Disclaimer: United European Gastroenterology (UEG) is not responsible for errors or omissions in the abstracts. This abstract book was finalized on August 26, 2013, any changes received after this date have not been incorporated. Changes to presenters received after August 26, 2013 have been included in the online version of the programme and can be obtained at: http://www.e-learning.ueg.eu.

Disclosure policy: The United European Gastroenterology (UEG) is committed to ensuring scientific rigour and objectivity in all of its educational activities. These include all aspects of the educational programme at UEG Week 2013. All presenters, whether invited Faculty or abstract presenters are required to make a formal disclosure of financial or other relationships that could influence the content of a presentation in the form of a disclosure statement. Conflict of interests does not preclude an individual from making a presentation providing the conflict was disclosed.
Aims and scope
Launched in 2013, United European Gastroenterology Journal is the official Journal of United European Gastroenterology (UEG), a professional non-profit organisation combining all the leading European societies concerned with digestive disease. UEG’s member societies represent over 22,000 specialists working across medicine, surgery, paediatrics, GI oncology and endoscopy, which makes UEG a unique platform for collaboration and the exchange of knowledge.

United European Gastroenterology Journal provides an international forum for research in gastroenterology, publishing original articles which describe basic research, translational and clinical studies of interest to gastroenterologists and researchers in related fields. Articles from across all fields of gastroenterology are welcomed by the Editor-in-Chief, including luminal, liver and pancreatic diseases, gastrointestinal surgery, gastrointestinal oncology, paediatric gastroenterology and nutrition as well as endoscopy.

Published article types include original research, reviews, guidelines papers and news items. The journal is a member of the Committee on Publication Ethics (COPE).

2014 annual subscription rates
United European Gastroenterology Journal ISSN: 2050-6406 (print) 2050-6414 (online) is published in February, April, June, August, October and December by SAGE Publications (London, Thousand Oaks, CA, New Delhi, Singapore and Washington DC).

Annual subscription (2014) including postage: Institutional Rate (combined print and electronic) £673/US$1246. Note VAT might be applicable at the appropriate local rate. Visit http://www.sagepublications.com for more details. To activate your subscription (institutions only) visit http://online.sagepub.com online. Abstracts, tables of contents and contents alerts are available on this site free of charge for all. Student discounts, single issue rates and advertising details are available from SAGE Publications Ltd, 1 Oliver’s Yard, 55 City Road, London EC1Y 1SP, UK, tel. +44 (0)20 7324 8500, fax +44 (0)20 7324 8600 and in North America, SAGE Publications Inc, PO Box 5096, Thousand Oaks, CA 91320, USA.

SAGE Publications is a member of CrossRef

Periodicals postage paid at Jamaica, NY. POSTMASTER Send address corrections to United European Gastroenterology Journal, c/o Worldnet Shipping NY Inc., 155-11 146th Street, Jamaica, New York, NY 11434, USA.

Manuscript submission guidelines
To view the manuscript submission guidelines, please visit the Manuscript Submission link at http://ueg.sagepub.com

Peer review policy
United European Gastroenterology Journal operates a conventional single-blind reviewing policy in which the reviewer’s name is always concealed from the submitting author. Papers will be sent for anonymous review by at least two reviewers who will either be members of the Editorial Board or others of similar standing in the field. The Editors’ decision is final and no correspondence can be entered into concerning manuscripts considered unsuitable for publication in United European Gastroenterology Journal. All correspondence, including notification of the Editors decision and requests for revisions, will be sent by email.

Commercial sales
For information on reprints and supplements please contact reprints@sagepub.co.uk and for advertising, please contact UKAdvertising@sagepub.co.uk.

Abstracting and indexing
Please visit http://ueg.sagepub.com and click on more about this journal, then Abstracting/Indexing, to view a full list of databases in which this journal is indexed.

© UEG 2013

Cover image:
© Shutterstock

Apart from fair dealing for the purposes of research or private study, or criticism or review, and only as permitted under the Copyright, Designs and Patents Act 1988, this publication may only be reproduced, stored or transmitted, in any form or by any means, with the prior permission in writing of the Publishers, or in the case of reprographic reproduction, in accordance with the terms of licences issued by the Copyright Licensing Agency or your equivalent national blanket licencing agency. Enquiries concerning reproduction outside of those terms should be sent to SAGE Publications.

Disclaimer: The authors, editors, and publisher will not accept any legal responsibility for any errors or omissions that may be made in this publication. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper by Page Bros., Norwich, UK.
Contents

Letter of Thanks ........................................................................................................ v
Thanks to Partners, Sponsors and Exhibitors .......................................................... vii

UEG Week 2013 Oral Presentations

Monday, October 14, 2013

Opening Plenary Session – Hall 1 ............................................................................. A1
Today’s science; tomorrow’s medicine: From genes to disease in IBD – Hall 2 . ....... A2
Paediatric IBD – Hall Helsinki ................................................................................. A3
H. pylori treatment – Hall 7 .................................................................................... A4
Endotherapy via the submucosal space – Hall 6 ...................................................... A6
Improving colonoscopic practice – Hall 9 ............................................................ A8
Advances in biliopancreatic imaging – Hall 10 .................................................... A9
Oesophageal cancer: Treatment, prognosis and prognosticators – Hall 8 .......... A11
Hepatocellular carcinoma – Salon 11/12 .............................................................. A13
Getting more out of anti-TNF therapy in IBD – Hall 2 ........................................ A16
Management of GI bleeding: A case based discussion – Hall 3 ......................... A18
IBD: Extraintestinal manifestations and complications – Hall Prague ................ A18
Improving survival in colon cancer: Lessons learned from rectal cancer – Hall Oslo A20
Colorectal sensory and motor dysfunction in constipation: Lessons for the management of adult and paediatric patients – Roof Garden ............................................. A20
Coeliac disease: From the tip of the iceberg to deep water – Hall Helsinki .......... A21
Prevention and early detection of upper GI cancer – Hall 7 ................................. A23
Endoscopy towards, into and through the stomach – Hall 6 .............................. A25
Today’s science; tomorrow’s medicine: Genetics of functional disorders – Hall 9 A26
Multimodal therapies in biliopancreatic diseases – Hall 10 ................................. A28
Basic Science Workshop 1: The metagenomic approach to GI disease – Hall 8 A30
Aids to improving endoscopic practice – Salon 11/12 ......................................... A31
Clinical challenges in the anorectal region – Hall 3 .......................................... A32
Long-term outcomes in IBD – Hall Prague .......................................................... A33
IBD: Top late breaking trials and other major advances – Hall Helsinki .......... A35
Clinical management of pancreatic diseases: Important updates – Hall 8 ........ A37
Update on Barrett’s oesophagus – Hall 7 ............................................................. A38
Endoscopic techniques which will change tomorrow’s practice – Hall 6 .......... A40
Today’s science; tomorrow’s medicine: Genetics and pathogenesis – Hall 9 .... A42
New developments in upper GI imaging – Hall 10 ............................................. A44
Basic mechanisms of GI motor and sensory dysfunction – Salon 11/12 ............. A45
Tuesday, October 15, 2013

New treatments for functional GI disorders – Hall Prague ........................................... A47
New tools for IBD diagnosis and monitoring – Hall Oslo ........................................... A50
Treatment of rectal cancer in 2013 – Roof Garden .................................................. A53
Management of pancreatic cancer – Hall Helsinki .................................................... A53
Upper GI interventions and advanced endoscopy: Top late breaking abstracts – Hall 7 .... A53
*H. pylori* and gastric cancer risk – Hall 9 .................................................. A54
Beneficial and detrimental effects of bacteria in the GI tract – Hall 10 ...................... A56
Nutrients, gut function and obesity – Hall 8 .................................................. A59
Endoscopic interventions in pancreatic diseases – Salon 11/12 ............................... A61
IBD therapy: Safety issues – Hall 2 .......................................................... A64
Non-invasive diagnosis and staging of liver disease – Hall Stockholm ...................... A66
Faecal calprotectin as a diagnostic tool in IBS and IBD – Hall Oslo ......................... A66
Role of gut microbiota in GI diseases – Hall Helsinki ............................................. A68
Colonoscopic screening: Top late breaking abstracts – Hall 7 ................................. A69
Viral hepatitis B – Hall 6 .......................................................... A70
Gastro-oesophageal cancer: The science behind the medicine – Hall 9 .................... A71
Challenges in diagnosis and treatment of colorectal cancer – Hall 10 ....................... A73

**Nutrition and gut function – Hall 8** .......................................................... A75
Pancreatitis: Lessons from animal models – Salon 11/12 ........................................ A76
Difficulties in the diagnosis of colitis – Hall Copenhagen ........................................ A78
IBD: Therapy beyond anti-TNFs – Hall Helsinki ...................................................... A78
Recent developments in upper GI and small bowel bleeding – Hall 7 ....................... A81
Complications of cirrhosis – Hall 6 .......................................................... A82
Prevention, detection and management of gastric tumours – Hall 9 ......................... A84
Optimising colonic polyp detection – Hall 10 ................................................... A85
Basic Science Workshop 2: Autophagy a common pathway in GI-inflammation – Hall 8 A87
Pancreatic cancer: Pre-clinical models – Salon 11/12 ............................................ A88
Video Cases – Hall 1 .......................................................... A90
Inherited liver diseases – Hall Stockholm ........................................................... A92
Controversies in oesophageal squamous cell cancer – Hall Prague ....................... A93
Interactions between *H. pylori* and epithelial cells – Roof Garden ..................... A94
IBD epidemiology: New insights – Hall Helsinki .................................................... A94
Screening for colorectal cancer: The facts and the future – Hall 7 ......................... A96
NAFLD and general hepatology: Important updates – Hall 6 .............................. A98
Improving EUS-guided diagnosis – Hall 9 ................................................... A100
Identifying high risk colonic polyps – Hall 10 ................................................... A101
Gastro-oesophageal and small intestinal tumours – Hall 8 .................................. A103
Autoimmune pancreatitis: Mechanism and treatment – Salon 11/12 ...................... A104
Wednesday, October 16, 2013

Safety profile of immunosuppressive therapy in IBD – Hall 2 ........................................ A106
East meets West: Colorectal cancer screening – Hall Stockholm ........................................ A107
Understanding new technologies: A session for the general gastroenterologist – Hall Oslo .... A107
New insights into the pathophysiology of functional GI disorders – Hall 6. ......................... A108
GORD: From diagnosis to treatment – Hall 9 .......................................................... A111
Viral hepatitis C – Hall 10 ................................................................................. A113
Therapeutic ERCP update – Hall 8. ................................................................................. A116
Management of early oesophageal tumours – Salon 11/12 ............................................. A118
Unravelling new pathways in IBD pathogenesis – Roof Garden .................................... A121
Oesophageal motility disorders and eosinophilic oesophagitis – Hall 9 ......................... A123
Capsule endoscopy: From mouth to anus – Hall 10 .................................................. A125
Management issues in pancreatic and biliary cancers – Hall 8 ........................................ A127
Colorectal cancer: Back to the basics – Salon 11/12 ..................................................... A128
Optimizing clinical outcomes in IBD – Roof Garden .................................................... A130
GI Surgery: What’s new in 2013? – Hall 6................................................................. A132
Nutrition: From the nursery to the nursing home – Hall 10 ............................................. A132

UEG Week 2013 Poster Presentations

Monday, October 14, 2013

POSTER PLUS VIDEO I – Poster Area ................................................................. A135
GENETICS OF GI AND LIVER DISEASES I – Poster Area ................................. A141
LIVER & BILIARY I – Poster Area ................................................................. A145
PANCREAS I – Poster Area ............................................................................... A158
ENDOSCOPY AND IMAGING I – Poster Area .................................................... A165
SURGERY I – Poster Area ................................................................................ A196
IBD I – Poster Area .......................................................................................... A201
OTHER LOWER GI DISORDERS I – Poster Area ................................................ A228
OESOPHAGEAL, GASTRIC AND DUODENAL DISORDERS I – Poster Area .... A248
H. PYLORI I – Poster Area ................................................................................ A267
SMALL INTESTINAL I – Poster Area .................................................................... A273
NUTRITION I – Poster Area ................................................................................ A280

Tuesday, October 15, 2013

POSTER PLUS VIDEO II – Poster Area ............................................................. A283
GENETICS OF GI AND LIVER DISEASES II – Poster Area ............................... A288
LIVER & BILIARY II – Poster Area ................................................................. A292
PANCREAS II – Poster Area .............................................................................. A304
ENDOSCOPY AND IMAGING II – Poster Area .................................................. A312
enhancing cell sensitivity to 5-FU. Furthermore, 5-FU exposure markedly downregulated the expression of daily MEK5 expression (p < 0.05), while inducing p53 and p21 expression (p > 0.05).

CONCLUSION: Overall, our results indicate that overactivation of MEK5/ERK5 pathway may contribute to CC aggressiveness and chemoresistance, suggesting the potential inhibition of MEK5/ERK5 signal may be a promising therapeutic approach for CC treatment, warranting further investigation.

(Supported by PTDC/SAU-ORG/119842/2010, PEstOE/SAU/UI4013/2011, Sociedade Portuguesa de Gastrenterologia, SFPH/BD/386/12 and SFPH/HD/79356/2011. The authors thank Dr. Robert Doebele for the kind gift of pWPI-MEK5AA and pWPI-MEK5DD constructs.)

Contact E-mail Address: dampererea@ff.ul.pt

Disclosure of Interest: None Declared

Keywords: Fluorouracil, Chemosensation, Colon Cancer, MEK5/ERK5 Signalling

TUESDAY, OCTOBER 15, 2013 11:00-12:30

Nutrition and gut function – Hall B

OP246 PPARGAMMA IS A MASTER REGULATOR OF LACTASE PRODUCTION BY INTESTINAL EPITHELIAL CELLS

M. Fumery1, A. Langlois2, S. Specia3, C. Dubuquoy2, M. Figeac2, R. Christel3, L. Dubuquoy2, S. Bellinovia4, P. Desreumaux2, B. Bertin3 and C. Dubuquoy3

INTRODUCTION: Lactate intolerance is a frequent condition that causes abdominal discomfort and diarrhea, resulting from lactase (LCT) enzyme deficiency produced by intestinal epithelial cells (IEC). Except for lactose free diet, no treatment can cure lactate intolerance and the regulation of LCT enzyme expression remains unknown. Peroxisome proliferator-activated receptor gamma (PPARγ) is a nuclear receptor highly expressed by IEC playing a key role in gut homeostasis and metabolism regulation.

AIMS&METHODS: Aim: To evaluate the roles of PPARγ in the regulation of lactase production in vivo and in vitro in IEC and in vivo in rodents. Methods: Caco2 cells were treated 24 hours with Pioglitazone (Pio; 1 µM) and with a new PPARγ modulator named GED (amino-phenyl-methoxy-propionic acid; 1mM) or 5-aminosalycilate (5ASA; 30mM). Transcriptional profiling was done using Agilent 2-colors 44K Gene Expression Microarrays. LCT mRNA expression was assessed by quantitative RT-PCR and immunostaining. LCT expression was evaluated in vitro by standard method measuring the amount of glucose after lactose digestion. In vivo involvement of PPARγ was confirmed using RNA interference, antago- nists and inducers in specific PPARγ knock-out C57BL/6 mice (PPARγ-/-). In vivo, LCT expression and activity were determined in the duodenum and jejunum of wild-type rodents orally treated with GED.

RESULTS: Both in microarray and qRT-PCR analysis, LCT mRNA expression was significantly increased by GED, Pio and 5ASA compared to control cells with a mean fold of 5.7 (p < 0.0001), 14.7 (p < 0.0001) and 9.5 (p < 0.05) respectively. LCT protein upregulation was also observed by immunostaining of stimulated Caco-2 cells. Importantly, GED and Pio treatments significantly increased LCT enzyme activity in Caco-2 cells (5.8 ± 2.2 fold; p < 0.005). LCT mRNA expression was significantly decreased both in duodenum (p = 0.028) and jejunum (p = 0.03) compared to control mice. Both LCT mRNA expression and activity were decreased in duodenum (p = 0.05) and jejunum (p < 0.01) of weaned C57BL/6 mice and Sprague-Dawley rats treated one week with GED compared to animals receiving vehicle.

CONCLUSION: PPARγ agonists are again able to increase lactase expression and activity in vivo and in vitro. These findings identify PPARγ as a new master regulator of LCT production by IEC and suggest that modulating PPARγ activity may be a new therapeutic strategy for the management of lactose intolerance.

Contact E-mail Address: benjamin.berti-2@univ-lille2.fr

Disclosure of Interest: None Declared

Keywords: Chronic obstructive pulmonary disease (COPD), effects of activities of daily living

OP247 DISTURBED INTESTINAL INTEGRITY IN PATIENTS WITH COPD: EFFECTS OF ACTIVITIES OF DAILY LIVING

K. Lenaerts1, E. Rutten2, W. Buurman3, E. Wouters4 and J. Elbers5

INTRODUCTION: Chronic obstructive pulmonary disease (COPD) is accepted to be a multicomponent disease with various comorbidities. The contribution of the gastrointestinal tract to the systemic manifestation of COPD has never been investigated. This metabolically active organ may experience recurring local oxygen deficits during daily life, leading to disturbed intestinal integrity in COPD patients.

AIMS&METHODS: 18 patients with moderate COPD (mean FEV1: 55±3%pred) and 14 matched healthy controls were tested on two occasions, a baseline measurement at rest and, at another day, during the performance of activities of daily living (ADLs). To assess enterocyte damage, plasma intestinal fatty acid binding protein (IFABP) levels were determined, whereas urinary excretion of orally ingested sugar probes was measured using liquid chromatography and mass spectrometry to assess gastrointestinal permeability.

RESULTS: Plasma IFABP concentrations were not different between COPD patients and healthy controls at rest. In contrast, 0-3h urinary lactulose:chamomose and sucralose:erythritol ratios and 5-24h urinary sucralose:erythritol ratios were significantly higher in COPD patients compared to controls, indicating increased intestinal permeability and colonisation. The performance of ADLs led to significantly increased plasma IFABP concentrations in COPD patients but not in control subjects. In line, the intestinal permeability difference between COPD patients and controls was intensified.

CONCLUSION: Besides intestinal permeability in COPD patients at rest, performing ADLs led to enteroxy damage in addition to intestinal hyper-permeability in COPD patients but not in controls, indicating functional alteration in the gastrointestinal tract. Hence, intestinal compromise should be considered as a new component of the multi-system disorder COPD.

Contact E-mail Address: kaatje.lenaerts@maastrichtuniversity.nl

Disclosure of Interest: None Declared

Keywords: Chronic disease, Intestinal epithelial barrier, Intestinal epithelial cells, Intestinal injury, Physical activity

OP248 FOLATE PRODUCTION IN BIFIDOBACTERIA FROM INFANT AND ADULT HUMANS

M. R. D’Aimmo1, M. Modesto2, P. Mattarelli3, B. Gorbati2, B. Biavati2, T. Andlidl1 and M. R. D’Aimmo1

INTRODUCTION: Folates – the natural chemically reduced forms of folic acid (vitamin B9) – are cofactors in essential metabolic pathways such as DNA synth- esis and methylation pathways. Humans cannot synthesise folate and depend on intake both from the diet (green vegetables, cereals, rice, milk, fermented milk products, etc.) and from indigenous folate synthesizing bacteria of the intestinal tract. Folate deficiency may cause health problems and increase the risk for neural tube defects and may increase the risk for e.g. certain cancer forms, cardiovascular disease and Alzheimer’s.

AIMS&METHODS: Screening for folate production of the bifidobacteria iso- lates from human adult and infant (1-6 month old) was performed. Strains typical of infants, such as Bifidobacterium longum subsp. infantis and B. breve, and of adults (B. adolescentis) were selected for characterization. The aim of the present work was to investigate bifidobacteria from human host of different age and feeding habits in order to establish a possible correlation between diet and the folate production. Folate is present in many different forms in humans. The detectable forms studied in the present work are 5-CH3-H4, H4 and total folate content. Bifidobacteria strains were cultivated in folate free synthetic media. Validated HPLC method was used to analyze deconvoluted folates extracted from bacterial biomass.

RESULTS: All bifidobacteria tested (both from adult and infant) were able to produce folate. Strains derived from adults were the higher producer of total folate (up to 40 µg/g dry matter) with the predominance of 5-CH3-H4 folate and a low amount of H4 folate. In infants we obtained the opposite results with strains typical of infant habitat producing low amounts of total folate (range from 35 to 200 µg/g dry matter) and an inverted ratio of 5-CH3- H4 folate in infant to total folate content.

CONCLUSION: In agreement with idea of coevolution of host-gut microbiome (Ley et al., 2008) we find that bifidobacteria present in the adult gut were able to produce a large amount of folate whereas strains derived from infant habitat were unable to produce folate. These findings correlate with the diet and the folate require- ment of the host: in infants, in fact, milk feeding is able per se to fulfill the folate needs of the individuals whereas in adults a more complex diet is sometime not able to cover all the folate need. The relevance of the different ratio of 5-CH3-H4 folate production in adults and infants has been studied only in few strains and further studies are requested in order to complete this finding and provide an ecological explanation.


Contact E-mail Address: paolla.mattarelli@unicibo.it

Disclosure of Interest: None Declared

Keywords: Bifidobacterium adolescentis, Bifidobacterium breve, Bifidobacterium longum subsp. infants, Folate production, Gut microbiota

OP249 UNDISSOCIATED GELATIN TANNATE REDUCES INTESTINAL LEAKINESS AND MUCOSA INFLAMMATION BY FORMING A PROTECTIVE BIOFILM: RESULTS FROM IN-ITO AND IN-VIVO STUDIES

I. Bueno1,2, S. Sekkat3, V. Theodore4, M. Dattilo5 and V.R. Taddei, France

INTRODUCTION: Gelatine (GEL) stabilised by cross-linking with tannic acid (TA) forms gelatine tannate (GT). GT is approved as medical device for the oral treatment of diarrhoea as Tassetan®. GT is considered as a protective biofilm on the gut mucosa and has been shown to cure diarrhoea but the mechanism of action needs further investigation.

AIMS&METHODS: We aimed at investigating the effect of GT and its com- pounds (GEL and TA) on the intestinal mucosa using both in vitro and in-vivo models.
The in-vitro "filming" activity was evaluated by Corrositol®; a standard method on Caco-Coated Bead monolayers. The effect of GT (5 or 20 mg/ml) on Caco-Coated monolayers was assessed by Tran-Epithelial Electrical Resistance (TEER) and Lucifer Yellow (LY) before and after apical exposure to S. typhimurium. The tight junction (TJ) proteins, aquaporin-3 (AQP3) and occludin (OCL) were assessed by RT-PCR as markers of TJ integrity.

In-vivo, Wistar rats received orally either GT (250 mg/kg), Gel (125 mg/kg), or TA (125 mg/kg), and 2 h; later were injected IP with LPS from E. coli. Jejunal strips were collected 6 hours later for in vitro TJ permeability measurement using FITC-dextran and mucosal myelo-peroxidase (MPO) activity as a marker of inflammation.

RESULTS: GT increased the corrosion time (hydrochloric ac. 37%) from 400 to 699 sec (p<0.001) suggesting a chemical biofilm protection. In addition, GT; marginally increased the electrical resistance (TEER) of Caco-Coated at 4 hours (from 180 to 260 ohm*cm², p<0.05) and decreased the basal permeability to LY in basal conditions at both 2 and 4h. The LY permeability increased from 1.18 to 7.54 after 2 hours of exposure to S. typhimurium whereas a pre-treatment with GT suppressed basal permeability and increased bacterial invasion by 72%, such been associated with overexpression of AQP3 and OCL at 4 hours (350 and 200% respectively for GT at 20mg/kg).

Six hours after LPS injection in rats, both jejunal TJ permeability and MPO activity were dramatically increased. Oral pretreatment with GT reduced by 78.1% the jejunal increase of permeability whereas Gel and TA did not affected it and subsequently reduced significantly the LPS-induced increase in MPO.

CONCLUSION: Our results confirm that GT acts by mechanical protection of the gut mucosa. The protective biofilm formed by GT prevents the leakiness of the tight Junctions both in basal conditions and after insult by bacteria (in-vitro) and by LPS (in-vivo). These effects cannot be replicated by either tannic acid or gallic acid confirming that GT is the active form to prevent gut leakiness and subsequent inflammation.

Contact E-Mail Address: maurozio.datto@gmail.com
Disclosure of Interest: None Declared

Keywords: Diarrhoea; Gastrointestinal inflammation; MPO, paracellular permeability

OP250 DIABETES AND GASTROINTESTINAL DISORDERS: THE EFFECT OF INTESTINAL METHANE PRODUCTION ON GLUCOCYTIC CONTROL

V. Cesario1, T. A. Di Rienzo1, D. Pitiocc2, M. Campana1, G. D’Angelo1, S. Pecere1, F. D’Aversa1, A. TORTORA1, F. Barbaro1, G. Vitale1, G. Gigante1, G. Latorre1, C. Vittorini1, V. Oliani1, Internal Medicine, Diabetology, POLICLINICO GEMELLI, Rome, Italy

INTRODUCTION: At the state of art it isn’t known the correlation between diabetes and lower gastrointestinal disorders. Some studies show a significantly higher prevalence of small intestinal bacterial overgrowth (SIBO) in patients with type 1 diabetes. No data exists about gastrointestinal methane (CH4) production in patients with diabetes.

AIMS&METHODS: Aim of our study was to evaluate the effect of methaogenic flora eradications on glycomic control and daily insulin requirements in patients with type 1 diabetes in order to identify a possible role of CH4 production on diabetes metabolism. 30 consecutive patients (9 males, 21 females; mean age 45 +/-7yrs) affected by type1 diabetes under H2/CH4 lactulose breath test to evaluate the presence of SIBO and CH4 production (CH4 concentration at 3 ppm over that of recovery) was evaluated trough glycemic hemoglobin and daily insulin requirement (ratio between total insulin units in a day and body weight). CH4 producers were treated with metromidazole (500 mg bid for 10 days) and underwent a control breath test 6 weeks after the end of therapy.

RESULTS: Data were analyzed using paired-data t-test.

RESULTS: 12/30 patients (40%) were methane-producers (mean baseline value 6 +/-2 ppm; mean peak 25 +/-3ppm); the mean glycemic control was 7.6% and the daily insulin requirements (0.12 UI/kg) in 12 patients (75%) showed a significant (P<0.001) reduction of their glycemic control (mean HbA1c 7.6% vs 6.8%) and daily insulin requirements (0.68+/-.012 vs 0.49+/-.008 UI/kg) after metronidazole therapy. CONCLUSION: Our study showed for the first time a possible role of CH4 production in diabetes metabolic control. In particular, the most interesting data is that poorly controlled diabetes seems to be related to a gut CH4 production as confirmed by its significant improvement after eradication therapy.

Contact E-Mail Address: valecesario@yahoo.it
Disclosure of Interest: None Declared

Keywords: diabetes mellitus type 1, glycemic control, methanogenic flora

OP251 MEDIUM-CHAIN TRIGLYCERIDE INDUCED LEUKOCYTE ACTIVATION IS NOT MEDIATED BY TOLL-LIKE RECEPTOR 4

E. D. Olofth1,*, A. F. Guech1, L. A. Joosten2, H. M. Schaap - Roelofs1, G. J. Wantien1, 1Department of Gastroenterology and Hepatology, 2Department of General Internal Medicine, RADBoud UNIVERSITY Nijmegen Medical Centre, Nijmegen, Netherlands

INTRODUCTION: Lipids, as part of parental nutrition formulations modulate the function of the immune system. For instance, medium-chain triglycerides (MCTs), but not long-chain triglycerides (LCT), as part of parenteral lipid emulsions activate leukocytes in vitro by mechanisms that are still unknown. It has been shown that saturated fatty acids can activate Toll-like receptor 4 (TLR-4) mediated pro-inflammatory signaling pathways in leukocytes.

AIMS&METHODS: Aim of our study was to investigate whether TLR-4 is also involved in MCT-induced leukocyte activation. We assessed the in vitro effect of the parental mixed lipid emulsion LCT/MCT, at a clinically relevant triglyceride concentration of 5 mmol/l, on the expression of leukocyte surface markers, leukocyte activation markers in the presence or absence of the specific TLR-4 inhibitors TAK-242 (0.5 and 5 µmol/l) and Bartonella quintana LPS (0.1, 1 and 2.5 µg/ml).

RESULTS: As expected, LCT MCT activated leukocytes, with an increase in expression of adhesion (55% and 41% in granulocytes and monocytes, respectively), azurophilic and specific degradation (19% and 22%, respectively in granulocytes) markers, and a decrease in L-selectin (14% and 20% in granulocytes and monocytes, respectively). Inhibition of TLR-4 by TAK-242 and Bartonella quintana LPS did not alter the LCT/MCT-induced decrease in L-selectin and increase in adhesion marker expression in granulocytes and monocytes. Furthermore, in granulocytes Bartonella quintana LPS did not change the MCT-induced increased expression of specific and azurophilic degradation markers whereas LPS did. Both TLR-4 markers in granulocytes was abolished during TLR-4 inhibition with 5 µmol/l TAK-242. However, a similar decrease in degradation marker expression was found after incubation with 5 µmol/l TAK-242 alone.

CONCLUSION: MCT-induced immune activation is not mediated by TLR-4 signaling.

Contact E-Mail Address: e.olofth@mlld.umcn.nl
Disclosure of Interest: None Declared

Keywords: immune activation, medium chain triglyceride, parental nutrition, toll like receptor-4

TUESDAY, OCTOBER 15, 2013
11:00–12:30
Pancreatitis: Lessons from animal models – Salon 11/12

OP252 IMPROVEMENT OF ENDOPLASMIC RETICULUM STRESS BY ENHANCED PERK PATHWAY REDUCES MURINE EXPERIMENTAL ACUTE PANCREATITIS

T. Okazaki1, A. Nishio1, T. Masahiro1, T. Inoue2, Y. Sakauchi1, T. Fukui2, K. Uchida1, K. Okazaki1. 1Gastroenterology and Hepatology, Kansai Medical University, 2Moriguchi, Gastroenterology and Hepatology, Kansai Medical University, Hirakata, Japan

INTRODUCTION: Endoplasmic reticulum (ER) stress causes the accumulation of misfolded proteins inside the ER and initiates unfolded protein response (UPR). UPR is activated during pancreatitis to restore ER homeostasis. Although protein kinase RNA-like ER kinase (PERK) is associated with the UPR through phosphorylation of eukaryotic initiation factor 2a (eIF2a), the role of PERK signaling pathway in pancreatitis is not fully clarified. We investigated the significance of PERK signaling pathway in severe acute pancreatitis in mice using an eIF2a dephosphorylation inhibitor, salubrinal.

AIMS&METHODS: Severe acute pancreatitis was induced by intraperitoneal injection of cerulein (CER) at a dose of 50 mg/kg six times at 1 hour intervals. Moreover, LPS was administered at a dose of 10mg/kg as the septic challenge immediately after the completion of CER injections. Salubrinal was administered intraperitoneally immediately after LPS injection and six hours later. Mice were sacrificed at 24 hours after the first injection of CER and the severity of pancreatitis was histologically graded with a scoring system. Serum amylase and pancreatic cytochrome C (CytC) were measured. Expression of ER stress-related proteins was examined by western blotting.

RESULTS: The severity of pancreatitis in mice treated with salubrinal was significantly attenuated compared with control mice. Serum amylase and proinflammatory cytokine levels were significantly reduced. Expression of CytC was suppressed.

CONCLUSION: Inhibition of eIF2a dephosphorylation decreased ER stress and reduced severe acute pancreatitis in mice. Augmentation of PERK signaling pathway could be a potential therapeutic option for the treatment of acute pancreatitis.

REFERENCES:

Contact E-Mail Address: okazaki-t@aki.km.ac.jp
Disclosure of Interest: None Declared

Keywords: ER stress, pancreatitis, PERK signaling, Salubrinal

OP253 SEROTONIN REGULATES PROGENITOR CELL-BASED BUT NOT CLONAL REGENERATION IN THE ADULT PANCREATIC ACINAR CELL

E. Saponara1,*, S. Sonda1, K. Grablauksaitė1, Y. Tian1, T. Reding1, R. Graf1. 1Department of General Internal Medicine, Radboud University Medical Centre, Nijmegen, Netherlands

INTRODUCTION: Progenitor cell-based regeneration of acinar cells is activated during cerulein-induced pancreatitis. This process requires a prelimi

Towards the question of differentiation via secretion of zymogens, followed by expression of progenitor cell markers and formation of acinar-to-ductal metaplasia (ADM). Clonal regeneration without loss of zymogens and cell de-differentiation is observed following 60% pancreatectomy. Previously, we demonstrated that