972nd Conference





11th Global Gastroenterologists Meeting

June 12-13, 2017 Rome, Italy

Keynote Forum Day 1

Gastro 2017



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Francesco Marotta

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Hormetic microbiota effect on the gut-based mechanism of metformin benefit

etformin is commonly used as the first line of medication for the treatment of metabolic syndromes, such as obesity and type 2 diabetes (T2D). Recently, this compound has gained an increasing interest within the scenario of pro-longevity medicine given also its peculiar increase in AMP-activated protein kinase (AMPK) thus beneficially affecting energy balance by maintaining a proper cellular AMP/ATP ratio through the increase of ATP consumption and decreasing ATP production, which is associated with AMPK activation. The gut microbiota is known to play an important role in harvesting energy from food, metabolic processes and immune modulation. An increasingly body of evidences proves that its composition is significantly associated with obesity, T2D, metabolic syndromes and other chronic diseases. Data show that when metformin is administered to high fat diet (HFD) animals, the composition of the phylum Bacteroidetes significantly increases (over 75%) similar to that in the normal diet-(ND)-fed animals. Moreover, the composition of Verrucomicrobia in the HFDMet group significantly increases, unlike what obtained by simple dietary change applied to HFD. In the HFD-Met group, the abundances of the families Bacteroidaceae, Verrucomicrobiaceae, and some specific Clostridia change significantly vs. those in the HFD and HFD-ND groups. Interestingly, metformin treatment also affects the composition of the gut microbiota in mice fed a ND. The families Rikenellaceae, Ruminococcaceae, and Verrucomicrobiaceae, as well as Alistipes spp., Akkermansia spp., and Clostridium spp., are more abundant in the ND-Met group than the ND group. All this is shedding new light on the AMPK-independent pathway of metabolic improvement by metformin treatment through targeting the microbiota. It is likely that metformin via changes in Akkermansia and lactobacilli bacteria regulation improves the metabolic profile of dietinduced obesity by ameliorating low-grade tissue inflammation and also up-regulating the intestinal expression of several endocannabinoids controlling inflammation, barrier function and peptide secretion in the gut. Till recently, a main hindrance in bacterial stool culture is represented by the major bias that only a few gut bacteria can be properly detected and cultivated in the laboratory. On the other hand, capillary sequencing or PCR-based approaches need culture medium with its inner complexities of multiple separate analyses. In this scenario, a rising star is represented by the next-generation sequencing (NGS) which by combining multiple samples in a sequencing run, is able to analyse the entire microbial community within a sample. Thus, this unique ability enables to catalog resident organisms within the very complex gut poly-microbial bacterial communities to make a DNA-stable sampling and producing a report intelligible to physicians and ideally endowed with a nutritionist and a gastroenterologist commented interplay so to make it a valid diagnostic, a treatment-guided result and a follow up tool as well. This has been quite recently achieved by a spin off dedicated geneticists and biologists (Next Genomics, Prato, Italy) using a kit allowing small and 14-day stable sampling of 0,0001% accuracy. This test is currently used also in clinical center (MMC Milano, Milan, Italy) identifying the enterotype and the presence of bacterial species correlated with diseases.

Biography

Francesco Marotta completed his MD and PhD with experience in Gastroenterology, Oxidative Stress, Aging and Nutrigenomics in USA, Cape-Town and Japan leading to extensive publications. He is a Research Professor in Department of Nutrition, Texas Women University, USA. He is in Advisory Board Panel of the Center for Life Science at Nazarbayev University, Kazakhstan and he is an External Examiner at McGill University, Canada and Osaka University, Japan. Currently, he serves as CMO at Milano Medical Center for Healthy Aging and also at a prime international clinical set up in central-Asia.

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Larry I Good

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Serum derived bovine immunoglobulin in the treatment of gastrointestinal disease

Serum derived bovine immunoglobulin was introduced as a medical food in the U.S. in 2013. Since then it has been studied in over 800 patients with a variety of gastrointestinal illnesses including diarrhea predominant irritable bowel syndrome (IBS-D), ulcerative colitis, Crohn's disease, pouchitis, *C. difficile* colitis and chronic mesenteric ischemia. Its effectiveness in these diverse disease entities is related to the ability of SBI to bind intraluminal pro-inflammatory mediators including enterotoxins, pro-inflammatory cytokines and bacterial degradation products, thereby, preventing the loss of intercellular tight junction proteins. By maintaining tight junction, integrity, dendritic antigenic stimulation in the mucosa is reduced, resulting in inhibition of the inflammatory cascade. This presentation will review the US FDA category of medical foods, the proposed mechanism of action SBI and published clinical data.

Biography

Larry I Good has been a practicing Gastroenterologist since 1978. He completed his Graduation at Colgate University in 1969; MD at Medical University of South Carolina in Charleston, SC in 1973 and; Medical Residency from 1973-76. He was Chief Medical Resident in 1976. He completed his fellowship trained in Gastroenterology from 1976-78. He served as the Director of Liver Diseases at Nassau County Medical Center and was for many years Chief in Division of Gastroenterology, Department of Medicine at South Nassau Communities Hospital. He is an Assistant Clinical Professor of Medicine at SUNY Stony Brook. He has given hundreds of lectures in his field and has authored numerous papers and abstracts. Recently, he presented the ACG Theater lecture at American College of Gastroenterology annual meeting in Chicago, Illinois in October, 2014. He was Chief Medical Officer at Ritter Pharmaceuticals in Los Angeles, California, where he expanded his research interest in the Intestinal Microbiome. His current clinical research activities involve the microbiome, inflammatory bowel disease, irritable bowel syndrome and the application of orally administered gamma globulin to patients with acute and chronic gastro-intestinal disorders.

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Notes:

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David H Van Thiel

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Subsequent clinical exocrine pancreatic insufficiency, a disease requiring recognition and therapy

Introduction: The criteria utilize for the identification of the diagnoses of exocrine pancreatic insufficiency (EPI) was established in the late 1950s and early 1960s. The possible criteria was the finding of high-grade steatorrhea equal to or greater than 20 g of fecal fat per day collected over a 72 hour. While the patient was ingesting a diet with a minimum of 100 g of fat daily. It was reported that this criterion was met when more than 90% of the function of the exocrine pancreas was lost. This requirement for the diagnoses of exocrine pancreatic insufficiency is difficult to achieve because the test is cumbersome and poorly accepted by patients, physicians and laboratory personnel. A multitude of alternative tests have been developed, but none of them has achieved universal acceptance and/or availability. The detection and quantification of exocrine pancreatic enzymes in stool has been evaluated for this purpose, but all of the enzymes with the exception of pancreatic elastase are destroyed during the transit through the intestine, making them unacceptable as an diagnostic tool. Pancreatic elastase measured in stool is greater than 200 ng/g stool and normal individuals. Those with less than 200 mg greater than 100 ng per male have subclinical pancreatic insufficiency. Those with less than 100 ng per male have overt pancreatic insufficiency as defined by the original definition for the detection of pancreatic insufficiency.

Aim: Identify an individuals with subclinical exocrine pancreatic insufficiency having stool he less based determination was less than 201 greater than 100 and evaluate the response to treatment with pancreatic enzyme supplementation.

Methods: #1 Consecutive patients seen in outpatient gastroenterology clinic were evaluated. #2 Stool elastase was measured in all enabling the segregation of the subjects into 3 distinct groups consisting of normal individuals(elastase >200, those with minimal to moderate pancreatic insufficiency less than 200 but greater than 100 ng elastase/gram of stool and those with overt pancreatic insufficiency having less than 100 ng/g pancreatic elastase in stool. #3 symptoms of abdominal pain, cramps, bloating, gas, flatulence, diarrhea, large, bulky stools, foul smelling stools, I requirement for double flushing, were each evaluated on a 0-10 scale@each clinic visit over a 6 month period. #4 6 laboratory parameters consisting of hemoglobin, BUN, creatinine, albumin, levels of vitamin A, vitamin D, and vitamin E were recorded and each clinic visit.

Results: Symptoms in most patients and groups to ON 3 improved with time and continued utilization of pancreatic enzyme replacement. The response occurred earlier in those with mild to moderate pancreatic insufficiency as compared to those with overt pancreatic insufficiency and no improvement was noted in those with normal levels of pancreatic elastase in stool.

Conclusions: #1 individuals with unexplained abdominal symptoms should be evaluated utilizing a stool elastase determination of the presence of pancreatic insufficiency. #2 individuals with pancreatic elastase in stool less than the lower limit of normal, but greater than 100 ng per gram stool have pancreatic disease that responds to therapy achieving a symptom response pattern, similar to that of normal individuals having a normal stool elastase determination greater than 200 ng/g stool. Those and groups 3 with overt pancreatic insufficiency response lower to a degree less than that observed by those in group to with subclinical pancreatic insufficiency.

Summary: Subclinical exocrine pancreatic insufficiency response to therapy consisting of esophagitis pancreatic enzyme supplementation and represents the clinical disease prosess that can and should be treated

Biography

David Van Thiel is a Gastroenterologist in Berwyn, Illinois. He is affiliated with multiple hospitals in the area, including Rush Oak Park Hospital and Rush University Medical Center. He completed his Medical degree from David Geffen School of Medicine at UCLA and has been in practice for 39 years. He is one of the 21 doctors at Rush Oak Park Hospital and one of 25 doctors at Rush University Medical Center who has specialization in Gastroenterology. He completed his Graduation from University of California at Los Angeles. He has obtained board certification from the member board for Internal Medicine and Hepatology.

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