



13th Euro-Global Gastroenterology Conference

August 20-21, 2018 | Rome, Italy

Keynote Forum

Day 1

Gastro Congress 2018

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David H Van Thiel

Rush University Medical Center, USA

Hepatitis C eradication: A promise unfulfilled

Hepatitis non-A and non-B hepatitis was recognized as a unique form of viral hepatitis distinct from hepatitis A, hepatitis B, and other unusual types of a chronic hepatitis such as CMV, EBV as well other more uncommon types of viral hepatitis in the late 1970s. His clinical characteristics, biochemical manifestations as well as its chronicity from its initial presentation followed by increasing stages of chronic hepatitis and hepatic fibrosis ultimately resulting in cirrhosis and occasionally progressing to hepatic cancer and required an additional 15-30 years. A host of potentially antiviral agents were utilized initially to treat the disease process with minimal or no success. With the introduction of interferons (alfa 2a or alfa 2b) with or without additional ribavirin, a modicum of success defined as a reduction in transaminase levels was achieved with little or no retrospectively determined viral clearance. With the isolation and characterization of the hepatitis C virus genome and the various polypeptides it codes for, a new era of treatment directed at inhibiting viral replication as opposed to enhancing the immune response against the virus began. The initial direct acting antiviral agents increased viral clearance rates to 40%. Agents more recently developed have increased the rates of viral clearance to 95 to 100%. This initiated reports (a promise) that hepatitis C would be eliminated as a disease process by 2020 with a progressive decline in the rates of cirrhosis and hepatocellular carcinoma thereafter through at least 2030. Unfortunately this does not appear to be the case as multiple obstacles prevent the favourable outcome. The issues and remaining and prohibit the promises full film and include the following: Lack of knowledge of primary care physician's that the disease is a serious hepatic disease that slowly and quietly progresses to cirrhosis and potentially hepatic cancer and is treatable. As a result large numbers estimated to be three quarters of the infected population failed to be identified. Secondly the cost of the drugs is prohibitive to those individuals with no insurance and contributes to the effort by third party peers and cover mental agencies to limit treatment to selected groups with advanced liver disease. As a result only a minor fraction of the infected population is identified for treatment and receives treatment. In addition, individuals with non-hepatic manifestations of hepatitis C are not recognized this having the disease process and are excluded from treatment despite the fact that this population represents the largest group of individuals perpetuating the disease in the community as they do not know they have the disease. In order with a promise of the elimination of hepatitis C and a reduction in long-term consequences of the infection universal defecation of infected patient's to include all forms of hepatic dysfunction as well as non-hepatic manifestations of the disease need to be recognized in treated. To accomplish this, the cost of treatment will have to be dramatically reduced and includes not only the cost of the therapeutic agent but also through numerous tests required to justify treatment. Some progress is being made by governmental agencies that are looking at the concept of micro-elimination as a potential means of reducing the prevalence of the disease in high prevalence groups such as men having sex with men, individuals enrolled in drug treatment programs, who said receive multiple transfusions as result of clotting disorders and/or hemolytic anemias. This is clearly a started but only if start.

Biography

David Van Thiel obtained his MD from the University of California at Los Angeles and completed his Internal Medicine residencies at Cornell University Hospitals and Boston University. He completed a Gastrointestinal/Hepatology fellowship at Boston University and the University of Pittsburgh. At the latter institution, he progressed from an Instructor of Medicine to Professor of Medicine and Director of the Gastroenterology & Hepatology Program and served as the medical Director of Liver transplantation. He has published more than 100 peer reviewed papers in a variety of journals and is on the Editorial Board of several journals as well as serves as a reviewer.

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Giovanni Gasbarrini

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Gut Microbiota: An epochal revolution

Few data exist on differences in gut microbiota composition among principal gastrointestinal diseases. We evaluated the differences in gut microbiota composition among uncomplicated diverticular disease (DD), IBS and IBD patients. DD, IBS and IBD patients along with healthy controls (CT) were enrolled in our Italian GI outpatient clinic. Stool samples were collected. Microbiota composition was evaluated through a metagenomic gene-targeted approach. GI pathology represented a continuous spectrum of diseases where IBD displayed one extreme while healthy controls displayed the other. Among Phyla, Biplot PC2/PC3 and dendrogram plot showed major differences in samples from IBS and IBD. DD resembled species CT composition, but not for *Bacteroides fragilis*. In IBS, *Dialister* spp and then *Faecalibacterium prausnitzii* were the most representative species. UC showed a reduced concentration of *Clostridium difficile* and an increase of *Bacteroides fragilis*. In CD, *Parabacteroides distasonis* was the most represented, while *Faecalibacterium prausnitzii* and *Bacteroides fragilis* were significantly reduced. Each disorder has its definite overall microbial signature, which produces a clear differentiation from the others. On the other side, shared alterations constitute the “core dysbiosis” of GI diseases. The assessment of these microbial markers represents a parameter that may complete the diagnostic assessment.

Biography

Giovanni Gasbarrini was President of the Italian Society of Alcology and Member of the Board of Directors of the Italian Society of Gastroenterology, of the Italian Society of Internal Medicine, from which he was awarded the title of Member of Honor. He is the founder and President of the “Club del Tenue” and of the “FONCRE” (Operative Cancer Rectus-Colon), and currently the corporate framework for digestive diseases (Club del Tenue, FONCRE, Hp Italian Section, Intestinal Motility). He was Vice-President of the International Association of Surgeons & Gastroenterologists. He was President and is now a Member of the European Helicobacter Study Group (of which the Italian section is also the President); he was President of EAGEN (European Associate Gastroenterol, Nutrition) and is currently past-President of EAGEN. He is a member of over 15 Italian and International Scientific Societies (Italian Society of Internal Medicine -Italian Geriatrics Society -Italian Gastroenterology Society - Italian Electronic Microscopy Society -Italian Society of Alcology - European Society Liver Study - French Society of Gastroenterology).

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Fong Fong Chu

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Reactive oxygen species generated by NADPH oxidase-1 and Dual oxidase-2 contribute to inflammatory bowel disease

Statement of the Problem: Gut microbes play an essential role in pathogenesis of inflammatory bowel disease (IBD). Host cells respond to microbe colonization by releasing cytokines and chemokines. Some inflammatory cytokines such as IL-4 and IL-13 induce NADPH oxidase-1 (NOX1) and dual oxidase-2 (DUOX2) gene expression in the epithelial cells. Elevated NOX1 or DUOX2 can produce reactive oxygen species (ROS) to regulate various cellular functions including cell proliferation, migration and apoptosis. NOX1 and DUOX2 have been linked to very-early-onset IBD, beginning before 6 years old. But the exact role of NOX1 and DUOX2 in IBD is not known.

Methodology: Mice deficient in antioxidant enzymes, glutathione peroxidase (GPx)-1 and -2, so called GPx1/2-DKO mice, develop ileocolitis around weaning. The hall-mark of pathology includes high crypt apoptosis, Paneth cell depletion, exfoliation and crypt abscess. Germ-free DKO mice are disease-free. To explore the role of Nox1 and Duox2 in gut inflammation, we studied the pathology and phenotype of Nox1-GPx1/2-triple KO (TKO) and Duox-GPx1/2-TKO mice at 35 days of age (comparable to human very-early-onset IBD).

Findings: Nox1-GPx1/2-TKO mice virtually do not have pathology. Duox-GPx1/2-TKO mice have intermediate pathology except crypt apoptosis remain as high as the DKO mice.

Conclusions & Significance: Both Nox1 and Duox2 contribute to inflammation, while Nox1 has a stronger impact than Duox2 probably because it is expressed in the crypt of the gland. Drugs that have been effective in treating IBD, such as dexamethasone and antibiotics, are likely mediated through suppression of NOX1 and DUOX2 gene expression.

Recent Publications

1. Chu F F et al. (2004) Bacteria-induced intestinal cancer in mice deficient in both Gpx1 and Gpx2 genes. *Cancer Res.* 64:962-968.
2. Hayes P et al. (2015) Defects in HADPH oxidase genes NOX1 and DUOX2 in very early onset inflammatory bowel disease. *Cell Mol. Gastroenterol. Hepatol.* 1(5):489-502.
3. Chu F F et al. (2017) Deficiency in Duox2 activity alleviates ileitis in GPx1- and GPx2-knockout mice without affecting apoptosis incidence in the crypt epithelium. *Redox Biology.* 11:144-156.
4. Liu H et al. (2017) Interleukin-4 and interleukin-13 increase NADPH oxidase 1-related proliferation of human colon cancer cells. *Oncotarget.* 8(24):38113-38135.

Biography

Fong Fong Chu has her expertise in gastrointestinal diseases especially in inflammatory bowel disease (IBD). Her team has established a mouse model of IBD which is very-early onset. These mice are deficient in two isoenzymes which reduce hydrogen peroxide named GPx1/2-double knockout (DKO). This model is a better model than chemical-induced colitis models because it is not injury based and mimic closely to human IBD. She has built this model through 20 years of research and has identified new targets for IBD therapy. She joined Beckman Research Institute of the City of Hope, Duarte CA USA (1987). She is currently associated with the Department of Gastroenterology & Hepatology and the First Affiliated Hospital and College of Clinical Medicine of Henan University of Science and Technology, Luoyang, Henan, P R China since 2016.

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Bashar Attar

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Subclinical exocrine pancreatic insufficiency (EPI): A disease that merits treatment

Introduction: Exocrine pancreatic insufficiency (EPI) is one of the long-term consequences of chronic pancreatitis (CP). Majority of patients with EPI are undiagnosed or undertreated.

Study Design: We prospectively evaluated 200 consecutive individuals seen in a pancreatic outpatient practice. These individuals were screened on 2 occasions to determine their baseline stool elastase excretion. The 200 individuals were segmented into 4 distinct groups: a) "Normals" (n=105) with stool elastase >500 ug/g stool, received no treatment; b) "Minimal EPI" (n=60) with stool elastase >200 to <500 ug/g stool, received 3000 IU of a standard pancreatic enzyme preparation (Creon) with their 2 ingested meals; c) "Moderate EPI" (n=23) with stool elastase >100 to 200 ug/g stool, received 12,000 IU of the same pancreatic enzyme preparation with each meal; d) "Severe/Overt EPI" (n=12) with stool elastase <100 ug /g stool, received 24,000 IU of the same pancreatic enzyme preparation with each meal and with a bedtime snack.

Results: These groups presented with abdominal pain, bloating, flatulence, diarrhea, large bulky stools, and greasy stools. Symptoms were graded (1-10) at entry and monthly for 3 months. Symptom scores decreased in all groups. The response to therapy was maximal in those with most severe disease identified by their greatest reduction in stool elastase at entry. Lesser responses were seen in the other groups and mirrored the severity of the disease at entry as defined by their stool elastase levels.

Conclusions: We conclude that 1) pancreatic elastase in stool enable the segmentation of individuals into distinct subgroups of EPI. 2) pancreatic elastase in stool enables identification of not only overt EPI but those with minimal and moderate EPI. 3) therapy with pancreatic enzyme preparations can be individualized based upon the concentration of pancreatic elastase in stools. 4) individuals with "subclinical" EPI with stool elastase level of 100-500 improve with treatment.

Recent Publications:

1. Wang Y and Attar B M (2017) Comment on Comparison of BISAP, Ranson, MCTSI, and APACHE II in predicting severity and prognoses of hyperlipidemic acute pancreatitis in Chinese patients. *Gastroenterol. Res. Pract.* 2017:1426486. Doi:10.1155/2017/1426486.
2. Wang Y et al. (2017) Evaluation of the prognostic value of neutrophil to lymphocyte ratio in patients with hypertriglyceridemia-induced acute pancreatitis. *Pancreatology.* 17(6):893-897. Doi:10.1016/j.pan.2017.10.001.
3. Wang Y et al. (2017) Concurrent diabetic ketoacidosis in hypertriglyceridemia-induced pancreatitis: how does it affect the clinical course and severity scores? *Pancreas.* 46(10):1336-1340. Doi:10.1097/MPA.0000000000000937.
4. Walter R J et al. (2012) Newcastle disease virus LaSota strain kills human pancreatic cancer tumor cells *in vitro* with high selectivity. *Journal of Pancreas.* 13(1):45-53.
5. Walter R J (2012) Two avirulent, lentogenic strains of Newcastle disease virus are cytotoxic for some human pancreatic tumor lines *in vitro*. *Journal of Pancreas.* 13(5):502-513. Doi: 10.6092/1590-8577/977.

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Biography

Bashar M Attar is a Professor of Medicine and Surgery at Rush University Medical Center in Chicago, Illinois, USA. He is also the system-wide Chairman of Gastroenterology and Hepatology at Cook County Health and Hospitals System. He has special interest acute and chronic pancreatitis as well as potential mechanisms contributing to pancreatic cancer. He has an avid interest in viral hepatitis, metabolic and cholestatic liver disorders including bile transport. He is the Recipient of the President Diversity Award by the ASGE (2010); Recipient of the prestigious National "Parker J Palmer Courage to Teach Award" by the ACGME (2015) which was granted in recognition of extraordinary accomplishment in Graduate Medical Education. He has been recently elected (2017) to the Humanism Honor Society in recognition of exemplary service, integrity, clinical excellence and compassion.

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Davor Štimac

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Endoscopic treatment of obesity: Challenge for gastroenterologists

According to emerging role of endoscopic procedures in the treatment of obesity and rapid changes in endoscopic technologies and techniques, the current state of endoscopic management of obesity will be presented. Endoluminal interventions performed entirely through the GI tract by using flexible endoscopy offer the potential for an ambulatory weight loss procedure that may be safer and more cost-effective compared with current surgical approaches. Endoscopic techniques attempt to mimic the anatomic features of bariatric surgery. Accordingly, there are two main endoscopic weight loss modalities - restrictive and malabsorptive. Restrictive procedures act to decrease gastric volume by space-occupying prosthesis and/or by suturing or stapling devices, while malabsorptive procedures tend to create malabsorption by preventing food contact with the duodenum and proximal jejunum. The former include intragastric balloon treatment, endoluminal vertical gastropasty, transoral gastropasty and transoral endoscopic restrictive implant system, while the latter include duodenojejunal bypass sleeve. Gastroduodenojejunal bypass sleeve is a combination of both procedures. Except for intragastric balloon, all mentioned procedures are rather new, tested on a small number of human subjects, with a high rate of success, but with limited knowledge on safety and long-term efficacy. The role of gastric electrical stimulation and intragastric injections of botulinum toxin in obesity treatment is also considered as is the role of minimally invasive bariatric endoscopic interventions.

Biography

Davor Štimac is a Professor in the university of rijeka. He is Director of Clinical Hospital Center Rijeka from 2016. Head of the Clinic for Internal Medicine of the Clinical Hospital Center Rijeka from the year 2014. Deputy Head of the Department of Internal Medicine of the Faculty of Medicine of the University of Rijeka from the year 2001. He is member in the following societies, Croatian Medical Association- member of the Executive Board, Croatian Gastroenterological Society - Member of the Steering Committee, Croatian Pancreatic Club –President, Croatian Society for Thickness - the President, Croatian Society for Health Care Quality Improvement-Vice President, World Gastroenterology Organization (WGO)- a member of the Global Guidelines and Publication Board and a member of the Trainers' Trainers (TTT), United European Gastroenterology Federation (UEGF)- a member of the General Assembly, European Association of Gastroenterology, Endoscopy and Nutrition (EAGEN)- Member of the Board of Directors, European Board for Gastroenterology and Hepatology (EBGH)- Member of the Management Board, UEMS Section for Gastroenterology- HLZ representative. He had published about 121articles. Editor for 7 books and had 21 chapters in the books, 71 work in other indices.

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