magnus Group

6TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

AUGUST 17-19, 2023

LONDON, UK | HYBRID EVENT

Venue:

Copthorne Hotel Slough-Windsor

Cippenham Ln, Slough SL1 2YE, United Kingdom

BOOK OF ABSTRACTS

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17-19 AUGUST

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ABSTRACTS

6TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

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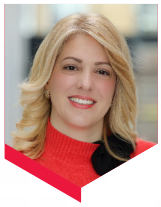

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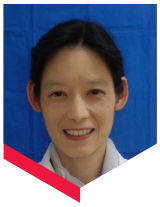

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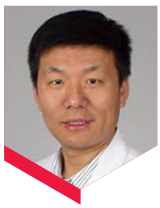

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University of Southern California, USA

Siyi He
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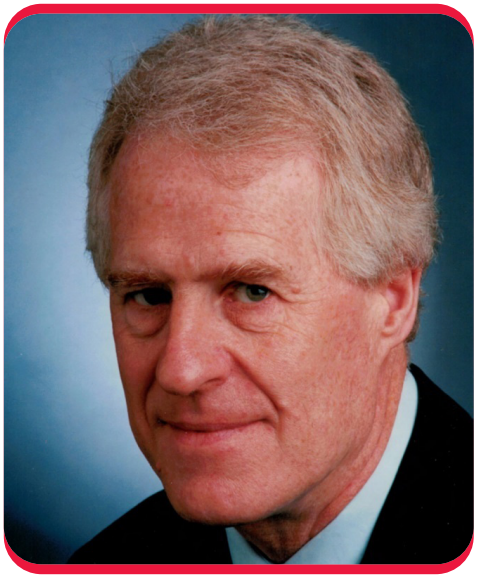
Xing Cui
Shandong University, China

Maria Zahra
WWL NHS trust hospital, United Kingdom

Thank You All...

Welcome Message



On behalf of the Scientific Committee, I take great pleasure in welcoming you to the 6th Edition of International Cancer Conference being held in the world class city of London, UK. The location for personal attendance at the Conference is very conveniently located close to Heathrow Airport. The theme of this year's hybrid conference is "Stretching Wings to Fight Cancer through Unified Research Approaches". This particularly comprehensive meeting will cover many aspects of research including cancer metabolism, cancer diagnosis, cancer prevention and detection, treatment and drug-based chemotherapy. While you are here, I sincerely hope that you take the opportunity to network, learn, share and collaborate with international experts. All of us on the Scientific Committee will take great pleasure in meeting you In-Person and learning more about your outstanding work. I sincerely wish you have an enjoyable and productive conference. I hope you enjoy your stay in this wonderful city and use pre and post conference times to enjoy the many exciting sites of London, such as the iconic Windsor Castle which is close by! We are enthusiastic about your attendance and participation. Enjoy the conference!

Michael Thompson

University of Toronto, Canada

Welcome Message

On behalf of the Scientific Committee, it is my distinct pleasure to extend a warm welcome to the 6th Edition of the International Cancer Conference in the vibrant city of London, England. Our gathering is graced by an extraordinary assembly of globally acclaimed leaders, converging to delve into a myriad of topics shaping the landscape of cancer care.

Anticipate dynamic discussions catalyzing advancements in clinical practices and sparking innovative research trajectories. This platform is designed to ignite dialogues that pave the way for enhanced patient care and pioneering ideas for the future. While here, I encourage you to seize the chance to connect, learn, exchange insights, and cultivate collaborations with the esteemed international leaders present.

All of us on the Scientific Committee would take great pleasure in meeting you and learning more about your amazing work. I hope that you enjoy this spectacular event and all of the amenities of London. Here's to a rewarding and engaging event!

Yan Leyfman

Icahn School of Medicine at Mount Sinai, USA

Welcome Message

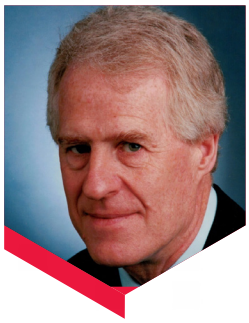
It is my privilege as a member of the Scientific and Organizing Committee in welcoming all of the professionals in the scientific and health care international community both In-Person and Virtually to the 6th Edition of the International Cancer Conference in London, England. The breadth of topics of this year's meeting is certainly quite wide including the evolving "sub-fields" of pathophysiology, basic science, epidemiology, biology, treatment, bedside patient care, and ongoing research involving hematologic malignancy and cancer. In addition, the confluence of cancer with other disease processes will be featured in a number of presentations highlighting its effects upon other disease processes.

The aim of the Conference is to stimulate the academic and clinical "appetites" among scientists, researchers, physicians, and other health care professionals involved in the battle against cancer. We very much look forward to meeting you and sincerely hope that you will immensely enjoy interacting with new and established colleagues in both a professional and personal capacity. Lastly, we hope that some time will be available for those present in order to visit and partake of beautiful and historic London.

Franco Musio

University of Virginia School of Medicine, USA

Keynote Speakers



Michael Thompson
University of Toronto, Canada

Yan Leyfman
Icahn School of Medicine at Mount Sinai, USA

Franco Musio
University of Virginia School of Medicine, USA

Jianhua Luo
University of Pittsburgh School of Medicine, USA

George Zachos
University of Crete, Greece

Marina De Rosa
University of Naples Federico II, Italy

Shinya Tajima
Shizuoka Medical Center, Japan

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 2023

We are thrilled to announce the upcoming ICC 2023, the 6th Edition of the International Cancer Conference, which will take place as a HYBRID EVENT from August 17-19, 2023. This year's conference theme is "Stretching Wings to Fight Cancer through Unified Research Approaches."

Since 2016, our conferences have brought together leading experts in the field of cancer, creating a platform for professionals worldwide. The ICC congress serves as a comprehensive forum for the oncology and cancer research community, encompassing doctors, scientists, researchers, patient activists, caregivers, journalists, pharmacists, oncologists, cancer experts, and industry representatives.

The global symposium will offer a hybrid platform, facilitating interaction, communication, and education on various oncology-related topics. Attendees will have the rare opportunity to share valuable work that integrates clinical, translational, and basic research. ICC 2023 will showcase the latest developments in cancer therapy and offer a robust educational program. It will provide enhanced In-Person and online experiences, promoting delegate interaction and engagement. Esteemed professionals in the field are invited to participate as keynote speakers, oral presenters, organizing panel members, poster presenters, and delegates. The conference agenda will cover a wide range of topics, including oncology, cancer metabolism, cancer diagnosis, and chemotherapy drugs. These areas, often under-researched, will receive special attention, encouraging additional study and knowledge dissemination.

Join us at ICC 2023 to be part of this dynamic event, gain valuable insights, and contribute to the fight against cancer through unified research approaches.

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17-19 AUGUST

DAY 01

KEYNOTE FORUM

6TH EDITION OF

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Sensor technology for detection of biomarkers in gynecological medicine

Detection of gynecological disorders of female reproductive organs, especially forms of cancer, typically involves screening of symptoms for the illnesses, followed by imaging with ultrasound or MRI. These techniques are time consuming and require trained professionals to perform, and thus are expensive and difficult to apply to general population screening. Faster, lower cost, and simple screening methods need to be developed as screening is essential to detecting these illnesses in an early stage, allowing their more effective treatment and improving outcomes for patients. The realm of sensor and biosensor technology offers a potential protocol for such large scale screening of blood, tissue, intertidal fluid and urine-based biomarkers for gynecological cancers. Devices based on luminescence, electrochemistry, acoustic wave, and surface plasmon resonance involving surface-attached enzyme, immunological and nucleic acid probes have figured predominantly in terms of biomarker detection. The main aim of research in this area appears to be targeted at either the typical central clinical biochemistry or point-of-care assay. A highly attractive possibility with regard to the former would be the incorporation of a biosensor into the conventional automated robotic system to process and test patient samples. Such a technology would require device reversible signalling or flow-through cleaning, appropriate sensitivity and, critically, the capability of operation in a biological fluid. The reality is that the issue of fouling by components of such fluids has constituted a major problem for the practical application of sensors for many years. Biosensor-based detection of ovarian cancer has been by the most studied in the gynaecological oncology field. This activity appears to have been spawned by the realization that early-stage detection of the disease can potentially result in a high level of patient survival. Such research has involved many assays of the well-known CA-125 antigenic species in addition to Human Epididymis Protein (HE4), Heat Shock Protein 10 (HSP10) and Lysophosphatidic Acid (LPA). This paper will attempt to address the efficacy of the various sensor approaches that have been employed for the detection of these markers. This will include our own work on the square wave electrochemical signalling of LPA interaction via a protein probe for the molecule attached to an electrode. Importantly, the assay is capable of function in biological fluid through the application of tandem antifouling surface chemistry.

Audience Take Away Notes

- How biosensor technology can employed for potentially large-scale screening of cancer biomarkers
- Assessment of biosensors with regard to device efficacy in gynecological cancer detection
- Example of biosensor for early-stage detection of ovarian cancer

Soha Ahmadi, Katharina Davoudian, Brian De La Franier, Navina Lotay, Michael Thompson*

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

Biography

Professor Michael Thompson obtained his PhD in analytical chemistry from Mc Master 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.

Revisiting the treatment of anemia in the setting of chronic kidney disease, hematologic malignancies, and cancer

Anemia has and will continue to be a central theme in medicine particularly as clinicians are treating a burgeoning population of complex multiorgan processes. As a result of multiple Randomized Controlled Trials (RCA), meta-analyses, and medical societal recommendations overly restrictive paradigms and under-administration of Erythropoiesis Stimulating Agents (ESA) have likely been followed by clinicians among all specialties. A review of anemia in the context of Chronic Kidney Disease (CKD), hematologic malignancies, and cancer is presented with focus on the establishment of ESAs as integral in the treatment of anemia. RCTs and meta-analyses studying the use of ESAs are presented with focus upon their application to clinical practice. A compendium will be presented describing the evolution, establishment, and implications of ESA administration initially among those with CKD with rapid subsequent application to the hematology-oncology population of patients. Upon evaluation of the risks and benefits of ESAs focused critique is made supporting more liberal use of these agents strongly suggesting that the current underlying treatment 'pendulum' has perhaps shifted too far to the 'under-treatment' side in many individual cases. In addition, a parallel critique will be made in favor of liberalizing the strict transfusion criteria among selected renal and oncology patients who generally present with multiple comorbid conditions.

Objectives:

- A focused appreciation of the history and evolution of erythropoiesis stimulating agents in clinical medicine
- A deeper understanding of the risks and benefits of ESA administration among patients with chronic kidney disease, hematologic malignancies, and cancer with a greater critique of the literature and practice guidelines in this field
- A greater discernment of the position that the current underlying treatment 'paradigm' in renal and oncologic disease has perhaps shifted too far to the 'under-treatment' side in many cases
- In parallel fashion, appreciation of the position that transfusion guidelines and the current practice of transfusion of packed red blood cells among renal and oncology patients may also have shifted too far to the 'under-treatment' side in many cases
- An appreciation that the treatment of anemia is predicated upon the art of medicine which is the synthesis of scientific data and the unique nature of each individual patient to include their comorbidities and emotional health

Franco Musio, M.D, FACP, FASN

University of Virginia School of Medicine (Inova Fairfax Campus), Falls Church, VA, United States of America

Biography

Dr. Franco Musio earned his undergraduate and medical degrees from Georgetown University (Washington, D.C.) with subsequent training in General Surgery, Internal Medicine, and Nephrology at Brooke Army (San Antonio, Texas) and Walter Reed Army Medical Centers (Washington, D.C.). Dr. Musio has subsequently been in the academic and clinical practice of nephrology for close to 30 years at Walter Reed and Inova Fairfax Hospital (Falls Church, VA) where he has collaborated with many hematology-oncology colleagues and has developed a specialty in caring for complex patients with microangiopathic hemolytic anemia as well multiple other types of anemia. Dr. Musio enjoys lecturing, writing, and discussing medical and human-interest topics on local and international radio stations and podcasts.

BOOK OF ABSTRACTS

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17-19 AUGUST

DAY 01

SPEAKERS

6TH EDITION OF

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Tattym E. Shaiken

Department of Medicine, Baylor College of Medicine Houston, TX, 77030, United States

PeriNuc Labs, University of Houston Technology Bridge, Houston, TX, 77023, United States

Coordination of the cytomatrix micromechanics and the warburg effect support survival of cancer cells

For decades cancer studies have focused on molecular genetics while the role of the cytoplasm has remained obscure. Separation of the viscous fluid cytosol and elastic-solid cytomatrix has offered an opportunity to solve an age-old mystery in biochemistry; how millions of complex chemical reactions can occur simultaneously within the cell cytoplasm. The cytomatrix contains structural proteins, ribosomes, and metabolome enzymes responsible for unique biosynthetic pathways that involve immobilized biocatalysis. Immobilizing these catalytic complexes overcomes the spatial limitations for biochemical processes and allows integration of the intracellular and extracellular matrices and receptors with nuclear processes. Together, the cytosol and cytomatrix produce an interconnected synergistic network that maintains the operational flexibility of healthy cells as well as the survival of malignant cells. The cytomatrix is also responsible for cellular micromechanics and cytoplasmic motion. The combination of mechanical and biocatalytic processes triggered by extracellular signals and gene mutations in malignant cells requires additional energy. Cancer cells, consequently, utilize aerobic glycolysis, the Warburg effect, to meet the energy demands of the matrix mechanics that arise in response to imbalanced signaling and excessive biocatalytic activity. Clinical cancer is a rare event despite a high frequency of mutations, as clinical cancer is limited by the requirement for alterations that result in a high energy production state. Without these transformations, potential cancers can only survive in the quiescent state or will be eliminated. Survival of cancer cells indicates that the cancer cells were able to synchronize energy output for matrix mechanics supplying sufficient energy for tumor growth. Thus, cancer cell survival or death depends on triggering the Warburg effect that links genetic aberrations and intracellular matrix mechanics with the ability to provide the energy supply through glycolysis and oxidative phosphorylation.

Audience Take Away Notes

The organization of biochemical processes within the cell has fascinated many generations of scientists because it directly impacts our understanding of Life and pathological processes. Classical biochemistry is founded on several assumptions valid in dilute aqueous solutions that are often extended without question to the interior milieu of intact cells. The cytoplasm is crowded and the diffusion and partitioning of macromolecules is restricted by steric hindrance as well as by unexpected binding interactions. The cytoplasm model consisting of elastic solid cytomatrix and viscous fluid cytosol derives from chemically separating these physical phases and showing their differences using conventional methods and high-throughput analyses such as mass-spectrometry proteome profiling, RNA-seq, and Ribosome footprint analyses. By separating the viscous fluid and elastic solid components of cytoplasm, we elucidated how millions of chemical reactions can potentially create life without interrupting each other by immobilizing on the cytomatrix platform. Mechanical activity of cytomatrix induces cytomatrix motion that requires ATP energy. Cancer cells, consequently, utilize aerobic glycolysis, the Warburg effect, to meet the energy demands of the matrix mechanics that arise in response to imbalanced signaling and excessive biocatalytic activity.

Our findings will help to better understand metabolic processes of the normal cell and elucidate mechanisms of the malignant transformation. Method of cellular fractionation into the cytosol, cytomatrix and core nucleus will help design experiments without mixing solid and liquid phases of the cytoplasm and the nucleus. The knowledge that clinical cancer is the combination of gene mutations, matrix mechanics, and the surplus energy known as the Warburg effect provide new information about cancerogenesis that can be used by scientists in their theoretical and experimental work and oncologists and medical professionals in their clinical approach to fight cancer and biotech companies to design anticancer drugs

Biography

Dr. Shaiken studied Biology at the Kazakh State University, Kazakhstan and graduated as MS. He received his PhD degree in 1991 at Russian Regional Institute of Biochemistry. Dr. Shaiken worked in Dr. Wakil's lab, Baylor College of Medicine, Houston, TX from 2001 to 2007 and worked as Sr. Scientist from 2007-2014 at the MD Anderson Cancer Center. He is associated with the Department of Medicine at Baylor since 2014 and Founded Peri-Nuc Labs. He has published more than 20 research articles and multiple patents. For the last ten years, Dr. Shaiken developed several methods to advance cell and cancer research.

Fleur M. Aung, MD[1*], Danielle Brown RN BSN[2], Yianna Arceneaux RN BSN, MD[3], Adrianna Knopfelmacher MD[4]

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[2]MD Anderson Blood Donor Center, Section of Transfusion Medicine, Department of Laboratory Medicine, Division of Pathology. The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Unit 0800, Houston, Texas 77030, USA
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[4]Assistant Professor, Acting Medical Director MD Anderson Blood Donor Center, Section of Transfusion Medicine, Department of Laboratory Medicine, Division of Pathology. The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Unit 0800, Houston, Texas 77030, USA

The daily availability of granulocyte transfusions in a large oncology center

Introduction: Granulocyte transfusions are requested to treat severely neutropenic adult/pediatric Leukemic and Stem cell transplant patients with overwhelming bacterial/fungal infections. Clinical indications range from organ specific to soft tissue infections infection.

Requesting Granulocytes: The process begins when the patient's primary team notifies the White Blood Cell Nurse Coordinators at the Blood Donor Center. Education material (Overview and Donor Process) with a link to a video presentation is forwarded to the donors. The eligibility and collection is a three-day process and logistics can be complicated as the donors may not always be available when the transfusion is needed. All donors (family, friend, relatives and volunteers) are eligible to be prescreened and consented for granulocyte apheresis.

Donor Eligibility: Donors are required to be eligible as per FDA/AABB guidelines, ABO compatible with the patient and female donors negative for HLA antibodies. If the patient has a red cell alloantibody, the designated donor is screened for the cognate antigen. CMV testing is not performed, and HLA compatible granulocytes are currently not available. All prescreened eligible donors are required to undergo a platelet apheresis collection prior to granulocyte collection. Medical exceptions are made when the patient and the care team agrees to an ABO incompatible transfusion.

After the platelet apheresis, the donor is notified by the Transfusion Medicine Physician. The nurses schedule the donor for pickup of G-CSF and Dexamethasone a day prior to the apheresis procedure. All donors are stimulated with 480 mg granulocyte colony stimulating factor (G-CSF) and 8 mg dexamethasone the night before the apheresis with few exceptions. At the time of medication pick up a second CBC, repeat Infectious disease testing is done, and vital signs documented.

Granulocytapheresis: The apheresis procedure may take up to 180 minutes depending on TBV, increment in WBC after stimulation and is optimized to reduce red cell collection. All units are drained by gravity with or without the addition of Hetastarch. A complete blood count is performed on each unit to obtain the unit wbc count and volume of donor red cells. The irradiated/labelled granulocyte unit(s) are available for transfusion approximately 3 hours after collection, stored at room temperature 22 C and expire 24 hours from the time of completion. A large unit dose may be split to support more than one patient. The Blood Donor center can perform Granulocytapheresis daily if there are eligible prescreened donors available.

Administration of Granulocytes: Compatibility testing is performed on all units. Granulocytes are transfused via a red cell infusion set not to exceed 4-hours. Patient response is monitored by treating team and the WBC nurse coordinators daily. The clinical decision to continue, stop or pause the transfusion is at the discretion of the primary care team.

Outcome: Our Blood Donor Center has performed 1530 granulocyte apheresis procedures since Dec 2015 to March 2023, the majority (63%) being male donors, median age 41 years (range 17-83), unit volume median 631 mL (229-1085) and Unit WBC median 10.3 10e10 (range 2.46-33.06) and continues to be a successful treatment modality.

Audience Take Away Notes

- The role of the clinical team in ordering Granulocyte Transfusions
- Outline the process of selection and triaging granulocyte donors
- Overview of the Granulocyte collection process and product modification
- The efficacy of Granulocyte Transfusions in severely Neutropenic Leukemic and Stem cell transplant adult and pediatric patients
- Potential benefits to the patient

Biography

Dr. Fleur Aung graduated from the Institute of Medicine I, Yangon, Myanmar where she completed a one-year rotating internship. She then came to the United States and trained in Anatomic and Clinical Pathology followed by a Surgical Pathology Fellowship. After practicing as a Surgical Pathologist, she returned to train in Transfusion Medicine and Histocompatibility and Immunogenetics (HLA) at MD Anderson Cancer Center where she started as an Assistant Professor. She is involved in IRB Approved clinical research protocols as a Principal Investigator, Co-Investigator and Collaborator related to Stem cell transplantation, Granulocyte and Mononuclear collections in Leukemia, Lymphoma and Stem Cell Transplantation.

Lashitha Reddy Konda
Medical student at University of Buckingham, United Kingdom

Sinonasal undifferentiated carcinoma: A case report and brief literature review

Sinonasal undifferentiated carcinoma is a rare malignancy, of the nasal cavity and/ or paranasal sinuses accounting to be less than 1% of all malignancies. This case report will discuss a young male patient diagnosed with poorly differentiated carcinoma in the lateral wall of the nasal cavity, who has subsequently undergone endoscopic excision and adjuvant chemotherapy. He is currently undergoing radiotherapy. The report will further review the literature to find the best management. Overall, it's concluded that multimodality treatment (surgery and chemoradiotherapy) is shown to be most effective but considering chemoradiotherapy toxicity the decision should be made on an individual basis. This also raised the need for research and standardization through evidence-based medicine while considering each patient as an individual and providing personalized care.

Audience Take Away Notes

- Some rare cancers do not gain stoplight as much as the common cancers do for research. Allow this poster to spread awareness and open a platform to draw attention to more research in the area
- This poster will discuss on the treatment of Sinonasal undifferentiated carcinoma through evidence based practice

Biography

Lashitha Reddy Konda is currently a second-year medical student at University of Buckingham, Buckingham, United Kingdom of a 4.5-year undergraduate program. She is passionate about medical education and surgery. And this is her first abstract.

Michell Fullmer RDN, LDN, CSP, CNSC
Mosely Center for Cancer and Blood Disorders/Nemours Wilmington Delaware United States of America

Skeletal health in childhood cancer: A novel team approach to addressing a common late effect of cancer treatment

Children with cancer are at an increased risk of developing osteonecrosis (ON) and decreased bone mineral density (BMD). In Acute Lymphoblastic Leukemia, patients aged 10 to 20 years are 10-20 times more likely to develop ON compared to younger children and adults. Although ON and decreased BMD can result in life-altering pain and disability, research regarding the appropriate screening, prevention, and treatment is lacking. To address this gap in knowledge, our institution created a multi-disciplinary team comprising of experts in oncology, radiology, endocrinology, physiatry, physical therapy, orthopedics, and nutrition. The Skeletal Health in Childhood Cancer (SHICC) Team was created in early 2022. The team meets quarterly to discuss clinical guidelines, develop research protocols, and provide education to families and medical staff A subgroup was formed in February 2023 to review individual patients' imaging and laboratory results to track incidence of ON and decreased BMD and opportunities to optimize bone health. The recommendations of the group are reviewed with the patient and/or guardians in the form of a skeletal health report card that was developed by the SHICC team.

Audience Take Away Notes

- Participants will demonstrate an understanding on the impact of cancer therapy on skeletal health in children with cancer
- Participants will understand the impact of interdisciplinary care in the management of the skeletal health in childhood cancer
- Participants will be exposed to algorithms to improve the vitamin D surveillance in the clinical setting

Biography

Michell Fullmer is a registered dietitian/nutritionist who received her bachelor's degree from Immaculata University in 1989. She is certified in pediatric nutrition from the Academy of Nutrition and Dietetics. She is also a certified nutrition support clinician through the American Society of Parenteral and Enteral Nutrition. She has published several articles in peer reviewed journals on various topics including novel research on Vitamin D insufficiency in newly diagnosed children with cancer. She leads a team within her institution that is currently developing protocols and further research on skeletal health and childhood cancer.

Ahmad M.S[1], Braoudaki M[1], Patel H[1], Ahmad I.[2], Shagufta[2], Shoib Sarwar Siddiqui[1*]

[1]School of Life and Medical Sciences, University of Hertfordshire, United Kingdom

[2]Department of Mathematics and Natural Sciences, School of Arts and Sciences, American University of Ras Al Khaimah, Ras Al Khaimah, United Arab Emirates (UAE)

Novel siglec-15-sia axis inhibitor leads to colorectal cancer cell death by targeting mir- 6715b-3p and oncogenes

Colorectal cancer (CRC) has been referred to as the fourth most diagnosed cancer worldwide and the third leading cause of patient mortality in humans. Approximately, 1,931,590 new cases of CRC were reported with 935,137 patient deaths worldwide in 2020. CRC has become increasingly prevalent worldwide as well, with statistical trends indicated cancer-related deaths could reach as high as 71.5% by 2035. Currently, conventional treatments for CRC patients have fallen short as successful therapeutic strategies, due to lacking in patient response, severe side effects and modest specificity consequently resulting in patient mortality and/or tumour recurrence. The recent trend of immunotherapy in solid cancers such as melanoma have shown success with blocking antibodies including Nivolumab and Pembrolizumab. Siglecs are well known immunotherapeutic targets in cancer. Current checkpoint inhibitors have exhibited limited efficacy, prompting a need for novel therapeutics for targets such as Siglec-15. Presently, small molecule inhibitors targeting Siglec-15 are not explored alongside characterised regulatory mechanisms involving microRNAs in CRC progression. Therefore, a small molecule inhibitor to target Siglec-15 was elucidated in vitro and microRNA mediated inhibitor effects were investigated. Our research findings demonstrated that the SHG-8 molecule exerted significant cytotoxicity on cell viability, migration, and colony formation, with an IC50 value of approximately 20μM. SHG-8 significantly reduced the SIGLEC15 expression at the gene and protein levels. Notably, miR- 6715b-3p was the most upregulated miRNA in high-throughput sequencing, which was also validated via RT-qPCR. MiR-6715b-3p may regulate PTTG1IP, a potential oncogene which was validated via RT-qPCR and in silico analysis. Additionally, molecular docking studies revealed SHG-8 interactions with the Siglec-15 binding pocket with the binding affinity of - kcal/mol, highlighting its role as a small molecule inhibitor. Importantly, Siglec-15 and PD-L1 are expressed on mutually exclusive cancer cell populations, suggesting the potential for combination therapies with PD-L1 antagonists.

Audience Take Away Notes

- We synthesized an in-house beta-amino ketone compound (SHG8) that kills colorectal cancer cells with an IC50 of 20 μM
- This novel compound can inhibit the Siglec-15-Sia axis which is very important axis in cancer progression
- This is the first reported inhibitor of Siglec-15 and reduce proliferation, migration and colonization of cancer cells
- We performed the small RNA Seq (sRNA seq) to find the differential expressed miRNAs with SHG8 treatment
- MiR-6715b-3p was found be the highly upregulated miRNAs upon SHG8 treatment

Biography

Dr. Shoib Siddiqui is a senior lecturer in the School of Life and Medical Sciences at the University of Hertfordshire, UK. Before starting his position at UH, he was working as an Assistant Professor at the American University of Ras Al Khaimah (AURAK), UAE. He worked as a postdoctoral researcher in the lab of Prof. Ajit Varki at University of California, San Diego (UCSD), California, USA from 2014 through 2018. His research work focused on the role of sialic acids and Siglecs (Sialic acid binding lectins) in cancer, Alzheimer's, and sepsis. He has done my PhD from ETH Zurich, Switzerland.

Duygu Duzgun*, Sebastian Oltean

Department of Clinical and Biomedical Sciences, University of Exeter Medical School, EX1 2LU, Exeter, United Kingdom

The role of SRPK1 in tumour chemoresistance

Resistance to chemotherapeutic drugs is a major setback in cancer therapy, which leads to a high proportion of relapses and poor survival outcomes in cancer patients. Chemoresistance is frequently elicited by abnormal pre-mRNA alternative splicing, regulated by crucial kinases such as the serine-arginine protein kinase 1 (SRPK1). While SRPK1 has been implicated recently in chemoresistance in several tumours, the molecular mechanisms of this process are not known. In the present study, we aim to investigate in depth the role of SRPK1 (and other kinases from the same family) in chemoresistance across multiple cancers. We first generated two cisplatin (CDDP)-resistant cell lines (breast cancer CDDP-4R and colon cancer HCT-116-4R) and a docetaxel (DTX)-resistant (prostate cancer PC-3-5R) cell line by continuous exposure (over a period of 8 months) of PC-3, MDA-MB-231 and HCT-116 cells to sub-lethal, stepwise increasing concentrations of drugs. Subsequently, we evaluated how sensitivity to chemotherapeutic drugs measured by the MTT assay changes with SRPK1 inhibition using SPHINX31 in the three cell lines. Combining SPHINX31 with chemotherapeutic drugs significantly sensitizes the parental (sensitive) cell lines but less prominently the resistant cell lines. Immunoblotting showed that the level of SRPK1 in the parental (sensitive) MDA-MB-231 cells increased when treated with CDDP alone as well as in combination with SPHINX31. In resistant MDA-MB-231 cells, CDDP did not increase SRPK1 expression on its own but did so when combined with SPHINX31. In the parental (sensitive) HCT-116 colon cancer cells, both CDDP on its own and in combination with SPHINX31 decreased the expression of SRPK1, while in the resistant line, both treatments increased the expression. Finally, in the parental (sensitive) PC-3 prostate cancer cells, treatment with DTX alone or in combination with SPHINX31 decreased SRPK1 expression, while in the resistant PC-3 line, DTX on its own did not affect SRPK1 expression; however, when combined with SPHINX31, expression of SRPK1 decreased. This indicates that SRPK1 protein levels might be differentially regulated in various resistant cell lines and support the hypothesis that SRPK1 is involved in chemoresistance. Potentially, our future studies will reveal the role of SRPK1 in the development of chemoresistance in cancer cells and suggest a potential therapeutic avenue for overcoming chemotherapy resistance.

Audience Take Away Notes

- To learn how to become chemoresistance in multiple types of cancer in experimental medicine
- Establishment models of acquired chemotherapeutic drugs resistance
- Generating experimental model related to chemoresistance

Biography

Duygu studied Bioengineering in Gaziosmanpaşa University, Turkey. This was followed by MSc qualification at Gaziosmanpaşa University (Turkey) where she became interest in studying Recombinant DNA Technology. She got a full scholarship by the Turkish Ministry of National Education to study her MSc and PhD education in the UK to become an expert in the field of molecular pathology. In 2021, she obtained a MSc (Genomic Medicine) under the supervision Professor Sebastian Oltean from the University of Exeter where she studied the alternative splicing in vitro, with focus the importance of FGFR2 in prostate cancer as well as a potential therapeutic strategy.

Lian Ulrich

Retired consultant from Department Gynaecology, Copenhagen University Hospital Rigshospitalet, Copenhagen, Denmark

Gynecological sarcomas - The challenges of diagnosis and treatment

Sarcomas are rare gynaecological tumours, but because most have a bad prognosis, it is important to know when to suspect a sarcoma and to be able to diagnose it from the common uterine fibroleiomyomas. The most common variety of gynaecological sarcomas is the leiomyosarcoma, which constitute about half of gynaecological sarcomas. It is followed by the low malignancy endometrial stromal sarcoma (about 20%) and the undifferentiated sarcoma (about 15%). The remaining 15% represent a long list of even more rare tumours. The suspicion of sarcoma should be raised if uterine tumours grow after menopause in women not receiving estrogen treatment. The mean age is 50-55 years, but sarcomas are seen also in young women. Furthermore, the incidence in women in their 40es and 70es is not much less than in the 50es and 60es. Gynaecological Ultrasound investigation may rise suspicion of malignancy sarcoma based on patterns, especially when supplemented by doppler. CT scans can be used to diagnose distant metastases, but MRI is better to differentiate between malignant and benign tumours, and to evaluate resectability. PET-CT is useful when high malignant potential is known, e.g., when recurrence is suspected, but not useful in case of stromal sarcomas of low malignant potential. If biopsy is considered, multiple samples should be taken. Surgery with wide excision and clear margins is the cornerstone of treatment and should be done in tertiary centres. Bilateral oophorectomy is recommended in endometrial stromal sarcoma patients, and subsequent estrogen therapy should be avoided. Lymphadenectomy in most cases does not result in a better prognosis, unless metastases to lymph nodes are present and resectable. Morcellation is contraindicated in sarcoma surgery. If surgery cannot be radical, it is best avoided. Adjuvant chemotherapy is generally not recommended, and radiation should be reserved for special cases. Both chemotherapy and radiation can be considered in stage III and IV patients.

Audience Take Away Notes

- When to expect a tumour is a sarcoma and not a fibroma
- How to investigate, if sarcoma is suspected
- Extent of surgery

Biography

Lian Ulrich is retired consultant from University Hospital in Copenhagen, Rigshospitalet, Dpt. of Gynecology, Oncological team, where she had special responsibility for the treatment of sarcomas. She holds the university's gold medal, has published +30 articles in international peer-reviewed journals and two chapters in medical textbooks. She is reviewer for international journals and author of Danish and international clinical guidelines. She has given +100 lectures nationally and internationally, served as chairman and chaired and/or taught +50 courses for medical personnel. She is in "Who is Who in the World" since 1999.

Akshay Kishore Nadkarni[1*], Aditi Nadkarni[2]

[1]MS/DNB/ Dip Lap Germany – Director & Consultant Cancer & laparoscopic surgeon at 21st century hospital Vapi Gujarat India

[2]DGO/Dip Lap Germany – Consultant gynecologist at 21st century hospital Vapi Gujarat India

Evolution of laparoscopic oncosurgeries in rural western India – Our experience at a tertiary level 100 bedded private hospital over last 12 years

Laparoscopy has been a boon to mankind. Its first use has evolved during the 80's & 90's only for benign diseases. Over the years, it has evolved in complex cancer surgeries mainly abdominal & thorasic cancers. Last 10 years has seen the outcomes of minimally invasive cancer surgeries & its advantages to open surgeries with the advances in technology-instruments & techniques specially for colorectal, gynecology & gastrointestinal cancers.

We started our laparoscopy career in 2010 & started off with benign surgeries & eventually started minimally invasive cancer surgeries in rural western India. The most common cancer in India in females is still cervical cancers & the main reason is poor socioeconomic class - poor personal hygiene leading to HPV virus infection & white discharge – lack of knowledge & lack of facilities in rural India – leading to late detection. In the last 12 years, as a single private institute for cancer, we have developed techniques & facilities for the poor patients making minimally invasive surgeries affordable for all by cost cutting methods & quality assurance along with abiding to oncologic principles – catering to over 20 lakh population covering a distance of 200 sq kms between two major cities of Mumbai & Surat- where the next nearest cancer facilities are available. This presentation shall share the experience of evolution from a basic laparoscopic centre doing benign work to eventually developing a cancer institute & robotic centre in a rural place along with developing a training academy for gynecologists for minimally invasive cancer surgeries – a pure hands-on experience. An experience of over 6000 benign gynecology (fertility enhancing & radical) laparoscopic procedures & 350 plus laparoscopic cancer cases in a single institute in rural India.

Audience Take Away Notes

- This presentation shall discuss the laparoscopic techniques for benign and cancer cases – share the experience of time taken to evolve from benign to cancer surgeries – share the numbers -complications & follow up of the cases and inspire & encourage surgeons to evolve into minimally invasive cancer pelvic surgeries
- It will also touch the problems we faced in evolving in a rural place away from cities & inspire young surgeons to settle in rural places & not cities
- It will show how a small team can do multiple things in a small place and still give good services with care & ethics. The author is a trainer in gynec endoscopy as well & trained over 600 gynecologists in last 6 years – he is one of the few who gives pure hands-on training to doctors on patients for evolving laparoscopy
- This presentation shall encourage people to share their knowledge- be open to sharing & replicating by standardizing the technique & steps inspiring youngsters to start teaching

Biography

MS, DNB cancer surgeon & director – 21st century hospitals vapi India born in 1983 in rural India to parents who sacrificed their life for rural patients. Topper in school & got Presidents award for max marks in biology in India At school 12th standard level. Eventually completed MBBS from a rural college in Gujarat with gold medal & topper. MS general surgery & DNB from Manipal institute with best outgoing student of the year 2010. Completed fellowship from tata memorial hospital Mumbai in cancer surgeries & did diploma in lap surgery from Geissen University Germany. Started cancer centre in rural India in 2012 & now director and cancer surgeon catering to rural people of western India & runs a complete cancer care centre with radiation, medical & surgical onco facilities & recently got the CMR robot for the rural people – the first of its kind in rural India. Has been awarded bharat jyoti award from govt for his tremendous efforts to develop cancer awareness campaigns for education rural people & encouraging early detection & has 8 international research papers to his name.

Nauf Bou Antoun*, Athina-Myrto Chioni
Biomolecular Sciences Department, Kingston University London, United Kingdom

Differentially expressed genes between wild type and drug resistant human cervical cancer cell lines

Background: Cervical cancer although is highly preventable, is still the 4th most common cancer in women globally. World Health Organization estimate 640,000 new cases and 342,000 deaths in 2020, indicating the need for effective treatment at late stages of the disease, when it is usually diagnosed. Fibroblast Growth Factor (FGF) signaling plays an important role in angiogenesis, cell differentiation, migration, proliferation, wound repair and apoptosis. However, when FGFR signaling is deregulated, for example due to activating mutations, gene amplification and oncogenic fusions, it can contribute to the progression of many cancers, including cervical. Interestingly, cancers with aberrant FGFRs face a major challenge which is the development of resistance against the FGFR inhibitors. To investigate possible mechanisms of drug resistance we have generated three Drug Resistant (DR) human cervical cancer cell lines (HCCCL; Caski, HeLa and SiHa) to an FGFR inhibitor, PD173074. The main aims of this project are to (1) characterise the PD173074-resistant cancer cervical cell lines and (2) investigate possible mechanism(s) of drug resistance by comparing their transcriptome to their equivalent wild type cell lines.

Methods: Cell proliferation, apoptosis and lateral cell migration were studied using Incucyte zoom system to examine the functional differences between the wild type and DR HCCCL. Western blot analysis were performed to investigate the activation of MAPK, AKT and S6 downstream signaling pathways upon stimulation with recombinant FGF2, FGF4 or FGF7 ligands in the presence and absence of the FGFR inhibitor, PD173074. Transcriptomic analyses were performed to detect Differentially Expressed Genes (DEG) and their protein-protein interactions between DR and wild type HCCCLs. Validation of transcriptome was done by real-time PCR and Western blot using specific primers and antibodies for the identified genes and proteins.

Results: PD173074-resistant HCCCL had higher IC50, were more proliferative and migratory and less apoptotic than the wild type cells with and without PD173074. Biochemical studies revealed Phospho-ERK activation after FGF2, FGF4 and FGF7 stimulation in both the wild type and drug resistant cells. Interestingly, in the presence of the FGFR inhibitor the effect of the phosphorylation was abolished in both cell lines indicating that the DR HCCCL might still have some sensitivity to the drug, despite having lower IC50. However, there was no difference in Phospho AKT expression level at either Ser (473) or Thr (308) sites in the presence or absence of the drug in both wild type and DR cell lines. Transcriptase analysis and subsequent validation via RT-PCR revealed five up regulated and nine down regulated DEGs in DR HCCCL as well as several protein-protein interactions between them. For example PHLDA1 was significantly dowregulated in DR HCCCLs and when overexpressed, DR cells had a less metastatic behavior similar to wild type. In contrast, PLCB4 gene was up regulated in DR HCCCLs.

Conclusion: Our data suggest that DR cell lines have a more metastatic signature compared to wild type HCCCLs. PHLDA1 and PLCB4 were two key genes that were highlighted as potential mechanisms of drug resistance in HCCCLs. Further investigation on the mechanisms of drug resistance will help to design therapeutic strategies to overcome it and improve treatment.

Audience Take Away Notes

- Highlight the involvement of FGFR in cervical cancer
- Tackle possible mechanisms of drug resistance in this disease
- Knowledge about the factors that underlie mechanisms of drug resistance in cervical cancer will encourage further investigation that will help designing therapeutic strategies to overcome chemoresistance and improve treatment

Biography

Mrs Nauf Bou Antoun, a part time PhD researcher in the department of Biomolecular Sciences at Kingston University London working under the supervision of Dr Athina-Myrto Chioni. Her research focuses on investigating the factors that underlie the mechanism of drug resistance in cervical cancer to help designing therapeutic strategies to overcome resistance and improve treatment in this disease. In 2017 she held a position of visiting researcher for 6 months in the Institute of Reproductive and Developmental Biology (IRDB) building at Imperial College London. During this period, she worked on a glioblastoma project from which she gained valuable skills on a wide range of laboratory techniques In January 2019; she got awarded from Advance HE and Kingston University an Associate Fellowship in the Higher Education Academy.

Akshay Kishore Nadkarni[1*], Aditi Nadkarni[2]

[1]MS/DNB /Dip Lap Germany – Director & Consultant Cancer & laparoscopic surgeon at 21st century hospital Vapi Gujarat India

[2]DGO/Dip Lap Germany – Consultant gynecologist at 21st century hospital Vapi Gujarat India

Laparoscopic radical hysterectomy – Our experince in rural south Gujarat India

Introduction: Laparoscopy has been a boon to mankind. Its first use has evolved during the 80's & 90's only for benign diseases. Over the years, it has evolved in complex cancer surgeries mainly abdominal & thorasic cancers. Last 10 years has seen the outcomes of minimally invasive cancer surgeries & its advantages to open surgeries with the advances in technology-instruments & techniques specially for colorectal, gynecology & gastrointestinal cancers.

We started our laparoscopy career in 2010 & started off with benign surgeries & eventually started minimally invasive cancer surgeries in rural western India. The most common cancer in India in females is still cervical cancers & the main reason is poor socioeconomic class - poor personal hygiene leading to HPV virus infection & white discharge – lack of knowledge & lack of facilities in rural India – leading to late detection. In the last 12 years, as a single private institute for cancer, we have developed techniques & facilities for the poor patients making minimally invasive surgeries affordable for all by cost cutting methods & quality assurance along with abiding to oncologic principles – catering to over 20 lakh population covering a distance of 200 sq kms between two major cities of Mumbai & Surat- where the next nearest cancer facilities are available. This presentation shall share the experience of evolution from a basic laparoscopic centre doing benign work to eventually developing a cancer institute & robotic centre in a rural place along with developing a training academy for gynecologists for minimally invasive cancer surgeries – a pure hands-on experience. An experience of over 6000 benign gynecology (fertility enhancing & radical) laparoscopic procedures & 350 plus laparoscopic cancer cases in a single institute in rural India. The first use of laparoscopic methods in gynecologic oncology dates back to the 1970s.Initially, laparoscopy was used as diagnostic tool for preoperative staging in patients with ovarian carcinoma. In 1990 Querleu first reported on the use of laparoscopy in pelvic lymphadenectomy procedures in patients with cervical cancer. Other reports soon followed, including a study on laparoscopy in paraaortic lymph node sampling, published by Herd an colleagues in 1992, and a publication in the same year by Nezhat et al. on laparoscopic radical hysterectomy to treat cervical carcinoma

Aims & Objectives: To create a data base for knowing the short and long term advantages and disadvantages of laparoscopic radical hysterectomy for CA cervix with review of literature and discussing pelvic laparoscopic anatomy.

Material & Methods: 144 cases of laparoscopic radical hysterectomy were operated at our centre between 2012-2018. Intra operative, pre and post operative parameters were noted. The technique of doing the surgery was the "Pune" technique, described by dr shailesh puntambekar. Short and long term post operative survival data and complications were analized.

Results: 144 cases of early ca cervix were operated by laparoscopic radical hysterectomy. Average operative time was 165min. average blood loss was 140ml. average hospital stay was 3 days. Vaginal margin was 2 cms.12 patients had recurrence at the end of 5years followup. Few complications were encountered.

Conclusions: Laparoscopic radical hysterectomy is here to stay and has sure short and long term benefits and comparable survival data compared to open surgeries. Better vision, better magnification, immediate post op recovery and lesser complications was seen. Over 10 years of experience & follow up is discussed, but have had an exciting experience with the technique and would love to discuss in the forum.

Audience Take Away Notes

- This presentation shall discuss our exp with lap radical hysterewctomy for ca cervix in a high volume centre in rural india- the laparoscopic techniques for benign and cancer cases – share the experience of time taken to evolve from benign to cancer surgeries – share the numbers -complications & follow up of the cases and inspire & encourage surgeons to evolve into minimally invasive cancer pelvic surgeries
- It will also touch the problems we faced in evolving in a rural place away from cities & inspire young surgeons to settle in rural places & not cities
- It will show how a small team can do multiple things in a small place and still give good services with care & ethics
- The author is a trainer in gynec endoscopy as well & trained over 600 gynecologists in last 6 years – he is one of the few who gives pure hands-on training to doctors on patients for evolving laparoscopy
- This presentation shall encourage people to share their knowledge- be open to sharing & replicating by standardizing the technique & steps inspiring youngsters to start teaching

Biography

MS, DNB cancer surgeon & director – 21st century hospitals vapi India born in 1983 in rural India to parents who sacrificed their life for rural patients. Topper in school & got Presidents award for max marks in biology in India At school 12th standard level. Eventually completed MBBS from a rural college in Gujarat with gold medal & topper. MS general surgery & DNB from Manipal institute with best outgoing student of the year 2010. Completed fellowship from tata memorial hospital Mumbai in cancer surgeries & did diploma in lap surgery from Geissen University Germany. Started cancer centre in rural India in 2012 & now director and cancer surgeon catering to rural people of western India & runs a complete cancer care centre with radiation, medical & surgical onco facilities & recently got the CMR robot for the rural people – the first of its kind in rural India. Has been awarded bharat jyoti award from govt for his tremendous efforts to develop cancer awareness campaigns for education rural people & encouraging early detection & has 8 international research papers to his name.

Hui Yu*, Haoyi Zhao, Ruifang An

Department of Gynecology and Obstetrics, The First Affiliated Hospital of Xi'an Jiaotong University, Xi'an, Shaanxi Province, China

A biomimetic nanoreactor for combined chemo/chemodynamic therapy on tumor through synergistic apoptosis and ferroptosis strategy

Chemotherapy is a traditional method in clinical cancer treatment. As a malignant solid tumor caused by abnormal proliferation of trophoblastic cells, choriocarcinoma can be cured by chemotherapy for preserving patients' fertility potential. But at present the biggest hurdle in cancer treatment is insufficient delivery of chemotherapeutics to solid tumors leading to serious side effects and drug resistance. The development of nanomedicine provides a new idea to solve the dilemma and have shown great potential in the application of tumor therapy. Biomimetic metal-organic framework loaded with antineoplastic drugs can achieve targeted drug delivery, which enriches drugs at tumor sites and reduces the toxic side effects in normal tissues. Therefore, we developed a biomimetic nanoreactor derived from cancer cell membrane coated metal-organic frameworks encapsulating methotrexate to achieve maximum therapeutic effects and minimum adverse side-effects. In this work, the biomimetic nanoreactor (MMC NPs) are built from Methotrexate (MTX)-loaded iron-based metal-organic Framework (Fe-MOFs) and cancer cell membrane camouflage (MMC NPs). The successful preparation of biomimetic nanoreactor was proved by a series of characterization methods, including Transmission Electron Microscopy (TEM), X-ray Diffraction (XRD), Fourier Transform Infrared (FTIR) spectroscopy, UV-visible spectrophotometer and western blotting. The biomimetic nanoreactor could be specifically targeted to the tumor site and accumulated in the tumor in biodistribution studies. There were potentiated anti-cancer effects of MMC NPs on JEG-3 cells, including an antiproliferative effect by G0/G1 phase cell cycle arrest and a reduction in endocrine activity and migration ability. Furthermore, the overproduction of highly toxic •OH by Fenton reaction led to excellent Chemodynamic Therapy (CDT) effect, Glutathione (GSH) scavenging caused intracellular redox dyshomeostasis, and lipid peroxidation resulted in cell death by concurrent apoptosis and ferroptosis. A subcutaneous xenograft tumor model of choriocarcinoma demonstrated that the biomimetic nanoreactor showed excellent tumor targeting and achieved satisfactory tumor inhibition effects without appreciable systemic toxicity. Further transcriptomics analysis showed that the specific mechanism of the MMC NPs is through apoptosis and ferroptosis. These elaborately synthesized nanoparticles are promising as multifunctional platforms for cancer therapy due to their excellent biocompatibility and easy preparation. In summary, this nanoreactor caused obvious tumor cells apoptosis by Chemotherapy (MTX) and ferroptosis by GSH depletion-enhanced CDT (MIL-100 NPs). This new strategy for the cooperative combination of chemotherapy and chemodynamic therapy provides a reference for cancer therapy due to their excellent performance.

Audience Take Away Notes

- JEG-3 membrane camouflage could deliver MTX to the tumor site, lessen the drug dosage and reduce side effects incidentally. Cooperation with CDT effect generating by Fenton reaction, the therapeutic efficiency of MMC NPs is further amplified basing on highly toxic
- OH, glutathione scavenging and lipid peroxidation. In vivo and in vitro experimental results clearly illustrate the ability of kill tumor cells and satisfactory biological safety of this nanoplatform

- This elaborately synthesized multifunctional nanoreactor could achieve accurate targeting oncotherapy by easy preparation, taking full advantage of nanomedicine to the cancer treatment
- Biomimetics have excellent tumor targeting ability which extracts different cancer cell membrane from different cancer cells which preserve homologous targeting to recognize tumor site
- Biomimetic nanoreactor loading first-line chemotherapeutics methotrexate maybe a new method to treat choriocarcinoma patients with excellent tumor killing ability and little side effects, thereby avoiding hysterectomy and preserving patients' fertility potential
- As the mechanism of tumor treatment is interrelated in some way, the achievements of our work could also be applied in other pathological types of tumors or molecular biology in future research. Other faculty can use biomimetic strategy from this research to expand their research about other cancer

Biography

Hui Yu obtained Bachelor Degree from Xi'an Jiaotong University in 2016. She then was trained as a resident doctor of Gynecology and Obstetrics Department in the First hospital of Xi'an Jiaotong University. She received her MS degree in 2019 at the same institution. She is a PhD candidate of Xi'an Jiaotong University, majoring in Obstetrics and Gynecology. She is conducting research about gynecological cancers supervised by Prof. Ruifang An. She has published 5 research articles in SCI journals and 15 articles in CSSCI journals.

Yanting Zhang
School of Public Health, Guangdong Medical University, Dongguan, Guangdong, China

The global landscape of nasopharyngeal cancer incidence and mortality in 2020 and projections to 2040

Background: Nasopharyngeal Cancer (NPC) is one of the most common cancers in head and neck. Understanding the current epidemiological profile of international variations in NPC incidence and mortality and predicting the future NPC burden allows policymakers to make evidence-based decisions for primary prevention and to optimize the allocation of resources to reduce the global burden of NPC. Our study aims to examine global epidemiological profile of NPC incidence and mortality in 2020 and the projected burden in 2040.

Methods: Estimates of NPC cases and deaths were extracted from the GLOBOCAN 2020 database. Age-Standardized Incidence Rates (ASIRs) and age-standardized mortality rates (ASMRs) per 100,000 person-years were calculated. The predicted burden of incidence and mortality in 2040 was calculated based on global demographic projections.

Results: Globally, approximately 133,354 new NPC cases and 80,008 deaths occurred in 2020, corresponding to ASIRs and ASMRs of 1.5 and 0.88 per 100,000, respectively. The incidence and mortality rates were approximately 2.7-fold higher among males (2.2 and 1.3 per 100,000, respectively) than females (0.82 and 0.47, respectively). Incidence rates varied markedly across world regions, approximately 29-fold among males and 63-fold among females, with the highest ASIRs for both males and females detected in South-Eastern Asia (7.7 and 2.5 per 100,000, respectively) and Eastern Asia (3.9 and 1.5, respectively) and the lowest in Central America (0.27) among males and Melanesia (0.04) among females. The highest ASMRs for both males and females were found in South-Eastern Asia (5.4 and 1.5 per 100,000, respectively). At the national level, among males, the both incidence and mortality rates were highest in Brunei Darussalam (13.4 and 8.0 per 100,000, respectively), Indonesia (10.7 and 7.7, respectively), and Maldives (10.7 and 7.7, respectively). Among females, the highest incidence rates were detected in Brunei Darussalam (6.4 per 100,000), Maldives (3.3), and Malaysia (3.1), whereas the highest mortality rates were found in Brunei Darussalam (3.4), Timor-Leste (2.3), and Lao People's Democratic Republic (2.2). By Human Development Index (HDI) group, the majority of NPC burdens (69.9% of new cases and 68.6% of deaths) occurred in high HDI countries. Global burden of NPC is predicted to increase to 179,312 new cases (34.5% more than in 2020) and to 113,603 deaths (42.0%) by 2040.

Conclusion: Geographical distributions of NPC incidence and mortality varied markedly worldwide. Our study highlights the urgent need of developing and accelerating NPC control initiatives for high-risk populations to tackle the global NPC burden and narrow its geographical disparities between countries.

Keywords: Nasopharyngeal cancer, Incidence, Mortality, Epidemiology, Worldwide.

Audience Take Away Notes

- The current epidemiological profile of international variations in nasopharyngeal cancer incidence and mortality in our study can provide important evidence for clinicians and public health policymakers to plan appropriate nasopharyngeal cancer control strategies and optimize the allocation of resources for screening, diagnosis, and therapy for high-risk populations to reduce the global burden of nasopharyngeal cancer and narrow its geographical disparities
- If no further nasopharyngeal cancer control action was taken, nasopharyngeal cancer is expected to contribute to a substantial number of cases and an important cause of cancer mortality worldwide

Biography

Dr. Zhang received her PhD degree in 2022 at Sun Yat-Sen University in China. Dr. Zhang studied global cancer burden and has published several related articles.

Salma Alawadi Dawood*, Ahmed Abdelatif
Department of biotechnology, American University of Cairo

Cytotoxic activity of Salvia Officinalis in targeting JAK2/STAT3 pathway in breast cancer

Background: Breast cancer is the most common type of invasive cancer in women in their forties and fifties. Recent evidence suggests that JAK2/STAT3 signaling is constitutively active in breast cancer. Previous studies suggest that plant extracts, including *Salvia Officinalis*, have strong cytotoxic effects on breast cancer cells. The differential expression of miRNAs is also strongly linked to cancer initiation and progression.

Aim: In the current study, we hypothesize that S. Officinalis extract suppresses JAK2 expression and has strong anticancer potential in MCF7 breast cancer cells in vitro.

Methods: GC-MS analysis showed the presence of flavonoids in Salvia officinalis Extract. The cytotoxicity of S. Officinalis was compared to Cisplatin on human breast (MCF-7) cells. Bioinformatic analysis was performed to detect the link between JAK2 and different microRNAs. qPCR assessment of microRNAs and JAK2 , BAX, Bcl-xL and BIRC5 mRNAs was performed. miR-216a-5p was overexpressed in MCF7 cells to test its anticancer potential.

Results: GC-MS analysis showed the presence of anticancer and antioxidant compounds (Linolein and Apigenin). S. *Officinalis* extract reduced cell proliferation of MCF-7 cells with an IC50 range from 5.123 to 6.345 mg/mL ($p<0.0001$) compared to cisplatin (IC50 =20 ug/ul). S. *Officinalis* was also safe for the human skin fibroblast, suggesting that S. *Officinalis* has anticancer activity and is less harmful to normal cells ($p<0.0001$). Morphological assessment of MCF-7 cells showed that untreated cells maintained their epithelial morphological shape, while those treated with S. *Officinalis* displayed morphological changes consistent with apoptosis. Bioinformatic analysis revealed that JAK2 contains two theoretical binding sites of miR-101, miR-216, and miR-204 in its 3` UTR. qPCR revealed that three miRs (miR-101, miR-216a-5p & miR-204-5p) were low expressed in breast cancer cell lines than in normal cell lines ($P=0.0022$). S. *Officinalis* and cisplatin reduced the expression of miR-101-5p ($p<0.0001$). While S. *Officinalis* reduced the expression of miR-216 and miR-204, Cisplatin, on the other hand, increased their expression ($p<0.005$). Also, qPCR showed that S. *Officinalis* and miR-216a-5p mimics significantly reduced JAK2 mRNA expression ($p<0.0001$). Both S. *Officinalis* and miR-216a-5p increased the expression of BAX and reduced the expression of Bcl-xL and BIRC5.

Conclusions: S. *Officinalis* has significant anticancer potential mediated through the increased expression of BAX and reduced expression of JAK2, Bcl-xL, and BIRC5 mRNAs. As well as the reduced expression of miR-101, miR-216, and miR-204.

Keywords: *Salvia Officinalis*, miR-101-5p, miR-216a-5p & miR-204-5p, JAK2, BAX, Bcl-xL, BIRC5

Audience Take Away Notes

- To explain the importance of genetics in understanding the development of breast cancer
- Using different types of treatments to see to what extent miRNA and plant extract can be a potential therapeutic role for breast cancer
- Illustrate the different laboratory methods that were used such as transfection

Biography

Salma Alawadi Dawood graduated from college of pharmacy in 2017. Then, in Fall 2020, she started her master's degree in biotechnology in American university in Cairo (AUC). After a year and half, she started her career to be a research assistant in AUC. In Spring 2023, she is going to present her thesis.

Dhanapal Sakthisekaran

Retired Professor and Head Department of Medical Biochemistry, University of Madras, Taramani Campus, Chennai-600 113, India

Role of natural antioxidants in cancer chemoprevention and chemotherapy

Cancer chemoprevention is the use of natural or synthetic compounds for the inhibition, delay, or reversal of carcinogenesis. Chemotherapy is one of the most common treatments for cancer. It uses certain drugs to kill cancer cells or to stop them from growing and spreading to other parts of the body. There are several approaches for the primary prevention and treatment of cancers. One effective way is to modify food habits by including dietary micronutrients. Hence, researchers have focused on micronutrients as the best way to cancer chemoprevention and chemotherapy. Several micronutrients have attracted the attention of the scientific community as potential cancer-preventive agents. Among them, curcumin, piperine and Tangeritin, etc. have been studied because of the protection they offer against oxidative stress. These antioxidants are found to be very effective in minimizing the side effects induced by anticancer drugs. The hemopreventive/chemotherapeutic efficacy of these natural antioxidants in experimental cancer will be discussed.

Audience Take Away Notes

- The Audience will be encouraged to use micro nutrients as natural anti-oxidants to prevent free radical-induced lipid peroxidation and other related cellular damage
- It is highly recommended to researchers to use the micronutrients as natural anti-oxidants along with known anti-cancer drugs as combination therapy in experimental cancer-bearing animals to explore the possibility of using the anti-oxidants in clinical studies

Biography

Professor Dr.Dhanapal Sakthisekaran has obtained his Ph.D., degree from the University of Madras. He has joined as Lecturer in the Department of Medical Biochemistry, University of Madras in 1978 and subsequently promoted as Reader and Professor in 1988 and 1994 respectively. He is having about 45 years of Professional standing. He has published more than 140 research papers in reputed journals and written four textbooks in Biochemistry as Chairperson. He has received many awards for his contributions to the field of Basic Medical Sciences. He has won Indian Council of Medical Research Award (Govt.of India) in 2003 and Tamil Nadu Scientist Award in the year 2005. He has received SOMPS Poster Award from ICACT, Paris, France in 2008. He has guided 49 PhD students and all of them are well placed in India and abroad. He was the Editor in Chief of the journal BIOMEDICINE published by the Indian Association of Biomedical Scientists, India. He has retired in 2014 as Professor and Head of the Department of Medical Biochemistry, University of Madras and served as UGC –BSR Faculty Fellow from 2014 -2017.

Abdulkadir Geylani Sahan
Suleymaniye Foundation Natural Medicine Institute, Turkey

Novel QRN substance effect of non small cell lung cancer cells

Introduction: Cancer is a scary name even though it is named after an innocent animal, even called a disease. It has sought the door to many different ideas working to experience many other theories and treatment alternatives as a disease. Despite the drugs that are still used in many types of treatment, the only real death that cannot be prevented due to the disease is the end. When the products needed are provided, the old chemical balance is restored, otherwise every cell must continue to do its job, which is to keep the organism alive, even if it is uninterrupted. For this purpose, we have been examining and working on various studies and various sources for 11 years, and we determined the name that enabled us to find this substance as the name of the raw material of the study. Like all scientists, our aim is to wake up to a better world the next day and compete in goodness.

Material-Method: An MTT assay is a colorimetric assay based on assessing the cell metabolic activity. A549 Lung adenocarcinoma cell line was used to see the cytotoxic potential of a new drug for initial screening of apoptosis or necrosis. The formed formazan can be dissolved with Dimethyl Sulfoxide (DMSO) to give a purple color with characteristic absorption at 540 nm. Intensity of purple color is directly proportional to the cell number and thus indicating the cell viability. We use MTT method evaluate QRN substance effect of A549 non-small cell lung cancer cell line.

Result: After culturing the A549 cell line, the drug was administered for 24, 48, 72 hours. While the number of cells in the control group increased over time, a significant decrease in cell proliferation was observed in the drug(QRN substance)-administered cell groups. In the control and bdH2O groups, the number of A549 cells increased with time. In the QRN administered group, the number of viable cells decreased by 62.8% in 24 hours, 52.6% in 48 hours and 35.8% in 72 hours.

Discussion: Cancer is still an incomplete treated disease with many treatment modalities. For this reason, there is a need for new treatment and alternatives in the birth, formation and development stages in the treatment treatment. Therefore, there is an urgent need for this and similar treatment studies. However, the study is the result of a study at the cell level only, and further analyzes are needed to become a treatment alternative.

Keywords: QRN substance, Novel substance, Non-small cell lung cancer, Novel therapy.

Biography

He have been medical doctor since 11 years. He have worked forensic science medicine, pediatric surgery, obstetric & gynecology, psychiatry, before being internal medicine specialist. After; he had worked intensive care unit about two years for educating internal medicine. Diabetes educator instructor for education, internal intensive care, CVVHD (continuous veno-venous hemodialysis), CAVHD (continuous arteriovenous hemodialysis), mechanical ventilation, non-invasive mechanical ventilation field training worked in internal intensive care over the two years as a practical bought. Two articles published in international Together, the two have been published in international conferences and congresses posters are available. I have been internal medicine specialist since one and half year in Baykan, Siirt and after that; I have been surgery, internal, coronary and cardiovascular intensive care administrator a year. Intensive care administratory; I have been internal medicine specialist in Bahat hospital Istanbul. One year after this; I am currently working ESHA surgery medical center internal medicine specialist. I defined myself prone to business life time work, open communication believed, results-oriented, open to development, as new scientific developments following, niggling personality.

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17-19 AUGUST

DAY 01

POSTERS

6TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

Paiboon Jungsuwadee

Department Pharmaceutical Sciences, Fairleigh Dickinson University School of Pharmacy & Health Sciences, Florham Park, New Jersey, United States of America

Analysis of adverse events of anaplastic lymphoma kinase inhibitors reported to FDA adverse event reporting system (FAERS)

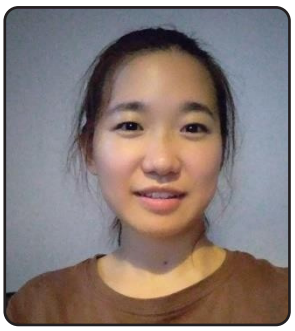
Targeted cancer therapy often causes fewer adverse effects than traditional chemotherapy drugs. Thus, targeted therapeutic drugs have become mainstream of several hematologic and solid malignancies including Non-Small Cell Lung Cancer (NSCLC). The first Anaplastic Lymphoma Kinase (ALK) inhibitor, crizotinib was approved by the US Food and Drug Administration (FDA) in 2011 for the treatment of NSCLC with alteration of ALK gene. Three more ALK inhibitors i.e., ceritinib (2014), alectinib (2015), and brigatinib (2017) were approved to enter the market. Since 2011, 19,298 adverse event cases associated with ALK inhibitors have been reported to FAERS (data as of September 30, 2022). 14,176 of those cases were serious cases (including deaths). The aim of this study was to explore the characteristics of adverse events associated with the ALK inhibitors. To establish an association between ALK inhibitors and adverse events, patient cases with polypharmacy were excluded, which brought the total number of cases down to 16,868 for analysis. Adverse reactions describing the adverse events in each individual case were mapped to System Organ Class (SOC) based on Medical Dictionary for Regulatory Activities (MedDRA) system. Of those 16,868 cases, total number of patients with at least one adverse event were on crizotinib (N=9,036), alectinib (N=3913), ceritinib (N=2183) and Brigatinib (N=1736). Common SOCs associated with ALK inhibitors were "General disorders and administration site conditions", "Gastrointestinal disorders", "Respiratory, thoracic and mediastinal disorders", "Neoplasms benign, malignant and unspecified (incl cysts and polyps)", "Nervous system disorders", "Investigations", "Cardiac disorders", "Injury, poisoning and procedural complications", "Vascular disorders", "Musculoskeletal and connective tissue disorders", and "Skin and subcutaneous tissue disorders". Since cardiac toxicities are often a major concern clinically, a breakdown of cardiac adverse reactions such as bradycardia, atrial fibrillation, pericardial effusion, dyspnoea, and cardiac arrest was further characterized.

Audience Take Away Notes

- The audience will be able learn and discuss real-world adverse event characteristics of ALK inhibitors
- The presentation will bring awareness to patients/prescribers/researchers about the organ systems that are commonly affected by the adverse effects of ALK inhibitors
- With the different adverse event characteristic profile among the four ALK inhibitors available in the market, the information presented could help the patients/prescribers as part of their informed decision-making to choose the most appropriate drug
- Other researchers could use this presentation to generate a research hypothesis or expand their own research work

Biography

Dr. Jungsuwadee received his Doctoral degree in Pharmacology and Toxicology from the University of Vienna, Austria. Dr. Jungsuwadee teaches pharmacology in several integrated pharmacotherapy courses including autoimmune diseases, cardiovascular diseases, and oncology at Fairleigh Dickinson University School of Pharmacy & Health Sciences. He also provides mentoring as well as advising to PharmD students. Dr. Jungsuwadee has published more than 30 original research articles, review articles and book chapters.



Tingting Ye*, Zifu Zhong, Bruno G. De Geest
Department of Pharmaceutics, Ghent University, Ghent, Belgium

A minimal epitope peptide lipid nanoparticle vaccine for the E7 HPV oncoprotein

Cancer vaccines based on peptide antigens aim at activating T cells and mediate tumor- specific killing, leading to durable tumor regression and prolonged survival. However, the poor efficiency of unformulated peptide antigen at generating antigen-specific CD8+ T cells in vivo remains a major limitation. Indeed, formulating peptide antigens into nanoparticles leads to improved uptake by antigen presenting cells and cross-presentation to T cells. Regarding the latter, co-delivery of peptide antigen with molecular adjuvants that induce innate immune activation, and thereby shape the magnitude and type of the adaptive T cell response, is of great relevance. Due to the broad variability in amino-acid sequence, nanoparticle formulation of peptides and co-formulation with molecular adjuvants that also differ in physicochemical properties remains a daunting task. Here we propose a generic strategy to formulated peptide antigens into Lipid Nanoparticles (LNP) by extending the amino acid sequence of the peptide antigen epitope with 10 glutamic acid residues, thereby endowing the resulting peptide with an overall anionic charge that allows for formulation into LNPs by electrostatic interaction with an - in house newly developed - cationic ionizable lipid and helper lipids in analogy to mRNA LNP formulation. Interestingly, formulation into LNPs containing an ionizable lipid can promote delivery of peptide antigen into the cellular cytoplasm and thereby improve cross-presentation of antigen. We demonstrate that in vitro and in vivo in mouse models, LNP-formulated peptide antigen shows a vast improvement in cellular uptake, while co-formulation with an imidazoquinoline TLR7/8 agonist IMDQ induces robust innate immune activation in a broad range of immune cell subsets in spleen. On the level of the adaptive immune response, we found that LNP loaded with E7 and IMDQ induces high levels of antigen-specific CD8+ T cells in the blood that can suppress the growth of E7-expressing tumors.

Audience Take Away Notes

- For clinical workers, they will find a novel cancer vaccine based on the peptide antigen and TLR agonist, this cancer vaccine performed good on tumor prophylactic model and therapeutic model
- For basic researchers, this work provides a simple, efficient and generic strategy to co-encapsulate peptide antigen and a TLR agonist IMDQ in a Lipid Nanoparticle (LNP) platform. This LNP platform was successfully used as COVID-19 mRNA delivery method, which demonstrates its potential for clinical translation

Biography

Tingting Ye graduated as a Master of Clinical Medicine at Zhejiang University, China. In 2019 she started her PhD research in the research group of Prof. Dr. Bruno De Geest at Ghent University. In her PhD research she focuses on self-assembled nanomaterials for anti-tumor immuno-engineering.

Young Sun Kim[1]*, Kyung Nam Kangb, Yong Sung Shinb, Ji Eun Leeb, Ji Young Jangb, Chul Woo Kim[2]

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Diagnostic value of combining tumor and inflammatory biomarkers in detecting common cancers in Korea

Background: The ultimate goal of cancer screening is to diagnose invasive cancers early, while they are still curable. We aimed to validate the diagnostic value of blood-derived protein biomarkers that we developed for six common cancer in Korea.

Methods: We have discovered 12 protein biomarkers that are useful in differentiating cancer patients from healthy controls using two-dimensional gel electrophoresis (2-DE), surface-enhanced laser desorption/ ionization time-of-flight mass spectrometry (SELDI-TOF-MS), and literature review. Cancer patients (stomach, colon, liver, lung, breast, and prostate) and control subjects were collected and tested data sets were used to generate predictive models that identify risk scores for each cancer. The validation study was done in serum samples of an independent patient cohort Receiver operating characteristic (ROC) analyses were conducted to evaluate the diagnostic performance of the biomarker combinations.

Results: The AUCs of the model in the test set were 0.971, 0.960, 0.969, 0.942, 0.834, and 0.985 for stomach, colon, liver, lung, breast, and prostate cancer, respectively.

Conclusions: Combining multiple tumor and systemic inflammatory biomarkers proved to be a valid strategy in the diagnosis of six common cancers in Korea. Further validation of appropriate screening populations through large-scale clinical trials are warranted.

Ji Hyun Song[1*], San Wang[2], Young Sun Kim[1], Sun Young Yang[1], Hae Yeon Kang[1], Marharyta Kurban[2], Sanghee Lim[2], Jeong Yoon Yim[1]

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Explainable gastric cancer risk prediction: Machine learning based models trained on data from regular health check-up programs of asymptomatic screening population

Background: Since early diagnosis and treatment of gastric cancer increases the survival rate, regular gastric cancer screening is recommended for the high-risk group of gastric cancer. Objective risk stratification and assessment would be helpful to develop personalized strategies for follow-up screening.

Objective: Using medical annual check-up data, we aim to develop Machine Learning (ML)-based risk stratification models for gastric cancer.

Methods: Comprehensive medical annual check-up data, including endoscopic findings and blood test results, were collected from 129,223 patients who visited one of the largest medical screening facilities in South Korea. We trained the models using several survival-based ML algorithms (e.g., Extreme Gradient Boosting [XGB] Survival, DeepSurv, Random Survival Forest) as well as a conventional Cox Proportional Hazards (CPH) regression. Our model performance was also compared to previous works' benchmark models and features. We also used SHapley Additive exPlanations (SHAP) analysis to explain the model's predictions.

Results: The XGBoost Survival model with sixteen clinically explainable features achieved the best performance (avg. c-index: 0.78). Among others, Helicobacter pylori (H. pylori) infection, chronic atrophic gastritis, and intestinal metaplasia are the most significant risk factors contributing to cancer development. Explicit explanations of how models make their predictions are well-aligned with clinical intuitions.

Conclusions: Our model could serve as a basis for creating a clinical decision support system to help clinicians for assessing a patient's individual gastric cancer risk. We expect that the results of this work will be helpful for further studies on the screening interval of gastroscopy according to the individual gastric cancer risk.

Audience Take Away Notes

- Helicobacter pylori (H. pylori) infection, chronic atrophic gastritis, and intestinal metaplasia are the most significant risk factors contributing to cancer development
- Our machine learning based model could serve as a basis for creating a clinical decision support system to help clinicians for assessing a patient's individual gastric cancer risk

Biography

Dr. Song studied Internal Medicine at the Ewha Womans University College of Medicine, Korea and graduated as MS in 2006. She received her PhD degree in 2009 at the same institution. She has worked as a clinical professor at the Seoul National University Hospital Healthcare System Gangnam Center.

Kyung Hwa Choi[1], Hyungryul Lim[1], Sanghyuk Bae[2], Mina Ha[1], Ho Jang Kwon[1], Mira Yoon[3], Seonmi Hong[4], Sang Yong Eom[4], Yong Dae Kim[4,5*], Heon Kim[4]

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[5]Chungbuk Environmental Health Center, Chungbuk National University Hospital, Cheongju, Chungbuk, R. of Korea

Cancer risk in the residents living near industrial waste incinerators in Korea

Three Industrial Waste Incinerators (IWIs) were built in 1999, 2001, and 2010, within a 3 km radius of a town with a population of around 5000 in Korea. This study evaluated whether residents near these three IWIs had increased cancer incidence than those from other areas in Korea using regional health data. Standardized Incidence Ratios (SIR) were calculated using the frequency of cancer cases in the National Cancer Registry of the exposed area (Buki-myeon), Chungcheongbuk-do (Chungbuk, state including Buki-myeon), and whole Korea from 1999 to 2017. A retrospective cohort was created using National Health Insurance System data from 2002 to 2018. The exposed group was defined as those having a residential history in the exposed area. The control group was defined as those having a residential history in nearby towns or counties in Chungbuk, excluding counties having living and cultural areas in other provinces and cities. Hazard Ratio (HR) and 95% Confidence Intervals (CI) were estimated using the Cox proportional hazard model adjusted for age, level of health insurance fee, and smoking history. In the ecological study using National Cancer Registry data, the risk of all cancers, all cancers excluding thyroid, esophageal, stomach, and lung cancers in the exposed area were 1.13 (95% CI 1.03–1.24), 1.15 (95% CI 1.04–1.26), 1.91 (95% CI 1.13–2.89), 1.39 (95% CI 1.14–1.66), and 1.29 (95% CI 1.03–1.57) times higher than in whole Korea among exposed males, respectively. In the retrospective cohort, 4300 males (26,821 person-years) and 3796 females (24,746 person-years) in exposed group, 150,964 males (1,212,010 person-years) and 134,535 females (1,104,025 person-years) in control group were analyzed. After adjusting for several confounding factors, the risks for gallbladder cancer among males and kidney cancer among females were 2.65 (95% CI 1.38–5.06) and 2.82 (95% CI 1.13–7.03) times higher in the exposed group versus the control group, respectively. In summary, cancer risk was higher in Koreans having residential history living near IWIs compared to the other areas. Further study warrants nationwide effects and longer follow-up of WIs for cancers in Korea.

Audience Take Away Notes

- This result helps to understand the effect of increasing cancer incidence due to incinerators
- Although a retrospective cohort study design is applied to an environmental epidemiological study, it has limitations in exposure assessment
- Other exposure sources other than incineration facilities (vehicle traffic, factories, etc.) need to be considered

Biography

Dr. Yong-Dae Kim studied Environmental Epidemiology research group of Prof. Heon Kim at the Chungbuk National University, Republic of Korea, and received PhD degree in 2002. He supervised by Prof. Toshihiro Kawamoto at University of Occupational and Environmental Health, Japan. He is currently a professor at the College of Medicine, Chungbuk National University, and a director of the Center for Environmental Health, Chunbuk, Korea. He has published more than 100 research articles in SCI (E) journals.

Su Jung Park*, Eun Jeong Lee

Department of Brain Science, Ajou University School of Medicine, Suwon 16499, Korea

Dual roles of TRAIL-TRAIL receptor signaling for low dose Decitabine-induced anti-tumor effects on glioblastoma (GBM) cells

Low doses of DNA methylation inhibitors (DNMTi), such as DAC, exert durable antitumor effects. In this study, we demonstrated that low doses of DAC exert anti-tumor effects, at least in part, by inducing TRAIL and TRAIL receptors. DAC-induced TRAIL signal regulates specific immune genes expression through NF-kB activation. Interestingly, TRAIL-induced NF-κB activation promotes further TRAIL production, creating a TRAIL autocrine loop. Furthermore, DAC-induced TRAIL signal mediates Cas3-dependent apoptosis-pyroptosis cascades. Thus, in cancer cells with methylation-mediated defective TRAIL signaling, TRAIL-TRAIL receptor signaling restoration using DAC, can promote dual effects: cancer cell immunomodulatory activity as well as cancer cell death through apoptosis and pyroptosis. Despite its promising anticancer activity, low-dose DAC therapy is associated with relatively low response rates. Induction of TRAIL itself along with its receptors may be a useful biomarker to predict responses to low-dose DAC therapy.

Biography

Mar 2022 – Present: Research Assistant Professor, Department of Brain Science, Ajou University School of Medicine. Sep 2014 – Feb 2022: Research Assistant Professor, Dept. of Pharmacology, Ajou University School of Medicine. Sep 2009 - Aug 2014: Research Instructor, Ajou University School of Medicine. Sep 2005 – Aug 2009: Ph.D., Dept. of Pharmacology, Ajou University School of Medicine. Mar 1998 – Feb 2000: M.S., Dept. of Bioloby, Kyungpook National University. March 1994 - February 1998: B.S., Dept. of Bioloby, Kyungpook National University.

Alex Chung Ming Wu, Yick Pang Ching*

School of Biomedical Sciences, The University of Hong Kong, Pokfulam, Hong Kong SAR

Characterization of the role of Salt-Inducible Kinase 2 in hepatocellular carcinoma metastasis

Primary liver cancer is one of the most diagnosed cancer and leading cause of cancer death worldwide with Hepatocellular Carcinoma (HCC) contributing the majority of all cases. Advanced HCC has very poor prognosis due to frequent recurrence and metastasis. Salt-Inducible Kinase 2 (SIK2), a member of the AMP-Activated Protein Kinase (AMPK) family, is reported to participate in a wide range of molecular pathways, such as the PI3K/Akt and LKB1-HDAC, vital to cell proliferation and migration in tumours, including breast and ovarian cancers. However, the role of SIK2 in HCC has not been well studied. Using stable SIK2 knockout cells, we showed that loss of SIK2 led to much slower growth rate and drastic increases in mobility suggesting that SIK2 affects the proliferation and migration of HCC cells. Interestingly, restoration of SIK2 significantly suppressed the HCC cell invasiveness. Among the chemo-drugs tested, SIK2 knockout cells were more sensitive to cisplatin treatment, indicating that loss of SIK2 might affect the efficacy of chemotherapy against HCC. Taken together, these results suggest the participation of SIK2 in the progression of HCC, making it a potential novel target for effective HCC treatment.

Audience Take Away Notes

- SIK2 is important in chemosensitivity of cancer
- SIK2 is an idea target for cancer therapy

Biography

Dr. Yick Pang Ching obtained his B.Sc. in Biochemistry at Imperial College, University of London UK. He continued his PhD training in Department of Biochemistry at University of Dundee, Scotland UK. He then returned back to Hong Kong and had his first post-doctoral training in Hong Kong University of Science and Technology. After that, he moved to The University of Hong Kong, working as postdoctoral fellow and research assistant professor. Since 2007, he has been appointed as Assistant Professor in the Department of Anatomy at the same university. He is currently an Associate Professor (tenured) at the School of Biomedical Sciences, HKU. He has published more than 80 research articles in SCI (E) journals.

BOOK OF ABSTRACTS

magnus Group

17-19 AUGUST

DAY 02

KEYNOTE FORUM

6TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

Emerging role of succinate dehydrogenase (SDH) alterations in the onset of Hamartomatous Polyposis Syndromes (HPS) through STK11 and PTEN tumor suppressor genes regulation

The hamartomatous polyposis syndromes are a heterogeneous group of rare syndromes showing autosomal dominant inheritance and characterized by the onset of gastrointestinal hamartomatous polyps and increased cancer risk. Among them there are the Peutz-Jeghers syndrome, juvenile polyposis, and PTEN-related hamartomatous tumor syndrome.

The aim of this work was to identify new genes involved in the pathogenesis of the diseases, new relationships between molecular pathways involved, and new alternative therapeutic targets useful for disease management.

To realize this aim, we planned the following research stages:

- NGS analysis of a specific gene panel in 20 subjects with clinical suspicion of familial hamartomatous polyposis.
- Investigation of the STK11 and PTEN genes/protein expression, in subjects carrier of SDHB and SDHD genes variants;
- Investigation of the SDH inhibition effect on the STK11 gene expression.

Using this method, we identified 6 subjects carrying a pathogenetic variant of the STK11 gene, 1 carrier of a VUS of the STK11 gene, 1 carrier of a VUS in the SDHB gene and 2 carriers of a VUS in the SDHD gene. It was therefore observed that the presence of the SDHD gene variants is associated with downregulation of the STK11 and PTEN tumor suppressor genes. Furthermore, we in vitro demonstrated downregulation of STK11 protein due to SDH inhibition.

In conclusion, a previously undescribed regulation between the SDH enzyme and STK11/LKB1 and PTEN genes emerges, which contribute to shed light on the pathogenesis of hamartomatous polyposis and opens new insight into the standardization of a targeted therapy for the management of affected patients.

Audience Take Away Notes

- The data presented will be useful to the audience investigating the molecular basis of solid tumors. Genes, such as STK11, PTEN and SDH, are involved in the onset of colon cancer, but also of other types of cancer
- The identification of a crosstalk between the genes coding for SDH and the STK11 and PTEN genes will be useful to clarify the effects of alterations in SDH genes and to improve the known on the molecular

Francesca Cammarota[1,2], Valeria D'Agostino[2], Mimmo Turano[3], Valentina D'Angelo[4], Giovanni Battista Rossi[4], Annamaria Staiano[5], Erasmo Miele[5], Francesca Duraturo[1,2], Paola Izzo[1], Marina De Rosa[1,2*]

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Biography

Marina De Rosa (MDR) is PhD professor of Biochemistry at University of Naples Federico II. Her research activity focused on the study of molecular basis of hereditary

basis of hereditary and sporadic tumors

- In our opinion these results will be useful for the identification of new therapeutic targets
- The workflow used in this study could represent a study model for other types of autosomal dominant diseases

and sporadic colorectal cancers and Inflammatory Bowel Disease (IBD). She is editorial Board Member of the Experimental and Therapeutic Medicine journal (Spandidos publication) and Biomedicines journal (mdpi). She is membership of the Italian Society of Biochemistry and Molecular Biology (SIB) and of the "Associazione Italiana Familiarità Ereditarietà Tumori (AIFET)". She is author of 51 articles on indexed journals with a total of 1079 citations, achieving an h-index of 20 (Scopus database).

Relation of luminal and myoepithelial cells in nuclear-inverse polarity papillary lesions lacking myoepithelail cells of the breast

Breast papillary lesions exhibit broad range. Tajima et al. reported in discrimination between benign Intraductal Papilloma (IDP) and malignant Endocrine Ductal Carcinoma In Situ (E-DCIS), new marker of CD56 is useful for discriminating between benign and malignant. Relationship between luminal cells and myoepithelial cells of cell-cell interaction is important for intraductal lesion to invasive lesion in recent time. This microenvironment is correlated many factors especially synaptophysin which marker is advocated Maeda I. and Tajima S. et al. and correlated neuroendocrine marker of CD56. Here, in relation to IDP, we would like to present new concept of two papillary lesions resembling IDP. In the past, lacking myoepithelial cells thought to be invasion and means malignancy. We will present distinctive subtype which pathologists over-diagnose malignant however benign truly. We demonstrate two cases of 68- (Case1) and 44-year-old (Case2) female. They have abnormality in the breast. And they came to the hospital for further examination. Radiologically, malignancy could not be excluded. Then, breast excision was performed. Histologically, both cases revealed papillary lesions lined by fibro vascular core and nuclear inverse-polarity without atypical. Loss of myoepithelial cells was observed by HE, p63, and calponin. Previous report indicate CK5/6, ER, p63 and MUC3 are important for distinguishing between papillary lesions according to the differential index Allred score of ([ER total score] + [MUC3 total score])/([CK5/6 total score] + [p63 total score] + 1). Based on this analysis, our 2 cases had benign lesions. Additionally, the Ki-67 index was <1% in both cases, and no disease was observed minimum 62 months of follow-up, despite additional treatment. Here, we newly experimented MUC immunostainings in these cases because MUC status is important in breast diseases. We did immunostaining of MUC1,2,4,5AC,5B and 6. The results are MUC2,4,5AC and 6 are negative. MUC1 revealed apical strong staining and also MUC5B was negative. MUC1 of apical staining means benign. MUC5B is thought the staining positivity means early cancer. Hence our staining status also benign without myoepithelial cells. In conclusion, MUC immunostaining status also proved “Nuclear inverse-polarity papillary lesion lacking myoepithelial cells” are benign lesions. Our lesion is distinctive and another term of “Tajima tumor” is accepted DOI. In this congress, I emphasize lacking of myoepithelial cells does not indicate malignancy as well as correlation of luminal cells. And I think it will be increase new histological subtype of benign however without myoepithelial cells in the future. Then, we might contributing for patients well-being as well as quality of life.

Audience Take Away Notes

- Through our new histological subtype, we can reduce unnecessary operation

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Biography

Shinya Tajima MD, PhD from Japan. I graduated Keio University School of Medicine. After graduated from the university, working in Department of Pathology at the same institution. He learned general pathology. Then he would like to be a specialist of breast pathology. He affiliated St. Marianna University which is the most breast operation number in Japanese university. He received PhD in Radiologic-Pathology from same Graduate School of Medicine in Japan. He worked at the Department of Pathology and Radiology of this latter institution. Now He is working at Department of Diagnostic Pathology of National Hospital Organization Shizuoka Medical Center.

- If our tumor is commonly diagnosed, it would be better for breast tumor patients
- Our knowledge will discover common sense and contribute to daily pathological diagnoses
- In the breast, lactifellous duct is maintained by two cells of cell-cell interaction. One is luminal cell and the other is myoepithelial cell. Usually myoepithelial cells control luminal cells for their invasion. However the two cells of cell-cell interaction disordered, invasive carcinoma arises from the lactifellous duct. Hence, lacking myoepithelial cells are considered invasion and malignancy. However our cases are indicated benign however lacking myoepithelial cells. To learn our new distinctive histological subtype, we can reduce unnecessary operation and increase patients' Quality of Life
- Through our new histological subtype, audience will learn surgical pathology of importance
- If our tumor is commonly diagnosed, it would be better for breast tumor patients
- Our knowledge will discover common sense and contribute to daily pathological diagnoses

Mutation evolution of human hepatocellular carcinoma

The protein diversity of mammalian cells is determined by arrays of isoforms from genes. Genetic mutation is essential in species evolution and cancer development. Accurate Long-read transcriptome sequencing at single-cell level is required to decipher the spectrum of protein expressions in mammalian organisms. In this report, we developed a synthetic long-read single-cell sequencing technology based on LOOPseq technique. We applied this technology to analyze 447 transcriptomes of hepatocellular carcinoma (HCC) and benign liver from an individual. Through Uniform Manifold Approximation and Projection (UMAP) analysis, we identified a panel of mutation mRNA isoforms highly specific to HCC cells. The evolution pathways that led to the hyper-mutation clusters in single human leukocyte antigen (HLA) molecules were identified. Novel fusion transcripts were detected. The combination of gene expressions, fusion gene transcripts, and mutation gene expressions significantly improved the classification of liver cancer cells versus benign hepatocytes. In conclusion, LOOPseq single-cell technology may hold promise to provide a new level of precision analysis on the mammalian transcriptome.

Audience Take Away Notes

- Mutation is the fundamental cause of human liver cancer
- Mutations evolve from a single nucleotide alteration of specific region in the HLA molecules and expanded to spread to other regions of the same alleles
- Hypermutation clusters are readily identified in HLA molecules that may be essential for cancer cells to evade immune surveillance

Jianhua Luo

Department of Pathology, High Throughput Genome Center, Pittsburgh Liver Research Center, , University of Pittsburgh, 3550 Terrace Street, Pittsburgh

Biography

Dr. Luo has been studying molecular mechanisms of human malignancies in the last 35 years. Currently, he is a Professor of Pathology and Director of High Throughput Genome Center at University of Pittsburgh. In the last 29 years, Dr. Luo has been largely focusing on the genetic and molecular mechanism of human cancers such as liver cancer. He is one of the pioneers in utilizing high throughput gene expression and genome analyses to analyze field effects in prostate cancer and liver cancer. He is also the first in using methylation array and whole genome methylation sequencing to analyze prostate cancer. He and his colleague helped to develop an ultra-low error synthetic long-read sequencing technology called LOOPSeq that can be utilized to quantify mRNA isoforms and mutation isoform distributions in single cell level. His group has discovered 21 novel fusion genes in prostate, liver and colon cancers. Subsequently, his group discovered that many of these fusion genes are recurrent in many other types of human cancers. His group also developed a genome intervention strategy targeting at the chromosomal breakpoint of fusion gene to treat cancers. Overall, these findings advance our understanding of how cancer develops and behaves, and lay down the foundation for better future diagnosis and treatment for human malignancies.

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SPEAKERS

6TH EDITION OF

INTERNATIONAL CANCER CONFERENCE

Sophie Jessop*, Shandelle Hill, Tom Revesz
Women's and Children's Hospital, Australia

Improving the patient journey for First Nations children diagnosed with cancer

Background: There are disparities in outcomes for Aboriginal children with acute leukaemia, with reduced enrolment onto clinical trials and increased rates of loss to follow-up. Inequalities can result from reduced access to health-care services due to remote living, cross-cultural misunderstandings and poor understanding surrounding a cancer diagnosis.

Objective: To understand the barriers and challenges associated with an Aboriginal child being diagnosed with cancer, in the hope to lead local, national and international initiatives for change.

Method: This service provision audit was conducted out of the Women's and Children's Hospital (WCH). Patients identifying as Aboriginal Australians, and healthcare staff, including Aboriginal healthcare workers, were approached to participate. Families of deceased patients were ineligible. Consenting participants were interviewed (face-to-face or via telephone) by study members on the cancer journey.

Results: Between 2006 and 2021, 65 families undergoing cancer treatment at the WCH identified as Aboriginal. 53 were ineligible (deceased, relapsed during study, under child protection, did not attend, un-contactable). 12 participants were interviewed (9 families, 3 healthcare staff). Main themes identified were delayed diagnoses, hardships with travel, lack of access to support services and poor communication surrounding the cancer diagnosis and treatment. Positives included local network involvement, telehealth services, social work input and support from Non-Government Organisations.

Conclusion: There are many challenges faced by Aboriginal families when a child is diagnosed with cancer. Our results support the necessity to implement changes surrounding cultural sensitivity, communication and culturally adapted education of patients, and improve assistance with travel and accommodation for the patient and wider family.

Audience Take Away Notes

- There are disparities in outcomes for Aboriginal children with cancer, particularly leukaemia, along with reduced enrolment on clinical trials and increased rates of loss to follow up
- There is inequitable service provision to Aboriginal patients and families, including poor cultural sensitivity, inadequate communication, education and support schemes
- We need to raise awareness of the gap in cares, affecting outcomes
- Cultural awareness and respect is inadequate amongst healthcare staff
- Healthcare staff need to advocate for local, national and international initiatives for improvements in service provision

Biography

Sophie Jessop is a Paediatric Oncologist from the Women's and Children's Hospital in South Australia and a ZERO2 research fellow with the Children's Cancer Institute, New South Wales (NSW). She underwent training at the Women's and Children's Hospital, and completed a clinical fellowship at Westmead Children's Hospital (NSW) and a research fellowship in precision medicine at Sydney Children's Hospital (NSW). Her research interests include paediatric solid tumours, Aboriginal cancer care, palliative care, having completed a Masters in Palliative care, and precision medicine.

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Effectiveness of 123 I-BMIPP myocardial scintigraphy in breast cancer patients

Cancer Therapeutics-related Cardiac Disfunction (CTRCD) is a cardiovascular disease caused by cancer therapies. It is essential to detect CTRCD at an early stage to prolong patients' lives. However, there aren't any established screening tests other than ultrasound cardiogram (UCG). For breast cancer patients who are unable to undergo UCG due to surgery wounds or radiation scars in their breasts, Iodine-123 β-methyl-P-iodophenyl-pentadecanoic acid (123I-BMIPP) myocardial scintigraphy will become the preferable second-choice screening test for CTRCD. 123I-BMIPP scintigraphy has been performed in Japan for over 2 decades, however it is not common elsewhere. More than 100 patients were involved in the study, and the results were analyzed with Heart Risk View-S software. Myocardial scintigraphy cannot serve as a perfect alternative to UCG, however, it will become the preferable second-choice screening test, as it could point out the early stage of CTRCD.

Audience Take Away Notes

- To our knowledge, this is the most extensive research of ^{123}I-BMIPP scintigraphy for CTRCD. The audience will learn how to diagnose CTRCD without using UCG
- This research will benefit the audience who are interested in CTRCD. The guidelines have been established recently by European Society of Cardiology. CTRCD is still a new research field

Biography

Yuko Harada, M.D., received her M.D. degree from the Keio University School of Medicine. Dr. Harada is currently Vice Director of the Department of Cardiology at Kawasaki Municipal Ida Hospital. From 2018 to 2020 she was Division Head of General Internal Medicine at Yamato Tokushukai Hospital. From 2014 to 2018 she was Director of the Department of Internal Medicine at Shin-yurigaoka General Hospital. Until 2014 she was Department Chief of Cardiology at Kawasaki Municipal Ida Hospital, where she also completed her residency. Dr. Harada received the Chairman's Award from the Japan Endocrinology Association for her life-saving work on thyroid storm. She has authored numerous pioneering research and medical papers in the fields of Internal Medicine, Cardiology, and Radiology. She has chaired and has presented at numerous medical conferences.

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Bioinformatics analysis of PDIA3 as an immune and prognostic biomarker in cervical and lung adenocarcinoma and pan-cancer

Protein disulfide isomerase A3 (PDIA3) is a kind of thiol oxidoreductase with a wide range of functions, and its expression is elevated in a variety of tumors, which is closely related to the invasion and metastasis of tumor cells, and has a significant impact on the immunogenicity of tumor cells.This study aims to analyze the differential expression of PDIA3 in cervical cancer, lung adenocarcinoma and pan-cancer, evaluate its clinical prognostic significance and possible mechanism of action in different cancers, and explore its ability to regulate tumor immunity as a biomarker. This study was performed by downloading multi-level data containing 33 cancers from The Cancer Genome Atlas (TCGA), UCSC Xena, Cancer Cell Lineage Encyclopedia (CCLE), Genotype Tissue Expression (GTEx), and GDC. R software was used to conduct log2 standardization on gene expression data. The Wilcoxon rank sum test was used to compare the differential expression of PDIA3 gene between cancer and adjacent tissue samples in different tumors. The survival prognosis was analyzed by Kaplan-Meier method, log-rank test and Cox proportional risk regression model. Correlation analysis between the two variables was performed using Spearman's test or Pearson's test. At the same time, 111 cases of cervical cancer tissue and 24 cases of paracancerous tissue samples containing clinical characteristics were collected for immunohistochemical test verification. Bioinformatics methods were applied to analyze the differential expression levels of PDIA3 in pan-cancer and its clinical prognostic significance. Based on the pan-cancer study, the differential expression profile, survival prognostic value and clinical significance of PDIA3 in cervical cancer and lung adenocarcinoma were further explored. The analysis results showed that: (1) PDIA3 was highly expressed in 19 types of cancers, but down-regulated only in THCA. The expression level of PDIA3 in THYM has the strongest correlation with TMB and MSI. The methylation level of PDIA3 promoter region was closely related to patient outcome in eight tumors. (2) The expression of PDIA3 in cervical cancer is higher than that in normal tissues, which was significantly increased with the progression of tumor stage; The high expression of PDIA3 gene is associated with poor prognosis in patients with cervical cancer. The genes positively correlated with PDIA3 expression included HSPA5, PPIB, and HSP90B1, which were mainly enriched in biological processes such as endoplasmic reticulum protein folding, endoplasmic reticulum stress response. (3) PDIA3 expression was higher in lung adenocarcinoma tissues than in normal tissues, and PDIA3 showed moderate staining in normal lung tissues and strong staining in tumor tissues. High expression of PDIA3 gene is associated with poor prognosis of LUAD. In TIMER, after adjustment for tumor purity, PDIA3 expression levels were significantly correlated with 21 of the 33 immune cell markers in LUAD and significantly positively correlated with immune checkpoint expression. This study suggests that PDIA3 plays an important role in the occurrence and development of KIRP, KICH, and CESC and in the immunotherapeutic response of THYM, READ, and LGG. PDIA3 may affect lung adenocarcinoma development by regulating tumor infiltrating cells in TME, and PDIA3 can be used as a prognostic biomarker for these tumors.

Audience Take Away Notes

- Existing studies have shown that PDIA3 is closely related to the occurrence and development of a variety of diseases. Detection or intervention of PDIA3 expression levels in relevant sites may become a new target for early diagnosis and treatment of diseases

- At present, the understanding of PDIA3 is not completely clear, and further research is needed. For example, in-depth exploration of the biological function of PDIA3 can promote the understanding of the mechanism of related diseases, and studying the changes of PDIA3 in various related diseases will help to have a more comprehensive understanding of PDIA3
- PDIA3 is a reactive change in the development of the disease, or the leading cause of the development of the disease is still worthy of in-depth exploration, at the same time, gene intervention will become the main means to deepen the study of the therapeutic effect of PDIA3

Biography

Dr. Li Hui received her Doctor's Degree of Medicine from Xinjiang Medical University in 2017. Her main research interests are the molecular biomarkers and pathogenesis of esophageal cancer with high incidence in China and the material basis and molecular mechanism of the anti-diabetic retinopathy of Xinjiang Coreopsis Tinctoria Nutt. In recent years, she has published more than 20 research papers.

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Disparities of cancer statistics between China and the USA: A secondary analysis on five aspects of cancer profiles

Background: China faces a disproportionate cancer burden to the population size and is undergoing a transition in the cancer spectrum. A comprehensive analysis of the current cancer profiles in China is still urgently needed to provide references for further formulation and adjustments of cancer prevention strategies.

Methods: We extracted data in five aspects of cancer incidence, mortality, survival, staging distributions, and attribution to risk factors in China, the USA and worldwide from open-source databases, including the Global Cancer Observatory Platform, the Global surveillance of trends in cancer survival 2000–14 (CONCORD-3) program, the Surveillance, Epidemiology, and End Results (SEER) program, and the published reports released by the National Central Cancer Registry of China (NCCR). We conducted a secondary analysis of cancer statistics in China in the above aspects, and compared between China and the USA.

Findings: A total of 4,546,400 new cancer cases and 2,992,600 deaths occurred in China in 2020, accounting for 25.1% and 30.2% of global cases, respectively. Lifestyle-related cancers including lung cancer, colorectal cancer, and breast cancer showed an upward trend and have been the leading cancer types in China. 41.6% of new cancer cases and 49.3% of cancer deaths occurred in digestive-system cancers in China, and the cancers of esophagus, nasopharynx, liver, and stomach in China accounted for over 40% of global cases. Infection-related cancers showed the highest population-attributable fractions among Chinese adults, and most cancers could be attributed to behavioral and metabolic factors. The proportions of stage I for most cancer types were much higher in the USA than in China, except for esophageal cancer (78.2% vs. 41.1%). The 5-year relative survival rates in China have improved substantially during 2000-14, whereas survival for most cancer types in the USA was significantly higher than in China, except for upper gastrointestinal cancers.

Conclusions: Our findings suggest that although substantial progress has been made in cancer control, especially in digestive system cancers in China, there was still a considerable disparity in cancer burden between China and the USA. More robust policies on risk factors and standardized screening practices are urgently warranted to curb the cancer growth and improve the prognosis for cancer patients.

Keywords: Cancer Burden; Survival Rate; Neoplasm Staging; Risk Factors; China

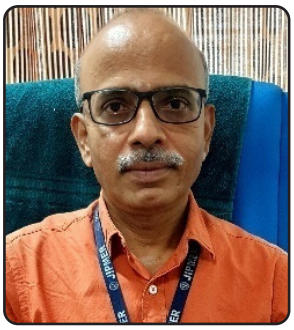
Audience Take Away Notes

- Chinese population accounted for 18% of the world's total population, whereas there were over a quarter of global cancer cases and deaths occur in China, which indicates a disproportionate cancer burden to the population at present.
- In both China and the USA, smoking and low fruit intake ranked the top five modifiable risk factors with high PAFs of cancer deaths. The highest PAFs of overall cancer deaths were also for behavioral and metabolic factors in the USA, while for infection-associated factors in China.

- The proportion of stage I diagnosis in the USA was higher than in China for cancers of colorectum, lung, breast, liver, and all cancers combined, whilst Chinese patients with esophagus cancer showed a higher proportion of stage I.
- Age-standardized 5-year survival in the USA was considerably higher than in China, except for stomach cancer and esophagus cancer.

Biography

Miss He studied Preventive Medicine and Public Health at Wuhan University, China and graduated as Bch in 2020. She then joined the research group of Prof. Wanqing Chen at the National Cancer Center of China as a PhD student, majoring in Epidemiology and Biostatistics, and is planned to receive the PhD degree in 2025 from Peking Union Medical College. Her doctoral dissertation focuses on the evaluation and optimization of endoscopic screening strategy of gastric cancer in China. She has published several research articles in SCI (E) journals.



Sharmila Baalaji MD, Neelaiah Siddaraju MD*, Debasis Gochhait MD, Debdatta Basu MD

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A comparison between the conventional and the sydney systems of reporting lymph node cytopathology

Background: In recent decades, various standardized classification systems for reporting cytopathology have been implemented, which have revolutionized the management of patients with cancerous and non-cancerous conditions by establishing effective communication between clinicians and cytopathologists. The present study evaluates the merits of the Sydney System of reporting lymph node cytopathology.

Study design: A comparison between the conventional and the recently proposed 'Sydney systems of reporting lymph node cytopathology' was carried out in the present study with 355 cases in each group. The fine needle aspiration cytological (FNAC) diagnoses and interpretations in both study groups were made as per the established criteria for each of the diagnostic entities, documented in the standard textbooks and literature, with the use of appropriate ancillary techniques, whenever possible. An algorithmic approach designed in one of our previous studies was stringently applied for all hematolymphoid neoplasms in both groups.

Results: There was no significant variation in terms of interpretative categories and the final diagnoses between the two groups. Both groups showed high sensitivity, specificity, positive and negative predictive values, as well as, diagnostic accuracy for lymph node FNAC. There was a slight decline in the rate of non-diagnostic aspirates, due to the consistent use of the ROSE technique in the Sydney group.

Conclusions: Though no significant differences were noted between the two systems of reporting, our study immensely contributed to implementing and streamlining the new system of reporting in our institute, with a more rigid and meticulous approach and the use of relevant ancillary techniques.

Keywords: Sydney system, lymph node, cytopathology, fine needle aspirates, neoplastic, non-neoplastic, ancillary studies.

Audience Take Away Notes

- As mentioned in the abstract, the standardized cytological reporting systems improve not only the pre-therapeutic diagnostic accuracy but also the effective follow-up of patients with improved communication between clinicians and cytopathologists
- The algorithmic approach used in the study for diagnosing a variety of hematolymphoid malignancies would be of great practical value, especially for budding pathologists in general and cytopathologists in particular
- The paper also emphasizes the importance of a meticulous approach to sampling, interpretation, and effective use of ancillary techniques such as immunocytochemistry and flow cytometry in the diagnosis and distinction of hematolymphoid malignancies. The aspects dealt with here are not just good teaching material but also have scope for further research

Biography

Dr. Siddaraju completed his postgraduate studies at Mysore Medical College, Mysore (Karnataka), India, in 1991. Currently, he is a senior professor in the Department of Pathology at the Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry, India. His field of interest is cytopathology. He is a postgraduate teacher and a Ph.D. guide. He has participated in various national and international conferences and published more than 130 scientific papers and a book chapter. He has been a reviewer for various peer-reviewed journals, and currently, he is also an Editorial Board Member of the Journal of Cytology.

Sujoy Neogi*, Meghna Kinjalk, Deepak Goyal, Simmi K Ratan
Maulana Azad Medical College, India

Notorious chemo-resistant childhood tumours – Is early diagnosis a saviour?

Neuroblastoma is a rare neoplasm whose prognosis becomes poor as age advances. It is a tumour of neural crest cell origin, primarily occurring in abdomen. Due to its poor morbidity and poor survival, there is no standard protocol.

We report three cases of Neuroblastoma which were resistant to treatment. A 5-year boy who presented with swelling on the back and fever. He was treated initially as a cold abscess secondary to tuberculosis. Subsequent imaging revealed it to be a metastatic lesion and biopsy along with urinary Vaninyllmandelic acid (VMA) proved it to be a Neuroblastoma. Second case was a 6-month-old girl with progressively increasing abdominal lump. Imaging and VMA levels diagnosed neuroblastoma. The third case was a 5-year-old boy presented with anasarca and respiratory distress. Initially the boy was diagnosed as Pott's spine and was treated for the same. On further imaging and investigation, it was diagnosed as abdominal Neuroblastoma with bone and pulmonary metastasis. In all these cases neoadjuvant chemotherapy (OPEC) was started but none of them responded. All the babies died of non-response and fast deterioration of the malignancy. The rising incidence of non-responsive Neuroblastoma and other malignancy is a matter of global concern and needs proper research to identify the cause and its prevention thereafter.

Keywords: Neuroblastoma, Pediatric Neuroblastoma, Non-responsive chemotherapy.

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Investigation of anticancer activity 2,3-dihydroxybenzoic acid (2,3-DHBA) against MCF-7 breast cancer cells and determination of lipid profile changes with untargeted lipidomic analysis

Breast cancer (BC) is the most commonly diagnosed cancer in women in the world. Many studies have shown that phenolic acids are anticancer agents. 2,3-dihydroxybenzoic acid (2,3-DHBA) is a natural phenolic acid found in different fruits or vegetables. Lipidomic approaches investigate changes in lipid types in both normal and pathological conditions. Various diseases, including cancer, have been shown to be associated with specific changes in certain types of lipids, and new treatment strategies involving lipid metabolism have been developed. However, no studies have been conducted to show the effects of 2,3-DHBA on the lipid species of MCF-7 human breast cancer cells. The in vitro anticancer activity of 2,3-DHBA (0.75-20 mM) against MCF-7 cancer cells and L-929 healthy mouse fibroblast cells determined by MTT (3- (4,5-dimethylthiazol-2-yl) -2,5-diphenyltetrazolium) method for 24h, 48h and 72h. 2,3 DHBA was found to have anticancer activity on MCF-7 cancer cells and this effect increased in a time and dose dependent manner. Our results indicates that the 2,3-DHBA more cytotoxic against breast cancer cells than L-929 healthy cells. The effects of 2,3 DHBA on the lipid profile of MCF-7 cancer cells were studied using the untargeted lipidomic approach for the first time. It was determined that incubation of MCF-7 cancer cells with 2,3 DHBA caused changes in glycerophospholipid, sphingolipid, glycerolipid and fatty acid lipid species. As a result, the relationship between the in vitro anticancer activity of 2,3-DHBA and the differentiation of the lipid profiles of MCF-7 breast cancer cellsthrough this activity was determined. “This work is supported by the Scientific Research Project Fund of Sivas Cumhuriyet University under the project number ECZ-049”.

Audience Take Away Notes

- The audience will gain insight into the role of 2,3-DHBA on lipid profile changes of MCF-7 breast cancer cells
- The audience will gain insight into lipidomic techniques in cancer studies
- Other faculty may shape their future research on effects of natural compounds on lipid metabolism in cancer
- The audience may apply the technique mentioned in this speech in their research

Biography

Assoc. Prof. Dr. Serap Sahin-Bolukbasi completed her Ph.D. studies on Biochemistry at The Sivas Cumhuriyet University. She performed post-doctoral studies at The University of Georgia (UGA) Collage of Pharmacy, Department of Pharmaceutical and Biomedical Sciences. She is an Assoc. Prof. Dr. at the Afyonkarahisar Health Sciences University, Faculty of Pharmacy, and Department of Biochemistry since 2021. She has published many research articles in SCI (E) journals and presented more than 80 presentations at national/international conferences. Dr. Sahin-Bolukbasi’s research focuses on lipidomic, lipid metabolism in cancer, lipid biomarkers for rare and common diseases, new treatment targets based on lipid metabolism, cell culture, development of biotechnological aptamers in cardiovascular diseases and cancer.

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Video assisted thymectomy- A new frontier in myasthenia gravis

Background: Thymoma comprises 20% of all mediastinal neoplasms and 50% of all primary tumours in the anterior compartment. Thymic surgery has undergone a paradigm shift in approach from being transcervical to Video assisted. Video assisted thymectomies in selected patients can decrease the inherent morbidities of a transternal approach, while achieving similar therapeutic benefits.

Aim: To analyse the technical key points, morbidity and outcomes associated with video assisted thymectomy in early stage thymoma (Masoaka stage I or II) with myasthenia gravis.

Method: This was a retrospective analysis of 24 patients with thymoma and Myasthenia Gravis Foundation of America (MGFA) class II to IV myasthenia gravis who underwent video assisted thoracoscopic thymectomy in our institution from May 2013 to May 2018. We included all patients with thymomas which on CECT did not show infiltration of the surrounding structures and were <5cm. All patients were operated under GA with a single lumen tube with controlled CO2 pneumothorax with right or left thoracocopic approach.

Results: 75% of the patients were male in our case series and 63% belonged to 20-40 years of age. Only 8% of the patients belonged to <20 years of age. Mean operative time was 164 +/- 8.65 min in VATS. Blood loss in VATS was 178 +/- 47ml. Mean chest tube duration was only 3.2 days. Duration of stay in the hospital was on an average only 3.2 +/- 1.45 days days. Mean VAS pain scale for VATS patients was only 3.5 +/- 1.08. Post operative complication occurred in 8.3% of the patients. 50% of the patients achieved complete remission of their myasthenia gravis symptoms and were free of any treatment.

Conclusion: From our study, it can be concluded that VATS thymectomy in selected patients is a safe and minimally morbid operation and is asscociated with decrease in length of hospital stay, decreased post operative complications, reduced pain and requirement of analgesia with reduced or similar operative time, while achieving excellent remission rates for myasthenia gravis.

Audience Take Away Notes

- This study enlists our institutional experience in managing Myasthenia Gravis patients with Thymoma using the technique of VATS thymectomy
- MGFA Class II and IV patients with <5cm tumors were involved in the study
- It provides the evidence that VATS thymoma is a safe, feasible and an excellent technique with good resolution of MG symptoms
- The result can help in designing randomized control trials for open trans-sternal and VATS thymectomy

Biography

Amiy Arnav is a surgical oncologist trained at Army Hospital Research & Referral, Delhi, India, with special interest in minimally invasive thoracic & GI malignancy management. He is Currently working as Assistant Professor, Department of Surgical Oncology, AIIMS, Deoghar since 14th August, 2021. He worked as Specialist in department of surgical oncology at Tata Main Hospital, Jamshedpur since 16th Nov, 2020 to 4th August 2021.He was involved and completed Four intramural research projects in my previous institutions. He was member of Editorial Review Board of Pubmed indexed international Journal " Cancer Treatment and Research Communications. He was the reviewer for Pubmed indexed international Journal" Cancer Treatment and Research Communications. He is also a Reviewer for Pubmed indexed international Journal "Cureus". He has published more than 12 papers on various topics in peer reviewed journals.

Mahmoud Ismail Nouh[1,2*], Hani Raka Karrar[1], Yousef Ismail Nouh[3], Mawadah Ismaeel Nooh[4], Rehab Salah Aldin Alhendi[5]

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Alpelisib plus fulvestrant in advanced breast cancer with hormone receptor-positive, human epidermal growth factor receptor-2 negative, and PIK3CA mutation

Breast cancer is a type of cancer that develops in the breast tissue. It is the most common cancer in women worldwide. Breast cancer developed silently without medical attention, and most cases are discovered during a routine clinic visit. Mortality rates of breast cancer getting improved, but still, the survival rate in the advanced stage getting decreased. Breast cancer is typically diagnosed through a combination of mammography, ultrasound, and biopsy. Treatment options can include surgery, radiation therapy, chemotherapy, hormone therapy, and targeted therapy, depending on the stage and characteristics of cancer. Chemotherapy considers the gold-standard approach for most cancer types and the modest improvement in both survival rates and toxicity reduction. Wherefore, this article aims to focus on the use of one of the modest drugs that treat advanced breast cancer, which is Alpelisib. Alpelisib is a type of kinase inhibitor that is prescribed alongside fulvestrant to treat advanced or metastatic breast cancer. This treatment is specifically indicated for individuals with hormone receptor-positive, human epidermal growth factor receptor 2 (HER2)-negative breast cancer that has one or more PIK3CA mutations. The most common adverse reactions include (Hyperglycaemia, nausea, rash, diarrhea, fatigue, decreased appetite, vomiting, weight decreased, stomatitis, aPTT prolonged, and alopecia). The recommended dose of alpelisib is 300 mg taken orally, once daily, with food. Currently, there is no data available regarding the potential risks associated with the use of alpelisib in pregnant women. However, animal studies conducted on pregnant rats and rabbits have shown that oral administration of alpelisib during a specific stage of fetal development, known as organogenesis, can lead to negative developmental outcomes, such as post-implantation loss, reduced fetal weights, and an increased likelihood of fetal malformations. There is currently no information available on whether alpelisib is present in human breast milk, how it may affect milk production, or what impact it may have on a breastfed infant. Due to the possibility of serious negative effects on the nursing child, it is recommended that lactating women avoid breastfeeding while undergoing treatment with alpelisib and for at least one week following the final dose. Animal studies have suggested that alpelisib may have negative effects on the fertility of individuals who are capable of reproduction, both males and females. There is no available evidence on the safety or effectiveness of alpelisib in pediatric patients. It is unclear what impact severe renal impairment (characterized by a creatinine clearance rate of less than 30 mL/min) may have on the way alpelisib is processed by the body (its pharmacokinetics). This oral presentation provides detailed information about various aspects of a particular topic, including its mechanism of action, recommended uses, situations where it should not be used, how it affects the body, how it is metabolized, appropriate dosages, and any potential adverse effects.

Audience Take Away Notes

- The audience will be able to know the pharmacokinetics and pharmacodynamics of Alpelisib and Fulvestrant
- The audience will be able to highlight the drug interaction and contraindications of Alpelisib and Fulvestrant

- The audience will be able to list the most common side effect of Alpelisib and Fulvestrant
- The audience will be able to recognize the overdose sign and symptoms related to Alpelisib and Fulvestrant
- The audience will be able to know the warnings and precautions related to Alpelisib and Fulvestrant

Biography

Dr. Mahmoud Nouh studied Clinical Pharmacy and graduated as a pharmacist with honors in 2020 from Umm Al-Qura University, Makkah, Saudi Arabia. He then works as a pharmacist in the Pharmaceutical Care Department at General Network for Healthcare Providers Hospital, Jeddah, Saudi Arabia. He then joined Ibn Sina National College for Medical Studies in Medicine Department to complete his double major in medical college. Currently, He is involved in various research projects related to breast cancer, respectively.

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Sox7 is a candidate tumor suppressor gene in multiple myeloma

Background: As a common and incurable cancer originating from plasma cells, multiple myeloma (MM) represents 10% of hematological cancers. Most cancer-related genes involved in MM development are currently unknown. Deletions in chromosome 8p23.1, harboring multiple candidate tumor suppressor genes, are observed in MM tumor samples. SOX7, a transcription factor located in 8p23.1, was reported to be silenced in many cancer types; and its ectopic expression inhibited growth of different tumor types. Herein, we investigated whether SOX7 acts as a tumor supressor in MM or not.

Methods: In MM cell lines, SOX7 copy number, transcript, and protein expression analyses were investigated with qPCR, qRT-PCR, and western blot, respectively. SOX7 promoter methylation analysis of MM cell lines was performed through bisulfite sequencing. The SOX7 coding sequence was cloned into the PMIG retrovirus for transduction into two MM cell lines. GFP competition, cell cycle, and apoptosis assays were performed using these two MM cell lines with ectopic SOX7 expression.

Results: We observed monoallelic deletion of SOX7 in two out of five (40%) MM cell lines. Of significance, SOX7 promoter was hypermethylated in four out of five (80%) of the MM cell lines. Consistent with these observations, SOX7 was under-expressed in most MM cell lines. Ectopic expression of SOX7 with transduction in two SOX7 non-expressing MM cell lines resulted in progressive decrease in the percentage of GFP+ cells, suggesting that SOX7 expression exerts negative selection pressure on these MM cell lines. Cell cycle analysis showed increase of cells in G1 phase and decrease in S phase in two MM cell lines with no endogenous SOX7 expression. Similarly, apoptotic cell percentage was higher in SOX7 non- expressing MM cell lines with ectopic SOX7

Conclusions: Based on our results, SOX7 is genetically and epigenetically inactivated in multiple myeloma. *In vitro* functional experiments showed that SOX7 may be acting as a tumor suppressor gene in multiple myeloma by inducing G1 cell cycle arrest and/or apoptosis. Altogether, these results suggest that loss of expression of SOX7 tumor suppressor may be contributing to development of multiple myeloma.

Audience Take Away Notes

- The audience will gain insight into the role of SOX7 tumor suppressor on multiple myeloma development
- Other faculty may shape their future research on SOX7 and/or multiple myeloma based on the results presented in this talk
- The audience may apply the techniques mentioned in this speech in their profession

Biography

Assoc. Prof. Dr. Can Küçük completed his Ph.D. studies on oncology and cancer biology at The University of Nebraska Medical Center (UNMC). He performed post-doctoral studies at UNMC and City of Hope Medical Center. Dr. Küçük has publications in high impact journals such as Nature Communications, Blood, or PNAS. He earned prestigious international awards from the American Society of Hematology and the National Natural Science Foundation of China. Dr. Küçük's research focuses on genomic, transcriptomic, and epigenomic aberrations causing lymphoid cancers to identify biomarkers that can improve diagnosis or prognostication of lymphoid cancers and to discover more effective therapeutic targets.

Sandhya Ojha, Punam Kumari Mandal*
Department of Community Health Nursing, Tribhuvan University, Institute of Medicine, Biratnagar Nursing Campus, Nepal

Close the cancer care gap, aware the elderly: A study to assess awareness of health effects of tobacco use among elderly, Nepal

Introduction: The tobacco epidemic is one of the biggest public health threats the world has ever faced, killing more than 8 million people a year, including around 1.2 million deaths from exposure to second-hand smoke. Every eight seconds someone, somewhere in the world, dies as a result of tobacco use. It is reported that by the year 2030, the death toll is likely to exceed eight million people a year. Tobacco use is widely recognized as the single most preventable cause of premature death due to these NCDs and the leading preventable cause of cancer and cancer deaths. The economic costs of tobacco use are substantial and include significant health care costs for treating the diseases as well as the lost human capital that results from tobacco-attributable morbidity and mortality. The aim of this study was to assess awareness of the health effects of tobacco use among the elderly.

Methodology: A cross-sectional study was conducted among the elderly and the sample was selected through a purposive sampling technique in 2022. Data was collected through face-to-face interviews using a semi-structured interview schedule. Data entry and analysis were done in SPSS version 16.0. Descriptive analysis was used and prevalence was estimated. In inferential analysis, a test of significance like chi-square was used to find the association between awareness and selected demographic variables. A p-value of <0.05 was considered to indicate statistical significance at a level of significance of 5% with a confidence interval (CI) of 95%.

Results: The findings of the study revealed that the mean age of the respondents was 70.17 ± 8.25 and the prevalence of tobacco use was 44.8%. Almost all of the respondents (99.4%) had knowledge about the serious illness caused by tobacco use whereas 98.7% knew lung and throat cancer caused by tobacco smoking. likewise, more than half of the respondents (57.1%) had good awareness regarding tobacco use.

Conclusion: The study concluded that less than half of respondents reported tobacco use in any form. Likewise, more than half of the participants have a good level of awareness regarding tobacco use. Furthermore, there is a significant association between the level of awareness with age group and advertisement. The study findings will be helpful in recommending local authorities for information dissemination about the health effects of tobacco use to prevent disease conditions. Besides this screening programs should be done by health workers to detect early signs of cancer. In order to bring awareness about the effects of tobacco use among the elderly, health education programs should be organized in the community.

Keywords: Awareness, Tobacco use, Health effects, Elderly.

Audience Take Away Notes

- The audience will be able to learn about the level of awareness of the health effects of tobacco use among the elderly and they will use these findings in designing the public health program focusing elderly for countries facing similar problems
- It will help to sensitize the audience that tobacco use among the elderly is an issue that needs to be considered and awareness programs should be organized to close the cancer care gap

- The findings will help them to initiate screening programs for the recognition of early warning signs of cancer. Additionally, it helps future researchers to conduct the study in different settings

Biography

Ms. Punam Kumari Mandal is an assistant professor at Tribhuvan University Institute of Medicine. She graduated MPH from the Institute of Medicine, Nepal in 2013. She is involved in teaching research, and biostatistics, and supervised research work for more than 8 years. She is a member of the research committee at this institute. She has been involved in quantitative, qualitative as well as collaborative research. She has been awarded the gold medal by the President of Nepal. She received Nepal Bidhya Bhushan and Nepal Chatra Bidhya Padak Awards in 2009. She has published more than 10 articles in NepJol journals.

Sneha Patnaik*, Kuan-Han Lin, Yin-Hwa Shih

Department of Healthcare Administration, Asia University, Taichung 41354, Taiwan

Prevalence and laboratory findings of cachexia in head and neck cancer patients

Background: Head and neck cancer patients experience cachexia that causes weight loss, poor response to treatment, and decreased overall survival rates. The study aims to determine the prevalence of cachexia in head and neck cancer patients and analyze their laboratory parameters by systematic review and meta-analysis.

Methods: A comprehensive literature search was carried out using five electronic databases (PubMed, Embase, CINAHL, ProQuest, and Cochrane Library). We included original articles published in English. Two reviewers evaluated the abstracts and titles before the full-text review. The quality of the study was appraised by the Joanna Briggs Institute's critical appraisal tool. The prevalence data and the laboratory findings were extracted to Review Manager and conducted the meta-analysis.

Results: After the removal of duplicates, a total of 1406 articles were retrieved from electronic databases. After title, abstract, and full-text screening, 14 articles were included in the review. The pooled prevalence of cachexia in the included studies is 54.97% (48.14-61.81). Decreased plasma haemoglobin (MD = -0.60, 95% CI = -1.11, -0.08) decreased serum albumin (MD = -0.22, CI = -0.37, -0.08) and increased serum C-reactive protein (MD = 3.43, CI = -14.79, 21.65) were found in patients with cachexia of head and neck cancer.

Conclusion: Except for body weight loss, the change of the plasma haemoglobin, serum albumin could be an indicator and the treatment effect of cachexia in head and neck cancer.

Keywords: Head and neck cancer, cachexia, prevalence, haemoglobin, albumin, C-reactive protein.

Audience Take Away Notes

- This study reports the pooled prevalence rate and laboratory data findings of cachexia in head and neck cancer. The outcome provided medical professionals with the indicators for cachexia either for prevention or treatment effect

Biography

Sneha Patnaik studied at KIIT University, India and graduated as MBBS (Bachelor of Medicine Bachelor of Surgery in 2014. She then did MPH from University of Sheffield, U.K in 2017 and joined as a Faculty in Dept. of Public Health in KIIT School of Public Health where she served 4 years. Currently, she is doing her PhD in Asia University, Taiwan with an MoE scholarship from the Taiwan Government.

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The role of high index of suspicion in early detection of pancoast tumours: A diagnostic dilemma and hidden menace

Introduction: Pancoast tumours, a subtype of non-small cell lung carcinoma (NSCLC) was an obscure entity until Henry Pancoast first described them in the 20th century. These tumours primarily involve the apical chest wall and thoracic inlet structures, and their diagnosis requires the presence of lung apices origin and associated neurological dysfunction. Pancoast tumours commonly manifest as shoulder girdle and arm pain, often along the C8, T1, and T2 dermatomes, accompanied by weakness or atrophy of hand muscles and Horner's syndrome, a constellation of symptoms collectively known as Pancoast Syndrome. Despite comprising 5% of all NSCLC cases, Pancoast tumours are frequently diagnosed at advanced stages, exhibiting a propensity for metastasis and leading to a generally unfavourable prognosis.

This abstract presents a clinical case study that provides an accurate depiction of the signs and symptoms associated with Pancoast tumours, underscoring the crucial importance of early recognition through maintaining a high index of suspicion, even in routine clinical scenarios.

Case Study: A 56-year-old male patient presented at the emergency department with a sudden onset of left leg weakness and mild sensory loss. Upon further inquiry, the patient reported clumsiness in the left hand. Initially, the initial suspicion was that of a stroke; however, a CT scan of the head yielded unremarkable results. Subsequent examination revealed classical Horner's syndrome, characterized by miosis, partial ptosis, and hemifacial anhidrosis, which raised suspicion of an apical pathology. Although the chest radiograph appeared normal, a CT scan of the chest was subsequently conducted, revealing the presence of a left-sided Pancoast tumour. A subsequent MRI demonstrated cord compression at T2-T3 levels, along with tumour infiltration into the left brachial plexus. CT-guided biopsy conclusively confirmed the diagnosis of lung adenocarcinoma with distant spread.

Results: During the course of treatment, the patient underwent five cycles of radiotherapy, which led to the development of neuropathic pain in the left shoulder and upper chest. Encouragingly, there was clinical improvement noted in the left-sided Horner's syndrome. While the clumsiness in the left hand did not deteriorate further, some residual weakness persisted. Regrettably, the patient experienced permanent loss of bilateral leg function as a result of spinal cord metastasis.

Conclusion: In conclusion, this case study highlights the critical importance of conducting a thorough medical history, including a detailed assessment of smoking history, and performing meticulous examinations when evaluating patients who present with seemingly unrelated symptoms. It is crucial not to disregard the possibility of Pancoast tumours, even in the presence of normal findings in physical and imaging studies. The delay in diagnosis and management often arises from the failure to consider Pancoast tumours in the differential diagnosis. Recognizing these tumours early and initiating timely treatment have a significant impact on patient survival rates in cases of lung adenocarcinoma. Therefore, maintaining a high level of suspicion for Pancoast tumours is vital to optimize prognosis and enhance patient outcomes.

Audience Take Away Notes

- This case demonstrates the significance of a thorough medical history and examination while assessing patients with seemingly unrelated symptoms. The presence of "normal" physical and imaging studies should provoke the clinician to consider an alternative possibility
- Pancoast tumors are challenging to be picked up on plain radiographs. It is always a good idea to seek radiologist input when there is a high suspicion of an apical tumor
- The inability to include a Pancoast tumour in the differential diagnosis, especially with a significant smoking history, most commonly causes a delay in diagnosis and management
- Early recognition is the key since the prognosis is directly dependent on timely treatment, which can affect patients' disability with lung adenocarcinoma and survival rate

Biography

Jeevitha is currently working as a doctor at Ashford and St Peter's NHS trust in Surrey. She aspires to be a surgeon, educator and a researcher. She aims to reach a level of expertise where she can effectively share her knowledge with others, enabling them to navigate the complexities of the medical field. By blending her theoretical knowledge with practical experience, counselling aptitude, teaching commitments and unwavering passion for surgery, she is committed to delivering the highest standards of care to patients. Additionally, she possesses a strong passion for clinical governance and holds a PG certification in medical education.

Arya Ashok

Department of Medical Affairs, Tempus Labs, Chicago, Illinois, United States of America

Cancer biomarkers and latest technologies for early cancer detection

This presentation will focus on the advancements made in cancer biomarkers. Cancer biomarkers can be important in identifying targeted treatment options and personalizing the cancer treatment based on the genomic profile of the tumor. Studies have been published that provide evidence on the benefit of personalized cancer care rather than a more generic route. This talk will also focus on exciting new technologies that can help detect cancer at an early stage, monitor cancer recurrence after surgery and also discuss the benefits of liquid biopsy as a tool to monitor the cancer and study the tumor evolution from a genomics standpoint. Liquid biopsy is a non-invasive tool and just a simple blood draw is sufficient to get genomic results. While tissue is always the gold standard for detecting biomarkers, when tissue is not available or at a site that is hard to biopsy, liquid biopsy is an easy blood draw that can provide some important genomic insights. However, there are limitations to the liquid biopsy assays and this will be discussed in the presentation as well.

Audience Take Away Notes

- This presentation will highlight important cancer biomarkers in two populations: 1. Patients with advanced disease and 2. Technology to identify cancer at a very early stage
- Someone we know or care about could have cancer, a friend, a colleague, a relative. This talk will empower you with the latest knowledge on cancer biomarkers including DNA and RNA sequencing
- Cancer can be cured if detected at early stages but in most cases, cancer is detected at a late stage. There are new technologies that are a simple blood test and will allow early detection of cancer. I will discuss this in detail

Biography

Dr Arya Ashok has a Masters and PhD in Biological Sciences. She has 12+ years of experience in the field of precision oncology. Her expertise includes genetics, molecular biology, immunology and next generation sequencing. She has 20+ publications, abstracts with close to 100 citations and also has experience launching clinical trials including face-to-face meetings with FDA. Currently, Dr Ashok works at Tempus Labs which is one of the leading next generation providers in USA.

Suresh Chava[1], Suresh Bugide[1], Parmanand Malvi[1], Romi Gupta[1,2]*

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Co-targeting of specific epigenetic regulators in combination with CDC7 potently inhibit melanoma growth

Melanoma is a highly aggressive skin cancer that frequently metastasizes, but current therapies only benefit some patients. Here, we demonstrate that the serine/threonine kinase Cell Division Cycle 7 (CDC7) is overexpressed in melanoma, and patients with higher expression have shorter survival. Transcription factor ELK1 regulates CDC7 expression, and CDC7 inhibition promotes cell cycle arrest, senescence, and apoptosis, leading to inhibition of melanoma tumor growth and metastasis. Our chemical genetics screen with epigenetic inhibitors revealed stronger melanoma tumor growth inhibition when XL413 is combined with the EZH2 inhibitor GSK343 or BRPF1/2/3 inhibitor OF1. Mechanistically, XL413 with GSK343 or OF1 synergistically altered the expression of tumor-suppressive genes, leading to higher apoptosis than the single agent alone. Collectively, these results identify CDC7 as a driver of melanoma tumor growth and metastasis that can be targeted alone or in combination with EZH2 or BRPF1/2/3 inhibitors.

Audience Take Away Notes

- Melanoma Treatment remains a challenge because of development of drug resistance, which allows disease to metastasize. Identification and targeting of CDC7 alone or in combination with EZH2 or BRPF1/2/3 inhibitors can be employed as a new therapeutic intervention for melanoma patients
- New epigenetic regulator along with kinase is involved in melanoma growth and progression
- Clinically relevant targetable mechanism for melanoma therapy

Biography

Dr. Gupta did her BS and MS in India. She then joined Prof. Knud Nierhaus group at Max Planck Institute for Molecular Genetics, Berlin, Germany for her PhD and obtained her degree in the area of ribosome biology and protein translation. After that she worked at Yale University as postdoc, where she extensively performed studies to identify new regulator of cancer growth and progression. Many of her studies are published in journals like eLife, PNAS, Cell Reports, Oncogene, iScience, Cancer Research etc. Currently she is an Assistant Professor in the UAB and Associate scientist at O'Neal Comprehensive Cancer Center at UAB. Her lab Our works on identifying new molecules and pathways and studying their role in tumor initiation and progression. Her long-term goal is to not only identify new molecules and signaling pathways that regulate the disease but also develop more effective and durable cancer therapies.

Amy Symington MSc[1,3*], Jaime Slavin RD[2], Meaghan Kavanagh[3], Tamara Saslove[2], Christine Hotz[1]

[1]Centre for Hospitality & Culinary Arts, George Brown College, Toronto, ON, Canada
[2]Independent Registered Dietitians, Toronto, ON, Canada
[3]Department of Nutritional Sciences, University of Toronto, Toronto, ON, Canada

Application of dietary & eating guidance to support cancer treatment side effects

Patients receiving treatment for cancer by any modality are likely to experience side effects that affect their ability or desire to consume a variety of nutritious foods. This is a critical issue as maintaining good nutrition and health during treatment may affect patient tolerance of treatments, treatment progression, and quality of life during treatment, which may ultimately affect patient outcomes. However, cancer care teams may not be equipped to provide appropriate, practical, non-medical advice or resources to help alleviate these side effects, or able to provide dietitian consultations on a routine basis. Common eating and diet related side effects include olfactory changes like loss of taste, odour sensitivities, or bitter or metallic tastes in the mouth; gastrointestinal ailments like dry mouth, sore mouth or throat, nausea, vomiting, diarrhea, bloating, and constipation; physical effects such as fatigue and decreased immunity; and neurological effects such as brain fog or mood disturbances. Poor appetite may occur as a direct side effect or secondary to these various side effects and, if not managed well, may lead to malnutrition. The appropriate selection of foods for taste, texture, energy and nutritional content or density, food preparation methods, and eating behaviours (e.g., meal size, frequency) can assist patients in managing these side effects and supporting adequate nutrition throughout treatment. We have compiled practical eating and dietary advice derived from published research articles and various cancer research authorities for addressing treatment related side effects. We have also compiled tailored recipes and culinary tips to help patients and caregivers directly implement this advice. These resources may be helpful tools for use by cancer treatment programs that may lack knowledge and training to assist patients with this critical component of health and treatment success. This information may also be delivered through a supper club format and live cooking demonstrations with instruction designed for patients and caregivers. These resources will be used as a teaching tool for physicians in a new course designed by the University of Toronto and George Brown College (Toronto, Canada).

Audience Take Away Notes

- This presentation will highlight the importance and practical role of diet and eating guidance for patients experiencing common side effects of cancer treatments
- The problem being addressed is that many oncology physicians do not have training or knowledge in eating, dietary, or culinary-related advice for cancer patients, and not all cancer treatment programs are able to provide access to dietitians or culinary professionals to assist them in the management of treatment symptoms
- The audience will come away with practical eating advice for patients, knowledge of available resources for this advice, and examples of specific recipes and food types that may help patients and their care givers manage common side effects at home
- Information in this presentation will help cancer treatment professionals to understand and access very practical advice for patients. The resources developed for this purpose may be accessed and used directly or can be used as a template to develop similar, culturally, and geographically appropriate resources for patients

Biography

Amy Symington, MSc, is a nutrition professor, research associate and plant-based chef at George Brown College, and is a healthy plant-based food advocate. She is also the culinary nutrition program coordinator at Gilda's Club Greater Toronto, a not-for-profit that provides social support for those who have been touched by cancer, and Culinary Specialist for the Food Forward program of the Humane Society International/Canada promoting the adoption of plant-based menus by commercial kitchens. Chef Symington is also a doctoral candidate in the Department of Nutritional Sciences at the University of Toronto, Canada.

Anyou Wang

Feinstone Center for Genomic Research, University of Memphis, Memphis, TN 38152, United States of America

Noncoding RNAs and deep learning neural network discriminate multi-cancer types

Detecting cancers at the population level can dramatically reduce mortality rates, but there is no way to systematically detect all cancers today. Here we develop an accurate cancer detection system to discriminate multiple types of cancers by integrating an artificial intelligence deep learning neural network and universal noncoding RNA biomarkers selected from massive data. Our system can accurately detect cancer vs. healthy objects with 96.3% of AUC of ROC (Area under Curve of a Receiver Operating Characteristic curve), and it surprisingly reaches 78.77% of AUC when validated by real-world raw data from a completely independent data set. Even validating with raw exosome data from blood, our system can reach 72% of AUC. Moreover, our system significantly outperforms conventional machine learning models, such as random forest. Intriguingly, with no more than six biomarkers, our approach can easily discriminate any individual cancer type vs. normal with 99% to 100% AUC. This detection system provides a promising practical framework for automatic cancer screening at the population level.

Biography

Anyou Wang received his PhD from University of California, Riverside. His research interest is in computational biology, artificial intelligence and big data. Dr. Wang develops computational algorithms to capture the big pictures from massive data and to understand the fundamental principles of biology (combai.org). He computed petabyte level data and revealed the distinctive functional regime of endogenous lncRNAs in dark regions of human genome and unearthed that noncoding RNAs endogenously rule the cancerous regulatory realm while proteins govern the normal.

John M. York, PharmD, MBA[1,2*], Mahesh Kandula, M.Tech, MBA[3,4], Subbu Apparsundaram, PhD[3,4]

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[3]Cellix Biosciences, Inc, Newark, New Jersey, United States of America
[4]Cellix Bio Private Limited, Hyderabad, India

Preclinical evaluation of CLX-155, a novel prodrug o 5-FU for the treatment of cancer

Introduction: Capecitabine is an oral prodrug of antimetabolite 5-fluorouracil (5-FU), used to treat metastatic breast and colorectal cancer. Variability in therapeutic levels of 5-FU and severe neurologic (e.g., hand-foot syndrome), gastrointestinal, and hepatic toxicities have limited this agent's use. The development of CLX-155 arose from the need to address these clinical issues. This novel oral 5-FU prodrug differs from capecitabine. Unlike capecitabine, CLX-155 design involves 5'-DFCR, an intermediate in generating 5-FU, independent of liver metabolism. CLX-155 can offer the potential for greater potency for anticancer effects at a lower dose, with less variability. Hence, this preclinical work addresses the research question – what is the relative efficacy of CLX-155 in a human colon cancer xenograft model in nude mice?

Methods: This study involved Foxn1 athymic nude female mice (7-8 weeks) implanted subcutaneously with HCT116 human colon cancer (5 million cells/site) in the dorsal right flank. The study randomized these animals into different treatment groups (N=10 per group) as vehicle control, CLX-155 (125, 250, and 500 mg/kg/day), or capecitabine (1000 mg/kg/day). Animals underwent treatment for three consecutive weeks (5 days/week; 2 days off). Investigators recorded tumor volumes thrice weekly and observed clinical signs of toxicity, mortality, and body weights. Analysis of tumor growth inhibition (TGI) percentage involved calculation based on the tumor volume on a given day compared to Day 1.

Results: CLX-155 demonstrated statistically significant, dose-dependent tumor growth inhibition at all doses compared to vehicle control ($p<0.05$). The tumor growth inhibition on Day 15 was 57.8, 70.4, and 90.6% at 125, 250, and 500 mg/kg/day, respectively. Two animals experienced complete tumor regressions at 500 mg/kg/day. Capecitabine at 1000 mg/kg/day showed 87.7% TGI, but no complete responders were in the capecitabine group. There were no clinical signs of toxicity at any CLX-155 dose levels, whereas 2 of 10 animals in the capecitabine group showed hunched back and scaly skin. All CLX-155 treated animals survived, whereas 2 of 10 capecitabine animals expired at 1000 mg/kg/day.

Conclusion: CLX-155 is expected to provide an additional therapeutic option with the potential for better efficacy and improved tolerability due to the potential reduction in dosing and non-requirement of liver carboxylesterase for producing the active drug. Such characteristics offer new opportunities for clinical investigators to engage in Phase 1 and 2a studies for managing colorectal cancer. Moreover, the preclinical and clinical efficacy and tolerability of CLX-155 in breast cancer treatment are to be investigated.

Audience Take Away Notes

- The audience will be able to use such data to evaluate the candidacy of CLX-155 for phase 1/2 study investigation in solid tumors, such as breast and colon cancer, where capecitabine might be used
- This research will add knowledge about antimetabolites, capecitabine, and their role in solid tumors. Such can be taught in both pharmacology and therapeutics class settings
- This research offers an avenue to examine drug design strategies to improve the therapeutic window of newer antimetabolite candidates for use in solid tumors

- This effort creates awareness about the antimetabolite and capecitabine alternatives. Such will help clinicians reevaluate the current and future roles of antimetabolites and capecitabine based on the therapeutic benefit and toxicity profiles, which might limit current use, particularly that of capecitabine

Biography

John M. York, PharmD, MBA is faculty at the University of California, San Diego, as co-director of the translational medicine capstone project and lead instructor at the Institute for the Global Entrepreneur. He also serves as faculty at Rutgers's Ernest Mario School of Pharmacy, overseeing scholarly projects within the industry post-doc program. York's oncology experience spans over 30 years, including efforts at Amgen, HDI, and Akita Biomedical. His activities include being Associate Editor for Cancer Control from 2008-2018 and collaborating with the H Lee Moffitt Cancer Center in Tampa, FL. He has over 45 peer review articles on clinical, translations, pharmacoeconomic, entrepreneurship, and management topics. His oncology consulting includes work with Celgene, Daiichi Sankyo, Genentech, HRA Pharma, Novartis, Pharmion, and Pfizer.

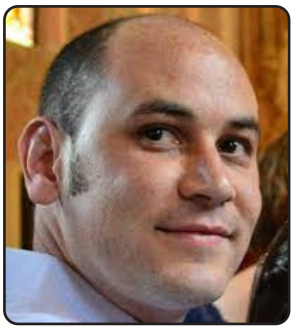
Brandon Lucke-Wold MD, PhD, MCTS
University of Florida, United States

Advanced breast cancer metastasized in the brain: Treatment standards and innovations

Breast cancer continues to be a difficult disease to treat due to high rates of metastasis. Metastasis to the brain presents a unique and often overlooked challenge. In this focused review, we discuss the epidemiology of breast cancer and which types frequently metastasize to the brain. Novel treatment approaches are highlighted with supporting scientific evidence. The role of the blood-brain barrier and how it may become altered with metastasis is addressed. We then highlight new innovations for Her2-positive and triple-negative breast cancer. Finally, recent directions for luminal breast cancer are discussed. This review serves to enhance understanding of pathophysiology, spark continued innovation, and provide a user-friendly resource through tables and easy-to-process figures.

Biography

Brandon Lucke-Wold was born and raised in Colorado Springs, CO. He graduated magna cum laude with a BS in Neuroscience and distinction in honors from Baylor University. He completed his MD/PhD, Master's in Clinical and Translational Research, and the Global Health Track at West Virginia University School of Medicine. His research focus was on traumatic brain injury, neurosurgical simulation, and stroke. At West Virginia University, he also served as a health coach for the Diabetes Prevention and Management program in Morgantown and Charleston, WV, which significantly improved health outcomes for participants. In addition to his research and public health projects, he is a co-founder of the biotechnology company Wright-Wold Scientific, the pharmaceutical company CTE cure, and was a science advocate on Capitol Hill through the Washington Fellow's program. He has also served as president of the WVU chapters for the American Association of Pharmaceutical Scientists, Neurosurgery Interest group, and Erlenmeyer Initiative Entrepreneur group. In addition, he has served as vice president for the graduate student neuroscience interest group, Nu Rho Psi Honor Society, and medical students for global health. He was an active member of the Gold Humanism Honor Society and Alpha Omega Alpha Honor Society. He is currently a member of the UF House Staff Council, Positive Culture Committee, Quality Improvement Committee, Board of Directors Alachua County Medical Society, and Accreditation Requirements Review Committee. He is married to Noelle Lucke-Wold and has two children. As a family, they enjoy running with their dogs, rock climbing, and traveling. In his spare time, Brandon frequently runs half marathons and 10ks together with is wife. Brandon also enjoys reading, playing piano, discussing philosophy, and playing chess. He is currently a Pgy5 neurosurgery resident at University of Florida with pursuing endovascular enfolded training and was awarded the Dempsey Cerebrovascular Research Fellowship.



Daniel L. Adams[1]*, Cha-Mei Tang[2]

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Liquid blood biopsy test for cancer screening, prognosis and drug prediction

Creatv Bio provides innovative liquid biopsy blood tests for cancer screening, cancer prognosis and predicting cancer treatment response. We were the first to discover and research the clinical applications of tumor macrophage fusion cells found in the blood of cancer patients. These tumor macrophage cells are commonly ignored, mis-identified or simply not properly collected by other liquid biopsy techniques. Our LifeTracDx blood tests have been shown to collect all tumor macrophage fusion cells, as well as circulating tumor cells, from the blood of cancer patients for numerous drug developments and clinical applications. LifeTracDx blood tests have been shown to be applicable to all solid tumors, all stages of cancer and all therapy types.

LifeTracDx liquid biopsies are applicable for cancer screening and providing patient prognosis in parallel from a single blood sampling, including: (1) Screening for single cancer or pan cancers with high sensitivity even in Stage I, (2) Detecting minimal residual disease at end of therapy treatment, and (3) Detecting recurrence earlier than standard imaging.

LifeTracDx liquid biopsies may also provide a spectrum of clinical utilities for patients with cancer, including: (1) Providing companion/complementary diagnostics (simpler and cheaper than tissue biopsies), (2) Predicting treatment response in 30 days after a new therapy induction, (3) Providing prognosis at any time during treatment, (4) Monitoring patient responses to treatments over time, and (5) Providing unfragmented tumor DNA for sequencing. Clinical applications of these highly sensitive and specific tests might reduce morbidity and significantly improve patient outcomes.

Our LifeTracDx blood tests will not only benefit patients, but also suited for cancer drug development and clinical trials.

Audience Take Away Notes

- The audience will learn about a novel type of circulating cancer biomarker in the blood of patients that had not been previously studied prior to our discovery
- The audience will learn about our ongoing research studies regarding this cancer biomarker in various cancer subtypes and their biological origins in cancer patients
- The audience will learn about the various clinical utilizations established using this novel cancer marker and its clinical applicability in specific cancer screening trials and in predicting drug responses

Biography

Daniel Adams is Director of Clinical R&D at Creatv Bio, Division of Creatv MicroTech, Inc., and currently a visiting scientist at Rutgers University, New Jersey. He received BS in Biochemistry-Molecular Biology from University of California Santa Barbara. Before joining Creatv MicroTech in 2007, he was Associate Research Scientist at SBH Sciences, a pre-clinical CRO for cancer drug development. He was first to publish the clinical applications of tumor macrophage fusion cells. He has been lead author on 20 peer-reviewed publications, 7 co-authorships, and written 4 book chapters on bio-detection of rare analytes and liquid biopsies for cancer screening and cancer diagnostics.

Jean Bao, MD[1*], Taylor N. Anderson, MD[1], Carlos Ayala, MD[1], Irene Wapnir, MD[1], Mardi R. Karin, MD[1]

Division of General Surgery, Department of Surgery, Stanford University, Palo Alto CA, United States of America

Contemporary surgical treatment algorithm for male breast cancer including nipple-sparing mastectomy and areolar-sparing mastectomy

Background: The vast majority of male breast cancer (MBC) is treated with total mastectomy without reconstruction, while female breast cancer patients are commonly offered less deforming surgical procedures such as partial mastectomy, nipple-sparing mastectomy (NSM) and areolar-sparing mastectomy (ASM). Many men report dissatisfaction with their appearance after total mastectomy due to chest wall concavity and loss of nipple. This underscores the importance of cosmesis-oriented procedures in this population. In this presentation, we will present our series of early stage MBC treated with NSM or ASM, including indications for NSM or ASM, surgical techniques, and clinical and oncologic outcomes. We will review the literature on various surgical operations for MBC, discuss their impact on quality of life, and introduce a modern surgical treatment algorithm for MBC.

Methods: A retrospective review was performed of all MBC patients treated with NSM or ASM during 2015-2021. Patient and tumor characteristics, mastectomy type, surgical margins, complications and recurrences were analyzed.

Results: Four MBC patients, with median age of 58 years (range 47-64), had node-negative invasive cancers ranging from 0.2-2.8 cm in size. Tumor-to-nipple distance ranged from 0.2-1.8 cm on imaging. NSM was performed in two patients; one of the two ASMs was converted from planned NSM due to a positive sub-nipple biopsy. All had negative surgical margins. There were no postoperative complications or recurrences at median follow-up 41 months (range 18-82 months). All verbally reported postoperative cosmetic satisfaction. A new surgical treatment algorithm for MBC was created, incorporating tumor-to-breast ratio, cancer distance to the nipple and results of sub-nipple biopsy into surgical decision making.

Conclusions: Surgical decision making for MBC should be based on similar criteria as for females. Our study established the feasibility of NSM and ASM in MBC with excellent oncologic outcomes and cosmesis. These contemporary surgical techniques should be considered as alternatives to total mastectomy for select MBC patients.

Audience Take Away Notes

- Understand the oncologic outcomes of less aggressive surgical treatment (ie breast conserving surgery) compared to total mastectomy for MBC
- Understand the impact of total mastectomy with removal of the nipple-areolar complex on quality of life for MBC patients
- Discuss indications for NSM and ASM for select MBC patients

Biography

Dr. Jean Bao graduated summa cum laude in Bioengineering from Rice University (Houston, Texas, USA) in 2006. She obtained her MD degree from the University of Chicago in 2010. After general surgery residency at the University of Texas Southwestern Medical Center (Dallas, TX) from 2010-2016, she completed a one-year fellowship in Breast Surgical Oncology at Cedars-Sinai Medical Center (Los Angeles, CA) under the mentorship of Dr. Armando Giuliano. She practiced breast surgery as an Assistant Professor of Surgery at the University of Chicago (2016-2020) and has been a Clinical Assistant Professor of Surgery at Stanford University since 2020.

Sweta Sharma Saha[1*], Lucy Gentles[2], Rachel Howarth[1], Stuart Rundle[3], Angela Ralte[4], Rachel O'Donnell[1,5], Nicola J Curtin[1], Yvette Drew[1,6]

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Understanding tumour heterogeneity following neoadjuvant chemotherapy in ovarian cancer

Neoadjuvant chemotherapy (NACT) has been known to induce genomic and transcriptomic alterations within tumours. Here, using transcriptome profiling along with histological analysis of pre and post NACT tumours, we show temporal and disease site-specific differences (omentum, ovary and other sites) in high grade serous ovarian cancer (HGSOC) and their association with progression free and overall survival. We observed a significant reduction in genome instability signature score following NACT irrespective of disease site, which correlated with homologous recombination repair (HRR) function measured by RAD51 foci formation. However, increase in immuno-oncology and inflammation signatures was only observed in the omentum and not in the ovary post-NACT. DNA damage immune response (DDIR) assay along with HRR function associated with CD8+ T-cell infiltration. Our study provides transcriptomic and phenotypic data highlighting temporal heterogeneity in HGSOCs and site-specific evolution of tumours in response to NACT which holds the potential to guide therapy selection.

Audience Take Away Notes

- Understanding cancer heterogeneity in high grade serous ovarian cancers
- Transcriptomic and molecular analysis approaches to characterize tumours

Biography

Dr. Sweta Sharma Saha did her PhD in cancer biology from the National Institute of Biomedical Genomics, India. Following that Dr. Sharma Saha joined Department of Hematology/Oncology at the University of Chicago as a post-doctoral scholar working on DNA damage response inhibition and immune modulation in prostate cancers. After that Dr Sharma Saha joined as a Research Associate at the Newcastle University Centre for Cancer studying DNA damage response regulation in ovarian cancers. Dr Sharma Saha has published 15 articles in her scientific career so far.

BOOK OF ABSTRACTS

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17-19 AUGUST

DAY 03

KEYNOTE FORUM

6TH EDITION OF

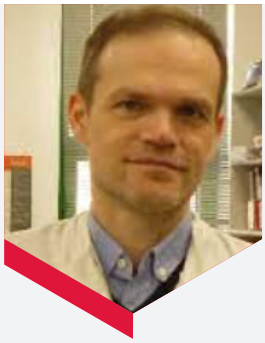
INTERNATIONAL CANCER CONFERENCE

Novel mechanisms of the abscission checkpoint in human cells

Chromatin bridges are strings of chromatin connecting the anaphase poles or daughter nuclei and have been linked to tumourigenesis. In response to chromatin bridges in cytokinesis, eukaryotic 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, which are associated with genomic instability and cancer predisposition. In mammalian cells, this abscission delay is called the "abscission checkpoint" and is dependent on optimal localization and catalytic activity of the Chromosomal Passenger Complex (CPC)-catalytic subunit Aurora B kinase at the midbody. There, Aurora B phosphorylates ESCRT-III components to prevent formation of ESCRT-III filaments at the abscission site, to delay the final cut. However, how chromatin bridges are detected by the abscission checkpoint and the molecular mechanisms by which the abscission-delay signal is transduced to the CPC have not been previously identified. Here, we present previously undescribed mechanisms that sense chromatin bridges in human cells. We also show that the molecular origin of chromatin bridges is important for the activation of the abscission checkpoint and describe a novel signaling pathway that transfers the abscission-delay signal to the CPC at the midbody to prevent chromatin bridge breakage. These results identify mechanisms that preserve genome integrity in human cells, by activating the abscission checkpoint to prevent chromatin breakage in cytokinesis.

Audience Take Away Notes

- Chromatin bridges
- Abscission checkpoint signaling
- Mechanisms that maintain genome integrity in cytokinesis



Eleni Petsalaki, Athina Kyriazi, Sofia Balafouti, George Zachos*

Department of Biology, University of Crete, Vassilika Vouton, Heraklion 70013, Greece

Biography

George Zachos completed his PhD at the University of Crete and received postdoctoral training at the Beatson Institute for Cancer Research, Glasgow, U.K. investigating DNA damage checkpoint mechanisms. In 2008, he moved to the Department of Biology of the University of Crete in Greece as an Assistant Professor in Cell Biology, became Associate Professor in 2015 and continues to hold this position today. Discoveries from the Zachos lab have identified novel mechanisms of the mitotic spindle and abscission checkpoints during cell division in human cells. He has published 39 papers in leading scientific journals and has received >2000 citations.

Novel mechanisms of the abscission checkpoint in human cells

Coronavirus disease 2019 (COVID-19), a respiratory illness caused by beta coronavirus SARS-CoV-2, has broad clinical presentations ranging from asymptomatic to fatal outcomes. Due to their immunocompromised status, cancer patients are at an increased risk for severe SARS-CoV-2 infection. Given the diverse clinical presentations, we developed a model to explain the pathogenesis of severe SARS-CoV-2 and its immunological interplay with cancer. Since SARS-CoV-2 causes multi-organ dysfunction through IL-6-mediated inflammation and hypoxia, while malignancy causes apoptosis through hypoxia-induced cellular metabolic alterations, we propose a mechanism by which both conditions resulted in IL-6 upregulation causing increased cytokine release and systemic injury with clinical trial support. Infection with severe SARS-CoV-2 in patients with malignancy results in increased IL-6 production leading to enhanced systemic injury as compared to either alone. Molecularly, this hypoxic interplay results in increased apoptosis, mitochondrial dysfunction and oxidative phosphorylation dysregulation. This results in the propagation of the inflammatory response through increased cytokine production resulting in systemic organ injury especially to the lungs, which can manifest as bronchoconstriction and pulmonary edema. Due to the limited therapeutic interventions for severe COVID-19 especially in the immunocompromised such as cancer patients, we propose the use of a stem cell therapy that has yielded promising efficacy in COVID-19 patients with severe disease. This therapy, mesenchymal stem cells, possesses regenerative, antiviral and immunomodulatory properties that can inhibit viral replication, while dampening the cytokine response with resulting systemic inflammation and injury. Clinically, it has demonstrated a 91% overall survival and 100% survival in patients younger than 85 years old within a month after treatment with results holding steady for 6 months. Thus, cancer patients can quickly contain SARS-CoV-2 with limited interruptions to their treatment schedule.

Audience Take Away Notes

- This presentation will feature our cohesive models for the mechanism of action for SARS-CoV-2 followed by the first proposed mechanism to explain the interplay between cancer & severe SARS-CoV-2
- This presentation will explain and highlight the efficacy, supported by clinical trial results, of the most effective therapies against severe SARS-CoV-2
- Novel therapies with multi-disease potential including mesenchymal stem cells and exosomes will be discussed.
- This presentation will drive great investment into cellular and immunotherapy research to further finetune their efficacy to treat more disease

Yan Leyfman[1*], Muskan Joshi[2], Gayathri Pramil Menon[2], William B. Wilkerson[3], Chandler Park[4], Pushpa Sharma[5]

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[5]Uniformed Services University of the Health Sciences, Bethesda, MD, United States of America

Biography

Dr. Yan Leyfman has been recognized as one of the top international researchers in oncology by ASH and ASCO. During the COVID-19 pandemic, he was recruited as the Director of the Immunology Division of the Global COVID-19 Taskforce, which produced one of the first mechanisms for SARS-CoV-2 and COVI-Flu along with therapeutic interventions for both. In 2021, Dr. Leyfman presented the first mechanism to explain the interplay between cancer and COVID-19 at the 2021 ASCO Annual Meeting. His work has been published as the

cover article in the journal, Shock, and in the textbook, Insights on a Post-COVID World. Over the past two years, Dr. Leyfman was recognized as the 2020 iCHEM Emerging International Scholar in Immunology & Immunotherapy, 2020 New York State & City Manhattan Hero, 2021 & 2022 Lymphoma, Leukemia & Myeloma Congress Hero in Healthcare, by Memorial Sloan Kettering Cancer Center for research excellence, and amongst the top 6 early career scientists for 2022 in recognition of his contributions to cell and immunotherapy with a 2023 Phacilitate Advanced Therapies Award.

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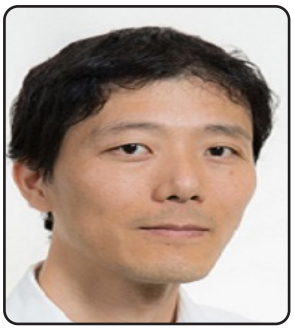

Kazuho Saiga
Aichi Medical University, Japan

PBRM1 immunohistochemical expression profile correlates with histomorphological features and endothelial expression of tumor vasculature for clear cell renal cell carcinoma

Polybromo-1 (PBRM1) is an epigenetic-related gene, and the loss of PBRM1 protein in tumor cells has been expected as a potential biomarker for Clear Cell Renal Cell Carcinoma (ccRCC). Whereas there is little knowledge about how PBRM1 immunohistochemical expression correlates with the histomorphological features of ccRCC and the endothelial expression of tumor vasculature. The present study evaluates the association of architectural patterns with the PBRM1 expression of cancer cells using a cohort of 425 patients with no metastatic ccRCC. Furthermore, we separately assessed the PBRM1 expression of the endothelial cells and evaluated the correlation between the expression of cancer cells and endothelial cells. PBRM1 loss in cancer cells was observed in 148 (34.8%) patients. In the correlation analysis between architectural patterns and PBRM1 expression, macrocyst/microcystic, tubular/acinar, and compact/small nested were positively correlated with PBRM1 expression, whereas alveolar/large nested, thick trabecular/insular, papillary/pseudopapillary, solid sheets, and sarcomatoid/rhabdoid were negatively correlated with PBRM1 expression. PBRM1 expression in vascular endothelial cells correlated with the expression of cancer cells (correlation coefficient = 0.834, $p < 0.001$). PBRM1 loss in both cancer and endothelial cells was associated with a lower recurrence-free survival rate ($p < 0.001$). Our PBRM1 expression profile indicated that PBRM1 expression in both cancer and endothelial cells may be regulated in an orchestrated manner.

Biography

Kazuho Saiga MD, PhD from Japan, graduate from Aichi medical University. After graduated the university, working in Department of Pathology at Saitama Medical Center. He learned general pathology. He then entered graduate school in the Department of Pathology at Kansai Medical University, where he studied renal pathology. Now he is working at Department of Diagnostic Pathology of Ijinkai Takeda General Hospital.

Takako Ooshio*, Masahiro Sonoshita

Division of Biomedical Oncology, Institute for Genetic Medicine/Hokkaido University, Sapporo, Hokkaido, Japan

Identifying RFK and MEK as potential therapeutic targets for pancreatic cancer through a *Drosophila* screening

Pancreatic ductal adenocarcinoma (PDAC) has a dismal patient prognosis with overall survival of merely ~10% mainly due to the lack of effective diagnostic and therapeutic measures. In PDAC, there have been identified high rates of alterations in the oncogene KRAS and the tumor suppressor genes TP53, CDKN2A, and SMAD4. Moreover, the patients with mutations in all these four genes display the worst prognosis among all the PDAC patients. To develop a novel therapy for PDAC, we have recently generated the first animal model, '4-hit' Drosophila, that mimics the four gene alterations. Most of these flies died before reaching adulthood due to the abnormal proliferation of the transformed cells. To determine signaling pathways causing the phenotypes, we performed comprehensive genetic screening for all the kinases in 4-hit flies. Consequently, we revealed that suppression of riboflavin (RF) kinase (RFK) or MEK inhibited their malignant traits. RFK is a rate-limiting enzyme in RF pathway which activates flavoproteins and regulates metabolic pathways, antioxidant activity, and so on with their roles unknown in PDAC pathogenesis. Furthermore, we conducted chemical testing and elucidated that a combination of roseoflavin (RoF), an inhibitor of RF pathway, and the MEK inhibitor drug trametinib significantly rescued the lethality of the 4-hit flies. Additionally, the combination also markedly suppressed the expansion of human PDAC xenograft in mice. These results indicate that the combination of RF pathway inhibitor RoF and MEK inhibitor drug Tr is a novel therapeutic candidate for PDAC.

Audience Take Away Notes

- The inhibition of RFK and MEK is a novel therapeutic candidate for PDAC
- The 4-hit Drosophila is valuable for developing novel therapeutics for PDAC

Biography

Dr. Ooshio studied cell biology at Osaka University, Japan, and received her PhD degree in 2008. After five-year post-doctoral fellowship researching stem cells at Kanazawa University, Japan, she became an Assistant Professor in the Department of Pathology at Asahikawa Medical University, Japan. Since 2019 she has been working on cancer research using Drosophila in her current position.

Qiang Wen[1*], Feng Shao, Li Yang, Feng Cheng, Huarong Tang, Wen Gao, Yingli Zhang, Xi Chen, Chenyan Fang, Tao Zhu, Jianqing Zhu, Zhuyan Shao[2]

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Para-aortic lymph node dissection with or without nerve-sparing in gynecological malignancies

Objective: Para-aortic lymph node dissection (PALND) is a widely used treatment that causes many complications. This study is to evaluate the efficacy and safety of nerve-sparing para-aortic lymph node dissection (NSPALND) by comparing it with conventional PALND in gynecological malignancies and to prove whether locating the superior hypogastric plexus (SHP) can help reveal the para-aortic nerves.

Methods: This is a retrospective study of the patients who underwent para-aortic lymphadenectomy from January 2020 to December 2022 at Zhejiang Cancer Hospital. All of them were divided into NSPALND and PALND groups according to whether or not nerve-sparing was performed. The surgical, functional and oncological outcomes were analyzed retrospectively.

Results: There were 43 patients enrolled, of which, 20 patients underwent NSPALND and 23 patients underwent PALND. The para-aortic nerves were successfully revealed by locating the SHP in all 20 cases of NSPALND. The post-operative anal exhaust time in the NSPALND group was significantly shorter than that in the PALND group (2.5 vs. 4 days, P=0.006), and the incidence of bowel obstruction in the NSPALND group was significantly lower than that in the PALND group (10% vs. 39%, P=0.029). There was no difference between the two groups in terms of catheterization duration, urinary retention, dysuria, as well as the number of lymph nodes removed and the para-aortic recurrence rate.

Conclusion: NSPALND can significantly reduce the rate of bowel obstruction and improve post-operative bowel function. Locating the SHP and using it as an anatomical landmark to reveal the para-aortic nerves is feasible. Its exact clinical value needs to be further studied.

Keywords: Lymph Node Dissection, Gynecologic Neoplasms, Nerve-sparing Surgery, Superior Hypogastric Plexus, Treatment Outcome.

Audience Take Away Notes

- This is the first study comparing NSPALND and PALND in gynecologic malignancies
- NSPALND significantly improved post-operative bowel function and reduced the rate of bowel obstruction
- Locating the SHP and using it as an anatomical landmark helps reveal the other para-aortic nerves

Biography

Dr. Qiang Wen graduated from Zhejiang University in 2001 and worked as a gynecologic oncologist from then. He had been a fellow for 7 years and has rich experiences in ovarian cancer, cervical cancer and endometrial cancer, etc. He attended the training program of observership in University of Miami, Miler school of medicine in 2014, participated in series of cilinical trials like SOLO2, NOVA, SUNNY, SENTICOL3. He has published more than 20 research articles. He is now focusing on the nerve-sparing surgeries in gynecologic malignancies.

Song Song[1*], Han Liu[1], Rongshou Zheng[2], Ni Li[1], Wanqing Chen[1], Wenqiang Wei[2], Jiansong Ren[1]

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[2]Office for Cancer Registry, National Cancer Center / National Clinical Research Center for Cancer / Cancer Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing 100021, China

Effectiveness of one-time endoscopy for upper gastrointestinal cancer screening in China: A multicenter population-based prospective cohort study

Objective: Upper gastrointestinal cancer is one of the malignant tumours which seriously threaten human health. However, there are no multicentre studies evaluating the effectiveness of the endoscopic screening for upper gastrointestinal cancer in urban area. We aimed to estimate the effectiveness of one-time endoscopic screening in prevention of upper gastrointestinal cancer in urban population of China.

Methods: From 2014 to 2015, we assessed 192 805 individuals aged 40 to 69 for eligibility to build a prospective cohort study in 7 cities (Jinan, Qingdao, Chongqing, Ningbo, Quzhou, Xuzhou, and Hefei) of 5 provinces in China. Participants were classified as high risk or low risk of upper gastrointestinal cancer using an established risk scoring system that included cigarette smoking, alcohol consumption, history of upper gastrointestinal disease, family history of upper gastrointestinal cancer and so on. Participants who were evaluated to be high risk for upper gastrointestinal cancer were recommended to undergo endoscopic examination and were classified into screened and non-screened groups on the basis of whether or not they had the endoscopy examination. The effectiveness of one-time endoscopic screening was evaluated by a comparison of the screened and non-screened groups in terms of upper gastrointestinal cancer incidence and mortality using Cox proportional hazards model.

Results: 39 986 participants at high risk were enrolled in the study: 10 433 in the screened group and 29 533 in the non-screened group. By the end of December 2020, after a median follow-up of 5.8 years (IQR 5.6–5.9), 211 had an upper gastrointestinal cancer diagnosis, of whom 154 had gastric cancer and 57 had oesophageal cancer. Compared with non-screened group, upper gastrointestinal cancer incidence density was 25% higher (HR = 1.25, 95% CI = 0.93-1.67), and upper gastrointestinal cancer mortality was 55% lower (HR = 0.45, 95% CI = 0.22-0.90) in the screened group. Mortality of gastric cancer and oesophageal cancer decreased by 31% (HR = 0.69, 95% CI = 0.31-1.50) and 88% (HR = 0.12, 95% CI = 0.02-0.87), respectively, in those having endoscopic examination. All-site cancer mortality and all-cause mortality was 38% lower (HR = 0.62, 95% CI = 0.47-0.83) and 36% lower (HR = 0.64, 95% CI = 0.52-0.78), respectively, for participants in the screened group.

Conclusion: Among high risk individuals aged 40 to 69 years in urban population of China, one-time endoscopic screening was associated with a significant decrease in upper gastrointestinal cancer mortality, all-site cancer mortality and all-cause mortality.

Keywords: upper gastrointestinal cancer, screening, endoscopy, effectiveness

Acknowledgments: We thank all the subjects who participated in the study and all the collaborators who contributed to its success.

Audience Take Away Notes

- Our findings show the effectiveness of one-time endoscopic screening in reducing upper gastrointestinal cancer mortality, all-site cancer mortality and all-cause mortality for a population at high risk of upper gastrointestinal cancer
- Previous research to evaluate the effectiveness for upper gastrointestinal cancer screening was mostly small-sample sized, single-center studies or based on rural population. This is the first study to evaluate the effectiveness of a one-time endoscopic screening in urban China in a multicenter cohort study
- The results indicate the feasibility of implementation of an endoscopic screening program for upper gastrointestinal cancer in urban China
- Our results point to the promise of one-time endoscopic examination in other developing countries with a heavy burden of upper gastrointestinal cancer and limited medical sources

Biography

Song Song, obtained Bachelor Degree from Nanjing Medical University in 2021. She, as a master student, is studying in the Office of Cancer Screening in Cancer Hospital, Chinese Academy of Medical Sciences. Her research interest: Cancer epidemiology and early-detection, effectiveness evaluation of cancer screening programs.

Krishna Misra*, Unnati Soni, Pritish Vardwaj
Indian Institute of Information Technology, Allahabad, India

Targeted drug delivery of anticancer drugs using platelet membrane from patient cells as nanocarriers

Nano medicines are promising strategies for anticancer therapy; however, camouflaging Nano medicines with cell based carrier i.e. Platelet membrane would significantly prolong its retention time in the bloodstream, enhance the targeting ability and reduce the off-target effects. Anticancer nanoparticles wrapped inside patient's own platelet membrane shall act as mimics which contain the complete set of surface receptors, antigens and proteins naturally present on platelet membranes This technique takes advantage of the unique natural properties of human platelet membranes, which have a natural preference to bind to certain tissues and organisms in the body, this targeting ability, which red blood cell membranes do not have, makes platelet membranes extremely useful for targeted drug delivery. Platelets have great prospective as drug delivery systems, proficient of causing extraordinary changes in pharmacokinetics, pharmacodynamics and immunogenicity. These were found ensuring better stability of the particles, better escape from reticuloendothelial system, biocompatible and non-toxic, hence not inducing immunological response. These particles evade immune system attack. The natural flexibility permits the platelet mimics to distort and travel through narrow capillaries. Attachment of some fluorescent materials based on FRET (Förster resonance energy transfer) makes the process of drug delivery more convenient to monitor. Our present work concerns preparation of human platelet membrane nanoparticles dispersed with anticancer drugs for oral cancer. In addition to using commercially available drugs, we are using herbal bioactive and other small molecules designed through in *silico* studies.

Biography

Prof (Mrs) Krishna Misra, PhD FNASc, FBRSI Ph.D. Delhi University, Department of taught Chemistry, Biochemistry and Biotechnology at University of Allahabad, Allahabad, India and Gen Secretary NASI, India. Honorary professor - Indian Institute of Information Technology Allahabad, also Chief advisor- India Pesticides Ltd., Lucknow and Chairperson of STEMM, DST, New Delhi. Research fields: Organic Chemistry, Biochemistry, Biotechnology, Bioinformatics, Bio-medical Engineering. Supervised 55 Ph.D., 260 publications, 6 books, 25 reviews, dozen book chapters and about 100 conference papers, Indian and US patents, visited Japan, U.K, USA (Invited talks/chair). One of the 50 Indian Women in S&T (i.e., book "Women in STEM" by CII TNTDPC).

Koustav Sarkar[1*], Geetha Shanmugam[1], Sudeshna Rakshit[1], Pradeep R[1], Melvin George[2]

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The role of epigenetic alteration in T helper cells in chronic obstructive pulmonary disease (COPD) and its relationship to non-small cell lung cancer (NSCLC)

Chronic Obstructive Pulmonary Disease (COPD) and Lung cancer are the major reasons for lung disease related mortality worldwide. Chronic inflammation is a key attribute of COPD and a potential driver of lung carcinogenesis. Among various environmental risk factors, cigarette smoke plays a crucial role in the development and progression of COPD and lung cancer. Several epidemiological studies show that COPD patients are at a greater risk of developing lung cancer independently of cigarette smoking which suggests the role of genetic predisposition in the disease development. Uncovering the mechanistic link between these two diseases is hampered due to their heterogeneous nature: each is characterized by several sub- phenotypes of diseases. Our laboratory is mainly focused to study the specific epigenetic mechanism that occur in both COPD and lung cancer. The purpose of the current study is to uncover the link between alterations in inflammatory cytokine levels and disease progression in CD4+T cells of patients suffering from COPD and lung cancer. We also investigated the epigenetic regulation of mitochondrial Transcriptional Factor A (mtTFA) to delineate the role of oxidative stress-mediated inflammation in Lung cancer and COPD. The RT2 Profiler PCR array was used to examine the differential expression pattern of inflammatory genes in $CD4^+$ T helper (Th) cells from COPD, NSCLC, and control subjects. Candidate inflammatory gene loci were selected and the enrichment of transcriptional factor and histone modifiers was analysed using ChIP-qPCR. In comparison to control subjects, a set of genes (e.g., BMP2, CCL2, IL5, VEGFA, etc.) are over-expressed whereas another set of genes (e.g., AIMP1, IFNG, LTA, LTB, TNF, etc.) are under-expressed in both COPD and NSCLC patients. The increased percentage enrichment of inflammation-associated transcription factors including NF- kB, CREB, HIF1, and MYC at the loci of inflammatory genes was revealed by our chromatin immunoprecipitation (ChIP) data. H3K4me3, H3K9me3, H3K14Ac, HDAC1, 2, 3, 6 all showed dysregulated enrichment at the VEGFA gene locus. One of the epigenetic modifications, histone methylation, was found to be abnormal in the mtTFA complex in COPD and NSCLC patients in comparison to controls. Although there is mounting evidence of several links between these disorders, therapeutic options remain inadequate. Our findings contribute to the body of knowledge about therapeutic techniques that use inflammatory cytokines as a prognostic marker and highlight the need for epigenetic therapy for these debilitating lung diseases.

Audience Take Away Notes

- Audience will learn that COPD patients are at a greater risk of developing lung cancer
- Mechanistic link between COPD and lung cancer will help the researcher to develop different types of immunotherapeutic strategies for lung disease
- Our research will help the other researchers to study the specific epigenetic mechanism(s) that occur in two interconnected diseases
- Our findings contribute to the body of knowledge about therapeutic techniques that use inflammatory

- cytokines as a prognostic marker and highlight the need for epigenetic therapy for the debilitating lung diseases
- Our study will definitely improve to find out several links between the lung disorders to develop different immunotherapeutic strategies to combat those diseases

Biography

Dr. Koustav Sarkar has completed his PhD at the age of 28 years from Chittaranjan National Cancer Institute/Jadavpur University, Kolkata, India. Currently, he is the Research Assistant Professor in department of Biotechnology, SRM Institute of Science and Technology, Chennai, India. He presented papers in more than 50 national and international conferences. Dr. Sarkar has been involved in research over the last twenty years (including a Ph.D. and three Post-Docs) and made several important contributions to the development of advanced science and technology. He was involved in understanding the molecular mechanisms of the development of human immune responses in health & disease. Dr. Sarkar has already published 46 high-impact scientific publications in internationally reputed journals. He was also co-author of four book chapters. During PhD, Dr. Sarkar has developed a process for isolating glycoprotein(s) from neem leaf, which has immunomodulatory and cancer preventive functions. One patent (Patent Number: 259434; Grant Date: 12- Mar-2014) has been granted for this invention. He found out that the neem leaf glycoprotein helped to generate carcinoembryonic antigen specific anti-tumor immune responses utilizing macrophage & dendritic cell mediated antigen presentation to T and B cells and the induction of type 1 protective immunity. To study the intricate molecular mechanisms involved in the type 1 protective immunity, Dr. Sarkar moved to USA. Research from his US laboratory was essential in revealing for the first time a novel nuclear function for a well- known cytoskeleton structure associated protein, Wiskott Aldrich Syndrome Protein (WASp) in the transcriptional regulation of T helper cell 1 (Th1)-differentiation through its effect on epigenetic modifications at the T-BET gene-promoter locus. Since that time, Dr. Sarkar has been actively involved in further understanding how different types of epigenetic mechanisms are involved in T helper cells of lung cancer in association with Chronic Obstructive Pulmonary Disease (COPD).

Mohite Utkarsha L

Department of Electrical Engineering, MET's League of Colleges, Bhujbal Knowledge City, Nashik, Maharashtra, India

Extended Kalman filter-oriented robust control of cancer chemotherapy

Amongst the various treatment models, chemotherapy is the most significant and widely practiced to cure cancer. More computerized mathematical models have been introduced for controlling the growth of tumors and also to maintain normal and immune cells in their desired values by adjusting the drug dosages. Because of the inaccuracies of measurement and biological changes, a proper quantity of model parameters is unavailable. Hence it is needed to model a controller in a way that should be robust over the parameter uncertainty and deviations. This paper introduces a new robust controller that manipulates the dosage of the drug and does parameter estimation as well. Further, a Nonlinear Error Function based EKF (NEF-EKF) is introduced to estimate the tumor cells as it is complex to measure at the time of vivo experiments. The performance of the proposed controller is compared over other conventional models, and the outcomes show the proper effect of drug dosage injection on immune, normal, and tumor cells. It is also guaranteed that the proposed controller does a robust performance on parameter uncertainties. Furthermore, it is proved that the proposed improved Kalman Filter has the ability on evaluating the tumor cells with high accuracy.

Audience Take Away Notes

- This research proposes a novel robust controller that manipulates the drug dosage and does the estimation of the parameters by the introduction of a new NEF-EKF
- The performance of the proposed controller is compared over other conventional models and proves the proper effect of injection of the drug on immune, normal, and tumor cells
- The performance of the proposed improved Kalman Filter is proven and it has the capability on estimating immune cells with high accuracy

Biography

Dr. Utkarsha L. Mohite is an Assistant Professor in the Department of Electrical Engineering at MET's League of Colleges Bhujbal Knowledge City. She received her B.E degree in Electrical Engineering from SPPU University, Pune, India, in 2010, and her M.E degree in Electrical Control Systems from SPPU University, Pune, India, in 2015. Her research interests are adaptive, robust, and optimal control and modeling of cancer systems. She has published more than 10 research articles in SCI (E) journals.

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Ketorolac modulates HIF-1α/DDX3/Rac-1/β-catenin axis via a tumor suppressor - Prostate apoptosis respose-4 (Par-4) against RCC

Renal cell carcinoma (RCC) is the most difficult-to-treat form of kidney cancer with a median 5-year survival of 10% under metastatic setting. Although cytoreductive nephrectomy is common, approximately 20–30% of patients will develop recurrent cancer after surgery, which highlights the need for an effective therapy. We therefore have elucidated the role of Ketorolac, a modulator Rho-GTPases against RCC through potentiation of tumor suppressor Par-4. The effect of Ketorolac alone and in combination with Sunitinib on proliferation, apoptosis, cell-cycle progression, migration, tumor inhibition and their related markers were studied. Moreover, Ketorolac's impact on metastasis by influencing Rac-1/HIF-1α/DDX3/β-catenin signalling was studied with respect to its ability to modulate the expression of tumor suppressor Par-4 and this mechanism was confirmed by siRNA knockdown studies. Ketorolac caused significant down regulation of proliferation (Ki-67, Cyclin D1, pRB and DDX3), migration/invasion (Rac-1, Cdc42, and Tiam1), and angiogenesis (HIF-1α and VEGF) markers as studied by gene and protein expression. Importantly, Ketorolac alone and in combination with Sunitinib showed tumor growth inhibition (TGI) of 73% and 86% respectively in xenograft model. This anti-tumor activity was further corroborated by down regulation of Rac-1/ Cdc42/HIF-1α/DDX3/β-catenin signalling. This is the first report which implicates the role of Ketorolac against RCC by acting as a small molecule secretagogue causing upregulation of Par-4 in autocrine and paracrine manner. Consequently, these findings suggest that Par-4 can serve as a valuable therapeutic target and a prognostic marker for the treatment of RCC.

Audience Take Away Notes

- This presentation will aid the audience to understand the path followed to identify drugs which can be repurposed for indications other than the approved ones
- De novo drug development is a lengthy, complex and costly process. Understanding the path to be followed for identification of approved drugs that can be repurposed can help conserve efforts, resources and at the same time shortening the period for approvals
- Our study has led to the identification of a new mechanism of action of Ketorolac as a Par-4 secretagouge. Par-4, which is a pro-apoptotic factor that brings about its action by both autocrine and paracrine manner is down-regulated in several cancers. Therefore, we think that Par-4 can serve as a valuable therapeutic and prognostic marker not only for RCC but for other solid tumors too
- Using this information More randomised clinical trials could be undertaken to prove their efficacy, and thus reduce financial burden on stressed health systems, particularly in poorer economies

Biography

Dr. Jeevan is currently associated with Cipla Ltd, one of the largest pharmaceutical firms in India as Director and Head of the Discovery Biology Division. He received his PhD degree in 2008 from Indian Council of Medical Research and University of Mumbai. Dr. Jeevan has more than 20 years of research experience in the field of Cell biology, Molecular Biology, and Drug Discovery. He has co-authored more than 20 patents, several publication and presentations, Pre-IND and IND packages submitted to the regulatory agencies in India and the US.

Aminul Islam[1,2], Ishtiaque M. Syed[2], S. Manjura Hoque[1*]

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Enhancement of Specific Loss Power (SLP) of MCFO-Chitosan Nanohybrids for cancer thermotherapy by hyperthermia

In this study $Mg_{1-x}Co_xFe_2O_4$ ($0 \le x \le 1$ with $\Delta x = 0.1$) nanoparticles were synthesized by chemical co-precipitation method and annealed at 200oC, 400oC, 600oC, and 800oC in order to study their potential applications for hyperthermia therapy of cancer. The structural properties of the materials were investigated by X-ray diffractometer (XRD), transmission electron microscopy (TEM), and Fourier-transform infrared spectroscopy (FTIR). The observed and calculated lattice parameter, the particle size, the X-ray density, the ionic radius of the tetrahedral and octahedral site, the hopping length of the tetrahedral and octahedral site, the bond length of the octahedral and tetrahedral site, cation-cation distance, and cation-anion distance increases with an increase in Co^{2+} content and annealing temperature. From FTIR spectroscopy we observed that the frequency band of both tetrahedral and octahedral site shifted towards the lower frequency region with an increase in Co^{2+} content and annealing temperature due to the extension of the covalent bond. Force constant decreases with an increase in Co^{2+} content and annealing temperature. The thermal characteristics of the $Mg_{1-x}Co_xFe_2O_4$ nanoparticles were investigated by gravimetric analysis (TG) and differential scanning calorimetry (DSC) measurement. Exothermic peak found near 400oC of the TG curve is a clear indication of good crystallinity which was also confirmed by XRD. The magnetic properties of our investigated sample were measured by the Physical Property Measurement System (PPMS). The saturation magnetization, coercivity, remanent magnetization, and anisotropy constant of the $Mg_{1-x}Co_xFe_2O_4$ nanoparticles increase with an increase in Co^{2+} content and annealing temperature. The hyperthermia properties of Chitosan-coated $Mg_{1-x}Co_xFe_2O_4$ nanoparticles annealed at different temperatures were investigated. Specific loss power (SLP) increases with an increase in Co^{2+} content. Initially, the value of SLP increases with increasing particle size but after a certain value of particle size the value of SLP fall abruptly with an increase in particle size. The peak of the SLP curve found at a comparatively lower value of particle size with an increase in Co^{2+} content. The higher the value of SLP, the more efficient the nanohybrid will be for hyperthermia therapy of cancer.

Audience Take Away Notes

- The audience will learn about a new approach of cancer treatment called hyperthermia, which is a minimally invasive technique
- This technique is applicable for localized or deeply seated tumor
- This is a nanohybrid based therapy. Audience will be able to learn how functionalization and structural adjustment of the nanohybrids enhance the ultimate engineering parameter called specific loss power for successful development of the media for hyperthermia treatment of cancer

Biography

Dr. Engr. Sheikh Manjura Hoque is serving as the Chief Scientific Officer (Grade-2) and the Head, Materials Science Division, Atomic Energy Centre Dhaka where she joined in 2001. Dr. Hoque received her B.Sc Engg.(1994) from Department of Materials and Metallurgical Engineering, Bangladesh University of Engineering and Technology (BUET); M.Sc. Engg.(2000) from Department of Materials Engineering, Indian Institutes of Science (IISc), Bangalore; and Ph.D.(2007) from Department of Physics, Bangladesh University of Engineering and Technology. Dr. Hoque conducted research on

bionanomaterials as MRI Contrast Dye and Hyperthermia under NIH Grant in 2012-2013 as a Postdoctoral Associate in the Department of Diagnostic Radiology, Yale University supervised by Prof. Fahmeed Hyder. She published 150 papers and contributed 130 presentations in conferences. She jointly supervised a total of 64 students and 3 postdocs. Dr. Hoque is the Coordinator of the Dhaka Nanomaterials Science Group, a BAN-02/2 program funded by the International Science Programme, Uppsala University, Sweden. She established laboratories of TEM, MRI, XRD, DSC, FT-IR, DLS etc.. Among the many honors, she is the recipient of BAS-Dr. Innas Ali Gold Medal Award (2013) and Best Paper Award from Materials Transactions, JIM(2000).

Nitin Khunteta, MBBS

MS (General Surgery), MCh, DNB (Surgical Oncology), Associate Professor, Department of Surgical Oncology, Sri Ram Cancer & Super speciality Centre, Mahatma Gandhi Medical College & Hospital, Jaipur, Rajasthan, India

Peroral submandibular & cervical approach for middle third oral cavity cancer radical resection - POSC technique

Cancer of oral cavity is commonest in india. Generally, patients come with locally advanced tongue cancer. The treatment for this cancer is multi-modality. For resectable cancer, multiple surgical approaches are described in the literature. Mandibulotomy for resection of the middle third oral cancer is a well-established technique but it has its attended morbidity. We present our results of a new surgical technique, called the peroral & submandibular cervical (posc) surgical approach where in tongue cancer of middle third is resected without mandibulotomy. The advantages of this technique are that the complications of malunion or non-union of bone are avoided, no chances of osteoradionecrosis or osteomyelitis, no need of periosteal elevation or damage, the malignancy is removed with wide margin with minimal blood loss, there is no scar on face or chin, morbidity of surgery is minimal. Also, the technique is easily reproducible.

Biography

Dr. Nitin Khunteta has many international and national publications in field of Surgical Oncology, including Head and Neck Oncology. He has been guide for thesis programs of many DNB Surgical oncology residents. He presented many papers and posters at National and International conferences. He has done more than 9500 cancer Surgeries & Procedures.

Neema Tiwari[1*], Nidhi Chaturvedi[2]

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Comparison of NLR, LMR, PLR, RDW, and platelet count in hematological malignancies at baseline and at intervals of 2 months in patients undergoing chemotherapy

Blood cancer has become quite common in all age groups, worldwide. The cancer ranges from Acute life-threatening leukemias to indolent chronic leukemias, Lymphoma spillovers causing morbidity and mortality to slow-growing indolent lymphomas. Since blood is present everywhere in the body hence the spread of hematological malignancies is massive. Chemotherapy is expensive and a must to treat blood cancers. However, the ancillary workups like flowcytometry etc in prognosticating and diagnosing blood cancers become quiet cumbersome and heavy on the pocket for an average Indian. Therefore, the authors planned the study aimed at analyzing-

-Trend in NLR,LMR, PLR RDW, Platelet count at baseline

-Trend in NLR,LMR, PLR RDW, Platelet count 2 months post-chemotherapy

-Comparison between 2 parameters for any significant change

The study was carried out on 11 cases as a prospective case-based study of 11 cases where pre and post-values of chemotherapy cases of newly diagnosed blood cancer cases were available with the author. The baseline CBC and post-induction 1 st CBC were used to record the variables under study and latest SPSS software was used to come to a conclusion through the results.

The findings stated that there was a decline in NLR, PLR and Platelet count at follow-up as compared to baseline and an increase in LMR and RDW at follow-up as compared to baseline, however, the difference was significant statistically only for PLR (p=0.028) and near significant (p=0.059) for platelet count.

Hence in view of significant findings seen only in 11 cases a larger cohort may be used to correlate these findings with follow up of such cases.

Audience Take Away Notes

- Trend in NLR, LMR, PLR RDW, Platelet count at baseline
- Trend in NLR, LMR, PLR RDW, Platelet count 2 months post-chemotherapy
- Comparison between 2 parameters for any significant change
- Hence the authors want to highlight the importance of Complete blood counts-a simple toll available at almost all the labs as an important diagnostic tool to assess the progress of patients on chemotherapy in cases of blood cancers

Biography

Neema Tiwari did her MBBS and MD in Pathology in Pathology from Eras Lucknow Medical college and Hospital, She is has worked as Senior Resident, Pathology in department of clinical hematology and hemato-oncology, King George Medical University, India and Post Graduate Institute of Child Health Noida, UP and has 4 years post PG experience. She is currently working as Assistant Professor in Subharti Medical College Meerut. She has done numerous intar-mural and extramural (ICMR,DST)research projects and has many national and international publications in indexed and peer reviewed journals(>40) to her credit. She is a reviewer for 3 journals to of which are pubmed indexed. She has presented papers in IAP,ICC and CAP conferences. She has recently presented a poster on MDS in the ISHBT-EHS TUTORIAL-2018 held in India.

Sonal M. Manohar[1]* and Kalpana S. Joshi[2]

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Exploring the efficacy and mechanism of action of multitarget cyclin-dependent kinase inhibitors against human colorectal carcinoma cells

Colorectal cancer (CRC) is one of the leading causes of cancer death in the world. Deregulation of cyclin- dependent kinases (CDKs) is often observed in CRC making them attractive drug targets. We investigated the efficacy of three multitarget CDK inhibitors viz. riviciclib (also known as P276-00) roscovitine and UCN-01 with varied specificities for CDKs against human CRC cell lines of different genetic background viz. Colo-205, HCT116 and HCT-15. Among the three drugs, UCN-01 was found to be the most potent followed by rivicilcib. Roscovitine was the least potent across these cell lines. Nonetheless, all these drugs exerted cytotoxicity and inhibited clonogenic potential of CRC cells. Cell cycle studies using flow cytometry revealed that these drugs abrogated cell cycle progression and lead to accumulation of cells in the sub G1 phase indicating increased apoptosis. Further, apoptosis was also confirmed by annexin V/PI, Terminal deoxynucleotidyl transferase–mediated nick end labeling (TUNEL) and caspase-3/7 glo assays. Protein expression analysis demonstrated that these drugs downregulated expression of their respective target CDKs and cyclins as well as their activity and also modulated expression of caspases and pro- and anti-apoptotic proteins. Further, riviciclib, roscovitine and UCN-01 were found to inhibit RNA polymerase II carboxy- temrinal domain (CTD) phosphorylation at Ser 2/5 thus inhibiting transcription via downregulation of CDK9/cyclin T1 and CDK7/cyclin H (i.e. transcription-regulatory CDK/cyclin complexes) activity. This lead to rapid downregulation of short-lived anti-apoptotic protein Mcl-1 which is implicated in not only cell survival but also drug resistance of CRC cells. Hypoxia-inducible factor 1 alpha (HIF-1□) overexpression is typically associated with mortality and metastasis of many solid tumors including CRC. Riviciclib and roscovitine specifically inhibited hypoxia-induced HIF-1 transcriptional activity in luciferase reporter assay and also downregulated hypoxia-induced HIF-1□ accumulation in metastatic CRC cell line HCT116. All the three drugs abrogated migration of HCT116 cells and also inhibited vascular endothelial growth factor (VEGF)- stimulated tube formation of human umbilical vein endothelial cells (HUVECs) indicating their anti- angiogenic potential. Moreover, these CDK inhibitors exhibited synergism when used in combination with standard chemotherapeutic drugs against CRC cells. Thus, we hereby provide proof-of-concept for the potential use of multitarget CDK inhibitors as promising therapeutic drugs against CRC.

Audience Take Away Notes

The underlying biology of CRC is complex and heterogeneous. There is an unmet medical need in order to treat CRC patients with surgically incurable disease. Trials with novel approaches and agents that attempt to minimize toxicity and improve efficacy are urgently needed

Given the hyperactivation of one or more redundant CDKs in CRC, it is implicated that multitarget CDK inhibitors are promising for CRC treatment. This study provides a preclinical framework for potential clinical application of such multispectrum CDK inhibitors against CR

Much of the anticancer drug development phase relies on ‘in vitro’ model systems i.e. cancer cell lines since these are less expensive and less time-consuming as compared to animal tumor models. Functional assays and mechanistic studies using these cell lines assess the potential of candidate drugs that alter the target and facilitate the selection of the most promising candidate drug e.g. CDK inhibitors

Biography

Dr. Sonal studied Life Sciences at the University of Mumbai and graduated as MSc in 2006. She then joined R & D of Piramal Life Sciences Ltd., Mumbai, India (a reputed pharmaceutical company) in the Department of Pharmacology as a research scientist. While working, she simultaneously completed her PhD degree in 2015 at the same institution. After three-year postdoctoral fellowship at IIT Bombay, Mumbai, India, she obtained the position of Assistant Professor at NMIMS (Deemed-to-be) University. She has published more than 30 research and review articles in reputed peer- reviewed journals.

Sinerik Ayrapetyan*, Vahe Hrutunyan

UNESCO Chair in Life Sciences, Life Sciences International Postgraduate Center
Yerevan, Armenia

The dysfunction of metabolic driving water efflux from the cells is primary mechanism carcinogenesis

Cancer cells are characterized by overhydrated state and contain more than 90% of water (Kircuita et al., 1973). Cell over hydration serves as one of the diagnostic parameters for carcinogenesis (Damadian 1971). However, the nature of metabolic mechanism, the dysfunction of which causes in cancerous cells has not been elucidated yet as well. The discovery of our laboratory, that the electrogenic Na/K pump –induced net water efflux from the cell is fundamental mechanism controlling semipermeable properties of cell membrane, dysfunction of which in common consequence of cell pathology, including cancer. Two quantum-sensitive families of high affinity (10^{-11}–10^{-10}M and 10^{-9}–10^{-8}M) ouabain sensors in cell membrane have been identified which unlike to low affinity to obtain(>10^{-7}M), having inactivation effect on Na efflux from the cells, they have activated effect on Na efflux from the cells, accompanied by water efflux from the cells. The highest affinity sensors by activation of cGMP-stimulated Ca efflux from the cell stimulating of Na/K pump while and receptors with middle affinity stimulate the cAMP-activated Na/Ca exchange in Reverse(R), mode controlling Na gradient on membrane by pushing out Na and decreasing membrane permeability for these ions. The dysfunction of cGMP-dependent Ca efflux from the cells leading to cAMP-dependent R Na/Ca exchange -induced elevation intracellular Ca is suggesting as a primary mechanism for cell pathology, including cancer and cGMP-stimulated Ca efflux from the cell as effective tool for earlier periods carcinogenesis. Target for tumor therapy.

Audience Take Away Notes

- The audience will be able to learn by lecture and publication materials in Research gate
- This research helps to other faculty to expand their research or teaching
- This provides a practical solution to a problem that could simplify or make a designer's job more efficient
- It will improve the accuracy of a design, or provide new information to assist in a design problem

Other benefits

The SGC as a novel target for cancer therapy

Cell over hydration as earlier diagnostic parameter for carcinogenesis

Biography

Prof. Sinerik Ayrapetyan has received his PhD in Cell Biophysics in the Institute of Physiology of Ukraine Academy of Sciences, Kiev. He is one of pioneers discovered electro genic Na-pump components of membrane potential of neurons. He was discover that quantum sensitive metabolic driving water efflux from the cells which is fundamental mechanism controlling of cell membrane semipermeable properties dysfunction of which is primary mechanism of cell pathology. He published more than 200 research articles in international journals. At present he is coordinator Interregional UNESCO/UNITWIN Network research and postgraduate education in in Life Sciences.

Manya Yu[1], Xing Cui[2]*

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A risk score model based on angiogenesis for survival prediction and immunosubtype screening in multiple myeloma and intervention with traditional Chinese medicine

Little is known about the potency of Angiogenesis-Associated Genes (AAGs) in the regulation of the immune microenvironment of Multiple Myeloma (MM). Traditional Chinese medicine has positive anti-angiogenesis and immunomodulatory effects, especially in the treatment of tumors. Although frequently used in the clinic, the mechanism remains elusive in MM. In this study, we divided MM patients into three angiogenesis subgroups based on 35 AAGs, and these subgroups showed significant differences in clinical outcome, biological functions, immune infiltration, and immune checkpoints. These results suggest that AAGs are critical in screening immune molecular subtypes as well as the response to checkpoint immunotherapy. Therefore, we established an AAG_score model with 11 selected immunity- and prognosis-associated Differentially Expressed Genes (DEGs) to quantify angiogenesis subgroups. There is a significant positive correlation between favorable prognosis-related molecules (LST1, IGHM, PD-L2, CD22, FUCA1, SLC7A7, and ADAM28) and the infiltration of most immune cells, while the relationship between risk-related molecules (NUF2, IFI16, and GEMIN6) and immune cell infiltration was the opposite. We also revealed that the expression of 11 molecules was negatively correlated with that of almost all immune checkpoints, indicating that these key molecules could mediate the formation of individual infiltration patterns of immune cells as well as the response to immune checkpoint blockade. In recent years, an increasing number of Chinese patients have tried Traditional Chinese Medicine (TCM) for complementary interventions following a hematologic malignant disease diagnosis, including MM. Previous studies have shown that TCM can regulate the immune system and angiogenesis. Therefore, we comprehensively analyzed the regulatory effects of Danggui-Sini Decoction (DSD) on MM angiogenesis and the immune microenvironment on the basis of the AAG_score model. UHPLC□MS/MS and network pharmacology were performed to analyze its chemical constituents and targets coincident with the AAG_score model. Five AAG_score genes could be targeted by DSD. Furthermore, the in vitro and in vivo experiments demonstrated that DSD could inhibit angiogenesis promoted by the secretion of U266 cells and reduce tumor burden. Therefore, we concluded that angiogenesis has a great influence on the immune phenotype and that the AAG_score has the potential to help clinicians choose precise immune checkpoint blockade treatments. Future studies defining the mechanisms of monomers or a combination of ingredients in DSD on MM with anti-angiogenesis and immune modulate signatures will be of great value in basic research and transformation.

Audience Take Away Notes

- We constructed the AAG_score model to predict patient prognosis and evaluate the immune microenvironment, and this model presented great robustness and independence
- Angiogenesis has a great influence on the immune phenotype and that the AAG_score has the potential to help clinicians choose precise immune checkpoint blockade treatments

- DSD could inhibit MM angiogenesis and reduce tumor burden, further exploration of the antiangiogenic effect of DSD on immune modulation will provide new insights into how TCM controls tumor growth

Biography

Prof. Cui studied Clinical Discipline of Chinese and Western at Shandong University of Traditional Chinese Medicine and received his PhD degree in 2011 at the same institution. After two years postdoctoral fellowship supervised by Prof. Wang at Shandong University, focus on the development of treatment for lymphoma and multiple myeloma, he obtained the position of an attending doctor. He then joined the research group of Prof. Janz at the Department of Pathology, University of Iowa (America). Now, he runs a department of Oncology and Hematology and has published about 20 research articles in SCI (E) journals.

Aggelos T Margetis, MD
Athens Naval and Veterans Hospital, Greece

Exercise for cancer patients: Supportive intervention or therapeutic perspective?

During the presentation, the role of exercise for oncology patients will be outlined. Within this context, established benefits and recommendations regarding exercise as a supportive treatment will be overviewed, as well as mechanistic insights. Additionally, preliminary evidence from preclinical and clinical studies dictating possible therapeutic usages of exercise will be briefly demonstrated so as to discuss future perspectives.

Audience Take Away Notes

- Can we prescribe exercise for oncology patients? if so, under which circumstances?
- Is exercise feasible and beneficial for cancer patients?
- Can exercise be used along with standard-of-care treatments for therapeutic purposes in the oncology setting?

Biography

Dr. Margetis graduated with honors as MD at National and Kapodistrian University of Athens (NKUA), Greece in 2017. He then joined the Molecular Carcinogenesis Group in the Department of Histology, NKUA, to study how metabolism rewiring contributes to cancer development and examine whether dietary restriction protocols can restrain tumor growth in breast cancer models. In 2020 he started his Internal Medicine residency training in Athens Naval and Veterans Hospital; during 2021-2023 he was certificated in Cancer Survivorship, Clinical Nutrition and Integrative Medicine from GW University Cancer Center, KU Leuven University and University of Minnesota, respectively.

Maria Braoudaki

Department of Clinical, Pharmaceutical and Biological Science, University of Hertfordshire, United Kingdom

Downregulation of mir-21 and coro1c in glioblastoma cells upon treatment with a new carbonyl compound

Glioblastoma multiforme (GBM) is considered the most aggressive brain malignancy associated with poor prognosis. MicroRNAs (miRNAs) are small RNA molecules that regulate gene expression and have been found to play a role in GBM progression. MiRNAs can serve as promising diagnostic and prognostic biomarkers, as well as targets for novel therapies. Additionally, □-amino carbonyl compounds are a class of synthetic molecules with potential anti-cancerous properties that might provide an alternative management strategy for GBM patients. This study sought to investigate the action of a new □-amino carbonyl compound, N-(4-Benzyloxybenzylidene)-2-bromo-4-methylaniline (SHG-8), and its effects upon miRNA regulation within GBM cell models.

In silico analysis was performed to identify the most deregulated miRNAs and their mRNA targets in GBM. Quantitative real-time polymerase chain reaction (RT-qPCR) was carried out to verify the expression profiles of selected miRNAs/mRNA targets. In vitro assays including 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-2H-tetrazolium bromide (MTT), cellular reactive oxygen species (ROS), colony forming, and wound healing were performed to assess the proliferative and migratory properties of U87MG cells prior- and post-exposure to SHG-8. DAPI staining was conducted to examine apoptosis. Small RNA-sequencing (sRNA-seq) was performed to identify the miRNAs that SHG-8 compound activates or supresses. Immunohistochemistry was carried out to evaluate CORO1C protein expression across brain cancer patients' samples. The absorption, distribution, metabolism, excretion, and toxicity (ADMET) properties of SHG-8 were predicted with admetSAR 2.0 using the canonical SMILES with the option 'ADMET properties for drug discovery' selected.

In silico analyses suggested that CORO1C was a direct target of miR-21 and miR-128a. RT-qPCR demonstrated that CORO1C was significantly downregulated within GBM cells upon 24h treatment with SHG-8, whilst sRNA-seq revealed significant downregulation of miR-21 and miR-128a. SHG-8 induced significant inhibition of cell viability at 80μM and 100μM. ROS were released following 30min incubation with 50μ□ of SHG-8. Presence of apoptotic bodies were observed post-treatment with 100μ□ SHG-8 in comparison to dimethyl sulfoxide (DMSO). SHG-8 (40μ□) mediated 94% and 93% open wounding areas at 24h and 48h post-treatment respectively compared to wound openings of DMSO; 1.6% and 0.36%, respectively. In total, 75% of the GBM cases exhibited moderate to high CORO1C expression, similar to 70% of medulloblastoma cases, which sustained strong expression levels. Bioinformatics analysis revealed that SHG-8 demonstrated absorption and distribution features comparable with ibuprofen and was able to cross the blood-brain-barrier.

Synthetic molecules provide a future opportunistic approach for the putative treatment of GBM via miRNA targeting. SHG-8 demonstrated favourable bioavailability and potential to modify the levels of GBM-associated miR-21 and miR-128a. These miRNAs could downregulate CORO1C, subsequently inhibiting tumorigenic processes such as proliferation and migration and alter the malignancy outcome. The high expression levels of CORO1C observed through immunohistochemistry further supported its involvement

in highly metastatic brain tumours and thus offers a potential target for future therapies. SGH-8 has shown promising preliminary in vitro results, and hence will be further investigated in regard to its effects upon miRNA regulation.

Audience Take Away Notes

- We synthesized an in-house beta-amino ketone compound (SHG8) that kills glioblastoma cells
- SHG-8 demonstrated favourable bioavailability and potential to modify the levels of GBM-associated miR-21 and miR-128a
- MiR-21 and miR128 could downregulate CORO1C, subsequently inhibiting proliferation and migration and alter the malignancy outcome
- High expression levels of CORO1C observed through immunohistochemistry indicated its involvement in highly metastatic brain tumours and thus offers a potential target for future therapies
- SHG-8 demonstrated absorption and distribution features comparable with ibuprofen and was able to cross the blood-brain-barrier

Biography

Dr Maria Braoudaki is a Senior Lecturer in Molecular Genetics at the University of Hertfordshire, UK. After graduating in 2004 with a PhD in Molecular Microbiology at Aston University, she worked as a Postdoctoral researcher at the Medical School of the National and Kapodistrian University of Athens in Greece, and at the same time continued her postgraduate studies in Medicine. Dr Braoudaki's expertise lies in microRNA cancer biomarkers. Dr Braoudaki has published work in more than 40 high-impact peer-reviewed journals. In 2016, she was awarded the prestigious L'OREAL-UNESCO Award for Women in Science for her work on pediatric cancer.

Eleni Petsalaki*, Sofia Balafouti, George Zachos
[1]Department of Biology, University of Crete, Heraklion, Greece

A novel mechanism promotes actin patch formation to prevent chromatin bridge breakage in cytokinesis

Chromatin bridges are strands of incompletely segregated DNA connecting the anaphase poles or daughter nuclei. If unresolved, chromatin bridges can break in cytokinesis leading to micronuclei formation and accumulation of DNA damage. To prevent this, human cells form accumulations of polymerized actin (actin patches) at the base of the intercellular canal to stabilize chromatin bridges; however, the molecular mechanisms involved are incompletely understood. In the present study, we identify small GTPases, which control the growth or contraction of filamentous actin fibers, that localize to actin patches and are required for stable chromatin bridges in cytokinesis. Inhibition of these actin regulators reduces actin patch formation and promotes chromatin bridge breakage by confocal microscopy analysis of fixed cells or live-cell fluorescence microscopy. Furthermore, chromatin breakage in cells deficient for the above proteins is not caused by premature abscission, but correlates with reduced actin patches compared with wild-type cells. We also propose that DNA bridges generate tension inside the nucleus which is then transmitted through specific mechanosensitive complexes to the cell cytoskeleton to promote generation of actin patches in the cytoplasm. This study identifies a novel signaling pathway that prevents chromatin bridge breakage by promoting actin patch formation in cytokinesis in human cells. Because chromatin breakage can lead to genomic instability that is associated with cancer formation or progression, understanding how cells stabilize chromatin bridges may help us understand mechanisms of tumorigenesis.

Audience Take Away Notes

- Genomic instability can be caused by chromatin bridge breakage in cytokinesis
- Actin fibers, called actin patches, are formed at the base of the intercellular canal to stabilize chromatin bridges and prevent them from breaking
- Novel signaling pathways preventing chromatin bridge breakage by promoting actin patch formation in cytokinesis

Biography

Dr Eleni Petsalaki completed her PhD in Molecular Biology and Biomedicine at the Department of Biology of the University of Crete in Greece in 2014 and has continued working as a post-doc in Dr George Zachos' lab since. Her main research interest is to understand how mis-regulation of mechanisms of cell division can lead to tumour formation, with the aim to identify potential targets for cancer therapy. She has published 15 papers in leading scientific journals such as the Journal of Cell Biology (5), Nature Communications (1), Journal of Cell Science (2) and others, and her work has received >300 citations.

Emanuele Calabro[1*], Salvatore Magazu[2]

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Amplification of ions flow through membrane channels in cancer cells induced by resonant electromagnetic radiation

Resonance phenomenon consists of the oscillation at high amplitude of a material if it is forced mechanically, acoustically or electrically at some specific frequencies named natural resonance frequencies. In particular, some researchers have hypothesized that biological systems have natural resonant frequencies in the microwaves region so that an applied electromagnetic field (EMF) can interfere with their sophisticated electromagnetic circuits. This phenomenon can be applied to cancer treatment using electromagnetic radiations at some natural resonance frequency of cancer cells. Modern wireless technology uses the transmission of electromagnetic radiation in radiofrequency and microwave regions and produced a huge development in human activities. In this regard, there is a large literature on harmful effects of EMFs on living beings that can be explained assuming that the frequency of electromagnetic radiation impinging on biological systems is close to natural resonance frequencies of such systems. On the other hand, beneficial effects of resonant EMFs could occur by using electromagnetic radiation at natural frequencies of cancer cells in order to damage them. In particular, this technique could be applied in damaged areas that cannot be treated surgically and in order to not damaging nearby healthy cells. Nevertheless, the problem is which frequencies to choose among the many resonance frequencies in order to damage cancer cells. Indeed, human cells are complex systems that have a very large number of natural resonance frequencies. In our opinion, the flux of ions across cellular membranes can be a significant parameter, whose alteration can change cell viability. In fact, the flux of ions across cellular membrane channels have been demonstrated to have a fundamental role in cellular functions of cancer cells as they can regulate cancer's initiation, progression and proliferation. In this regard, the displacement of α-helices in membranes channels of cancer cells caused by resonant frequencies of an EMF should alter the flux of ions across cellular membranes channels, damaging cancer cells. In contrast, normal cells would not be damaged, as their natural resonant frequencies should be different from those of cancer cells. Experimental results have already provided proof of the correctness of this theory. Indeed, previous studies in vitro showed that there is a correlation between proteins α-helix (represented by Amide vibration bands, observed by infrared spectroscopy techniques) and the frequency of applied EMF. In addition, exposure of neuronal-like cells to EMFs showed a direct proportionality between displacement of cell channels α-helix and EMF frequency. The result of these studies leads us to look further into the frequencies in which this effect is amplified, since an alteration of cellular membrane channels should induce a change in ions flux across channels and the resulting damage of unhealthy cells. Indeed, the rotation of α-helices in cellular membrane channels of cancer cells due to torque effect induced by an applied EMF should be amplified if the frequency of the applied EMF is close to a natural frequency of channels α-helix in that type of cells. Indeed, cellular membrane channels are formed by proteins, that is α-helices, so that exposure to EMF should induce the orientation of these α-helices along the direction of the applied EMF, causing the enlargement of the channel and inducing an increasing of ions flow across the channel, such as schematized in Figure 6 (A-B) of [14]. Otherwise, a large amount of studies in literature has shown that even a minimum alteration of ions flux across cells membrane channels should cause significant cells damage.

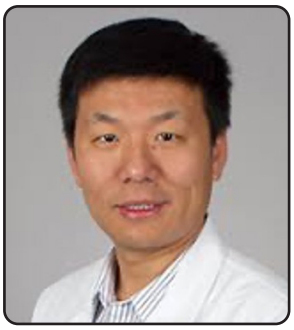
In conclusion, these results suggest to project the application of this methodology to the treatment of tumor cells, irradiating them by electromagnetic radiation at resonant frequencies of their cellular membrane channels α-helix, once those frequencies have been found by spectroscopic techniques. Also, neighboring healthy cells should not be significantly damaged because natural resonant frequencies of channels α-helix in healthy cells should be significantly different from natural resonant frequencies of channels α-helix in cancer cells. This could be a relevant vantage with respect traditional chemotherapy or other radiation therapy techniques.

Audience Take Away Notes

- At the end of this presentation the audience will be able to undertake a project aimed at finding resonant frequencies to damage cancer cells
- The audience will be able to teach the mechanism of ions flow in cellular membrane channels and an application of resonance phenomenon
- In particular, the physicians will learn a new methodology for the treatment of cancer that can be an alternative to the traditional ones

Biography

Emanuele Calabrò is full Professor of Physics and Environmental Physics at the Technological Technical Institute of Messina (Italy). He got the National Scientific Qualification for University Associate Professor in “Applied Physics” and in “Experimental Physics of Matter”. He is a PhD expert collaborator at the Department of Mathematics and Informatics Sciences, Physics Sciences and Earth Sciences, University of Messina, Italy. He is member of the “Interuniversity Consortium of Applied Physical Sciences” (CISFA), Italy. He has published more than 130 refereed papers in ISI journals and he is author of several monographs, book chapters and editor of prestigious special issues.



Hong-Tao Li
Department of Urology, University of Southern California, Norris Comprehensive Cancer Center, Los Angeles, CA, 90033, USA

Combination treatment of DNA Hypomethylating Agents targeting SETD2-defficient kidney cancer

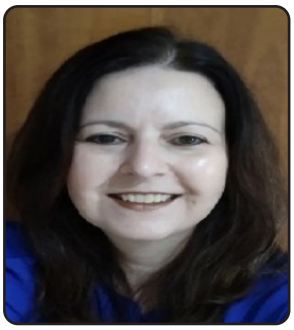
DNA methylation is one of the most well-studied epigenetic regulation factors, which involved in almost all DNA metabolism pathways including gene expression, DNA replication, DNA damage repair, chromatin structure et al. Like genetic alterations, DNA methylation aberrancies commonly happen in many types of cancer and could play a driver-role during tumorigenesis. DNA methylation changes may lead to the silencing of tumor suppressor genes (TSGs) by promoter hypermethylation and activation of oncogenes by gene body hypermethylation. DNA Hypomethylating Agents (HMA), such as 5-Aza-CR and 5-Aza-CdR, can reverse the gain-of-methylation, turn on the expression of TSGs, and decrease oncogenes' expression, which will consequently inhibit tumor cell growth. These two HMAs have been used for the treatment of liquid tumor patients. But the attempts to treat solid tumors using HMAs have failed in clinic trials. We found that SETD2-defficient kidney cancer cells were more sensitive to 5-Aza-CdR. SETD2 is the only known H3K36me3 methyltransferase in mammalian cell and commonly mutated in ccRCC (15%–23%). 5-Aza-CdR induced intronic transposon element (TE) by the enhanced mis-splicing and stimulated viral mimicry. This result suggests that SETD2 is a potential therapeutic target of HMAs. Drug resistance is one of the major reasons for the failure of cancer treatment. In our studies, we revealed the synergetic anti-tumor effects of HMA combined with immunotherapy drug (anti-PD-L1) or PARP1 inhibitor in mouse model. Our results suggest that combination treatments of HMA with other anti-tumor drugs might be an efficient therapeutic strategy.

Audience Take Away Notes

- SETD2 deficiency is a potential therapeutic target of DNA Hypomethylating Agents (HMA).
- HMA could induce viral mimicry and stimulate immune response.
- Combination treatment of different types of anti-tumor drugs might be a solution for the drug resistance.

Biography

Dr. Hong-Tao Li received his PhD degree in 2009 at Chinese Academy of Sciences. He got postdoctoral training in Shanghai Institute of Biochemistry and Cell Biology, China. Then he joined Dr. Gangning Liang's lab in University of Southern California, where his study focus on cancer epigenetics and translational medicine.



Aline do Carmo França-Botelho
Foundation for Children and Adolescents of Araxá, Araxá, Minas Gerais, Brazil

Evidence of the positive impact of breastfeeding on reducing cancer

Breastfeeding is a highly accessible and low-cost public health measure. Several studies have shown the benefits of breastfeeding for the baby and the mother. Aspects such as strengthening the mother-child bond, adequate nutrition, and immunological protection were widely evaluated, and there is no doubt that breastfeeding is highly positive in all these parameters. On the other hand, knowledge of the impact on disease reduction, especially cancer reduction, is still partially understood. There is strong evidence of cancer decrease in women who breastfed their children, which has been reported as one of the protective aspects of breastfeeding. For breast cancer, the most incidence cancer in women worldwide, there are reports of the protective effect of breastfeeding due to the differentiation of mammary cells, reduction in the number of ovulatory cycles, and excretion of estrogens and carcinogens by human milk. Colostrum and human milk have antimicrobial components, which reduce diseases in infants, especially respiratory and gastrointestinal infections. But the health benefits do not end with weaning; there is evidence that human milk can confer long-term benefits such as reduced risk of certain autoimmune diseases, inflammatory bowel diseases, and certain malignancies. Childhood cancer is the leading cause of death in children worldwide, and leukemia is one of the most common cancers. It also suggests possible effects in reducing cases of different types of cancer in children, including leukemia, who were breastfed as recommended. A discussion focused on this theme is proposed to search the understood aspects and stimulate new research for those who are still partially understood.

Audience Take Away Notes

- Elucidation of aspects of the relationship between breastfeeding and cancer
- Contribution to public health campaigns and promotion of breastfeeding
- Encouraging new research and improving information for teaching in the health area

Biography

Dr. Aline is graduate in Biology and Odontology and is master in Immunology and Parasitology (Federal University de Uberlândia - 2004). She completed her PhD in 2009, in Biological Sciences, at the Federal University of Minas Gerais, Belo Horizonte, Brazil. She has almost 20 years of experience in multidisciplinary research and health teaching at several Brazilian Universities. She has active and regular scientific production, with more than 40 published articles.

Parvin Mehdipour

Department of Medical Genetics, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

Novel evolutionary models in cancer: Bridging functional and molecular platforms to cancer therapy

A. Hit evolutionary hypothesis:

Alphabetic code for cancer management, based on the comprehensive strategic network by considering single cell based analysis/pedigree platform/ Micro-,macro/environmental factors which have significant role through the pre- and post-embryonic periods. Cancer translational insight relies on the diverse or harmonic behavior at functional and molecular levels. So, it is crucial to unmask the sequential/ constructive/cascade events at genomics/tumor levels. Detecting D1853N polymorphism of Ataxia telangiectasia mutated gene (ATM) gene is the key target in cancer- and non-cancerous individuals, inherited, or as a de novo alteration, located at 11q22q23 and Involved in DNA repair pathway and cell cycle checkpoint.

Material and methods: Includes molecular diagnosis by PCR/sequencing, protein expression (PE) assay at single cell level, and In sillico analysis.

Introduction: Cell cycle outlines the initiation/progression and therapeutic approaches of neoplasms. An uncontrolled cell proliferation and growth are the key characteristics of neoplasms. Normal checkpoints regulate the machinery of phases through the barriers. So, balancing the oncogenic processes inhibit progression and facilitates the personalized therapy.

Results/discussion: Three-hit includes D1853N polymorphism, as the first predisposing hit, IVS 35-63T□A as second hit deriving from the first somatic evolution before differentiation and third-hit (IVS35-30 A□G) through the tumor development.

Five- hit in breast cancer (BC) includes IVS 36-91 AA>TT, IVS 36-8 T>C, D1853N, IVS 37+47 A>G, IVS 37+60 Del T. IVS 36-8 T>C and D1853N in blood and tumor tissue.Splicing variants occurred at tumor level. Missense D1853N was effective on 2D and 3D structure of ATM protein. PE of ATM also confirmed the functional alterations. Conclusively, five-hit influence the guard of genomic stability, at molecular/cellular/structural levels.

Eight-hit includes D1853N (1st hit), IVS 36-8 T>C as 2nd hit, V1833M as 3rd hit (at pre-differentiation stage), followed by L1888L as the 4th, and somatic variants including IVS 36-46 C>T, L1842L, H1864H, and S1872R, as 5-8th hits. Low PE of ATM was also confirmed, by the diverse expression of cyclin E, CDC25A, P53, and Ki-67.

B. Evolutionary Mosaic phases in cell cycle of BC patients:

Methods: Evolution was traced in interphase to detect the Mosaic Phases (MPs) by Fluorescence In Situ Hybridization (FISH) and PE including immunofluorescence and flow cytometry.

Results: Novel hypothesis reflects the incidence of dual and/or multi-phases, as minor clones in single cells of BC patients. This definition initiated a model, based on the ratio and diverse MPs comprising G1/S, S/G2 and G1/S/G2, and normal phases (G1, S, G2). Significant-harmonic manner was traced between: 1)

signal copy numbers, 2)equivalent PE, dual- and triple- co-expression of the cyclins E/B1, D1/E/B1. The ratio between gain/normal signals led to a good prognosis for chromosome 1, but longer survival related to this ratio in chromosome 3.

Key words: Cell cycle, Mosaic Phases, Evolutionary Hypothesis, Breast Cancer, FISH

Conclusions:

A. Hit-hypothesis provides: Reliable platform for early detection/tracing Predisposing/ Prediction/ Prognostic/Preventive (4xP) packages for clinical management.

B. The cell cycle based panel innovates the CDKs inhibitor-based therapy by considering MPs Model in BC and other cancers.

Audience Take Away Notes

- To believe in bridging Science to Medicine and the patients' right
- To trust the bridging strategy between Science and Medicine
- To apply both global- and single cell based investigations
- To consider Predisposition, Prognosis, Prediction, Prevention (4xP strategy) early detection, and early therapy for the cancer patients and their target relatives through their pedigree
- To consider the hazard of micro and macro-environmental factors from the embryonic to the adult periods for each individual
- They need to feel and respect the Scientific and medical philosophy, cancer evolution, and 4xp strategy
- They need to apply for the Academic-research based centers
- This research offer other faculty to expand their research or teaching
- This provide a practical solution to a problem that could simplify or make a designer's job more efficient
- It will improve the accuracy of a design, or provide new information to assist in a design problem

List all other benefits:

- Reliable translational insight
- Pedigree-based service to the predisposed family to cancer. Translational insight from single cells' biological identities to the cancer clinics
- Considering environmental factors including nutrition
- Considering the appropriate style of life from childhood, all stages of life, pre- pregnancy and through the 9 month of pregnancy
- Highlighting the importance of Macro- and Micro-environmental factors all through the human's life, including pre- and post- pregnancy and improve the link between Science and Oncology

Biography

Professor Dr. Parvin Mehdipour studied at Tehran University, and graduated as M.Sc. in Genetics. She joined Professor S.Walker, Department of Genetics, and She received her PhD degree in University of Liverpool (1982). She had scientific links with DKFZ/Institutes of Human Genetics/and Institute of Medical Genetics in Erlangen-Nuerenberg/Bonn/ Humboldt/berlin/Dresden/Manchester-University. She focused on evolution/hypotheses in different cancers and tracing cancer biomarkers. Her aims included early detection through Circulating Tumor Cells and innovating modeling in cancer. She has published 3 books on Cancer In Farsi with the University publisher, and 5 books on different aspects in Cancer-Genetics by Springer-Nature (2013-2017&2022). Her interests include painting/writing/nutrigenomics.

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POSTERS

6TH EDITION OF

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Mechanisms of the role of FOXM1 in promoting the proliferation of breast cancer stem cells

In clinical practice, it is also common to see tumor recurrence with distant metastasis in breast cancer patients after chemotherapy, which leads to increased mortality of breast cancer patients. Therefore, it is of great theoretical significance and practical clinical application to further investigate the molecular mechanism of enhanced invasive metastatic ability caused by chemotherapy drugs, and to further search for feasible target molecules that can effectively interfere with them to prevent the occurrence of enhanced invasive metastatic properties of tumors during chemotherapy. Real-time fluorescence PCR showed that the expression levels of tumor tissue mesenchymal cell-related markers: VIM, CDH1 and TWIST1 genes were significantly increased in patients who received neoadjuvant chemotherapy compared with breast cancer patients who did not receive neoadjuvant chemotherapy, while the expression of epithelial cell-related marker CDH1 was decreased, and the results indicated that neoadjuvant chemotherapy could inhibit EMT in breast cancer cells. Flow cytometry detected the expression of stem cell marker CD44/CD24 in primary breast cancer cells before and after chemotherapy, and found that the proportion of tumor stem cells was significantly increased in primary tumor cells treated with neoadjuvant chemotherapy. To further clarify the role of chemotherapy on EMT transformation of breast cancer cells, we used qPCR to detect the expression levels of CDH1 and VIM in MCF-7 and MDA-MB-231 cells after treatment with adriamycin, and the results showed that adriamycin treatment significantly promoted EMT in MCF-7 and MDA-MB-231 cells. Moreover, we used trans well assay to detect the alteration of cell migration ability, and we observed that adriamycin treatment significantly promoted the migration ability of MCF-7 and MDA-MB-231 cells after adriamycin treatment. We found a significant increase in the expression level of FOXM1 in breast cancer cells that underwent mesenchymal transformation in response to chemotherapeutic agents. qRT-PCR and WB results showed that the expression of FOXM1 mRNA and protein in MCF and MDA-MB-231 cells was significantly increased. Subsequently, we found by flow cytometry that overexpression of FOXM1 in MCF and MDA-MB-231 cells promoted an increased CD44+/CD24- cell ratio and an increased number of microsomes. CCK8 experiments further showed that the proliferation capacity of MCF and MDA-MB-231 cells overexpressing FOXM1 was significantly increased compared with control normal cells. FOXM1 can affect EMT in breast cancer cells, thereby promoting tumor stem cell proliferation. Inhibition of FOXM1 expression may affect clinical chemotherapeutic efficacy in breast cancer patients.

Audience Take Away Notes

- Neoadjuvant chemotherapy could inhibit EMT in breast cancer cells
- We found a significant increase in the expression level of FOXM1 in breast cancer cells that underwent mesenchymal transformation in response to chemotherapeutic agents
- FOXM1 can affect EMT in breast cancer cells, thereby promoting tumor stem cell proliferation. Inhibition of FOXM1 expression may affect clinical chemotherapeutic efficacy in breast cancer patients

Biography

Dr. Chanchan Gao studied clinical medicine at the Southeast University, China and graduated as Bachelor in 2004. She received her MS degree in 2017 at the same institution. She then joined Department of Oncology, Zhongda Hospital affiliated Southeast University, Nanjing, China. She is a PhD candidate in Oncology at Southeast University. She has published more than 10 research articles in SCI (E) journals.

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T-cell exhaustion prediction algorithm in tumor microenvironment for evaluating prognostic stratification and immunotherapy effect of esophageal cancer

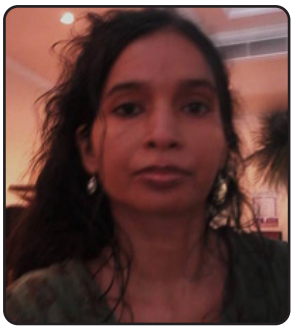
Esophageal cancer(EC) is a common digestive malignancy that ranks sixth in cancer deaths, with a 5-year survival rate of 15-25%. As a result, reliable prognostic biomarkers are required to accurately predict the prognosis of EC. T-cell exhaustion(TEX) is associated with poorer prognosis and immune infiltration in EC. In this study, nine risk genes were finally screened to constitute the prognostic model using LASSO analysis. Patients were divided into two groups based on the expression of the TEX-related genes: high-risk group and low-risk group. The expression of TEX-related genes differed significantly between the two groups. The findings revealed that the risk model developed was highly related to the clinical prognosis and amount of immune cell infiltration in EC patients. It was also significantly correlated with the therapeutic sensitivity of multiple chemotherapeutic agents in EC patients. Subsequently, we successfully constructed drug-resistant cell lines KYSE480/CDDP-R and KYSE180/CDDP-R to verify the correlation between PDCD1 and drug resistance in EC. Then, we examined the mRNA and protein expression levels of PDCD1 in parental and drug-resistant cells using qPCR and WB. It was found that the expression level of PDCD1 was significantly increased in the plasma red of drug-resistant cells. Next, we knocked down PDCD1 in drug-resistant cells and found that the resistance of EC cells to CDDP was significantly reduced. And the proportion of apoptotic cells in cells treated with 6 μM CDDP for 24h was significantly in increase. The TEX-based risk model achieved good prediction results for prognosis prediction in EC patients. And it was also significantly associated with the level of immune cell infiltration and drug therapy sensitivity of EC patients. The high-risk group had lower TIDE scores, indicating that the high-risk group benefits more after receiving immunotherapy. Thus the TEX-based risk model can be used as a novel tumor prognostic biomarker.

Audience Take Away Notes

- The TEX-based risk model can be used as a novel tumor prognostic biomarker for risk stratification and EC patient prognosis prediction
- Our study provides a new biomarker for predicting the prognosis of patients with esophageal cancer and could help physicians to more accurately predict patient prognosis and response to treatment
- Our study found that PDCD1 plays an important role in drug resistance in esophageal cancer, which provides new ideas for the development of new treatment strategies

Biography

Dr. Xiangyu Su studied clinical medicine at the Southeast University, China and graduated as Bachelor in 2005. He received his MS degree in 2008 at the same institution. He then joined Department of Oncology, Zhongda Hospital affiliated Southeast University, Nanjing, China. He is a PhD candidate in Oncology at Southeast University. He has published more than 10 research articles in SCI (E) journals.



Saumya Pandey* **(M.Sc. Biochemistry, Ph.D. Life Science)**
Department of Clinical Research, Indira-IVF Hospital, Udaipur-Lucknow, India (formerly)
University of Texas Medical Branch, Galveston, Texas, USA (formerly)

Autophagy as an immunomodulatory "molecular rheostat" in toll-like receptors/wnt-Frizzled mediated inflammatory "gastrohepatic disease-web" in hepatocellular/colorectal/cholangio-carcinomas: Translational research and public health impact in American Cohorts of Texas, Nebraska and New York States in USA

Objective: Dissecting the cellular/molecular/genetic regulatory biochemical signaling networks: Autophagy/Toll-like Receptors/Wnt-Frizzled in inflammatory "gastrohepatic disease-web" primarily hepatocellular/colorectal/cholangiocarcinomas is essential for diminishing the disproportionate share of morbidities and mortalities in susceptible "at-risk" American cohorts of Texas, Nebraska and New York states in USA. I aimed to investigate the potential immunomodulatory role of complex Autophagy, Toll-like Receptors and Wnt-Frizzled signaling networks/cross-talks in pathobiology and prevention of gastrohepatic cancers viz. hepatocellular/colorectal/cholangiocarcinomas for eventual design of promising patient-friendly cost-effective predictive and prognostic biomarkers and/or pharmacological scaffolds for future immunotherapeutically potent drugs with minimal adverse effects.

Material and Methods: Cell-viability/MTT-proliferation assays were performed under basal and Glucose-deprived metabolic/physiological conditions in TLR-4 agonist Lipopolysaccharide (LPS/endotoxin)-treated cells in culture: HepG2, HT-29, SW480, Caco-2 gastro-hepatic-colorectal carcinoma cells. Autophagy-flux monitoring by assessing relative ratios of LC3-II vs LC3-I in 0-12-24-48 hours' time-course in GD-triggered cells in absence and/or presence of TLR-4 agonist. Cytotoxicity assays, semi-quantitative Reverse Transcriptase-Polymerase Chain Reaction (RT-PCR), immunofluorescence and DAPI/Hoechst-staining were performed followed by protein isolation by RIPA-method, protein estimation by Bradford's method, Western blotting (primary antibodies from Cell Signaling Tech. USA). Immunoblots were subjected to densitometric scans and relative protein expressions/fold-changes determined; Glyceraldehyde-3-Phosphate-Dehydrogenase (GAPDH) and/or beta-actin were used as internal controls/housekeeping proteins.

Results: HepG2, HT-29, SW480, Caco-2 gastro-hepatic-colorectal carcinoma cells were ≥80% viable under basal and Glucose-deprived metabolic/physiological conditions in TLR-4 agonist LPS/endotoxin-stimulated sterile culture in vitro conditions; autophagy-flux was significant in GD-triggered/starvation and/or hypoxic conditions with relatively higher expression levels of LC3-II (16 kDa) vs LC3-I (14 kDa) in 0-12-24-48 hours' time-course. Wnt 1/2/4/5/11 and Fzd 1/2/5 yielded relatively low mRNA expression levels in HepG2/Caco-2 cell-lines. Further, TLR4 agonist LPS-modulated autophagic flux was significant in SW480 adenocarcinoma cells in 48 hours with differential LC3-II vs LC3-I expression levels; interestingly, the protein expression patterns of LC3-II isoform were significantly higher than LC3-1 over 0-12-24-48 hours in HepG2, HT-29, SW480 and Caco-2 cells ($p \geq 0.05$).

Conclusions: My promising translational research study strongly highlights the emerging immunotherapeutic potential of Autophagy/Toll-like Receptors/Wnt-Frizzled cross-talks/signaling-networks hepatocellular

in /colorectal/cholangio-carcinomas for future pharmacogenetics /genomics/metabolomics-based public health-oriented studies in ethnically disparate population-subsets of States of Texas, Nebraska, New York, USA as well as Asia-Pacific region (North+South India). Autophagy markers LC3-II and LC3-I appear promising targets for future development of patient-friendly cost-effective predictive and prognostic biomarkers and/or pharmacological scaffolds for immunotherapeutically potent drugs with minimal adverse effects in susceptible cohorts worldwide.

Acknowledgements: Dr. Pandey acknowledges NIH, USA for previous funded-postdoctoral/doctoral biomedical/translational research experiences at Schools of Medicine, UTMB, Galveston, Texas/Creighton University, Omaha, Nebraska/New York Presbyterian-Weill Cornell Medical College, New York, USA.

Biography

Dr. Pandey possesses brilliant academic credentials with earned Post-Doctorate: Biochemistry-Molecular Biology, Graduate School of Biomedical Sciences, University of Texas Medical Branch (UTMB), Galveston, TX, USA/Visiting Scientist: Urology (Robotic-Prostatectomy), James Buchanan Brady Foundation,-Lefrak Center of Robotic Prostatectomy, Department of Urology, New York Presbyterian-Weill Cornell Medical College, New York, NY, USA/Doctorate: Ph.D. Life Sciences, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, UP, India–Chhatrapati Shahuji Maharaj University, Kanpur, UP, India/Doctoral Research Fellowship: Biomedical Sciences, Creighton University, Omaha, Nebraska, USA/M.Sc. Biochemistry, University of Lucknow, Lucknow, UP, India, and recently worked as Head-Clinical Research, IndiraIVF-Hospital, Udaipur-Lucknow, India with 61 scientific publications in international journals.

Diana Elisa Zamora Avila[1*], Pablo Zapata Benavides[2], Sibilina Cedillo Rosales[3], Aime Jazmin Garza Arredondo[4], Andrea Gonzalez Baez[5], Luis Edgar Rodriguez Tovar[6], Gustavo Moreno Degollado[7,] Gustavo Hernandez Vidal[8]

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From a one health perspective: WT1 as a biomarker and therapeutic target for cancer

The One Health Initiative integrates the interactions between the environment, animal and human health, which opened the doors to work in a multidisciplinary way and address problems from a more comprehensive perspective. The translational or comparative medicine contemplates the study of cancer in animals and humans. We have the opportunity as animal and human health researchers to provide solutions to problems that impact the One Health triad. Comparative oncology looks for a positive impact for animals and humans with cancer. Here, the dogs are a potential model for cancer study for many reasons, for example, both humans and dogs, have a natural development of neoplasms, also they have a similar immune system, and share a high percentage of genetics similarity and a common environment. Actually, there are prognostic molecular markers for tumors in human medicine, which have gradually been integrated into veterinary medicine. One of the genes studied in humans is Wilm's Tumor or WT1 gene, which is related with the development of the genitourinary system during embryonic development. At the beginning it was classified as a tumor suppressor gene, however, it has been found to be overexpressed in leukemia and different solid tumors in humans, being recognized as an oncogene and biomarker associated with malignancy, chemoresistance, and poor prognosis. However, there are few reports on its possible biological role in different animal species. Our researching group has focused on research on melanoma in horses, lung cancer using murine models, and cancer in dogs. Cancer in companion animals is an important disease and there are few records in veterinary medicine and specifically in canines, unlike human medicine. A study was carried out with the objective of analyzing the expression of WT1 in different neoplasias in canines to evaluate its potential as a possible biomarker. We made the histopathological diagnosis of 50 samples of macroscopic lesions with a tumor-like appearance in dogs collected at the Small Species Veterinary Hospital of the Faculty of Veterinary Medicine of Universidad Autonoma de Nuevo Leon. Samples were collected from the mammary gland, vulva, scrotum, spleen, prepuce, head and neck region. 82% of the samples corresponded to neoplastic processes and 18% to pre-neoplastic processes. The neoplasms were classified by their histological behavior as benign or malignant, where

benign neoplasms developed more frequently in 54% and malignant ones in 28%. In the malignant tumors, we find the following histopathological subtypes: anaplastic carcinoma, inverse squamous cell carcinoma and mixed lymphoma. RNA extraction from tumors and complementary DNA synthesis were performed, and WT1 gene expression was analyzed by RT-PCR, finding expression in all the malignant spleen tumors and in a high percentage of breast cancer samples. When we analyzed the correlation of WT1 expression with histopathological diagnosis, we observed higher expression in malignant tumors in regions of the spleen, mammary gland, and neck. Also, within the line of research, we are working on the evaluation of a canine training protocol using cell lines for cancer detection.

Audience Take Away Notes

- The audience will be able to visualize the relevance of addressing the topic of oncology from the point of view of One Health
- The audience, more specifically, will be able to analyze the role of the WT1 gene in cancer in animals and its comparison in humans
- The research projects that will be shown to be being developed, opened the doors to work in a multidisciplinary way and address problems from a more comprehensive perspective
- The different health professionals, can observe the opportunity as human and animal health researchers to provide solutions to problems that impact the One Health triad
- The audience will be able to observe how, through comparative oncology, it is possible to achieve a positive impact for animals and humans with cancer

Biography

Dra. Zamora graduated as Biologist from the Faculty of Biological Sciences of the Autonomous University of Nuevo Leon (UANL). Master's Degree in Sciences with a specialty in Microbiology and a Doctoral Degree in Sciences with a specialty in Microbiology from the Faculty of Biology of the UANL. Member of the National System of Researchers Level I. Research Professor B and Coordinator of the Department of Genetics of the Faculty of Veterinary Medicine and Zootechnics- UANL. She has advised more than 48 undergraduate and postgraduate theses in the area of molecular biology and published more than 20 scientific articles.

Maria Zahra

Department or Division Acute medicine RAEI Organisation/Affiliation, Wigan, Lancashire, UK

Posterior reversible encephalopathy syndrome

A case-based discussion will be done by me about PRES (posterior reversible encephalopathy syndrome). Firstly, I will discuss a case to help the audience to understand the disease with good understanding of how the syndrome presents then I will go through Clinical features, pathophysiology, diagnosis, Findings on neuroimaging & treatment.

Audience Take Away Notes

- After presenting a case, which will include the clinical features, investigations for diagnosis, neuroimaging results, D/D , Causes and appropriate treatment the audience will be able to understand the pathophysiology of one of these rare syndromes with good understanding
- After understanding about PRES, the audience would be able to diagnose this rare syndrome timely which can prevent the lifelong complications of PRES & Also the number of diagnosed case will increase which is very important to provide data for future research
- There is very less data available about this syndrome because of a smaller number of patients diagnosed with this syndrome. By understanding this rare condition and getting more patients with diagnoses more data can be collected, and research can be expanded which will help us to treat this condition timely

Biography

Dr. Maria Zahra studied from university of health sciences in Pakistan and graduated as MBBS in 1999. I have worked as a Junior clinical fellow for 2 years in Pakistan and later on joined NHS as a junior clinical fellow in Royal albert Edward infirmary Wigan.

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Notes

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