Scientific Tracks & Abstracts
November 29, 2017

Cell Science & Pharmacology 2017

Annual Congress on

Cell Science, Stem Cell Research & Pharmacological Regenerative Medicine

November 29-30, 2017 | Atlanta, USA

Lung cancer and liquid biopsy

Nefize Sertac Kip
Icahn School of Medicine at Mount Sinai, USA

Greatest advances have recently been made in genetics to increase our understanding of the genetic basis of human cancers. Many familial and somatic cancer genes with high-penetrance mutations have already been identified, but the situation is more complex for the contribution of the low-penetrance variants. Circulating tumor DNA (ctDNA) and cell-free DNA (cfDNA) have been shown to be elevated in the plasma and urine of patients with malignancy, offering clinical utility based on a blood draw and/or urine collection, in multiple solid tumors to improve our understanding of low-allelic fraction somatic variants. Multiple tumor type–specific next-generation sequencing assays are currently available which enable detection of somatic mutations in plasma, down to an analytical sensitivity of 0.1% in clinically relevant genes. This oral presentation will focus on the recent advances achieved in the field of liquid biopsy, along with our recent experience in Sema4 Laboratory, especially in non-small cell lung cancer (NSCLC) specimens. Paired plasma and/or urine samples with respective lung tissue appear to ensure a deeper understanding of the molecular pathogenesis of NSCLC, determine diagnosis and prognosis, predict response to therapy, identify resistance variants, assess minimal residual disease, characterize evolution of the tumor, as well as shed light onto signaling networks that orchestrate tumor behavior in a longitudinal manner, to ultimately result in superior patient care.

Speaker Biography

Nefize Sertac Kip is a Pathologist who has completed her training at Mayo Clinic, Rochester MN five years ago. She has triple boarded for anatomic, clinical and molecular genetics pathology. She is currently working as the Director of Oncology at Sema4 Genomics Laboratory at CT, which is a spinoff company out of Mount Sinai, New York.

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Microenvironment changes cell non-autonomously impose functional phenotypes in epithelial progenitors causing increased susceptibility to breast cancer

Mark A LaBarge
Beckman Research Institute at City of Hope, USA

Microenvironment is a crucial determinant of tissue and lineage specificity of cells. My lab mainly studies human mammary epithelia in the context of the aging process to explore this relationship because more than 75% of women diagnosed with breast cancer each year are over 55 years of age. We have found that mammary epithelial cells are unable to maintain strict lineage specificity with age, which results in deleterious tissue-level changes making the tissue more susceptible to malignant transformation. Two processes likely contribute to these changes and increased cancer incidence the accumulation of mutations over time, along with numerous changes to the tissue microenvironment. Microenvironment changes are essential for allowing expression of malignant phenotypes in cells and for imposing metastable non-genetic functional phenotypes in progenitors, which leads to an epithelium that is more susceptible to transformation. Microenvironment is defined as the combination of cell-cell, -ECM and -soluble factor interactions surrounding each cell in a tissue. These components exchange information with cells via a combination of physical, chemical and electrical signals, frequently activating or deactivating the same pathways triggered by oncogenes. To better understand, the genesis of age-related states of breast, we developed cell culture systems that maintain primary human epithelial cells in ways that maintain the molecular and functional fingerprint of chronological age and enable robust and repeated experimentation relevant to *in vivo*. We examine consequences of the age-related changes by examining functional responses in bioengineered cell culture substrata and *in vitro* microtissue assemblies that recapitulate aspects of *in vivo* tissues. We found that the aging process results in a continuum of different microenvironments that impose aging phenotypes that are metastable. We provide evidence that aging in epithelial progenitors and soma is cell non-autonomously communicated by microenvironment cues over at least one cell diameter.

Speaker Biography

Mark A LaBarge is a Cell Biologist with expertise in aging in the context of breast cancer. He has earned a PhD in Molecular Pharmacology from Stanford and then performed his Post-doctoral training with Dr. Mina Bissell in the field of Microenvironment Biology. During that period of training, he has focused on development of novel technologies that would enable cell-based functional interrogation of tissue microenvironments (e.g. the combinatorial microenvironment microarray (MEMA)). As a Junior Faculty at the Lawrence Berkeley National Lab, his lab focused on the role of microenvironment in age-related breast cancers. He is a recipient of the prestigious Era of Hope Award for his research in Breast Cancer and is currently a Professor at the Beckman Research Institute at City of Hope near Los Angeles.

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ID4 regulates prostate development and stem cell population in mice prostate

Jaideep Chaudhary, Dhanushka Hewa Bostanthirige, Jugal Joshi, Shravan Kumar and **Divya Patel**
Clark Atlanta University, USA

Inhibitor of differentiation 4 (Id4), a member of the helix-loop-helix family of transcriptional regulators is a novel prostate cancer (PCa) tumor suppressor. Recent studies have shown that Id4 is highly expressed in the normal prostate and decreases in prostate cancer (PCa) due to epigenetic silencing. Genetic ablation of Id (Id4-/-) in mice leads to underdeveloped prostate without the loss of Androgen Receptor (AR) expression but with re-directed activity. In this study, we demonstrate that prostates from the Id4 knockout (Id4-/-) mice show hyperplasia associated with increased stem cell population that was evident by increased Sca-1 and p63 expression. Histological analyses of adult Id4-/- mice prostate shows increased Amacr expression, a biomarker for early prostatic epithelial neoplasia (PIN) but without a clear evidence of PCa. Immuno-histochemical analysis demonstrated undetectable Nkx3.1 and Pten tumor suppressors suggesting lack of epithelial differentiation. Although Pten protein was not present in Id4-/- mice, the presence of the corresponding Pten mRNA suggested intact transcription of the gene with a possible translational or post-translation defect. These results suggested that Id4 plays a role in regulating the translation of the Pten mRNA. The results suggested that Id4-/- results in PIN lesions that may be in part due to a block in Pten translation. These data suggest that loss of Id4 can initiate PIN like lesions through multiple mechanisms such as by maintaining stemness (Sca-1) and down-regulating known tumor suppressors (Pten) and promoters of epithelial differentiation (Nkx3.1) while having no effect on AR expression and function that is reminiscent of castration resistant prostate cancer. We are currently investigating the possible mechanisms by which Id4 regulates cell fate and translational/post-translational mechanisms involved in the regulation Pten.

Speaker Biography

Jaideep Chaudhary has his expertise in Bioinformatics and Molecular Biology. He uses large datasets (microarray and NGS) to develop molecular pathways involved in cell differentiation and diseases, primarily cancer. He is passionate about teaching and mentoring Undergraduate and Graduate students. As a Scientist and an Administrator, he works across the aisle to create educational programs that help developing the new generation of productive scientists and educators.

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Computational and molecular analysis of RTN4 as novel therapeutic option for axonal regeneration

Sumaira Kanwal
COMSATS Institute of Information Technology, Pakistan

Demyelinating diseases, such as multiple sclerosis (MS), Charcot Marie Tooth (CMT) are accountable for a significant portion of the neurological disability burden worldwide, especially in young adults. Communication among the presynaptic terminus of a motor neuron and the postsynaptic membrane of a skeletal myofiber take place at the neuromuscular junction (NMJ). The fully-grown mammalian central nervous system (CNS) has a deprived aptitude to restore and return function after damage. Reticulon-4, also known as Neurite outgrowth inhibitor or Nogo, that has been identified as an inhibitor of neurite outgrowth specific to the central nervous system. A large number of genes have been associated with axonopathies and one of the emerging subgroups encodes membrane-shaping proteins with a central reticulon homology domain. This suggests that membrane-shaping disorders might be considered as a continuous disease-spectrum of the axon. The ligand shows the interaction with the residues at position of LEU133, lLE134, Pro135, LEU136, ILE155, ILE157, LEU158, ASP160, TYR161 These are the conserved residues and also found in the favored region. The analysis of RTN4 gene was performed on the DNA sample by amplifying and sequencing all the coding exons and their flanking intronic regions. However a missense mutation was found in exon 2 of RTN4 gene in the proband and later on whole family was analyzed. The pathogenicity of the mutation was checked by *in silico* analysis by using the SIFT and Polyphen. As it is well established, that RTN4 is involved in demyelinating diseases like MS so it can be directly involved in CMT 1 disease. On the other hand it can also be a genetic modifier through NgR-p75(NTR)-Mediated Signaling.

Speaker Biography

Sumaira Kanwal is working as Assistant Professor at Department of Bioscience in COMSATS institute of Information Technology, Sahiwal Pakistan. She was graduated from Kongju National University, South Korea as a human geneticist. Her basic interest in research is to find the genetic contribution primarily in Neuromuscular disorders, Neurological disorders and epigenetic of complex phenotypes of the epilepsy Genetics. Recently she did her post-doctoral research on the influence of micro RNAs on clinic-genomics of CMT1A. She has numerous international Publications on diverse subjects including Suicide, Polio eradication and role of Mitofusion2 in the development of diversity of phenotypes. She has been an invited speaker at various prestigious forums including International conference of the Genetics Society of Korea, 2016.

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Targeting FOXM1 in cancer

Andrei L Gartel
University of Illinois at Chicago, USA

The outcomes for acute myeloid leukemia (AML) have remained abysmally poor for the past 30 years. 20-40% of patients fail to achieve remission with induction chemotherapy and 50 -70% of patients who achieve a complete remission relapse within 3 years. A breakthrough in dissecting out prognostic subgroups came with the discovery of the nucleophosmin (NPM1) mutation in 40%-60% of CN-AML cases. In subsequent analyses it has been shown that AML patients with wild-type FMS-like receptor tyrosine kinase (FLT3), bearing mutated NPM1 (*NPM1*^{mut}) showed improved overall survival (OS) and relapse-free survival (RFS). We proposed that mutated NPM1 (*NPM1*^{mut}) confers this advantage in CN-AML via sequestration of FOXM1 in the cytoplasm where FOXM1 is inactive. We have demonstrated that FOXM1, an oncogenic transcription factor, co-localizes with NPM in AML cells. Mutations in NPM1 resulting in its nuclear export will drive FOXM1 to the cytoplasm where it is inactive as a transcription factor. We have shown a correlation between the expression of nuclear FOXM1 and the outcome for AML patients using primary AML samples. Stable knockdown of FOXM1 in AML KG-1 cell line resulted in increased sensitivity to this chemotherapeutic agent This data suggests that targeting FOXM1 in AML could increase sensitivity to standard chemotherapy. Knockdown of NPM1 in cancer cells led to significant down-regulation of FOXM1 suggesting that NPM/FOXM1 interaction is required for FOXM1 expression. in preliminary experiments we identified two compounds that inhibit NPM/FOXM1 interaction and suppress FOXM1 expression in AML cell lines. These compounds preclude binding of NPM and FOXM1 and modulate the suppression of FOXM1. We found that these compounds suppress FOXM1 in a variety of human cancer cell lines of different origin. Overall, our data validate FOXM1 as important target in human cancer and novel NPM/FOXM1 inhibitors that could be developed for cancer patients.

Speaker Biography

Andrei L Gartel is an Associate Professor in the Department of Medicine at the University of Illinois at Chicago and is the Academic Editor of PLOS ONE. He is the author of 89 peer-reviewed publications that include more than 20 reviews. He has more than 10000 citations and his h-index is 39. His scientific interests include: cancer, cell cycle, protein-protein interactions, regulation of CDK inhibitor p21 and regulation of oncogenic transcription factors FOXM1 and c-Myc. Specifically, his lab is interested in identification of new FOXM1 inhibitors. He received his funding from NIH, DOD and private companies/foundations.

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Talin2 mediates traction force generation and matrix metalloproteinase secretion to regulate cell invasion

Cai Huang
University of Kentucky, USA

Invadopodia, the key structures that cell invasion, are a therapeutic target for cancer metastasis. However, the molecular mechanism that regulates invadopodium maturation remains to be elucidated. Talin activates integrins and regulates cell migration, invasion and metastasis. Talin is localized to invadopodia and is essential for invadopodium formation. There are two talin genes, Tln1 and Tln2, which encode talin1 and talin2. It was widely believed that talin2 and talin1 function redundantly, but our recent studies show that talin2 regulates traction force generation, matrix metalloproteinase (MMP) secretion, invadopodium formation and cell invasion independently of talin1. In this talk, I will discuss how talin2 mediates traction force generation and MMP secretion and their role in invadopodium maturation and cell invasion. Our studies significantly advance our understanding of the molecular mechanisms by which traction force regulates cell invasion.

Speaker Biography

Cai Huang's research interest is to understand the signaling mechanisms that regulate cell migration and invasion, key steps in metastasis, that are highly dynamic processes requiring temporal and spatial regulation of integrin activation, traction force generation, focal adhesion dynamics and invadopodium formation. He has a broad background in the study of focal adhesions and cell migration, with expertise in protein phosphorylation, ubiquitination and live cell imaging.

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Novel, non-toxic rifamycin that reverse drug resistance in cancers through modulation of oxidative stress: Dual mode of action

Seyed H Mousavi-Fard, Deeann Wallis, Nian Zhou, Dwight Baker, Kimberly Loesch, Stacy Galaviz, Steve Maxwell and **James C Sacchettini**
Texas A&M University, USA

We have discovered a novel chemosensitizer (RTI-79, a rifamycin-derivative) with a broad spectrum of action that includes ovarian cancer and double and triple hit non-Hodgkin's lymphoma. RTI-79 is relatively non-toxic and has favorable *in vivo* safety and pharmacokinetic (PK) profiles. RTI-79 in combination therapies is effective in multiple drug resistant cancers in mouse models. RTI-79 works by dramatically increasing intracellular reactive oxygen species (ROS), primarily superoxide, through redox cycling. The level of ROS induction is directly correlated with drug sensitivity. Importantly, RTI-79 also triggers the unfolded protein response (UPR) that results in increased ubiquitination and loss of Nuclear factor erythroid–related factor 2 (Nrf2), the primary sensor for intracellular ROS. Thus, RTI-79 both increases ROS and squelches Nrf2's ability to respond to ROS. This unique mechanism provides a broad and novel approach for the very safe application of RTI-79 and other rifamycin, in treating drug resistant cancers.

Speaker Biography

Seyed H Mousavi-Fard has a demonstrated history of working in the higher education academia. He has advanced experimental skills in diverse fields ranging from Diagnostics, Molecular and Cellular Biology, Genetics, Cancer Biology and Virology area. He is a Research Professional with a PhD in Medical Sciences focused on Cancer Biology from Texas A&M University System Health Science Center, College of Medicine. He is effectively collaborating with several scientists with minimal supervision. He is responsible for study protocol design and maintenance, data generation and collection, resulting in expedited study completion and data output in undertaking projects.

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Epigenetic regulation of bone metastasis and osteoclast differentiation

Woojin An
University of Southern California, USA

Osteoclasts are multinucleated bone-resorbing cells and generated by the fusion of mononuclear precursor cells of the monocyte-macrophage lineage. A group of genes regulating osteoclast differentiation positively or negatively have been identified and the deregulated expression of these genes has been documented to cause various skeletal diseases. MMP-9 is a member of MMP family that has been studied mainly with respect to its role in extracellular matrix remodeling. Unexpectedly, however, we found that MMP-9 moves into the nucleus and mediates histone H3 N-terminal tail proteolysis at osteoclastogenic genes in RANKL-induced osteoclast precursor (OCP) cells. Our observation that MMP-9 knockdown abrogates H3 N-terminal tail proteolysis and osteoclastogenic gene expression is supportive of the idea that MMP-9 is the major protease responsible for H3 N-terminal tail proteolysis-mediated gene activation occurring in OCP-induced cells. Furthermore, our follow-up studies indicate that specific patterns of histone posttranslational modification are key regulators of MMP-9 protease activity toward target chromatin domains in OCP-induced cells. Cancer cells frequently spread to bone and secrete soluble signaling factors to accelerate osteoclast differentiation and bone resorption. Since chromatin signaling and regulatory factors have been implicated in epigenetic control of cancer metastasis, we also investigated their possible roles as modulators of metastatic potential of cancer cells to bone. We show that specific histone modification and histone variant tightly regulate cancer bone metastasis and osteoclast differentiation. The observed effects require epigenetic control of genes encoding secreted factors that influence cancer cell metastasis and osteoclast differentiation. Consistent with these data, osteoclastogenesis and osteoporosis are significantly affected following the administration of recombinant forms of secreted factors into mice. More interestingly, our mechanistic studies reveal that histone modification functionally interacts with histone variant to alter the expression and functional properties of metastasis-associated genes in cancer cells in the bone microenvironment.

Speaker Biography

Woojin An investigates the biological role of chromatin modification and its basic concept and mechanism of action in gene regulation and cell differentiation. By using multiple new technologies, his recent studies are mainly directed toward understanding chromatin reorganization and histone modification-mediated recruitment of novel regulatory factors to specific target genes.

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Enriched platelet factors (EnPLAF™), an alternative to PRP for regeneration and rejuvenation applications

Nathan Katz and **Nishit Pancholi**
NeoKine Laboratories Inc., USA

Platelet rich plasma (PRP) is basically a preparation of autologous concentrated platelets derived from one's own blood. Over the years, PRP has been used for multiple clinical and aesthetic applications. Multiple commercial kits are available to derive PRP and mostly all the companies selling them have their own protocols that they claim can best concentrate higher number of platelets. When it comes to deriving the PRP in clinical practice the actual number of platelets concentrated from each blood sample is more so often an unknown variable. Therefore, PRP has been unsuccessful at delivering consistent results in clinical practice especially in treatments related to pain management and tissue rejuvenation. Also, there are several drawbacks to PRP like it has no shelf life and loses activity and potency upon minimal storage and PRP injections are painful. Moreover, evidence from scientific research shows that it is the growth factors that reside in the platelet granules and which are secreted by the cells upon activation, that are responsible for inducing healing and regeneration. This is called paracrine action of cells where they release these beneficial growth factors at site of injury. The cells themselves do not survive in this external environment, but it is these growth factors and cytokines that help healing and regeneration. So, the solution to the drawbacks of PRP is a process that can concentrate just the growth factors using a standardized protocol. We have perfected this process to develop Enriched platelet factors (EnPLAF™) technology which can derive beneficial biological factors from patient's own blood platelets, but do not contain any live cells. EnPLAF™ is rich in growth factors like platelet derived growth factor (PDGF), vascular endothelial growth factor (VEGF). Since EnPLAF™ is a non-cellular, growth factors only product it can be stored, has great shelf life and is not painful to inject. Also, since EnPLAF™ is derived from the patient's own blood, there is no risk of having an allergic reaction. When used as directed, EnPLAF™ can stimulate faster healing and regeneration as well as reduce pain and inflammation.

Speaker Biography

Nathan Katz has 20 year's experience in the field of stem cells, human biology and genetics, with a solid background in commercialization of scientific achievements in this area. He is recognized as one of most experienced professionals practicing single cell micro-manipulation, nuclear transfer and genetic diagnosis. His record includes top notch peer-reviewed publications and dozens of scientific presentations. He is involved in commercial projects around the world utilizing his professional knowledge and experience, promoting new methodological approaches in private markets. Being involved daily in clinical embryology, he has been exposed to field of embryonic stem cells; adult stem cells came into focus of attention as less controversial and potentially powerful source for tissue regeneration. His personal experience and leadership qualities are key factors that led to founding and early success of Jointechlabs Inc., stem cells technology venture. He is also the Co-founder and Scientific Director of NeoKine Laboratories.

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Science of omics and its role in human health care

Nawin Mishra
University of South Carolina, USA

Omics is derived from term Ome which means to include a complete description of a system. Thus, genome is derived from gene to become genome and genomics means the complete description of all genes or DNA sequences in a cell or in an organism. Likewise, proteome is derived protein and proteomics included the description of entire proteins of a cell or an organism. Advances in Genetics led to the development of the Sciences of omics and system Biology. They provide the tools for a better understanding of human diseases and for the development of new drugs and ultimately the possibility of personalized medicine. It is now possible to determine the entire DNA sequence of a genome as well as the entire protein sequence of a proteome in any organism because of the coming of throughput technologies and Bioinformatics. Thus omics includes genomics, epi-genomics, proteomics, metabolomics and similar branches of science which describe the characteristics of a cell and its components. Several conceptual and technological advances in Genetics, Bioinformatics and Molecular Biology made possible the emergence of the science of omics. My presentation will discuss some of these advances and the role of the science of omics in human health care including precision medicine.

Speaker Biography

Nawin Mishra received his B. S (Honors) and M.S degrees from Patna University (then nicknamed as Oxford of the East) in India and Ph. D Degree from McMaster University. He received his post –doctoral training with the late Nobel Laureate Professor E. L. Tatum at the Rockefeller University. He was a Fellow for Medical Research of the Jane Coffin Child Fund of the Yale University at the Rockefeller University for two years and then Research Associate with Professor Tatum where he initiated his work in what is now called as Proteomics and Metabolomics. There he also devised the first gene transfer in a eukaryote, Neurospora crassa. Later he joined the University of South Carolina Molecular Biology Group and Chairman of the Microbiology dept in the Medical School and remained as Professor of Genetics in the Dept. of Biological Sciences. He was also a Visiting Professor at the Max Planck Institute for Molecular Biology in Heidelberg, Germany and in Genetic Institute of Greenwood, SC. In addition to a large number of articles published in leading journals, he has published two books by John Wiley & Sons of New York, one on Proteomics in 2010, this book has been endorsed by Nobel Laureate Professor Gunter Blobel.

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An *ex vivo* gene therapy strategy to treat Duchenne muscular dystrophy using a novel 3D muscle stem cell culture system and CRISPR/Cas9–mediated genome editing

Wen-Shu Wu

University of Illinois at Chicago, USA

Duchenne muscular dystrophy (DMD) is a genetic disorder caused by mutations in *dystrophin* gene and characterized by progressive muscle degeneration and a shortened life span and there is no effective treatment available. Muscle stem cells (MuSCs) exhibit robust myogenic potential *in vivo*, thus they provide a promising curative treatment for DMD and other muscle disorders. Because of scarce native adult MuSCs in muscle tissue and very limited source of biopsies from patients, *ex-vivo* expansion of freshly isolated MuSCs is highly desired to achieve a therapeutic cell dose. Here we develop a soft 3D salmon fibrin gel culture system that can selectively expand MuSCs from a small number of crude skeletal muscle cell preparations and faithfully maintain their regenerative capacity for at least two weeks in culture. Moreover, we used CRISPR/Cas9-mediated genome editing to correct the *dystrophin* gene mutation in MuSCs expanded by this system and restored the skeletal muscle *dystrophin* expression upon transplantation in mdx mice. Our studies established a reliable and feasible platform for *ex-vivo* expansion and direct gene editing of MuSCs, thus greatly advancing MuSC-based gene therapies for various muscle disorders.

Speaker Biography

Wen-Shu Wu has received his Doctoral degree in Cancer Biology from University of Texas MD Anderson Cancer Center and completed his Post-doctoral training at Dana-Farber Cancer Institute, Harvard Medical School. He has held faculty positions at several research institutes. He was an Instructor at Harvard Medical School, a Principal Investigator at Main Medical Center Research Institute and an Assistant Professor of the Program in Cell, Molecular and Developmental Biology at Tufts University School of Medicine. Before joining UIC, he was an Associate Scientist at Children's Hospital Oakland Research Institute (CHORI) and a Principal Investigator at the University of California Berkeley Stem Cell Center. He was recruited to University of Illinois at Chicago as a tenured Associated Professor in 2013.

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First report of citrus yellow mosaic virus from sweet oranges in Pakistan

Shazia Mannan
COMSATS Institute of Information Technology, Pakistan

Citrus fruits are one of the most important fruits in term of consumption as well as nutritional value. However, being such an important crop, it is infected by many diseases. One of the important diseases that can cause significant loss is citrus yellow mosaic virus disease. Citrus yellow mosaic virus causes severe mosaic disease in sweet oranges and has been reported earlier from India. In 2016, mosaic symptoms were observed in sweet oranges orchards in the Okara district at Punjab province of Pakistan. Leaf samples were collected from the 50 trees with symptoms. DNA was extracted by using sodium sulphite method. Conserved region of CYMV was amplified with PCR using primer pair (5GTGGCTTTCATCAGGTAGC, forward and 5CATGCATCCATCCGTTTCG, reverse). Approximately 640 bp Product was obtained. The PCR product was sequenced and BLASTn was performed and 96% similarity was found with the Indian isolate (GenBank accession no. AF347695). CYMV particles were propagated and transmitted to five healthy sweet orange trees through grafting. CYMV symptoms appeared after 65 days of grafting. After 80 days of transmission DNA was isolated from leaves and PCR for CYMV was conducted as described earlier. A Product of 638 bp was obtained. Thus, fulfilling Koch's postulates. To our knowledge this is the first report of CYMV in citrus in Pakistan.

Speaker Biography

Shazia Mannan from the city of Sahiwal which is located in the south of Punjab province of Pakistan. After completing her graduation from Baha-ud-din Zakariyya University, Multan, Pakistan. She got her post graduate degree in Biochemistry/Molecular biology from Quaid-i-Azam University Islamabad, Pakistan in 2001. Then she did her Doctorate from the same university in Biochemistry/Molecular Biology in 2007. After that she won Charles Wallace Fellowship of British Council and did her post doctorate research at Central Sciences Laboratories Yorkshire, UK in 2008. Then she joined department of Biosciences, COMSATS Institute of Information Technology Sahiwal campus in 2008 as Assistant Professor. Her research interest subjects are Molecular Phytopathology and drug designing. She has been working on plant pathogens including Xanthomonas Oryze pv. Oryze, phytoplasma, cotton leaf curl viruses and maize dwarf mosaic virus. She is also working on computational drug designing against cancers. She is supervising research of undergraduate and post graduate students at COMSATS Sahiwal campus

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Culture of animal cell

Sudha G Bansode
Shankarrao Mohite College, India

Cell culture has become an indispensable technology in many branches of the life sciences. It provides the basis for studying the regulation of cell proliferation, differentiation and product formation in carefully controlled conditions, with processes and analytical tools which are scalable from the level of the single cell to in excess of 10 kg wet weight of cells. Cell culture has also provided the means to define almost the entire human genome and to dissect the pathways of intracellular and intercellular signaling which ultimately regulate gene expression. Roller bottle culture is considered the first scale-up step for anchorage-dependent cells from stationary flasks or bottles. This is achieved by using all the internal surface for cell growth, rather than just the bottom of a bottle. The added advantages are that: A smaller volume of medium and thus a higher product titre can be achieved; the cells are more efficiently oxygenated due to alternative exposure to medium and the gas phase; and dynamic systems usually generate higher unit cell densities than stationary systems.

Speaker Biography

Sudha Bansode is an Associate Professor in Zoology at Shankarrao Mohite College, Akluj, Maharashtra State, India. Recently she was a Visiting Scholar at University of California, Riverside, USA. She is an active researcher & passionate teacher in India. Still she has been published above 20 research papers in International Journals & she is interested on Bone Research. Also she has honor of Distinguished Editorial Board Member of several International Journals. She is an own author of "Textbook Histological Techniques" & "Outlines of Physiology" and now she is working on another own reference book "Rhythms in Freshwater Crustaceans". She is a University recognized research guide for Ph.D. students in India. She was an invited Indian Speaker of "OXFORD SYMPOSIUM" on 27-29 August, 2014 at Balliol College, Oxford, United Kingdom. She was academic visitor of Bangkok- Thailand, Colombo-Sri Lanka, Daira-Dubai-UAE. Her recent intellectual Interaction is with many International Professional groups.

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Prostaglandin E2 receptor (EP2): A novel target to attenuate inflammation and excessive bone loss during autoimmune arthritis

Rangaiah Shashidharamurthy
Philadelphia College of Osteopathic Medicine, USA

Prostaglandin-E2 receptors (subtype EP2) are known to be activated during various autoimmune inflammatory disorders including rheumatoid arthritis (RA) and play an essential role in exacerbating the bone damage during RA. Herein, we have shown that EP2 antagonism attenuates the ongoing inflammation and excessive bone loss in collagen-induced arthritis model. Further, EP2 antagonists significantly down-regulated the serum pro-inflammatory cytokine response compared to untreated arthritic mice. We have also investigated the anti-osteoclastogenic activity of EP2 antagonists using an *in vitro* osteoclastogenesis model using mouse monocytic cell line. We observed significantly increased size and number of osteoclasts by both PGE2 and butaprost (selective EP2 agonist) compared to receptor activator of nuclear factor kappa-B ligand (RANKL) alone treated cells. We did not observe significant difference in number of osteoclasts between PGE2 and butaprost. In addition, 10μM concentration of various EP2 specific antagonists inhibited RANKL-induced osteoclast formation. Western blot analysis revealed that EP2 antagonists decreased the expression of c-Fos but not NFATc1 and NFkB, which are the master regulators of osteoclastogenesis. These data indicates the direct effect of EP2 antagonists on going inflammation and bone cells in preventing the severe bone damage implying EP2 receptors play a major role during osteoclast formation. Therefore, EP2 receptors should be explored as a therapeutic target to blunt the ongoing inflammation as well as excessive bone loss during autoimmune arthritis.

Speaker Biography

Rangaiah Shashidharamurthy is Associate Professor of Department of Pharmaceutical Sciences, PCOM-School of Pharmacy, Georgia campus. He has published more than 38 papers in peer reviewed journals and also serving as an external reviewer and editorial board member for many of the international peer reviewed journals. Dr. Shashidharamurthy research interest is in investigating the pathogenesis of chronic autoimmune/inflammatory disorders such as vasculitis and arthritis.

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Cis-vaccenic acid induces differentiation and up-regulates gamma globin synthesis in K562, JK1 and transgenic mice erythroid progenitor stem cells

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Gamma globin induction remains a promising pharmacological therapeutic treatment mode for sickle cell anemia and beta thalassemia, however, hydroxyurea remains the only FDA approved drug which works via this mechanism. In this regard, we assayed the γ-globin inducing capacity of Cis-vaccenic acid (CVA). CVA induced differentiation of K562, JK1 and transgenic mice primary bone marrow hematopoietic progenitor stem cells. CVA also significantly up-regulated γ-globin gene expression in JK-1 and transgenic mice bone marrow erythroid progenitor stem cells (TMbmEPSCs) but not K562 cells without altering cell viability. Increased γ-globin expression was accompanied by KLF1 suppression in CVA induced JK-1 cells. Erythropoietin induced differentiation of JK-1 cells 24 h before CVA induction did not significantly alter CVA induced differentiation and γ-globin expression in JK-1 cells. Inhibition of JK-1 and transgenic mice bone marrow erythroid progenitor stem cells fatty acid elongase5 (Elovl5) and Δ9 desaturase suppressed the γ-globin inductive effects of CVA. CVA treatment failed to rescue γ-globin expression in Elovl5 and Δ9-desaturase inhibited cells 48 h post inhibition in JK-1cells. The data suggests that CVA directly modulates differentiation of JK-1 and TMbmEPSCs and indirectly modulates γ-globin gene expression in these cells. Our findings provide important clues for further evaluations of CVA as a potential fetal hemoglobin therapeutic inducer.

Speaker Biography

Idowu A Aimola is a Senior Academic Staff at the Department of Biochemistry, Ahmadu Bello University, Zaria. He is also a member of the Africa Centre of Excellence on Neglected Tropical Diseases and Forensic Biotechnology at the University. He obtained his PhD in Biochemistry from Ahmadu Bello University, Zaria and was a Fulbright Scholar at the Department of Cell and Developmental Biology, Mount Sinai School of Medicine New York. He is a member of the Sickle Cell Support Society of Nigeria. His main research interest is in red blood cell developmental biology and hematological disorders. His research work involves studying molecular mechanisms, genetics, epigenetics and small molecules induction of fetal hemoglobin expression. He has scholarly publications in renowned peer reviewed international and national journals. He is an Editorial Member of some international journals.

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