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15th World Congress on

CANCER THERAPY, BIOMARKERS & CLINICAL RESEARCH

December 05-07, 2016 Philadelphia, PA, USA

Scientific Tracks & Abstracts

(Day 1)

Cancer Therapy & Biomarkers 2016

Sessions:

Day 1 December 05, 2016

Cancer Stem Cell Therapy | Cancer Biomarkers | Cancer Chemotherapy | Clinical Oncology | Gynecological Cancer | Radiation Oncology | Surgical Oncology | Anti-Cancer Agents in Medicinal Chemistry

Session Chair
Huber Colleen
Naturopathic Oncology Research Institute, USA

Session Chair
Ying Mu
Center for Devices and Radiological Health-US FDA, USA

Session Introduction

Title: Overcoming therapy-resistant cancer stem cells
Hideshi Ishii, Osaka University Graduate School of Medicine, Japan

Title: Phenylethyl maleimide derivatives as novel apoptosis inducers on L5178-Y murine leukemia cells (in silico, *in vitro* and *in vivo* study)
Erik Andrade Jorge, Escuela Superior de Medicina, Instituto Politécnico Nacional, Mexico

Title: From multi-drug treatment to a single multitarget drug; A new path in the treatment of cancer
Jose Guadalupe Trujillo Ferrara, Escuela Superior de Medicina, Instituto Politécnico Nacional, Mexico

Title: ImmTACs: Bi-specific TCR-based reagents for targeted cancer immunotherapy
Cheryl McAlpine, Immunocore Ltd., UK

Title: Pfetin as a risk factor of recurrence in gastrointestinal stromal tumors
Shunsuke Sakuraba, Juntendo Shizuoka Hospital, Japan

Title: ERC/mesothelin as a biomarker for mesothelioma and gastrointestinal cancer
Tomoaki Ito, University School of Medicine, Japan

Title: Association between levels of 5-hydroxymethylcytosine and clinical/histopathologic features in locally advanced breast cancer: Results from the Biomarkers Breast Cancer Cohort of Mexican Women (2012-2015)
Diddier Prada, National Institute of Cancerology, Mexico

Title: Prognostic microRNA signature of triple-negative breast cancer identified by cross-validated Cox model development
Jianying Zhang, Ohio State University, USA

Title: Serum fatty acid synthase as a marker of digestive neoplasia
Hajime Orita, Shizuoka Hospital-Juntendo University, Japan

Title: *Ex vivo* apoptotic, autophagic and angiogenic influence of an estradiol analogue on platelets
Lisa Repsold, University of Pretoria, South Africa

Title: Breast cancer amongst old and young women: Prevalence, risk factors in Lviv city, Ukraine
Obianuju Efobi Chinyere, Ukraine

Hideshi Ishii, Oncol Cancer Case Rep 2016,2:3(Suppl)
http://dx.doi.org/10.4172/2471-8556.C1.002

Overcoming therapy-resistant cancer stem cells

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Till date, international pharmaceutical companies have produced numerous anti-cancer reagents, including molecular targeting strategies and immunotherapies, but resistance exists even for these up-to-date medicines. The most important factors that make present therapeutic strategies ineffective are tumor heterogeneities. To visualize and collect cancer stem cell fractions, we transfected cancer cells with a green fluorescent protein-fusion one-carbon metabolism monitoring cassette. The monitoring system allowed visualization of cancer cell populations with therapy-resistant cancer stem cell properties. The present study revealed that polyamine flux plays a critical role in cancer stem cell properties, and polyamine metabolism is linked with epigenetic regulation of downstream gene expression. Epigenetic studies demonstrated the uncharacterized mechanism of transcription cycles and underscored the significance of molecular profiling in the discovery of novel therapeutic targets for retractable cancer cells. These novel approaches are beneficial for cancer research and may open avenues for treating gastrointestinal cancers that present challenges for treatment.

Biography

Ishii has completed his postdoctoral studies from Thomas Jefferson University, Philadelphia, PA. He is the professor of cancer profiling discovery, Osaka University, Japan. Specialty and Present Interest: intractable cancer stem cells, epigenome, metabolome, gastrointestinal tumors.

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Phenylethyl maleimide derivatives as novel apoptosis inducers on L5178-Y murine leukemia cells (*in silico*, *in vitro* and *in vivo* study)

Erik Andrade Jorge and **José Guadalupe Trujillo Ferrara.**
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Conventional cancer therapies have been shown to have many side effects, the main challenge is to find molecules with higher selectivity to tumor cells rather than normal cells. A main differences between cancer and normal cells, are the levels of thiol-containing compounds, a scavenging mechanism of Reactive Oxygen Species. Cancer cells seem to have higher levels of glutathione than normal cells, and this allow them to survive in adverse conditions, even in chemotherapy. Therefore, glutathione has become a target for the new anticancer therapy. The aim of this contribution was to develop a series of α,β-unsaturated compounds derivate of endogenous amines that may deplete the levels of glutathione, as well as, induce cancer cells to death by apoptosis. Pharmacokinetic evaluation (*in silico*) showed a good score on the the parameters such as human intestinal absorption, plasma protein binding, biotransformation evaluated by cytochrome CYP2C9 affinity and CYP2D6 affinity, P-glycoprotein substrate, LopP, etc. The *in vitro* assays showed a EC50 of 5μM for molecule MF01 and a EC50 of 30 μM for molecule MF02 evaluated by MTT method at 24 and 48h, *in vivo* experiments include LD50 and survival experiment. It was estimated a LD50 for MF01 of 8mg/kg and 80mg/kg for molecule MF02, which means that molecule MF02 is 10 times less toxic that molecule MF01. There wasn't significant difference on the survival experiment at the dose used, but there was a delay on the tumor's development on the treated group. These results allow us to try others candidates which might possess the same properties.

Biography

Erik Andrade-Jorge is a Doctorate student in the Department of Biochemistry at Instituto Politecnico Nacional. He is a chemist-pharmaceutical-biologist and has a Master degree in Pharmacology and is currently in the seventh semester of the Doctorate in research in medicine. Currently, he has two different lines research one of these is cancer cell proliferation and another one is in Parkinson's disease. He has been focused on the rational drug design based on the molecular mechanisms of different pathologies and in the physicochemical properties of the ligands.

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José Guadalupe Trujillo Ferrara et al., Oncol Cancer Case Rep 2016,2:3(Suppl)
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From multi-drug treatment to a single multitarget drug: A new path in the treatment of cancer

José Guadalupe Trujillo Ferrara and **Erik Andrade Jorge**
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Drug discovery and development is a resource-intensive endeavor that does not always end in success. One of the particularly illustrative examples of this challenge is the development of drugs for cancer treatment. Cancer is one of the leading causes of death worldwide and currently used chemotherapy causes several serious side effects. Hence it is advantageous to combine traditional methodology with new computer-assisted technology to increase the success and lower the investment involved in drug research. The aim of the present work was to develop new compounds with an epigenetic approach for treating cancer cells. This means generating a compound that could inhibit HDAC and ODC while depleting the high level of thiol-containing compounds existing in cancer cells. Overall, the objective is to have an extended conformation of DNA because this tends to silence proliferative genes and activate anti-cancer genes. Additionally, the greater oxidative stress has been shown to lead to death by apoptosis in mitochondrial cells. *In silico* results allow us to predict that α,β-unsaturated compounds will react with thiol-containing compounds in a selective way by a Michael type 1,4-addition reaction. Moreover, molecular docking clearly demonstrates that one moiety of the ligand recognizes the catalytic site of HDAC while the other one recognizes ODC, a theoretical result that has been corroborated by *in vitro* studies. Cell viability did not decrease in noncancerous cells (epithelial cells, HaCaT, THLE-3), it did indeed do so in human cancer cells (HuH7, HepG2, Hela), it was found an inhibition constant of 1.5μM estimated for ODC and HDAC. Finally, the administration of one or more of these compounds in an *in vivo* model was able to extend the life of mice in survival experiments, reaching up to double the time found in the control. This is a preliminary contribution to pave the way for clinical testing of this kind of molecule.

Biography

Pioneering in Mexico in the field of medicinal chemistry, Dr. Trujillo has focused on rational drug design based on the physicochemical properties of ligands and receptors, by using new technology. His different research lines include cancer, obesity, adrenoceptors, Parkinson's, diabetes, and many others. Dr. Trujillo has authored more than 125 publications and has more than 876 citations. He has been the advisor for 72 students to obtain a university degree. He is a member of the National System of Researchers, level III, and is currently in charge of the Ministry of Research and Graduate Studies of the Instituto Politecnico Nacional.

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Cheryl McAlpine et al., Oncol Cancer Case Rep 2016,2:3(Suppl)
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ImmTACs: Bi-specific TCR-based reagents for targeted cancer immunotherapy

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Immune system based therapies constitute a promising class of treatment for many types of cancers. Whilst T cells can mediate tumor destruction, their effectiveness is limited due to negative thymic selection, down-regulation of HLA expression by cancer cells and an immunosuppressive microenvironment. To overcome the poor immunogenicity of tumors, Immunocore has developed ImmTACs (immune mobilizing monoclonal T cell receptors (mTCR) against cancer). A soluble mTCR, engineered to recognise a given tumor associated peptide-HLA complex with exceptionally high sensitivity and specificity, redirects host polyclonal T cells via an anti-CD3 antibody fragment, facilitating targeted T cell recognition of tumors. Antigens that exist on tumor cells but not on normal cells are rare, thus the selection and validation of appropriate target antigens and the testing of ImmTACs for specificity is critical. Antigen candidates are selected based on their levels of expression in cancer vs healthy tissues by RT-PCR and the presence of the targeted peptide on the cell surface is confirmed by mass spectrometry. As ImmTACs are specific for humans, efficacy, specificity and off target effects are determined through a detailed molecular and cellular testing programme, using antigen positive tumor cells and HLA appropriate primary human cell lines representing a range of tissues. IMCgp100, the lead ImmTAC, is currently in a Phase I/II clinical trial for the treatment of advanced melanoma. The maximum tolerated dose has been established and emerging data demonstrate several durable responses. IMCgp100 is well tolerated and there is evidence of T cell mobilisation in the tumor microenvironment, release of cytokines and tumor shrinkage.

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Shunsuke Sakuraba, Oncol Cancer Case Rep 2016,2:3(Suppl)
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Pfetin as a risk factor of recurrence in Gastrointestinal Stromal Tumors

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We have proposed Pfetin as a new, strong prognostic biomarker in resected Gastrointestinal Stromal Tumors (GISTs). Pfetin is a potassium channel protein. It is highly expressed in fetal cochlea and in the brain, consistent with the fact that the origin of GIST is Cajal cells, and neuronal cells in the gut. Pfetin was discovered by using a proteomics approach and its usefulness as a prognostic biomarker has been reported. We examine Pfetin expression immunohistochemically using paraffin-embedded tissues, and when more than 20% of tumor cells are stained with the anti-Pfetin antibody, they are considered to be Pfetin-positive. Pfetin expression and tumor metastasis were inversely related and GIST with Pfetin-negative status has a poorer prognosis than Pfetin-positive GIST. We also have reported the fact that Pfetin is an independent predictor of recurrence/metastasis for completely resected primary, localized GIST. In our study, 13 cases were Pfetin-negative and 5/13 of these cases recurred. Conversely, 32 cases were Pfetin-positive and 2/32 of these cases recurred. (p=0.002) Pfetin is a strong prognostic biomarker and useful in selecting adjuvant therapy. We reviewed literatures and summarized Pfetin in GIST.

Biography

Shunsuke Sakuraba has completed his MD from Juntendo University. He works in the Department of Surgery at Juntendo Shizuoka Hospital and specializes in Open Surgery, Laparoscopic Surgery and Endoscopic Surgery. He has published 2 papers about GIST and MALT lymphoma of GI tract.

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Tomoaki Ito, Oncol Cancer Case Rep 2016,2:3(Suppl)
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ERC/mesothelin as a biomarker for mesothelioma and gastrointestinal cancer

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Previously, we found that the ERC (Expressed in Renal Carcinoma) gene was preferentially expressed in renal cancers in the Eker rat. Furthermore, we subsequently confirmed that ERC is a homolog of the human mesothelin gene, a gene that is strongly expressed in normal mesothelial cells, mesotheliomas, and non-mucinous ovarian carcinomas. The ERC/mesothelin gene (MSLN) encodes a 71kDa precursor protein, which is cleaved to yield 31kDa N-terminal (N-ERC/mesothelin) and 40 kDa C-terminal (C-ERC/mesothelin) proteins. N-ERC/mesothelin (also known as megakaryocyte-potentiating factor; MPF) is a soluble protein and is released into the extracellular space and blood. C-ERC/mesothelin is a glycoprotein that is tethered to the cell surface by a glycosylphosphatidylinositol anchor. C-ERC/mesothelin expression of tumor by immunohistochemistry can be correlated with patient's survival in several human cancers. Soluble Mesothelin-Related Peptide (SMRP) has proven to be a promising biomarker in the sera of patients with mesothelioma and ovarian cancer. As for secreted N-ERC/mesothelin, we previously devised a novel Enzyme-Linked Immunosorbent Assay (ELISA) system for determining its concentration in serum and showed that it is useful for diagnosing human mesothelioma and ovarian cancer. In addition, we demonstrated that C-ERC/mesothelin was expressed in gastric cancer and pancreatic cancer tissues. Meanwhile, increased serum N-ERC/mesothelin concentrations are not specific to these patients with gastric cancer or pancreatic cancer. Although N-ERC/mesothelin is established as a reliable marker for mesothelioma, N-ERC/mesothelin is not useful as a diagnostic marker of gastric cancer and pancreatic cancer.

Biography

Tomoaki Ito received the MD in 2000 and the PhD in Medical Science from Juntendo University, Tokyo, Japan. He completed Post-doctoral studies from Stanford University School of Medicine. He is an Assistant Professor of Department of Surgery, Juntendo Shizuoka Hospital, Juntendo University School of Medicine, Shizuoka, Japan. His research interests include oncology and cancer biomarker.

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Association between levels of 5-hydroxymethylcytosine and clinical/histopathologic features in locally advanced breast cancer: Results from the biomarkers breast cancer cohort of Mexican women (2012-2015)

Diddier Prada, Omar Peña Curiel, José Díaz Chávez, Cynthia Villarreal Garza, Nancy Reynoso, Paula Cabrebra Galeana, Enrique Bargalló Rocha, Yolanda Villaseñor Navarro, Claudia Caro Sánchez, Luis A Herrera, Ramirez-Otero, Miguel, Justo and **Montserrat**
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Breast cancer is a leading cause of cancer death in the world. In developing countries, most patients are still diagnosed in locally advanced stages (LABC), which highlight the importance of identifying prognostic and predictive biomarkers. In this study, we determined the level of 5-hydroxymethylation (5hmC) in the biopsy at diagnosis. Then, we determined its association with clinical and histopathologic characteristics in a prospective cohort of LABC patients (N=84), diagnosed between 2012 and 2015; using a semi-quantitative enzyme-linked immunosorbent assay. We found a statistically significant association between low 5hmC levels with histological grade: Mean %5hmC in low grade tumors 0.17%, (95% CI 0.07-0.26%), intermediate-grade tumors 0.15% (95% CI 0.10-0.19%), high grade tumors 0.066% (95% CI 0.05-0.08%; p<0.001). We also observed an association between 5hmC levels with histological type: Invasive ductal carcinoma 0.10% (95% CI 0.07-0.12%) vs. non-ductal invasive carcinoma 0.22% (95% CI 0.07-0.36; p=0.008). Additionally, we found a negative and significant association with Ki67 (β=-0.012, standard error [SE]=0.0061, p=0.047), a known marker for cellular proliferation. Multivariate analysis confirmed the association between lower levels of 5hmC with age (β=-0.066, SE=0.031, p=0.036) and histological grade (β=-1.197, SE=0.589, p=0.042). No association was observed with therapeutic response or free-relapse survival, probably attributed to only 2-3 years of follow-up, and to few deaths (N=2) and relapse (N=7) that have been observed. This is the first report on the association between levels of 5hmC with the histological type and histopathologic grade in a prospective cohort of LABC. Our findings suggest that 5hmC levels may be a potential biomarker for tumor aggressiveness in LABC.

Biography

Diddier Prada graduated as Medical Doctor from the Universidad Industrial de Santander, Colombia. Then, he pursued a PhD in Biomedical Science at the Universidad Nacional Autónoma de México (UNAM). He was a Post-doctoral Fellow at Harvard T.H. Chan School of Public Health, from 2013 to 2016; his stay included one-year and a half as Research Associate in the laboratory of Environmental Epigenetics in the Department of Environmental Health. He is currently working as Associate Researcher at the National Cancer Institute of Mexico, and he is also Associate Professor of Biomedical Informatics in the Faculty of Medicine at the UNAM. He has published papers in highly reputed peer-reviewed journals (e.g. *Circulation, Environmental Research*, and *Environmental Health Perspectives*). He has also published one book and five chapters in internationally recognized books, plus one that is currently in press

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Prognostic microRNA signature of triple-negative breast cancer identified by cross-validated Cox model development

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Cox regression models have been used for prognostic prediction based on omics data for years. But due to model over-fitting and the improper way of model development and various other factors, very few published prognostic signature has found its clinical success in applications. This study illustrated how an improper model develop or testing procedure could mislead the result and applied the proper cross-validated (CV) Cox model building and testing procedure to identify a prognostic microRNA signature based on 125 triple negative breast cancer (TNBC) patients. In each CV procedure (K-fold), the full training data was split into train data and test data first. Then four steps were followed: feature selection, model selection, addition of significant clinical covariates, and model performance assessment. For each of the four steps, various methods were compared. For example, univariate cox model or progression vs progression-free comparison was applied to feature selection. Stepwise and penalized Cox model were used for model selection for the training data. Model performance was assessed on the test data by cross-validated AUC of 3-year or 5-year recurrence and ROC calculated using the time-dependent ROC method. The proposed optimal procedure is cross-validated stepwise selection based on the feature screening by progression vs progression-free. The final proposed cox model contained 5 miRs: miR-363, miR-155, miR-142-5p, kshv-miR-K12.11, and miR-1307. When the significant pathologic predictor NODES (positive vs negative) was added to the 5-miR model, the mean cross-validated AUC was increased by 0.06 on average. Permutation method was used to test if the cross-validated AUC of a model was significantly greater than 0.5. Enrichment analysis and supportive literature further verified the association between the five proposed oncomiRs and TNBC progression (recurrence/death). The five-miR model was validated in an independent group of N=34 TNBC patients with 2-year, 3-year and 5-year progression AUC 00.84, 0.72, 0.77, respectively. Survival analysis on the dichotomized risk groups based on the predictive risk score resulted in a P value of 0.065 (by median of validation risk score) or 0.0008 (by median of train risk score) for the log-rank test.

Biography

Jianying Zhang received her PhD degree in Statistics from Purdue University in 2008. She has worked at the Center for Biostatistics at the Wexner Medical Center of the Ohio State University, Columbus, OH for about 8 years. She has collaborated extensively with investigators in the Comprehensive Cancer Center on basic science and translational laboratory experiment and clinical trials and has been the Co-investigator of over 10 P01/U01/R01 and industrial grants. She has published over 40 collaborative papers in reputed journals.

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Serum fatty acid synthase as a marker of digestive neoplasia

Hajime Orita
Shizuoka Hospital-Juntendo University, Japan

The aim of this study is to evaluate FAS as a marker of digestive cancer to measure FAS serum levels in patients with digestive cancer and explore each digestive cancer metabolic features. Cancer metabolism is a futuristic strategy point for diagnosis and management. Lipid metabolism in particular is a vast uncharted territory for targeting tumor control. It is well known that many cancer cells up-regulate of fatty acid synthesis. Cancer cells require fatty acid for building blocks of new organelles and cells. Fatty Acid Synthase (FAS) is the enzyme responsible for fatty acid biosynthesis. It is over-expressed in many human cancers, reported to be correlated with cancer growth, contributes to poor prognosis and inhibition of FAS results in decreased cell proliferation, and loss of cell viability. Previously we have reported high expression not only in tissue, but also in serum in patients with digestive cancers. Although FAS is found to be over-expressed in many solid tumors, its role in digestive cancer has not been extensively evaluated. We have reported that it seems to be up-regulated during the early stages of tumorigenesis. In Digestive neoplasm, Upper GI (esophageal and gastric) and colorectal ones have different characteristics due to carcinogenesis. Upper GI neoplasm results from the continuous inflammation from Helicobacter pylori and various other factors. Therefore colorectal one arises from adenoma carcinoma sequences. Our results show a different tendency for each other. Compare between premalignant status and malignancy, by using serum and tumor FAS level we can approach the mechanism of reprogramming the regulation of metabolic pathways.

Biography

Hajime Orita graduated and took the surgical training from Juntendo University School of medicine in Japan. Now he is the associate professor of dept. of upper GI and especially do laparoscopic surgery. He had been post doctal studies and recived the adjunct associate professor degrer from Johns Hopkins University School of medicine. He has published more than 10 papers in reputed journals and has been serving as an editorial board member of repute.

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Ex vivo apoptotic, autophagic and angiogenic influence of an estradiol analogue on platelets

Lisa Repsold
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Platelets are known contributors to vascularization, metastasis and growth of tumors. Upon their interaction with cancer cells they are activated resulting in the release of angiogenic activators thereby promoting angiogenesis. Angiogenesis-regulating proteins are ideal biomarkers in the study of cancer pathophysiology and represent desirable therapeutic- and diagnostic targets.

The *in silico*-designed analogue of 2-methoxyestradiol, namely 2-ethyl-3-O-sulphamoyl-estra-1,3,5(10)16-tetraene (ESE-16), binds to carbonic anhydrase II delaying early metabolism and is thus carried into the circulation. The effect it potentiates on blood components, especially on platelets, is of significance in cancer progression. This study thus investigated the possible ex vivo apoptotic, autophagic and angiogenic effects of ESE-16 on platelets.

Scanning electron microscopy was used to assess morphological changes in platelets after exposure to ESE-16 and no changes were observed in ESE-16-treated platelets. The possible induction of apoptosis and autophagy was determined by annexin V-FITC, measurement of caspase 3 activity, autophagy-related protein 5 levels, light chain 3-I to light chain 3-II conversion and monodansylcadaverine staining which indicated that there was no increase in apoptosis or autophagy when platelets were exposed to ESE-16. The expression of the angiogenic proteins namely vascular endothelial growth factor, platelet derived growth factor and matrix metallopeptidase-9 was also assessed and results showed that levels were significantly increased after platelets were added to MCF-7 cells.

This is the first *ex vivo* study to highlight possible involvement of angiogenesis, apoptosis, autophagy in platelets after exposure to this potential anti-cancer compound warranting further investigation concerning these signaling pathway targets on platelets of cancer patients.

Biography

Lisa Repsold is a PhD student at the Department of Physiology, University of Pretoria, South Africa. She completed her Masters degree cum laude and is currently conducting her PhD study with a title: 'Angiogenic, apoptotic and autophagic profiling of chronic myeloid leukaemia patients' platelets ex vivo before and after treatment with Imatinib'. She has published three papers in internationally accredited peer-reviewed journals and presented her research at national conferences. In her research career she has mastered several scientific techniques including scanning and transmission electron microscopy, flow cytometry, cell culture techniques and western blot.

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Breast cancer amongst old and young women: Prevalence, risk factors in Lviv city, Ukraine

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Ukraine

Background: Breast cancer is becoming a silent killer amongst women of younger age due to negligence and belief that it occurs only in old women. As long as there are presence of breast tissues in a female, breast cancer can occur from any age.

Methods: A qualitative study was carried out for 1 week, offline forms were filled by women of different races, ages diagnosed of cancer. Data was compiled, AGE 15-35 was grouped as young and 36 > was grouped as old.

Result: Among the participants, were 6 cases of younger women and 39 cases of older women. Most women of the young age group linked their diagnosis to genetic predisposition while women of older age groups could not determine a link to their diagnosis which rarely occurs.

Conclusion: Prevalence of breast cancer in younger women is quite low and can be linked to family history, unlike in older women it is very high and can be due to various risk factors which cannot be stated specifically.

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CANCER THERAPY, BIOMARKERS & CLINICAL RESEARCH

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Scientific Tracks & Abstracts

(Day 2)

Cancer Therapy & Biomarkers 2016

Sessions:

Anti-Cancer Drugs and Delivery | Biomarkers | Novel Approaches to Cancer Therapeutics | Chemotherapy | Cancer and Lifestyle Connection | Cancer Biomarkers | Cancer Cell Biology, Diagnosis and Applied Research

Session Chair
Al Charest
Beth Israel Deaconess Medical Center Cancer Center, USA

Session Chair
Andrea Nicolini
University of Pisa, Italy

Session Introduction

Title: Preoperative neutrophil to lymphocyte ratio predicts central compartment lymph nodes metastasis in papillary thyroid microcarcinoma
Xiequn Xu, Peking Union Medical College Hospital, China

Title: Inhibitive effect of Non-viable derivatives of Clostridium sporogenes on colorectal cancer cells
Madhura Satish Bhave, Nanyang Technological University, Singapore

Title: Breast carcinoma metastasis suppressor gene 1 (BRMS1): Update on its role as the suppressor of cancer metastases in the context of combinatorial cancer treatment
Magdalena Kodura, Karolinska Institute, Sweden

Title: Anaemia in cancer patients undergoing radiotherapy and chemotherapy in National Hospital Abuja, Nigeria
Chinedu S Aruah, National Hospital Abuja, Nigeria

Title: Simvastatin induces apoptosis of Osteosarcoma cells: A novel potential therapeutic approach
Walied A Kamel, Keio University, Japan

Title: Decision-making of cancer patients about End-of-Life: The lived experience
Angela Katrina G Fonte, Far Eastern University, Philippines

Title: Endothelium-derived 5-Methoxytryptophan acts as a therapeutic biomarker for systemic inflammation
Cheng Chin Kuo, National Health Research Institutes, Taiwan

Title: An *in vitro* evaluation of carmustine-loaded Nano-co-Plex for potential magnetically targeted intranasal delivery to the brain for brain tumor management
Olufemi David Akilo, University of the Witwatersrand, South Africa

Title: How many cell death pathways that Doxorubicin can affect HepG2 cells?
Noor Mohammed, 7 goss croft birmingham, UK

Title: Protection against oral mucositis in patients undergoing radiotherapy of the head and neck with topical mucoadhesive gel containing Propolis
Vagner Rodrigues Santos, Universidade Federal de Minas Gerais, Brazil

Xiequn Xu et al., Oncol Cancer Case Rep 2016,2:3(Suppl)
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Preoperative neutrophil to lymphocyte ratio predicts central compartment lymph nodes metastasis in papillary thyroid microcarcinoma

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Background: The incidence of Papillary Thyroid Microcarcinoma (PTMC) is rising in most countries, which is a consequence of efficacy earlier diagnosis. Even though the central lymph nodes metastasis has be considered to be a risk factor of poor prognosis in PTMC, there is no reliable preoperative assessment for it. Our study was to confirm the value of preoperative Neutrophil To Lymphocyte Ratio (NLR) to predict the central lymph nodes metastasis of PTMC by retrospective case control study.

Methods: A total of 142 patients who underwent total thyroidectomy with prophylactic central lymph nodes dissection at a single institution between Oct 2013 and Jan 2015 were analyzed. Patients were categorized into central lymph node metastasis positive and negative, those were confirmed by histological. Age, gender, blood cells counts with differential counts, medical history were measured or recorded in each patient before operation.

Results: In our study, 37.14% (n=52) were CLN metastasis positive while 62.86% (n=88) were negative. The factors that correlated (P<0.05) with the presence of central lymph node metastases were age and NLR. The NLR were significantly higher in the Central lymph node negative group (p=0.027). The optimum NLR cut-off value obtained from ROC analysis was 2.32 (Sensitivity 44% and Specificity 87.1%). The optimum Age cut-off value obtained from ROC analysis was 45(Sensitivity 53.1% and Specificity 76.3%).

Conclusions: Higher preoperative NLR could be a reliable negative marker for central compartment lymph node metastasis and cut-off value of 2.32 be supposed.

Biography

Xiequn Xu has completed his MD at the age of 27 years from Peking Union Medical College Hospital. He is the associate professer of general surgery in Peking Union Medical College Hospital, a ranked No 1 hospital in China. He has published more than 30 papers in reputed journals.

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Inhibitive effect of Non-viable derivatives of *Clostridium sporogenes* on colorectal cancer cells

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Traditional cancer treatments like chemotherapy and radiation therapy continue to have limited efficacy due to phenomena like tumor hypoxia and multi-drug resistance. Bacterial cancer therapy has the potential to overcome these problems, through the use of anaerobic spores of bacteria such as the proteolytic *Clostridium sporogenes*. However, the use of spores or live bacteria comes with the risk of toxicity and infection. To circumvent these issues, the anti-cancer effect of heat-inactivated *C. sporogenes* bacteria (IB) and its secreted bacterial proteins, known as Conditioned Media (CM) was investigated. These non-viable bacterial derivatives were administered to CT26 and HCT116 colorectal cancer cells in a 2-Dimensional (2D) and a 3-Dimensional (3D) platform. IB significantly inhibited cell proliferation of CT26 in a dose-dependent manner to 6.3% of the control in 72 hours for the 2D monolayer culture. In the 3D spheroid culture, cell proliferation of HCT116 spheroids notably dropped to 26.2%. Similarly the CM also remarkably reduced the cell-proliferation of the CT26 cells to 2.4% and 20% in the 2D and 3D models, respectively. Results suggest that physical interaction between the IB and the cancer cells lead to their inhibition, while the secreted proteins present in CM were responsible for anti-cancer effect observed. The bacterial derivatives exhibited strong inhibitive effects on colorectal cancer cells, indicating that there is a safer alternative to the use of spores and live bacteria. With further research, these non-viable derivatives could be developed as an alternative or adjunct to traditional cancer treatments.

Biography

Madhura is currently pursuing her PhD at Nanyang Technological University (NTU), Singapore. She completed her Bachelor of Engineering in Bioengineering from NTU where her final year honours thesis was on the subject of Bacterial Cancer Therapy. She was on the Dean's List for the academic year of 2014/2015. Her work has been published in the Scientific Reports journal, by the Nature Publishing Group.

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Notes:

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Breast carcinoma metastasis suppressor gene 1 (BRMS1): Update on its role as the suppressor of cancer metastases in the context of combinatorial cancer treatment

Magdalena Kodura
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Background: BRMS1 (Breast Cancer Metastasis Suppressor-1) protein was discovered over a decade ago as a potential tumor suppressor gene. Our review summarizes the recent findings about the structure of BRMS1, mechanisms of its action and the role of BRMS1 in the cancer progression. In addition, I would like to discuss the BRMS1 findings in a broader context. Combinatorial treatment of breast cancer with the joined forces of chemotherapy, adjuvant therapy, cytotoxic agents and radiation has had a great impact on the prolonged survival of breast cancer patients. Metastasis remains however the major reason behind the mortality rate in this group of patients and as such requires more attention in understanding the potential influence of other parallel pharmaceutical treatments.

Objectives: The aim of my presentation will be to summarize the recent findings regarding the effect of BRMS1 protein on the suppression of cancer metastasis as well as to explore the correlation between the activity of BRMS1 protein, growth factors (TGF-β and EGF), antidepressants (fluoxetine and amitriptyline) and the stimulation of cell survival and migration of breast cancer.cells in a combinatorial cancer treatment.

Results: As a suppressor of metastasis, BRMS1 has demonstrated a variety of ways to act on the cell functions, such as cell migration, invasiveness, angiogenesis, cell survival, cytoskeleton rearrangements, cell adhesion, and immune recognition. This variety of effects is a likely reason behind the robustness of anti-metastatic influence of BRMS1. Intracellular signaling mechanisms employed by BRMS1 include regulation of transcription, EGF/HER2 signaling, and expression of NF-kB, fascin, osteopontin, and IL-6. Recently reported clinical studies confirm that BRMS1 can indeed be used as a prognostic marker. Approaches to employ BRMS1 in a development of anti-cancer treatment have also been made. The combinatorial influence of growth factors and antidepressants showed a dynamic modulation depending 2 on the presence of BRMS1 protein. It indicates the existence of a correlation between BRMS1, TGF-β, EGF, fluoxetine and amitriptyline. Furthermore, the effect of antidepressants differed depending on the kind of parallel treatments and therefore underlined the significance of drug-drug interactions. Antidepressant amitriptyline strongly promoted colony formation in MDA-MB-231-pMEP4 cell line, which was observed in both membrane migration assay and clonogenic assay. The same treatment resulted in a complete inhibition for MDA-MB-231-pMEP-BRMS1 cell line. Although the study requires to be confirmed by larger number of experiments, it may suggest that breast cancer patients taking amitriptyline (while not expressing BRMS1 protein) are exposed to increased risk of metastasis. On the other hand, for breast cancer patients who express BRMS1 protein and are treated with amitriptyline, it may imply an outstanding inhibiting effect of amitrypryline treatment on metastasis. Moreover, BRMS1 proved to have an impact on fluoxetine activity by inhibiting the stimulating effect of fluoxetine on the treatments with TGF-β, which were earlier observed in MDA-MB-231-pMEP4 cell line. In contact inhibition assay the stimulation of senescent cells by BRMS1 may suggest the role of BRMS1 in the inhibition of uncontrolled cancer.cell.proliferation. Conclusions: The studies reviewed here with respect to BRMS1 structure, cellular effects, intracellular signaling, and clinical value consolidate the importance of BRMS1 in the development of metastasis. In addition, the results of our study imply a significant correlation between BRMS1 protein, growth factors and antidepressants. A strong, opposite impact of amitriptyline on colony formation in both BRMS1-expressing and non-expressing cell lines requires a further investigation of the mechanism of interactions between BRMS1 and the treatment agents used in the study. It is advised in order to improve the outcome of the cancer treatment as well as the cancer related depression treatment. Furthermore, the results indicate that the future cancer treatment needs to consider not only drug-drug interactions but also a cross-talk between the drugs and the proteins involved in the cell growth and metastasis.

Biography

Two Master of Science degrees (Medical Physics, AGH, Krakow, Polen and Molecular Biotechnology, KTH, Stockholm, Sweden). Both of my theses were performed and defended with highest grades at Karolinska Institute in Stockholm, Sweden. The results of my Master of Science thesis in Molecular Biotechnology turned out to be so interesting that I had a pleasure to present them at Personalized Cancer Care Symposium in Oslo (Norway) in 2012. My supervisor at Karolinska Institute, Serhiy Souchelnytskiy, saw so much scientific potential in me during my work at his lab that he invited me to write a review which was published in Cancer and Metastasis Reviews Journal and which was met with a worldwide interest and many invitations to conferences. The findings of my review as well as my own personal experimental results are so interesting and crucial for the development of combinatorial cancer treatment that I hope I will have a pleasure to present them at your esteemed congress in Philadelphia in December and inspire a positive development in combinatorial cancer therapy.

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Chinedu S. Aruah et al., Oncol Cancer Case Rep 2016,2:3(Suppl)
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Anaemia in cancer patients undergoing radiotherapy and chemotherapy in national hospital Abuja, Nigeria

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Introduction: Many cancer patients present with anaemia prior to radiotherapy and chemotherapy or may experience anaemia /worsening of anaemia at some point during treatment.

Aims and Objectives: The aim of the study was impact of anaemia in cancer patients undergoing Radiotherapy and Chemotherapy.

Methodology: 201 cancer patients of both sexes with histopathologically confirmed malignancies (solid cancers). Patient's pre-treatment Hb was taken. Patients were distributed into Radiotherapy, Chemotherapy and Chemoradiation. Their Hb were measured once every 2 weeks. The blood film pictures of the patients were examined. The whole process was terminated after 3 consecutive Hb reading or after week 6. Anaemia was classified into:

Less than 10g/dl	-	Severe anaemia
10 - 10.9g/dl	-	moderate anaemia
11 - 12 g/dl	-	mild anaemia
12 g/dl and above	-	no anaemia.

Results and Analysis: Out of 201 cancer patients, 86.1% were female and 13.9% were male. Age range, 25 - 75 years, 100 patients were on Chemotherapy, 63 patients on Radiotherapy and 38 patients on Chemoradiation. The prevalence in anaemia in cancer patients undergoing radiotherapy and chemotherapy was found to be 63% as shown by blood film picture (i.e average of 72%, 42.9% and 73.7%). At the end of therapy, 62% (100) patients on Chemotherapy and 55.6% (63) patients on Radiotherapy had their Hb level between 11-12g/dl, 39.5% (38) cancer patients on Chemoradiation arm had Hb value of 10-10.9 g/dl. At P-value > 0.05, there was no statistical significance on distribution of mean Hb, standard deviation based on sex and treatment type.

Conclusion: Prevalence of anaemia in the study group was found to be 63% while 37% had adequate haemoglobin (Hb) after the therapy as reflected in the blood film picture. At 95% confidence interval, Chemotherapy had greatest impact on Hb level during therapy. Thus Chemotherapy; 9.60-10.62g/dl, Radiotherapy; 11.52-12.1 3g/dl, Chemoradiation therapy; 10.98-11.3 6gIdl.

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Simvastatin induces apoptosis of osteosarcoma cells: A novel potential therapeutic approach

Walied A Kamel,[1,2,7] Eiji Sugihara,[1] Sayaka Yamaguchi,[1] Koichi Matsuo,[3] Akihiro Muto,[2] Hideyuki Saya,[1] Takatsune Shimizu[1,2]
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Osteosarcoma (OS) is the most common, non-hematopoietic, primary malignant bone tumor. Previously, we developed an OS mouse model by overexpressing c-MYC in bone marrow stromal cells derived from Ink4a/Arf knockout mice. We isolated highly tumorigenic cells (designated AXT cells) from tumors after serial transplantation. To obtain the novel candidate agents for OS, we performed drug screening and found that statins strongly suppressed AXT cell growth.

Simvastatin treatment inhibited cell proliferation and induced apoptosis, which was almost fully rescued by the supplement of mevalonate and geranylgeranyl pyrophosphate but modestly by farnesyl pyrophosphate, suggesting that protein geranylgeranylation has a greater impact on OS cell viability.

Simvastatin treatment inactivated RhoA through translocation of RhoA from membrane to cytosol and RhoA-GTP was accumulated by disruption of the interaction between RhoA and Rho-GDI.

As a downstream signaling of RhoA, AMPK-p38MAPK pathway was strongly activated by simvastatin treatment, with AMPK functioning as an upstream effector of p38MAPK. Inhibition of AMPK or p38MAPK activation rescued apoptosis induced by simvastatin treatment, indicating that simvastatin exerts antitumor activity in OS via activation of AMPK- p38 MAPK pathway.

Although treatment with simvastatin alone did not inhibit OS tumor growth *in vivo*, its combination with a fat-free diet induced a significant antitumor effect that was further enhanced by metformin administration. These findings suggest that the activation of AMPK- p38 MAPK pathway by statins become a potential therapeutic option for OS.

Biography

Walied kamel, 32 years old, a PHD student at keio unviersity, school of medicine, Tokyo, Japan.

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Angela Katrina G et al., Oncol Cancer Case Rep 2016,2:3(Suppl)
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Decision-making of cancer patients about end-of-life: The lived experience

Angela Katrina G. Fonte and **Rn Bs Man Smrin**
Far Eastern University, Philippines

Understanding the perception of an end-stage cancer patient about end-of-life decision making can help the patient's relatives, healthcare providers, and the person himself or herself in attaining the best quality of life in their exit event. The aim of this study is to deeply gain an understanding of the voice and feelings of stage 4 cancer patients in making decisions for end-of-life. The study was conducted using a qualitative phenomenological approach. Five participants who are of sound mind and able to make rational decisions shared their preferences. The participants were selected using a non-probability, criterion, purposive sampling. Data were gathered through the use of a semi-structred interview. Four major themes emerged from the analysis of the data. The themes were leaving protracted misery, divesting the burden, feeling of complacency and living in a former time.

These themes encircles mainly on the issue of cycle of suffering anf prolonging one's agony with the use of life-saving measures which can reduce the quality of life. Findings of the study revealed that end-of-life deision making is encapsulated with different factors which include physical discomfort and exhaustion, emotional distress, spiritual dilemma and financial burden. Recommendations include educational training for nurses about end-of-life care and discussion of ethical issues, culturally competent care, and management of patients who are facing end-of-life decision making. It is also recommended that physicians should take the lead and explore the end-of-life preferences of patients and their families.

Biography

Angela Katrina G. Fonte is a Philippine Registered Nurse. She completed Bachelor of Science in Nursing at the Far Eastern University Manila, in the year 2010 and earned Master of Arts in Nursing Major in Medical-Surgical, year 2015 at the same university. As a passionate and dedicated registered nurse, she practice critical care nursing for more than two years at Marikina Valley Medical Center and later joined Asian Hospital and Medical Center in their Medical-Surgical Intensive Care Unit. She is a Fellow of the Royal Institute of Nurses, Singapore.

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Cheng Chin Kuo, Oncol Cancer Case Rep 2016,2:3(Suppl)
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Endothelium-derived 5-methoxytryptophan acts as a therapeutic biomarker for systemic inflammation

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National Health Research Institutes, Taiwan

Systemic inflammation has emerged as a key pathophysiological process which induces multi-organ injury and causes serious human diseases. Endothelium plays a critical role in maintaining cellular and inflammatory homeostasis, systemic inflammation and progression of inflammatory diseases. We postulated that endothelium produces and releases endogenous soluble factors to modulate inflammatory responses and protect against systemic inflammation. We found that conditioned medium (CM) of Endothelial Cell (EC) inhibited Cyclooxgenase-2 (COX-2) and interleukin-6 expression in macrophages stimulated with lipopolysaccharide (LPS). Analysis of CM extracts by Liquid Chromatography–Mass Spectrometry (LC-MS) showed the presence of 5-Methoxytryptophan (5-MTP) but no other related tryptophan metabolites. Furthermore, endothelial cells-derived 5-MTP suppressed LPS-induced inflammatory responses and signaling in macrophages and endotoxemic lung tissues. LPS suppressed 5-MTP level in EC-CM and reduced serum 5-MTP level in the murine sepsis model. Intraperitoneal injection of 5-MTP restored serum 5-MTP accompanied by inhibition of LPS-induced endothelial leakage and suppression of LPS- or cecal ligation and puncture (CLP)-mediated pro-inflammatory mediators overexpression. 5-MTP administration rescued lungs from LPS-induced damages and prevented sepsis-related mortality. Importantly, a considerable amount of 5-MTP was detected in healthy subjects (1.05±0.39 mM) while 5-MTP level in septic patients (0.37±0.15 mM, $p<0.0001$) was significantly reduced in septic patients. We conclude that 5-MTP belongs to a novel class of endothelium-derived protective molecules which defend against endothelial barrier dysfunction and excessive systemic inflammatory responses. Being an endogenously produced compound, 5-MTP has the advantage of having less unexpected adverse effects. Thus, 5-MTP will be a valuable lead compound for new inflammatory drug development. Another potential clinical application of 5-MTP is its use as a biomarker of sepsis and other systemic inflammatory disorders. Hence, it may be useful as a "Therapeutic" biomarker for selecting sepsis patients for 5-MTP therapy.

Biography

Cheng-Chin Kuo has completed his PhD at the age of 30 years from National Defense Medical Center, Taipei, Taiwan and postdoctoral studies from Academia Sinica, Taipei. In 2007, he joined the National Health Research Institutes as Assistant Investigator. In 2013, he got promotion to Associate Investigator. He has published more than 34 papers in reputed journals and has been serving as an journal reviewer. His current researches are to use comparative metabolomics analysis coupled with cellular biochemical approaches and animal model to determine physiological relevance and pathophysiological connection between physiological metabolites and inflammatory diseases such as systemic inflammation, vascular diseases, and cancer.

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An *in vitro* evaluation of carmustine-loaded Nano-co-Plex for potential magnetically targeted intranasal delivery to the brain for brain tumor management

Olufemi David Akilo
University of the Witwatersrand, South Africa

The aim of this work was to synthesize polyvinyl alcohol/polyethyleneimine/folate (Polyplex) complex coated Magnetite (Nano-co-Plex) with superparamagnetic capability and targeting attributes loaded with carmustine (BCNU) for potential magnetically targeted delivery of BCNU to the brain following intranasal administration for brain tumor management. This was achieved by co-precipitation reaction of $Fe2^{+}$ and $Fe3^{+}$ at high pH under N2, epoxidation of PVA and EDC/NHS coupling reaction of Folate with PEI. Drug loading was carried out in the dark. The release study was carried out by dispersing the dried BCNU-Nano-co-Plex in PBS pH 7.4 at 37°C. The formulation was characterized employing Fourier Transform Infrared Spectroscopy (FTIR), Thermogravimetric Analysis (TGA), Transmission Electron Microscope (TEM), Zetasizer, Superconducting quantum interference device (SQUID) and X-ray Diffraction (XRD) techniques. FTIR spectra confirmed the synthesis of BCNU-loaded Polyplex coated Magnetite. The morphology, size and stability of the formulation showed hexagonally shaped particles with average size of 45nm with zeta potential of +21mV and poly dispersity index (PDI) of 0.22. XRD results further confirmed the crystalline nature of the formulation. The thermal analysis indicated that 1/3 of the total weight of the formulation constituted the drug-loaded polymeric coating. The magnetic studies showed superparamagnetic attribute of the formulation with high magnetization value. The loading capacity of the synthesized Nano-co-Plex was efficient for BCNU. The release profiles indicated a sustainable release of BCNU, with up to 75% of drug released after 72 hours. BCNU-loaded Nano-co-Plex was synthesized successfully.

Biography

Olufemi David Akilo has just completed his PhD in Pharmaceutics specializing in drug delivery systems at the Department of Pharmacy and Pharmacology, University of the Witwatersrand, Johnnesburg South Africa. He currently works with Faculty of Health Sciences, University of the Witwatersrand University. He has authored a book chapter, research paper in a reputed journal and has filed a patent with South Africa patent Agency. He has also presented papers in several local and international conferences.

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Noor Mohammed, Oncol Cancer Case Rep 2016,2:3(Suppl)
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How many cell death pathways that *Doxorubicin* can affect HepG2 cells?

Noor Mohammed
University of Birmingham, UK

Doxorubicin (DOX) is a potent antibiotic anti-cancer drug that is used either in isolation or in combination, for treating ovary, haematological, breast, stomach, liver, and prostate cancers. This drug has the ability to damage DNA and inhibit macromolecules (DNA and RNA) by producing free-radicals. Several studies have shown that Dox induces P53 activation leading to apoptosis in both normal and tumour cells, by causing cytochrome c release from the mitochondria which ultimately leads to apoptosis via caspase 3. We have investigated the molecular mechanisms of DOX induced hepatic cell death. This study shows that DOX can induce cell death in HepG2 cells through two different mechanisms. The use of caspase substrates and caspase inhibitors confirm that apoptosis through caspase 9 and caspase 3 are involved. Using HepG2 cells transfected with LC3-GFP, it was also noted that a high percentage of LC3-GFP punta were seen using fluorescence microscopy, following DOX treatment, which suggests that autophagy is also involved. However, lactate dehydrogenase release assays and the use of necrostatin, on DOX treated cells indicate that necrosis is unlikely to be involved.

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Protection against oral mucositis in patients undergoing radiotherapy of the head and neck with topical mucoadhesive gel containing Propolis

Vagner Rodrigues Santos, Vladimir R.A.S. Noronha, Henrique C. Meira, Ruan S. Silva, Mariana T. Rodrigues, Luis Guilherme V. Madeira, Mayara F. Paiva and **Barbara F. G. Queiroz.**
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The objective of this phase II study was to determine the effectiveness of a mucoadhesive propolis gel in the prevention of radiation-induced oral mucositis. Thirty-four patients who were selected to undergo radiation therapy for oral cancer were included in this open-label trial. They were advised to use a mucoadhesive gel containing propolis 5,0% w/v three times a day starting one day before the course of radiation therapy and concluding after 2 weeks of radiation therapy. A weekly follow-up for evaluation of food intake, pain and grading of mucositis was performed. In order to confirm the absence of Candida-related mucositis in patients who developed mucositis, it was performed exfoliative cytology of buccal mucosa, palate and tongue and the material for Candifast(®) Candida species identification. At the end of the study was made the compliance of patients, quality, appreciation and acceptance of product evaluation. Twenty patients did not develop mucositis, two patients developed grade 1 mucositis and two patients developed grade 2 mucositis. None of the patients discontinued food intake and no pain was observed during the study. Candidosis was not detected in any patient. Mucoadhesive propolis gel could be considered as a potential topical medication for preventing radiation-induced oral mucositis.

Acnowledgements: FAPEMIG/CNPq/ CENEX-FOUFMG/PROEX-UFMG.

Biography

Vagner Rodrigues Santos is Graduate in Dentistry, Associate Professor at School of Dentistry of the Minas Gerais Federal University(SD/UFMG). He has PhD in Oral Pathology (Faculty of Medicine/UFMG), Master in Microbiology(INRA–France), and Post-doctor in Natural Products at University of California Berkeley, USA. He is coordinator of Dentistry Care Clinic to Irradiated in head and neck Patients. Participates in the post-graduate program , guides doctoral, master's and undergraduate research students. Search developing new drugs based on propolis and medicinal plants for prevention and treatment of oral lesions. He has 50 published papers with 387 citations by ResearchGate.

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Session Chair
David Chafin
Roche Tissue Diagnostics (RTD), USA

Session Chair
Jianhua Luo
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Session Introduction

Title: Gut microbiota modulates cisplatin mediated systemic toxicity
Soumen Roy, Cancer and Inflammation Program, NCI, NIH, USA

Title: Formulation of liposomes for oral delivery of phyllanthin and hypophyllanthin
Thahera Parveen Dandu, Oman Pharmaceutical Products (Gulf), Oman

Title: A conceptual study on understanding an ayurvedic concept of cancer and its supportive treatment majors in Ayurveda
Nikhil M Dongarkar, Parul Institute of Ayurved, India

Title: Method development for quantification of circulating cell free HPV DNA
Zhigang Kang, National Cancer Institute, Bethesda, USA

Title: Predicting wound healing complications: a personalized medicine approach to wound care
Kara Spiller, Drexel University, USA

Title: The comprehensive analysis of complex genomic data such as exome sequencing presents several challenges to a clinical laboratory
Avni Santani, Children's Hospital of Philadelphia, USA

Title: Biomarker concentration acting as the indicators for chemicals health risk assessment: The case study in Thailand
Nalinee Sripaung, Bureau of Occupational and Environmental Diseases-Ministry of Public Health, Thailand

Title: The evaluation of concentration of calprotectin, in pleural fluid with causes of exudative pleural effusion
Mohammad Reza Hashempour, Azar Hospital-Golestan University of Medical Sciences, Iran

Title: Regulatory aspetcs of co-development of biomarkers and companion diagnostics
Soma Ghosh, Center for Device and Radiological Health, FDS, USA

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Gut microbiota modulates cisplatin mediated systemic toxicity

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Anticancer chemotherapy has achieved a significant milestone in increasing the number of cancer survivors over past decades, while leaving behind the survivors with various toxic side effects, which are nephrotoxicity, ototoxicity and intestinal damage. Challenges remain to reduce systemic toxicity as well as retaining the anticancer therapy. Gut microbiota modulates cancer chemotherapy, however little is known about the role of gut microbiota in modulating systemic toxicity. We hypothesized that gut microbiota regulates systemic toxicity. Four groups (n=10/group) of 8 weeks old C57B/6 mice were treated with cisplatin, cisplatin+antibiotics cocktails (ABX), ABX only and untreated. ABX cocktail contained primaxin, vancomycin and neomycin, which depletes broad spectrum gut microbiota. This experiment was validated using C57B/6 germ free mice (contains no microorganisms). We performed anti-p-γ-H2AX and anti-ATM based DNA-double stranded break (stains foci in the nuclei) based toxicity assay in kidney and gut (small bowel). H&E and 4 color immunostaining (anti-p-γ-H2AX, anti-ATM, Actin and DAPI) were done. DNA-DSBs were evaluated using Zeiss 780 confocal and quantified by 3-D reconstruction using IMARIS. There were reductions in γ-H2AX$^+$ DAPI$^+$ (DNA damaged) cell populations compared to only cisplatin treated mice, indicated protection in the kidney. Both nuclear foci counts as well as the pathological scores indicated gut microbiota associated modulation in the glomeruli of kidney and in the villi of small bowel. Our data leads to a possibility to develop microbiota based therapy which might be utilized to reduce chemotherapy associated systemic toxicity and for better management of chemotherapy.

Biography

Roy is currently working in the field of cancer and gut microbiota at the National Cancer Institute (NCI), NIH in the laboratory of Dr. Giorgio Trinchieri. His main focus is to investigate the role of gut microbiota in chemotherapeutic drug and radiation therapy induced local and systemic toxicity. Prior joining the National Cancer Institute, Dr. Roy worked at the National Institutes on Deafness and other Communication Disorders (NIDCD) in the Laboratory of Dr. Lisa Cunningham, where he contributed to the development of a sound conditioning based co-therapy, which inhibits cisplatin and aminoglycoside mediated hearing loss in mice. Roy received his doctorate degree under the supervision of Anneliese Schrott Fischer in the field of targeted nanomedicine and hearing from University of Innsbruck, Austria in 2011

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Formulation of liposomes for oral delivery of phyllanthin and hypophyllanthin

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Formulation of liposomes in order to enhance the oral bioavailability of phyllanthin and hypophyllanthin with proven anticancer activity. The bioactive lignans, Phyllanthin and hypophyllanthin are formulated in to conventional and PEGylated liposomes using different ratios of DSPC, DSPE-MPEG2000 and cholesterol by film hydration technique. Evaluation of the prepared liposomes was done by the determination of encapsulation efficiencies, particle size analysis, polydispersity index (PDI), zeta potential, TEM analysis, IR studies, DSC studies and powder X-RD analysis. The drug retention *in vitro* and pharmacokinetic properties *in vivo* are investigated. A new, simple and sensitive analytical method using HPLC with photodiode array (PDA) detection was developed for the determination of phyllanthin and hypophyllanthin in solvent system and in plasma.Conventional and pegylated liposomes are successfully formulated using film hydration technique with encapsulation efficiencies of 86.47%±0.13% and 83.68±0.22% (phyllanthin), 84.83±0.19% and 81.87±0.54% (hypophyllanthin). The HPLC method was successfully applied for quantification of lignans with recorded LOD and LOQ values of 56.15 ng/mL & 169.99 ng/mL (phyllanthin) and 56.04 ng/mL and 169.82 ng/mL (hypophyllanthin), respectively. From the *in vivo* pharmacokinetic studies, it was observed that the oral bioavailability of lignans was enhanced as indicated by AUC values of 5265.30±275.52 ng.h/mL (phyllanthin), 15217.60±987.96 ng.h/mL (conventional liposomal phyllanthin), 30810.23±2587.96 ng.h/mL (pegylated liposomal phyllanthin) and 7354.42±578.2 ng.h/mL (hypophyllanthin), 29222.4±1951.8 ng.h/mL (conventional liposomal hypophyllanthin), 58631.87±2515.46 ng.h/mL (pegylated liposomal hypophyllanthin). The developed liposomal formulations of both the lignans, showed extended drug release over 24 h in *in vitro* drug release studies. Pharmacokinetic studies showed the enhancement of oral bioavailability by several folds for liposomes. The enhanced oral bioavailability of lignan loaded liposomes will be helpful for the production of desired pharmacological activity relatively at a lower dose when compared to their respective free drugs.

Biography

ThaheraParveenDandu has completed her PhD at the age of 35 years from Andhra University. She is working as Deputy Manager, Formulation R&D atOman Pharmaceutical Products, Gulf based pharmaceutical company. She has published more than 9 papers in reputed journals.

Madhukiran Parvathaneni has completed his PhD at the age of 26 years from NAIP/ICAR Project, Andhra University. He is working as Senior Regulatory Associate with a global pharmaceutical CRO company. He has published more than 14 papers in reputed journals and serving as an editorial board member and reviewerfor more than 10 PubMed, Springer and Elsevier Journals

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A conceptual study on understanding an ayurvedic concept of cancer and its supportive treatment majors in ayurveda

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In the present era when the modern thinking and the various research are taking place here is an attempt made to understand the basics of cancer in terms of Ayurveda principles. The Ayurvedic description of the pathophysiology of cancer uses traditional concepts translated into a modern context. Although the biomedical treatment of cancer is considered valuable, from an Ayurvedic perspective it results in degeneration and depletion. Taking this into consideration an Ayurvedic approach focusing on strengthening digestion, eliminating toxins, reducing tumor growth, and improving tissue metabolism is useful. An Ayurvedic approach to cancer supportive care focuses on restoring equilibrium, building strength, and rejuvenation.To understand these concept various Ayurved Classical Books and modern textbooks about cancer along with interviews were conducted with 10 experienced Ayurvedic clinicians in verbal manner. Hence on over all of this discussion it has be concluded that Ayurvedic medicine offers a unique perspective on the biomedical diagnosis of cancer that emphasizes restoring wholeness, uses natural remedies, includes a focus on emotional health, and emphasizes prevention strategies which can be a major part in pallivative and supportive care.

Biography

Nikhil M. Dongarkar, have completed my graduation from Rajive Gandhi university of helth science bangalore and now pursuving post graduation from Parul Institute of Ayurveda vadodara, Gujarat Ayurved University.

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Predicting wound healing complications: a personalized medicine approach to wound care

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Background: Wound healing can be impaired by bacterial infection or patient factors including diabetes, autoimmune disease, chemotherapy, and immunosuppression. Failure to achieve complete closure of wounds, whether they are combat wounds or chronic ulcers that are commonly observed in the veteran population, leads to impaired mobility, hospitalization, amputation, and even death. The selection of an appropriate, individually tailored treatment strategy from the myriad choices available on the market is critical to successful wound healing. Unfortunately, there is no accurate, objective way to determine if a wound is healing or not. For example, in clinical practice, chronic diabetic ulcers that fail to show a 40% reduction in surface area (length x width) over 4 weeks are considered non-healing. However, this method of healing diagnosis has low accuracy (~60%) because these assessments are subject to error, can differ from one physician to the next, and convey only superficial characteristics of the wound. As a result, patients are treated with ineffective treatments for far too long (leading to amputation or death), or expensive therapies like synthetic skin substitutes and hyperbaric oxygen therapy are used when they are not necessary (causing waste to our healthcare system). Therefore, we developed a quantitative molecular assay that uses debrided wound tissue that would otherwise be discarded to evaluate the behavior of wound macrophages, the inflammatory cells recognized as the major regulators of healing, in order to determine if a wound is on a healing trajectory or is likely to develop complications and fail to heal.

Methods: In the pilot study, debrided wound tissue, collected during routine wound care that would otherwise be discarded, was collected from the ulcers of 21 diabetic patients over 4 weeks. Treatment and follow-up were conducted for an additional 8 weeks to determine if the ulcer had completely healed at 12 weeks. Gene expression data of a panel of 7 biomarkers related to the behavior of macrophages was converted to a single score that is indicative of the inflammatory status of the wound. Briefly, the score represents a ratio based on the M1/M2 paradigm of macrophage behavior, in which M1 macrophages are inflammatory yet initiate the healing process, while M2 macrophages are anti-inflammatory and facilitate resolution of the healing process. Non-healing wounds are believed to be stalled in the M1 phase.

Results: In the pilot study of 21 patients with diabetic ulcer, the M1/M2 score decreased over a 3-week period for all wounds that ultimately healed by 12 weeks of treatment (n=9 patients). In stark contrast, the scores stayed the same or increased for all wounds that ultimately failed to heal (n=12 patients). In fact, the fold change in the score at 4 weeks from the initial visit was almost 100 times higher for nonhealing wounds compared to healing wounds ($p<0.0001$). Using a 4-week fold-change cutoff of 1 (so that an increase was used to classify non-healing wounds and a decrease was used to classify healing wounds), the score successfully predicted healing or nonhealing in 19/21 patients in this study (100% positive predictive value, 83.3% negative predictive value, and 90% overall accuracy). Interestingly, healing wounds generally had higher M1/M2 scores than non-healing wounds at the start of the study, suggesting that inflammation is critical for wound healing and that with further optimization, analysis of a single sample may be predictive of likelihood of developing wound complications.

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Conclusions: These results indicate that analysis of wound tissue that would otherwise be discarded can be used as a quantitative, accurate indicator of healing. Such an assay will be critical for employing a precision medicine approach to wound care.

Next steps: More patients are needed to further optimize this diagnostic assay. Wound samples can be stored in a buffer and shipped at room temperature to our lab for analysis. If you are interested in collaborating on this project. Future directions include expanding to other wound types (combat wounds, burns, chronic venous ulcers, etc.) and developing a rapid assay for point-of-care diagnosis. Our pilot data suggest that this method will be readily applicable to burn wounds without the need for further optimization.

Citation: Nassiri, S., I. Zakeri, M.S. Weingarten, K.L. Spiller. "Relative expression of pro-inflammatory and anti-inflammatory genes reveals differences between healing and nonhealing human chronic diabetic foot ulcers." Journal of Investigative Dermatology 2015 (135) 1700-1703.

Biography

Kara received her bachelor's and master's degrees in biomedical engineering from Drexel University in 2007. She conducted her doctoral research in the design of semi-degradable hydrogels for the repair of articular cartilage in the Biomaterials and Drug Delivery Laboratory at Drexel and in the Shanghai Key Tissue Engineering Laboratory of Shanghai Jiao Tong University. After completing her PhD in 2010, when she received the award for Most Outstanding Doctoral Graduate: Most Promise to Enhance Drexel's Reputation, she conducted research in the design of scaffolds for bone tissue engineering as a Fulbright Fellow in the Biomaterials, Biodegradables, and Biomimetics (the 3Bs) Research Group at the University of Minho in Guimaraes, Portugal. She is currently conducting research in the design of immunomodularatory biomaterials, particularly for bone tissue engineering. Her research interests include cell-biomaterial interactionns, biomaterial design, and international engineering education.

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Method development for quantification of circulating cell free HPV DNA

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In the study, we present a method of automated circulating cell-free (ccf) DNA isolation and digital droplet (dd) PCR-based single DNA molecule testing for detecting and typing circulating human papillomavirus (HPV) DNA. To detect single copies of HPV16 or HPV18 DNA, a digital droplet PCR (ddPCR) method was developed using the sequences of HPV16 or HPV18 E7 gene that are common amongst difference subtypes. The probes for HPV16 and HPV18 are differentially fluorescence-tagged for the simultaneous typing and quantification of both types to enhance the confidence in the tests. Two (2) different sets of primers were designed for each HPV type to produce amplicons of different length. Using ccfDNA from a few cervical cancer patients for analysis, it was apparent that the short amplicons of 70 and 88 bps gave much higher HPV copy numbers with ccfDNA than the long amplicons of 208 and 273 bps for HPV18 and 16, respectively. While the assays have maximum sensitivity of a single copy, the quantification ranges are between 10-100,000 copies of both HPV16 and HPV18 DNA which is within its linear dynamic range with coefficient of variation (CV) of less than 20% throughout the entire quantification range. To determine the spike recovery as a part of analytic validation, low amounts of known DNA template of HPV16 or HPV18 were spiked into serum samples. DNA was subsequently isolated and the purified DNA was then analyzed by ddPCR for HPV16 or HPV18 copy numbers. Approximately 75% of the spike DNA could be recovered and quantified. Thus, the HPV16 and HPV18 ccfDNA tests were specific and quantitative.

Biography

Zhigang Kang has completed his PhD from University of Helsinki, Finland and Post-doctoral studies from NIMH, NIH. He is a Research Scientist in Molecular Target Core, Genetics Branch, Center for Cancer Research, National Cancer Institute and an employee of Basic Science Program, Leidos Biomedical Research, Inc., Frederick. He has been working in the field of cancer biomarkers for the past 7 years. His research interests focus on the detection of invasive tumor biomarkers include circulating tumor cell (CTC), cell free circulating tumor DNA and clinical relevance protein markers from peripheral blood of patients with various types of tumors.

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Biomarker concentration acting as the indicators for chemicals health risk assessment: The case study in Thailand

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According to the usage of type of metabolites in blood and urine acting as the biomarker and the concentration of biomarker acting as the indicator for health risk assessment. The health risk management concerning with chemicals toxicity faces to the problem of how to use the concentration of biomarker to be the suitable indicator for health risk assessment. Presently, there is no any safety value adjustment of health risk assessment for people's health in community. Therefore, in the field of prevention and control of occupational and environmental diseases has to use the safety value of biomarker concentration for worker's health to be the safety value for people's health indicator in community for health risk assessment resulted from chemicals pollution. The study of using the safety value of worker' health to identify people's health in community in case of health surveillance of chemicals incidents was proceeded during the year 2015-2016. It was found that biomarkers concentrations of VOCs (Volatile Organic Solvents) of worker's health were higher than the actual baseline chemicals exposure of People's health in community. The result from the adjustment the risk group of people in community by worker' s health safety value indicated the lower amount of people than the actual amount risk group. Thus, the baseline concentration of biomarker should be further studied to be the health surveillance value for people' health risk assessment caused by chemicals toxicity.

Biography

Nalinee Sripaung has completed her PhD from Tokyo Medical and Dental University, Japan. She is the Assistant Director of Bureau of Occupational and Environmental Diseases, Department of Disease Control, Ministry of Public Health, Thailand. She has experience working with the Thai National Chemicals Management Strategy for prevention and control of occupational and environmental diseases. She has published more than 30 papers in chemicals dissemination and reputed journals and has been serving as an Editorial Board Member of reputed journal in Thailand.

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Comparative study of the levels of interleukin-27 and interleukin-17 in pleural fluid of patients with cause of exudative pleural effusions

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Pleural effusion is one of signs and complications resulting from malignant diseases such aslung and breast cancer, and also tuberculosis and infective lung disease. Diagnostic evaluationfor patients with pleural effusion include history, physical exam, chest x-ray and if necessarythoracocentesis and cytologic analysis of pleural fluid. According to cytologic evaluation ofpleural fluid, pleural effusion divide in two group: exudative and transudative, that exudativepleural effusion require more diagnostic evaluations. By cytologic analysis of pleural fluid wecan use of tumor markers and other biomarkers to better diagnose malignant pleural effusion. Inthe previous studies by using immunochemical approach, interleukin-17 and also CEA expressin more amounts in malignant pleural effusion than in benign pleural effusion, in contrast tointerleukin-27. In this study we examined the concentration of Interleukin-27 & 17 in PleuralFluid with Causes of Exudative Pleural Effusion in the Patients Referred to educational Hospitalof Gorgan of Iran in 2015-16. This is a descriptive-analytical and case-control study and 130patient with exudative pleural effusion were enrolled in the study after an informed consent.Samples collected from the patients divided in two main group including 88 patients withmalignant pleural effusion and 42 patients with benign pleural effusion. In next step by using ofthe same previous pleural fluid samples, the concentration of Interleukin-27 & 17 was measuredwith ELISA by an specific Kit. After entering to computer through SPSS-18 statistical software,description of data was done into frequency and percentage. Interleukin-27 concentration was(203.05±76.03) in patients with malignant causes and (344.15±236.42) in benign causesspecially tuberculosis. Interleukin-17 concentration was (69.73±64.58) in patients with malignantcauses and (55.32±43.60) in benign causes.The results showed that these difference werestatistically significant (p=0.000 for IL27) and (p=0.02 for IL17). In other word interleukin-27level, is higher in the benign pleural effusions and interleukin-17 level, is higher in the malignantpleural effusions. According to higher levels of interleukin-27 in benign pleural effusions andhigher level of interleukin-17 in malignant pleural effusions, maybe we can achieve importantresults in differentiating between malignant and non-malignant pleural exudate, without the needfor invasive procedures, by putting together the clinical symptoms, the interleukins concentrationin pleural fluid and pleural fluid cytology results.

Biography

Mohammad Reza Hashempour has completed his Doctorate from Army University of Medical Sciences and Postdoctoral studies in Surgery from Golestan University School of Medicine. He has published papers in reputed journals.

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Regulatory aspects of co-development of biomarkers and companion diagnostics

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An important requirement in the development of targeted therapies is identification of clinically relevant biomarkers, such as DNA mutations or protein overexpression. The detection of appropriate biomarkers is essential for the safe and effective use of targeted therapies and serves as the basis for development of companion diagnostic devices. There are several regulatory considerations for successful biomarker-targeted therapy-companion diagnostic co-development programs. My talk will highlight challenges and opportunities associated with these programs.

Biography

After completing her doctoral degree from the School of Life Sciences at Jawaharlal Nehru University, New Delhi, India, Dr. Ghosh continued her training in molecular biology at the National Institutes of Health (NIH/NICHD), Bethesda, MD, where her work dealt with the mechanisms that regulate cellular DNA replication during animal development. Her focus then shifted to development of sequencing-based assays to support clinical decision making in cancer therapy and management, an area she pursed as a molecular geneticist at the Sidney Kimmel Comprehensive Cancer Center in Johns Hopkins Medical Institute. Currently, Dr. Ghosh is a regulatory scientist at the FDA where she is actively involved in the review and approval of companion diagnostic devices.

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