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4TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

17-18 September 2021

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ICC 2021

BOOK OF ABSTRACTS

4TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

17-18 SEPTEMBER 2021

Theme:

Cancer Free World - Possible or Not

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About MAGNUS GROUP

Magnus Group (MG) is initiated to meet a need and to pursue collective goals of the scientific community specifically focusing in the field of Sciences, Engineering and technology to endorse exchanging of the ideas & knowledge which facilitate the collaboration between the scientists, academicians and researchers of same field or interdisciplinary research. Magnus group is proficient in organizing conferences, meetings, seminars and workshops with the ingenious and peerless speakers throughout the world providing you and your organization with broad range of networking opportunities to globalize your research and create your own identity. Our conference and workshops can be well titled as 'ocean of knowledge' where you can sail your boat and pick the pearls, leading the way for innovative research and strategies empowering the strength by overwhelming the complications associated with in the respective fields.

Participation from 90 different countries and 1090 different Universities have contributed to the success of our conferences. Our first International Conference was organized on Oncology and Radiology (ICOR) in Dubai, UAE. Our conferences usually run for 2-3 days completely covering Keynote & Oral sessions along with workshops and poster presentations. Our organization runs promptly with dedicated and proficient employees' managing different conferences throughout the world, without compromising service and quality.

About ICC 2021

ICC 2021 aims to bring together specialists from various medical and surgical branches, researchers, clinicians, faculty, students, Directors in the field of Oncology under one roof to share and exchange clinical experiences with the ultimate aim of intensifying practical knowledge. With delegates from all around the world attending, it will provide the ideal forum to discuss the latest findings to put them into context and really understand how they affect your practice day to day.

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KEYNOTE FORUM

DAY 1

4TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

SEP 17-18, 2021

Michael Thompson
Department of Chemistry, University of Toronto, Toronto, Ontario, Canada

Point-of-care tool for early diagnosis of Ovarian Cancer

High grade serous ovarian cancer (HGSOC) histologic subtypes make up the majority of epithelial ovarian cancer and 85% of cases are diagnosed at advanced stages, in which the 5-year relative survival could be as low as 20%. Unfortunately, only 20% of patients are diagnosed at stages and when treatment of the disease is more effective. Moreover, there are no mass screening techniques that are cost-effective and reliable. Our research involves the development of both a low-cost general screening test for early detection of OC as well as a multiplexed precise medical device that can be used for point-of-care testing (POCT) at the bedside to monitor the progress of the disease during treatment. The former detects the lysophosphatidic acid (LPA), a highly promising biomarker, which was found to be elevated in 90% of stage OC and gradually increases as the disease progresses to later stages. And the latter detects LPA together with the known cancer antigen-125 (CA125) biomarker to use as a POCT device. We are employing electrochemical techniques, which are highly sensitive and rapid, to develop the proposed devices. Electrochemical devices can be easily miniaturized, which will reduce the cost of the fabrication. Such devices that can accurately detect early-stage OC as well as monitor the progress of the disease is highly desired for i) mass screening that reduces fatality rates; ii) avoiding false negatives or false positives that can lead to higher health-care costs and undesired stress to the patient iii) monitoring the progress of the disease during and after the treatment or surgery; and iv) screening drug candidates that accelerating the clinical trials. Our research includes a novel and unique strategy to avoid fouling of devices surfaces by components of biological fluids. This involves silane-based interfacial chemistry for the mitigation of non-specific adsorption. We are tailoring this strategy by designing novel trichlorosilane molecules using molecular dynamic (MD) computer simulations to provide a scaffold for developing the biorecognition surfaces. Using this scaffold, we are developing electrochemical biosensors for LPA and CA125 detection by following affinity-based and aptamer-based approaches, respectively. We will combine the above biosensors in a miniaturized setup using a microfluidic system to fabricate a POCT device capable of detecting LAP and CA125, which is simple, easy to use, and cost-effective.

Audience Take Away:

- Appraisal of possible biomarkers for ovarian cancer
- Application of biosensor technology to point-of–care assays
- Possibility for rapid, sensitive screening for stage 1 ovarian cancer

Biography:

Professor Michael Thompson obtained his PhD in analytical chemistry from McMaster University. He was Lecturer in Instrumental Analysis at Loughborough University, UK before moving to the University of Toronto where he is now Professor of Bioanalytical Chemistry. Thompson has served on the Editorial Boards of a number of major international journals and is Editor-in-Chief of "Detection Science" for the Royal Society of Chemistry, UK. He has been awarded many prestigious international prizes for his research including The Robert Boyle Gold Medal of the Royal Society of Chemistry, The Elsevier Prize in Biosensor and Bioelectronic Technology and the E.W.R. Steacie Award of the Chemical Society of Canada. He has published over 300 papers.

Alain Chapel

Radiological Protection and Human Health Division, Institute of Radiological Protection and Nuclear Safety, Fontenay-aux-Roses, France

Clinical trial evaluating the efficacy of systemic mesenchymal stromal cell injections for the treatment of severe and chronic radiotherapy-induced abdomino-pelvic complications refractory to standard therapy

The late adverse effects of pelvic radiotherapy concern 5 to 10% of patients, which could be life threatening. However, a clear medical consensus concerning the clinical management of such healthy tissue sequelae does not exist. Our group has demonstrated in preclinical animal models that systemic mesenchymal stromal stem cells (MSCs) injection is a promising approach for the medical management of gastrointestinal disorder after irradiation. In a phase 1 clinical trial, we have shown that the clinical status of four first patients suffering from severe pelvic side effects (Epinal accident) was improved following MSC injection (figure 1 and 2). Two patients revealed a substantiated clinical response for pain and hemorrhage after MSC therapy. The frequency of painful diarrhea diminished from 6/d to 3/d after the first and 2/d after the 2nd MSC injection in one patient. A beginning fistulization process could be stopped in one patient resulting in a stable remission for more than 3 years of follow-up. A modulation of the lymphocyte subsets towards a regulatory pattern and diminution of activated T cells accompanies the clinical response. MSC therapy was effective on pain, diarrhea, hemorrhage, inflammation, fibrosis and limited fistulization. No toxicity was observed. We are now starting a clinical research protocol for patients with post-radiation abdominal and pelvic complications (figure 3) who have not seen their symptoms improve after conventional treatments (NCT02814864, Trial evaluating the efficacy of systemic MSC injections for the treatment of severe and chronic radiotherapy-induced abdomino-pelvic complications refractory to standard therapy (PRISME). It involves the participation of 6 radiotherapy services for the recruitment of 12 patients. They will all be treated and followed up in the hematology department of Saint Antoine Hospital. The cells will be prepared in two production centers (EFS Mondor and CTSA). Treatment is a suspension of allogeneic MSCs. Eligible patients must have a grade greater than 2 for rectoragy or hematuria at inclusion and absence of active cancer. Each patient receives 3 injections of MSCs at 7-day intervals. Patients will be followed up over a 12-month period. The main objective is a decrease of one grade on the LENT SOMA scale for rectorrhagia or hematuria. The secondary objective is to reduce the frequency of diarrhea; analgesic consumption, pain and improved quality of life.

Audience Take Away:

- The audience will be learn about the treatment of pelvic cancer and how the stem cell therapy can be used to manage the late adverse effects of pelvic radiotherapy refractory to conventional therapy
- This help the audience in their job by providing new approach of side effect of radiotherapy
- This research could be used to expand their research in term of regenerative medicine of healthy tissues exposed to irradiation
- This provides a practical solution to problem of radiotherapy where healthy tissue surrounding the tumor is in the field of irradiation.

Biography:

For 25 years, he has been developing gene and cell therapy using non-human primates, immune-tolerant mice and rats to protect against the side effects of radiation. He collaborates with clinicians to develop strategies for treatment of patients after radiotherapy overexposures. He has participated in the first establishment of proof of concept of the therapeutic efficacy of Mesenchymal stem cells (MSCs) for the treatment of hematopoietic deficit, radiodermatitis and over dosages of radiotherapy. He has contributed to the first reported correction of deficient hematopoiesis in patients (graft failure and aplastic anemia) thanks to intravenous injection of MSCs restoring the bone marrow microenvironment, mandatory to sustain hematopoiesis after total body irradiation. He is scientific investigator of Clinical phase II trial evaluating the efficacy of systemic MSC injections for the treatment of severe and chronic radiotherapy-induced abdomino-pelvic complications refractory to standard therapy (NCT02814864Hirsch Index 27)

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SPEAKERS DAY 1

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SEP 17-18, 2021

Mauriane Maja*[1], Danahe Mohammed[2], Andra C. Dumitru[2], Sandrine Verstraeten[3], Maxime Lingurski[1], Donatienne Tyteca[1]

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[2]Louvain Institute of Biomolecular Science and Technology (LISBT), UCLouvain, Louvain-la-Neuve, Belgium

[3]Cellular and Molecular Pharmacology Unit (FACM), Louvain Drug Research Institute, UCLouvain, Brussels, Belgium

Role of membrane cholesterol in Breast Cancer cell invasion

Breast cancer mortality is almost exclusively due to metastatic disease. Better molecular characterization of the primary tumor is therefore crucial for a good prediction of the clinical outcome. Breast cancer cells are characterized by aberrant lipid accumulation and metabolism. However, how those changes influence plasma membrane biophysical properties and cell invasion is not understood. Taking benefit from our expertise in membrane lipid imaging and biophysics, the goal of the present study is to evaluate whether and how plasma membrane cholesterol composition and distribution contribute to breast cancer cell invasion. Using the malignant MCF10CAIa, the pre-malignant MCF10AT and the normal human mammary epithelial MCF10A cells which offer the same background, we showed that, as compared to normal and premalignant cells, malignant cells exhibit (i) a decreased cytocortex stiffness, (ii) an increased plasma membrane stiffness, (iii) a higher cholesterol content at the cell dorsal face and its distribution in submicrometric lipid domains, and (iv) a higher matrix metalloprotease (MMP)-dependent capacity of oriented invasion through Matrigel towards serum. To explore the potential involvement of cholesterol in cell invasion, cells were partially cholesterol-depleted with methyl-β-cyclodextrin (mβCD). We found a specific inhibition of oriented invasion in the malignant cells, in perfect correlation with the residual cholesterol content. Furthermore, while cholesterol depletion similarly decreased the plasma membrane stiffness of the 3 cell lines, it differentially modulated the cell cholesterol surface distribution in domains and cell:cell contacts in the malignant cells vs the premalignant and normal cells. Moreover, F-actin-enriched cortactin-positive invadopodia structures were specifically found at the ventral surface of malignant cells grown on fibronectin-coated coverslips and cholesterol depletion decreased their abundance and size. Cholesterol depletion also inhibited extracellular matrix degradation, as evidenced by the decreased size and abundance of fluorescent gelatin degradation areas. The level of inhibition was similar to the one observed upon inhibition of MMP activity by GM6001 whereas the combination of mβCD and GM6001 abrogated the effect of drugs alone, suggesting same mechanism of action/target. Altogether, our data suggest the specific dependence of malignant MCF10CAIa cells to cholesterol surface distribution for invadopodia outgrowth, extracellular matrix degradation and cell invasion. Such dependence was similarly observed for MDA-MB-231, another invasive breast cancer cell line. Our research provides new clues for the understanding of the molecular events underlying cellular mechanisms in breast cancer, a crucial step before the development of novel therapies based on targeting cholesterol in cancer cells to increase sensitivity to chemotherapeutic agents and consequently defeat multidrug resistance.

Audience Take Away:

- Our data suggest the specific dependence of malignant breast cancer cells to cholesterol surface distribution for invadopodia outgrowth, extracellular matrix degradation and cell invasion
- This present research combines the use of methodologies such as atomic force microscopy to study plasma membrane biophysical properties, live imaging confocal microscopy to evidence membrane cholesterol distribution and organization in submicrometric domains, and Matrigel invasion assay upon pharmacological treaments. Those methodologies can be applied to other cancer research projects which focus on the biology of cancer

- Our research provides new clues for the understanding of the molecular events underlying cellular mechanisms in breast cancer, a crucial step before the development of novel therapies based on targeting cholesterol in cancer cells to increase sensitivity to chemotherapeutic agents and consequently defeat multidrug resistance.

Biography:

Mauriane Maja studied Biomedical Sciences at the Université Catholique de Louvain, Belgium and graduated as MS in 2018. After a research internship at iTeos Therapeutics where she participated in the development of a new generation of highly differentiated immune-oncology therapeutics, she decided to start a PhD at the de Duve Institute under the supervision of Prof. D. Tyteca (CELL Unit). For the last three years, she has been focusing on the role of plasma membrane lipids in breast cancer cell migration and invasion. She has published a first research article in Advanced Science.

Anna Lian*, Jie Yang and Li Zhou
Department or Medicine, Brigham and Women's Hospital/Harvard Medical School, Boston, MA, USA

Identification of key pathological features associated with Melanoma survival by artificial intelligence NLP

Immunotherapy has reshaped the landscape of cancer treatment, showing promising treatment responses in melanoma. Currently, there are no pathological features which consistently predict treatment responses in melanoma; existing studies have involved small cohorts and shown conflicting results for all features except the depth of tumor invasion. Inflammatory regression and TILs are two pathological features that reflect host immune responses against cancer2-4. Currently, TILs are classified into three groups based on the presence of lymphocytes at the site of a tumor: absent, non-brisk and brisk5-6. However, there is no clear agreement as to the prognostic significance of these classifications7-10. In this study, we evaluate the prognostic value of host immune responses to melanoma by artificial intelligence (AI) approaches using natural language processing (NLP). In this retrospective cohort study that included over 2,500 patients with primary cutaneous melanoma were analyzed, NLP achieved an accuracy of 98% in feature extraction. Of the melanoma patients identified, 507 (19.3%) were deceased and 5-year survival was 74.3% (95% CI, 72.1% to 76.5%). Younger age, female sex, brisk TILs, lower Breslow thickness, lower mitotic rate, and absence of ulceration, microscopic satellites, or vascular/lymphatic invasion were independently associated with the increased survival probability (P <0.05). Absent, non-brisk, and brisk TILs were identified in 434 (16.5%), 1,916 (73.0%) and 274 (10.4%) patients, respectively. Compared to other status of TILs including absence and non-brisk TILs, brisk TILs had a 14.2% overall survival advantage at 5 years (adjusted HR, 0.7; 95% CI, 0.4 -1.0; P =.045). Our study indicate that the presence of brisk TILs is an independent prognostic factor for the overall survival of melanoma patients. This study also demonstrated that NLP can be a very effective and promising approach to build large longitudinal patient cohort that supports survival analyses.

Audience Take Away:

- Natural language processing is a very effective and promising AI approach to analyze large amount of pathology reports of patient cohort and supports survival analyses
- Our study show that the presence of brisk TILs is an independent prognostic factor for the overall survival in our melanoma cohort, as determined by natural language processing analysis of pathological reports

Biography:

Anna Lian has been working as trainee on natural language processing project to analyze pathology reports of cancer patients in Dr. Li Zhou's laboratory at Brigham and Women's Hospital.

Eleni Petsalaki*[1], Nikos Boutakoglou[1], Sergio Lilla[2], Sara Zavivan[2] and George Zachos[1]

[1]Department of Biology, University of Crete, Heraklion, Greece
[2]Cancer Research UK Beatson Institute, Glasgow, Scotland, UK

A novel mechanism that promotes mitotic spindle formation in cancer cells

During cell division, the mitotic spindle consists mainly of microtubules (MTs) and is essential for accurate distribution of the genetic material to the two daughter cells. Errors in spindle formation can lead to incorrect separation of chromosomes that is associated with tumorigenesis or developmental disorders; however, the molecular mechanisms of mitotic spindle assembly are incompletely understood. In the present study, we show that Chk1, a kinase involved in the cellular response to DNA damage, is essential for optimal density and effective polymerization of spindle MTs in human cells. Chk1 localizes to the centrosomes (the main centers of MT-organization in animal cells) in mitosis by confocal microscopy. Chk1 phosphorylates purified β-tubulin in kinase reactions in vitro at several conserved residues which were identified by mass ,spectrometry. Furthermore, reduced microtubule density in Chk1-deficient cells associates with formation of disorganized spindles. We propose that Chk1 phosphorylates β-tubulin to promote optimal spindle MT polymerization and spindle assembly. In conclusion, these findings describe a novel mechanism that could protect against tumorigenesis, through regulating mitotic spindle formation.

Audience Take Away:

- Mitotic spindle formation and tumorigenesis
- Phosphorylation of β-tubulin
- Spindle microtubule polymerisation

Biography:

Dr Eleni Petsalaki is a Post-Doctoral Research Scientist in Dr George Zachos' lab at the University of Crete, in Greece. She completed her PhD in 2014 in Molecular Biology and Biomedicine at the Department of Biology in Crete. Her main research interest is to understand mechanisms of mitotic cell division in human cells, such as the mitotic spindle and abscission checkpoints. She has published 14 papers in leading scientific journals (13 first name publications) such as the Journal of Cell Biology (5), Nature Communications (1), Journal of Cell Science (2) and others, and her work has received >200 citations.

Kalliopi Megari
Aristotle University of Thessaloniki, Thessaloniki, Greece

Neuropsychology of breast cancer patients

Post chemotherapy cognitive impairment (PCCI), is referred to a decrease in neuropsychological performance of neurocognitive measures after chemotherapy for the treatment of cancer. Chemotherapeutic drugs are often affecting both normal and cancer cells and the cause of cognitive impairment observed in some individuals following chemotherapy treatment. Breast cancer patients complain about cognitive difficulties during and after cancer treatment. We investigated the manifestation of cognitive impairment related to chemotherapy, before chemotherapy (T1), immediately after chemotherapy-1 day (T2) and 6 months later (T3), among 187 adult patients with different types of cancer (breast, colorectal, prostate and thyroid cancer). Cognitive functions were assessed, such as attention and working memory, visuospatial perception, executive functions, complex scanning and visual tracking, as well as short and long-term memory using a battery of neuropsychological tests. We had an assessment of emotions, such as anxiety, depression, positive and negative mood to investigate the emotional functioning of cancer patients. Results revealed a statistical significance in performance, immediately and 6 months post-chemotherapy (T3), although no statistically significant differences were found between the groups in any of the neuropsychological test, before chemotherapy. Patients showed lower performance immediately post-chemotherapy (T2) that remained stable 6 months post-chemotherapy (T3), compared to T2 in all cognitive domains ($p<0,001$). Patients with breast cancer showed significantly lower performance on all cognitive domains compared to other patients. In addition, all patients had a lower performance at T2, which means low emotional functioning with no statistical significant changes. At T3 all patients, had an increased performance with increased emotional functional 6 months post-chemotherapy. Cognitive change that can be detected with repeated testing is essential for an accurate interpretation of neuropsychological performance in studies with cancer patients.

Biography:

Dr. Kalliopi Megari is an experienced psychologist working in the hospital & health care industry. She is a lecturer at University of Western Macedonia in Greece. Skilled in Clinical Neuropsychology, Clinical Research and Learning Disabilities. Graduated from Aristotle University of Thessaloniki and attended further education from University of Macedonia, in people with special needs and disabilities. She holds undergraduate degrees in Nursing and Psychology, as well as a Master's and a PhD in Neuropsychology from Aristotle University of Thessaloniki. She has many years of experience working with chronic disease patients as well with people with disabilities. Her work has earned her many prestigious international awards. She has given lectures at Aristotle University of Thessaloniki and University of Warsaw. She is postdoctoral researcher and has published more than 10 research articles in journals. She is the Global Engagement Representative of International Neuropsychological Society, General Secretary of the booard of directors and member of the Ethics Committee of Hellenic Neuropsychological Society.

Izabela Mlynarczuk-Bialy*[1]. Agata Gawel[2], Pawel Tyrna[2], Lukasz P. Bialy[1]

[1]Department for Histology and Embryology, Medical University of Warsaw, Chalubinskiego 5, 02-004 Warsaw, Poland
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Entosis in cancer: Death or survival?

The process of entosis is not widely known phenomenon, that can impact diagnosis and prognosis of cancer. The phenomenon is characterized by the active invasion of one cell into another and the formation of "cell in cell structures" (CIC), with the participation of Rho-ROCK kinase and actin-myosin complexes. Clinical studies show that the presence of CIC can result in worse prognosis. This process may be also important in the biology of cancer since more malignant tumors, under conditions of starvation, hypoxia and metastasis, show a higher percentage of entoses. Several molecular agents involved in entosis process like Aurora A Kinase, CDC42, Atg5, Atg7, LC3, mTOR are identified. Little is known about triggering factors of entosis, why some tumors demonstrate CIC and the other (also of the same type), not. Studying of entosis is also challenging: the dynamics of cell in cell invasion can be observed in cell culture, while the post-operative cancer tissue represent some fixated stage like the "still nature", where dynamic studies are impossible. Despite these difficulties, we know quite lot about the phenomenon and its possible impact on cancer prognosis and diagnosis. In the research on entosis we need introduction of international standards and unification in distinguishing entosis from other processes, compilation of cell culture studies with histopathological studies. The first definition of entosis implicated that it is a new cell death since most entotic cells in that experiments died within lysosome of the host cell. Other data, including clinical ones, indicate that entosis process can be beneficial for tumor cells: supporting their survival, gain of nutrients and promoting more genetically instable cancer cells. Summarizing, it is good to consider once again what is entosis: death or survival. Especially in the context of clinical cancer biology.

Audience Take Away:

- The audience will be familiar with the entosis process in cancer
- Entotic index can be used as additional prediction factor in the cancer research
- Discussion on unification and setting multicenter standards in the field of entosis research
- Can entosis be a novel diagnostic factor indicating cases that need special care because of worse prognosis?

Biography:

Dr. Izabela Mlynarczuk-Bialy studied Medicine at the Medical University of Warsaw. Before graduation she was involved in following projects: the role of statins in cancer; biology of the proteasome system within National Grant to Dr C. Wójcik. She graduated as MD in 2001. Pre-doc and post-doc fellowship in Institute for Biochemistry, Charite Medical School, Berlin, Germany. Tutor: Prof PM Kloetzel. Characteristics of novel proteasome inhibitor BSc2118 in preclinical cancer therapy. Scholarship holder of DAAD, UICC, EMBO and Charite. 2004 - PhD degree. 2019 - Habilitation at WUM. Associate Professor at WUM. Tutor of the Students Scientific Association at the Department for Histology and Embryology. Research on experimental cancer therapy, intracellular protease systems and entosis process.

Sergio Alexandre Alcantara dos Santos* Ph.D, Ana Carolina Lima Camargo Ms; Flavia Bessi Constantino Ms; Ketlin Thassiani Colombelli Ms; Luiz Marcos Frediani Portela; Sergio Luis Felisbino Ph.D; Luis Antonio Justulin Junior Ph.D

Sao Paulo State University, UNESP, Brazil

Can prostate cancer originate in utero? Prostate carcinogenesis induced by maternal low protein diet in older off spring

Although the carcinogenesis is frequently linked to genetic background, exposure to environmental risk factors has gained attention as the etiologic agent for several types of cancer. The intrauterine microenvironment has been described as preponderant factor for offspring health; and maternal exposure to insults have been linked to chronic disease in aged offspring. Using a model of maternal exposure to low protein diet (LPD; 6% protein), we demonstrated that impairment of offspring rat prostatic growth at post-natal day (PND) 21 was associated with prostate carcinogenesis in aged offspring.

Audience Take Away:

- The audience will can use the information on fetal programming, relating diseases that occur in adults with changes that occur during the ges.ing (mainly metabolic diseases) have their beginning in the embryonic/fetal period, so we to instigate the study of the development of prostate cancer early in the development.
- Intrauterine alterations caused by fetal programming, observed using global analysis tools (proteomics, mirnome and transcriptome) that remain in the individual and are related to the development of different types of cancer, giving special attention to prostate cancer.

Biography:

Sergio Alexandre Alcantara Dos Santos is a postdoc at the State University of São Paulo, UNESP, with experience in the areas of prostatic morphology, analysis of signaling pathways and bioinformatics tools. We sought markers for the prostatic cancer development induced by fetal programming by maternal low protein diet.

Ana Luiza de Castro Conde Toscano
Medical Division, Emilio Ribas Institute of Infectology, Sao Paulo, SP, Brazil

AIDS related Kaposi sarcoma in Brazil

Kaposi's sarcoma (KS) is a mesenchymal tumor, caused by herpes virus type 8 (HHV-8), and a characteristic neoplasm of acquired immunodeficiency syndrome (AIDS). After the emergence of AIDS in 1980, a more aggressive form of the disease, more common among homosexual or bisexual men and associated with the human immunodeficiency virus (HIV), was documented. It was soon recognized as an epidemic form of KS, which had a big impact on the Public Health due to its high magnitude and mortality. KS was the first opportunistic infection recognized in association with HIV and is still the most common neoplasm related to AIDS. The introduction of highly active antiretroviral therapy (HAART) helped to strengthen the immune system of people infected with HIV and lowered the risk of these people to develop KS. Nevertheless, KS is still considered the most prevalent tumor among this population. Although HAART does not interfere directly with the replication of HHV-8, cases of regression of KS-AIDS lesion have been observed frequently after the use of HAART in combination with chemotherapy or radiotherapy,5 though the mechanism by which HAART leads to the regression of KS remains controversial.2,3 The incidence of KS has substantially declined due to HAART,4 in the same way that KS-AIDS became less aggressive when compared to KS among individuals with no antiretroviral treatment.6,7 In Brazil, from 1996 to 2010 - already in the post-HAART era -, the incidence of KS was still 2.5 times higher than in the United States of America (USA), and it remains as the most common neoplasm in HIV carriers.8 The aim of this presentation is to describe trends in prevalence of Kaposi's sarcoma in patients with AIDS and identify the factors associated with the occurrence of this neoplasm in Brazil.

Audience Take Away:

- Background: Aids treatment in Brazil
- Kaposi´s Sarcoma Prevalence and risk factors;
- Treatment Brazilian Consensus
- Current Chalenges in diagnosis and treatment

Biography:

Ana LCC Toscano, MD, holds a master's degree in Infectious and Parasitic Diseases from the Faculty of Medicine of the University of São Paulo, Brazil (2015) and a Medical Residency in Infectious and Parasitic Diseases from the Emilio Ribas Infectious Diseases Institute (2002). She graduated in Medicine from the Faculty of Medical Sciences of Santos (1998). She is currently Medical Team Supervisor at Emilio Ribas Infectious Diseases Institute and a member of the Research Ethics Committee (IRB). She is clinical researcher in national and international clinical projects. Hers research articles have been cited more than fifty times.

Paola Marcato*, Marie-Claire Wasson, Justin Brown
Department of Pathology, Dalhousie University, Halifax, NS, Canada

Genome-wide comparative analysis of long non-coding RNAs versus protein-coding transcripts reveals distinct expression profiles and patient survival correlations across cancer types

Long non-coding RNAs (lncRNAs) are emerging targets for the diagnosis and therapeutic treatment of cancer. LncRNAs are non-protein-coding transcripts that regulate gene expression by modulating chromatin structure or by interacting with other RNA species. Additionally, they exhibit high tissue- and context-specific expression, marking them as attractive biomarkers. Of the 12,727 lncRNAs identified in human cancers, the vast majority of these remain completely uncharacterized, limiting our understanding of the role of this group of non-coding RNAs. In contrast, the role of protein-coding transcripts as a class has been extensively characterized in cancer biology. To gain insight into the relative impact of lncRNAs in cancer progression in comparison to mRNAs, we conducted a comparative analysis of the two RNA species utilizing patient tumour expression and survival data in 6 cancer types. Our analysis revealed that lncRNAs exhibit distinct expression patterns across cancers. We determined which lncRNAs are associated with patient outcomes through conducting survival analyses for each lncRNA and mRNA in each cancer type assessed. This determined that lncRNAs are as involved as mRNAs in impacting patient outcomes. Additionally, we found that the function of lncRNAs is highly cancer-dependent, with some lncRNAs playing oncogenic roles in some cancers while exhibiting benign functions in others. Our results indicate that lncRNAs enriched in tumour tissues are more oncogenic, while those depleted in tumor tissues are more tumor suppressive. This study demonstrates that lncRNAs are clinically relevant players in the progression of cancer and merit further investigation as potential therapeutic targets.

Audience Take Away:

- Learn about the long non-coding RNA and their role in cancer
- Gain an expanded appreciation of now long non-coding RNAs can be targeted in the treatment of cancer
- Learn about how long non-coding RNA genes as a class compare to protein coding genes in their overall impact in cancer.

Biography:

Dr. Paola Marcato obtained her BSc (Cell Biotechnology) and PhD (Medical Microbiology and Immunology) from the University of Alberta. Dr. Marcato went on to complete postdoctoral training on cancer research with Dr. Patrick Lee at Dalhousie University. In 2012, Dr. Marcato started a breast cancer research laboratory at Dalhousie University. The research projects in Dr. Marcato's laboratory have the long-term goal of developing improved therapeutic strategies for breast cancer based on a precision medicine approach. They use transcriptome and genome-wide functional assays, cell lines, patient-derived xenografts (PDXs), mouse tumor models, and analysis of published patient datasets to identify biomarkers, study breast cancer stem cells (CSCs) and understand factors important in breast cancer progression. These factors include epigenetic modifications such as DNA methylation and long non-coding RNA.

Luigi Marongiu
Ruprecht-Karls University of Heidelberg, Germany

Increased viral richness in colorectal cancer tissues

Colorectal cancer (CRC) is a major burden for the Public Health worldwide. The modification of the relative prevalence of the micro-organisms present in the patient's gut (dysbiosis) has been shown to be linked to an increased risk of CRC. Nevertheless, the connection between dysbiosis and CRC is still poorly recognized. In the present study, 24 patients with matched normal/cancer colon tissues and liver metastases were analyzed using Illumina-based massively parallel sequencing (MPS). Viral and bacterial richnesses were evaluated. Virome analysis showed that phages were the most preponderant viral species (46%), and primary CRCs were enriched for bacteriophages. Bacteriome analysis showed Fusobacterium nucleatum, *Streptococcus sanguinis, F. Hwasookii, Anaerococcus mediterraneensis* and further species enriched in primary CRCs. In conclusion, these data supported and extend the notion that dysbiosis is associated with an increased risk of CRC and strengthened the case for further investigation of the role of bacteriophages in human cancer.

Audience Take Away:

- This presentation is related to a microbiome assessment of colorectal cancer tissues
- Human cancer might not always be due to oncogenic viruses
- In planning research related to cancer with an infectious aetiology, the role of bacteriophages should be taken into account.

Biography:

Dr. Marongiu studied Molecular Biology at the University of Rome 3, Italy, and received his PhD degree in Virology at the University College London, UK. He completed one postdoctoral project at the University of Cambridge, UK, on the genetic analysis of Noroviruses, and a second at the University of Edinburgh, Scotland, on veterinary viruses. He carried out the project on the metabiome analysis of cancer tissues presented herein at the University of Heidelberg. He is now employed as Research Associate at the University of Hohenheim, Stuttgart, Germany, studying the connection between viral infection and vitamin C.

Romi Gupta[1*], Suresh Bugide[1], Douglas B Johnson[2], Michael Green[3], Narendra Wajapeyee[1]

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[3]Department of Molecular, Cell and Cancer Biology, UMASS, Worcester, Massachusetts, USA

KLF7 promotes pancreatic cancer growth and metastasis by up - Regulating ISG expression and maintaining Golgi complex integrity

Pancreatic ductal adenocarcinoma (PDAC) is the fourth- leading cause of cancer-related deaths in the United States and is expected to become the second-leading cause by 2020. The five-year disease-free survival rate for patients with PDAC is extremely low and has remained <10% for several decades. Currently, there is no effective therapy for PDAC, and even immunotherapies that have worked effectively in patients with other cancer types have failed to provide meaningful clinical benefits in patients with PDAC. Therefore, further molecular and functional evaluation of PDAC is needed to identify and develop better therapeutic strategies. In our study we show that the transcription factor Krüppel-like factor 7 (KLF7) is overexpressed in PDACs, and that inhibition of KLF7 blocks PDAC tumor growth and metastasis in cell culture and in mice. KLF7 expression in PDACs can be up-regulated due to activation of a MAP kinase pathway or inactivation of the tumor suppressor p53, two alterations that occur in a large majority of PDACs. ShRNA-mediated knockdown of KLF7 inhibits the expression of IFN-stimulated genes (ISGs), which are necessary for KLF7- mediated PDAC tumor growth and metastasis. KLF7 knockdown also results in the down-regulation of Discs Large MAGUK Scaffold Protein 3 (DLG3), resulting in Golgi complex fragmentation, and reduced protein glycosylation, leading to reduced secretion of cancer-promoting growth factors, such as chemokines. Genetic or pharmacologic activation of Golgi complex fragmentation blocks PDAC growth and metastasis similar to KLF7 inhibition. Our results demonstrate a therapeutically amenable, KLF7-driven pathway that promotes PDAC growth and metastasis by activating ISGs and maintaining Golgi complex integrity.

Biography:

Dr. Gupta did her BS in microbiology and MS biochemical technology in India. She further obtained her PhD from Max Planck Institute for Molecular Genetics, Berlin, Germany in the area of ribosome biology and protein translation. After that she moved to Yale University where she performed extensive studies on identification of new regulators of cancer growth and progression. Many of her studies are published in journals like eLife, PNAS, Cell Reports, Oncogene etc. Currently she is Assistant Professor in the UAB and Associate scientist at O'Neal Comprehensive Cancer Center at UAB.

Tapasree Roy Sarkar*[1,2], Nathalie Sphyris[1]

[1]Department of Translational Molecular Pathology, University of Texas MD Anderson Cancer Center, Houston, TX

[2]Department or Biology, Texas A&M University, College Station, TX 77843

The role of EMT in angiogenesis-mediated tumor progression

Hypoxia stimulates angiogenesis, promotes tumor growth, and triggers the epithelialmesenchymal transition (EMT), which bestows cells with mesenchymal traits and multi-lineage differentiation potential. In this study, we investigated whether EMT can confer endothelial attributes upon carcinoma cells, augmenting tumor growth and vascularization. Human epithelial breast cancer cells (MCF7) were orthotopically injected into mice. The tumors of different sizes were harvested and immunostained for markers of hypoxia and EMT. Larger tumors were found to be well-vascularized with CD31-positive cells of human origin. It was observed that hypoxic regions in the tumors (demarcated by HIF-1α staining), exhibited E-cadherin loss and elevated levels of mesenchymal markers such as vimentin and FOXC2. When MCF-7 cells were implanted, co-mixed with human mammary epithelial (HMLE) cells overexpressing the EMTinducer Snail, markedly potentiated tumor growth and vascularization, compared with MCF-7 cells injected alone or co-mixed with HMLE-vector cells. Intra-tumoral vessels contained CD31-positive cells derived from either donor cell type which indicated the mesenchymal to endothelial transdifferentiattion. FOXC2 knockdown was found to abrogate the potentiating effects of HMLE-Snail cells on MCF-7 tumor growth and vascularization, and compromised endothelial transdifferentiation. Therefore, we concluded cells that have undergone EMT can promote tumor growth and neovascularization by promoting endothelial transdifferentiation of carcinoma cells, with FOXC2 playing key roles in these processes.

Audience Take Away:

- How EMT-induced cells can transdifferentiate into endothelial cells and augments tumor growth
- How tumors cells can be independent and can make their own blood vessels
- How FOX2 plays an important role in EMT-mediated endothelial Trans differentiation.

Biography:

Tapasree Roy Sarkar received her PhD degree from Purdue University. Then she did her postdoctoral research at National Cancer Institute/ National Institute of health (supervised by Dr. Esta Sterneck) and University of Texas M.D. Anderson Cancer center (Supervised by Dr. Sendurai Mani). After her postdoctoral work she joined Department of Biology at Texas A&M University as a faculty. She has published more than 21 research articles in reputed journals such as PNAS, Oncogene, Cancer research, Oncotarget etc.

Amarasooriya M. D. S. Jayawardhana*[1], Morgan Stilgenbauer[1], Payel Datta[1], Zihan Qiu[1], Sarine Mckenzie[1], Han Wang[1], David Bowers[1], Manabu Kurokawa[2] and Yao-Rong Zheng[1]

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[2]Department of Biological Sciences, Kent State University, Kent, OH 44242, USA

Fatty acid-like Pt(IV) prodrugs overcome cisplatin resistance in ovarian cancer by harnessing CD36

This is the first study of engineering mitochondria-damaging fatty acid-like Pt(IV) prodrugs (FALPs) to harness CD36, a fatty acid translocase, to treat drug resistant ovarian cancer. Mitochondria-damaging therapeutics have been proven effective against drug resistant ovarian cancer cells, but their systematic toxicity is higher. To lower the toxicity and increase the therapeutic window, there is a need to generate mitochondria-damaging therapeutics with high specificity against ovarian cancer cells. CD36 is a transmembrane protein responsible for facilitating uptake of free fatty acids, and it is upregulated in ovarian tumors. We hereby employed Pt(IV) prodrugs that mimic the fatty acid structure and act as "Trojan horse" to exploit CD36 for ovarian cancer treatment. First, the CD36-dependent cell entry of FALPs was confirmed by graphite furnace atomic absorption spectroscopic (GFAAS) analysis using ovarian cancer cells with different levels of CD36 expression. Unlike fatty acids that fuel lipid metabolism, these FALPs induce mitochondrial damage and kill ovarian cancer cells. Mitochondrial accumulation and activation of FALPs were attested using fluorescein imaging, and GFAAS analysis. Corresponding mitochondrial damages were validated by MitoSOX and Mitostatus flow cytometric analysis. Finally, the high potency against cisplatin-resistant ovarian cancer cells was confirmed by cell viability assays. Overall, this work demonstrates an innovative design that allows for selective activation of mitochondria-damaging Pt therapeutics at chemo-resistant ovarian cancer cells.

Audience Take Away:

- This research explains that Pt (IV) compounds with fatty acid like structure use CD36 (a transmembrane protein that transports lipids into cells) for higher cellular uptake.
- Fatty acid like Pt (IV) compounds having Positively charged moieties accumulates in mitochondria and damage them to eradicate cancer cells (mainly cancer stem cells that have higher mitochondria mass)
- By eradicating cancer stem cells (CSCs) these Pt (IV) compounds can overcome drug resistance in ovarian cancer.
- Overall, this project demonstrates an innovative idea for a researcher to design and improve cancer drugs to overcome drug resistance.

Biography:

Amarasooriya Jayawardhana studied chemistry in University of Peradeniya, Sri Lanka and graduated in Chemistry with a second-class upper in 2008. At the same university she completed Master of Philosophy degree in Bioinorganic chemistry in 2012. During that period, she published five articles in Sri Lankan national journals. In 2017, she started her PhD degree in Chemistry at Kent State University, USA. Amarasooriya has successfully completed several projects related with Pt(IV) therapeutics to treat cancer. She has published two first author papers and two co-author papers in high rank journals. Currently, she is studying on Photoactivatable Pt(IV) therapeutics for Light-controlled elimination of CSCs with CD36 targeting Pt (IV) compounds.

George Zachos* and Eleni Petsalaki
Department of Biology, University of Crete, Heraklion 70013, Greece

Delaying the final cut: An ATM-Chk2-INCENP pathway prevents chromatin bridge breakage in cytokinesis

Chromatin bridges are strands of missegregated chromatin connecting the anaphase poles or daughter nuclei and have been linked to tumourigenesis. In response to chromatin bridges in cytokinesis, cells delay abscission, the severing of the narrow cytoplasmic canal that connects the two daughter cells, to prevent chromatin breakage or tetraploidization by regression of the cleavage furrow that are associated with genomic instability and cancer predisposition. In mammalian cells, this abscission delay is called "the abscission checkpoint" and is dependent on the localization of the Chromosomal Passenger Complex (CPC) at the midbody. The CPC comprises the catalytic subunit Aurora B kinase, the scaffolding protein INCENP and the nonenzymatic subunits Survivin and Borealin; however, the molecular mechanisms that signal chromatin bridges to the CPC are incompletely understood. In the present study, we show that inhibition of the DNA damage kinases ATM or Chk2 impairs CPC localization to the midbody and correlates with premature abscission and chromatin breakage in cytokinesis with trapped chromatin in human carcinoma cell lines. ATM phosphorylates Chk2 threonine 68 (T68) to activate Chk2 at the midbody. In turn, active Chk2 phosphorylates INCENP at the newly identified site serine 91 (S91) to promote CPC localization to the midbody, to delay abscission. Expression of siRNA-resistant phosphomimetic mutant INCENPS91D, but not the wild-type protein, rescues CPC localization to the midbody and prevents chromatin breakage in Chk2-deficient or ATM-deficient cells. In contrast, in the absence of the endogenous INCENP, the nonphosphorylatable mutant INCENPS91A does not localize to the midbody and its expression promotes chromatin breakage. These results identify an ATM-Chk2-INCENP pathway that prevents chromosome breakage in cytokinesis with chromatin bridges, by promoting CPC-midbody localization, through Chk2- mediated INCENPS91 phosphorylation.

Audience Take Away:

- Chromatin bridges and carcinogenesis
- Mechanisms that maintain genome integrity in cytokinesis
- The abscission checkpoint

Biography:

George Zachos completed his PhD at the University of Crete in 1997. He then received postdoctoral training in the Beatson Institute for Cancer Research, Glasgow, U.K. before moving, in 2008, to the Department of Biology, University of Crete, Heraklion, Greece as an Assistant Professor in Cell Biology. In 2015, he became Associate Professor and continues to hold this position today. Discoveries from the Zachos lab have identified mechanisms that regulate the fidelity of chromosome segregation in mitotic cell division in higher eukaryotic cells. He has published over 40 papers in leading scientific journals and his work has received ~2,000 citations.

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POSTERS DAY 1

4TH EDITION OF

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SEP 17-18, 2021

ICC 2021

[1]*Yevheniia Radzishevska, [2]Leonid Vasil'ev

[1]Department of Medical and Biological Physics and Medical Informatics of the Kharkiv National Medical University, Head of group for Medical Informatics of the State organization «Grigoriev Institute for Medical Radiology and Oncology of the National Academy of Medical Sciences of Ukraine», Kharkiv, Ukraine
[2]Head Doctor of the State Institution "Institute of Medical Radiology after S.P. Grigoriev "of the National Academy of Medical Sciences of Ukraine, Senior Researcher, Kharkiv, Ukraine

Oncologic patients have mutual relations of nosological form of the first tumour and second cancers

The aim of presentstion is to demonsrate the relationship between nosological forms of the primary and second neoplasms in patients who have undergone special treatment for cancer, and who have developed second neoplasms three and more years after the end of treatment. It was realized by means of comparing the nosological structure of second neoplasms that developed in 203 patients undergoing treatment for oncological pathology with the official data on the specific weight of 10 major nosological forms of malignant neoplasms in Ukraine. The statistical significance of the obtained have been estimated using confidence intervals normalized taking into account the specificity of the patients of the clinic where the study was conducted. In accordance with the specific weight of the nosological forms of the primary neoplasms, conditioned first by the specialization of our clinic, the study groups have been divided into 5 nosological subgroups: patients with breast, thyroid, uterine, cervical, ovarian, and subgroup with other forms of cancer. For each of the subgroups, the nosological structure of the second neoplasms have been investigated and compared with the overall structure of malignant neoplasms according to the official statistics. It was shown that the frequencies of five allocated nosological forms calculated for the clinic correspond fairly well to the population mean, while for two most representative nosological forms the correspondence is within the expected stochastic variability. Thus, it was demonstrated that there are no statistically significant differences between the percentage composition of 10 major nosological forms of cancer in Ukraine and the structure of nosological forms of second neoplasms in the study group of patients. The nosological form of second neoplasms is not a consequence of the primary cancer, but reproduces the overall nosological structure of the incidence of malignant neoplasms.

Audience Take Away:

- Effective methods of treatment and early diagnosis have resulted in a significant increase in the survival of cancer patients. For example, in the USA, the number of patients who have survived after the primary cancer is 3.5% and increases annually by almost 1,000,000. However, against the background of a general increase in life expectancy, another problem arises sharply: the problem of the secondary cancer. According to statistical data, the incidence of recurrent neoplasms in surviving patients is 16%. Ambiguous in its solution is the relationship between the nosological forms of the primary and second neoplasms. The overwhelming number of numerous foreign studies aimed at studying the problems of second neoplasms, are carried out within the framework of one primary oncological pathology and investigate the connections between the primary and second cancers, taking for the dogma the dependence of the second-cancer nosologies on the nosological form of the primary tumor.
- It will be demonstrated that there are no statistically significant differences between the percentage composition of 10 major nosological forms of cancer in Ukraine and the structure of nosological forms of second neoplasms in the study group of patients
- The nosological form of second neoplasms is not a consequence of the primary cancer, but reproduces the overall nosological structure of the incidence of malignant neoplasms.

Biography:

Dr. Radzishevska studied mathematics at the Kharkiv University, Ukraine. Then she began working at the Institute of Medical Radiology as a researcher and, in addition, from 1998 she began to teach medical informatics at the Kharkiv National Medical University (KhNMU). She completed her Ph.D. in 1997 and now holds the position of Associate Professor at KhNMU and the head of the medical informatics group at the Institute of Medical Radiology. She is engaged in the statistical processing of medical data. She has published more than 160 scientific papers, in particular, 7 papers in SCI (E) journals and has certificates for 38 objects of the right of intellectual property of Ukraine.

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KEYNOTE FORUM

DAY 2

4TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

SEP 17-18, 2021

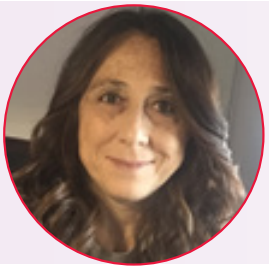
Yves-Marie Robin
Biology-pathology wing, Oscar Lambret Cancer Center, France

Real-time molecular diagnosis of soft tissue tumors using water-assisted laser desorption/ionization mass spectrometry technology

A substantial number of lesions in soft tissue pathology requires diagnostic precision for adequate patient care management and follow-up. Also, margin status of surgical specimens must be unequivocally defined, preferably intra-operatively, because of the tight relation which exists between the quality of safe tumor margin delineation and remission. New discriminative diagnostic tools are needed to deal with these issues. Using canine tissues, we assessed the performance of a recently developed system, termed SpiderMass, relying on mass spectrometry (MS) for molecular lipid-based real-time diagnosis of sarcomas using endogenous water as matrix. SpiderMass uses a laser microprobe operating under ambient conditions and remotely fromrm the MS instrument with excitation of endogenous water molecules generating negligeable tissue destruction. 1-mm-thick sections obtained from 33 canine biopsies of various sarcoma subtypes such as fibrosarcoma, osteosarcoma and undifferentiated sarcoma were subjected to SpiderMass analysis. Results indicated that classification models based on moprhological criteria such as normal, cancerous and necrotic tissue , tumor grade, and subtypes showed a minimum of 97.63% correct classification using data collected with the system. Specific markers of normal, cancer, and necrotic regions were identified by tandem MS and validated by MS Imaging. SpiderMass differentiated grade 3 sarcomas from normal tissue and from grade 1 and 2 lesions and further subdivided specific tumor types with a high accuracy. Real-time detection capabilities were demonstrated by ex vivo analysis . We are currently focusing on applying the technique on human soft tissue tumors (maligant and benign) for a proof of concept with clinical samples aimed toward two novel developements : first, set up databases in the form of classification models of tumor sub-types and grades with their corresponding specific lipidomic profiles so as to construct histo-lipidomic classfications and, secondly, introduce in the instrumentation an artificial intellignence-type algorithmic bioinformatics system enabling it to rapidly consult, process and translate the databases into a point-of-care real-time diagnostic tool.

Biography:

Yves-Marie Robin, MD, was born in Haiti on may 24th 1959 and graduated from Vassar College in Poughkeepsie, New York (USA). He has been since 2006 head of the department of Morphological and Molecular Pathology Unit at the Oscar Lambret Cancer Center, Lille, (France), one of the twenty anti-cancer institutions in France which he joined upon a completing his internship and graduationfrom the faculty of medicine of Clermont-Ferrand in 1999. Dr Robin is a member of the French Sarcoma Group which is known for its expertise and research accomplishments in the field of soft tissue tumors, and has published or collaborated on a number of publications in different areas of interest, mainly soft tissue tumors. Dr Robin is a research member of the Mass Spectrometry INSERM laboratory (PRISM) at Villeneuve d'Ascq (France) directed by Pr Michel Salzet. He has recently been appointed chief coordinator of the biology-pathology wing at Oscar Lambret Center whose main objective is to set standards of laboratory practice in pathology and molecular oncology adapted to clinical needs and translational research.



Marina De Rosa

Department of Molecular Medicine and Medical Biotechnology, University of Naples Federico II, 80131 Naples, Italy

Familial gastrointestinal polyposis syndromes: Lesson from next-generation sequencing analysis

Gastrointestinal polyposis syndromes are a heterogeneous group of rare precancerous syndromes classified as adenomatous and hamartomatous, based on the polyps' histology. Among the adenomatous polyposis syndromes are familial adenomatous polyposis (FAP), MUTYH-associated polyposis (MAP) and Polymerase proofreading associated polyposis (PPAP). Hamartomatous polyposis syndromes include: Peutz-Jeghers syndrome, juvenile polyposis and PTEN-linked hamartomatous tumour syndrome. Two gene panels, specific for the adenomatous (including the APC, MUTYH, POLE, NTHL1, AXIN2, POLD1 genes) and hamartomatous (including the PTEN, STK11, SDHB, SDHD, BMPR1A, AKT1, CDH1, AKT1, SMAD4, PI3KCA, ENG genes) polyposis diagnosis, have been designed and validated. Using these panels, 83 subjects with clinical suspicion/diagnosis of polyposis, and previously analyzed by Sanger sequencing of one of the following genes: APC, MUTYH, PTEN and STK11, were re-analyzed by NGS sequencing. Afterwards, variants identified of unknown pathogenetic significance (VUS) and the variants that had Minor Allele Frequency (MAF) <0.05, were subjected to in silico analysis, to evaluate their possible involvement in the pathological phenotype, using the following software and databases: Mutation Taster, Human Splicing Finder, Polyphen-2, UMD Predictor, Sift, InterVar, InSiGHT, ClinVar. All variants were also classified according to the American College of Medical Genetics and Genomics (ACMG) criteria. The results obtained by NGS analysis showed a 100% concordance with those obtained by Sanger sequencing. Pathogenic nucleotide variants were identified in 42% of FAP patients, 23% of MAP patients, 45% of PJS patients and 0% of PHTS patients. In addition, we identified a pathogenetic variant in the AXIN2 gene, in a woman who presented attenuated polyposis, with autosomal dominant inheritance. Among the rare variants in silico analyzed, additional 27 variants were found to be VUS/potentially pathogenic. However, a discrepancy emerged by the classification of these 27 variants between the verdict according to the ACMG criteria and that reported in the InSiGHT and ClinVar databases. Seven variants that were benign or probably benign according to the ACMG criteria, were found to be VUS according to the InSiGHT and/or ClinVar databases; on the contrary, 3 mutations that were probably pathogenetic according to the ACMG criteria, were found to be VUS according to InSiGHT and ClinVar. In agreement with the ACMG criteria, other probably pathogenic variants of the MUTYH gene were identified in 3.6% of patients, while VUS were identified in POLE (3.6%), APC (2.4%), NTHL1 (2.4%), STK11 (1.2%), SDHD (1.2%), PI3KCA (1.2%) and ENG (1.2%) genes. All these variants were not reported into the InSiGHT and ClinVar databases. We emphasize the need to follow common and shared guidelines for the clinical interpretation of DNA variants identified by NGS. In our opinion, databases and bioinformatic analysis are currently of great utility to select VUS/probably pathogenic variants, whose role will then have to be clarified through molecular biology studies. Furthermore, many VUS are in genes not often tested for molecular screening of colorectal polyposis. A big deal of work will have to be done in the next future to clarify the clinical significance of these variants, and therefore the role of the respective genes in the pathogenesis of familial colorectal polyposis.

Audience Take Away:

- The presentation will expose the clinical and molecular characteristics of hereditary gastrointestinal polyposis, highlighting their heterogeneity, both at molecular and clinical level.
- The relevance of molecular screening to allow a precise diagnosis and to prevent cancer onset will be clarified.
- The need to use NGS techniques for analysis of multigenic panels to perform an adequate molecular characterization of the disease will be underlined.
- The main problems related to the interpretation of the variants identified by sequencing of gene panels with NGS technology will then be discussed.
- I believe that the talk will be very useful to all researchers interested in molecular diagnostics of hereditary colorectal cancers, but also of other hereditary diseases. Indeed, the communication will show the role of molecular diagnosis of polyposis syndromes, our best methodology to perform it and the main problem that, nowadays, must be addressed.

Biography:

Marina De Rosa (MDR) is Associated Professor of "Biochemistry" at the University of Naples Federico II. She received the title of PhD in Biotechnology in 1998. In 2000 she obtained the title of specialist in Biochemistry and Clinical Chemistry. She is Editorial Board Member of the Experimental and Therapeutic Medicine journal-Spandidos publications. Reviewer Board member of the "Cancers" journal-MDPI journal. and "Membranes" journal-MDPI journal. She is author of more than 95 papers. Of these, 45 are articles on indexed scientific journals with a total of 862 citations, achieving an h-index of 18, according to Scopus and PubMed database.

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SPEAKERS DAY 2

4TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

SEP 17-18, 2021

Mehrasa Nikandish
University of Georgia, Georgia

Types of cancer

As it is obvious, nowadays cancer is one of the most common disorders all around the world and nobody can ignore the importance of getting educated about them, types, how they spread around the body and how they can be treated and also some procedures after treatment because every person has his/her own individual body and depend on their body immune system it's important to do some caring after the treatment. In this presentation, I try to explain about different types of cancer and how we can recognize the types. Actually there are four main types of cancer that include the carcinomas that begins with the tissues that cover the surface of the body and covers the internal glands and organs as well, sarcomas that begins in the tissues that thir function is to support and connect the body. Leukemias that is the cancer of the blood and lymphomas that is the cancer of lymph. The most common cancer among women that is reported in the U.S. is breast cancer and among the men is prostate cancer. Among different types of cancers the most curable cancers are breast cancer, prostate cancer, testicular cancer, thyroid cancer, melanoma, cervical cancer, Hodgkin lymphoma and the hardest cancer for treating is the pancreatic cancer because it develops quickly with few symptoms that makes it one of the most deadly forms of cancer and also pancreatic cancer is resistant to chemotherapy and the new clinical trials are taking place to develop the alternative treatments instead of chemotherapy.

Audience Take Away:

- Introduction of cancer
- Introduction of different types of cancer
- Cancer statistics
- The most curable cancers and the hardest type of cancer for treating and why?

Biography:

Miss Mehrasa Nikandish, high GPA (4) and third year student in pharmacy department from university of Georgia, Tbilisi. I start doing researchers from 2018 with some abstracts and articles and participate in different international conferences as the speaker. Last year my first full article was published in ESJ journal and I am doing my best job to become one of the good researchers. Also I did some researches in social media and also I have one Instagram page @ourinfinity_med with more than 100k followers, there are a team behind the posts that we shared in this scientific page.

Ozlem Dilek
University of the District of Columbia, USA

Small molecule-based probes for monitoring carbonylation in live cells

Small-molecule based probes have been used to tag biomolecules site-selectively on molecular basis to enhance diagnostic approaches in cancer. We designed a small molecule-based probe which incorporated with the carbonyl moiety of biomolecules through a click reaction to form a fluorescent product. In terms of fluorescence perspective, our non-fluorescent synthesized chemical probe can make fast covalent binding with carbonyl moieties at neutral pH to form a stable product leading to spectroscopic alteration in live cells. Spectroscopic and confocal microscopy results were used to analyze the exogenous and endogenous ROS induced carbonylation profile in human dermal fibroblasts along with A498 primary site and ACHN metastatic site renal cell carcinoma (RRC) cell lines. Our results showed that oxidative stress- induced carbonylation level responses varied in exogenous and endogenous stress in healthy and cancer cells. When cells were exogenously ROS induced, A498 cell line demonstrated higher carbonylation level than the ACHN cells. On a broader perspective, the results we reported introduced a new synthetic probe-biolabeling approach for understanding the critical importance of carbonylation that can trigger cancer or metabolic diseases.

Audience Take Away:

- It will give new insights for future fluorescence based diagnostic approaches
- It will introduce new fluorescent probe synthesis and its applications in cancer
- It will demonstrate how novel small molecules can selectively target certain functional groups in biomolecules in live cells.

Biography:

Dr. Dilek studied B.Sc. and M.Sc. in Chemistry at the Middle East Technical University, Ankara, Turkey. She then completed her Ph.D. in Chemistry/Chemical Biology at SUNY-Binghamton, USA. After two years postdoctoral studies at Cornell University and SUNY institutions, she joined Istanbul Altinbas University, Medical School in 2013 and got her tenure. She returned to USA in 2017 and worked as a faculty in Husson University and University of St Joseph. She recently joined as a faculty to University of District of Columbia, Washington, DC. She has published more than 15 research articles, book chapters and presented in more than 35 national and international conferences.

M. Cerasuolo*[1], L. Turner[1], A. Burbanks[1], R. Ronca[2]

[1]School of Mathematics and Physics - University of Portsmouth, Portsmouth
[2]University of Brescia, Department of Molecular and Translational Medicine, Brescia, Italy

In silico experiments on the effect of drug interaction in the treatment of prostate cancer in TRAMP mice

Prostate cancer is the fifth most common cause of death from cancer, and the second most common diagnosed cancer in men. Androgen deprivation therapy ability to reduce tumour growth represents a milestone in prostate cancer treatment, nonetheless most patients eventually become refractory and develop castration-resistance prostate cancer (CRPC). Enzalutamide is a second-generation androgen receptor antagonist approved for the treatment of CRPC in chemotherapy-naïve as well as in patients previously exposed to chemotherapy. However, cases of tumour resistance to enzalutamide have now been reported. In this context, preclinical models and in silico experiments are key to understand the mechanisms of resistance and to assess therapeutic settings that may delay or prevent the onset of resistance. In this study the multistage model of TRAMP mice and TRAMP-derived cells have been used to extensively characterise in vitro and in vivo response and resistance to second-generation enzalutamide. Within the study a multiscale hybrid mathematical model has been developed. The proposed mathematical model, whose development strongly relied on experimental data and their statistical analysis, was used to assess the role of cells' and chemicals' diffusion on the dynamics of prostate cancer in the multistage murine model TRAMP (transgenic adenocarcinoma of the mouse prostate) under different therapeutic strategies, with single- or combined-drug therapies. The model well describes the interdependence of cancer cells on tumour microenvironment as well as the onset of resistance following treatment with enzalutamide. Further, in silico experiments revealed that combination therapies can delay the onset of resistance to enzalutamide, and in the suitable scenario with alternating drug therapies, can eliminate the disease. The numerical simulations also showed that some of the drug combinations can cause the formation of smaller-size tumour clusters, which could give rise to metastasis.

Audience Take Away:

- The audience will learn a new experimental framework to experiment drug therapy in prostate cancer.
- The audience will learn about new hypothesis on possible drug combinations for the eradication of prostate cancer.
- The audience will be able to use some of the modelling tool as they can made available to researchers who are interested in the approach.

Biography:

Marianna Cerasuolo is a Senior Lecturer in the School of Mathematics and Physics at the University of Portsmouth. She is a mathematician whose research focuses on the mathematical modelling for medical, biological, and environmental applications. Her expertise ranges from modelling complex dynamical systems to handling and analysing experimental data. She held postdoctoral positions at the University of Naples and at the University of Durham (UK). Following these, she worked as Mathematical Modeller at Rothamsted Research (UK). She is currently working at a model for multi-drugs therapies for prostate cancer, and she is task leader in the H2020 EU-funded project Diverfarming where she is responsible for the modelling of soil carbon dynamics under different agricultural practices and climate conditions.

Sathish Kottaisamy* and Madhu Dayal

Department of Anaesthesia and Intensive Care, Vardhman Mahavir Medical College and Safdarjung Hospital, Guru Gobind Singh Indraprastha University, New Delhi, India

Comparative evaluation of characteristics of neuropathic pain and its effect on quality of life in cancer patients in oncology ward and palliative care unit

Technological advancements and recent discoveries in cancer therapy have decreased cancer-related mortality. But the principal objective of improving quality of life through advanced cancer treatments is compromised by poor management of pain (especially Neuropathic Pain (NP)) in the oncology ward compared to palliative care unit. The quality of life of cancer patients can be effectively improved by treating NP and addressing physical, psychological, social, and environmental factors associated with the disease. This study attempts to characterize and quantify neuropathic pain in terms of quality of life and determine if treatment of NP can improve the overall quality of life in cancer patients. The study included 26 patients from the oncology ward and 20 patients from the palliative care unit at Safdarjung Hospital diagnosed with cancer and presenting with pain. The study utilized the Self Complete Leeds Assessment of Neuropathic Symptoms and Signs Scale (S-LANSS) Pain Score for NP assessment, Edmonton Symptom Assessment System (ESAS), and Short Form - Brief Pain Inventory (SF-BPI) were used for assessing pain characteristics, Eastern Cooperative Oncology Group - Performance Status (ECOG-PS) for determining the patient performance, and WHO Quality Of Life - BREF (WHOQOL-BREF) for assessing the quality of life.Based on the S-LANSS score, 57% (15) of the oncology patients and 70% (14) of the palliative unit patients had NP. The non-NP patients were excluded from further analysis. The ESAS (#1) and the SF-BPI (#5) scores were lower in palliative unit patients than oncology ward patients. The ECOG-PS score was lower in palliative unit patients than oncology ward patients suggesting that patients in the palliative unit performed better than patients in the oncology ward. The WHOQOL-BREF scores (especially physical health and psychological) were higher in palliative patients than oncology patients. Cancer itself or the treatment modalities of cancer could be causing NP in patients which are evident from the high incidence of NP in both the groups. The data from this study also demonstrates that NP and quality of life has an inverse relationship and that the oncology ward patients are treated inadequately for NP and other factors affecting the quality of life of cancer patients are not addressed properly. The oncology ward patients need to be treated for their pain (especially NP) (with opioids, adjuvant analgesics, NSAIDs, Amitriptyline, Gabapentin, Pregabalin or a combination) and provided early palliative care in addition to their cancer treatment for improving the overall treatment efficacy and their quality of life.

Note: The participants for this study are still being actively recruited and we hope to collect a total of 60 patients in each palliative and oncology ward group and further analyze the same results.

Audience Take Away:

Although the cancer patients are getting treated for their ailment, advanced stage presentation of the disease and the consequences of multiple cycles of chemoradiation pose a greater challenge in managing pain which is the major factor affecting the quality of life of such patients. This study demonstrates that patients with cancer-related pain have differences in pain management between the palliative unit and the oncology ward. We see that oncology ward patients have a higher NP score indicating that they need to be treated for NP with drugs like (Amitriptyline, Pregabalin, Gabapentin or a combination) in addition to their cancer treatment to improve their quality of life.

- This study gives practitioners an insight into the importance of considering NP and early palliative care for better treatment of their patients.
- The major problem in pain management of cancer patients is simplified by treating NP as it is present in a majority of patients and adversely affects their quality of life.
- This study emphasizes the importance of Palliative care as an approach in terms of patient performance and quality of living especially in cancer-related pain.

Biography:

Dr. Sathish Kottaisamy completed his M.B.B.S. degree from The Tamil Nadu Dr. M.G.R. Medical University in 2018. Dr. Kottaisamy is currently pursuing M.D. in Anesthesia and Critical Care at Vardhman Mahavir Medical College and Safdarjung Hospital affiliated to Guru Gobind Singh Indraprastha University in New Delhi, India. As a part of this degree, Dr. Kottaisamy is involved in cancer research especially in evaluating the characteristics of Neuropathic Pain and its effect on quality of life and attending routine duties at Emergency Care, Operation Theatre, ICUs, COVID-19 ICUs, Palliative and Pain clinic.

Isha Amatya
Nepal Health Research Council, Nepal

A cross sectional study of knowledge and attitude regarding breast cancer among the students of higher secondary schools of Bhaktapur district, Nepal

Breast cancer in women is a major health burden. The leading cancer site in females was found to be breast according to population based cancer registry in Nepal. Prevention, early detection, effective treatment, and palliative care are the four major ways in controlling cancer. Knowledge plays an important role in improvement of health seeking behavior. Not only that, knowledge might significantly improve attitude, wrong beliefs, and misconceptions and consequently enhance screening practice. Providing knowledge regarding breast cancer, its risk factors, symptoms, signs and screening methods to adolescents may positively affect their attitude and practice as adults. So, this study aims to assess the knowledge and attitude regarding breast cancer among students of higher secondary schools of Bhaktapur district and also to compare knowledge and attitude regarding breast cancer according to gender.

Methodology: A cross sectional analytical study was done among the students of higher secondary schools of Bhaktapur district. There were total of 990 participants; out of which 452 were males and 538 were females. The questionnaire assessed respondents' knowledge and attitude towards breast cancer. Knowledge and attitude were categorized according to their score into poor and good. Further, correlation between knowledge and attitude and effect of socio-demographic factors on knowledge and attitude towards breast cancer were assessed.

Results: Majority of the respondents (89.6% in male and 88.5% in female) had poor knowledge regarding breast cancer. Similarly, out of the total respondents, 10.4% of males and 11.5% of females had good knowledge. Out of the total respondents nearly two-thirds of the female respondents (63.2%) and half (50.9%) of the male respondents had good attitude towards breast cancer. Similarly, nearly half (49.1%) of male and more than one third (36.8%) of female respondents had poor attitude towards breast cancer. A positive correlation was seen between knowledge and attitude of overall students ($r= 0.274$, $p= <0.001$). Similarly, a positive correlation was seen between knowledge and attitude of male students ($r= 0.302$, $p= <0.001$) and female students ($r= 0.215$, $p= <0.001$). Religion, education of parents and ethnicity of respondents showed positive association with respondent's knowledge of breast cancer. Gender, education and ethnicity of respondents showed positive association with respondent's attitude of breast cancer.

Conclusions: The study revealed that majority of the respondents (nearly 90%) had poor knowledge regarding breast cancer and more than half of the respondents had good attitude towards breast cancer. This shows that there should be more educational programs and awareness campaigns to improve the knowledge and attitude of adolescents regarding breast cancer, this ultimately may help in their screening practice in future. As awareness can play a vital role in prevention, early diagnosis and treatment of breast cancer.

Biography:

Dr. Isha Amatya studied MBBS at Institute of Medicine, Tribhuvan University, Nepal in 2014. She graduated as MD in Community Medicine at the Kathmandu Medical College, Kathmandu University, Nepal in the year 2020. She then joined as research officer in department of infectious disease at Nepal Health Research Council, Nepal. She has published more than 15 articles in National and International journals.

Sourabh Singh Gour*, Ritu Pandey and Kamlesh Choure
Department of Biotechnology, AKS University Satna (M.P) 485001, India

Potential bioactivity of *Cordyceps militaris* against viability of lung cancer cell line A549

Cordyceps militaris is an effective medicinal mushroom having reported potential bioactivity against several diseases. In this present study, we have extracted biochemical compounds present in Cordyceps militaris, using HPTLC and analyzed and identified by HPLC. The compound was detected as Cordycepin. MTT cell viability assay was performed on A549 (lung cancer cell line). For this, extract having cordycepin, was completely dried firstly and then dissolved in DMSO. A549 cell line was maintained in DMEM with10%FBS, 1% L-glutamine and1% penstrep. 5000 Cells were seeded in each well of 96 well plates; at 60-70% confluency cells were treated with different extracts of Cordyceps militrais at various concentrations. Further, plate was read by ELISA plate reader at 570 nm for absorbance density value to determine cell viability. Viable cells were capable to reduce the tetrazolium salt to formazon resulting in blue color whereas dead cells were not able to do so. Fractions of viable cells were calculated and it was found that the extracts of Cordyceps militaris having the anti-viable property against human lung cancer cell line A549.

Biography:

Sourabh Singh Gour has completed his graduation and post-graduation in biotechnology, currently he is pursuing Ph.D in Biotechnology under the guidance of Dr.Kamlesh Choure Head Department of Biotechnology, Faculty of life Science AKS University (M.P). His research area is mushroom technology and cancer study, during his research he isolated different industrial applicable compound.

Dellasie Aning
Panalove LLC, Ghana

The rise of skin cancer prevalence in Africa and the Diaspora

My objective is to bring awareness to the skin cancer trends rising within the Diaspora. My theory is in order to reduce the mortality rate, broader effort towards public awareness and education is key. Skin lightening is a wildly popular (multi-billion-dollar) global industry. This phenomenon has hit Africa and the Diaspora particularly hard. The World Health Organization states 1 in 3 South African women and 77% of Nigerians (60 million people) bleach their skin. The numbers are estimated to be anywhere from 25 -77% of African nations actively bleaching. This practice is extremely dangerous. Bleaching strips skin of its natural properties (melanin) leaving the skin vulnerable to infection and disease. This has been proven to lead to skin cancer. Compounding the issue is that there is a lack of dermatologists in sub-Saharan Africa. This means diagnoses is often too late. There is also a stigma attached to skin bleaching, making it difficult to ascertain the true numbers of people at risk. This issue motivates me because when I was younger, my aunt passed due to skin cancer that was attributed to bleaching. My uncle (the late Kofi Annan) was always very encouraging of my philanthropy goals, and his recent passing gives me even more motivation to make a difference. I believe there needs to be a higher regulation practice of toxic lightening products being imported into these nations. They are illegal, but the laws are not properly enforced. Consumers need to be better educated on cancer risks in skin-lightening products containing chemicals like glutathione, mercury and hydroquinone. Marketing companies should be taken to task for subliminal messaging that encourages consumers to bleach their skin for overall life improvement. Governments in sub- Saharan Africa should consider subsidizing dermatology-focused grants for young students. Awareness leads to change. That change begins with us!

Biography:

Dellasie Aning is a humanitarian, e-commerce business owner, recording artist and public speaker. Her expertise in public speaking lies in her philanthropy work, advocacy, and strides to create a healthier, sustainable world for the future. She is an alumna of Emory University with a degree in political science and marketing. Her passion and determination for women's health is rooted in her own personal connection to cancer. Her aunt died of melanoma in Ghana when Dellasie was only 12 years old. Her parents are both cancer survivors (with her father having beat prostate cancer and her mom beating cervical cancer). Over the past 5 years, Dellasie has been extremely driven and determined to combat this issue through her art, her mentorship, passionate speaking engagements and fundraising work on the ground. IN 2018 she established PANALOVE, LLC – a platform to support her initiatives, brand and business through her collaborations and social activations around the world. Her passion and tenacity is unparalleled.

Darko Katalinic
Josip Juraj Strossmayer University of Osijek, Croatia

Clinical and molecular features of Pleural Mesothelioma

Mesothelioma is an aggressive tumor caused by neoplastic transformation of mesothelial cells of the serous membranes. Although it can occur at any anatomical localization that contains a mesothelium, it most often occurs in the pleura. Most cases of mesothelioma are caused by occupational and environmental exposure to asbestos compounds, and are diagnosed with a latency period of several decades, very often in an advanced clinical stage when therapeutic options are significantly limited, and the overall survival rate is extremely unfavorable. The pathophysiology of mesothelioma is based on the development of a chronic inflammatory process potentiated by the influence of mutagenic oxygen ion radicals and the protein molecule HMGB1 (High Mobility Group Protein B1), which leads to activation of the NF-κB (Enhancer of Activated B-Cells) signaling pathway within mesothelial cells. The resulting microenvironment leads to neoplastic transformation of mesothelial cells, especially those with previously accumulated genetic damage. The presentation summarizes the epidemiology, etiopathogenesis and clinical course of mesothelioma, and additionally discusses the roles of different imaging techniques as well as application of serum and molecular biomarkers on top of standard diagnostic modalities. Finally, the presentation presents currently available treatment options that include a surgical approach, chemoimmunotherapy, radiotherapy, and multimodality treatment.

Audience Take Away:

- Epidemiology of mesothelioma
- Association and relationship between asbestos compounds and mesothelioma
- Etiology and molecular pathophysiology of mesothelioma
- The importance of early detection of mesothelioma
- Diagnostic modalities of mesothelioma
- Multimodality treatment of mesothelioma

Biography:

Darko Katalinić, M.D., Ph.D., FRSM (UK), FESE, FESMO, FEACR (Ambassador), internist, haematologist and medical oncologist, studied Medicine and Molecular Biology at the University of Rijeka and University of Zagreb, Croatia. He holds a Ph.D. degree from Zagreb University in Molecular Biology. A consummate clinician scientist, Assoc. Prof. Katalinić has authored more than 150 scientific papers. His clinical and research interests include medical oncology, haematology, molecular oncology, adjuvant immuno/chemotherapy and targeted therapy as well as discoveries on predictive and prognostic cancer markers in lung cancer, breast cancer, gastrointestinal cancer, urogenital cancer, neuroendocrine tumours, GIST, melanoma, sarcoma, lymphoma and MDS.

M.Bassam. Aboul- Nasr*[1], Sabah S. Mohamed[1] and Nehad A. El-Araby[2]

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Aspergillus ochraceus strain MW671551 isolated from Al Fawakhir mine soil used to produce gold nanoparticles and their efficacy against MDAMB-231 breast cancer cells

Aim: This study aimed to determine the ability of Aspergillus ochraceus MW671551strain from Al-Fawakhir Gold Mine, Egypt in biosynthesis of gold nanoparticles (AuNPs) and study their efficacy against MDAMB-231 breast cancer cells.

Methods and results: The extracellular culture filtrate of the fungus was challenged with 0.25 mmol of tetrachloroauric acid trihydrate (HAuCl4•3H2O) and the antitumor studies effect of AuNPs were estimated on MDAMB-231 cancer cell lines. The fungal strain showed an excellent capability to synthesize gold nanoparticles (AuNPs). The synthesized AuNPs were characterized using UV-vis spectroscopy, transmission electron microscopy (TEM), Fourier-Transform infrared spectroscopy (FT-IR) and X-ray, The results revealed that Aspergillus ochraceus could produce AuNPs with particle sizes ranged from 5-. 26 nm to 21.6 nm and Bragg reflections 111, 200, 220, and 311, respectively. MTT assay showed that AuNPs had high toxic effect on MDAMB-231 cells with IC-50 value 32.83µM/ml and 33.55µM/ml. The impact of A. ochraceus-synthesized AuNPs was assayed on MDAMB-231 cancer cells where the early and late apoptotic cells showed 0.27 % and 0.15 % apoptotic cells, respectively. The percentage of the treated cells decreased from 52.06 % to 38.45 % in G0/G1 phase and from 39.88 % to 36.99 % in S phase, while, the percentage of the treated cells in G2/M phase increased to reach about 14.56 % relative to 8.06 % in control.

Conclusions: In this study, we proved the ability of Aspergillus ochraceus MW671551 to induce highly stable and ecofriendly AuNPs. The impact of AuNPs on the proliferation of MDAMB-231 cancer cell lines showed significant results against cancer cell line.

Significance and impact of the Study: We showed that Aspergillus ochraceus MW671551 strain which isolated from gold mine soil had a wide scope of extracellular enzymatic profile that reduce tetrachloroauric acid trihydrate (HAuCl4•3H2O) into gold nanoparticles .The highly stable colloidal solution of these nanoparticles had a great selectivity to kill cancer cell in very minimal concentration. This study gave a promising role in demolition breast cancer and feather medical and pharmaceutical are needed.

Wei Qiu*, and Hao Wang
Department of Surgery, Loyola University Chicago, Maywood, USA

EPHA2, a promising therapeutic target for hepatocellular carcinoma

Hepatocellular Carcinoma (HCC), a primary type of liver cancer, remains one of the deadliest malignancies worldwide. Despite decades of targeted therapy advancements, the currently approved medications for advanced HCC provide patients with limited clinical benefits. One major obstacle to treatment is a lack of effective molecular-targeted therapies. Here we identified EPHA2 as a novel oncotarget that promotes HCC initiation and progression. We found that high EPHA2 signaling and expression is observed in over 30% of HCC patient samples and is associated with worse clinical outcomes. Loss of EphA2 suppressed the initiation and growth of HCC both in vitro and in vivo. Furthermore, CRISPR/CAS9 mediated EphA2 inhibition significantly delayed tumor development in a genetically engineered murine model of HCC. Mechanistically, we discovered that targeting EphA2 suppressed both AKT and JAK1/STAT3 signaling in HCC. We also demonstrated that treatment with a small molecule inhibitor of EPHA2, ALW-II-41-27, suppressed HCC progression in mice. In summary, our results suggest EphA2 as a promising therapeutic target for HCC.

Audience Take Away:

- The audience will get to know the current challenges in treating HCC, and the novel role of EphA2 in HCC as well as its action mechanisms. Further, our findings support the further clinical investigation to assess the safety and efficacy of EPHA2 inhibitors in the treatment of advanced HCC, especially in patients showing activation of EPHA2.

Biography:

Dr. Wei Qiu obtained his Ph.D. from the Chinese Academy of Science in 2005. He is a tenured Associate Professor at the Department of Surgery. Over the last 16 years, he has been studying the molecular mechanisms of tissue injury, inflammation, and tumorigenesis using many in vitro and mouse models. He has published 45 peer-reviewed papers in high-profile journals, including Cell Stem Cell, JCI, Gastroenterology, PNAS, Hepatology, Cancer Research, and Cell Reports. He has been serving as an editorial board member for several reputed journals.

Danielle Rossini-Dib
School of Medicine University of Sao Paulo, Brazil

3 reflections that can help us not die before die: considerations from a oncological psychologist that turn herself an oncological patient

The objective of this presentation is to report a personal experience with oral cancer, after more than 10 years of working on the psychological aspects of cancer patients in a private chemotherapy service. To support this, some psychiatric triage data collected in the service will be presented, as well as elements of Positive Psychology and Cognitive Behavioral Therapy that can be applied from diagnosis to follow-up or palliative phase.

Audience Take Away:

- The points covered can help the professional in trying to approach the client centered;
- Through active listening, the professional can guide the client to develop effective strategies that can help him or her cope better with cancer;
- The practitioner can recognize their personal elements involved in this emphatic approach.

Biography:

Graduate at Psychology from the Unifmu University Center (2002). Current institutional activities: Master in Sciences by the Department and Institute of Psychiatry, Hospital das Clínicas, Faculty of Medicine, University of São Paulo (IPq - HCFMUSP); Collaborating Psychologist at IPq-HCFMUSP: Integrated Outpatient Clinic for Impulse Disorders (Organization of the screening and referral system for patients; Coordination of the neuropsychology area); Area of expertise: Psychology, with an emphasis on Neuropsychology, Positive Psychology, Cognitive Training and Cognitive Psychotherapy.

PARTICIPANTS LIST

We wish to meet you again at our upcoming Conference:

ICC 2022 | September 16-17, 2022 | Paris, France

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