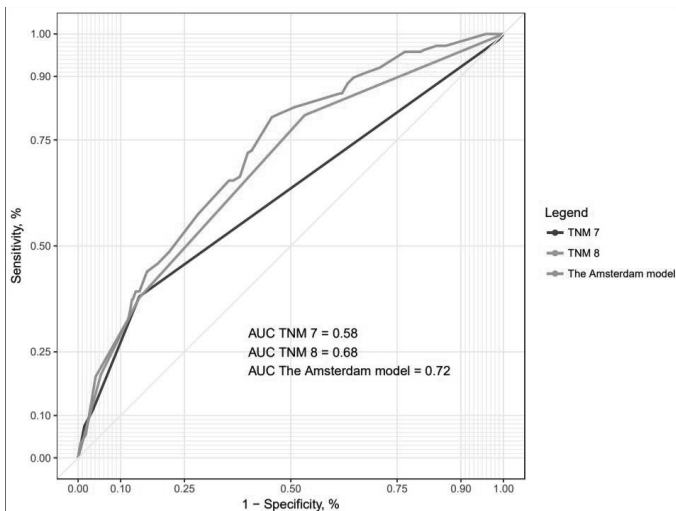


Figure 1: Depletion of the gut microbiome decreases tumor burden. Mice were randomized to receive oral saline or antibiotics. After 2 weeks of antibiotics or saline treatment, splenic injections were performed with KPC cells. Treatment with antibiotics or saline continued until sacrifice. Mice were sacrificed 21 days after splenic injection. Livers were harvested and weighed as shown. A) C57BL6 mice, p value 0.0013. B) TLR4<sup>-/-</sup> mice, p value 0.03. C) TLR2<sup>-/-</sup> mice, no significance.

## ORAL ABSTRACTS *(continued)*

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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](http://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 pancretoduodenectomy for pancreatic cancer. Model performance was

## ORAL ABSTRACTS *(continued)*

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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*

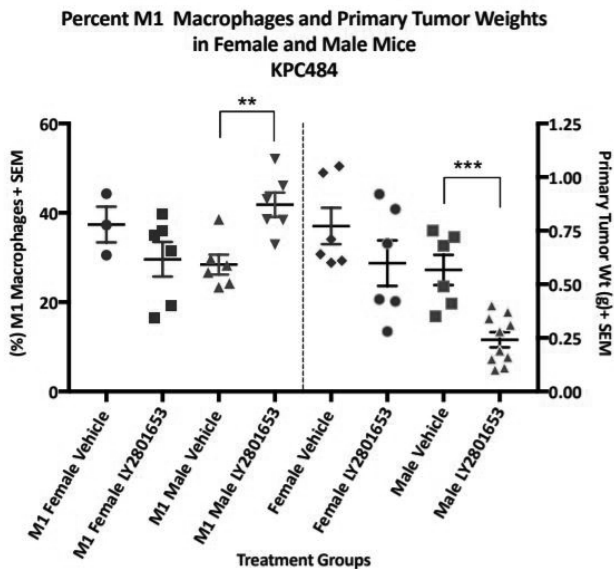
**Presenter:** Ning Pu MD | Johns Hopkins University School of Medicine

**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 *in 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.



## 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.

## ORAL ABSTRACTS *(continued)*

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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.

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

AA Javed, VPGroot, G Gemenetzis, A Hasanain, D Ding, AV Oosten, M Debeljak, SL Riel, JA Teinor, J Yu, L Zheng, EK Fishman, RH Hruban, M Goggins, JL Cameron, RA Burkhart, J He, MJ Weiss, JR Eshleman, CL Wolfgang

**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,

## ORAL ABSTRACTS *(continued)*

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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 anti-CEA-antibodies was 6.3 (imaged at 48 hours), PEG-ylated anti-CEA-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-INTEGRIN 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/ $\beta$ -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 ( $3.2 \pm 1.46$ ; p 4-fold), Enrichr analysis using the NCI pathway interaction database revealed significant enrichment in  $\beta$ -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.  $\beta$ -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.

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

*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.

## ORAL ABSTRACTS *(continued)*

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**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*

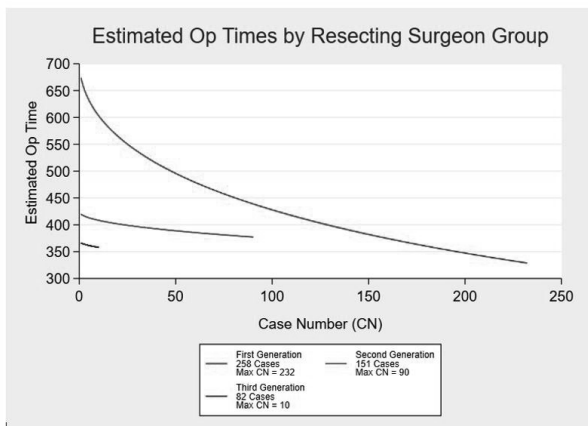
**Presenter:** Flavio Rocha MD | Virginia Mason Medical Center

**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.

# ORAL ABSTRACTS *(continued)*



Time (in minutes)	Generation 1	Generation 2	Generation 3
<b>Mean</b>	450.8	388.5	348.6
<b>SD</b>	121.0	76.7	52.6
<b>25<sup>th</sup> Percentile</b>	366	336	307
<b>Median</b>	425.5	378	342
<b>75<sup>th</sup> Percentile</b>	539	427	383

## **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

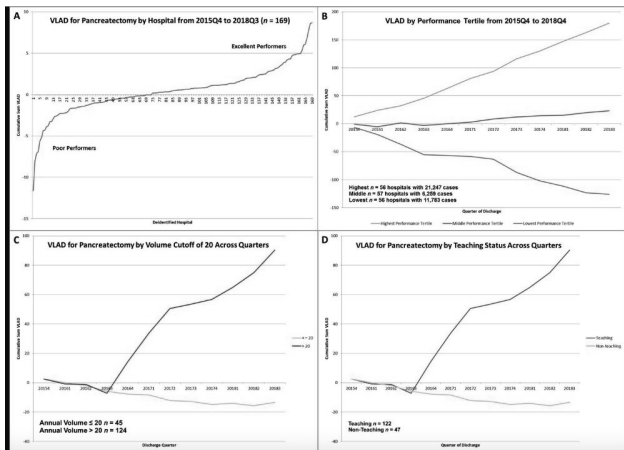
**Background:** Although distal pancreatectomy (DP) using reinforced stapler is expected to reduce PF, no multicenter RCT has been performed. The aim of this study was to investigate in the multicenter randomized controlled trial (RCT) whether reinforced stapler can reduce the incidence of clinically 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.

# ORAL ABSTRACTS *(continued)*



## **11. MENTORSHIP AND FORMAL ROBOTIC PROFICIENCY SKILLS CURRICULUM IMPROVE SUBSEQUENT GENERATIONS' LEARNING CURVE FOR THE ROBOTIC PANCREATODUODENECTOMY**

*ME Hogg, MJ Rice, J Hodges, J Bellon, J Borrebach, A Hamad, A Al-abbas, LM Knab, AH Zureikat, HJ Zeh III*

**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 pancreatoduodenectomy 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 pancreatoduodenectomies 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 $\geq$ 3).

**Results:** 514 cases were evaluated: average ORT=413.1 $\pm$ 106.4min, CR-POPF=7.6%, and Clavien $\geq$ 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 $\geq$ 3. Patient factors predictive of CR-POPF were increased ORT, soft gland, no neoadjuvant, and smaller duct. Patient factors predictive of increased Clavien $\geq$ 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 pancreatoduodenectomy. Complications were largely dependent on patient factors and not affected by introduction

# ORAL ABSTRACTS *(continued)*

of next generation surgeons. Health care delivery systems should seek to explicitly incorporate these strategies to maximize highest value and quality patient outcomes.

**TABLE 1. Univariate and Multivariate analysis of all cholangitis and/or stricture**

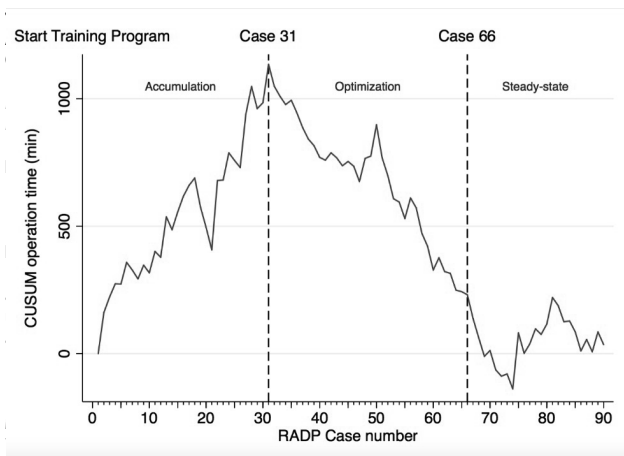
Variable	Univariate		Multivariate	
	Odds Ratio (95% CI)	p-value	Odds Ratio (95% CI)	p-value
Age > 65 years	0.623 (0.286, 1.361)	0.235	0.407 (0.165, 1.000)	0.050*
Sex (female)	0.587 (0.255, 1.349)	0.210		
BMI $\geq$ 30 kg/m <sup>2</sup>	1.585 (0.706, 3.555)	0.264	2.493 (0.999, 6.221)	0.050*
Prior abdominal surgery	0.624 (0.284, 1.371)	0.240		
Pre-op albumin	2.073 (0.918, 4.682)	0.080		
Preoperative radiation	5.160 (0.825, 32.284)	0.079	9.341 (1.283, 68.000)	0.027*
Tumor size $\geq$ 3.4 cm	2.348 (1.050, 5.252)	0.038*	2.937 (1.182, 7.296)	0.020*
Classic vs PPPD	0.529 (0.107, 2.624)	0.436		
Vascular resection	1.994 (0.816, 4.872)	0.130		
Total operative time	1.005 (1.000, 1.010)	0.052		
Time to complete HJ	1.014 (0.976, 1.053)	0.473		
Preoperative biliary stent	0.526 (0.234, 1.185)	0.121		
Cystic duct incorporated into HJ	1.490 (0.523, 4.247)	0.455		
Duct diameter, mm	0.797 (0.679, 0.937)	0.006*		
Thickness of bile duct, mm	1.080 (0.495, 2.359)	0.846		
Intra-operative biliary stent, yes	2.035 (0.926, 4.476)	0.077		
Length of HJ from hilar plate				
<10mm	<i>Ref</i>	-	-	-
10 – 20 mm	2.367 (0.776, 7.222)	0.130	3.569 (1.062, 11.996)	0.040*
> 20 mm	1.571 (0.369, 6.694)	0.541	1.453 (0.303, 6.982)	0.641
Mobilization of bile duct stump	1.032 (0.464, 2.296)	0.938		
HJ technique				
Running	<i>Ref</i>			
Interrupted	0.955 (0.365, 2.494)	0.924		
Hybrid	1.464 (0.163, 13.114)	0.733		
Number of posterior row stitches	0.732 (0.561, 0.956)	0.022*	0.720 (0.546, 0.949)	0.020*
Space between stitches, mm	0.443 (0.185, 1.059)	0.067		



# ORAL ABSTRACTS *(continued)*

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## ORAL ABSTRACTS *(continued)*



United States between FY 2016-2018. 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.

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# ORAL ABSTRACTS *(continued)*

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Median Overall and Progression Free Survival in 282 Patients

Parameter	SOC		SOC + HAP <sup>a</sup>		p-value
	FOLFIRINOX N=95	Gemcitabine/ Nab-Paclitaxel N=45	FOLFIRINOX N=97	Gemcitabine/ Nab-Paclitaxel N=45	
Overall Survival (months)	14.4	13.9	15.3	12.7	0.36
Progression Free Survival (months)	14.1	11.2	13.7	12.3	<b>0.55</b>

<sup>a</sup>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 PANCREATODUODENECTOMY**

*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

## ORAL ABSTRACTS *(continued)*

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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.

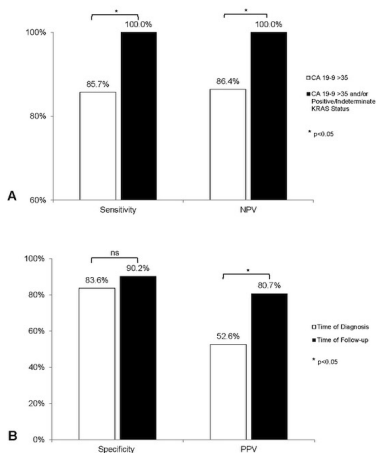
## 14. IMPLEMENTING ROBOT-ASSISTED DISTAL PANCREATECTOMY THROUGH A PROCEDURE-SPECIFIC TRAINING PROGRAM: AN ACADEMIC CENTER'S EXPERIENCE

S Klompmaker, WJ Van der Vliet, SJ Thoolen, AS Ore, K Verkoulen, M Solis-Velasco, E Canacari, JB Kruskal, KO Khwaja, JF Tseng, MP Callery, TS Kent, AJ Moser

**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



**Figure 1:** Sensitivity and negative predictive value (NPV) increased to 100% after including a positive/ indeterminate KRAS status with an elevated CA 9-9 for a marker of metastatic disease (A). Specificity and positive predictive value (PPV) of a positive/indeterminate KRAS as a single diagnostic test for metastatic disease increased to over 90% and 80% respectively after assessing disease status after a median follow-up of 3.5 months (B).

**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

# ORAL ABSTRACTS *(continued)*

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after 65 cases.

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# ORAL ABSTRACTS *(continued)*

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## **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.

**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.

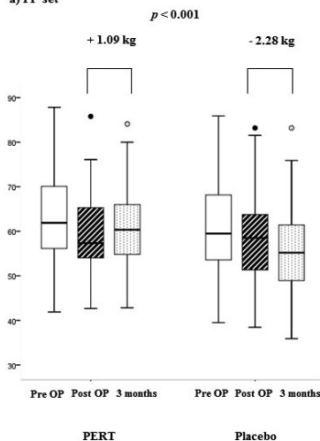
**Results:** Between May 2013 and December 2015, 282 patients were treated from 32 participating sites. Median overall survival was 14.5 months in the SOC group (N=140) compared with 14.3 months in the HAPa group (N=142) (stratified adjusted hazard ratio (HR) 0.99; 95% CI 0.63-1.55). Median progression-free 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

# ORAL ABSTRACTS *(continued)*

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Figure 2

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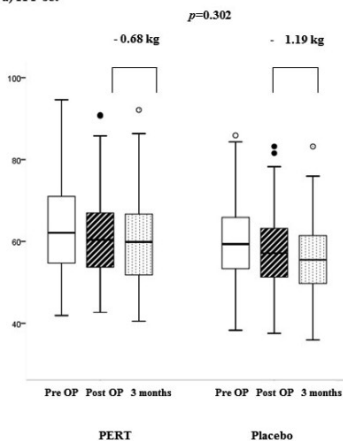


**Conclusion:** Ir  
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## **17. GASTRIC CANDIDIASIS PREOPERATIVELY EXAMINED IS A CRUCIAL PREDICTOR OF POSTOPERATIVE INFECTION-RELATED COMPLICATIONS AFTER PANCREATODUODENECTOMY: THE RESULTS OF PROSPECTIVE STUDY**

*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

# ORAL ABSTRACTS *(continued)*

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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.

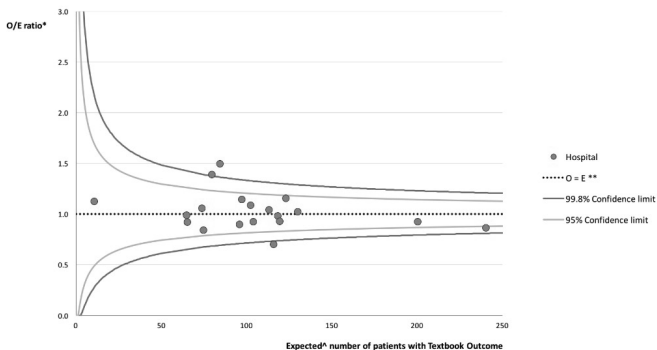
## **18. MOLECULAR STAGING IN PANCREATIC ADENOCARCINOMA UTILIZING KRAS MUTANT CELL-FREE DNA ANALYSES IN PERIPHERAL BLOOD AND PERITONEAL FLUID: ARE WE THERE YET?**

*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



**Figure 1.** Funnel-plot of between-hospital variation in Textbook Outcome after pancreatoduodenectomy during 2014-2017.

\*O/E ratio: observed number of Textbook Outcome patients divided by expected<sup>a</sup> number of Textbook Outcome patients.

<sup>a</sup>O = E: the observed  $\mu$

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 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.

## ORAL ABSTRACTS *(continued)*

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## **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 MIDP implementation rates.

## ORAL ABSTRACTS *(continued)*

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## **20. RISK FACTORS FOR MULTI-DRUG RESISTANT BACTERIA INFECTION AMONG RECTAL CARRIERS SUBMITTED TO PANCREATICOUDENECTOMY: 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.

# ORAL ABSTRACTS *(continued)*

Table 1. Cost of pancreaticoduodenectomy in patients undergoing laparoscopic vs. open approach

	Unadjusted			Adjusted*		
	Cost	95% CI		Cost	95% CI	
<b>Hospital volume, terciles</b>						
1-16	\$8,043	-\$3,271	\$19,357	\$9,390	\$2,948	\$15,831
17-127	\$4,717	-\$2,314	\$11,748	\$5,579	\$1,783	\$9,376
>127	-\$1,802	-\$8,819	\$5,216	\$616	-\$1,703	\$2,936

\*Adjusted for age, male gender, comorbidities, insurance, malignancy, and length of stay

## 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

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 PANCREATODUODENECTOMY**

*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*

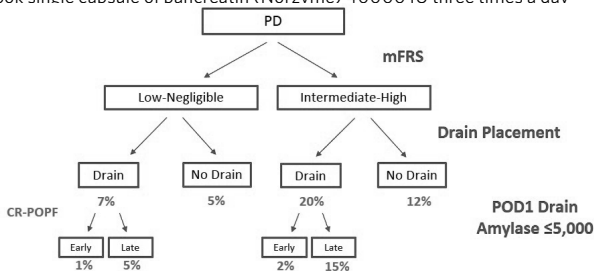
**Presenter:** Hongbeom Kim MD | Seoul National University Hospital

**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

# ORAL ABSTRACTS *(continued)*

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



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)*

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## **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  $\geq 60$  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

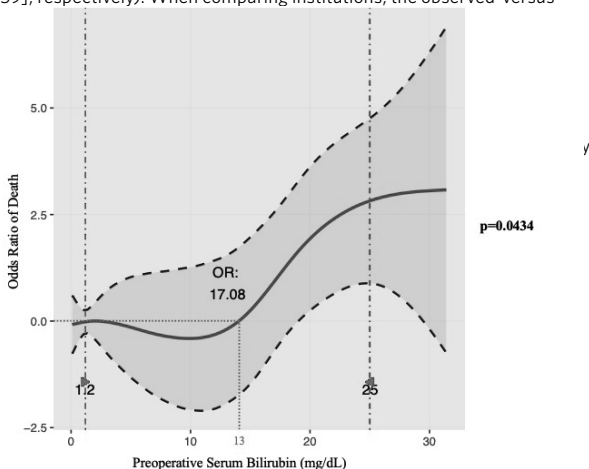
**Background:** Quality assurance programs are becoming increasingly popular in surgery but require objective assessment of surgical outcome. Textbook Outcome (TO) is a multidimensional measure, reflecting the 'ideal' surgical outcome. TO has shown to be a valuable indicator in other surgical fields,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

**Methods:** Patients who underwent pancreatoduodenectomy (PD) or distal pancreatectomy (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

## ORAL ABSTRACTS *(continued)*

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, low 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-



**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, R Fogg, EP Kennedy, H Lavu, JM Winter, S Cannaday, 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

## ORAL ABSTRACTS *(continued)*

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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 methachronous 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.

# ORAL ABSTRACTS *(continued)*

**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 PANCREATOCODUODENECTOMY 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

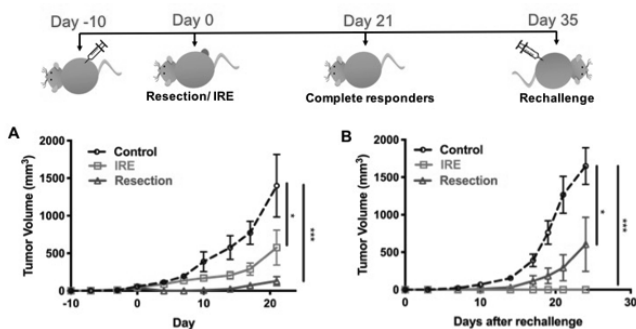


Figure: Growth curve of SQ KPC tumor at the primary site (A) and the rechallenge tumor growth on the contralateral site (B)

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,

## ORAL ABSTRACTS *(continued)*

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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 1.90; 95% CI [1.50, 2.42]), 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.

## 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 pancreatoduodenectomy (PD) have proposed omission of perioperative drains for negligible/low-risk patients and early drain removal ( $\leq$ POD3) for moderate/high-risk patients with POD1 drain amylase levels  $\leq$ 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 ( $\leq$ POD3) versus late ( $\geq$ POD4) drain removal was assessed among patients with POD1 drain amylase levels  $\leq$ 5000 U/L.

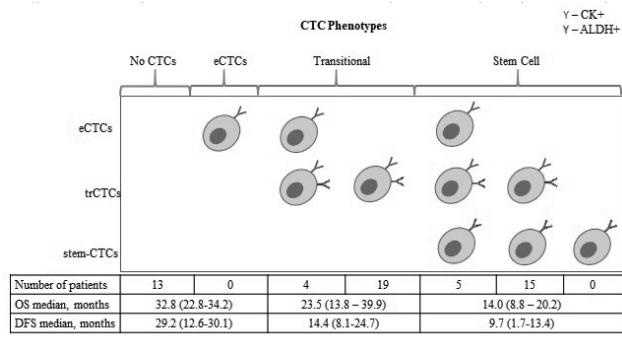
**Results:** Among 6730 patients undergoing PD, 3375 (50%) were high-risk and 3355 (50%) were low-risk (Figure). Among high-risk patients, drain placement ( $n=3093$ , 92%) was associated with a higher rate of POPF (26%vs16%,  $p=0.0003$ ), clinically relevant (CR)-POPF (20%vs12%,  $p=0.0015$ ), and 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  $\leq$ 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  $\leq$ 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



# ORAL ABSTRACTS *(continued)*

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  $\leq 5000$  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  $\leq 5,000$  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

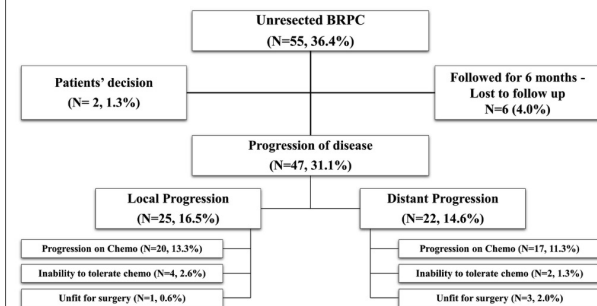
**Presenter:** Yoonhyeong Byun MD | Seoul National University Hospital

**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

## Reasons for unresectability in patients with unresected BRPC



**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**

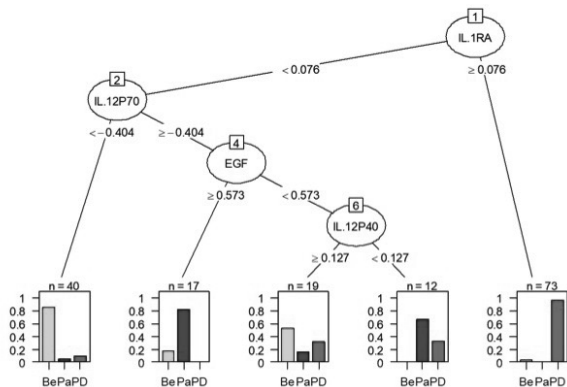
*B Chen, MT Trudeau, L Maggino, BL Ecker, LJ Keele, CM Vollmer*

**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 spline 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  $\leq 1.2$  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



**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 RECURRENCE AFTER CURATIVE RESECTION OF PANCREATIC NEUROENDOCRINE TUMORS**

*A Pulvirenti, J He, L Landoni, N Jamieson, JF Chou, M Miotto, A Javed, M Gonen, A Pea, LH Tang, C Nessi, S Cingarlini, MI D'Angelica, A Gill, TP Kingham, A Scarpa, MJ Weiss, VP Balachandran, J Samra, JL Cameron, WR Jarnagin, R Salvia, CL Wolfgang, PJ Allen, C Bassi*

**Presenter:** Alessandra Pulvirenti MD | University of Verona

**Background:** Among patients undergoing resection of pancreatic neuroendocrine tumors (PanNETs), approximately 17% 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

## ORAL ABSTRACTS *(continued)*

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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.

## **32. IMMUNE MODULATION BY LIPOPOLYSACCHARIDE SUPPRESSES PANCREATIC CANCER PROGRESSION**

*A Ferrantella, P Sharma, M Tarique, S Kurtom, V Sethi, B Giri, H Jacob, P Roy, S Lavania, S Ramakrishnan, A Saluja, V Dudeja*

**Presenter:** Anthony Ferrantella MD | University of Miami

**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.

# ORAL ABSTRACTS *(continued)*

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

	Gene	Number of patients with variant (%)	Agent Available
Likely Pathogenic	ATM	1 (2%)	Carboplatin Cisplatin Oxaliplatin
	FH	1 (2%)	No agent available
	TP53	6 (12%)	No agent available
Pathogenic	ARID1A	3 (6%)	No agent available
	ATM	8 (16%)	Carboplatin Cisplatin Oxaliplatin
	BRCA1	2 (4%)	Carboplatin Cisplatin Oxaliplatin Mitomycin C Olaparib
	BRCA2	1 (2%)	Carboplatin Cisplatin Oxaliplatin Mitomycin C Olaparib
	CDKN2A	9 (18%)	No agent available
	GNAS	1 (2%)	No agent available
	KRAS	32 (65%)	No agent available

# ORAL ABSTRACTS *(continued)*

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# ORAL ABSTRACTS *(continued)*

Table 1. Patient Demographics, Perioperative Variables & Overall Survival				
Total (n = 5364)	pCR (n = 41)	nCR (n = 57)	iCR (n = 5266)	p value
Age, yr, median (IQR)	63 (52-69)	64 (57-68)	64 (57-70)	0.29
Male, n (%)	20 (49%)	34 (60%)	2203 (51%)	0.43
<b>Race, n (%)</b>				
White	34 (85%)	47 (85%)	4613 (89%)	0.68
Black	4 (10%)	7 (13%)	467 (9%)	
Asian	2 (5%)	1 (2%)	120 (2%)	
Hispanic, n (%)	3 (8%)	3 (5%)	180 (4%)	0.29
<b>Morbidity, n (%)</b>				
Charlson-Deyo 0	31 (75%)	43 (75%)	3599 (68%)	0.38
Charlson-Deyo 1	10 (25%)	12 (21%)	1358 (26%)	
Charlson-Deyo 2+	0 (0%)	2 (4%)	309 (6%)	
Time to Chemotherapy, d, median (IQR)	28 (20-36)	29 (20-41)	27 (18-40)	0.83
Time to Radiation, d, median (IQR)	51 (36-98)	45 (26-95)	68 (31-128)	0.14
Time to Surgery, d, median (IQR)	195 (124-276)	157 (127-212)	139 (106-186)	<b>0.0001</b>
<b>Clinical Stage, n (%)</b>				
Stage I	6 (15%)	17 (30%)	1251 (24%)	0.06
Stage II	28 (68%)	24 (42%)	3091 (59%)	
Stage III	7 (17%)	16 (28%)	924 (17%)	
<b>Procedure, n (%)</b>				
Pancreaticoduodenectomy	30 (73%)	43 (75%)	3873 (75%)	0.51
Distal Pancreatectomy	7 (17%)	4 (7%)	558 (11%)	
Total Pancreatectomy	4 (10%)	10 (18%)	742 (14%)	
<b>Resection Margin, n (%)</b>				
Negative	41 (100%)	54 (95%)	4152 (82%)	<b>0.03</b>
Microscopic	0 (0%)	2 (3%)	528 (10%)	
Macroscopic	0 (0%)	0 (0%)	20 (1%)	
NOS	0 (0%)	1 (2%)	328 (7%)	
LOS, d, median (IQR)	8 (7-12)	7 (6-10)	8 (6-12)	0.23
Readmission, 30d	3 (8%)	6 (11%)	418 (8%)	0.80
Mortality, 90d	0 (0%)	2 (4%)	141 (3%)	0.23
Overall Survival, m, median (IQR)	43 (29-54)	24 (16-37)	23 (15-35)	<b>0.0001</b>

yr, year; IQR, Interquartile range; d, day; LOS, length of stay; m, month

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 Gemenetzi, 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

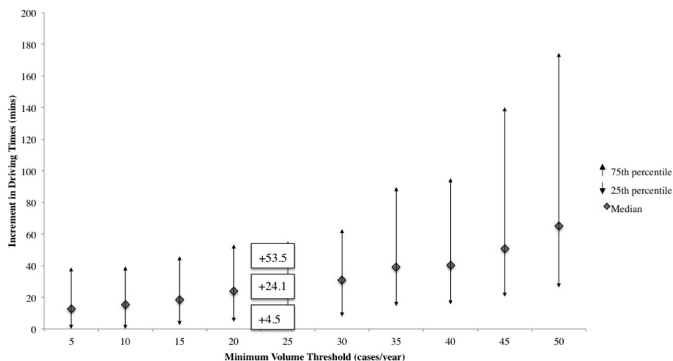
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(OS) and disease free survival (DFS) for each population were evaluated using Kaplan-Meier curves.

**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.



**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.

## ORAL ABSTRACTS *(continued)*

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**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.

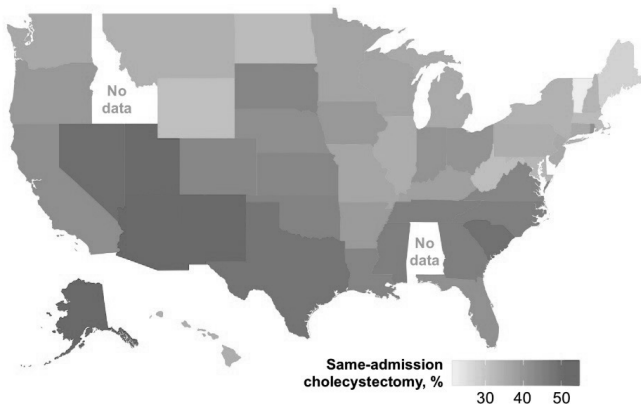
**Results:** The mean protein concentration obtained by FNA was 10.4 mg/ml and 30.6 mg/ml for 22-gauge and 19-gauge needles ( $p = 0.03$ ), respectively. The mean protein concentration in tissue homogenates was 1.49 mg/ml in PDAC, 1.46 mg/ml in pancreatitis, and 2.65 mg/ml in other benign tissue. To 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

# ORAL ABSTRACTS *(continued)*

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non-concertive

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



## **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-1 $\beta$  (IL-1 $\beta$ ) and prostaglandin E2 (PGE2), have been individually suggested as indicators of IPMN dysplasia. We sought to determine if IL-1 $\beta$  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-1 $\beta$  by ELISA. Patients underwent surgical resection with pathology-proven IPMN. Threshold values of PGE2 (>1100pg/mL), IL-1 $\beta$  (>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.

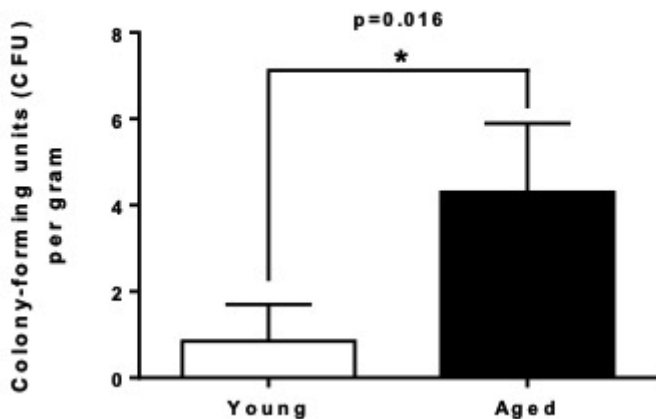
**Results:** Levels of IL-1 $\beta$  were higher in HGD/Invasive-IPMN (n=42) compared to Low/Moderate-IPMN (n=37) (median:range 54.6: 0-2671 vs. 5.9: 0-797pg/mL; P<0.001; AUC 0.766). Similarly, PGE2 was higher in HGD/Invasive-IPMN (n=45) compared to Low/Moderate-IPMN (n=47) (median:range 1790: 20-15180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

**Conclusion:** Cyst fluid PGE2, IL-1 $\beta$ , 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.

## ORAL ABSTRACTS *(continued)*

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## Pancreatic Infection AP 24h



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

## ORAL ABSTRACTS *(continued)*

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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

## ORAL ABSTRACTS *(continued)*

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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.



# ORAL ABSTRACTS *(continued)*

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## 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

*Table 1: Success rates and time to definitive treatment by initial treatment strategy in disconnected pancreatic duct syndrome (DPDS)*

Initial treatment strategy	n (%)	Success as definitive therapy, n (%)	Median time to definitive treatment (range)
Debridement and external drainage	91 (33%)	25 (27%)	237 days (10-3482)
Cystogastrostomy	78 (28%)	53 (68%)	163 days (13-2439)
Endoscopic	36 (13%)	23 (64%)	
Surgical	42 (15%)	30 (71%)	
Percutaneous drain	51 (19%)	17 (33%)	192 (22-1352)
Distal pancreatectomy (+/- splenectomy)	26 (9%)	23 (88%)	112 days (30-2482)
Internal drainage (pancreatico-/cysto-jejunostomy)	20 (7%)	15 (75%)	144 days (23-2481)
None	8 (3%)	---	---
Died before treatment	15	---	---
p-value			0.27

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.

# ORAL ABSTRACTS *(continued)*

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## ORAL ABSTRACTS *(continued)*

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## **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)*

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## **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.

# ORAL ABSTRACTS *(continued)*

Table 1. Morbidity - 264 total events in 111 patients (53%)

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

\*26/28 required percutaneous drainage

\*\*required reoperation



# ORAL ABSTRACTS *(continued)*

Table. Summary of outcome measures in meta-analysis comparing endoscopy and minimally invasive surgery for infected necrotizing pancreatitis

Outcome measure	Number of patients (n)		Pooled Estimate: mean % (95% CI)		Pooled risk ratio (95% CI)	p-value
	Endoscopy	Surgery	Endoscopy	Surgery		
Death	95	89	14.5 (8.3 - 22.1)	16.1 (5.0 - 31.8)	1.02 (0.42 - 2.51)	0.963
New onset multiple organ failure	95	89	5.2 (1.7 - 10.4)	19.7 (6.1 - 38.7)	0.34 (0.12 - 0.98)	0.045
Enterocutaneous fistula/perforation	95	89	3.6 (0.2 - 11.2)	17.9 (10.8 - 26.3)	0.34 (0.13 - 0.92)	0.034
Pancreatic fistula	86	83	4.2 (0.4 - 11.8)	38.2 (19.9 - 58.5)	0.13 (0.05 - 0.37)	<0.001
Intraabdominal bleeding	95	89	6.2 (0.2 - 27.0)	12.3 (3.5 - 25.4)	0.60 (0.10 - 3.60)	0.575
New onset diabetes mellitus	85	79	22.1 (14.1 - 31.3)	27.3 (18.0 - 37.7)	0.78 (0.45 - 1.36)	0.380
Pancreatic exocrine insufficiency	85	79	44.6 (7.5 - 85.8)	63.3 (30.9 - 90.1)	0.99 (0.72 - 1.37)	0.971
Length of hospital stay (days)	85	79	-	-	-0.41 (-0.72 to -0.10)	0.010

## **44. PATHOLOGY HAS THE LAST WORD - PANCREATIC FIBROSIS IS A BETTER PARAMETER FOR PREDICTION OF PANCREATIC FISTULA THAN TEXTURE - A RETROSPECTIVE ANALYSIS FROM THE RECOPANC TRIAL**

*E Petrova, S Timme, L Bolm, M Werner, T Keck, P Bronsert, U Wellner*

**Presenter:** Ekaterina Petrova MD | University Clinic Schleswig-Holstein (UKSH) Campus Lübeck, Germany

**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.

# ORAL ABSTRACTS *(continued)*

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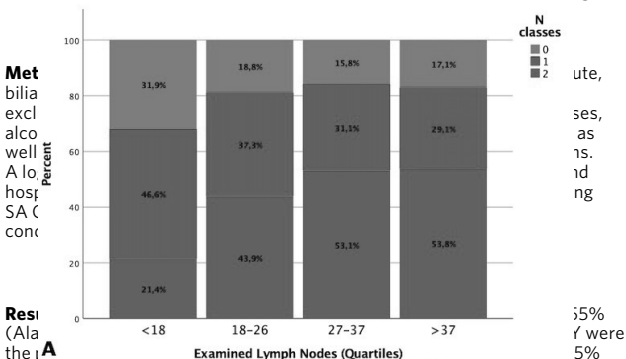
# ORAL ABSTRACTS *(continued)*

## 45. PREDICTORS OF SAME-ADMISSION CHOLECYSTECTOMY IN MILD, ACUTE, BILIARY PANCREATITIS

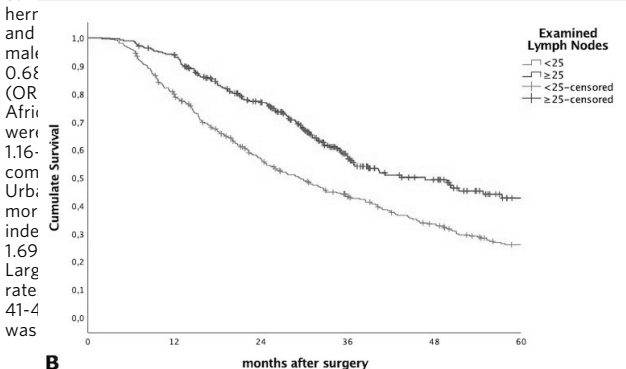
A Shmelev, SC Cunningham

**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 associated with same-admission cholecystectomy.

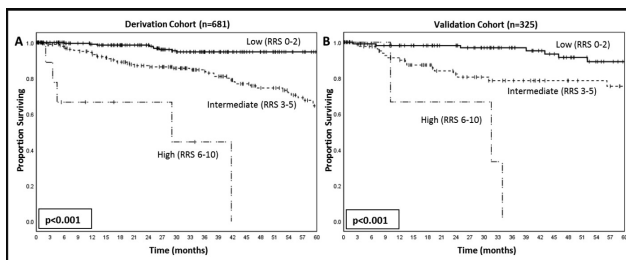


confidence interval [CI] 26.4-27.4), and the presence of ventral or umbilical



# 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.



## **46. INTERLEUKIN 4 RECEPTOR SIGNALING MEDIATES A REGENERATIVE RESPONSE IN THE DUCTAL EPITHELIUM IN RESPONSE TO PANCREATITIS**

KN Von Alt, M Mino-Kenudson, KD Lillemoe, C Fernández-Del Castillo, AL Warsaw, AS Liss

**Presenter:** Kate Von Alt BS | Massachusetts General Hospital

**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-4R $\alpha$ ). In this study we investigate the role of IL-4R $\alpha$  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-4R $\alpha$ -/- 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-4R $\alpha$  contributes to proliferation in the intrapancreatic biliary ductal system, we employed mice deficient in this receptor (IL-4R $\alpha$ -/-). Similar to WT mice, non-injured IL-4R $\alpha$ -/- 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

15 post-injury.

**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-4R $\alpha$  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- $\alpha$ , 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- $\alpha$  levels were observed in the aged group compared to the young group

# ORAL ABSTRACTS *(continued)*

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

Variable, n (%)	Malignancy				
	Univariable analysis		p-value	Multivariable analysis*	
	Yes 19 (10.9)	No 155 (89.1)		Hazard Ratio (95% CI)	p-value
Type of Institution			0.229		
Low-volume	4 (6.3)	59 (93.7)			
High-volume	15 (13.5)	95 (86.5)			
Age			0.068	1 (ref)	-
≤ 65	9 (7.6)	110 (92.4)			
> 65	10 (18.2)	45 (81.8)		<b>4.217 (1.348-13.188)</b>	<b>0.013</b>
Sex			1.000		
Male	10 (11.2)	79 (88.8)			
Female	9 (10.6)	76 (89.4)			
Number of Tumors			0.318		
1	16 (9.9)	145 (90.1)			
≥2	3 (23.1)	10 (76.9)			
Tumor Location			<b>0.023</b>		
Head	5 (15.6)	27 (84.4)			
Body-Tail	11 (8.2)	123 (91.8)			
Diffuse	3 (37.5)	5 (62.5)			
Tumor size			<b>0.009</b>	1 (ref)	-
≤ 2cm	1 (1.6)	60 (98.4)			
> 2cm	18 (15.9)	95 (84.1)		<b>12.320 (1.480-102.554)</b>	<b>0.020</b>
Bile Duct Dilation			0.506		
No	18 (10.7)	150 (89.3)			
Yes	1 (16.7)	5 (83.3)			
Pancreatic Duct Dilation			0.193		
No	17 (10.3)	148 (89.7)			
Yes	1 (14.3)	6 (85.7)			
Unknown	1 (50.0)	1 (50.0)			
Septations			0.098		
No	10 (7.9)	117 (92.1)			
Yes	8 (18.6)	35 (81.4)			
Unknown	1 (25.0)	3 (75.0)			
Solid Component			0.104		
No	7 (7.1)	92 (92.9)			
Yes	12 (16.0)	63 (84.0)			
Calcifications			0.402		
No	15 (9.9)	136 (90.1)			
Yes	4 (19.0)	17 (81.0)			
Unknown	0 (0)	2 (100)			
Hypervascular Rim			0.444		
No	9 (11.2)	71 (88.8)			
Yes	7 (8.9)	72 (91.1)			
Unknown	3 (20.0)	12 (80.0)			
Wall Thickness			0.638		
Thin	10 (13.5)	64 (86.5)			
Thick	7 (8.9)	72 (91.1)			
Unknown	2 (9.5)	19 (90.5)			
Radiologically Suspect Lymph Nodal Involvement			0.918		
No	18 (10.6)	152 (89.4)			
Yes	1 (25.0)	3 (75.0)			
Initial management			0.394		
Upfront Surgery	18 (12.1)	131 (87.9)			
Surveillance > 6 Months Before Surgery	1 (4.0)	24 (96.0)			

\*Also adjusted for the date of diagnosis



# ORAL ABSTRACTS *(continued)*

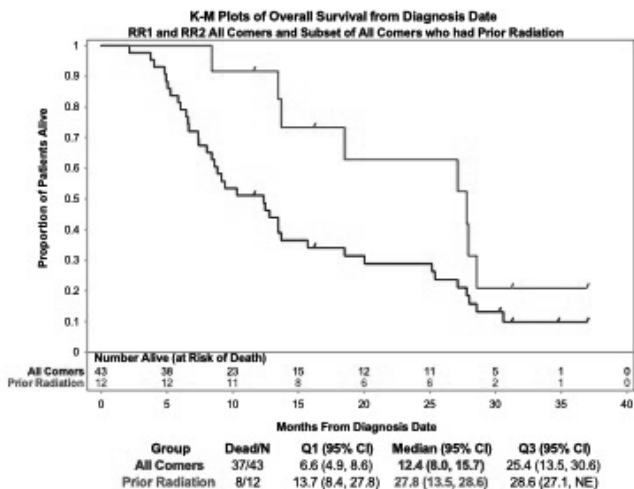
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# ORAL ABSTRACTS *(continued)*

## 48. THERAPEUTIC USE OF ADIPOSE-DERIVED STROMAL CELLS IN A MURINE MODEL OF ACUTE PANCREATITIS

AM Roch, TK Maatman, TG Cook, H Wu, KL March, NJ Zyromski

**Presenter:** Alexandra Roch MD | Indiana University School of Medicine



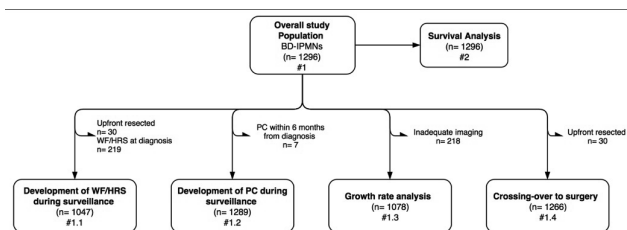
Notes: Forward slashes (/) indicate censored (alive) subjects. Month = Number of Days/30.44.

... mice treated with ASCs had less severe ... 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 TNF $\alpha$  ( $p<0.05$ ). 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

# ORAL ABSTRACTS *(continued)*

inflammation and end organ injury in a murine model of acute pancreatitis. ASCs represent an efficient and attractive therapy for acute pancreatitis.



## **49. THE CLINICAL COURSE AND DIAGNOSTIC WORK-UP OF IDIOPATHIC ACUTE PANCREATITIS, A POST-HOC ANALYSIS OF A PROSPECTIVE MULTICENTER OBSERVATIONAL COHORT**

*ND Hallensleben, DS Umans, SA Bouwense, RC Verdonk, MG Besselink, JE van Hooft, MJ Bruno*

**Presenter:** Devica Umans | Erasmus Medical Center

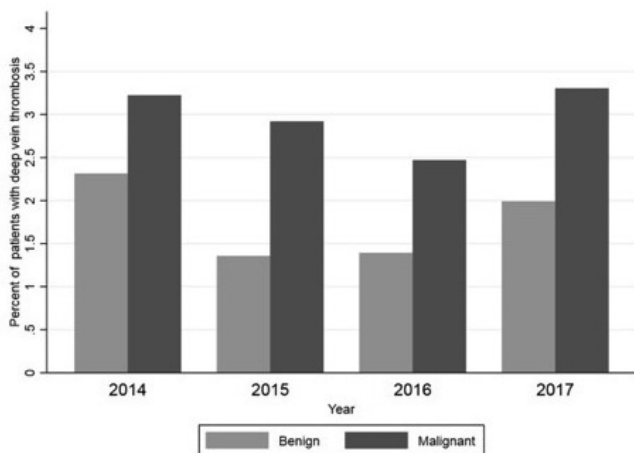
**Background:** After standard diagnostic work-up, the etiology of acute pancreatitis remains unknown in up to 25% of cases, a condition referred to as idiopathic acute pancreatitis (IAP). Determining the etiology of pancreatitis is essential, as it may direct treatment in the acute phase of the disease 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

## ORAL ABSTRACTS *(continued)*

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%).



## **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 (pancreatico- 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.

## ORAL ABSTRACTS *(continued)*

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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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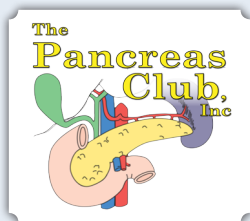
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