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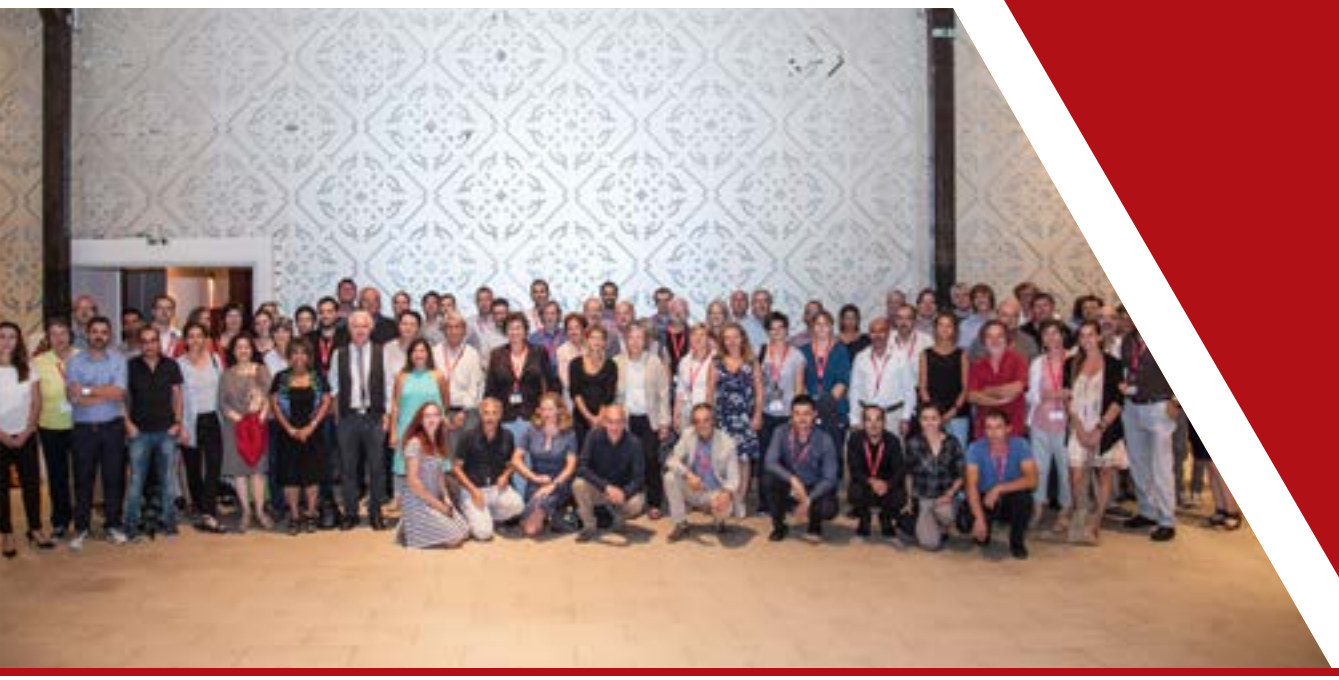

International Conference on

HEART CONGRESS, VASCULAR BIOLOGY AND SURGEON'S MEETING

December 04-05, 2017 Dallas, USA

Workshop

Day 1

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December 04-05, 2017 Dallas, USA

Candece L Gladson

Cleveland Clinic, USA

Therapeutically targeting angiogenesis: Characteristics of tumor-associated endothelial cells and a new mechanism of resistance to bevacizumab

Tumor-associated endothelial cells express unique markers and exhibit differences in blood vessel structure, permeability, internalization of monoclonal antibodies (mAbs), and transcytosis as compared to normal organ endothelial cells. In tumors, endothelial cells are near cancer stem-like cells (CSCs) or tumor cells. The direct contact of the two cell types through integrin αvβ3 on endothelial cells binding L1CAM on CSCs/tumor cells promotes pro-migration signaling in endothelial cells causing increased angiogenesis. This signaling pathway is blocked by cyclic Arg-Gly-Asp-(RGD)-peptide. Bevacizumab, a humanized monoclonal antibody to VEGF, is used routinely in the treatment of patients with recurrent glioblastoma (GBM), renal and metastatic colon and lung cancer. However, little is known regarding bevacizumab effects on cells in the perivascular tumor space. We used established orthotopic xenograft and syngeneic mouse models of GBM to determine entry of bevacizumab into, and uptake by cells in, the perivascular space. We also examined CSCs isolated from GBM for bevacizumab internalization, trafficking and effects on cell survival. In the GBM models, we found that administered bevacizumab entered the perivascular tumor niche and was internalized by CSCs. In the perivascular CSCs, bevacizumab was detected in the recycling compartment or the lysosome, and increased autophagy was found. In CSCs propagated *in vitro*, bevacizumab was internalized rapidly through macropinocytosis with a fraction being trafficked to a recycling compartment and a fraction to lysosomes. We demonstrate that bevacizumab is internalized by CSCs residing in the perivascular tumor niche and macropinocytosis of bevacizumab and trafficking to the lysosome promotes the survival of CSCs, as does the autophagy induced by bevacizumab depletion of VEGF-A. In the workshop, we will discuss the above data, the protocols used to develop the data, how these data fit into our current understanding of anti-angiogenic therapeutics and the development of resistance, as well as the impact on future anti-angiogenic therapy.

Biography

Candece L Gladson has her expertise in the modeling and analysis of angiogenesis and anti-angiogenic therapeutics utilizing both *in vitro* and *in vivo* model systems. Her laboratory focuses on the mechanisms by which the perivascular niche and extracellular environment regulate angiogenesis in vascular tumors; this has included the identification by her laboratory of a cell-cell signaling pathway that modulates angiogenesis and a new mechanism of resistance to anti-angiogenic therapeutics. The overall goal of her laboratory is to develop more efficacious anti-angiogenic therapeutics, aided by the identification of better therapeutic targets.

gladsoc@ccf.org

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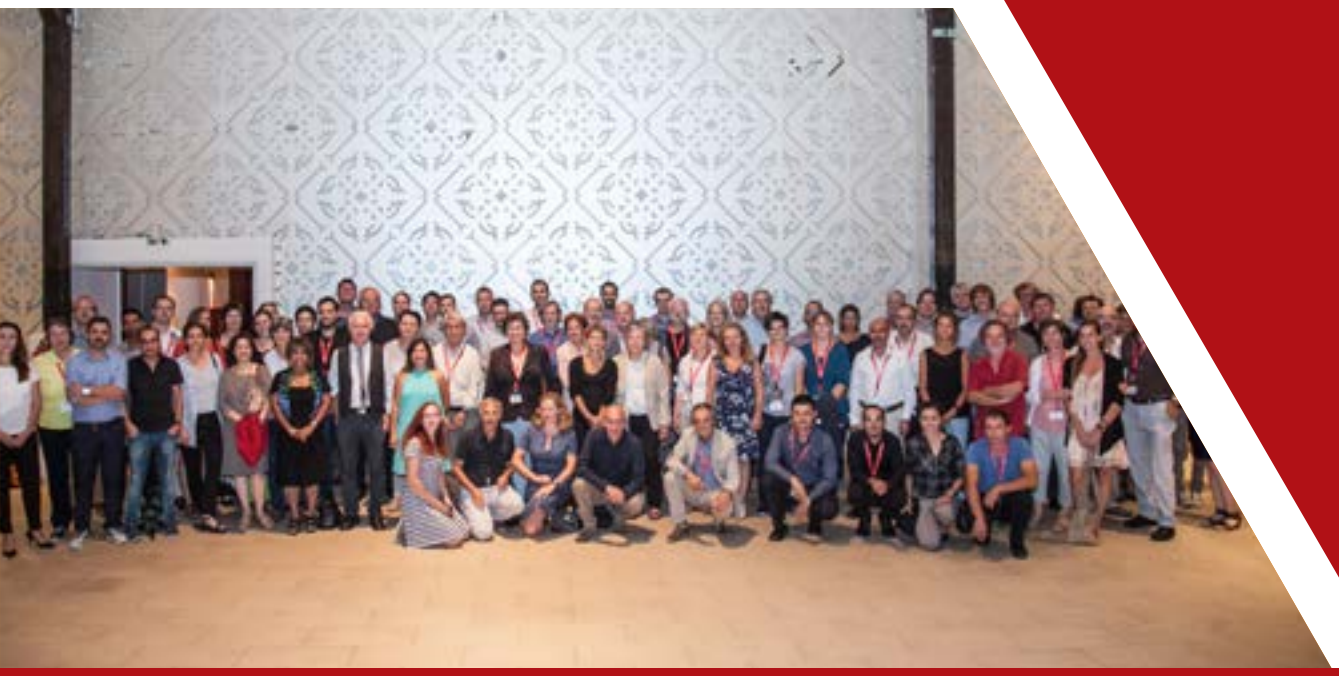

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Scientific Tracks & Abstracts

Day 1

Analysing novel regulators that may affect vascular integrity and function

Vishwanie Budhram-Mahadeo
University College London, UK

Arterial stiffness is associated with chronic hypertension and strongly linked to adverse vascular changes commonly associated with ageing and metabolic diseases e.g. type 2 diabetes. Such changes arise in response to stresses/injuries that transform vascular smooth muscle cells (VSMCs) from differentiated cells into proliferative, migratory phenotypes that synthesise extracellular matrix (ECM) proteins such as collagen 1 (Col1). However, the factors that control phenotypic switching in VSMCs are not clear. I will present results of recent studies which have identified the Brn-3b/POU4F2 transcription factor (TF) as a potentially novel regulator of VSMC fate which may link vascular integrity and function to metabolic function. Brn-3b is expressed in aortic VSMC and Brn-3b knock-out (KO) mutants have reduced arterial compliance and hypertension linked to hyperglycaemia at relatively young ages (by 2 months) and this is associated with significant remodelling in large blood vessels such as the aorta e.g. increased extracellular matrix (ECM) protein deposition, neo-intimal hyperplasia etc. As a transcription factor, Brn-3b can act as a master switch to control which genes are produced in cells. High throughput RNA sequencing analysis has identified significant changes in genes that are required for maintaining vascular integrity when comparing aortas from Brn-3b KO mutants with normal control tissues. Moreover, we have characterised the effects of this regulator in controlling expression of ECM such as Col1a1, which are deregulated in vascular diseases. These findings will increase our understanding of the factors that are required to maintain healthy blood vessels, but also identify how Brn-3b may prevent stiffening to blood vessels and whether its loss is associated with vascular disease development and progression.

Biography

Vishwanie Budhram-Mahadeo has been actively involved in medical research since 1992 and is currently a Tenured Scientist, working as a Principal Investigator (PI) within the UCL Institute of Cardiovascular Science. Her main research interest is focused on elucidating the mechanisms controlling transcriptional regulation of tissue-specific gene expression and cell fate in normal tissues (during embryogenesis or in adults) and changes which contribute to disease initiation or progression [e.g. cancer, cardiovascular disease (CVD) and metabolic diseases e.g. obesity and type 2 diabetes]. The studies utilized the related homeobox transcription factors, Brn-3b/POU4F2 and Brn-3a/POU4F1, to show how complex regulatory effects can be mediated, depending on cell type specificity and/or growth conditions. Data from this research has been pivotal for establishing novel paradigms to explain how different transcription factors can drive similar or antagonistic effects on key cellular functions. e.g. proliferation, survival/apoptosis or differentiation, depending on co-expression and interactions with other regulators such as the p53 tumor suppressor protein. More recently, novel roles for these regulators in the cardiovascular system and potential links to metabolic dysfunction have begun to identify increasing complexity for these regulators in controlling cell fate either during development or in disease.

v.budhram-mahadeo@ucl.ac.uk

Notes:

Endothelial intracellular molecule *LSP1* regulates the chemotactic directionality of extravascular migrating neutrophils

Liu Lixin
University of Saskatchewan, Canada

The recruitment of leukocytes from the flowing bloodstream into inflamed tissue is of key importance in inflammation. This recruitment is characterized as the initial tethering and rolling of leukocytes along the endothelium, followed by leukocyte activation and firm adhesion to the endothelium, leukocyte transmigration across the endothelium (diapedesis), and chemotactic migration of emigrated leukocytes toward the site of infection or injury (chemotaxis) in tissue. The role of intracellular signaling molecules in leukocytes and endothelial cells involved in leukocyte recruitment is investigated in the microvasculature *in vivo*. *LSP1* (leukocyte-specific protein 1), an F-actin-binding, intracellular phosphoprotein, is expressed in leukocytes and endothelial cells with different intracellular localization pattern. In endothelial cells, *LSP1* is mainly expressed in the nucleus with small proportion as cytosolic and cytoskeletal protein. The role of endothelial *LSP1* in neutrophil recruitment is investigated and endothelial *LSP1*-regulated mechanism for extravascular neutrophil chemotaxis is revealed in our study. Using intravital microscopy in *LSP1*-deficient and *LSP1*-chimeric mice, we show that endothelial *LSP1* plays a permissive role in controlling neutrophil transendothelial migration during neutrophil recruitment and regulates extravascular migration directionality but not the migration velocity of emigrated neutrophils. We found that the expression of $\alpha 6\beta 1$ integrins on the emigrated neutrophils was blunted when *LSP1* was deficient in the endothelium of LSP1-deficient or chimeric mice, and that neutrophil $\alpha 6\beta 1$ integrin expression dictated the directionality of emigrated neutrophils *in vivo* and *in vitro*. *LSP1*-deficiency or *LSP1*-targeted siRNA silencing in endothelial cells reduced endothelial adhesion molecule PECAM-1 expression through GATA-2-dependent mechanism. *LSP1* overexpression in endothelial cells has unregulated endothelial PECAM-1 expression. It was the reduced endothelial PECAM-1 expression in *LSP1*-deficient endothelium that down-regulated $\alpha 6\beta 1$ integrin expression on the transmigrating neutrophils and that, through PECAM-1-sensitive, down-regulated $\alpha 6\beta 1$ integrin expression, mitigated the migration directionality of transmigrated neutrophils in tissue. Thus, endothelial *LSP1* regulates vascular PECAM-1-sensitive and neutrophil integrin $\alpha 6\beta 1$ dependent directionality of extravascular neutrophil migration in inflamed tissue during neutrophil recruitment.

Biography

Liu Lixin had research training with Dr. Dirk Roos (CLB, The Netherlands) and completed his PhD study with Dr. Per Venge at Uppsala University (Sweden). In Europe, he has studied the regulatory mechanisms of granulocyte transmigration across epithelial cell monolayer. Thereafter, as a Post-doctoral Fellow, he has joined the lab of Dr. Paul Kubes in the University of Calgary, in Alberta, Canada, where he became experienced in the techniques of intravital microscopy and started to explore the role of intracellular protein molecules in neutrophil transendothelial migration and chemotaxis. In late 2006, he has joined the faculty in the University of Saskatchewan (Saskatchewan, Canada) and currently he is Associate Professor in Pharmacology. Using intravital microscopy and other imaging techniques and biochemical and cell biological approaches, his lab is now investigating the role of intracellular signaling molecules in neutrophil-endothelial cell interactions, with current research interests in the signaling mechanisms of *LSP1* (leukocyte-specific protein 1) and PI3K (phosphoinositide 3-kinases) in neutrophil recruitment during inflammation.

lixin.liu@usask.ca

Notes:

The effects of a combined *Bryophyllum pinnatum*, *Moringa olefeira* and vitamin C phytotherapeutic agent on cholesterol levels

Alfred Sparman
Sparman Clinic, USA

Statins have been regarded globally as the standard therapeutic agent for the treatment of high cholesterol levels. This treatment has become necessary as research has established a direct relationship between high cholesterol levels and the incidence of cardiovascular disease. In some developing countries, it has been observed that populations living in rural areas and poor communities find it difficult to access and afford key therapies such as, statin therapy. And so, often do not meet their treatment goals which eventually lead to progression of their disease and the onset of associated conditions. The perennial herbs, *Bryophyllum pinnatum* and *Moringa oleifera*, have been extensively researched in the countries to which they are indigenous, and have been shown to exhibit antidiabetic, antihypertensive and cholesterol lowering effects. Like these herbs, vitamin C has also been shown to assist with the management of blood glucose. And so, it was inferred that a combination of these phytotherapeutics and vitamin C, should prove effective in managing key risk factors for cardiovascular disease; in this instance high cholesterol. In my recent uncontrolled, randomized study consisting of 16 participants, 10 females and 6 males, total cholesterol levels at baseline (one year prior to the study) and post treatment with the combined phytotherapeutic were investigated. Total cholesterol levels decreased in 93.75% of participants. There was a significant difference in total cholesterol levels from baseline (M=4.5325, SD=1.0719) and post treatment (M=3.9050, SD=0.7057); t (15)=5.1236, p=<0.05. Therefore, I concluded that the use of a *Bryophyllum pinnatum*, *Moringa oleifera* and vitamin C combined phytotherapeutic agent reduces total cholesterol levels. In this seminar, I will discuss the origin of these perennial herbs and previous research on their ability to reduce cholesterol levels; with an emphasis on their cholesterol lowering capabilities when used as a combined *Bryophyllum pinnatum*, *Moringa oleifera* and vitamin C phytotherapeutic agent.

Biography

Alfred Sparman is an Interventional Cardiologist and Pioneer of Angioplasty in Barbados. He is the CEO of one of the premier healthcare facilities in the Caribbean. With years of experience in the field of Cardiology and a successful practice at The Sparman Clinic and 4H Hospital, he continues to build on his knowledge of chemistry and research. Concerned about the growing number of patients being diagnosed with cardiovascular disease annually, and in tune with patients' apprehensions about using drugs, he has taken a keen interest in developing viable alternatives which will diminish these apprehensions, and allow patients and their physicians to meet their treatment goals.

nutrition@thesparmanclinic.com

Notes:

Inflammation and vascular disease

Pavel Poredoš
University Medical Centre Ljubljana, Europe

During the last decade, the role of inflammation in the etiopathogenesis of arterial thrombosis has been elucidated. Inflammation is basic pathogenetic mechanism of atherosclerosis thromboembolic complications. However, little is known about the relationship between inflammation and venous thrombosis. Recently, inflammation has been accepted as a possible mechanism through which different risk factors trigger thrombus formation in arteries as well as in veins. The data indicate that inflammation of the vessel wall initiates thrombus formation in an intact vein and that inflammation and coagulation systems are coupled by a common activation pathway. The first event in thrombus formation is most probably activation of endothelial cells, platelets and leucocytes, with initiation of inflammation and formation of micro particles that trigger the coagulation system through the induction of a tissue factor. Therefore, the key event in the initiation of venous thrombus formation is most probably vein wall inflammation. However, expected relationship between inflammatory markers as indicators of inflammatory process and clinical venous thromboembolism (VTE) has not yet been elucidated. C-reactive protein does not appear to be useful in predicting future venous thrombosis or to be useful in the diagnosis of VTE. Recently, it was demonstrated that probable association between VTE and several other markers of inflammation such as: interleukin (IL)-6, IL-8 and tumor necrosis factor-a exists. While these markers of inflammation were studied during or after acute venous thrombosis, further prospective studies are needed to determine the predictive value of inflammatory markers for VTE. The identification and elucidation of inflammatory markers relevant to venous thrombosis could provide targets for future therapy. That inflammation is the basic etiopathogenetic process of VTE is also supported by the relation of some risk factors to both arterial and venous thrombosis: age, increased body mass index, hypercholesterolemia, hypertension, lupus anticoagulant and hyperhomocysteinemia. A relation was also found between preclinical and clinical atherosclerotic disease and VTE. Also in line with these arguments are the preventive effects of aspirin and statins in both arterial and venous disease.

Biography

Dr. Poredos graduated in the year 1974 from Medical Faculty Ljubljana, Slovenia with the specialization in internal medicine. He did his master of science and PhD in the year 1989. He is now the head of clinical department for vascular disease Medical Center Ljubljana and also as full professor of internal medicine at Medical faculty in Ljubljana.

pavel.poredos@kclj.si

Notes:

Neutrophil to lymphocyte ratio is related to ischemic event and short-term mortality in patients with acute coronary syndrome

Noujoum Zmouli[1,2*], Arslan Bettayeb[1,2], Wissam Tidjane[1,2], Nadjet Tighezza[1,2], Nadia Benatta[1,2], Houari Toumi[1,2] and **Mohamed Hammadi[1,2]**

[1]University of Medicine in Oran, North Africa

[2]University hospital of Oran, North Africa

Background: The inflammation can play a role in the myocardial ischemia and the number of leukocytes allows estimating the inflammatory status to the coronary. The aim of this study was to examine the prognostic utility in the short term of the neutrophil to lymphocyte ratio (NLR) measured in the admission of the patients with acute coronary syndrome (ACS).

Materiel & Methods: A total of 60 coronary patients admitted in the service of cardiology and in the USIC from Mars 2016 to December 2016 in the EHUO. The neutrophil and lymphocyte counts were obtained using a Coulter ADVIA® 2120i Hematology System with Autoslide* (Siemens Healthcare Headquarters, Erlangen, Germany). NLR is then calculated and a questionnaire is completed. Patients are contacted again 6 months later for research of a possible recurrence or mortality. First, a descriptive analysis is realized on the whole sample. Secondly, a Kaplan-Meier survival curve and a Cox regression analysis are established as well as the determination of the value threshold for predicting recurrence by receiver operating curve (ROC).

Results: The mean age 60.09±23.67 and NLR 4.52±7 sex ratio M/F=3.6. Among ACS: 73.3% ST segment elevation myocardial infarction (STEMI), 23.3% non STEMI (NSTEMI) et 3.3% Unstable angor (UA). The recurrence concerned 23% of the patients with 5% of mortality. There is a statistically significant difference of the mean age between men and women (P=0,035). There is no statistically significant difference of the other cardiovascular risk factors between the groups low NLR and high NLR as well as the groups with and without recurrence (P>0,05). The Kaplan-Meier survival curves are significantly different between both categories low NLR and high NLR (P=0,004). The Cox Model reveals that the NLR is the only predictive marker of a possible recurrence (HR=10.92). The cutoff of the NLR=3.63 has 85.71% sensibility and 65.22% specificity.

Conclusion: The NLR is a biomarker little expensive, easily available and obtained quickly by the realization of the simple NFS. It allows stratifying the patients at risk of making a recurrence or a death of cardiovascular origin.

Biography

Noujoum Zmouli is an Assistant Professor in Biological Hematology and Blood Transfusion in University of Medicine and Hospital Practitioner in University Hospital in Oran, Algeria. He has published several articles in French and in English. Currently, he is preparing a Doctoral thesis in Medical Sciences for which the title is: biological evaluation of the platelet reactivity in thienopyridines by the measure of the intra-platelet VASP (vasodilator stimulated phosphoprotein) in patients with ACS.

noudjoum@yahoo.fr

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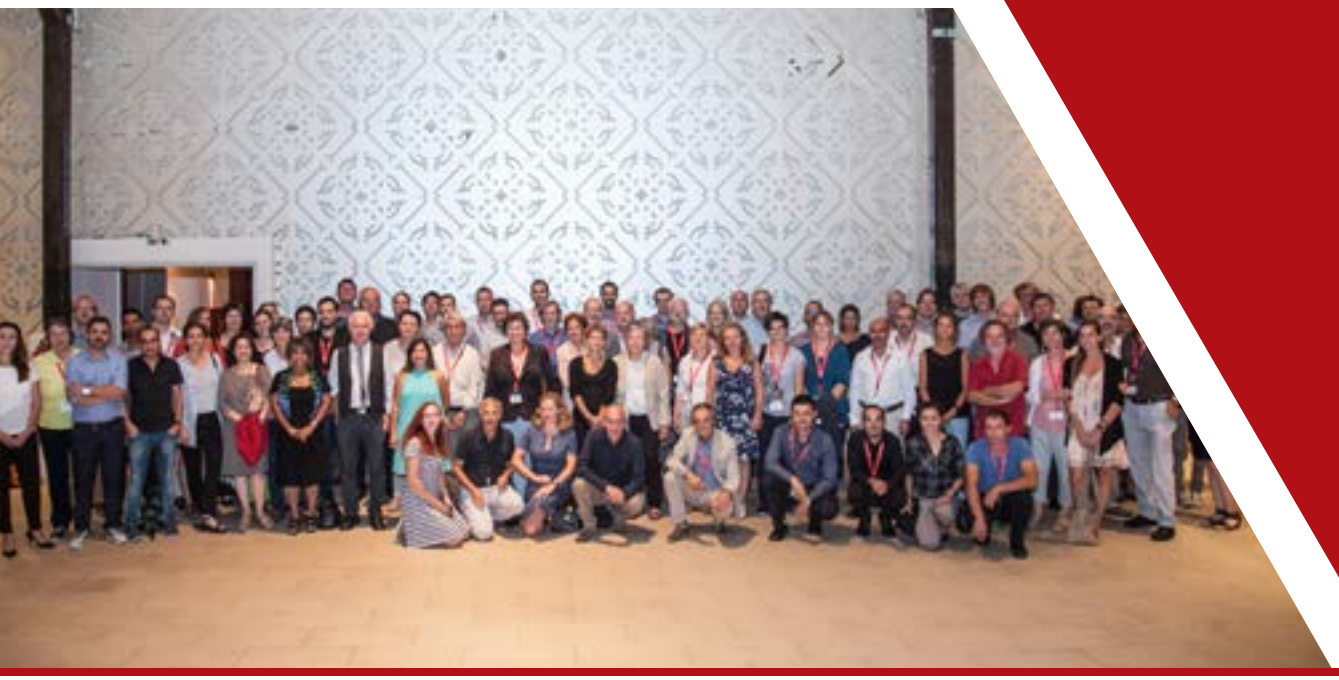

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Scientific Tracks & Abstracts

Day 2

On the regulation of local cerebral blood flow by neurons and astrocytes

Adam Institoris* and **Grant R Gordon**
University of Calgary, Canada

Neurovascular coupling requires intricate communication among neurons, astrocytes and vascular contractile cells that make fine adjustments to microvascular diameter. To meet the energy requirements of the resting and active brain, astrocytes and neurons must regulate microvascular diameter tonically, in a manner that is independent of neural activity, as well as dynamically in response to rapid changes in neural activity. Surprisingly, little is known about how the brain coordinates these distinct modes of blood flow control. We have discovered a key role for resting astrocyte Ca2+ in the steady-state dilation of micro-vessels. Specifically, we show that reducing basal Ca2+ in astrocyte 'endfeet', which are the specialized astrocytic compartments that directly appose microvascular elements, causes a vasoconstriction, in the absence of neural influences. Furthermore, we find that astrocytes are not necessary for vasodilations in response to brief, physiologically relevant increases in neural activity. We provide evidence that preventing Ca2+ transients specifically in the astrocytes that surround a micro-vessel, has no impact on activity-dependent vasodilations. These evoked increases in microvascular diameter are, however, largely eliminated by synaptic AMPA receptor and cyclooxygenase-2 blockade. Furthermore, this type of synaptically driven vasodilation occurs independent of vascular conduction, suggesting a local synaptic effect on nearby microvasculature. Our data suggests that astrocytes and neurons act in parallel to regulate blood flow on distinct timescales.

Biography

Adam Institoris is a research analyst, Hotchkiss Brain Institute, Department of Physiology and Pharmacology, Cumming School of Medicine, University of Calgary.

ainstito@ucalgary.ca

Notes:

CD45+ fibroblast in maladaptive cardiac fibrosis and its suppression by a caveolin-1 surrogate peptide

Dhan Kuppuswamy
Medical University of South Carolina, USA

In response to pathological stimuli such as hypertension, myocardial infarction (MI) and valvular defects, the heart undergoes both qualitative and quantitative changes that contribute to the progression of congestive heart failure (CHF), which develops in concert with myocardial fibrosis. We have used two independent models (transverse aortic constriction and angiotensin II infusion) of pressure overload (PO) to induce cardiac fibrosis that results in compromised ventricular function. Based on the idea that caveolin-1 deficiency in fibroblasts and monocytes contributes to fibrosis, we explored the role of caveolin-1 in the fibrotic processes using the caveolin-1 scaffolding domain peptide (CSD, a 20-amino acid segment of caveolin-1 that acts as a functional surrogate). While it is accepted that bone marrow (BM)-derived cells play a key role in cardiac fibrosis, it is controversial whether this is solely due to monocytes differentiating into macrophages that secrete factors that activate resident fibroblasts, or whether BM monocytes also differentiate into a major portion of the fibroblasts that overexpress collagen I (Col I) in PO myocardium. Our recent studies support the latter idea that PO causes a major increase in the levels of CD45+/ HSP47+ cells in the fibrotic heart which could be blocked by treating mice with CSD. Suppression of fibrosis by CSD was accompanied with improved ventricular function. Moreover, when fibroblast cultures are initiated using cells from fibrotic heart, the fibroblasts continue to express CD45, strongly suggesting that CD45+ cells are indeed fibroblast precursors. Our results strongly suggest that monocytes differentiate into a major portion of the fibroblasts that overexpress Col I in the fibrotic heart and that the recruitment of monocytes, their differentiation into fibroblasts, and the overexpression of Col I by fibroblasts are all potential points of regulation of fibrosis by CSD. Our ongoing studies are focused to validate the importance of caveolin - 1 and CD45+ BM-derived cells in heart fibrosis so that they set the stage for developing CSD as a treatment for cardiac fibrosis.

Biography

Dhan Kuppuswamy is an Associate Professor of Medicine, Cardiology Division at the Medical University of South Carolina (MUSC), Charleston, South Carolina. As a faculty member for the past 23 years at MUSC, he has been studying cellular and molecular mechanisms involved in cardiac hypertrophy that often leads to congestive heart failure. He got his Masters and PhD degrees at the University of Madras, India and moved to US as a postdoctoral fellow in 1984. After joining as a faculty at MUSC, he employed much of his expertise into cardiology on how integrin-mediated tyrosine kinase signaling promotes growth, survival and differentiation of cardiac muscle cells and fibroblasts. Studies from his lab show that β3 integrin mediated signaling mechanisms activate nonreceptor tyrosine kinases that contribute to cardiomyocyte growth and survival on one hand and promote cardiac fibroblast proliferation and extracellular matrix accumulation on the other hand. His recent collaborative studies shows very compelling data that ventricular pressure overload in mice can cause increased levels of CD45+ cells (monocyte-derived cells) in the heart that express fibrogenic markers and that treating these mice with the caveolin-1 scaffolding domain (CSD) peptide can suppresses recruitment and differentiation of CD45+ cells into fibrogenic cells, decrease cardiac fibrosis, and improve ventricular function. He has trained several PhD students and postdoctoral fellows and he is actively involved in graduate school.

kuppusd@musc.edu

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Short term outcome of thoracic endovascular aortic repair in patients with thoracic aortic diseases

Huthayfa Ghanem[1*] and **Mohammed N. El-Ganainy**[2]
[1]Alexandria University, Egypt
[2]National Heart Institute, Egypt

Aim and Background: Open surgical repair for thoracic aortic diseases is associated with a high perioperative mortality and morbidity. Most of type B aortic dissections are uncomplicated and are medically treated which carries a high mortality rate. Thoracic endovascular aortic repair is the first-line therapy for isolated aneurysms of the descending aorta and complicated type B aortic dissection. The aim of this study is to test the efficacy and safety of thoracic endovascular aortic repair in patients with uncomplicated type B aortic dissection and patients with descending thoracic aortic aneurysms.

Methods: A total of 30 patients (24 men and 6 females; mean age 59±8 years) with uncomplicated type B aortic dissection and descending aortic aneurysm who underwent endovascular aortic repair in National Heart Institute and Cairo University hospitals were followed up. Clinical follow-up data was done at one, three and twelve months thereafter. Clinical follow-up events included death, neurological deficits, symptoms of chronic mal-perfusion syndrome, and secondary intervention. Multi-slice computed tomography was performed at an average of three and six months after intervention.

Results: Of the 30 patients, 24 patients had aortic dissection, and 6 patients had an aortic aneurysm. Seven patients underwent hybrid technique and rest underwent the basic endovascular technique. Success rate was 100% for the basic endovascular procedures. Two patients developed complications, type Ia endoleak and type IIa endoleak, however both improved after short term follow up. The total mortality rate was 10% throughout the follow-up. Early thoracic endovascular aortic repair showed better results and less complication.

Conclusion: Along with medical treatment, early thoracic endovascular aortic repair should be considered as the gold standard in uncomplicated aortic dissections type B and aortic aneurysms.

Biography

Dr. Huthayfa Ghanem did his Bachelor of Medicine and Surgery from College and University of Alexandria, Egypt.`

avihoses@hotmail.com

Notes:

Central obesity and its implicated adverse health conditions are a major concern of some people

Iwezu Happy Nonso
University of Nigeria, Nigeria

Central obesity is one of the predisposition factors to cardiovascular disease, respiratory conditions, type 2 diabetes mellitus, cancer and others. The purpose of this study was to determine the relationship between central obesity, cardio respiratory fitness and physical activity level among adults in Enugu State. Three hundred and seventeen subjects (158 obese and 159 non-obese) who met the inclusion criteria and gave their informed consent participated in the study. Their waist to hip ratio was determined using measuring tape. Body Mass Index measured with stadiometer and weighing scale. Cardio respiratory fitness was determined using Harvard Step Test and International Physical Activity Questionnaire (IPAQ) was used to determine their physical activity level. Data collected was analyzed descriptively and inferentially using correlation test. The level of significance was set at p=0.01 The physical activity level of obese adults in Enugu state increases in an ascending order of vigorous, low, moderate while the cardiorespiratory fitness decreases in reverse direction as low, average, good, excellence and very low. There was a significant relationship between cardio respiratory fitness and physical activity (r=0.146, sig=0.009). There was also a significant relationship between waist-hip ratio and cardiorespiratory fitness (r=-0.221, sig=0.000).

Biography

Dr. Iwezu Happy Nonso is having his expertise in sports medicine (physiotherapy) and passion in improving the health and wellbeing. His open and contextual evaluation model based on responsive constructivists creates new pathways for improving healthcare and also developed this model after years of experience in research, evaluation, teaching and administration both in hospital, outreach programs and education institutions. This will go a long way in restoring man back to form and function physically via spatial neuroplasticity.

iwezuhappy@yahoo.com

Notes:

Influence of combined conventional and modified ultrafiltration in neonates on coagulation, hemodynamic and blood loss after pediatric cardiac surgery

Yopie A Habibie
Syiah Kuala University, Indonesia

Background: Combined Conventional (CUF) and Modified Ultrafiltration (MUF) in pediatric cardiac surgery with Cardiopulmonary Bypass (CPB) may offer advantages in comparison with conventional or modified ultrafiltration itself.

Methods: From January 2008 to October 2009, a total of 105 pediatric patients undergoing cardiac surgery were collected prospectively and analyzed retrospectively, divided into 2 groups. Group I undergo CUF, group II was CUF + MUF. The using of filtration was randomized. Sub analysis was between patient with Aristotle score above five and preoperative Pulmonary Hypertension (PH). Preoperative, intra operative, during CPB, post-operative data in ICU such as hemodynamics, hemoglobin, transfusion of blood products, Low Cardiac Output Syndrome (LCOS) such as lactate, arterial, vein saturations, mean arterial pressure were compared between groups.

Results: There was one operative mortality. Total patients with CUF (n=52) and CUF+MUF (n=53). Patients Aristotle score above 5 (n=101), median age 24 (1:204) months and median weight 9.5 (4:51) kg. In neonates, CUF+MUF increased hemoglobin level up to 2.1 (-1.6, 8.9 p=0.206) mg/ml post operatively, significantly reduced total transfusion volume of coagulation factors in ICU (RBC 225(90, 360) mL; p=0.099; FFP 110 (60:160) mL; p=0.075; TC 67, 50 (65, 70) m; p=0.365), also increased mean arterial blood pressure 12 (-26, 62) mmHg. Patients preoperative PH (n=95), median age 24 (1, 168) months and median weight 9,2 (4, 44) kg. In neonates, CUF+MUF also increased hemoglobin level up to 2,1 (-1.6, 8.9 p=0.224) mg/ml, but not significantly reduced total transfusion volume of coagulation factors (RBC 225 (90, 360) mL; p=0.154; FFP 110 (60, 160) mL; p=0.104; TC 67,50 (65, 70) mL ; p=0.400), also increased mean arterial blood pressure 11 (-26, 41) mmHg. LCOS have significant lower in all groups. In both groups, ventilation duration longer in CUF+MUF groups, but there was no significant difference among groups in ICU stay, central venous pressure post operatively.

Conclusions: Combination UF is effective and safe in pediatric patients undergoing cardiac surgery, make LCOS decreased, better hemodynamics and coagulation function if compare with other's strategy. We recommend the use of combined UF is safe in high-risk pediatric patients for hemoconcentration after cardiac surgery. Optimal use of combined UF includes patients with preoperative PH and with Aristotle score>5.

Biography

Yopie A Habibie has completed his Bachelor's degree in Medical Majors at Syiah Kuala University Banda Aceh (2005), graduated from Specialist Program 1 (Sp-1) Thoracic Cardiac and Vascular Surgery at the Faculty of Medicine University of Indonesia, Jakarta in 2012. In 2005-2006, he has served as a General Practitioner at Harapan Bunda Hospital, Banda Aceh and also in Refugee's Camp Lhokseumawe. He has under went for advanced fellowship training as Junior Consultant at 2013 for Overseas Adult Cardiac Surgery Fellow at Narayana Health Institute of Cardiac Science's in Bangalore India. In 2014, he has achieved Fellow of Indonesia Heart Association (FIHA) title from Indonesian Heart Association. Currently, he is serving as Medical Staff of Surgical Sciences at Dr. Zainoel Abidin General Hospital, as Head of Thoracic Cardiac and Vascular Surgery and Vice Chairman of Integrated Heart Center of Zainoel Abidin General Hospital. He has engaged actively in various organizations of the European Heart Surgery profession (EACTS), Asia (ASCVTS and ATCSA), American (STS) and WSPCHS. From 2006 up to now, he is appointed as a permanent Lecturer at the Medical Faculty of Syiah Kuala University.

yopie98@yahoo.com