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July 23-24, 2018 | Rome, Italy

DAY 1

Scientific Tracks & Abstracts

Day 1

SESSIONS

July 23, 2018

Geriatric Cardiology | Cardiomyopathy and Heart Failure | General Microbiology | Sports Cardiology | Oral Microbiology |Cardiac Surgery | Cardic Nursing | Women Heart Health | Oral Microbiology

Session Introduction

Session Chair
Peter P Karpawich
Wayne State University
School of Medicine, USA

Title: **Effect of xanthine oxidase inhibitors on cardiovascular outcomes: Trying to make sense of the contradictory evidence**
Markus Bredemeier, Hospital Nossa Senhora da Conceição, Brazil

Title: **Shah vs. Backman vs. Abbot's cut off for hsTnI showed different possibilities in patients with chest pain profile?**
Marta Noemi Monari, Humanitas Clinical and Research Center, Italy

Title: **Electromagnetic properties of the arterial blood flow**
Merab Beraia, Tbilisi State Medical University, Georgia

Title: **HcpE and DsbK, novel contributors to inflammation caused by human gastric pathogen *Helicobacter pylori***
Carole Creuzenet, The University of Western Ontario, Canada

Title: **Possible strategies to overcome drug resistance in biofilm**
Claudia Vuotto, Microbial Biofilm Laboratory, Italy

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Markus Bredemeier, Biomed Res 2018, Volume 29 | DOI: 10.4066/biomedicalresearch-C1-002

EFFECT OF XANTHINE OXIDASE INHIBITORS ON CARDIOVASCULAR OUTCOMES: TRYING TO MAKE SENSE OF THE CONTRADICTORY EVIDENCE

Markus Bredemeier
Hospital Nossa Senhora da Conceição, Brazil

Xanthine oxidase inhibitors (XOI), classified as purine-like (allopurinol and oxypurinol) and non-purine (febuxostat and topiroxostat) XOI, present antioxidant properties by reducing the production of reactive oxygen species derived from purine metabolism. Oxidative stress is an important factor related to endothelial dysfunction and ischemia-reperfusion injury and may be implicated in the pathogenesis of heart failure, hypertension and ischemic heart disease. However, there is contradictory evidence regarding the possible cardiovascular (CV) protective effect exerted by XOI, especially regarding the results of randomized controlled trials. In the present lecture, we discuss in deep the reasons for the disparity in results of different studies from a pharmacodynamic and clinico-epidemiologic point of view, considering the emergence of recent evidence in this field.

BIOGRAPHY

Markus Bredemeier is graduated from Medical School at Federal University of Rio Grande do Sul (1994) and obtained master's (2003) and PhD (2006) in Medicine: Medical Sciences. Currently, he is practicing at Grupo Hospitalar Conceição (GHC). He worked as Professor of Clinical Pharmacology at the Faculty of Dentistry, Lutheran University of Brazil, from 1997 to 2005. He has 55 scientific articles published in medical journals, of which 42 are indexed in PubMed. He founded and supervises the residency program in Rheumatology of the GHC. In October 2013, he received the award Luiz Felipe Cunha Mattos (first place) for the work low- versus high-dose rituximab for rheumatoid arthritis: a systematic review and meta-analysis. The updated version of this study was presented orally at the EULAR 2015 Congress. In 2017, presented orally the study effect of xanthine oxidase inhibitors on the incidence of cardiovascular events: a systematic review and meta-analysis of randomized controlled trials at the EULAR Congress Madrid.

markbred@terra.com.br

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Marta Noemi Monari et al., Biomed Res 2018, Volume 29 | DOI: 10.4066/biomedicalresearch-C1-002

SHAH VS. BACKMAN VS. ABBOT'S CUT OFF FOR HSTNL SHOWED DIFFERENT POSSIBILITIES IN PATIENTS WITH CHEST PAIN PROFILE?

Marta Noemi Monari[1], L Motta[2], A Molteni[2,3], A Voza[4], P Bianchi[1], F Maura[1], Barbieri B[1], M Pedretti[1] and M F Rossi[1]

[1]Humanitas Clinical and Research Center, Italy
[2]IRCCS Ca' Granda Ospedale Maggiore Policlinico, Italy
[3]ASST Lecco, Italy
[4]Humanitas University, Italy

Background: Chest pain is a common cause of worldwide hospital admission and is a major burden on health-care resources. Cardiac troponin has substantially improved the accuracy of diagnosis and prognostic assessment of patients with suspected acute coronary syndrome (ACS). We wanted to investigate the use of a high sensitive assays for cardiac troponin (hsTnl) in the emergency department and its influence in patients admission or discharge (according to assigned color code and pain during triage), in order to identify the best one in terms of accuracy between the gap from the cut off and the need of hospitalization.

Methods: We have conducted a retrospective analysis based on 1758 (three month) accessions in emergency department (ED). We have focused our attention between 1014 patients (534 men, 480 women) having cardiological profile, excluding thoracic trauma or other non cardiological pain. We compared three different possible scenarios to ruled in chest pain patients to intepretate the hsTnl: from literature Shah 12 ng/L, for Abbott hsTnl 34 ng/L for men, 15 ng/L for woman; and Beckman Coulter hsTnl 19.8 ng/L for men and 11.6 ng/L for woman.

Results: The need of hospitalization was associated with a value above the cut-off of each method taken into in a statistically significant way (Abbott, $p<0.001$; Beckman, $p<0.001$; Shah, $p<0.001$). Moreover, the gap from the cut-off is associated with an increased probability of admission, corrected for age, gender and color code (Abbott OR 7.74, 95% CI 2.89-20.75, $p<0.001$; Backman 3.93, 95% CI 1.89-8.18, $p<0.001$; Shah 5.06, 95% CI 2.51-10.22, $p<0.001$). The hospitalization is highly associated with the color code ($p<0.001$) given during the triage.

Conclusion: In this population, there is not a statistically significant difference between the three different interpretative cut off taken into consideration in identifying hospitalized patients. There is a statistically significant association between the color code given during the triage, the hsTnl level and the hospitalization, so the real key of the use of this marker is strongly related to the correct diagnosis.

BIOGRAPHY

Marta Noemi Monari is working in Humanitas clinical and research center, Italy

marta_noemi.monari@humanitas.it

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Merab Beraia et al., Biomed Res 2018, Volume 29 | DOI: 10.4066/biomedicalresearch-C1-002

ELECTROMAGNETIC PROPERTIES OF THE ARTERIAL BLOOD FLOW

Merab Beraia[1] and **Beraia G**[2]
[1]Institute of Clinical Medicine, Georgia
[2]Tbilisi State Medical University, Georgia

Introduction: Blood flow acceleration increases from the left ventricular outflow tract, to the sinotubular junction and the ascending aorta, while it must be decreasing due to the flow turbulences in the Valsalva sinuses and increased diameter of the vessel. Total energy of the pulse wave in the arterioles is up to 7.2 times higher, than in the ascending aorta, while it must be low due to the energy dissipation in the viscous flow, with the distance from the heart. Work made by the left ventricle, at least 2.0-2.5 times lower to the work needed for the blood displacement, in the systemic capillaries.

Purpose: The purpose of the study is identifying the additional possible energy source, for the arterial blood flow.

Methods & Materials: 12 healthy volunteer students (male) underwent echocardiography, ECG gated MRI of the heart for the visualization intracavitary flow in the ventricles, MR angiography of the aorta. Blood flow velocities and acceleration were studied in the different sites of the heart and the aorta.

Results: With the DU in the left ventricular outflow tract blood acceleration is 1430±120 cm/sec^2, in the sinotubular junction and ascending aorta 2395±195 cm/sec^2, at the aortic arch 1390±225 cm/sec^2, isthmus of aorta 2180±135 cm/sec^2, middle thoracic aorta 1260±140m/sec^2. With the MRI (TrueFisp. mean curve), blood acceleration from the left ventricular outflow tract to the sinotubular junction is 3.5±0.3 times higher and to the ascending aorta 2.5±0.2 times higher. Systolic blood pressure from the ascending aorta to the femoral and saphenous elastic arteries enhancing 1.3±0.1 times, increasing energy transmitted to the blood. Direction of the electric charge in the heart's ventricles from the circulating erythrocytes and in the fibres of the Purkinje (ECG), mathematically are coincident.

Conclusion: Availability of the heart, as the possible single tool for the blood flow, looks imperfect. Electric oscillate field from the heart dipoles can be impact to the blood charged particles. Erythrocyte forms the modulated naturally ultrasound vibration and associated with it colloid vibration current propagating distally to the all cell membranes. Blood motion in the heart chambers and arteries has the additional basis, besides the heart contraction: rotating blood particles in the heart chambers and in the arterial branching sites or the high resistive areas, with the concomitant oscillating electric field triggered from the heart, creates to the additional electromagnetic repulsing force, providing to the flow. Modulating ac electric field, transmitting by the oscillate blood particles, besides the flow, creates additional energy/signal source, enabling the spontaneous chemical reactions proceed across the cell membranes. Electromagnetism can be affect gas exchange in the systemic and pulmonary capillaries due to the different affinity of the oxygen and carbon dioxide in the diamagnetic/paramagnetic haemoglobin.

BIOGRAPHY

Merab Beraia has been graduated from Tbilisi State Medical University in 1986, as a Medical Doctor, with the specialty of Internal Medicine and took a Diploma in Neurology from the Institute of Clinical and Experimental Neurology Tblisi, Georgia. Later he obtained his post-graduation diploma in Radiology from University of Graz, Austria and then started working at The Institute of Clinical Medicine Tbilisi, Georgia, where he has continued his research. Presently he is working at the Tbilisi.

mberaia@hotmail.com

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HCPE AND DSBK, NOVEL CONTRIBUTORS TO INFLAMMATION CAUSED BY HUMAN GASTRIC PATHOGEN *HELICOBACTER PYLORI*

Carole Creuzenet
The University of Western Ontario, Canada

H. pylori causes gastritis, gastric ulcers and cancers but the mechanisms of virulence are not fully understood. It produces secreted proteins which may play a role in eliciting gastric inflammation, including the Helicobacter cysteine rich protein HcpE (HP0235) whose biological function is unknown. Our goal was to investigate if HcpE is secreted by *H. pylori* and is involved in host/pathogen interactions and identify components essential for its production. Using a combination of anti-HcpE ELISA and Western blots, knockout mutagenesis, phenotypic analyses and biochemical assays, we demonstrate that HcpE is secreted by many strains as a soluble protein and in association with outer membrane vesicles. We show that infected patients produce anti-HcpE antibodies, indicating *in situ* HcpE production. We show that HcpE comprises many disulfide bonds and identify DsbK (HP0231) as a folding factor necessary for HcpE production, and show that recombinant DsbK can refold unprocessed, reduced HcpE *in vitro*. This highlights the first biologically relevant substrate for DsbK. Furthermore, we show that DsbK has DiSulfide Bond (Dsb) forming activity and has DsbA-like activity despite its similarity with DsbG. Also, we show a role of DsbK in redox homeostasis in *H. pylori*. Finally, we show an important role for DsbK and HcpE in host-pathogen interactions, including murine gastric colonization and pro-inflammatory cytokine production in human gastric explants, gastric cell lines and in murine splenocytes. Both proteins will be investigated as therapeutic targets to treat *H. pylori* infections and prevent gastric ulcers and cancers.

BIOGRAPHY

Carole Creuzenet has completed her PhD in Biochemistry at the University of Nantes and the National Institute for Agronomical Research (France) and her postdoctoral studies at the Massachusetts Institute of Technology (USA) and the University of Guelph (Canada). She is Associate Professor at the University of Western Ontario (London, Canada), where her lab focuses on virulence factors from bacterial gastrointestinal pathogens such as *Campylobacter jejuni, Helicobacter pylori and Yersinia pseudotuberculosis*. Her focus is on glycolipids and glycoproteins as well as on novel secreted proteins and their folding partners. She has published 38 papers in reputed journals with h-index of 19.

ccreuzen@uwo.ca

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POSSIBLE STRATEGIES TO OVERCOME DRUG RESISTANCE IN BIOFILM

Claudia Vuotto and **Gianfranco Donelli**

Fondazione Santa Lucia IRCCS, Microbial Biofilm Laboratory, Italy

Biofilm-growing nosocomial pathogens represent a serious public health concern. In fact, the biofilm mode of growth is highly relevant in clinical settings where these microbial communities, displaying higher levels of antimicrobial resistance, cause several difficult-to-treat chronic infections. The reduced effectiveness of many antibiotics against bacteria grown as biofilm is due to the presence of the exopolysaccharide matrix, the reduced growth rate of microbial cells and the significant increase in the level of horizontal gene transfer. The relapsing nature of biofilm-related infections makes increasingly necessary to discover new antimicrobial agents able to interfere with biofilm formation and maturation, without inducing antibiotic resistance. This presentation will focus on biofilm resistance in aerobic and anaerobic species, underlining their response to antibiotics in terms of matrix production and induction of a viable but non-culturable state. Refractory medical devices and not-inducing resistance, antibacterial compounds will be presented as possible anti-biofilm strategies.

BIOGRAPHY

Claudia Vuotto is currently working in the Microbial Bioflim Laboratory and she is also an associate with Fondazione Santa Lucia IRCCS.

c.vuotto@hsantalucia.it

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DAY 2

Scientific Tracks & Abstracts

Day 2

SESSIONS

July 24, 2018

Geriatric Cardiology | Cardiomyopathy and Heart Failure | General Microbiology | Sports Cardiology | Oral Microbiology |Cardiac Surgery | Cardic Nursing | Women Heart Health | Oral Microbiology

Session Introduction

Session Chair
Ebtesam Al-Ali
Environmental and Life Sciences Research Centre, Kuwait

Title: **Epigenetic regulation of F508del-CFTR cystic fibrosis lung disease**
Roopa Biswas, Uniformed Services University of the Health Sciences, Bethesda, USA

Title: **CABG in diffuse coronary artery disease**
Shyam K Ashok, Aster CMI Hospital, India

Title: **ROTEM and human fibrinogen concentrate use in pediatric cardiac surgery**
Christopher F Tirotta, Nicklaus Children's Hospital, USA

Title: **Estimate pulmonary arterial hypertension by heart sound**
Hamza Cherif Lotfi, University of Abou Bekr Belkaid, Algerian

Title: **Global threats of antibiotic resistance**
Reza Nassiri, Michigan State University, USA

Title: **Epidemic flu**
Guilio Tarro, Foundation de Beaumont Bonelli for Cancer Research, Italy

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EPIGENETIC REGULATION OF F508DEL-*CFTR* CYSTIC FIBROSIS LUNG DISEASE

Roopa Biswas[1], Parameet Kumar[1], Raymond A Frizzell[2] and Harvey B Pollard[1]

[1]Uniformed Services University of the Health Sciences, USA
[2]University of Pittsburgh, USA

Cystic fibrosis (CF) is the most common life limiting recessive disease in the US and is due to mutations in the *CFTR* gene. CF mutations, of which the most common is F508del-*CFTR*, cause a massive pro-inflammatory phenotype in the lung arising from dysregulated expression of inflammatory genes. Recently, endogenous non-coding RNA (ncRNA) molecules, including long non-coding RNAs (LncRNAs) have emerged as important targets in the frontier of biomedical research. These ncRNAs coordinate with epigenetic factors to play a crucial role in the regulation of biological processes as well as in diseases. Long noncoding RNAs (LncRNAs) have emerged as novel regulators of gene expression, including inflammatory genes. Various diseases have been associated with the aberrant expression of LncRNAs. Here we report the role of LncRNA and associated epigenetic factors in the pathogenesis of CF lung disease. LncRNA nuclear enriched abundant transcript 1 (*NEAT1*) is aberrantly upregulated in CF cells including, IB3-1, CFPAC-1 and CFBE CF cells as well as in lung tissues of CF patients compared to the respective control cells. *NEAT1* has been shown to regulate the expression of pro-inflammatory cytokine IL-8 in other diseases. Consistently, we find that suppression of *NEAT1* in CF lung epithelial cells leads to reduced expression of IL-8. Additionally, *NEAT1* is induced by p38-MAPK signaling pathway, which is activated in CF, and our results indicate that inhibition of this pathway suppresses both *NEAT1* as well as IL-8. Our data indicate that SFPQ, a *NEAT1* interacting protein, is down-regulated in F508del-*CFTR* CF lung epithelial cells compared to WT-*CFTR* control cells and perhaps also contributing to increased expression of IL-8. Consistently, we find that increased exogenous expression of SFPQ not only attenuates expression of the pro-inflammatory IL-8 gene, suppresses pro-fibrotic CTGF protein, but also rescues F508del- *CFTR* expression in CF lung epithelial cells. Understanding these mechanisms will lead to novel therapeutic targets for CF and related pulmonary diseases.

Chronic diseases and even aging itself are known to damage the body by dys-regulated inflammatory processes. Dysregulated expression of the pro-inflammatory cytokine and chemokine genes are known to contribute to chronic inflammatory diseases. Recently, endogenous non-coding RNA (ncRNA) molecules, including long non-coding RNAs (LncRNAs) and microRNAs (miRNAs, miRs) have emerged as important targets in the frontier of biomedical research. These non-coding RNAs have been proven to be key regulators of gene expression. The ability to detect non-coding RNAs in biofluids has highlighted their usefulness as non-invasive markers of diseases, including lung diseases. The expression of specific non-coding RNAs is altered in many lung diseases and their levels in the circulation often reflect the changes in expression of their lung-specific counterparts. Therefore, exploiting these biomolecules as diagnostic tools seems an obvious goal. Our goal is to investigate the role of non-coding RNAs in Cystic Fibrosis lung disease and develop novel anti-inflammatory therapeutics for pulmonary disorders.

BIOGRAPHY

Roopa Biswas is an Associate Profeesor in Anatomy, Physiology and Gentics Department in University of Health Science, USA.

roopa.biswas@usuhs.edu

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CABG IN DIFFUSE CORONARY ARTERY DISEASE

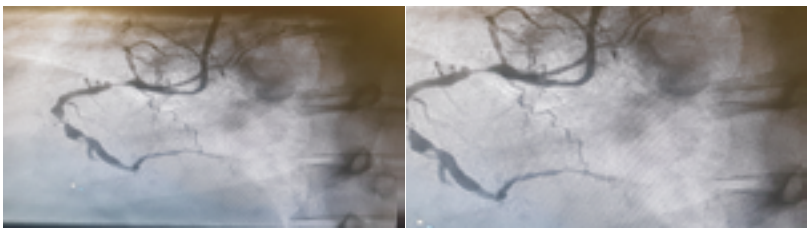
Shyam K Ashok
Aster CMI Hospital, India

Statement of the Problem: In India 2.78 million death are due to cardiovascular diseases of which 50% are due to CAD. Peculiarities of CAD patterns in Indian patients- Younger age at presentation, high incidence of DVD and TVD, diffuse involvement, distal disease and significant LV dysfunction at presentation. Diffuse CAD: Length of significant stenosis >20 mm, multiple significant stenosis (>70% narrowing) in the same artery separated by segment of apparently normal vessel and significant narrowing involving the whole length of coronary artery.

Methodology: We in our institute, perform OP CAB and use LIMA and veins as conduits to perform the surgery. Once the conduits are harvested, we heparinize with I.V. Heparin 3 mg/Kg given to achieve an ACT >300. Using the octopus as stabilizer, we perform an endarterectomy of the LAD first and then use a vein patch to cover the defect. LIMA is then used to anastomose the LAD on the vein patch. Veins are used to bypass the LCX and RCA, as deemed appropriate. The proximal ends of the vein grafts are anastomosed to Ascending Aorta with side clamp and heart beating. Intra op we start Lomodex infusion 20 ml/hr which is continued for 24 hours and the inotropes used are adrenaline and dobutamine as and when necessary. Postoperatively aspirin 75 mg is given, and heparin infusion started after six hours to maintain ACT of around 150 for 24 hours. Patients are usually extubated after four hours provided they are hemodynamically stable. Anticoagulation by acitrom is commenced orally from day one to maintain an INR of two for three months.

Result: Out of the 20 patients in last 18 months outcomes have been excellent with no in-hospital mortality or cerebrovascular incidents.

Conclusion: Off pump CABG with coronary end-arterectomy offers a good solution to the problem of diffuse coronary artery disease.



BIOGRAPHY

Shyam K Ashok after completing his MBBS and MS in General Surgery, he did his MCh in CVTS from Seth GS Medical College, Mumbai in 2008. He later joined Narayana Hrudayalaya, Bangalore in 2008, which is a 1000 bedded hospital executing close to 600 open heart surgeries in a month. He worked as a Fellow in Adult Cardiothoracic Department in Royal Melbourne Hospital, Australia, which is the second largest cardiothoracic unit in the whole of Australia. After working in Australia for two years he re-joined Narayana Hrudayalaya, as Consultant Cardiothoracic Surgeon in 2012, and worked there till 2015. He has independently performed about 1000 open heart surgeries, consisting of coronary artery bypass surgeries and valve replacements. His area of interest is coronary artery bypass, especially total arterial revascularization. He joined Aster CMI Hospital in Feb 2016, as Consultant Cardiothoracic Surgeon.

shyams2u@yahoo.co.uk

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Christopher F Tirotta, Biomed Res 2018, Volume 29 | DOI: 10.4066/biomedicalresearch-C1-002

ROTEM AND HUMAN FIBRINOGEN CONCENTRATE USE IN PEDIATRIC CARDIAC SURGERY

Christopher F Tirotta
Nicklaus Children's Hospital, USA

Human Fibrinogen Concentrate (HFC or RiaSTAP) is a purified fibrinogen concentrate derived from the plasma of healthy donors that undergoes virus inactivation and removal for safety purposes. HFC is indicated for the treatment of acute bleeding in patients with congenital fibrinogen deficiency (CFD), including afibrinogenemia and hypofibrinogenemia. Treatment with fibrinogen is also used for acquired fibrinogen deficiency caused by placental abruption, massive transfusion, liver failure, disseminated intravascular coagulation, and cardiac surgery. The ROTEM (Tem International GmbH, Munich, Germany) is an enhanced modification of thromboelastography (TEG), first described in 1948. Both are point-of care (POC) coagulation monitoring instruments that test the viscoelastic properties of whole blood. Use of the ROTEM has been shown to reduce the need and amount of transfused blood products in pediatric cardiac surgery patients. Tirotta et al. demonstrated that administering HFC at a dose of 70 mg/kg to neonates and infants undergoing cardiac surgery reduced the need for fresh frozen plasma (FFP) and cryoprecipitate. HFC can also be dosed depending on the actual and target fibrinogen levels using the formula:

Dose (mg/kg body weight) = target level (mg/dL) − measured level (mg/dL)

1.7 (mg/dL per mg/kg body weight)

Tirotta et al also demonstrated the Maximum Clot Firmness (MCF) of the ROTEM FIBTEM can be used as a surrogate of the fibrinogen level to dose the HFC via the formula: predicted fibrinogen=78.61+12.38 MCF. This formula suggests that a 1 mm of increase in MCF will correspond to a 12.38 increase in fibrinogen level. Using this formula and the POC ROTEM device, practitioners can tailor the transfusion therapy to reduce transfusion volume and donor exposure. CPB induced profound perturbations in ROTEM values. The administration of platelet pheresis (25 cc/kg) while on CPB improved the HEPTEM from 48 to 73 and the FIBTEM MCF from 4.8 mm to 8.3 mm; plasma fibrinogen levels increased from 105 mg/dL to 175 mg/dL. The administration of HFC (55 mg/kg) after termination of CPB improved the FIBTEM MCF from 7.9 mm to 10.3 mm and the plasma fibrinogen level from 175 mg/dL to 240 mg/dL.

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BIOGRAPHY

Christopher F Tirotta has been an active member of Miami Children's Hospital medical staff since 1991, practicing with the Department of Anesthesiology; he has served as the Director of Cardiac Anesthesia since 2002. He has served as Chief of the Department of Anesthesia since July 2017. He also has a clinical appointment with the Department of Anesthesiology at The University of Miami School of Medicine. He received his BA from Cornell University in 1982 and his MD from New York University School of Medicine in 1986. He also received an MBA degree from Columbia University in 1999. He completed his internship in Internal Medicine at SUNY at Stony Brook in 1987. He completed his residency training in Anesthesiology at the University of Miami/Jackson Memorial Hospital in 1990; he sub-specialized in pediatric and cardiovascular anesthesia, including heart transplantation.

christirotta@att.net

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ESTIMATE PULMONARY ARTERIAL HYPERTENSION BY HEART SOUND

Hamza Cherif Lotfi

University of Abou Bekr Belkaid, Algeria

Stenosis and mitral insufficiency may induce pulmonary arterial hypertension. The underlying mechanisms depend on the intrinsic characteristics of the pulmonary circulation and the acute or chronic character of left atrial hypertension. Since the main complication is right heart failure, treatment of left pathology (valve replacement) is aimed at reducing pulmonary pressures and decreasing the post-load of the right ventricle. Finally, various treatments, mainly medications, can be considered to reduce the effect of pulmonary hypertension and correct its symptoms. The results obtained show the clinical utility of our extraction methods for the recognition of heart sounds (or PCG signal), the estimation of pulmonary arterial hypertension. The results obtained also show that the severity of mitral stenosis involves severe pulmonary arterial hypertension.

BIOGRAPHY

Hamza Cherif Lotfi has received his PhD in Biomedical Electronics from the Faculty of Technology, University of Aboubekr Belkaid Tlemcen, Algeria in 2013. He is currently a Researcher in audible and ultrasonic processing physiological signals in the Genie-Biomedical Laboratory (GBM), Department of Genie-Biomedical, University of Tlemcen, Algeria. His current interests include phonocardiogram signal processing by applying the transform discrete wavelet transform and wavelet packet and spectro-temporal internal components of the first and second heart sound.

amzacherif@yahoo.fr

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THREATS OF ANTIBIOTIC RESISTANCE

Reza Nassiri
Michigan State University, USA

Global consumption of antibiotics has increased nearly 40% in the last decade. The incredible rapid resistance of antibiotic resistance which is taking place worldwide is not only a serious threat to the practice of modern medicine, but equally important, a threat to global public health. This urgent issue is so alarming that it caught the attention of G-20 Summit in both China (2016) and Germany (2017), let alone the U.N. Assembly in 2016 had called for a special meeting of "superbugs" which focused on the escalating drug resistance with respect to the sexually transmitted disease gonorrhea and carbapenem resistant *Enterobacteriaceae*. While the causes of antibiotic resistance are complex, certainly human behavior play a significant role in the spread of antibiotic resistant genes. In addition to the human behavior, the drivers of resistance include agriculture sector, animal husbandry, household and industry – these factors contribute significantly to the spread of the resistant genes within the ecosystem. Such resistant mechanisms are continuously emerging globally, which threatens our ability to treat common infections, resulting in increased death, disability and costs. Since the development and clinical use of penicillins, nearly 1000 resistant-related beta-lactamases that inactivate various types of antibiotics have been identified. The emergence of resistance to last-resort treatments known as extended-spectrum cephalosporins (ESCs) is now eminent. The five riskiest superbugs are recognized as (1) the original one: *Staphylococcus Aureus* (MRSA), (2) the hospital lurkers: Clostridium Difficile and Acinetobacter, (3) the food borne pathogens: *Escherichia Coli* and *Salmonella*, (4) The sexually-transmitted infections: Gonorrhea and Chlamydia, and (5) TB. India is a typical example of encountering the deadly bacterial resistance. The high prevalence of the *mrc-1* gene in *E. Coli* samples both in animals and raw meat, with the number of positive-testing samples are increasing each year in China. On average, more than 20 percent of bacteria in the animal samples and 15 percent of the raw meat samples carried the *mrc-1* gene. Numerous European countries have reported the existence of *mrc-1* gene in the isolates from human, isolates from animals used for food, isolates from food, and isolated from the environment. In conclusion, pathogens rapidly develop mutations that render current treatments ineffective – resistance to carbapenems, one of the 'last lines' of antibiotics, is widespread and has been observed in numerous countries. Therefore, there is an urgent need between research universities and industry aimed at developing novel antimicrobial agents to save the practice of modern medicine.

BIOGRAPHY

Reza Nassiri is a former Associate Dean of Global Health at the Michigan State University (MSU). He also served as MSU director of Institute of International Health. He is currently Professor of Pharmacology and Toxicology, Professor of Family and Community Medicine, and, lecturer in Global Health, Infectious Diseases and Tropical Medicine. He currently works on international public health issues relating to infectious diseases and has expertise in global health. He has made contributions in various fields of medical sciences including clinical investigation and health education. On the basis of his extensive experience and expertise in chronic infectious diseases including HIV/AIDS, TB, antimicrobial resistance and human gut microbiome, he has developed clinical research programs in Brazil, Haiti, Dominican Republic and Mexico. He had served as editorial board member for the journal of HIV and AIDS Review.

Reza.Nassiri@hc.msu.edu

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EPIDEMIC FLU VIRUS

Giulio Tarro
Foundation de Beaumont Bonelli for Cancer Research, Italy

For the emergency created by the epidemic of influence of the pigs in Mexico it was correct not to create alarmism's being victims of a bad information. The possibility that the virus arrives in other parts of the world is real as for all the types of influence virus. In order that a strain has a wide distribution, its antigenic characteristics must ensure that it escapes the neutralization of antibodies of the host and of the surrounding population. So, the outbreaks will happen with those strains that have dominant antigens that fit the deficiency, or better, the absences of antibody in the population. It seems, in conclusion that the flu virus shows an ability and an aptitude for survival built on the possibility of emergence of new models that allow the virus being confused easily through populations still partly immune to previous antigenic forms. According to this view, the changes in the influenza A can be designed in single meaning, in the context of a principle and of an evolutionary progress, from Burnet said immunological drift or steering immunology. The antiviral drugs (inhibitors of the neuraminidases, receptor of the virus surface) should be assumed within 48 hours by the appearance of the influence symptoms and for the subjects that have had a close contact with people infected by the flu virus. The vaccination against the influence is the most effective method to prevent the illness. From the moment that we find the isolation of a new flu virus, we must wait for the preparation of a new specific vaccine that will be ready for the next influence season in Autumn.

BIOGRAPHY

Giulio Tarro is graduated from Medicine School, Naples University (1962). He is the Research Associate, Division of Virology and Cancer Research, Children's Hospital (1965-1968), Assistant Professor of Research Pediatrics, College of Medicine (1968-1969), Cincinnati University, Ohio. He is also the Oncological Virology Professor, Naples University (1972-1985). Chief Division Virology (1973-2003), Head Department Diagnostic Laboratories, (2003-2006). Since 2007, he was the Chairman of Committee of Biotechnologies and VirusSphere, World Academy Biomedical Technologies, UNESCO, Adjunct Professor Department of Biology, Temple University, College of Science and Technology, Philadelphia, recipient of the Sbarro Health Research Organization lifetime achievement award (2010). His researches have been concerned with the characterization of specific virus-induced tumor antigens, which were the finger-prints left behind in human cancer. Achievements include patents in field; discovery of respiratory syncytial virus in infant deaths in Naples and of tumor liberated protein as a tumor associated antigen, 55 kilodalton protein overexpressed in lung tumors and other epithelial adenocarcinomas.

giuliotarro@gmail.com

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