10TH

10TH CONFERENCE OF THE HUNGARIAN PANCREATIC STUDY GROUP

PROGRAM BOOK

VECSÉS, HUNGARY

25-27 FEBRUARY 2022

HUNGARIAN PANCREATIC STUDY GROUP

CONTENTS

WELCOME

Dear Colleagues,

We thank you for celebrating with us the 10th Anniversary of the Hungarian Pancreatic Study Group. We do appreciate that you are all here either online or in person.

We believe that we have planned a conference with an exciting mix of contemporary content, and cutting-edge science from the field of pancreatology. True to our aim of facilitating and supporting networking, we have prepared the programme so that there is time and opportunity for interactions with colleagues, during and after the sessions.

Lately, we have not had many opportunities to catch up with each other in person, thus we have organized various social activities in the evenings that we hope will strengthen old friendships and help to make new acquaintances.

On behalf of HPSG and the local organising committee, we would like to welcome you to the 10th Conference of the Hungarian Pancreatic Study Group and wish you all a happy and productive stay in Vecsés and Budapest.

Péter Hegyi
co-chair

Miklós Sahin-Tóth
co-chair

Scientific and Organizing Committee

SCIENTIFIC COMITTEE

Péter Hegyi
Semmelweis University, Budapest and University of Pécs, Pécs, Hungary

Miklós Sahin-Tóth
University of California Los Angeles, Los Angeles, California, USA

Andrea Párniczky
Heim Pál National Pediatric Institute, Budapest, Hungary and University of Pécs, Pécs, Hungary

Bálint Erőss
Semmelweis University, Budapest and University of Pécs, Pécs, Hungary

Andrea Szentesi
University of Pécs, Pécs, Hungary

CONFERENCE VENUE

Hotel Stáció
Széchenyi utca 20, Vecsés, Hungary, H-2220
Phone: + 36 29 353 053
E-mail: info@hotelstacio.hu
www.airporthotelbudapest.hu

CONFERENCE OFFICE

Máté Lukácsi, event organiser
Centre for Translational Medicine,
Semmelweis University, Budapest, Hungary
Phone: +36 30 450 76 65
E-mail: 10hpsg@gmail.com

INVITED FACULTY

Maisam Abu-El-Haija, Cincinnati Children's Hospital Medical Center, Cincinatti, Ohio, USA

Katalin Borka, Semmelweis University, Budapest, Hungary

Marco Bruno, Erasmus University, Rotterdam, The Netherlands

Stefania Bunduc, 'Carol Davila' University of Medicine and Pharmacy, Bucharest, Romania

Daniele Campa, University of Pisa, Pisa, Italy

Federico Canzian, German Cancer Research Centre, Heidelberg, Germany

László Czakó, University of Szeged, Szeged, Hungary

Alexandra Demcsák, University of California Los Angeles, Los Angeles, California, USA

Juan Enrique Domínguez-Muñoz, University of Santiago de Compostela, Santiago de Compostela, Spain

Attila Doros, Semmelweis University, Budapest, Hungary

Bálint Erőss, Semmelweis University, Budapest and University of Pécs, Pécs, Hungary

Pramod Garg, All India Institute of Medical Sciences, New Delhi, India

Andrea Geisz, Boston University, Boston, Massachusetts, USA

Cristian Gheorghe, 'Carol Davila' University of Medicine and Pharmacy, Bucharest, Romania

Thomas Gress, University of Marburg, Marburg, Germany

Andrea Párniczky, Heim Pál National Pediatric Institute, Budapest, Hungary and University of Pécs, Pécs, Hungary

Ole Petersen, Cardiff University, Cardiff, Wales, United Kingdom

Zoltán Rakonczay, University of Szeged, Szeged, Hungary

Vinciane Rebours, Beaujon Hospital, Clichy, France

Jonas Rosendahl, Martin Luther University, Halle-Wittenberg, Germany

Miklós Sahin-Tóth, University of California Los Angeles, Los Angeles, California, USA

Ashok Saluja, University of Miami, Miami, Florida, USA

Vijay Singh, Mayo Clinic, Phoenix, Arizona, USA

Andrea Szentesi, University of Pécs, Pécs, Hungary

Attila Szijártó, Semmelweis University, Budapest, Hungary

Ákos Szücs, Semmelweis University, Budapest, Hungary

Imola Török, George Emil Palade University of Medicine, Pharmacy, Science and Technology of Targu Mures, Targu Mures, Romania

Viktória Venglovecz, University of Szeged, Szeged, Hungary

David Whitcomb, University of Pittsburgh, Pittsburgh, Pennsylvania, USA

László Zubek, Semmelweis University, Budapest, Hungary

GENERAL INFORMATION

CONFERENCE VENUE

The scientific sessions take place in the Neumann meeting room of the Hotel Stáció.

REGISTRATION

The registration desk is in the hotel lobby.
Registration hours:
Thursday, 24 February 2022: 17:00-20:00
Friday, 25 February 2022: 08:00-18:00
Saturday, 26 February 2022: 08:30-17:30
Sunday, 27 February 2022: 08:30-14:00

REGISTRATION ITEMS

Your conference registration includes access to the scientific programmes, final programme book, coffee breaks, lunches, dinner at the conference venue on Thursday and Friday, the cultural programme at the conference hotel on Friday evening, and the conference dinner in Downtown Budapest on Saturday evening.

ORAL PRESENTATIONS

Presentations will be uploaded with the help of our technician inside the conference room. Speakers can upload their presentation during breaks. Speakers in morning sessions are kindly requested to upload their presentation before the first session of the day or on the day before. Speakers in afternoon sessions should upload their slides during the lunch break at the latest.

Invited lecture duration: 16 minutes followed by 4 minutes of discussion. Free paper presentation duration: 8 minutes followed by 2 minutes of discussion.

SOCIAL PROGRAMMES

Thursday, 24 February 2022 **20:00 – 22:00**	Informal dinner in the restaurant of the conference venue
Friday, 25 February 2022 **19:00 – 19:45** **20:00 – 23:00**	Cultural programme in the conference room of the Hotel Stáció Dinner in the restaurant of the conference hotel
Saturday, 26 February 2022 **19:00**	Meeting in the hotel lobby and departure for the dinner in Downtown Budapest. (Bus transfer will be provided.)
20:00 – 22:30 **22:30**	Dinner in Downtown Budapest Bus transfer back to Hotel Stáció

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CONFERENCE PROGRAM

FRIDAY 25 FEBRUARY 2022

08:00–18:00
REGISTRATION DESK
REGISTRATION

08:30-09:00
ROOM NEUMANN
CONFERENCE OPENING

General introduction
Péter Hegyi
Hungarian Pancreatic Study Group

09:00-10:20
ROOM NEUMANN
SESSION 1 - ACUTE PANCREATITIS 1

Chairs: *Julia Gerasimenko*, Cardiff University, Cardiff, Wales, United Kingdom
Gábor Varga, Semmelweis University, Budapest, Hungary

09:00-09:20 **HPSG INTRODUCTION**
Discoveries by Hungarian researchers in the field of acute pancreatitis: Basic research overview
Zoltán Rakonczay, University of Szeged, Szeged, Hungary

09:20-09:40 **Calcium-dependent interactions between pancreatic acinar and stellate cells as well as pancreatic macrophages drive the development of acute pancreatitis**
Ole Petersen, Cardiff University, Cardiff, Wales, United Kingdom

09:40-10:00 **The role of Orai1 channel in pancreatic ductal physiology and pathology**
József Maléth, University of Szeged, Szeged, Hungary

10:00-10:10 **SARS-CoV-2 S protein subunit 1 elicits Ca2+ influx-dependent Ca2+ signals in pancreatic stellate cells and macrophages in situ**
Julia Gerasimenko, Cardiff University, Cardiff, Wales, United Kingdom

10:10-10:20 **Islet damage and regeneration in a mouse model of chronic pancreatitis**
Attila Ébert, Univesity of Szeged, Szeged, Hungary

10:20-10:40
FOYER
COFFEE BREAK

10:40-12:10 ROOM NEUMANN — SESSION 2 - ACUTE PANCREATITIS 2

Chairs: *Ferenc Izbéki*, Szent György Teaching Hospital of Fejér County, Székesfehérvár, Hungary
László Czakó, University of Szeged, Szeged, Hungary

10:40-11:00 **Advanced pre-clinical therapies for acute pancreatitis**
Vijay Singh, Mayo Clinic, Phoenix, Arizona, USA

11:00-11:20 **Summary of the 10-year clinical research experience of HPSG**
Andrea Szentesi, University of Pécs, Pécs, Hungary

11:20-11:40 **Organ failure in acute pancreatitis: a call for action**
Pramod Garg, All India Institute of Medical Sciences, New Delhi, India (online presentation)

11:40-12:00 **Accurate prediction of risk of organ failure within the first day of acute pancreatitis using mathematical modeling and a smart phone**
David Whitcomb, University of Pittsburgh, Pittsburgh, Pennsylvania, USA

12:00-12:10 **HORDEN - NOVEL multipronged method of treatment of acute pancreatitis**
Aleksei Kashintsev, PanDx LLC, St. Petersburg, Russia and PanDx Ltd, Whittlesford, Cambridge, UK

12:10–13:00 RESTAURANT — LUNCH BREAK

13:00–14:20 ROOM NEUMANN — SESSION 3 - EARLY CHRONIC PANCREATITIS 1

Chairs: *Ákos Pap*, Semmelweis University, Budapest, Hungary
Áron Vincze, University of Pécs, Pécs, Hungary

13:00–13:20 **HPSG INTRODUCTION**
Early chronic pancreatitis from the HPSG perspective
Katalin Márta, Semmelweis University, Budapest, Hungary

13:20-13:40 **Heterozygous SPINK1 deficiency promotes progressive pancreatitis in mice**
Alexandra Demcsák, University of California Los Angeles, Los Angeles, California, USA

13:40-14:00 **Cystic pancreatic incidentaloma**
Vinciane Rebours, Beaujon Hospital, Clichy, France

14:00-14:10 **The EFFect Of dietary fat content on the Recurrence of pancreaTitis (EFFORT): protocol of a multicenter randomized controlled trial**
Márk Félix Juhász, University of Pécs, Pécs, Hungary and Semmelweis University, Budapest, Hungary

14:10-14:30 FOYER — COFFEE BREAK

14:30-15:50 ROOM NEUMANN — SESSION 4 - EARLY CHRONIC PANCREATITIS 2

Chairs: *Miklós Sahin-Tóth*, University of California Los Angeles, Los Angeles, California, USA
László Czakó, University of Szeged, Szeged, Hungary

14:30-14:50 **The role of cathepsin B in the early events of pancreatitis**
Andrea Geisz, Boston University, Boston, Massachusetts, USA

14:50-15:10 **Observational longitudinal multicentre investigation of acute pancreatitis (GOULASH PLUS)**
Alexandra Mikó, University of Pécs, Pécs, Hungary

15:10-15:30 **A simple method for diagnosing early chronic pancreatitis**
Péter Jenő Hegyi, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary

15:30-15:50 **Screening, surveillance and targeted therapy in pancreatic cancer**
Julia Mayerle, University of Munich, Munich, Germany

15:50-16:10 FOYER — COFFEE BREAK

16:10-17:50
ROOM NEUMANN

SESSION 5 - PEDIATRIC PANCREATITIS

Chairs: *Andrea Párniczky*, Heim Pál National Pediatric Institute, Budapest, Hungary and University of Pécs, Pécs, Hungary
Maisam Abu-El-Haija, Cincinnati Children's Hospital Medical Center, Cincinatti, Ohio, USA

16:10-16:30 **HPSG INTRODUCTION**
Pediatric pancreatitis from the HPSG perspective
Andrea Párniczky, Heim Pál National Pediatric Institute, Budapest, Hungary and University of Pécs, Pécs, Hungary

16:30-16:50 **Pediatric acute pancreatitis in the twenty-twenties**
Maisam Abu-El-Haija, Cincinnati Children's Hospital Medical Center, Cincinatti, Ohio, USA

16:50-17:10 **Surgical approach to debilitating pancreatitis in children: past, present and future**
Jaimie Nathan, Nationwide Children's Hospital, Columbus, Ohio, USA

17:10-17:30 **Tackling the global problem of drug adverse events, using the prototypic example of asparaginase-associated pancreatitis**
Sohail Husain, Stanford University School of Medicine, Palo Alto, California, USA
(online presentation)

17:30-17:50 **The quest for treatments for pancreatic diseases**
Stephen Pandol, Cedars-Sinai Medical Center, Los Angeles, California, USA
(online presentation)

19:00-19:45
ROOM NEUMANN

SOCIAL EVENT - CULTURAL PERFORMANCE

20:00
RESTAURANT

DINNER

SATURDAY 26 FEBRUARY 2022

08:30–17:30
REGISTRATION DESK
REGISTRATION

09:00-11:00
ROOM NEUMANN
SESSION 6 - CHRONIC PANCREATITIS

Chairs: *Andrea Geisz*, Boston University, Boston, Massachusetts, USA
Balázs Németh, National Institute of Oncology, Budapest, Hungary

09:00-09:20 **HPSG INTRODUCTION**
Genetic risk of chronic pancreatitis in Hungarians: 10 years of HPSG studies
Miklós Sahin-Tóth, University of California Los Angeles, Los Angeles, California, USA

09:20-09:40 **Genetics in chronic pancreatitis: can we still find novel associations?**
Jonas Rosendahl, Martin Luther University, Halle-Wittenberg, Germany

09:40-10:00 **Controversial genetic risk factors in chronic pancreatitis: from CASR to CEL**
Eszter Hegyi, University of Pécs, Pécs, Hungary

10:00-10:20 **Promoter variants in the chymotrypsinogen C gene in patients with chronic pancreatitis**
Balázs Németh, University of Szeged, Szeged, Hungary

10:20-10:30 **In-hospital patient education markedly reduces alcohol consumption after alcohol-induced acute pancreatitis**
Rita Nagy, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary and Heim Pál National Pediatric Institute, Budapest, Hungary

10:30-10:40 **Metabolic associated fatty liver disease is associated with a more severe acute pancreatitis: a prospective cohort analysis of 2053 cases**
Szilárd Váncsa, University of Pécs, Pécs, Hungary and Semmelweis University, Budapest, Hungary

10:40-10:50 **In-hospital rate of gastrointestinal bleeding among acute pancreatitis patients: A systematic review and meta-analysis**
Marie Anne Engh, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary

10:50-11:00 **Risk of chronic pancreatitis in carriers of loss-of-function CTRC variants: A meta-analysis**
Gergő Berke, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary

11:00-11:10 **Dabigatran therapy in trypsin-dependent pancreatitis: preclinical studies**
Zsófia Gabriella Pesei, University of California Los Angeles, Los Angeles, California, USA

11:10-11:30 FOYER — COFFEE BREAK

11:30-12:40 ROOM NEUMANN — SESSION 7 - PANCREATIC SOLID TUMORS 1

Chairs: *Tibor Gyökeres*, Medical Centre Hungarian Defense Forces, Military Hospital, Budapest
Jonas Rosendahl, Martin Luther University, Halle-Wittenberg, Germany

11:30-11:50 **HPSG INTRODUCTION**
Pancreas tumors from the HPSG perspective: Future plans
Bálint Erőss, Semmelweis University, Budapest and University of Pécs, Pécs, Hungary

11:50-12:10 **Identification of novel genetic risk factors for PDAC by SNP functional annotation**
Federico Canzian, German Cancer Research Centre, Heidelberg, Germany

12:10-12:30 **Polygenic and multifactorial risk scores in PDAC risk prediction**
Daniele Campa, University of Pisa, Pisa, Italy

12:30-12:40 **The effect of bile on pancreatic cancer, the importance of mucins**
Eleonóra Gál, University of Szeged, Szeged, Hungary

12:40–13:30
RESTAURANT

LUNCH BREAK

13:30-15:10
ROOM NEUMANN

SESSION 8 - PANCREATIC SOLID TUMORS 2

Chairs: *Miklós Sahin-Tóth*, University of California Los Angeles, Los Angeles, California, USA
Péter Hegyi, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary

13:30-13:50 **Managing of individuals at high risk for familial pancreatic cancer**
Thomas Gress, University of Marburg, Marburg, Germany
(online presentation)

13:50-14:10 **Recent advances in pancreatic cancer therapy**
Ashok Saluja, University of Miami, Miami, Florida, USA

14:10-14:30 **Pancreatic cancer surveillance: current state of affairs and future opportunities**
Marco Bruno, Erasmus University, Rotterdam, The Netherlands

14:30-14:50 **Advanced EUS-guided imaging and tissue acquisition for the differential diagnosis of solid pancreatic lesions**
Juan Enrique Domínguez-Muñoz, University of Santiago de Compostela, Santiago de Compostela, Spain
(online presentation)

14:50-15:10 **ZIP4 is a novel therapeutic target in pancreatic cancer**
Min Li, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, USA

15:10-15:30
FOYER

COFFEE BREAK

15:30-16:50
ROOM NEUMANN

SESSION 9 - CYSTIC FIBROSIS

Chairs: *Gábor Varga*, Semmelweis University, Budapest, Hungary
István Balogh, University of Debrecen, Debrecen, Hungary

15:30-15:50 **HPSG INTRODUCTION**
Physiological and pathophysiological roles of CTRF in pancreatic exocrine and endocrine function
Viktória Venglovecz, University of Szeged, Szeged, Hungary

15:50-16:10 **Phosphatidylserine, CFTR and NBCe1-B: everything is local**
Shmuel Muallem, National Institutes of Health, Bethesda, Maryland, USA
(online presentation)

16:10-16:30 **Early results from the Hungarian CFRPD registry data**
Adrienn Kéri, Heim Pál National Pediatric Institute, Budapest, Hungary

16:30-16:50 **Personalized medicine: pancreas-on-a-chip to study CFRD**
Anjaparavanda Naren, Cedars-Sinai Medical Center, Los Angeles, California, USA
(online presentation)

16:50-17:20
ROOM NEUMANN

SESSION 10 - HIRSHBERG LECTURE IN PANCREAS SURGERY

16:50-17:00 **Welcome and introduction**
Agi Hirshberg, Hirshberg Foundation for Pancreatic Cancer Research, Los Angeles, California, USA
(online presentation)

17:00-17:20 **Surgical management of pancreatic cancer**
Joe Hines, University of California Los Angeles, Los Angeles, California, USA
(online presentation)

19:00
HOTEL LOBBY

GALA DINNER

Meeting in the hotel lobby followed by a bus transfer to the dinner venue in Downtown Budapest.

SUNDAY 27 FEBRUARY 2022

08:30–14:00
REGISTRATION DESK — REGISTRATION

09:00-11:00
ROOM NEUMANN — SESSION 11 - RARE DISEASES AND BIOMARKERS

Chairs: *Stefania Bunduc*, 'Carol Davila' University of Medicine and Pharmacy, Bucharest, Romania
Péter Jenő Hegyi, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary

09:00-09:20 **HPSG INTRODUCTION**
Autoimmune pancreatitis from the HPSG perspective
László Czakó, University of Szeged, Szeged, Hungary

09:20-09:40 **Is there a chance for simple pancreatic ductal adenocarcinoma biomarkers?**
Ewa Małecka-Wojciesko, Medical University of Lodz, Lodz, Poland

09:40-10:00 **EUS with FNA/FNB for pancreatic cancer: The future "standard of care" in the field of individualized medicine**
Cristian Gheorghe, 'Carol Davila' University of Medicine and Pharmacy, Bucharest, Romania

10:00-10:20 **Exosomes as prognostic biomarkers in pancreatic ductal adenocarcinoma**
Stefania Bunduc, 'Carol Davila' University of Medicine and Pharmacy, Bucharest, Romania

10:20-10:40 **Evolution in management of acute pancreatitis over the past 7 years in collaboration with HPSG**
Imola Török, George Emil Palade University of Medicine, Pharmacy, Science and Technology of Targu Mures, Targu Mures, Romania

10:40-11:00 **Complicated cases of pancreatic pathology in clinical practice**
Natalya Gubergrits, Donetsk National Medical University, Ukraine (online presentation)

11:00-11:20
FOYER — COFFEE BREAK

11:20-13:40 ROOM NEUMANN	SESSION 12 - CENTER OF EXCELLENCE IN PANCREATIC DISEASES (CEPD) CORNER

Chairs: *Péter Hegyi*, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary
Attila Szijártó, Semmelweis University, Budapest, Hungary

11:20-11:40 **Multidisciplinary approach in pancreatology - first experiencing and potentials**
Tamás Hussein, University of Pécs, Pécs, Hungary

11:40-12:00 **Management of pancreatic fluid collections - guidelines and daily practice**
Attila Szijártó, Semmelweis University, Budapest, Hungary

12:00-12:20 **Pancreas diseases at Semmelweis University: on the way to Champions League**
Ákos Szücs, Semmelweis University, Budapest, Hungary

12:20-12:40 **Hungarian interventional radiological solutions of hepato-pancreatico-biliary diseases**
Attila Doros, Semmelweis University, Budapest, Hungary

12:40-13:00 **Intensive therapeutic aspects of acute pancreatitis**
László Zubek, Semmelweis University, Budapest, Hungary

13:00-13:20 **Pathological aspects of pancreatic diseases**
Katalin Borka, Semmelweis University, Budapest, Hungary (online presentation)

13:20-13:40 **Step-up approach in the management of acute necrotizing pancreatitis in our practice**
István Hritz, Semmelweis University, Budapest, Hungary

13:40-14:00 ROOM NEUMANN	CLOSING REMARKS

Péter Hegyi, Semmelweis University, Budapest, Hungary and University of Pécs, Pécs, Hungary

14:00-15:00 RESTAURANT	LUNCH

PROGRAM AT A GLANCE

FRIDAY, 25 FEBRUARY 2022

TIME	SESSION CODE	SESSION TITLE	LECTURE TITLE	PRESENTER
08:30-09:00			**Conference opening General introduction**	***Péter Hegyi* Hungary**
09:00-09:20	Session 1	Acute pancreatitis 1	HPSG introduction: Discoveries by Hungarian researchers in the field of acute pancreatitis: Basic research overview	*Zoltán Rakonczay* Hungary
09:20-09:40			Calcium-dependent interactions between pancreatic acinar and stellate cells as well as pancreatic macrophages drive the development of acute pancreatitis	*Ole Petersen* United Kingdom
09:40-10:00			The role of Orai1 channel in pancreatic ductal physiology and pathology	*József Maléth* Hungary
10:00-10:10			SARS-CoV-2 S protein subunit 1 elicits Ca2+ influx-dependent Ca2+ signals in pancreatic stellate cells and macrophages in situ	*Julia Gerasimenko* United Kingdom
10:10-10:20			Islet damage and regeneration in a mouse model of chronic pancreatitis	*Attila Ébert* Hungary
10:20-10:40			**Coffee break**	
10:40-11:00	Session 2	Acute pancreatitis 2	Advanced pre-clinical therapies for acute pancreatitis	*Vijay Singh* USA
11:00-11:20			Summary of the 10-year clinical research experience of HPSG	*Andrea Szentesi* Hungary
11:20-11:40			Organ failure in acute pancreatitis: a call for action	*Pramod Garg* India
11:40-12:00			Accurate prediction of risk of organ failure within the first day of acute pancreatitis using mathematical modeling and a smart phone	*David Whitcomb* USA
12:00-12:10			HORDEN - NOVEL multipronged method of treatment of acute pancreatitis	*Aleksei Kashintsev* Russia
12:10-13:00			**Lunch break**	

12:10-13:00	**Lunch break**			
13:00-13:20	Session 3	Early chronic pancreatitis 1	HPSG introduction: Early chronic pancreatitis from the HPSG perspective	*Katalin Márta* Hungary
13:20-13:40			Heterozygous *SPINK1* deficiency promotes progressive pancreatitis in mice	*Alexandra Demcsák* USA
13:40-14:00			Cystic pancreatic incidentaloma	*Vinciane Rebours* France
14:00-14:10			The EFFect Of dietary fat content on the Recurrence of pancreaTitis (EFFORT): protocol of a multicenter randomized controlled trial	*Félix Juhász* Hungary
14:10-14:30	**Coffee break**			
14:30-14:50	Session 4	Early chronic pancreatitis 2	The role of cathepsin B in the early events of pancreatitis	*Andrea Geisz* USA
14:50-15:10			Observational longitudinal multicentre investigation of acute pancreatitis (GOULASH PLUS)	*Alexandra Mikó* Hungary
15:10-15:30			A simple method for diagnosing early chronic pancreatitis	*Péter Jenő Hegyi* Hungary
15:30-15:50			Screening, surveillance and targeted therapy in pancreatic cancer	*Julia Mayerle* Germany
15:50-16:10	**Coffee break**			
16:10-16:30	Session 5	Pediatric pancreatitis	HPSG introduction: Pediatric pancreatitis from the HPSG perspective	*Andrea Párniczky* Hungary
16:30-16:50			Pediatric acute pancreatitis in the twenty-twenties	*Maisam Abu-El-Haija* USA
16:50-17:10			Surgical approach to debilitating pancreatitis in children: past, present and future	*Jaimie Nathan* USA
17:10-17:30			Tackling the global problem of drug adverse events, using the prototypic example of asparaginase-associated pancreatitis	*Sohail Husain* USA
17:30-17:50			The quest for treatments for pancreatic diseases	*Stephen Pandol* USA
19:00	**Social event**			

SATURDAY, 26 FEBRUARY 2022

TIME	SESSION CODE	SESSION TITLE	LECTURE TITLE	PRESENTER
09:00-09:20	Session 6	Chronic pancreatitis	HPSG introduction: Genetic risk of chronic pancreatitis in Hungarians: 10 years of HPSG studies	*Miklós Sahin-Tóth* USA
09:20-09:40			Genetics in chronic pancreatitis: can we still find novel associations?	*Jonas Rosendahl* Germany
09:40-10:00			Controversial genetic risk factors in chronic pancreatitis: from CASR to CEL	*Eszter Hegyi* Hungary
10:00-10:20			Promoter variants in the chymotrypsinogen C gene in patients with chronic pancreatitis	*Balázs Németh* Hungary
10:20-10:30			In-hospital patient education markedly reduces alcohol consumption after alcohol-induced acute pancreatitis	*Rita Nagy* Hungary
10:30-10:40			Metabolic associated fatty liver disease is associated with a more severe acute pancreatitis: a prospective cohort analysis of 2053 cases	*Szilárd Váncsa* Hungary
10:40-10:50			In-hospital rate of gastrointestinal bleeding among acute pancreatitis patients: A systematic review and meta-analysis	*Marie Anne Engh* Hungary
10:50-11:00			Risk of chronic pancreatitis in carriers of loss-of-function CTRC variants: A meta-analysis	*Gergő Berke* Hungary
11:00-11:10			Dabigatran therapy in trypsin-dependent pancreatitis: preclinical studies	*Zsófia Gabriella Pesei* USA
11:10-11:30	**Coffee break**			
11:30-11:50	Session 7	Pancreatic solid tumors 1	HPSG introduction: Pancreas tumors from the HPSG perspective: Future plans	*Bálint Erőss* Hungary
11:50-12:10			Identification of novel genetic risk factors for PDAC by SNP functional annotation	*Federico Canzian* Germany
12:10-12:30			Polygenic and multifactorial risk scores in PDAC risk prediction	*Daniele Campa* Italy
12:30-12:40			The effect of bile on pancreatic cancer, the importance of mucins	*Eleonóra Gál* Hungary
12:40-13:30	**Lunch break**			

13:30-13:50	Session 8	Pancreatic solid tumors 2	Managing of individuals at high risk for familial pancreatic cancer	*Thomas Gress* Germany
13:50-14:10			Recent advances in pancreatic cancer therapy	*Ashok Saluja* USA
14:10-14:30			Pancreatic cancer surveillance: current state of affairs and future opportunities	*Marco Bruno* The Netherlands
14:30-14:50			Advanced EUS-guided imaging and tissue acquisition for the differential diagnosis of solid pancreatic lesions	*Juan Enrique Domínguez-Muñoz* Spain
14:50-15:10			ZIP4 is a novel therapeutic target in pancreatic cancer	*Min Li* USA
15:10-15:30	**Coffee break**			
15:30-15:50	Session 9	Cystic fibrosis	HPSG introduction: Physiological and pathophysiological roles of CTRF in pancreatic exocrine and endocrine function	*Viktória Venglovecz* Hungary
15:50-16:10			Phosphatidylserine, CFTR and NBCe1-B: everything is local	*Shmuel Muallem* USA
16:10-16:30			Early results from the Hungarian CFRPD registry data	*Adrienn Kéri* Hungary
16:30-16:50			Personalized medicine: pancreas-on-a-chip to study CFRD	*Anjaparavanda Naren* USA
16:50-17:00	Session 10	Hirshberg lecture	Welcome and introduction	*Agi Hirshberg* USA
17:00-17:20			Surgical management of pancreatic cancer	*Joe Hines* USA
19:00	**Gala dinner**			

SUNDAY, 27 FEBRUARY 2022

TIME	SESSION CODE	SESSION TITLE	LECTURE TITLE	PRESENTER
09:00-09:20	Session 11	Rare diseases and biomarkers	HPSG introduction: Autoimmune pancreatitis from the HPSG perspective	*László Czakó* Hungary
09:20-09:40			Is there a chance for simple pancreatic ductal adenocarcinoma biomarkers?	*Ewa Małecka-Wojciesko* Poland
09:40-10:00			EUS with FNA/FNB for pancreatic cancer: The future "standard of care" in the field of individualized medicine	*Cristian Gheorghe* Romania
10:00-10:20			Exosomes as prognostic biomarkers in pancreatic ductal adenocarcinoma	*Stefania Bunduc* Romania
10:20-10:40			Evolution in management of acute pancreatitis over the past 7 years in collaboration with HPSG	*Imola Török* Romania
10:40-11:00			Complicated cases of pancreatic pathology in clinical practice	*Natalya Gubergrits* Ukraine
11:00-11:20	**Coffee break**			
11:20-11:40	Session 12	Centre of Excellence in Pancreatic Diseases (CEPD) Corner	Multidisciplinary approach in pancreatology - first experiencing and potentials	*Tamás Hussein* Hungary
11:40-12:00			Management of pancreatic fluid collections - guidelines and daily practice	*Attila Szijártó* Hungary
12:00-12:20			Pancreas diseases at Semmelweis University: on the way to Champions League	*Ákos Szücs* Hungary
12:20-12:40			Hungarian interventional radiological solutions of hepato-pancreatico-biliary diseases	*Attila Doros* Hungary
12:40-13:00			Intensive therapeutic aspects of acute pancreatitis	*László Zubek* Hungary
13:00-13:20			Pathological aspects of pancreatic diseases	*Katalin Borka* Hungary
13:20-13:40			Step-up approach in the management of acute necrotizing pancreatitis in our practice	*István Hritz* Hungary
13:40-14:00	**Closing remarks**			***Péter Hegyi*** **Hungary**
14:00-15:00	**Lunch**			

FREE PAPER ABSTRACTS

ISLET DAMAGE AND REGENERATION IN A MOUSE MODEL OF CHRONIC PANCREATITIS

A. Ébert(1), T. Gajdos(2), M. Erdélyi(2), R. Molnár(3), D. Kelemen(5), P. Hegyi(3,4,6), V. Venglovecz(1)

(1) Department of Pharmacology and Pharmacotherapy, Univesity of Szeged, Szeged, Hungary
(2) Department of Optics and Quantum Electronics, University of Szeged, Szeged, Hungary
(3) 1st Department of Medicine, University of Szeged, Szeged, Hungary
(4) MTA-SZTE Momentum Translational Gastroenterology Research Group, University of Szeged, Szeged, Hungary
(5) Department of Surgery, Medical School, University of Pécs, Pécs, Hungary
(6) Institute for Translational Medicine/1st Department of Medicine, University of Pécs, Pécs, Hungary

OBJECTIVES

The literature on regenerative therapy in diabetes and pancreatitis has been rapidly growing in the last decade, concentrating mainly on embryonic- and induced pluripotent stem cells. This study aims to identify non-endocrine cell types that serve as a natural source of endocrine cells after repetitive inflammatory injury, and want to investigate if the CFTR chloride channel has any role in beta-cell regeneration.

METHODS

Chronic pancreatitis was induced in FVB/N mice by administration of cerulein. Following completion of disease development, we performed ip. GTT and ELISA. Freshly dissected pancreatic tissues were either fixed in paraformaldehyde for pathology and immunofluorescence or digested enzymatically for isolation of islets. Islets were dispersed and the cells were stained for CFTR and insulin, and imaged with direct stochastic optical reconstruction microscopy.

RESULTS

GTT after four weeks of disease development shows elevated blood glucose levels and decreased, elongated insulin response to glucose load. Based on immunofluorescence, LGR5 positive acini are developed during the disease, with a subset also expressing insulin. The expression of CFTR in beta-cells is decreased after two weeks of disease development, but exceeds the control in the third week, proceeding the increased glucose intolerance.

CONCLUSION

Our results suggest that acinar cells may undergo transdifferentiation into insulin producing cells, and CFTR expression does not correlate with endocrine function, but may have significance in beta-cell differentiation.

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IN-HOSPITAL RATE OF GASTROINTESTINAL BLEEDING AMONG ACUTE PANCREATITIS PATIENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS

Marie Anne Engh(1,2), Levente Frim(2), Dániel Sándor Veres(1), Lajos Szakó(2), Nóra Vörhendi(2), Endre Csiki(1,3), Péter Hegyi(1,2,4), Bálint Erőss(1,2,4)

(1) Centre for Translational Medicine, Semmelweis University, Budapest, Hungary

(2) Institute for Translational Medicine, University of Pécs, Pécs, Hungary

(3) Department of Gastroenterology, Mureş County Clinical Hospital, George Emil Palade University of Medicine, Pharmacy, Science and Technology of Târgu-Mureş, Faculty of Medicine, Târgu-Mureş, Romania

(4) Division of Pancreatic Diseases, Heart and Vascular Center, Semmelweis University, Budapest, Hungary

BACKGROUND

Gastrointestinal bleeding is a complication of pancreatitis, affecting clinical outcome, increasing severity and mortality rates. The aim of this study was to perform a systematic review and meta-analysis of the rate of gastrointestinal bleeding in pancreatitis patients, including sources of bleeding.

METHODS

A systematic search was performed of PubMed, Embase and Cochrane Library until March 2021. Selection, data extraction and risk assessment were done in parallel by two independent investigators, with level of agreement for selection measured by Cohen's kappa. A pooled event rate was calculated for gastrointestinal bleeding with 95% confidence intervals (CI). Separate analyses were performed on the rates of gastrointestinal bleeding in severe acute pancreatitis (SAP), of variceal bleeding following splenic vein thrombosis, and of pseudoaneurysmal bleeding.

Statistical heterogeneity was analyzed using the I2 and $\chi 2$ tests where possible. Risk assessment was done according to the Newcastle Ottawa scale for cohort studies, and a specially adapted tool for case-series.

RESULTS

11,363 records were found, of which 22 studies (121,057 patients) fulfilled the selection criteria. Some studies covered the same population and were combined.

The Cohen's kappa for title, abstract, and fulltext selection showed moderate, almost perfect, and substantial agreement, respectively. Eight included studies were given a risk level of "low", nine a level of "moderate" and three were judged to have a "high" level of risk. The rate of gastrointestinal bleeding was 4% (CI: 2%—6%). Comparing the studies including patients before versus after the Atlanta Classification showed no significant difference. The rate of pseudoaneurysmal bleeding (6 studies) was 0.4% (CI: 0.1%—1.4%). The rate of variceal bleeding (5 studies) was 0.2% (CI:0%—1.2%). The rate of gastrointestinal bleeding in SAP (6 studies) was 16% (CI:13%—18%). The I2 test showed significant heterogeneity in the analysis of GIB overall.

CONCLUSION

Gastrointestinal bleeding is a common complication of pancreatitis. Pseudoaneurysmal and variceal bleedings are rare. GI bleeding is a frequent complication in SAP that deserves further attention.

THE EFFECT OF BILE ON PANCREATIC CANCER, THE IMPORTANCE OF MUCINS

E. Gál(1), Z. Veréb(2), D. Rakk(4) ,A. Szekeres(4), E. Becskeházi(1), L. Kemény(2), L. Tiszlavicz(3), L. Czakó(5), T. Takács(5), P. Hegyi(5,6,7), V. Venglovecz(1)

(1) Department of Pharmacology and Pharmacotherapy, University of Szeged, Szeged, Hungary

(2) Regenerative Medicine and CellularPharmacology Research Laboratory, Department of Dermatology and Allergology, University of Szeged, Szeged, Hungary

(3) Department of Pathology, University of Szeged, Szeged, Hungary

(4) Department of Microbiology, University of Szeged, Szeged, Hungary

(5) First Department of Medicine, University of Szeged, Szeged, Hungary

(6) Institute for Translational Medicine, Medical School, Szentágothai Research Centre, University of Pécs, Pécs, Hungary

(7) Division of Gastroenterology, First Department of Medicine, Medical School, University of Pécs, Pécs, Hungary

Pancreatic cancer (PC) is one of the leading causes of death worldwide and is usually associated with obstructive jaundice (OJ). There is no clear consensus on whether biliary decompression should be performed prior to surgery and how high levels of serum bile affects the outcome of PC. Therefore, our aim was to characterise the effect of BAs on carcinogenic processes using a pancreatic ductal adenocarcinoma (PDAC) cell line and to investigate the underlying mechanisms. Liquid chromatography-mass spectrometry was used to determine the serum concentrations of Basin human serums. The effects of BAs on tumour progression were investigated using different assays. Mucin expressions were studied in normal and PDAC cell lines and in human samples at gene and protein levels and results were validated with gene silencing. The levels of BAs were significantly higher in the PDAC+OJ group compared to the healthy control. Treating PDAC cells with different BAs or with human serum obtained from PDAC+OJ patients enhanced the rate of proliferation, migration, adhesion, colony forming and the expression of MUC4. In PDAC+OJ patients, MUC4 expression was higher and the 4-year survival rate was lower compared to PDAC patients. Silencing of MUC4 decreased BAs-induced carcinogenic processes in PDAC cells. Our results show that BAs promote carcinogenic process in PDAC cells, in which the increased expression of MUC4 plays an important role. These results indicate that in PC patients, where the disease is associated with OJ, the early treatment of biliary obstruction improves life expectancy.

SARS-CoV-2 S protein subunit 1 elicits Ca2+ influx-dependent Ca2+ signals in pancreatic stellate cells and macrophages in situ

Julia V. Gerasimenko, Ole H. Petersen, Oleg V. Gerasimenko
School of Biosciences, Cardiff University, Cardiff, Wales, United Kingdom

The current global pandemic with coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS- CoV-2) has led to million deaths worldwide. It has been shown that the S protein subunit 1 (S1) of SARS-CoV-2 is responsible for the binding of the virus to host cell receptors. However, the subsequent mechanisms of the initial intracellular signalling that follows receptors activation of cells in the exocrine pancreas are still unknown. Recently we have employed the resident pancreatic macrophages in intact live mouse pancreatic lobule preparation to serve as a convenient model of studying immune responses in intact live mouse pancreatic lobules in situ. Using this experimental approach, we observed that S1 elicited Ca2+ signals in pancreatic stellate cells (PSC) and pancreatic macrophages (PM), but not in pancreatic acinar cells. The S1-induced Ca2+ signals were depended on the S1 concentration (70 - 600 nM). PSC responded much faster to application of S1 than PM. The interleukin-18 binding protein (IL-18BP) abolished the responses in PM without affecting the Ca2+ responses in PSC. The S1-elicited Ca2+ signals were dependent on the presence of external Ca2+ and were abolished by a selective blocker (CM4620) of Orai1/Ca2+ Release Activated Ca2+ (CRAC) channels. The S1-elicited Ca2+ signals in PSC and PM may play an important part in the development of the inflammatory process and contribute to the progression to acute pancreatitis.

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The EFFect Of dietary fat content on the Recurrence of pancreaTitis (EFFORT): protocol of a multicenter randomized controlled trial

Márk Félix Juhász(1,2), Zsófia Vereczkei(1), Klementina Ocskay(1), Lajos Szakó(1), Nelli Farkas(1), Zsolt Szakács(1), Noémi Zádori(1), Michael Wilschanski(3), Stephen J. Pandol(4), Francisca Joly(5), Gabriele Capurso(6), Paolo Giorgio Arcidiacono(6), Ferenc Izbéki(7), László Czakó(8), Mária Papp(9), László Czopf(10), Péter Hegyi(1,2,11), Andrea Párniczky(1,2,12*)*

on behalf of the Hungarian Pancreatic Study Group

(1) Institute for Translational Medicine, Medical School, University of Pécs, Pécs, Hungary
(2) Centre for Translational Medicine, Semmelweis University, Budapest, Hungary
(3) Hadassah Hebrew University Medical Center, Jerusalem, Israel
(4) Cedars-Sinai Medical Center and University of California, Los Angeles, USA
(5) Centre for Intestinal Failure, Department of Gastroenterology and Nutritional Support, Hôpital Beaujon, Clichy, France
(6) Pancreato-Biliary Endoscopy and Endosonography Division, Pancreas Translational and Clinical Research Centre, IRCCS San Raffaele Scientific Institute, Milan, Italy
(7) Szent György Teaching Hospital of County Fejér, Székesfehérvár, Hungary
(8) First Department of Medicine, University of Szeged, Szeged, Hungary
(9) Division of Gastroenterology, Department of Internal Medicine, Faculty of Medicine, University of Debrecen, Hungary
(10) First Department of Internal Medicine, Medical School, University of Pécs, Pécs, Hungary
(11) Division of Pancreatic Diseases, Heart and Vascular Center, Semmelweis University, Budapest, Hungary
(12) Heim Pál National Pediatric Institute, Budapest, Hungary

PURPOSE

Approximately 10-30% of patients with acute pancreatitis (AP) develop recurrent acute pancreatitis (RAP) and a third of those with RAP develop chronic pancreatitis (CP). Due to the lack of randomized control trials, recent international guidelines failed to include recommendations aimed at decreasing recurrences. In the everyday practice - without any evidence - a low-fat diet is often favored after AP to avoid subsequent attacks.

AIMS

To provide a high level of evidence concerning nutritional interventions in RAP in order to prevent further episodes.

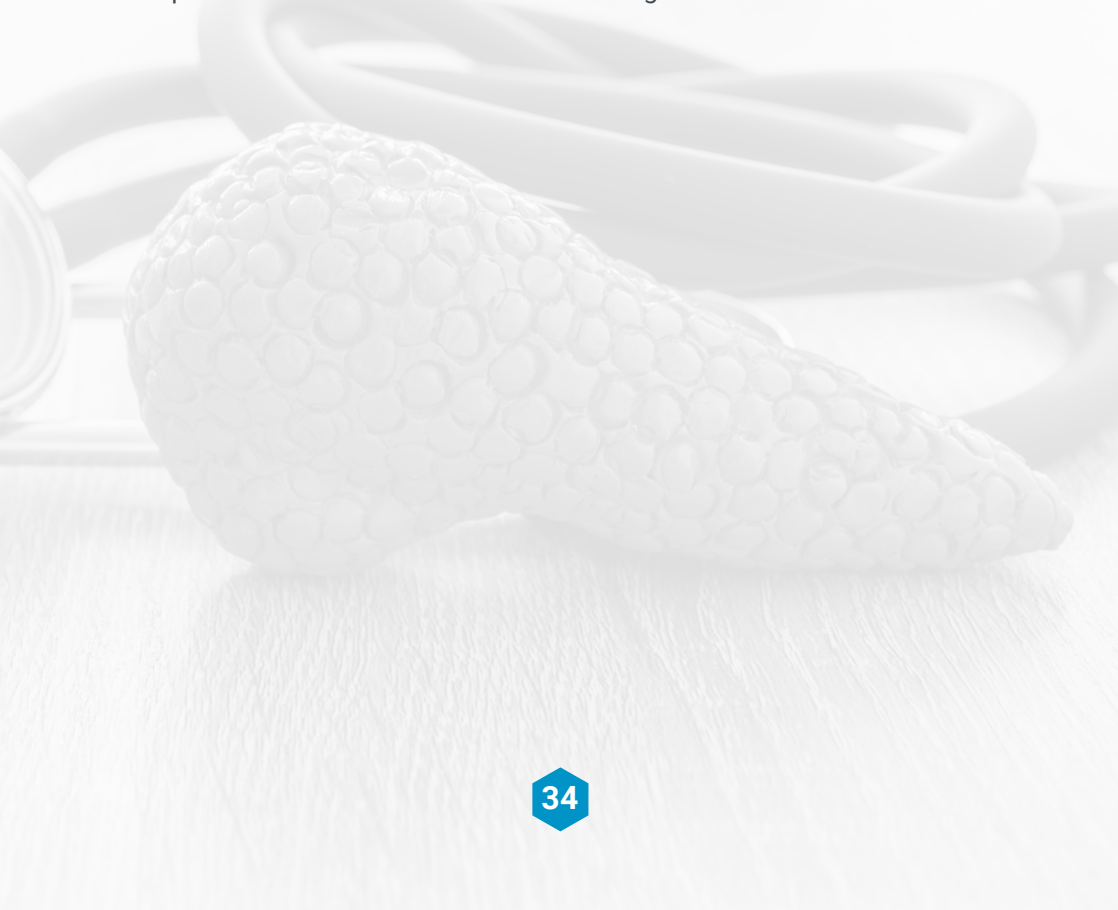
MATERIALS AND METHODS, RESULTS

The Hungarian Pancreatic Study Group (HPSG) has launched the EFFORT-(Effect of dietary fat content on the recurrence of pancreatitis) randomized controlled trial.

384 eligible patients – at least two episodes of AP in the preceding two years, of which the last one was idiopathic – will be randomized to one of the following diets with different fat contents: (1) 'reduced fat diet' (15% fat, 65% carbohydrate, 20% protein) or (2) 'standard healthy diet' (30%-50%-20% respectively, as per WHO recommendations). Participants will be followed-up for two years (visits at months 3, 6, 12, 18 and 24) receiving repeated sessions of nutritional guidance and completing food frequency questionnaires. Primary endpoint is a composite of AP recurrence and all-cause mortality. Secondary endpoints are CP, pancreatitis specific and cardiovascular (CV) cause mortality, body mass index, serum lipid parameters, dietary adherence, adverse events, etc.

CONCLUSIONS

Based on results of the EFFORT trial an evidence-based nutritional recommendation in order to prevent relapses of AP could be stated and incorporated into the international treatment guidelines.



HORDEN - NOVEL multipronged method of treatment of acute pancreatitis

Aleksei Kashintsev, Vitali Proutski, Eldar Kutumov, Anastasya Nadeeva, Oleg Granstrem, Sergei Anisimov
PanDx LLC, St. Petersburg, Russia; PanDx Ltd, Whittlesford, Cambridge, UK

BACKGROUND

Novel method of treatment of acute pancreatitis (AP) is proposed based on creating isolated area with negative pressure in duodenum while preserving connectivity of gastrointestinal tract. The method dubbed HORDEN reduces Humoral stimulation of pancreas, facilitates the Outflow of pancreatic fluid, prevents Reflux of bile and intestinal contents into the main pancreatic duct, allows for GI Drainage and decompression, and enables administration of Enteral Nutrition.

CLINICAL STUDY

An exploratory multicenter, prospective, randomized, controlled, open-label study is evaluating effectiveness of HORDEN in combination with SoC vs. SoC alone. The study enrolls ~150 patients with moderate and severe, biliary and non-biliary AP, randomized 1:1 into Group 1 – SoC+HORDEN, and Group 2 – SoC. HORDEN treatment in patients randomized to Group 1 is initiated within 48hrs post-hospitalization. Patients are evaluated during the entire stay in a study hospital and followed-up, whenever possible, in the event of transfer due to comorbidity. Effectiveness is evaluated with respect to frequency of complications, hospital mortality and duration of hospitalization.

RESULTS AND DISCUSSION

Preliminary analysis based on >100 patients suggests that study Groups are balanced for gender, age, BMI and etiology of AP. A marked reduction is observed in Group 1 with respect to infectious complications as well as the development and persistence of SIRS and MODS. Hospital mortality is considerably lower in Group 1 while LoS in intensive care and overall LoS are also reduced. Interim analysis is planned to provide a more objective assessment of the trends observed so far and to inform further study conduct.

IN-HOSPITAL PATIENT EDUCATION MARKEDLY REDUCES ALCOHOL CONSUMPTION AFTER ALCOHOL-INDUCED ACUTE PANCREATITIS

Rita Nagy(1,2,3), Klementina Ocskay(1,2), Alex Váradi(2), Andrea Párniczky(1,2,3), Mária Papp(4), Zsuzsanna Vitális(4), Ferenc Izbéki(5), Eszter Boros(5), László Gajdán(5), Andrea Szentesi(1,8), Bálint Erőss(1,2,6), Péter Jenő Hegyi(2,6), Áron Vincze(7), Judit Bajor(7), Patricia Sarlós(7), Alexandra Mikó(2), Katalin Márta(1,6), Dániel Pécsi(7), Péter Hegyi(1,2,6)

(1) Centre for Translational Medicine, Semmelweis University, Budapest, Hungary

(2) Institute for Translational Medicine, Szentágothai Research Centre, Medical School, University of Pécs, Pécs, Hungary

(3) Heim Pál National Pediatric Institute, Budapest, Hungary

(4) Division of Gastroenterology, Department of Internal Medicine, University of Debrecen, Debrecen, Hungary

(5) Szent György University Teaching Hospital of Fejér County, Székesfehérvár, Hungary

(6) Division of Pancreatic Diseases, Heart and Vascular Center, Semmelweis University, Budapest, Hungary

(7) Division of Gastroenterology, First Department of Medicine, University of Pécs, Medical School, Pécs, Hungary

(8) Centre for Translational Medicine, Department of Medicine, University of Szeged, Szeged, Hungary

Acute pancreatitis (AP) is an inflammatory disease which can recur in 20-30% of the cases. Although excessive alcohol consumption is by far the most frequent cause of recurrent cases, specific therapy is still not well established to prevent recurrence. Generally, psychological therapy (e.g. brief intervention (BI) is the cornerstone of cessation programs, however, is not yet widely used in everyday practice. Our aim was to investigate the effect of in-hospital brief intervention on alcohol consumption in alcohol-induced AP.

Patients suffering from alcohol-induced AP were consecutively enrolled between 2016 and 2021. During hospital stay patients received 30-min BI by a physician based on the FRAMES model. Patients reported alcohol consumption, serum gamma-glutamyl-transferase (GGT) level and mean cellular volume (MCV) of red blood cells were collected on admission and at the 1-month control visit to monitor patients' drinking habits.

Ninety-nine alcohol-induced AP cases were enrolled in the study (mean age: 50±11, 89% male). Significant decrease was detected both in mean GGT value (294 ± 251 U/L vs 103±113 U/L, $p<0.001$) and in MCV level (93.7±5.3 U/L vs 92.1±5.1U/L, $p<0.001$) in patients with elevated on admission GGT levels. Importantly, 79 % of the patients (78/99) reported alcohol abstinence at the 1-month control visit. Only three recurrent AP cases were observed within the investigated period.

Brief intervention is an effective tool to reduce alcohol consumption and to prevent recurrent AP. Longitudinal randomized clinical studies are needed to identify the adequate structure and frequency of BIs in alcohol-induced AP.

DABIGATRAN THERAPY IN TRYPSIN-DEPENDENT PANCREATITIS: PRECLINICAL STUDIES

Zsófia Gabriella Pesei, Zsanett Jancsó, Balázs Csaba Németh, Miklós Sahin-Tóth
Department of Surgery, University of California Los Angeles, Los Angeles, California, USA

Pancreatitis, the inflammatory disorder of the pancreas, has no specific therapy. Genetic, biochemical and animal model studies revealed that trypsin plays a central role in the onset and progression of pancreatitis. A recent article reported that dabigatran etexilate, the prodrug for the anticoagulant dabigatran, markedly improved experimental pancreatitis in transgenic mice expressing a mutant form of human cationic trypsinogen [J Clin Invest 2020, 130:189-202]. We hypothesized that the therapeutic effect of dabigatran etexilate was due to the trypsin inhibitory activity of dabigatran. Here, we performed biochemical and preclinical mouse experiments to offer proof of concept that orally administered dabigatran etexilate can inhibit pancreatic trypsins and show therapeutic efficacy in trypsin-dependent pancreatitis. We found that dabigatran competitively inhibited all human and mouse trypsin isoforms (Ki range 10-79 nM) and dabigatran plasma concentrations in mice given oral dabigatran etexilate well exceeded the Ki of trypsin inhibition. In the T7K24R trypsinogen mutant mouse model, a single oral gavage of dabigatran etexilate was effective against cerulein-induced progressive pancreatitis with a high degree of histological normalization. Taken together, our observations confirmed that benzamidine derivatives such as dabigatran are potent trypsin inhibitors and show therapeutic activity against trypsin-dependent pancreatitis in T7K24R mice.

RISK OF CHRONIC PANCREATITIS IN CARRIERS OF LOSS-OF-FUNCTION CTRC VARIANTS: A META-ANALYSIS

Amanda Takáts(1), Noémi Gede(1), Gergő Berke(1), Péter Hegyi(1,2,3), Miklós Sahin-Tóth(4), Eszter Hegyi(1)*

(1) Institute for Translational Medicine, Medical School, University of Pécs, Pécs, Hungary
(2) First Department of Medicine, University of Szeged, Szeged, Hungary
(3) Centre for Translational Medicine, Semmelweis University, Budapest, Hungary
(4) Department of Surgery, University of California Los Angeles, Los Angeles, California

The digestive protease chymotrypsin C (CTRC) protects the pancreas against pancreatitis by degrading potentially harmful trypsinogen. Loss-of-function genetic variants in CTRC are associated with chronic pancreatitis (CP), however, the effect size, as judged by reported odds ratio (OR) values, has been variable. Here, we performed a meta-analysis of published studies on four CTRC variants that alter the CTRC amino-acid sequence, are clinically relatively common (global carrier frequency in CP >1%), reproducibly showed association with CP and their loss of function phenotype was verified experimentally. We found strong enrichment of CTRC variants p.A73T, p.V235I, p.K247_R254del, and p.R245W in CP cases versus controls, yielding OR values of 6.5 (95% confidence interval (CI) 2.4-17.8), 4.5 (CI 2.2-9.1), 5.4 (CI 2.6-11.0), and 2.6 (CI 1.6-4.2), respectively. Subgroup analysis demonstrated disease association of variants p.K247_R254del and p.R245W in alcoholic CP with similar effect sizes as seen in the overall CP group. Homozygosity or trans-heterozygosity were rare and seemed to be associated with higher risk.

The results indicate that heterozygous loss-of-function CTRC variants increase the risk for CP approximately 3-7-fold. This meta-analysis confirms the clinical significance of CTRC variants and provides further justification for the genetic screening of CP patients.

METABOLIC ASSOCIATED FATTY LIVER DISEASE IS ASSOCIATED WITH A MORE SEVERE ACUTE PANCREATITIS: A PROSPECTIVE COHORT ANALYSIS OF 2053 CASES

Szilárd Váncsa(1,2,3), Zoltán Sipos(1), Rita Nagy(1,2,3), Klementina Ocskay(1), Félix Juhász(1), Mária Földi(4), Áron Vincze(5), Patricia Sarlós(5), Judit Bajor(5), Ferenc Izbéki(6), Mária Papp(7), Zsuzsanna Vitális(7), József Hamvas(8), László Czakó(9), Nándor Faluhelyi(10), Orsolya Farkas(10), Emőke Miklós(1), Alexandra Mikó(1), Péter Jenő Hegyi(1,3), Melania Macarie(11), Shamil Galeev(12), Elena Ramirez Maldonado(13), Ville Sallinen(14), Imola Török(11), Artautas Mickevicius(15,16), Bálint Erőss(1,2,3), Andrea Párniczky(1,4), Andrea Szentesi(1,2,17), Gabriella Pár(5), Péter Hegyi(1,2,3,17*)*

(1) Institute for Translational Medicine, Szentágothai Research Centre, Medical School, University of Pécs, Pécs, Hungary

(2) Centre for Translational Medicine, Semmelweis University, Budapest, Hungary

(3) Division of Pancreatic Diseases, Heart and Vascular Center, Semmelweis University, Budapest, Hungary

(4) Heim Pál National Pediatric Institute, Budapest, Hungary

(5) Division of Gastroenterology, First Department of Medicine, Medical School, University of Pécs, Pécs, Hungary

(6) Szent György University Teaching Hospital of Fejér County, Székesfehérvár, Hungary

(7) Department of Internal Medicine, Division of Gastroenterology, University of Debrecen, Debrecen, Hungary

(8) Péterfy Hospital, Budapest, Hungary

(9)Department of Medicine, University of Szeged, Szeged, Hungary

(10) Department of Radiology, Medical School, University of Pécs, Pécs, Hungary

(11) County Emergency Clinical Hospital - Gastroenterology and University of Medicine, Pharmacy, Sciences and Technology, Targu Mures, Romania

(12) Saint Luke Clinical Hospital, St. Petersburg, Russia

(13) General Surgery, Consorci Sanitari del Garraf, Sant Pere de Ribes, Barcelona, Spain

(14) Department of Transplantation and Liver Surgery, Helsinki University Hospital and University of Helsinki, Helsinki, Finland

(15) Vilnius University Hospital Santaros Clinics, Vilnius, Lithuania

(16) Clinics of Abdominal Surgery, Nephrourology and Gastroenterology, Faculty of Medicine, Vilnius University, Vilnius, Lithuania

(17) Centre for Translational Medicine, Department of Medicine, University of Szeged, Szeged, Hungary

BACKGROUND

We have shown in a meta-analysis that fatty liver disease (FLD) influences the outcomes of acute pancreatitis (AP). The aim of this study was to further analyze the prognostic role of metabolic-associated fatty liver disease (MAFLD) in AP in a prospective cohort.

MATERIALS AND METHODS

We identified our cohort from the multicentric prospective International Acute Pancreatitis Registry run by the Hungarian Pancreatic Study Group. AP was diagnosed by the revised Atlanta criteria. For the diagnosis of MAFLD, the presence of liver steatosis on abdominal imaging was mandatory, in addition to obesity, type 2 diabetes mellitus, or metabolic dysregulation. Outcomes of interest were in-hospital mortality, AP severity, length of hospital stay, local, and systemic complications of AP.

RESULTS

Out of the 2053 AP cases analyzed, 801 (39%) were diagnosed with MAFLD. Compared to the non-MAFLD group, MAFLD patients were more likely man (65.5 vs 50%, p=0.001), to have alcohol (28.2 vs 16.5%, p=0.001) and hypertriglyceridemia (13.5 vs 2.6%, p=0.001) induced AP. Regarding severity, MAFLD patients were more likely to develop moderately severe (28.1 vs 20.4%, p=0.001) and severe AP (7 vs 4.1%, p=0.001). Similarly, the proportion of local and systemic complications was higher in the MAFLD group. Length of hospitalization was significantly longer in cases with MAFLD (11.5±11.2 vs 10±8.9 days, p=0.001). In-hospital mortality rate in the MAFLD group was similar to the non-MAFLD group (3 vs 2.9%, p=0.874).

CONCLUSION

MAFLD increases severity and causes longer length of hospitalization in AP.

KEYWORDS

Acute pancreatitis, severity, mortality, metabolic associated fatty liver disease, prognosis

INDEX

NOTES

NOTES

NOTES

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