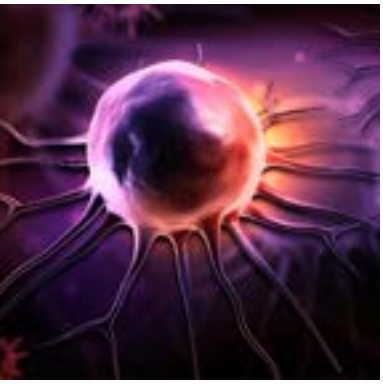
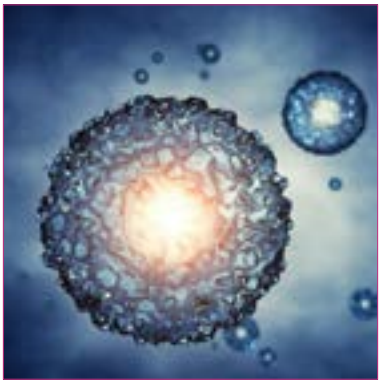
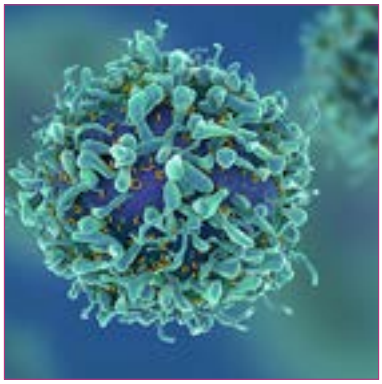
Scientific Tracks & Abstracts
October 30, 2017

Oncology and Therpeutics 2017







International Conference on

Oncology and Cancer Therapeutics

October 30- November 01, 2017 | Chicago, USA

Histone modification enzymes are critical for N-Myc-driven gene transcription and tumourigenesis

Tao Liu
The University of New South Wales, Australia

Myc oncoproteins exert tumorigenic effects by regulating the expression of target oncogenes. We previously show that the histone H3 lysine four presenters WDR5 promotes N-Myc-mediated gene transcription and tumour cell proliferation. Histone H3 lysine79 (H3K79) methylation at Myc-responsive elements of target gene promoters is a strict prerequisite for Myc-induced transcriptional activation. *DOT1L* is the only known histone methyltransferase that catalyses H3K79 methylation. Here, we showed that N-Myc up-regulated *DOT1L* mRNA and protein expression by binding to the *DOT1L* gene promoter. Knocking down *DOT1L* reduced mRNA and protein expression of the N-Myc target genes *ODC1* and *E2F2*. *DOT1L* bound to the Myc Box II domain of N-Myc protein, and knocking down *DOT1L* reduced histone H3K79 methylation and N-Myc protein binding at the *ODC1* and *E2F2* gene promoters and reduced neuroblastoma cell proliferation. Treatment with the small molecule *DOT1L* inhibitor SGC0946 reduced H3K79 methylation and proliferation of *MYCN* gene-amplified neuroblastoma cells. In mice xenografted with neuroblastoma cells stably expressing doxycycline-inducible *DOT1L* small hair-pin RNA, ablating *DOT1L* expression with doxycycline significantly reduced *ODC1* and *E2F2* expression, reduced tumor progression and improved overall survival. In addition, high levels of *DOT1L* gene expression in human neuroblastoma tissues correlated with high levels of *MYCN*, *ODC1* and *E2F2* gene expression, and independently correlated with poor patient survival. Taken together, our data identify *DOT1L* as a novel co-factor in N-Myc-mediated transcriptional activation of target genes and neuroblastoma oncogenesis, and *DOT1L* inhibitors as novel anticancer agents against *MYCN*-amplified neuroblastoma.

Speaker Biography

Tao Liu is originally trained as a Medical Practitioner specializing in Neurology and an Associate Professor. He studied for a PhD degree at The University of New South Wales, Sydney, Australia on the role of inflammatory mediators in chronic pain due to nerve injury. He then worked on the role of MIC-1, a new member of the transforming growth factor beta superfamily, in cancer cell proliferation, survival/apoptosis and metastasis at St. Vincent's Centre for Applied Medical Research, Sydney, Australia. He is an Associate Professor and joined Children's Cancer Institute Australia for Medical Research ten years ago. He has been focusing his research on the roles of histone deacetylases, histone demethylases, and histone methyltransferases, BET bromodomains proteins and long noncoding RNAs in modulating gene transcription and tumourigenesis, and the roles of histone deacetylase inhibitors, histone methyltransferase inhibitors and BET bromodomain inhibitors as anticancer agents *in vitro* and in mouse models of cancer.

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Notes:

Developing an advanced formulation of curcumin for targeted therapy of triple negative breast cancer

Pegah Varamini
University of Sydney, Australia

Breast cancer is the most common malignancy and the second leading cause of cancer-related death among Australian women despite existing progress in the development of novel therapeutic strategies. Triple-negative breast cancer (TNBC) accounting for 10-17% of all breast carcinomas, is an aggressive histological subtype. It represents an important clinical challenge because these cancers do not respond to the available targeted agents. Thus, there is an urgent demand for specific therapies that target other receptors that are overexpressed in TNBCs. We have designed and synthesized a novel drug delivery system, which targets curcumin to the breast cancer cells through a ligand of luteinizing hormone-releasing hormone (LHRH) receptors. LHRH receptors are overexpressed in breast cancer cells including MBC and TNBC cells while they are not expressed detectably in most visceral organs. We have taken advantage of this differential receptor expression by attaching a new derivative of the LHRH peptide (as a targeting moiety) to the outer surface of novel polymer nanoparticles. These nanoparticles encapsulate curcumin, a non-toxic plant extract that has recently attracted much attention in medicine due to its remarkable therapeutical actions. It is called the next generation multi-purpose drug and is the active constituent of the Indian spice turmeric. However, it suffers from a very poor metabolic stability and bioavailabilty due to low water solubility. We have used an advanced formulation strategy to overcome hurdles to make it effectively used as a medication and also target it specifically to the TNBC cells via LHRH receptors.

Speaker Biography

Pegah Varamini is an early career Researcher, Lecturer and Group Leader in Cancer Theme within the Faculty of Pharmacy. She is the Leader of Breast Cancer Targeting-Drug Delivery Group. She was awarded the prestigious National Breast Cancer Foundation (NBCF) fellowship in Jan 2016. She completed her Doctorate degree in Pharmacy (PharmD) in May 2005 and was awarded her PhD degree in Medicinal Chemistry and Pharmacology in December 2012 (UQ, Australia). She has won 2012 Dean's Award for Research Higher Degree Excellence. Her work was selected by the Australian Academy of Science in August 2016, resulting in her personal presentation at the inaugural Falling Walls Lab in Canberra (a gathering of 25 selected Australian and New Zealand researchers, entrepreneurs, engineers and innovators).

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Hidden correlations and symmetries determine per-residue interaction free energy contributions of protein: Small molecule/biomolecule binding

Lawrence J Williams
Rutgers University, USA

Protein interaction free energy is the lynchpin to understanding, identifying, and targeting cancer. Despite the common practice of describing key interactions in terms of ionic, polar, hydrogen bonding, and hydrophobic contacts, the interactions are too numerous, varied, weak and delicately balanced to allow meaningful prediction of per-residue contributions to interaction free energy. All-atom physics-based (molecular dynamics and free energy perturbation) simulations have struggled to provide per-residue contributions as well. The ideas presented here constitute an alternative physics-based approach to describe protein interaction energies without resorting to atomistic methods or rationales. Dilation symmetry, simple accommodations of protein backbone fluctuations and renormalization-based approaches provide a straight forward description of the individual and correlated effects of per-residue contributions to interaction free energy. This approach enables protein residues to be mapped according to how 'hot' or 'sticky' each residue is. Protein interiors are dominated by hot residues and exteriors are dominated by cool residues. Hot patches on protein surfaces correspond to the substrate binding surfaces of enzymes (Bcl kinase), protein-protein interfaces (Mcl-1/Bim), and protein-peptide interfaces (MHCs). Additionally, the model can be used to classify mutations as primarily impacting ground state conformations (Class I mutation) or ground and/or excited state conformations (Class II mutation) and to compute protein stability (*ddG*, lysozyme). The data suggest that sequence space – so abundant in the wake of the genomic revolution – can be converted into energy space and that it may be possible to navigate and interpret genomic and protein structure data directly in terms of interaction energy signatures.

Speaker Biography

Lawrence J Williams completed his PhD at the University of Arizona and held Post-doctoral fellowships at MIT and Memorial Sloan-Kettering where he studied molecular structure and synthesis of natural and engineered peptides and tumor-associated glycopeptides for immune activation. His independent research has focused on developing synthetic methods and strategies to understand structure, reactivity, materials and biological function of complex organic molecules, especially natural products, peptides and proteins. Recently, he has worked in the fields of antibody drug conjugates, biomarker diagnostics and protein biophysics.

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Notes:

ZEB1 deletions in brain cancer

Lincoln A Edwards
Weill Cornell Medical College, USA

Glioma stem cells (GSCs) drive the propagation of glioblastomas and can affect patient survival by imparting the virulence of unabated tumor growth through cancer stem cell self-renewal with a resistance to GSC differentiation. How GSCs achieve these characteristics are poorly understood. We have identified ZEB1 as a mediator of resistance to differentiation, and stem cell self-renewal. IFN-γ which causes ZEB1 induction aborts these GSC characteristics. We show that ZEB1 negatively regulates the stem cell self-renewal factor LIF, through newly identified E-box binding sites within the LIF promoter. Targeted suppression of ZEB1 resulted in the induction of LIF commensurate with GSC self-renewal and an inhibition to GSC differentiation. Interrogation of over 500 patient glioblastomas along with primary patient GSCs identified a significant number of glioblastoma patients harboring a ZEB1 deletion and frequent loss of heterozygosity (LOH). These findings are not in line with the present literature, which suggests that ZEB1 expression increases tumorigenicity. Surprisingly, our findings illustrate that the loss of the ZEB1 gene is common in glioblastoma and that ZEB1 loss is associated with propagation of the glioma stem cell population. These findings link ZEB1 loss to stemness with important implications for prognosis and treatment.

Speaker Biography

Lincoln A Edwards completed his PhD at the University of British Columbia, (Canada) and his Post-doctoral studies from the National Institutes of Health, National Cancer Institute in the Department of Neuro-Oncology. He then went to the Department of Neurosurgery at Cedars-Sinai Medical Center serving as a Research Scientist before moving to New York where he is currently an Instructor of Neuroscience, Neuro-Oncology at Cornell University, Weill Cornell Medical College. He has been serving as a review board member for the journal Frontiers of Oncology and has published in journals like *JNCI, Cancer Cell, Scientific Reports and Molecular Cancer Therapeutics*. His work has led to the initiation of clinical trials for the treatment of brain cancer.

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Notes:

Targeting FOXM1 in cancer

Andrei L Gartel
UIC, 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 major 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 (NPM1mut) showed improved overall survival (OS) and relapse-free survival (RFS). We proposed that mutated NPM1 (NPM1mut) 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 result in its nuclear export which 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, PhD, 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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Notes:

Recent advancements in cancer genomics

Shrikant M Mane
Yale Center for Genome Analysis, USA

The Yale Center for Genome Analysis (YCGA) is a state-of-the-art DNA Sequencing Center launched in 2010 to provide an open access centralized facility for services, equipment and expertise required for carrying out large-scale sequence analysis studies. Our group foresaw scientific opportunities for the development and use of exome sequencing in Mendelian genetics and was the first to develop the method for exome capture on the NimbleGen/ Roche platform. We were also the first to demonstrate the biological utility of exome sequencing for clinical diagnostic applications. Currently, YCGA is a part of the NHGRI supported Yale Center for Mendelian Genomics that uses NGS and computational approaches to discover the genes and variants that underlie Mendelian conditions. In the last four years, the use of next-gen sequencing has led to the publications of >375 articles in peer reviewed journals, including >40 in high profile journals such as Science, Nature, Cell, New England Journal of Medicine and Nature Genetics reporting new variants in various disorders, including cancer, hypertension, autism, several types of cancers, Gaucher disease, skin disorders, and cortical malfunctions, all using exome analysis. The presentation will focus on recent biomarker discoveries in cancer and clinical applications.

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Notes:

Metabolic flux analysis of mantle lymphoma cells upon Bruton tyrosine kinase inhibition

Seung-Cheol Lee
University of Pennsylvania, USA

Ibrutinib, a Bruton tyrosine kinase inhibitor, is being popularly used for treatment of relapsed/refractory mantle cell lymphoma (MCL) as well as chronic lymphocytic leykemia/ small lymphocytic lymphoma (CLL/SLL). We are working on metabolic pathway analysis of MCL cells upon ibrutinib treatment using novel 13C NMR and mass spectrometry technique and flux analysis methods. Ibrutinib sensitive MCL-RL cells and ibrutinib less sensitive Jeko-1 cells were studied. Cells were incubated in the medium containing 1,6-13C glucose, 1,2-13C glucose or U-13C glutamine for 8 hours to reach steady state of labeling enrichment of intracellular metabolites, and 13C labeling information was obtained using NMR or liquid chromatography mass spectrometry (LC-MS) techniques. Bonded cumomer and fragmented cumomer analysis methods were employed for analysis of NMR and LC-MS data. Significant changes were observed in the fluxes of glycolysis, glutaminolysis, reductive carboxylation and fatty acid syntheis in MCL-RL cells after ibrutnib treatment while less or no changes in JeKo-1 cells. Glycolytic flux changed to 1/4 in MCL-RL cells while to 1/2 in in JeKo-1 cells. Glutaminolysis changed by 90% in MCL-RL cells while no change in JeKo-1 cells. When a glutaminase inhibitor, CB-839, was added to medium, JeKo-1 cells exhbited remarkable response in cell growth while MCL-RL cells did not. This study demonstrates that metabolic flux analysis provides an important clue of what pathway is being affected and what pathway is not to specific kiniase inhibitors and which metabolic pathway should be further targeted with additional drugs.

Speaker Biography

Seung-Cheol Lee has finishes his BS (Physics) from Korea Advanced Institute of Science and Technology in 1993 and MS (Solid State Physics) from Korea Advanced Institute of Science and Technology in 1995. After that, he did his Post-Graduate Training (Postdoc in Biophysics) from Korea Basic Science Institute (2001-2004) and Postdoc in Radiology from University of Pennsylvania (2004-2007). He has expertise in MRI and MRS of cancer cells, animal models and human patients' prediction and early detection of therapeutic response in non-Hodgkin's lymphoma.

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Notes:

Onco-histone H3.3K36M reprograms the epigenome of chondroblastomas

Fang Dong
Columbia University, USA

With the expansion of cancer genome sequencing, many chromatin-regulating genes are found mutated. The surprising finding is that of histone proteins, the basic structural components of the human chromatin and is mutated in a variety of cancers. Specifically, a somatic histone H3.3 lysine 36 to methionine (K36M) mutation is identified in over 90% of chondroblastomas. In human genome, there are 13 genes encoding canonical histone H3 proteins that differ from two H3.3 genes by four or five amino acids. H3K36 is conserved among all these histone proteins. Therefore, it is unknown how mutations at one allele of 15 histone H3 genes are linked to Tumorigenesis. We have shown that the levels of H3K36 di- and tri-methylation (H3K36me2/me3) are reduced dramatically in chondroblastomas and chondrocytes bearing the same the genetic mutation as chondroblastomas. Mechanistically, we show that H3.3K36M mutant proteins inhibit enzymatic activity of some, but all H3K36 methyltransferases. In addition, chondrocyte cells with H3.3K36M mutant proteins exhibit several hallmarks of cancer cells. Based on these studies, we propose that H3.3K36M mutant proteins alter epigenomes of specific progenitor cells, which in turn lead to cellular transformation and tumorigenesis.

Speaker Biography

Fang Dong is an Associate Research Scientist in Institute of Cancer Genetics of Columbia University. He has expertise in evaluation and passion in improving the health and wellbeing. His open and contextual evaluation model based on responsive constructivists creates new pathways for improving healthcare. He works under the Leadership of Dr. Zhiguo Zhang.

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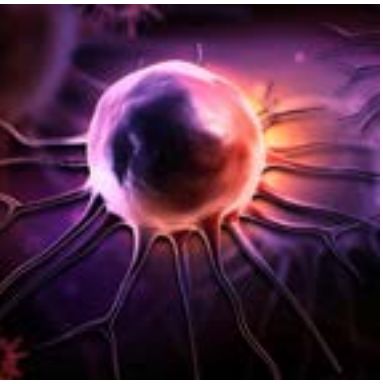

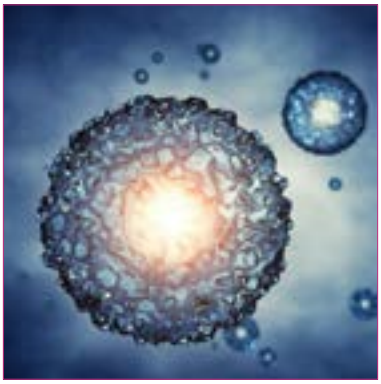

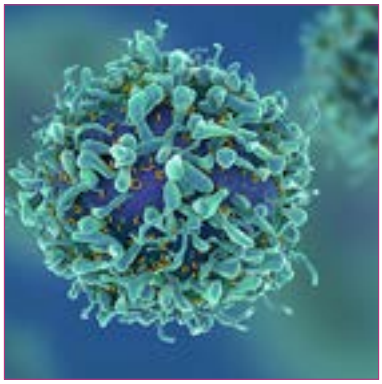

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Comparison of efficacy and toxicity of carboplatin or cisplatin based chemo-radiotherapy treatment among elderly locally advanced non-small cell lung cancer patients

Mohamed Sheta, Omnia Abd –El-Fattah and **Hanan A Alshenawy**
Tanta University Hospital, Egypt

Concurrent chemo-radiotherapy (CCRT) is the standard management for locally advanced non-small cell lung cancer (LA-NSCLC), but the definite choice of carboplatin or cisplatin-based chemo-radiotherapy as a treatment for elderly patients with LA-NSCLC has not yet been defined. In this study, we compared the efficacy and toxicity of Carboplatin vs. Cisplatin-based (CCRT) for elderly patients with LANSCLC. A study was conducted on 50 elderly patients (> 65 years) where 25 patients received. Carboplatin (area under the curve [AUC] 2) and Paclitaxel (45 mg/m2) administered on days 1, 8, 15, 22, 28, and 35 over a 6-week period; concurrent thoracic radiotherapy (RT) followed by two cycles of Paclitaxel 200 mg/m2 and Carboplatin AUC 6. The other 25 patients received 50 mg/m2 of Cisplatin administered on days 1, 8, 29, and 36, and 50 mg/m^2/day of etoposide delivered on days 1–5 and 29–33; concurrent thoracic RT followed by Cisplatin 50 mg/m^2 and etoposide 50 mg/m2 for two additional cycles. Both groups received thoracic RT dose ranged from 60 Gy to 70 Gy in 2 Gy per fraction, five fractions a week over six to seven weeks. Both Carboplatin and Cisplatin-based regimens had the similar overall survival but the Carboplatin is less toxic when combined with RT in elderly LA-NSCLC treated patients.

Speaker Biography

Mohamed Sheta is a Lecturer of Clinical Oncology and Nuclear Medicine and Consultant of Clinical Oncology at Nile Hospital for Medical Insurance, Cairo in Tanta University. He has published his papers in reputed journals.

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Notes:

The NAE inhibitor MLN4924 inhibits the nucleotide excision repair pathway to enhance the efficacy of cisplatin treatment in both BRCA1-proficient and –deficient triple negative breast cancers

Alo Ray
Ohio State University Medical Center, USA

Development of new drugs for TNBC (Triple negative breast cancer) is urgently needed due to lack of therapy. NEDD8-activation enzyme (NAE) inhibitor, MLN4924, is currently in clinical trials. We show that the TNBC cells show higher sensitivity compared to other breast cancer subtypes to MLN4924. Furthermore, MLN4924 enhances the cytotoxicity of the approved TNBC chemotherapeutics cisplatin but not doxorubicin. Importantly, both BRCA1-proficient and –deficient cells show re-replication with >4N DNA content accumulating cells in S phase leading to apoptosis and senescence. However, the BRCA1-deficient cells show less re-replication and a higher fraction of cells progressed to G1 undergoing more senescence. The re-replication is attributable due to the CDT1 accumulation via blocking its degradation which triggers DNA damage. Mechanistic investigation of increased sensitization upon MLN4924/cisplatin co-treatment revealed that neddylation substrates, nucleotide excision repair (NER) proteins, DDB2 and XPC play a key role. Neddylation of CUL4 helps DDB2 and XPC ubiquitination, which is essential for cisplatin-DNA adducts repair by NER. As expected, DDB2 accumulated and the XPC ubiquitination reduced upon MLN4924 treatment. Surprisingly, the reduced ubiquitination of XPC promotes decrease in XPC level. The alterations in the DDB2 and XPC ubiquitination and their protein levels inhibit NER by affecting the stoichiometry of repair protein assembly at the damage sites, and consequently the MLN4924/cisplatin co-treatment accumulated more cisplatin-DNA adducts. MLN4924 treatment showed activation of both ATR-Chk1 and ATM-Chk2 cell cycle checkpoint pathways, but the cells cannot repair the extensive DNA damage. Since MLN4924/cisplatin treatment shows sensitization of both BRCA1-proficient and –deficient TNBCs to cisplatin compared to PARP inhibitor, which sensitizes only BRCA1-deficient TNBC, this combination will have greater efficacy for all TNBC patients. We demonstrate a novel mechanism of MLN4924 and cisplatin sensitization and provide a strong rationale for the clinical investigation of this combination in highly drug resistant TNBC.

Speaker Biography

Alo Ray has extensive experience in the area of DNA damage repair, cell cycle checkpoint, and DNA replication all of which play a crucial role in cancer development and progression. Her research is aimed to investigate the molecular mechanisms and biological functions of basic cellular processes required for maintaining genomic stability and integrity with a future goal of developing therapeutic interventions of cancer. She is using multidisciplinary approaches to target the DNA damage response repair and cell cycle checkpoint pathways with a goal to enhance the chemo-and radio-therapy of cancer patients. She has authored several peer-reviewed high-profile journals such as *Nature Genetics, PNAS, Molecular Cell, NAR, MCB*, and *DNA repair*. She was granted the Leukemia & Lymphoma Society Special Fellowship Award. Additionally, she was granted several grants as Principal Investigator and Co-Principal Investigator from American Cancer Society, OSU Cancer Center and NIH.

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Notes:

Chronic inflammatory diseases, malignancies and nutritional intervention

Helieh S Oz
University of Kentucky, USA

Chronic inflammatory diseases including periodontitis, hepatitis, pancreatitis, gastrointestinal complications which can further lead to malignancies. Inflammation and immune response are required for the tissue defense, regeneration and healing process. Yet, exaggerated and chronic inflammation can advance to life-long debilitation, loss of tissue function and organ failure. Despite the millennial advancements in diagnostic technology and therapeutic modalities, there remains no effective cure for patients who suffer from inflammatory diseases and malignancies. Therefore, over 40% of patients with inflammatory complications seek some form of complementary and alternative medical (CAM) agents as adjunct therapeutic modalities, to alleviate symptoms and possibly to prevent outcomes of inflammation, whether or not to consent their clinicians. There is not sufficient scientific information or international regulatory enforcements regarding the most available CAM agents which some may interact with patients' current therapies with severe consequences. One of the most investigated agents is Green tea and it polyphenols (GrTP) with potent antioxidants effects. GrTP have important roles in regulating vital signaling pathways comprise transcription nuclear factor-kappa B mediated I kappa B kinase complex pathways, programmed cell death pathways like caspases and B-cell lymphoma-2 and production of cyclooxygenase. This presentation will review inflammatory disease and malignancies and explore mechanism of actions for protective effects of nutritional interventions and some reported adverse effects as well as some food safety applications.

Speaker Biography

Helieh S Oz has DVM and MS (U. IL); PhD (U. MN) and clinical translational research certificate (U. KY Med Center). She is an active member of American Association of Gastroenterology (AGA) and AGA Fellow (AGAF). She is a microbiologist with expertise in inflammatory and infectious diseases, innate and mucosal Immunity, drug discoveries, pathogenesis, and micronutrient. She was PI on different NIH and NIH-NCCAM grants to investigate Chronic Inflammatory Diseases and nutritional interventions. She has over 90 publications in the areas of chronic inflammatory disorders (pancreatitis, hepatitis, colitis and periodontitis) and micronutrients. She serves as the Lead editor for some specials issues and book Chapters.

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Notes:

Ayurvedic management of myeloid leukemia (myelo proliferative disorder) vis-à-vis-a clinico-theoretical review

Nishant Shukla
Shree Balahanuman Ayurved Mahavidyalaya, India

Myeloid leukemia (ML) is very commonly myelo proliferative disorder presented with leukocytosis usually >50K. It is clinically presented with varied symptom like anemia, bleeding symptoms, etc. In recent years disease having increased viscosity are managed and named as hyper viscosity state - one common pathological change of observed due to increase in cellularity. This condition is named as lohitabhisyanda and in CML myeloid leukocytes increases. This is the characteristic change observed in raktapitta. Thus it is kept at equality. Triyak raktapitta is advanced phase of the raktapitta where the blood oozes out even from the romakupa. This is tridoshaja condition and bears poor prognosis, because of non-effectiveness of therapeutic purification processes or limited availability of tridosha shamak drugs. It is observed that on the basis of the clinical experience that the chronic myeloid leukemia (CML) is managed effectively with providing good symptom relief and improving general wellbeing. Patient diagnosed CML attending clinic of Dr. Shukla were treated with ayurvedic treatment with combination of Shatavari (Asparagus), Yastimadhu and Dhatriloha. The clinical progress of the patients were recorded and based on their clinical progress and periodical pathological reports shown improvement in pathological parameters TLC reduced markedly from 21500/cumm to 7390/cumm, Hemoglobin increased from 7.58Gm/dl to 11.21gm/dl, marked clinical improvement is observed in all cases general wellbeing was marked improved. The average life span of the patient is believed to be five to ten years, but it is observed that the life span of the patients managed with ayurvedic live even more than fifteen years. Tools for evaluation for the life expectancy are yet not very much perfect, so calming the improvement of life expectancy is not justified, but it is certain that the wellbeing of the patients improves with symptom relief.

Speaker Biography

Nishant Shukla has completed his MD and PhD (Ayu) from IPGT&RA, GAU, Jamnagar. He worked as Lecturer in SGAM, GAU, Jamnagar in Kayachikitsa (Eternal Medicine) for approximately 8 years. He has good academic records and stood university 2nd in Post-graduate studies. He presented more than 20 papers in international and national seminars. He was invited to deliver speech four times in seminar organized by Rastriya Ayurved Vidyapith, New-Delhi and Royal Asiatic Society, Kolkata. More than 10 scientific papers are published in internal peer-reviewed journals and he is author of two ayurvedic books. He is a renowned Clinician and has rendered medical service through camps near Jamnagar. He served in medical camps organized by Borivali Gujarati Seva Mandal, Mumbai thrice.

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Notes:

Targeting cancer stem cell by oncolytic viruses

Faris Farassati
Midwest Biomedical Research Foundation, USA

According to the recent models for tumor development, a fraction of cells within tumors play the role of stem cells (i.e. cancer stem cells or CSC) and are in charge of giving rise to all other kinds of cells needed to maintain tumor integrity. An effective therapy must be able to destroy CSCs. Lack of such feature in our current chemotherapy agents leads to the eventual tumor relapse. There are no pharmacological agents currently available for specific targeting of CSCs. In this talk, we present our data on targeting CSCs from both preventive and therapeutic points of view. This is accomplished by developing Oncolytic Viruses that can target CSCs in a cell specific manner. Oncolytic viruses are novel tools for targeting human malignancies that are capable of infecting cancer cells while sparing normal cells. With the FDA approval of the first member of this family in 2015 for treatment of melanoma, we are entering a new phase in the use of these agents in clinical practice. A range of oncolytic viruses are used for this purpose such as genetically engineered viruses (e.g. Herpes and Adenoviral models) or viruses that are used in their natural form (e.g. Reoviruse). Other than killing cancer cells by infection (the signature effect of an oncolytic virus), an important anti-cancer mechanism of these agents is their stimulatory effects on anti-tumoral immunity. This is caused by release of a host of viral elements as well as cancer associated antigens. One of the focus areas of Dr. Farassati's team is to develop oncolytic viruses that can target and destroy CSCs which will be reviewed in this presentation.

Speaker Biography

Faris Farassati is a Translational Cancer Scientist whose research is focused on development of novel anti-cancer therapeutics. His research team focuses on intervention with pro-oncogenic cell signaling machinery in order to treat human malignancies. Therapeutic targets which are identified to be "Cancer-Specific" are pursued by both gene and drug therapy strategies. Transcriptional targeting of Oncolytic Viruses is a major focus of research of his group.

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Notes:

Chronic inflammatory mediators in progression of oral cancer

Shrihari
Krishna Devaraya College of Dental Sciences and Hospital, India

Oral cancer is a major cause of mortality and morbidity across the world. Because of extensive use of carcinogenic products such as tobacco, alcohol and some cases are due to viruses such as human papilloma virus induced oro- pharyngeal cancer. These carcinogens induce inflammatory changes in the inflammatory microenvironment of oral mucosa. chronic inflammatory mediators in tumor microenvironment are innate and adaptive immune cells and their secreting factors such as chemokine's, cytokine's, growth factors, and proteolytic enzymes activates transcriptional factors (NF-KB, STAT-3) expressed by immune cells and tumor cells promotes malignant changes.

Speaker Biography

Shrihari is working in the Department of Oral Medicine and Radiology at Krishna Devaraya College of Dental Sciences and Hospital, Bangalore, India. He has published many papers in reputed journals.

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Notes:

Femtomedicine in cancer: Discovery of new antitumor molecules for natural targeted chemotherapy and radiotherapy of cancers

Qing-Bin Lu
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The conquest of cancer continues to pose great challenges to medical science. There is a compelling need for innovative cancer research integrating biomedical sciences with physical sciences in order to ultimately conquer cancer. *Femtomedicine (FMD)*, which integrates femtosecond time-resolved laser spectroscopy with biomedical sciences, was recently coined to advance fundamental understanding and therapies of human diseases notably cancer. Our studies in FMD have led to the discoveries of the reductive damaging mechanism in DNA and living cells and the molecular mechanisms of action of existing anti-cancer agents. These have offered unique opportunities to develop new effective drugs for high-performance therapy of cancer. We have particularly found a new class of non-platinum-based anticancer compounds (called *FMD compounds*) for natural targeted chemotherapy and radiotherapy of a variety of cancers, e.g., cervical cancer, ovarian cancer, head and neck cancer, breast cancer, lung cancer, etc. Treatments of various cancer cells *in vitro* and *in vivo* mouse xenograft models with *FMD compounds* led to effective chemotherapy and enhanced radiotherapy, while the compounds themselves induced no or little systemic and radiation toxicity. These compounds are therefore a new class of potent antitumor agents that can be translated into clinical trials for targeted chemotherapy and radiotherapy of multiple types of cancer. The results also show that FMD can bring breakthroughs in understanding fundamental biological processes and lead to advances in cancer therapy. This presentation will discuss some progress in this new frontier—*FMD in Cancer.*

Speaker Biography

Qing-Bin Lu received his PhD from the University of Newcastle, Australia, and completed his postdoctoral positions at Rutgers University, University of Sherbrooke and California Institute of Technology. He is a full professor and a University Research Chair at the University of Waterloo, Canada. His research in *femtomedicine (FMD)*, which fuses ultrafast laser techniques with biomedical sciences to advance fundamental understanding and treatment of human diseases, notably cancer, has led to the discoveries of novel anti-cancer agents for targeted chemotherapy and radiotherapy of multiple types of cancers. He has published over 50 papers in prestigious journals.

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Scientific Tracks & Abstracts
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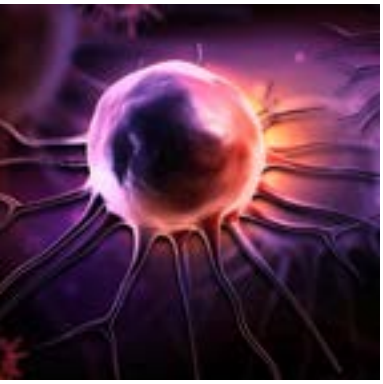
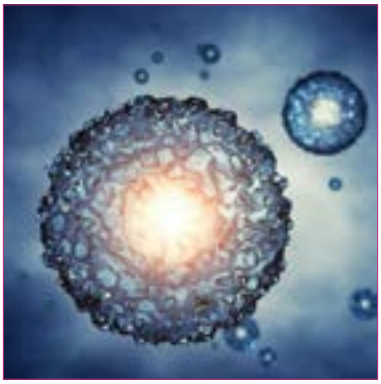
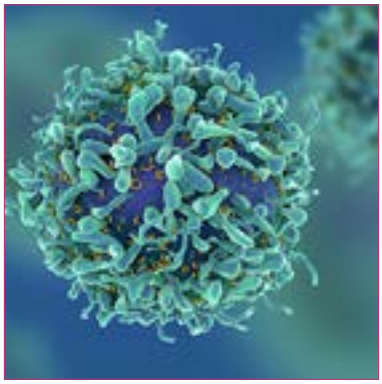
Oncology and Therpeutics 2017







International Conference on

Oncology and Cancer Therapeutics

October 30- November 01, 2017 | Chicago, USA

Novel immunotherapy for non Hodgkin lymphoma using vaccine of cancer cells with anti-IL-19 antibodies

Manal Mohamed Saber
Minia University, Egypt

Background: Non-Hodgkin's lymphoma (NHL) is a group of lymph proliferative malignant disorders with heterogeneous histological and clinical features. Higher IL-19 serum levels were associated with treatment failure and relapse in NHL. Despite major advances in treatment, a proportion of patient relapses highlighting the need for new immunotherapy.

Objective: To develop a novel cancer vaccine expressing anti-IL-19 mAbs in NHL

Methods: The antitumour effect of the vaccine was verified by therapeutic animal experiments *in vivo*. The antitumour mechanism was analysed using flow cytometry, immunohistochemistry, immunofluorescence, ELISA and T-lymphocytes assays.

Results: Novel cancer vaccine inhibited tumour growth and extended the survival of the mice compared to vaccine-untreated group. A strong T cell response by more CD4-positive T cells, CD8-positive T cells, NK cells and tregs appeared in the vaccine-treated group. Accompanying the antitumor responses, there were increases in IFN-γ and IL-12. Furthermore, novel cancer vaccine decreased the tumour-induced apoptosis of T cells.

Conclusion: This study has demonstrated a novel promising cancer vaccine in tumour immunotherapy.

Speaker Biography

Manal Mohamed Saber has completed her PhD from Nottingham University. She is an Associate Professor of Clinical Pathology, Minia University, Egypt. She has published papers in peer reviewed journals and has been serving as an Editorial Board Member of others.

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Identification of novel tumor suppressor through methods of reverse genetics

Zhenglun Zhu[1], Hong Gao[1, 2, 3], Yi Le[1] and **Ronald Bleday[1]**
[1]Brigham and Women's Hospital, USA
[2]Harvard Medical School, USA
[3]Tufts Medical Center, USA

Identifying novel tumor suppressor holds the promise for improving cancer treatment. Forward genetic screening has been the primary method for identifying tumor suppressors and oncogenes. While, the potential of reverse genetics in deciphering genes critical for tumorigenesis has been widely expected, the application of the approaches has reminded limited. By exploring the molecular mechanisms underlying dorsoventral axis formation during early vertebrate embryogenesis, we identified the human homeobox protein VentX as a novel tumor suppressor. We demonstrated that VentX exerts its function through mechanisms of anti-proliferation and pro-differentiation. Importantly, we found that VentX expression can be induced by chemotherapeutic agents and caused apoptosis of cancer cells in p53-independent manner. Taken together, our study revealed the application of reverse genetics in identifying novel tumor suppressors and the role of VentX as a novel therapeutic target in cancer treatment.

Speaker Biography

Zhenglun Zhu is an expert in Fundamental Biology and Translational Medicine. He is the elected member of the prestigious American Society of Clinical Investigation (ASCI). He discovered novel principle governing cell fate determination during early embryogenesis and translated the principle into identifying novel tumor suppressor.

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Double rolling circle replication (DRCR): A mode of amplification of oncogene as well as drug-resistant genes and replication of HSV and chloroplast DNA

Takashi Horiuchi
Tokai University School of Medicine, Japan

It is well established that eukaryote nuclear chromosomes are duplicated from multiple origins of replication. It remains a mystery, however, how genomes of some viruses, such as HSV (Herpes simplex virus) and Baculovirus, or chloroplasts, are replicated. We have found recently that (i) double rolling circle replication (DRCR), originally found responsible for replication of yeast 2µ plasmid DNA, can lead to amplification of oncogenes as well as drug resistance genes, and (ii) that DRCR is highly recombinogenic. In addition, we will present our model, based on these findings, that DRCR is involved in DNA replication of HSV-1, chloroplasts and some mitochondria. The model could explain how DRCR contributes to replication-recombination coupling of HSV, and also how it promotes amplicon shortening during gene amplification.

Speaker Biography

Takashi Horiuchi has received his BS degree in 1969 from Department of Agriculture, Kyoto University, and Kyoto, Japan. He did his MS degree in 1971 from Department of Agriculture, Kyoto University and another MS degree in 1973 from Department of Science, Kyoto University. He completed his PhD degree in 1980 from Department of Science (Institute for Virus Research), Kyoto University. He received Kihara Prize for "Identification and characterization of DNA replication fork blocking event".

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Neuropathic pain in prostate cancer with bone metastasis: Therapeutic approach

Beatriz Losada Vila, Maria Victoria De Torres Olombrada, David Gutiérrez Abad, Laura Rodriguez Lajusticia and **Juan Antonio Guerra Martínez**
Hospital University of Fuenlabrada, Spain

Pain is a frequent symptom in the evolution of the cancer in patient often acquiring chronic character as a consequence of the progression of the disease. More than 70% of patients are not under control. In this case review we will expose the management of different types of pain, side effects and education, taking also into account the fragility of the elderly patient.

Speaker Biography

Beatriz Losada Vila has completed her studies in Medicine at Complutense University (2012). She is persuing the speciality of Medical Oncology. She has studied the Master of Molecular Oncology at CNIO (2016-2017) expert title of thromboembolic disease at Alcala de Henares University and Post-grado of methodolody of investigation in Oncology at ICO too. She has published more than 30 papers in reputed journals and has been serving as an Editorial Board Member of repute. She has also presented more than 100 posters and oral communications.

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Targeting escape signaling in resistant non-Hodgkin's lymphoma

Lalit Sehgal
The University of Texas MD Anderson Cancer Center, USA

Diffuse large B cell lymphoma (DLBCL) is the most common subtype of non-Hodgkin lymphoma (NHL) with 1400 cases diagnosed yearly in the USA. While some patient can be cured for like, approximately 30% of these patients have lymphoma that comes back despite therapy and will die prematurely. In recent years advances in treatment have shown Ibrutinib blocks a driver of cancer termed BTK. While ibrutinib is initially effective, most individuals with different types of lymphomas develop resistance to and have a short survival. With this growing problem on potentially curable lymphoma, we plan to learn how ibrutinib stops working and overcome this problem with mechanistically derived new treatments for DLBCL, which will apply to many blood cancers. There are two major problems that stand in the way of identifying curative therapy. One is an incomplete understanding of drugs blocking the driver Bruton tyrosine kinase (BTK) which loses its effectiveness and allows regrowth of DLBCL. A second problem is suppressed immune cells which would normally recognize and eliminate DLBCL, but in relapsed DLBCL fail to eliminate DLBCL. Said another way, the immune cells have the brakes applied and are not free to eliminate DLBCL cells. We have made progress to show possible ways to understand how ibrutinib drug loses its effectiveness. The information contained in this e-mail message may be privileged, confidential, and/or protected from disclosure. This e-mail message may contain protected health information (PHI); dissemination of PHI should comply with applicable federal and state laws. If you are not the intended recipient, or an authorized representative of the intended recipient, any further review, disclosure, use, dissemination, distribution, or copying of this message or any attachment (or the information contained therein) is strictly prohibited. If you think that you have received this e-mail message in error, please notify the sender by return e-mail and delete all references to it and its contents from your systems.

Speaker Biography

Lalit Sehgal has research interests focused on the communication between lymphoma cells and stromal cells. His recent finding revealed that communication between the tumor and stroma could modulate the expression of key oncogene, which can be further targeted for effective therapy in MCL relapse. The findings have forwarded the hematology field by exploring new targets for therapy.

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Retrograde tracheal intubation in Mongolia

Bolormaa Batnasan
National Cancer Center, Mongolia

Anesthesia Process: The patient's back and place the O2 mask using the 20 G intravenous IV Fentanyl 100 µg. We reported successful and unsuccessful anesthesia retrograde tracheal intubations in NCC.

Case I: In 03 June 2015, a 30 year-old male patient was posted for elective surgery head and neck department. The surgery was required to recurrent tumor (d=6cm) of Rt. Sub mandible gland T2N1M0 do MND tumor remove. On examination of the airway, all parameters such as mouth not opening (he had big accidence and neck surgery in 2002, 2007, 2012). Chin-thyroid distance: less than 2 cm. Dentures, removable teeth.

Case II: In 19 Sep 2015, a 66 year-old male patient posted for emergency case head and neck surgery department. The patient had two surgeries NCC. First elective surgery was 17 Sep 2015 (required to big tumor resection and reconstruction by ALTFF in cancer mandibles) with normal intubation. Second emergency surgery was 19 Sep 2015 (free plat to restore the blood supply and airway oxygen supply to increase) with retrograde intubation. He was breathing periodically interrupted.

Case III: In 11 Apr 2016, a 46 year-old male patient was posted for elective surgery head and neck department. He was very (Fibroptic picture 3) difficult slowly breathing. The patient had tongue (root) cancer surgeries NCC. Elective surgery required to big tumor resection with tracheostomy. We can`t put retrograde intubation. Because he has trachea d=0.2-0.3mm. After resection we came to know that the intubation tube (size number: 4-5.5) was too big.

Case IV: In 13 Jan 2013, a 57 year-old male patient posted for elective surgery head and neck department. The patient had surgery big tumor resection and reconstruction in cancer mandibles with successful anesthesia retrograde tracheal intubations in NCC.

Discussion: Number of retrograde intubations in the literature makes an effort 539 patients and 137 Cadavers. If high professional anesthesia team puts retrograde intubation successful then, low trauma in patient, may be easier surgical team as fiberoptic and tracheostomy.

Speaker Biography

Bolormaa Batnasan did her Master's degree (2008) at World Federation Society, Anesthesiology–Training course in Thailand and Hospital Management course (2009) in Arab Republic of Egypt, and Anesthesiology Fellowship course (2010) Soul, Korea. She has studied Doctorate in September 2010, in Medical University of Mongolia and Anesthesiology Fellowship course (2012) in Lausanne, Switzerland.

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Tumor heterogeneity driven by sharing genetic and signaling code between microbiota and breast carcinoma

Nilesh Kumar Sharma
Dr. D Y Patil Biotechnology & Bioinformatics Institute, India

There are emerging views to substantiate the inumerable population of microbioata making their choice of residence within human body parts including intestine and mammay glad tissue. In a focussed way, possibly the presence of microbes in mammar gland could be explained by the entry of microbes from the skin. There are possibility of presence of proteobacteria including taxa such as Bacillus, Acinetobacter, Pseudomonas, Staphylococcus and Propionibacterium. It is true that presence of these microbes can impact the normal and tumor mammay glad tissue. In other way, the role of these microbes may be extended to possibility that descernible presence of intra-tumoral heterogeneity in breast tumor could be driven one of potential routes through microbes and cellular communities including cancer cell, immune cells and stromal cells. In view of plethora of microbiot and tumor cellular components interactions, first elucidation may come from the study of molecular signaling crosstalk between microbes and tumor cell. Possibly, molecular signaling crosstalk needs to be focussed at the level of metabolites secretion and sharing between microbes and tumor cells. Therefore, metabolome of tumor tissue needs to be highlighted with reference to microbes and tumor tissue niches. Another possible communication between microbes and tumor cell could be possible through the transfer of genetic materials between in the form of extra-chromosomal circular DNA like plasmid and small non-coding RNAs. In this era of science, theer are evidence of natural transfer of extra-chromosomal circular DNA and small RNAs may be possible through natural genetic mateiral exchange process among prokaryotes and eukaryotes. Hence, one potential question emerges that whether transfer of microbe origin plasmid with a potential of drug resistance gene can be transfered to cancer cells within the tumor niches. These plasmid genetic materials with drug resistance may be able to confer the reinforcing capability to the heterogeneous nature of tumor cellular communities for better growth, suvival and drug resistance. Currently, we are investigating the contribution of microbiota mediated plasmid transfer to push tumor heterogeneity. To achieve this goal, we use next generation sequencing, metabolomic profiling and molecular techniques. Therefore, this paper highlight the need for scienetific attempt to address the interplay of microbes and tumor heterogeneity. Also precisely unravell the contribution of transfer of drug resistance plasmid from microbes to tumor cell by generating drug resistant and robust heterogeneous population with their niches.

Speaker Biography

Nilesh Kumar Sharma has completed his PhD from Indian Institute of Technology Roorkee, India in the year 2009 within Health Science specialization. He completed his Post-doctoral research training for more than three years in DNA repair genetic and cancer biology at NIEHS, NIH, USA and Rutgers University, NJMS, NJ, USA. Since July 2016, he is working as an Associate Professor at Dr. D Y Patil Biotechnology & Bioinformatics, Dr. D Y Patil, Vidyapeeth, Pune, India as a faculty and Principal Investigator in DST and DPU funded research project. He has been credited with more than 25 research publications in indexed international journals and two book chapters.

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The Effect of the Anti-Cancer Preparation NSC-631570 on Prostate Cancer

Wassil Nowicky
Ukrainian Anti-Cancer Institute, Austria

Unusual for an anticancer agent NSC 631570 possesses some distinct immune properties. In several immune target-effector systems NSC 631570 significantly amplified the malignotoxic activity of macrophages, lymphocytes and NK cells, and stimulates dendritic cells maturation in vitro. While the parameters like B-lymphocytes count, immune globulin concentrations, complement and acute phase proteins did not changed significantly, it can be postulated NSC 631570 modulates the cellular part of the immune system whereas the humoral part remains unaffected. Besides, the NSC631570 has a selective effect – thus, it kills only cancer cells and the healthy cells remain undamaged. First indications on the selective effect of NSC 631570 on the cancer cells were provided in an early study when different oxygen consumption by normal liver cells and Ehrlich's tumor ascitic cells after the incubation with NSC 631570 was revealed. A radio protective effect was found in normal human fibroblasts. NSC 631570 caused the accumulation of prostate cancer cells as well as epidermoid carcinoma cells in the G2/M phase, however, not of normal cells. The efficacy of NSC-631570 in prostate cancer has been confirmed in a controlled clinical study. In the study patients, all standard treatment modalities had been exhausted. The cancer relapsed and/or progressed and no therapy protocol was available. The patients were treated with NSC-631570 and partially with local hyperthermia. Following results were achieved: full remission in 54 patients (73%), partial remission in 16 patients (22%). Only in 4 patients (5%) the therapy did not affect the course of the disease.

Speaker Biography

Wassil Nowicky — Dipl. Ing., Dr. techn., DDDr. h. c., Director of "Nowicky Pharma" and President of the Ukrainian Anti-Cancer Institute (Vienna, Austria). Has finished his study at the Radiotechnical Faculty of the Technical University of Lviv (Ukraine) with the end of 1955 with graduation to "Diplomingeniueur" in 1960 which title was nostrificated in Austria in 1975. He became the very first scientist in the development of the anticancer protonic therapy and is the inventor of the preparation against cancer with a selective effect on basis of celandine alkaloids "NSC-631570". He used the factor that cancer cells are more negative charged than normal cells and invented the Celandine alkaloid with a positive charge thanks to which it accumulates in cancer cells very fast. He has received the award for merits of National guild of pharmasists of America. The award of Austrian Society of sanitary, hygiene and public health services and others.

Total Number of Patients	Full Remission	Partial Remission	Disease Progression
74	54	16	4
100%	73%	22%	5%

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Feasibility of breast self-examination and clinical breast examination as a screening tool for breast cancer in a low resource setting: A pilot study

Meesha Iqbal
Aga Khan University, Pakistan

Breast cancer is the most common cancer in women both in the developed and less developed world. Breast cancer survival rates vary greatly worldwide, ranging from 80% or over in North America, Sweden and Japan to around 60% in middle-income countries and below 40% in low-income countries. The low survival rates in less developed countries can be explained mainly by the lack of early detection programs, resulting in a high proportion of women presenting with late-stage disease, as well as by the lack of adequate diagnosis and treatment facilities. The menace of breast cancer has not spared Pakistan with its incidence reaching up to almost 35,000/100,000. 30.8% of all cancer deaths in Pakistan are due to breast cancer. Given that Pakistan is a low resource setting, we designed a screening program based on examination for the detection of breast cancer. The study was piloted in the rural area of Rehri-goth. Pre-medical volunteer students were trained on breast self-examination. The volunteers went door to door creating awareness in the community regarding the danger signs of breast cancer and the importance of self-examination. All participants who identified any danger sign were called to the outreach center of AKU for clinical breast examination followed by diagnostic mammography. Although mammography has been established as the gold standard for screening breast cancer in the community, yet, in a low resource setting like Pakistan, breast self-examination followed by clinical breast examination can serve as a useful tool.

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Identification of canine papillomavirus in the transmissible venereal tumor using the polymerase chain reaction technique in canines (Canis lupus familiaris)

Sergio Ayala-Díaz[1], Joaquín Manzo-Merino[2], Marcela Lizano[2] and **Jaime Arroyo Ledezma[1]**
[1]Universidad del Mar, México
[2]Instituto Nacional de Cancerología, México

The Venereal transmissible tumor (TVT), also known as infectious sarcoma, venereal granuloma, transmissible lymphosarcoma or Sticker´s sarcoma, is a neoplastic disease affecting dogs and its propagated mainly during the intercourse. The TVT is located mainly in the genital area with a lower frequency at the oral cavity, nasal cavity, eyes and skin. The disease is presented as a tumoral mass at the glans bulb in males, and in the vaginal vestibule. Up to date, there is no evidence for a viral agent as the causative agent for TVT development. The present work was aimed to analyze 21 samples from canines with TVT for clinical, cytological and histopathological evaluation, as well as for blood count, clinical chemistry, bacterial culture and molecular analysis to identify papilloma virus DNA sequences. Clinical diagnostic confirmed the clinical and biochemical features for TVT and molecular analysis demonstrated the viral DNA presence in the samples through the amplification of the viral sequence L1 (major capsid coding gene of papilloma virus) using different primer sets, the MY primers amplified a 450 bp band in seven out of 23 samples (33%). L1 positive samples were sequenced to analyze the identity of the PCR product. The PVF and Fap-64 primer set, targeting the L1 sequence of Canine Papilloma Virus (CPV), showed positivity in 16 out of 21 samples (76%). On the other hand, the amplification using the CP4/5 primer set, aimed to amplify the E1 region of CPV, showed no amplification at all. These results support the possible causative association between CPV and TVT; nevertheless, confirmatory studies are required to confirm such as statement. This work represents the first evidence indicating that a viral agent might be involved in the pathogenesis of TVT with high impact in the understanding of TVT pathogenesis.

Speaker Biography

Sergio Ayala-Díaz is graduated in Zootechny from Universidad del Mar, holds a degree in Hematology from the Universidad Nacional Autónoma de México and a Master of Science from Universidad del Mar in collaboration with the Instituto Nacional de Cancerología, México. He has worked in collaboration with other researchers in the area of Epidemiology and Molecular Biology of oncogenic viruses and transmissible tumors such as canine transmissible venereal tumor to generate timely diagnostic tools.

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