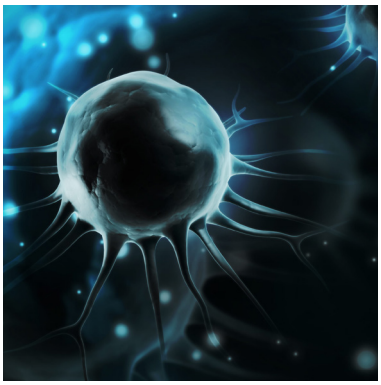
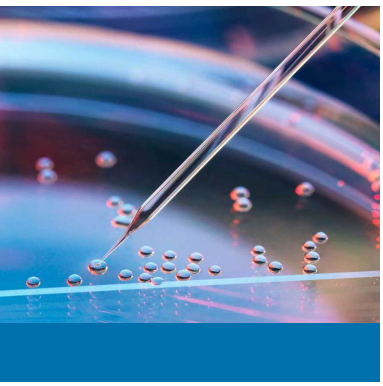
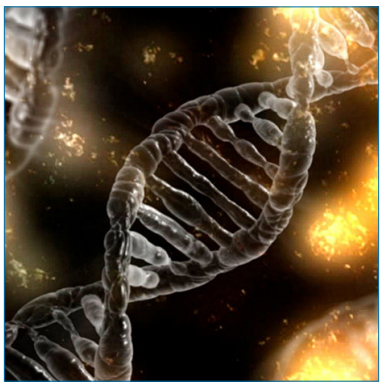
Video Presentation

Molecular Biology, Tissue Science & Heart Congress 2018







Joint Event

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4th World Heart Congress

November 19-20, 2018 | Paris, France

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Hypertension of moon walkers

William J Rowe
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Hypertension of 12 moon walkers, James Irwin on day after return from Apollo 15 mission, showed extraordinary bicycle (B) stress test (ST) hypertension (275/125) after 3 minutes exercise; supervising >5000 maximum treadmill ST, author never witnessed ST- blood pressure approaching this level. Symptom-limited maximum B stress test showed "cyanotic fingernails"; Possibly venous blood trapped peripherally, supporting author's "Apollo 15 Space Syndrome," postulating that severe fingertip pain during space walks, triggered by plasma fluid, trapped distally; Mechanism could be related to endothelial dysfunction, providing "silent ischemia" warning. Neil Armstrong returned to earth with severe diastolic hypertension (160/135), consistent with ischemic left ventricular dysfunction; 50 mm increase in comparison with resting BP 110/85. With inhalation of lunar dust, brought into habitat on space suit, with high lunar iron (I) this dust inhalation, along with reduced (R) space flight-transferrin, R antioxidant, calcium (Ca) blocker-magnesium, conducive to severe oxidative stress, Ca overload with potential endothelial injuries. Using moon walker studies as example, my recent editorials show that I dust, released from brakes, with over 90% of brakes made of I, is a major hypertension factor and may also contribute to myocardial infarctions.

Speaker Biography

William J Rowe is a board certified specialist in Internal Medicine. He received his MD at the University of Cincinnati and was in private practice in Toledo, Ohio for 34 years. He is a former assistant clinical professor of Medicine at the University of Ohio, School of Medicine at Toledo. Of only 4 space syndromes, he has published 2: "The Apollo 15 Space Syndrome" and "Neil Armstrong Syndrome". He published Neil Armstrong's probable lunar acute heart failure. He has been listed in the Marquis Whos Who of the World from 2002-2009,2013, 2014, 2015, 2016.

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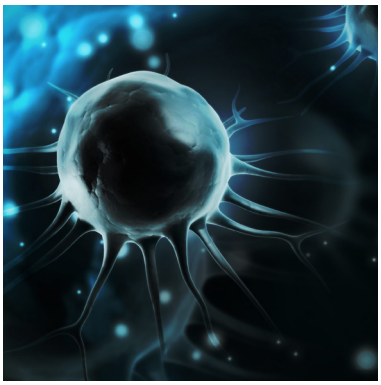
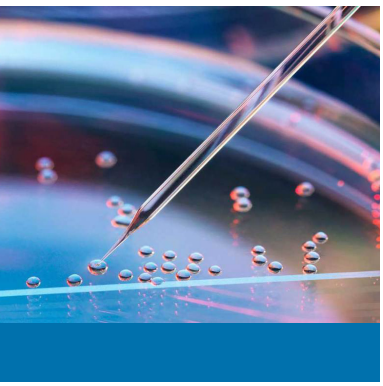
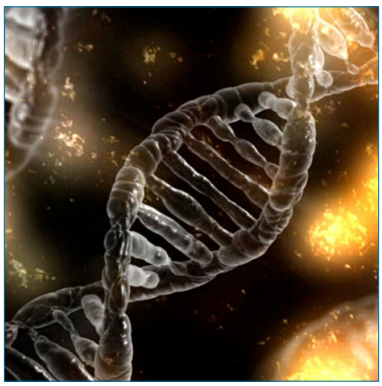
Poster Presentation

Molecular Biology, Tissue Science & Heart Congress 2018







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Arshi Malik, Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

To evaluate the effect of ellagic acid treatment on the cell viability of human prostate cancer cells

Arshi Malik
King Khalid University, Saudi Arabia

Objective: To evaluate the effect of ellagic acid treatment on the cell viability of human prostate cancer cells.

Methods: Ellagic acid (10-100 mol/L) treatment (48h) of human prostate carcinoma PC3 cells was found to result in a dose-dependent inhibition of cell growth and apoptosis of PC3 cells as assessed by MTT assay, western blotting, flow cytometry and confocal microscopy.

Results: We observed that ellagic acid treatment of PC3 cells resulted in a dose dependent inhibition of cell growth/cell viability. This ellagic acid caused cell growth inhibition was found to be accompanied by induction of apoptosis, as assessed by the cleavage of poly (ADP-ribose) polymerase (PARP) and morphological changes. Further, induction of apoptosis accompanied a decrease in the levels of antiapoptotic protein Bcl-2 and increase in proapoptotic protein Bax, thus shifting the Bax: Bcl-2 ratio in favor of apoptosis. Ellagic acid treatment of PC3 cells was also found to result in significant activation of caspases, as shown by the dose dependent decrease in the protein expression of procaspase−3, −6, −8 and −9. This ellagic acid-mediated induction of apoptosis was significantly (80%-90%) inhibited by the caspase inhibitor N-benzyloxycarbonyl-Val-Ala-Asp (OMe)-fluoromethylketone (Z-VAD-FMK). Thus, these data suggested an essential role of caspases in ellagic acid-mediated apoptosis of PC3 cells.

Conclusions: It is tempting to suggest that consumption of tropical pigmented fruits and vegetables could be an effective strategy to combat prostate cancer.

Speaker Biography

Arshi Malik is currently an assistant professor at the College of Medicine, Department of Clinical Biochemistry, King Khalid University, Kingdom of Saudi Arabia. His work mainly focusses on chemoprevention and chemotherapy by various natural agents. He has an extensive experience with human tissue culture, orthotopic/ecotopic implantations of tumors in rodents, tail vein injections, surgery of small animals. Before coming to King Khalid University in Saudi Arabia, he also taught/mentored undergraduate students at Harvard Medical School, Boston as well as at the University of Wisconsin, Madison, United States. Arshi earned his PhD degree in Biochemistry in the year 2002 from Aligarh University in India.

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November 19-20, 2018 | Paris, France

The validation of hla-b*13:01 as genetic biomarker of trichloroethylene-induced hypersensitivity syndrome in a prospective cohort study

Qiang Jia
Shandong Academy of Medical Sciences, China

Trichloroethylene (TCE)-induced hypersensitivity syndrome (HS) has become one of the serious public health issues. Previous case-control study found that HLA-B*13:01 is a high-risk marker of TCE-induced HS (TCE-HS) with odds ratio of 27.5. This study aimed to validate HLA-B*13:01 as a genetic biomarker of TCE-HS and evaluate its effectiveness for screening TCE-HS in population occupationally exposed to TCE, for which 1,764 new workers firstly exposed to TCE were enrolled in this prospective cohort study. During the three-month period of follow-up, 16 patients were found with the incidence of TCE-HS being 0.97%. Based on genotyping of HLA-B, the study subjects were divided into HLA-B*13:01 positive group and negative group. We found the incidence of HS was 7.59% in HLA-B*13:01 carriers and 0.27% in non-carriers of HLA-B*13:01. The relative risk (RR) is as high as 28.35 (95% CI: 9.25 to 86.85). Receiver operating characteristic curve analysis showed that HLA-B*13:01 allele screening yielded an area under curve (AUC) of 0.83 with sensitivity of 75% and specificity of 91.1% in discriminating TCE-HS. The study firstly reported the incidence of TCE-HS in southern Chinese population and validated HLA-B*13:01 as a susceptible biomarker in TCE-exposed cohort. The effectiveness analysis highlighted that HLA-B*13:01 allele screening is a cost-effective means in prevention of TCE-HS among TCE-exposed workers.

Speaker Biography

Qiang Jia is now currently working in Key Laboratory of Chemical Safety and Health, National Institute for Occupational Health and Poison Control, Chinese Centre for Disease Control and Prevention, Beijing, China and also in Shandong Academy of Occupational Health and Occupational Medicine, Shandong Academy of Medical Sciences, Shandong, China.

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Accepted Abstracts

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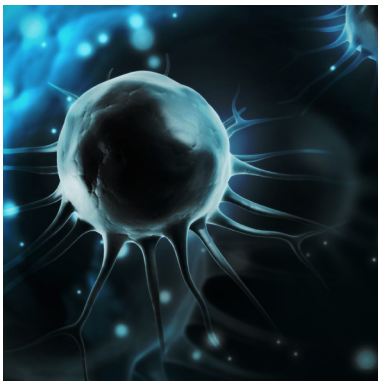

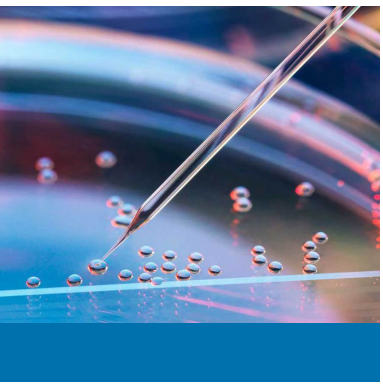

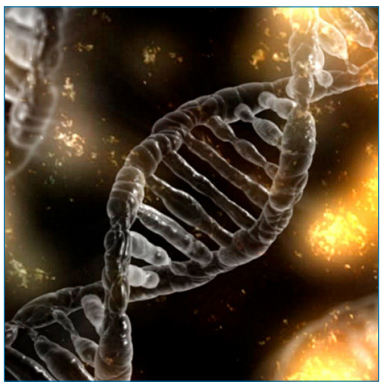

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Leveraging NQO1 bioactivatable drugs for tumor-selective PARP inhibitor synergy for pancreas, breast and nonsmall cell lung cancers

David A Boothman
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Therapeutic drugs that block DNA repair, including poly (ADP-ribose) polymerase (PARP) inhibitors, fail due to lack of tumor-selectivity. When PARP inhibitors and NQO1 bioactivatable drugs are combined, synergistic antitumor activity results from sustained NAD(P)H levels that refuel NQO1-dependent futile redox drug recycling. Significant oxygen consumption rate/reactive oxygen species cause dramatic (DNA) lesion increases that are not repaired due to PARP inhibition. In NQO1+ cancers, such as non-small cell lung, pancreatic and breast, the cell death mechanism switches from PARP1 hyperactivation-mediated programmed necrosis with NQO1 bioactivatable drug monotherapy to synergistic tumor-selective, caspase-dependent apoptosis with PARP inhibitors and NQO1 bioactivatable drugs. Complete metabolic profiling of cells containing or lacking NQO1 and treated with PARP inhibitor Rucaparib along with the NQO1 bioactivatable drug, ß-lapachone, demonstrated dramatic shifts from suppression of glycolytic and KREB's cycle with NQO1 bioactivatable drugs alone to apoptotic activation with PARP inhibitors along with the NQO1 bioactivatable drug. Synergistic antitumor efficacy and prolonged survival were noted in human orthotopic pancreatic and non-small cell lung xenograft models, expanding use and efficacy of PARP inhibitors for human cancer therapy. Cell death was found independent of oncogenic driver mutations or over-expressed oncogenes or loss of tumor suppressors. This work was supported by NIH/NCI R01 grants, R01 CA102792-16 and R01 CA221158-01 to DAB.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Next generation sequencing: A novel approach to distinguish multifocal primary lung adenocarcinomas from intrapulmonary metastases

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Distinguishing between multiple lung primaries and intrapulmonary metastases is imperative for accurate staging. The American Joint Committee on Cancer (AJCC) criteria are routinely used for this purpose but can yield equivocal conclusions. We evaluated whether next generation sequencing using the 50 gene AmpliSeq Cancer Hotspot Panel v2 can be used to facilitate this distinction. Sequencing was performed on known primary-metastatic pairs (8 patients) and multiple lung adenocarcinomas (11 patients). Primary-metastatic pairs showed high mutational concordance: Seven pairs shared mutations and 1 was concordant for having no mutations. Driver mutations in KRAS (4), EGFR (2) and BRAF (1) were always concordant. Multiple lung tumors from 3 patients were completely concordant and therefore, were predicted by sequencing to be intrapulmonary metastases, whereas 8 had completely discordant mutations and therefore, were predicted to be primaries. The sequencing prediction correlated with the AJCC (8th edition) prediction in all patients for whom the latter was unequivocal (8 of 11). Furthermore, it separated patients by overall survival: Patients with predicted multiple primaries had better survival than those with distant metastases (p=0.016, log-rank test), whereas those with predicted intrapulmonary metastases showed no difference (p=0.527). With further validation, the 50 gene panel has the potential to serve as an adjunct to the AJCC criteria, especially in patients for whom the latter is equivocal.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Silicosis-induced fibrosis: Pathogenesis, intervention and treatment

D Cheng Peng
Queensland Alliance for Environmental Health Sciences, Australia

Long term exposure to silica can induce silicosis, a globe disease with higher incidence in developing countries. Although extensive efforts have been made, the molecular mechanisms remain to be fully elucidated for this disease. It is believed that the general process of the lung fibrosis includes the cell damage, formation inflammation, epithelial mesenchymal transition (EMT), extra cellular matrix and collagen deposition and consequent fibrosis. Due to the pathogenic complexity of the disease, together with the irreversibility of the fibrosis, silicosis is currently a progressive and incurable disease. Pharmacological treatment methods targeting on above process have been proved to be largely unsatisfactory. As an emerging treatment protocol, cell therapy through stem cell transplantation is promising in treatment of many disease including lung fibrosis. In this talk, the molecular mechanisms of silicosis, current treatment and our work to investigate the intervention effects of stem cells from various sources on the formation and development and lung fibrosis in animal model will be discussed.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

CTGF/CCN2 a profibrotic factor to target in neuromuscular diseases

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Muscular fibrosis is an excessive accumulation of extracellular matrix (ECM) replacing functional tissue, characteristic of several myopathies and neuropathies. The knowledge of pro-fibrotic factors biology and regulation is critical to develop new therapeutic strategies. Upon unilateral sciatic section, we observe in denervated hindlimb accumulation of ECM proteins such as collagen and fibronectin together with an increase of profibrotic factors TGFβ and CTGF/CCN2. We use hemizygous mice for CTGF/CCN2 and treatments with a blocking antibody against CTGF/CCN2 and we observe reduced denervation-induced fibrosis when compared to control mice suggesting a direct role for CTGF/CCN2 on denervation-induced fibrosis. In time course experiments, we observe that ECM proteins and CTGF/ levels are increased early after denervation (2-4 days), while TGFβ signalling present a delayed kinetics of appearance (1-2 weeks). Furthermore, blockage of TGFβ signalling does not decrease fibronectin or CTGF levels 4 days after denervation. These results suggest that in our model CTGF/CCN2 is not up-regulated by canonical TGFβ signalling early after denervation and other factors might be involved in the fibrotic response very soon after skeletal muscle denervation.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

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The extracellular matrix molecule tenascin-C promotes metastasis by multiple mechanisms

Gertraud Orend
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Today, an orchestrating role of the tumor microenvironment is widely accepted where especially stromal cells and soluble factors are recognized as active players. Yet, the extracellular matrix, although highly abundant, is often considered as passive bystander. A better understanding of the functions of the extracellular matrix in cancer is largely hampered by the lack of relevant models. This applies also to the extracellular matrix molecule tenascin-C, which is a marker of the cancer specific tumor microenvironment. We used a comprehensive approach comprising novel immune competent mouse models (with engineered tenascin-C levels) and demonstrated that tenascin-C plays multiple roles in cancer. Tenascin-C is dangerous as soon as it is expressed out of control. Through assembly into "Tumor Matrix Tracks", tenascin-C impacts tumor and stromal cells including immune cells thereby regulating tumor immunity. Tenascin-C also enhances formation of new but leaky blood vessels. Direct interactions with tenascin-C causes endothelial cell rounding and death or survival upon induction of an insulating pericellular fibronectin coat. Tenascin-C enhances the angiogenic switch and upregulates a proangiogenic secretome. Finally, tenascin-C is an important component of metastatic vascular invasions by promoting endothelialization and cellular plasticity thereby increasing lung metastasis.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

VEGF-A, a potential biomarker for systems medicine

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Vascular endothelial growth factor–A (VEGF–A) is implicated in angiogenesis, lymphangiogenesis, vascular permeability and haematopoiesis. It is associated with numerous pathologies including cardio-vascular diseases and several types of cancer. We specifically developed an integrative systems biology strategy for clinical improvement of this biomarker.

A high heritability of this trait, 60% was estimated in the STANISLAS cohort giving us the needed arguments to continue for a deep characterization of the genetic component of VEGF–A levels. Therefore, we searched, by a Genome Wide Association Study (GWAS), the VEGF–A genetic variants and the inter-connexions of these biomarkers with other disease-associated molecules in healthy populations. Functional transcriptomic analyses were performed in peripheral blood mononuclear cells (PBMCs). Four polymorphisms (rs6921438, rs4416670, rs6993770, rs10738760) explaining ~50% of VEGF–A heritability were identified. These variants, directly or via gene x environment interactions had significant effects on HDL, LDL, TNF-a, IL-6, E selectin and ICAM-1 plasma levels. SNP rs6993770 was shown to increase VEGF121 mRNA levels and rs4416670 was associated with L-selectin expression.

Recently, thanks to a meta-GWAS, we identified 6 additional rs further explaining VEGF–A levels variability and ongoing investigations focus on clinical implementation of the '–omics' determinants of this biomarker.

Our integrative strategy illustrates an improved exemple to be applied for every biomarker with high heritability levels, consequently with potential interest in personalised medicine

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Diagnostic biochips for the analysis of tumor biomarkers

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Engelhardt Institute of Molecular Biology, Russia

Genetic aberrations in leukemia often lead to the formation of expressed chimeric genes, which should be assessed for proper diagnosis and therapy. We developed a biochip-based assay for the analysis of 22 most clinically relevant fusion genes occurring in pediatric leukemia, including 8 recurrent translocations involved the MLL gene and different gene partners. The method includes a multiplex reverse transcriptase–polymerase chain reaction (RT–PCR) to amplify and fluorescently label a fusion transcript fragments and subsequent hybridization of on a biochip with immobilized oligonucleotides complementary to different parts of fusion genes. The fine structure of the MLL fusion genes, including localization of the MLL breakpoints, was performed in 43 infants and 28 pediatric cases. Other important cancer biomarkers predicting prognosis and therapeutic targets are somatic mutations in solid tumors. A diagnostic biochip for the detection of 39 clinically relevant somatic mutations in the BRAF, NRAS, KIT, GNAQ, GNA11, MAP2K1 and MAP2K2 genes has been developed. The multiplex LNA-clamp PCR was used for the preferable amplification of mutated over wild type DNA. The approach could detect 0.5% of mutated DNA in the sample analyzed.

The biochip-based assay is a robust and highly sensitive method for the detection of fusion genes and somatic mutations in cancer patients.

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Tibetan patients with hepatic hydatidosis can tolerate hypoxic environment with no incident increase of pulmonary hypertension - An echocardiograph study

Lixue Yin

Sichuan Academy of Medical Sciences & Sichuan Provincial People's Hospital, China

To evaluate the characteristics of right ventricular and pulmonary anatomical structure, function and hemodynamics of Tibetan patients with hepatic hydatidosis living in high plateau using echocardiography. This prospective study is involved 262 Tibetan patients diagnosed with hydatidosis from June 2016 to June 2017 in Shiqu and Seda areas (4178 meters above sea level in Shiqu County and 3,878 meters above sea level in Seda County). The anatomical structure, function and hemodynamic parameters of the right ventricle and pulmonary between the Tibetan patients with hepatic echinococcosis and the high plateau control group were compared.

In the hydatidosis group, there was no significant differences in the detection rate of TR and PR (χ^2=1.993, p=0.158 and χ^2=3.468, p=0.063, respectively). There was no significant difference in the incidence of PAH (χ^2=1.456, p=0.228) and also no significant difference in the degree of PAH between the two groups (38.93 ±4.60mmHg vs. 41.50 ±6.55mm, p>0.05). The OR value of PAH risk in patients with hydatidosis was 0.708 and the 95% CI was 0.317-1.582. There was no significant difference in the detection rate of TR and PR between the subgroups of hydatidosis and the plateau control group (χ^2=2.323, p=0.508 and χ^2=7.455, p=0.059, respectively). There was no significant difference in the incidence of PAH (χ^2=2.086, p=0.555) and also no significant difference in the degree of PAH among the four groups (F=0.738, p=0.535).

There was no significant effect on the incidence of pulmonary hypertension and its pressure level in Tibetan patients, suggesting that the pulmonary circulation system of Tibetan might have a strong tolerance to hepatic hydatidosis and hypoxic environment simultaneously.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Molecular biology in Kinshasa

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Molecular biology has reached its peak in the Democratic Republic of Congo (DRC) and particularly in Kinshasa due to major epidemics outbreaks in the region such as EBOLA haemorrhagic fever, HIV, swine virus, cassava mosaic virus and so many others. First introduced in the medical curriculum, she quickly evolved in the field of biomedical research and various community services. Nearly 9 Molecular biology laboratories exist across the country, including 4 in Kinshasa and all in the public sector. At the National Institute of Biomedical Research (INRB), the work of molecular biology is mainly focused on the research and therapeutic development of EBOLA fever. At the Faculty of Medicine of the University of Kinshasa (UNIKIN), researches are more diversified. They range from HIV (diagnosis, management, genetic diversity and resistance) to plasmodium (diagnosis and resistance) through viral hepatitis and others parasites. In addition, molecular biology has made it possible to identify the viruses responsible for African cassava mosaic, the genotypes involved in sickle cell disease, the mapping of HIV strains circulating in the DRC and the different mutations associated with ARV resistance in Kinshasa. All molecular biology laboratories in Kinshasa have adopted the standards of good practice and research. Nevertheless, the challenges are still huge for the evolution of this new technology; In other, weak state funding and lack of partnership to support the different areas of research are the main struggle.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Stress cardiomyopathy: Emerging concepts on diagnosis and management

Samer Ellahham
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Stress cardiomyopathy or takotsubo cardiomyopathy, is a syndrome characterized by transient regional left ventricular dysfunction in the absence of significant coronary artery disease. Possible pathogenic mechanisms include catecholamine excess, microvascular dysfunction and multivessel coronary artery spasm. The diagnosis should be suspected in adults who present with a suspected acute coronary syndrome when the clinical manifestations are out of proportion to the degree of elevation in cardiac biomarkers. A physical or emotional trigger is often but not always present. Wall motion abnormalities in patients with stress cardiomyopathy are typically the apical type and atypical variants including mid-ventricular, basal, focal and global types. The differential diagnosis of stress cardiomyopathy includes acute coronary syndromes, coronary artery spasm, myocarditis and pheochromocytoma. A high index of suspicion is key in the diagnosis and management.

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The centres of premeltons signal the beginning and ends of genes

Henry M Sobell
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Premeltons are examples of emergent structures (i.e., structural solitons) that arise spontaneously in DNA due to the presence of nonlinear excitations in its structure. They are of two kinds: B-B or A-A premeltons form at specific DNA-regions to nucleate site-specific DNA melting. These are stationary and being globally nontopological, undergo breather motions that allow drugs and dyes to intercalate into DNA. B-A or A-B premeltons, on the other hand, are mobile and being globally topological, act as phase-boundaries transforming B- into A- DNA during the structural phase-transition. They are not expected to undergo breather-motions. A key feature of both types of premeltons is the presence of an intermediate structural-form in their central regions (proposed as being a transition-state intermediate in DNA-melting and in the B- to A- transition), which differs from either A- DNA or B- DNA called beta-DNA, this is both metastable and hyperflexible and contains an alternating sugar-puckering pattern along the polymer-backbone combined with the partial-unstacking (in its lower energy-forms) of every other base-pair. Beta-DNA is connected to either B- or to A- DNA on either side by boundaries possessing a gradation of nonlinear structural-change, these being called the kink and the antikink regions. The presence of premeltons in DNA leads to a unifying theory to understand much of DNA physical-chemistry and molecular-biology. The premeltons are predicted to define the 5' and 3' ends of genes in naked-DNA and DNA in active-chromatin, this having important implications for understanding physical aspects of the initiation, elongation and termination of RNA-synthesis during transcription. For these and other reasons, the model will be of broader interest to the general audience working in these areas. The model explains a wide variety of data and carries within it several experimental predictions all readily testables as will be described in my talk.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Beneficial effects of adipose-derived-mesenchymal stem cells (ad-mscs) versus anti-parkinson drug in a rat model of parkinson's disease: Relationship to the molecular genetic expressions, ultrastructural and physiological responses

Naglaa K Idriss
Assiut University, Egypt

Backgrounsd: Parkinson's disease is the most common chronic progressive neurodegenerative disorder after Alzheimer's disease. The effectiveness of anti-Parkinson treatments gradually diminished by the progressive degeneration of the dopaminergic terminals. The current research investigated the effect of adipose-derived mesenchymal stem cells (AD-MSCs) versus anti-Parkinson drug in a Parkinsonism rat model.

Methods: Forty adult rats divided into 4 equal groups; Group I; Control group received the vehicle. Group II; Parkinson's disease group, received rotenone 2mg/kg daily intraperitoneally for one month. Group III received rotenone at the same previous dose then received isolated AD-MSCs on day 14. Group IV received rotenone at the same previous dose then received carbidopa/levodopa on day 14. Behavioral tests were carried out and midbrain specimens were processed for light and electron microscopy. Genetic expression of glial fibrillary acidic protein (GFAP) and nestin mRNA were assessed by real time-PCR. Lamin-B1 and vimentin genes were detected by gel electrophoresis and plasma levels of angiopoietin-2 and dopamine were measured by ELISA.

Results: Rotenone induced pronounced motor deficits, neuronal and glial alterations AD-MSCs group showed improvements in the motor function and microscopic picture. Fold change of both genes (GFAP and Nestin) were decreased significantly in AD-MSC and carbidopa/levodopa group compared to parkinson's disease. Lamin-B1and vimentin genes were highly expressed in parkinson's disease. Plasma levels of angiopoietin-2 and dopamine were significantly increased after treatment ($P<0.001$) compared to parkinson's disease.

Conclusions: Adipose-derived-mesenchymal stem cells reduced neuronal degeneration more efficiently than the anti-parkinson drug did in a parkinsonism rat model.

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Joint Event

Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

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Cartography of meningococcal meningitis in Mali: Serogroups, sequences and clones from 2006 to 2016

Coulibaly S
National Institute for Public Health Research of Bamako, Mali

Introduction: Mali, a country in the heart of West Africa, is part of the Lapeyssonnie meningitis belt. Traditionally, neisseria meningitis serogroup A is responsible for most major epidemics. After the introduction of MenAfriVac conjugate vaccine A in Mali in 2010, serogroup A declined sharply in the country.

Method: This is a retrospective study of surveillance data from 2006 to 2016 for meningococcal meningitis at the National Institute of Public Health Research in Bamako. The data collection concerned all confirmations of Neisseria meningitis by bacteriological and PCR. The data was processed and analyzed using Excel, Epi Info 3.5.4 and Health Mapper Version 4.2.

Results: A total of 5549 CSF were registered at INRSP between 2006 and 2016, of which 1122 positive or 20.22% positivity. The typical sequences and clones of neisseria meningitis circulating in Mali in the last 10 years were NmA-ST_7_CC5, NmA-ST_2859_CC5, NmC-ST_12446_CC10217, NmW-ST_11_CC11, NmW-ST_11_CC11ET17 and NmX-ST181_CC181.

Conclusion: Our data show that before introduction of "MenAfriVac" in Mali, N. meningitis serogroup A ST_7 and ST_2859 clonal complex 5 was the cause of most epidemics. After introduction there is the appearance of N. meningitis C-ST_12446 and N. meningitis X-ST_181.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Exogenous hydrogen sulfide ameliorates high glucose-induced myocardial injury & inflammation via the CIRP-MAPK signaling pathway in H9c2 cardiac cells

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Aims: Hydrogen sulfide (H_2S) is a novel signaling molecule with potent cytoprotective actions. In this study, we hypothesize that exogenous H_2S may protect cardiac cells against high glucose (HG)-induced myocardial injury and inflammation with the involvement of the CIRP-MAPK signaling pathway.

Main Methods: H9c2 cardiac cells cultured under HG conditions were transfected with siRNA and different inhibitors for detecting the effects of sodium hydrogen sulfide (NaHS) (a H_2S donor) on cell biological processes. The cardiac cell viability and LDH activity were determined by CCK-8 and LDH kit. ELISA was employed to measure the levels of inflammatory factors, while 2',7'-dichlorofluorescein diacetate (DCFH-DA) to evaluate reactive oxygen species (ROS). Mitochondrial membrane potential (MMP) was identified by rhodamine 123 staining. TUNEL staining and Hoechst 33258 staining were employed to observe cardiac cell apoptosis. Besides, we determined the expression of CIRP-MAPK signaling pathway- and apoptosis-related factors by protein immunoblot analysis.

Key Findings: HG culturing induced toxicity, LDH, higher level of inflammatory factors, ROS, MMP, and apoptosis in cardiac cells, attenuated the viability of cardiac cells, and activated the CIRP-MAPK signaling pathway. Notably, CIRP silencing aggravated the above condition. H_2S or blockade of the MAPK signaling pathway reversed the above conditions induced by HG.

Significance: The present study provides evidence for the protective effect of exogenous H_2S on HG-induced myocardial injury and inflammation in H9c2 cardiac cells and suggests that the activation of CIRP-MAPK signaling pathway might be one of the mechanisms underlying the protective effect of H_2S.

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Molecular Biology & Heart Congress 2018, Volume 29
DOI: 10.4066/biomedicalresearch-C8-023

Microfluidic-assisted formation of highly monodisperse and ordered mesoporous silica bioglasses microcapsules

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In this work developed recently an original approach using droplet-based microfluidics and the ESE techniques for the fabrication of well-defined monodisperse mesoporous silica bio glasses (85S5) hollow microspheres (microcapsules) whose size, shape and composition can be varied on demand. Our original approach allows producing highly monodisperse mesoporous and hollow silica bio glasses microspheres which is much simpler and straight forward than the time consuming and tedious standard technique as the latter relies on the use of a sacrificial hard template on which silica solidifies and forms a mesoporous shell, followed by the selective removal of the template. Our approach consists of one-step procedure as the formation of the silica shell is driven only by a control of the balance between the solvent evaporation and the silica solidification rates at the surface of the microdroplets. A step in our approach is that the control of the evaporation process is conducted outside the microfluidic channels since we use highly diluted solutions of the silica precursor (TEOS) droplets and a fluorinated oil as a carrier medium in which solvents (water ad ethanol) contained in the droplets do not solubilize. Understanding the broad variety of the observed behaviours is highly relevant to many applications. We vary the size of the microdroplets, the concentration of the precursor molecules (TEOS), the nature of the surfactant used against coalescence.

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