magnus Group

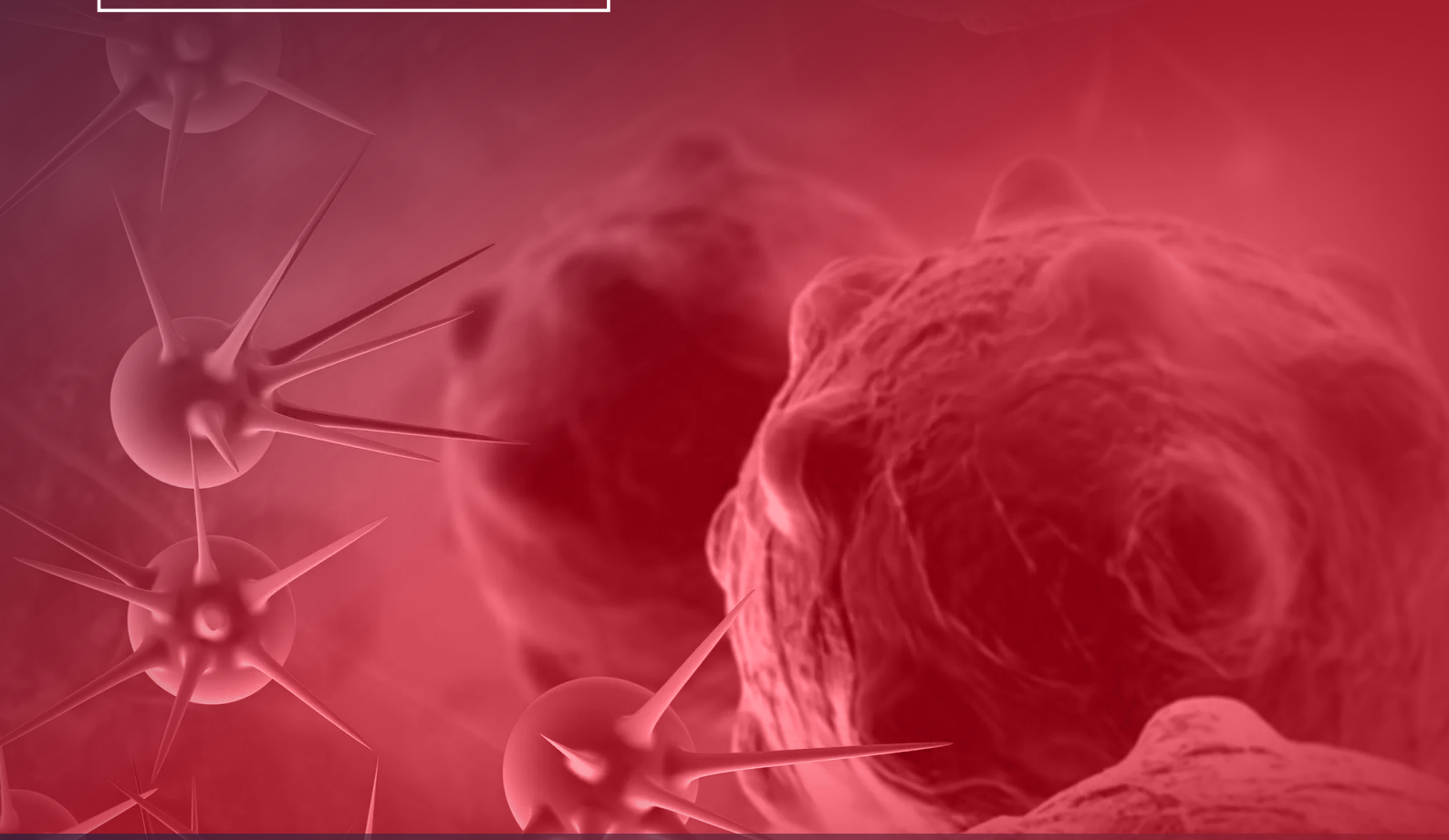
International

CANCER RESEARCH CONFERENCE

MARCH 24-26

2025

SINGAPORE



Venue : Village Hotel Changi 1 Netheravon Rd, Singapore 508502

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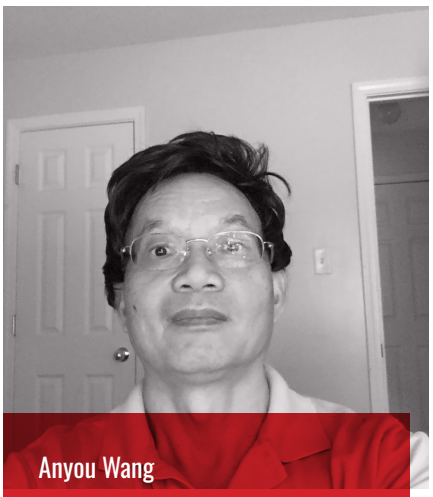
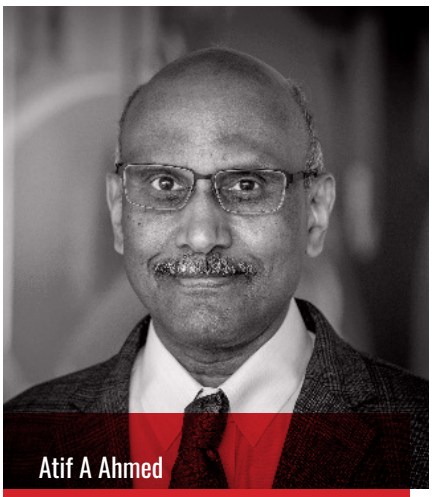


Anyou Wang
DIFIBER LLC, United States



Atif A Ahmed
University of Washington-Seattle Children's Hospital, United State

Bene Ekine-Afolabi
University of East London, United Kingdom

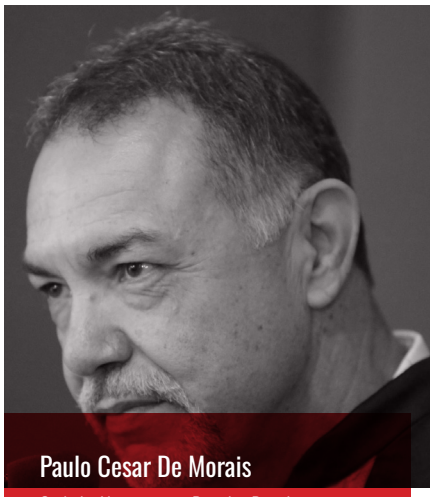
Farid Moinfar
Ordensklinikum Linz Barmherzige Schwestern, Austria

Marika Crohns
Impactful Innovations Management Consultants LLC, United Arab Emirates

Michael Thompson
University of Toronto, Canada

Michele Mishto
Francis Crick Institute, United Kingdom

Patricia Tai
UpToDate, Canada



Paulo Cesar De Morais
Catholic University of Brasilia, Brazil

Thank You All...

Keynote Speakers

Pietro Salvatori

Independent H&N Surgeon, Italy

Rajvir Dahiya

University of California San Francisco, United States

Vandana Tiwari

Dr. Ram Manohar Lohia Institute of Medical Sciences, India

Thank You All...

Welcome Message

Hello

My name is Rajvir Dahiya, Professor Emeritus of Urology Oncology at the University of California San Francisco School of Medicine (UCSF), San Francisco, California, USA

As an organizing committee member, it is with immense enthusiasm and deep gratitude that I welcome you all to this International Cancer Research Conference at Singapore On March 24-26, 2025.

Dear Distinguished Guests, Esteemed Scientists, Honored Attendees, Organizing Committee members, Chairs, Co-chairs, Speakers, and Students, all of you are a premier gathering of brilliant minds dedicated to transforming the future of cancer diagnosis, treatment, and care. Today, we stand on the frontline of innovation, where cutting-edge science meets a shared vision to conquer one of humanity's greatest challenges.

This conference serves as a dynamic platform for collaboration, uniting renowned researchers, clinicians, industry leaders, and young scientists from across the globe. Together, we will explore groundbreaking discoveries, novel technologies, and revolutionary therapeutic strategies that have the potential to redefine cancer care.

Cancer research is not just a scientific endeavor; it is a mission of hope, perseverance, and impact. Every breakthrough we discuss today represents a step toward earlier detection, more effective treatments, and ultimately, saving lives. Your dedication, passion, and relentless pursuit of knowledge are the driving forces behind these advancements, and this event is a testament to your invaluable contributions.

Over the next few days, we will witness thought-provoking keynote lectures, groundbreaking presentations, and collaborative discussions that will inspire new ideas and ignite innovation. Let this conference not only be a forum for exchanging knowledge but also a catalyst for forging meaningful collaborations that accelerate progress in the fight against cancer.

As we embark on this remarkable journey together, I encourage you to engage, challenge ideas, and push the boundaries of what is possible. The future of cancer research is being shaped here, in this very room, by each one of you.

With that, I officially welcome you to an exciting and transformative conference. Let's make history together!

Thank you.

Rajvir Dahiya

University of California San Francisco, United States

Welcome Message

Dear Conference Attendees,

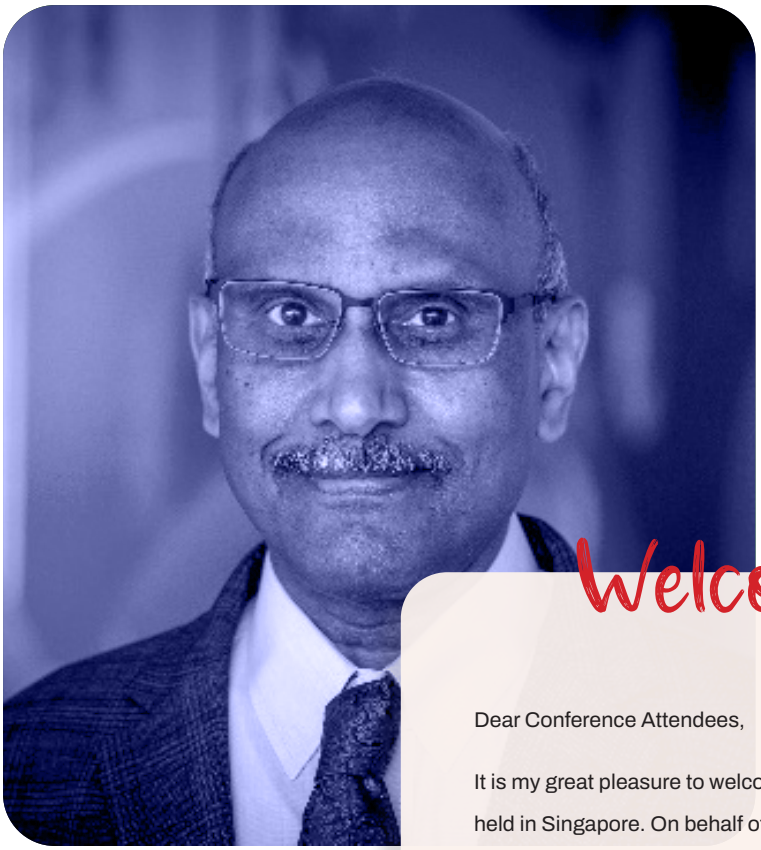
Cancer Research is a well organized conference that every researcher and layperson will enjoy. As there are more cancer researches reported every day, the topic is so relevant to all walks of life. Pollution, virus, sun damage, genetics, cigarette smoke, etc all increase cancer risk that we are all exposed to.

In line with this, my keynote speech will discuss new updates on cancer management and I shall focus on lung, prostate, skin, brain cancers and briefly mention other cancer sites if I have the time. It will be a great opportunity for all participants to know these recent advances. I promise to make it easy to understand with laymen terms as much as I can, despite the slides will show abstracts, diagrams and titles full of medical jargons! So much to tell you in 40 minutes. Indeed other speakers will offer you also super nice experience as they are from famous international universities and institutes.

The venue, or city that holds this conference is very pleasant to visit, with relatively inexpensive hotels that I enjoy to go often! The shopping is lovely and you can always find the bargain or the dream gift you want to bring home to your loved ones.

Professor Patricia Tak Hing Tai

UpToDate, United States



Welcome Message

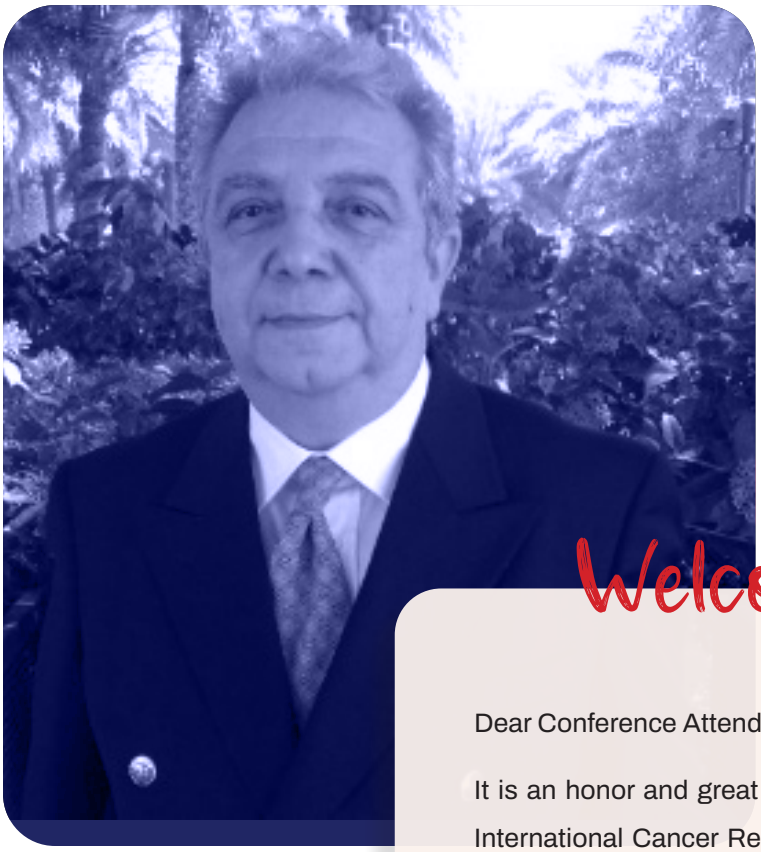
Dear Conference Attendees,

It is my great pleasure to welcome you to the “International Cancer Research Conference” held in Singapore. On behalf of the conference organizers, we extend a hearty welcome to in-person participants as well as to those who are joining us virtually from around the world. We are living in a crisis era of increasing cancer incidence in both adults and children, and conferences like this one are desperately needed to advance our understanding of the growing cancer epidemic. This multi-disciplinary conference links basic, clinical and public health research on cancer. We attempt to address a variety of preclinical and clinical research topics on cancer including clinicopathologic presentations, prognostic features, genetics, various treatment modalities including immunotherapy and other targeted therapy, biologic basis of treatment, multi-omics analysis, preclinical drug research, biomarkers, and targeted therapy. This will be a great opportunity for all types of participants including young and senior researchers, scientists, clinicians and academicians to gain knowledge with the up-to-date information on various aspects of cancer. I encourage you to actively listen, engage in live discussions, network with your peers and widen your scope with better knowledge and research opportunities.

Sincerely yours,

Atif A. Ahmed, MD

University of Washington, Seattle, United States



Welcome Message

Dear Conference Attendees,

It is an honor and great pleasure to write a few welcome notes for the International Cancer Research Conference, whose theme is: Breaking Barriers, Building Hope: Advancing Cancer Research and Care. Undoubtedly, the main goal of those involved in the diagnosis and treatment of cancer remains the increase in survival, but it is increasingly evident that the improvement of the quality of life must go hand in hand. The multi-disciplinary approach - in other words, synergy - is the necessary key to effectively achieve the aforementioned objectives. I sincerely hope that my modest contribution brought during this exciting International Conference can help stimulate the development of this strategy.

Sincerely yours,

Pietro Salvatori

Independent H&N Surgeon, Italy

ABOUT
MAGNUS GROUP

Magnus Group, a distinguished scientific event organizer, has been at the forefront of fostering knowledge exchange and collaboration since its inception in 2015. With a steadfast commitment to the ethos of Share, receive, grow, Magnus Group has successfully organized over 200 conferences spanning diverse fields, including Healthcare, Medical, Pharmaceutics, Chemistry, Nursing, Agriculture, and Plant Sciences.

The core philosophy of Magnus Group revolves around creating dynamic platforms that facilitate the exchange of cutting-edge research, insights, and innovations within the global scientific community. By bringing together experts, scholars, and professionals from various disciplines, Magnus Group cultivates an environment conducive to intellectual discourse, networking, and interdisciplinary collaboration.

Magnus Group's unwavering dedication to organizing impactful scientific events has positioned it as a key player in the global scientific community. By adhering to the motto of Share, receive, grow, Magnus Group continues to contribute significantly to the advancement of knowledge and the development of innovative solutions in various scientific domains.

ABOUT
Cancer Research 2025

International Cancer Research Conference" (**Cancer Research 2025**) a premier gathering will takes place during **March 24-26, 2025**, in **Singapore** and **online,** themed around "Breaking Barriers, Building Hope: Advancing Cancer Research and Care."

The Cancer Research 2025 Conference will bring together oncologists, researchers, healthcare professionals, and industry leaders to explore groundbreaking advancements in cancer treatment and research. Key topics will include innovative therapies, AI in oncology, precision medicine, immunotherapy, and the latest developments in cancer diagnostics.

Attendees will benefit from keynote addresses, oral and poster presentations, and panel discussions, offering valuable insights into how emerging technologies and research are transforming cancer care and improving patient outcomes.

Participants of Cancer Research 2025 will have unparalleled opportunities for professional growth and networking. The conference will provide access to the latest research, cutting-edge technologies, and innovative practices in oncology. It will serve as a dynamic platform for exchanging ideas with leading experts, engaging in collaborative discussions, and contributing to the future of cancer research and treatment.

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KEYNOTE PRESENTATIONS

Anyou Wang

DIFIBER LLC, USA

Theory and perspective: How big data and AI accelerate the discovery of a universal cancer principle from the noncoding RNAa regime

Numerous cancer-related mechanisms are reported daily, yet a universal theory for eradicating all cancers remains elusive. Fortunately, recent advances in big data and AI have identified noncoding RNAs as central players in all cancers, challenging the conventional protein-centric view. However, unlike proteins, noncoding RNAs evolve dynamically rather than following conserved patterns.

Moreover, they remain poorly defined, and no systematic framework exists for their study, hindering the progress toward universal cancer theory. Although big data and AI hold promise for accelerating this discovery, significant challenges persist in biological data collection and computational algorithm development. Overcoming these barriers requires groundbreaking new scientific concepts, algorithms, and technologies in big data and AI.

Biography

Dr. Anyou Wang, a graduate of the University of California, Riverside, is passionate about computational biology, big data, and AI. He develops computational algorithms to extract fundamental insights from large-scale biological data. He is a pioneer in utilizing the world's largest database to uncover noncoding RNAs (ncRNAs) as central players in all cancers and as evolutionary drivers of lifespan across animal species, challenging the traditional protein-centric paradigm. Additionally, Dr. Wang has developed innovative algorithms to systematically analyze all SARS-CoV-2 genomes, uncovering the virus's evolutionary trajectory and origins, which contributed to the COVID-19 pandemic.

Atif A Ahmed, MD

Department of Pathology, Seattle Children's Hospital-University of Washington Seattle, Washington, United States

RNA binding proteins in the pathogenesis of pediatric cancer

RNA Binding Proteins (RBPs) are regulators of RNA metabolism and influence protein expression in various physiologic and pathologic conditions through regulation of RNA splicing, stability and translation. Several studies have demonstrated that RBPs play a role in cancer development through dysregulation of RNA-RBP interaction and disruption of RPB expression. One such protein, the Polypyrimidine Tract Binding protein (PTB) is a member of the heterogeneous nuclear Ribonucleoproteins (hnRNP) family and functions as suppressor of splicing and an activator of protein translation. Its accumulation in the Perinucleolar Compartment (PNC) promotes cancer progression and metastasis. Its expression in various adult cancers has been identified to correlate with tumor metastatic potential and patients' prognosis. PTB expression has not yet been studied in pediatric malignancies.

Retrospective clinical information was collected on 65 patients <22 years of age with Osteosarcoma (OS), Ewing's Sarcoma (ES), or Rhabdomyosarcoma (RMS) over a 10- year period. Formalin-fixed paraffin-embedded slides of primary or metastatic tumor samples were immunostained with SH54, a monoclonal antibody that specifically recognizes PTB that is highly concentrated in the PNC. Scoring for PTB staining was determined by estimating the percentage of cells showing positive staining. Statistical analysis was performed to demonstrate any association between PTB prevalence and demographic characteristics of patients and tumor types.

Biography

Dr. Atif Ali Ahmed is a professor and senior pediatric pathologist at Seattle Children's Hospital, Seattle, WA, USA. Graduated from medical school in 1988, completed pathology residency and fellowship training in the U.S.A. and is board-certification in Anatomic, Clinical and Pediatric Pathology. He has been in academic practice for more than 20 years with clinical and research experience in pediatric pathology. Research interests include pediatric tumors, bone pathology and pediatric cancer biology. Has over 100 academic publications including peer-reviewed articles, books and meeting abstracts. He is a member of several pathology and cancer research organizations.

PTB expression differs by tumor type with osteosarcoma (mean 58.3%, n=13) having higher prevalence than ES (mean 40.9% n=26) and RMS (37.7%, n=36) and the difference was statistically significant (p=0.0569 and p=0.0297 respectively). Bone tumors (ES and OS) had higher expression rate at metastatic biopsies compared to primary tumor biopsies (99% CI: 0.2865187 – 52.60305, p= 0.0031). PTB expression in rhabdomyosarcoma was higher in embryonal than alveolar RMS. In ES, PTB overexpression was associated with significantly shorter disease-free survival and early recurrence compared to those with tumors with low expression. PTB expression is not affected by patient's age or gender.

Studying PTB highlights its role in the pathogenesis of pediatric cancer. Targeting PTB by inhibitory therapy results in important potential clinical applications in pediatric cancer. Further studies are needed to determine its prevalence in other pediatric tumors and its relationship to prognosis.

Bene Ekine-Afolabi (PhD)

ZEAB Therapeutic, 49 Hitchin close RM3 7EQ, London, University of East London, Stratford, Water Lane, E15 4LZ

The future of pharmacogenetic polymorphism, pharmacogenomics and pharmamicrobiome in cancer treatment

Drug metabolism is crucial in any functioning of a drug, particularly in drugs for cancer treatment. Several drugs do not function in human body in their original state unless metabolised to its required functioning metabolite to affect its target organ/site or tissue. Drug metabolism occurs via certain drug metabolising enzymes and transporters which are responsible for drug biotransformation and function. The increasing understanding of the implications of genetic variation, genomics, and microbes in disease, is paramount in identifying and validating clinically relevant biomarkers to improve patient outcomes during chemotherapy.

The human genome has variation occurring in every 300-1000 nucleotides, with over 14 million Single Nucleotide Polymorphisms (SNPs) across the entire genome. The individual differences in traits, characteristics and behaviour can determine patients' response administered with same drug.

Conclusion: To reduce toxicity and improve efficiency of therapy, it is crucial to understand variations in genetics and genomic that impacts on drug response, to optimize cancer therapy and individual side effects. Researchers have intensely focused on human genomes and genetics; understanding the roles of microbiome in drug metabolism and function, could further define patient's response to therapy?

Biography

Bene is a graduate of River State University of Science & Technology in Applied Biology (Medical Microbiology option); with an MRes degree at University of East London, United Kingdom. She had her PhD study & worked at the Department of Natural Sciences, Middlesex University, UK. Trained in practical approach to toxicology in drug development (American College of Toxicology/British Toxicology Society). Bene had Harvard University part-sponsored training in Cancer Biology & Therapeutic, and received a 2nd Harvard award for a complementary training in COVID-19 and Mental Health for Medical Professionals. Bene has her expertise in evaluation and passion in improving the health and wellbeing. Her open and contextual evaluation model based on responsive constructivists creates new pathways for improving healthcare. Researching in Microbiology, Molecular Biology and Cancer: Her current focus of research (which has yielded eight designed drug models), is on the Investigation of molecular mechanism of colorectal cancer and due to the year 2020 pandemic, has been involved in drug development for COVID-19;

she set up a diagnostic laboratory for COVID-19 testing service, and now expanding diagnostic scope. Bene has been speaking in several conferences and published over seven peer reviewed articles, as well as written a chapter in Springer Nature book series on Cancer & Immunology. And a manuscript on COVID-19 was submitted to the Chief Medical Officer of United Kingdom to assist in response to the pandemic. Bene sits on journals' editorial boards, and review panels. Bene sits on congress' committee Board (European Congress on Human Genetics 2024, Oncology & Cancer Conference 2024). She is expanding through her years of research evaluation and teaching to establish Hospital projects in Africa, starting with Nigeria's state-of-the-art Hospital project. Bene is the Founder & CEO of ZEAB Therapeutic LTD, UK, ZEAB Medical Centre, Lagos Nigeria; as well as the founder of two non-profit organizations: Citizen's Wellbeing Intervention UK, and Earthwise & Citizens' Empowerment Nigeria. She was recently endorsed by and in collaboration with the Ministry of Environment Nigeria for promotion of transition to Green Renewable system. She is a member of the Medical Research group council at University of East London, a research consultant with Norxin Medical research Cooperation. She is honoured with an affiliation to Universal Scientific Education & Research Network (USERN), London, England United Kingdom. Bene is also involved in advocating for liveable environment; with recent approval for National Summit on Green-Renewable Energy & Climate in Nigeria by President of Nigeria.

Farid Moinfar[1*], Jonathan Burghofer[2], Theodora Malli[2], Melanie Rammer[2], Gerald Webersinke[2]

[1]Department of Pathology, Hospitals of sisters of charity, Linz, Austria, Upper Austria

[2]Department of Molecular genetic diagnostics, Hospitals of sisters of charity, Linz, Austria, Upper Austria

Molecular-genetic study of low-grade and high-grade "basal-like" (triple-negative) breast carcinomas

Introduction: Basal-like breast carcinomas are immunohistochemically positive for basal-type keratins such as K 5/6, K14, and K17 and mostly are triple-negative (negativity for ER, PR, and Her2/neu). "Basal-like" or triple-negative breast carcinoma is mostly known as a distinct clinicopathologic entity with aggressive clinical behavior. Therefore, most patients with this type of breast carcinoma are treated with chemotherapy. There are, however, a number of rare examples of BLC/TNC such as Adenoid-Cystic Carcinoma (ACC), Fibromatosis-Like Carcinoma (FLC), Secretory Carcinoma (SC), etc., that are associated with low-grade atypia, low mitotic activity and good prognosis. The aim of our study was to compare several example of high-grade BLC/TNC with rare variant of low-grade BLC/TNC by means of Next Generation Sequencing and Copy Number Variation (CNV)-Assay.

Material and Methods: Thirty cases of BLC/TNC (15 low-grade and 15 high-grade) were examined. For mutational analysis, we used NGS performed by FoundationOne (Foundation Medicine Inc. Cambridge, MA, USA) with a NGS panel of 322 genes. We also used OncoScan (Thermo Fisher Scientific, Walham, MA, USA) in order to evaluate CNVs and Loss of Heterozygosity (LOH) of

Biography

Dr. Moinfar studied Medicine at the Vienna and Graz University in Austria and graduated in 1991. He completed the resindency program of Pathology in 1996 followed by 2 years at fellow-ship in Breast & Gynecologic Pathology at the AFIP, Washington, DC, USA. After returning to Austria in 2000, he optained the position of associate Prof. of Pathology, medical University at Graz. Since 2014, he is the Chairman of the Department of Pathology, Hospitals of sisters of charity in Linz, Austria. He has published more than 84 research articles in SCI Journals.

the whole genome. All molecular-genetic analyses were performed on formalin-fixed, paraffin-embedded material.

Results: Low-grade and high-grade BLC/TNC are molecular-genetically drastically different with regard to Mutations and CNVs. The NGS analysis revealed significantly higher tumor mutation burden in high-grade (TMB: 95% CI: 44.51–79, 48) compared to TMB of the low-grade group (TMB: 95% CI: 20, 96–35, 03). The difference of TMB in both groups were highly significant (p=8.5x 10-3). High-grade BLC/TNC showed almost always (93.33%) TP53 mutations, while not a single case of low-grade BLC/TNC revealed TP53 mutations. While multiple amplifications of oncogenes were identified in the high-grade group, amplification of oncogenes could hardly be observed in the low-grade group.

The CNV Assay revealed much higher number and larger changes (gains and losses) in the high-grade group as compared to those identified in the low-grade group. The percentage of genome change (combination of CNVs and LOH) was significantly different in the two groups (p=8.63x 10-6, 95% CI: 1.21%-5.46% in the low-grade group and 37, 21%-60,43% in the high-grade group).

Conclusion: Basal-like or triple negative breast carcinomas do not represent a homogenous entity. The oncologists needs to recognize the rare low-grade BLC/TNC of the breast, which are morphologically, molecular-genetically, and clinically drastically different from the more common and high-grade BLC/TNC. The distinction between the two groups has a major impact on the therapeutic management of the patients, particularly with regard to the chemotherapy.

Dr. Marika Crohns MD, PhD

CEO, Impactful Innovations Management Consultants LLC, Dubai, UAE

The role of Artificial Intelligence (AI) in oncology

The Role of AI in Oncology

Artificial Intelligence (AI) has emerged as a transformative force in oncology, offering unparalleled potential to revolutionize cancer care. By leveraging advanced algorithms and machine learning models, AI enhances every phase of the cancer care continuum—from early detection and diagnosis to treatment planning and post-treatment monitoring.

In diagnostics, AI-powered image recognition systems enable rapid and accurate analysis of radiological and pathological data, improving detection rates for cancers such as breast, lung, and skin. Predictive algorithms analyze genetic, clinical, and demographic data to identify high-risk populations, facilitating personalized screening programs.

AI's role in treatment is equally groundbreaking. Predictive models guide oncologists in selecting the most effective therapies by analyzing large datasets of clinical outcomes, genomic profiles, and real-time patient data. This individualized approach minimizes adverse effects and optimizes therapeutic outcomes. Furthermore, AI-driven tools assist in radiotherapy planning by automating target delineation and optimizing dose delivery.

In clinical research, AI accelerates drug discovery and trial design by identifying potential drug targets and predicting patient responses. Natural Language Processing (NLP) facilitates the synthesis of vast scientific literature, ensuring oncologists stay abreast of emerging evidence.

Biography

Dr. Marika Crohns is a distinguished healthcare executive with over 20 years of international leadership experience. She holds MD, PhD, Board Certification in Oncology and Radiation Therapy, as well as certification in Pharmaceutical Medicine in addition to commercial training in the UK. As an award-winning keynote speaker, she is a passionate advocate on novel technologies in healthcare. She is also the author of a groundbreaking research book. Currently, Dr. Crohns acts as the CEO of two international companies, and serves on the boards of several international companies and organizations. Her dynamic career reflects a commitment to innovation, transformative oncology, leadership, and advancing global health. She is currently the CEO for Impactful Innovations Management Consultants LLC and PRiMe International LLC.

Beyond the clinic, AI enhances survivorship care by monitoring patients remotely through wearable devices and mobile health applications, providing real-time feedback and early detection of recurrence. However, challenges remain, including ethical considerations, data security, and the need for transparency in AI decision-making processes.

This abstract explores the transformative role of AI in oncology, emphasizing its ability to personalize care, improve efficiency, and enhance outcomes. By addressing challenges and fostering interdisciplinary collaboration, AI has the potential to redefine the future of cancer care.

Michael Thompson*, Soha Ahmadi Katharina Davoudian, Navina Lotay and Lidia Nemtsov

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

Biosensor-based detection of cancer biomarkers

Biosensor technology represents an attractive strategy for the detection and monitoring of biomarkers for disease states within the context of precision medicine. This approach offers the possibility for biomarker assay via incorporation into an 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.

In the present paper we discuss the application of biosensor technology for the early-stage detection of ovarian cancer. This disease results in some 150,000 deaths worldwide of nearly 300,000 new cases each year. Unfortunately, only 20% of patients are diagnosed at the early stages (I and II) of the disease when treatment is most effective, leading to a 5-year relative survival rate of only 20%. Early diagnosis of OC improves survival rate to 93%; however, there is a lack of early diagnose due to few specific symptoms being observed, and the absence of reliable, cost-effective mass screening techniques. Several biomarkers have been identified for OC, of which cancer antigen-125 (CA125) is the only one currently clinically approved.

In our research, we are working on the development of seniors for the multiplexed assay of markers for OC. Lysophosphatidic Acid (LPA) is a distinctly attractive potential biomarker with high sensitivity (98%) and

Biography

Professor Michael Thompson was appointed Lecturer in Instrumental Analysis at Loughborough University in 1971. He then moved to the University of Toronto where he is now Professor of Bioanalytical Chemistry. He is recognized internationally for his pioneering work over many years in the area of research into new biosensor technologies. Thompson has served on the Editorial Boards of a number of major international journals including Analytical Chemistry and The Analyst and is currently Editor-in-Chief of the monograph series “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, the E.W.R. Steacie Award of the Chemical Society of Canada, and recently the 2023 Royal Society of Chemistry Horizons Prize in Analytical Science.

specificity. The normal level of LPA in the body is 0–5μM, but increases to 5–50μM in OC, even in stage I. In our research, we are employing three different biosensor-based strategies for LPA detection in tandem with that for CA-125. These techniques include an ultra-high frequency acoustic wave device, a chemiluminescence-based Iron Oxide Nanoparticle (IONP) approach and electrochemical detection based on both square wave and differential pulse voltammetry. For assay of LPA all these methods incorporate the protein complex gelsolin-actin, which enables testing for detection of the biomarker binding to the complex results in separation of gelsolin from actin. In proof of concept experiments, each of the approaches is capable of the detection of LPA at the sub micromole level. In addition to the work with LPA we are developing an electrochemical system for the tandem assay of CA-125 which is based on an aptamer probe for the marker.

Michele Mishto

King's College London (UK), The Francis Crick Institute (UK)

Targeting noncanonical epitopes in anti-cancer immunotherapy

MHC class I complexes can present antigenic peptides that have a sequence produced by post-translational mechanisms such as peptide splicing. Some examples of tumour-associated spliced epitopes have been investigated for their immunogenicity in the context of cancer so far. We developed several pipelines to identify and predict these noncanonical epitopes, which are freely available. In addition, we tested the immunogenicity and the potential for therapeutical applications for those associated to various forms of cancer.

Biography

Prof. Michele Mishto is Professor in Immunobiology at the King's College London and Senior Group leader at the Francis Crick Institute in London (UK). PhD in Medical Biotechnology at University of Bologna (ITA) and a long post-doc and project leader experience at the Institute of Biochemistry at Universitätsmedizin Charite' Berlin (GER). His research focus on antigen presentation and proteasomes.

Biography

Patricia Tai[1*], Edward Yu[2], Michael Veness[3], Avi Assouline[4], Arbind Dubey[5], Rashmi Koul[5], Jidong Lian[6], Song Park[7], Vimal H. Prajapati[8], Osama Souied[9], Aoife Jones Thachuthara[10], Kelvin Wong[11], Kurian Joseph[12]

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[2]Division of oncology, Western U., London, Ontario, Canada

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[4]Oncologue Radiothérapeute, Centre de Cancérologie de la Porte de Saint-Cloud (CCPSC), Boulogne-Billancourt, France

[5]Dept of radiation oncology, Manitoba Cancer Care; U. Manitoba, Winnipeg, Manitoba, Canada

[6]U. Toronto, Toronto, Ontario, Canada

[7]U. Washington U., Seattle, USA

[8]U. Calgary, Calgary, Alberta, Canada

[9]U. Saskatchewan, Saskatoon, Saskatchewan, Canada

[10]Dept of medical oncology, Cork U. Hospital, Cork, Ireland

[11]Astellas, Canada

[12]Dept of radiation oncology, Cross Cancer Institute; Division of oncology, U Alberta, Edmonton, Alberta, Canada

Professor Patricia Tai graduated with a gold medal from U. of Hong Kong in 1984. Since then she became an experienced clinical oncologist with expertise in skin and urologic cancers. She is one of the international experts on Merkel cell carcinoma, the author in UpToDate since 2000. Being the author of 149 full publications and 167 abstracts, honorary Professor of University of Hong Kong and clinical professor of U. Saskatchewan, Canada, she now works with UpToDate and welcomes collaborations on photodynamic therapy, Merkel cell carcinoma and prostate cancer.

Updates on cancer research for detection and management

About 2 in 5 Canadians are expected to be diagnosed with cancer in their lifetime and 1 in 4 die of it. I would discuss interesting updates of advances in cancer research.

Biomarker assays have become routine for some cancers. Circulating tumor cells/cell-free deoxyribonucleic acid (DNA) can detect cancer cells at initial diagnosis, during treatment and

follow-up. Photodynamic therapy can treat several precancerous lesions, superficial skin cancers, as well as heal superficial wounds, including radiation-induced ulcers (for which we documented the first Canadian clinical case).

In April 2024 the Food and Drug Administration (FDA) approved vorasidenib for Grade 2 astrocytoma/oligodendroglioma with Isocitrate Dehydrogenase (IDH)1 or IDH2 mutation. Tumour treating fields were also FDA-approved for slowing down/stopping glioblastoma cell division.

Lung cancer screening in high-risk smokers is performed nowadays with low-dose computerized tomography scans. Early stage inoperable Non-Small-Cell Lung Cancer (NSCLC) benefits from the highly focused Stereotactic Ablative Radiotherapy (SABR) or Stereotactic Body Radiation Therapy (SBRT) with 3-year local control of 70-90% and 2-year survival of 50-70%. For advanced NSCLC, oral systemic therapy improves accessibility and quality of life. The landmark phase III PACIFIC trial compared durvalumab with placebo in patients with unresectable, stage III NSCLC and no disease progression after concurrent chemo-radiotherapy. Consolidation durvalumab significantly improved Overall (OS) and Progression-Free (PFS) survivals, with manageable safety. New ADAURA data show adjuvant osimertinib significantly improved OS, cutting the risk of death in half when compared with placebo for Epidermal Growth Factor Receptor (EGFR)-mutated NSCLC. The FLAURA trial studied osimertinib versus gefitinib or erlotinib in treatment-naive patients with advanced NSCLC with EGFR exon 19 deletion or L858R mutations. Median PFS with osimertinib is significantly longer at 18.9 months compared with 10.2 months with standard EGFR Tyrosine Kinase Inhibitors (TKIs). Subgroup analysis also demonstrated a PFS benefit in patients with CNS metastases!

Locally advanced or metastatic basal cell carcinomas may benefit from vismodegib, a Hedgehog pathway inhibitor. Recently, cemiplimab, a PD-1 inhibitor, was approved for advanced squamous and basal cell carcinomas. Other PD-L1 inhibitors, including avelumab, pembrolizumab and retifanlimab, have received FDA approval for advanced Merkel cell carcinomas. In addition to multiple immune checkpoint inhibitors, Tumor-Infiltrating Lymphocyte (TIL) therapy is used in metastatic melanoma. Skin brachytherapy delivers localized radiation with excellent cosmetic outcomes. Mohs surgery enhances cosmetic results, reduces positive margins and facilitates tissue reconstruction. Lymphovenous Anastomosis (LVA) effectively improves lymphedema in 23-100% of patients by redirecting lymphatic flow to nearby veins.

Androgen Receptor Pathway Inhibitors (ARPI) are used as concurrent and adjuvant therapy for node-positive prostate cancer. Radio-ligand treatment for castrate-resistant prostate cancer improves OS. Renal cell carcinoma can now be cured by SABR, again an achievement for the inoperable elderly.

In conclusion, advances in cancer detection and treatment have improved cancer outcome and quality of live in recent decades. Future challenges are controlling health care costs (sustainability issues) and increasing access in developing countries. Lastly, I would like to thank each member of my team, the International Oncology Cancer Research Group (IOCRG).

Paulo Cesar De Morais

Genomic Sciences and Biotechnology, Catholic University of Brasilia, Brasilia, DF, Brazil

Institute of Physics, University of Brasilia, Brasilia, DF, Brazil

Successful tumor growth control using PLGA nanoparticles loaded with selol and nanomaghemite

Poly (Lactic-Co-Glycolic Acid) (PLGA) nanoparticle has proven extremely useful for drug delivery, thanks to its high biocompatibility, biodegradability, and toxicologically safe degradation products. This talk will emphasize the use of nanosized (~140 nm mean diameter) PLGA loaded with selol and maghemite nanoparticles (~9 nm mean diameter). The platform comprising selol-maghemite-loaded PLGA nanoparticles is very promising for solid tumor growth hindering in combination with magnetohyperthermia. The effectiveness of using the as-fabricated selol-maghemite-loaded PLGA nanoparticles (NcSel) associated with different tumor treatment protocols will be explored. One-week treatment of combined therapy comprising magnetic hyperthermia and selol-loaded nanoparticles in reduced dosages is able to hinder primary breast tumor growth in aged animal model. Moreover, different biological observations are in excellent agreement with the parameters extracted from the logistic modelling. Importantly, biocompatibility analyses revealed that the adverse effects of the treatments are negligible. Taken together, the results herein reported lead us to suggest the translation of this nanotherapeutic approach for future clinical trials. In short, the use of NcSel plus MHT as a short-term neoadjuvant therapy, for example, may represent a less costly, safe and effective alternative in cancer control in aged patients. It is also expected that the shorter treatment period will contribute to the adherence of cancer patients.

Biography

Professor Paulo César De Morais (H60), PhD, was full Professor of Physics at the University of Brasilia (UnB)–Brazil up to 2013. Appointed as UnB's (Brazil) Emeritus Professor (2014); Visiting Professor at the Huazhong University of Science and Technology (HUST)–China (2012-2015); Distinguished Professor at the Anhui University (AHU)–China (2016-2019); Full Professor at the Catholic University of Brasília (CUB)–Brazil (2018); CNPq-1A Research Fellow since 2010; 2007 Master Research Prize from UnB. He held two-years (1987-1988) post-doc position with Bell Communications Research, New Jersey–USA and received his Doctoral degree in Solid State Physics (1986) from the Federal University of Minas Gerais (UFMG)–Brazil. With more than 13,000 citations, He has published more than 500 papers (Web of Science), delivered more than 200 international invited talks, and filed 16 patents.

Dr. Pietro Salvatori

Independent H&N Surgeon, Italy

Principles of oral rehabilitation in H&N cancer patients

Treatment goals for Head & Neck cancers can be indicated as: 1) Eradication of the tumor (ablative surgery or XRT, combinations), 2) Restoration of the anatomy (reconstructive surgery), 3) Rehabilitation of the function.

Of course, the main goals are to increase survival and recovery rate, but equally important is to maintain and restore a good quality of life.

This presentation deals with the identification of the most common problems that can affect oral function (mainly chewing and swallowing), their possible solutions and the results. Numerous clinical examples will be presented.

Biography

Dr. Pietro Salvatori graduated at the University of Florence Medical School, Italy. He earned specialization in General Surgery, Otorhinolaryngology, and Maxillo-Facial Surgery. He was Research Fellow at the University of Liverpool, served in several Institutions, and ended his hospital career as Head of ENT-H&N Department of the Humanitas San Pio X Hospital, Milan, Italy. At present, Dr. Salvatori acts as freelance Head & Neck Surgeon. Most of both his work and research dealt with head and neck cancer. Dr. Salvatori published more than 70 papers and gave about 150 speeches. During recent pandemic, he made research with international colleagues and published on ethanol inhalation to treat SARS-CoV-2 infection and Covid-19.

Yutaka Hashimoto, Shiv Verma, Guoren Deng, Dharam P Chauhan, Sujit Nair, Hirofumi Yoshino, Junya Arima, Yutong Liu, Koji Hatano, Yogesh Shivakumar, Raghunath S. Krishnappa, Chitra Chandran, Narasimhan Ragavan, Hideki Enokida, Norio Nonomura, K C Balaji, Sudhir K Rawal, Ashutosh K. Tewari, Sanjay Gupta, Rajvir Dahiya*

VA San Francisco Health Care and UCSF School of Medicine, San Francisco, CA; Case Western Reserve University and University Hospitals Cleveland Medical Center, Cleveland, OH; Geneverify Inc, Hayward, CA; Tisch Cancer Institute, Icahn School of Medicine at Mount Sinai, New York, NY; Kagoshima University Hospital, Kagoshima, Japan; Osaka University Medical School, Osaka, Japan; HCG Cancer Hospital, Bengaluru, India; Apollo Cancer Center Hospital, Chennai, India; University of Florida College of Medicine, Jacksonville, FL; Rajiv Gandhi Cancer Institute & Research Centre, New Delhi, India; UCSF School of Medicine, San Francisco, CA, USA

A novel mRNA-genomic technology for screening, diagnosis, prognosis, personalized medicine and treatment of prostate cancer

Background: Current screening methods, have limitations in the diagnosis and prognosis of prostate cancer. This study examined whether mRNA-genomic biomarkers can improve screening and monitoring of prostate cancer.

Biography

Rajvir Dahiya (born May 30, 1956) is an American Indian medical oncology scientist with expertise in urology oncology diagnosis, prognosis and risk assessment through genetic and epigenetic technology. Dahiya retired in 2021 as a Professor Emeritus and Director of Urology Oncology Research Center at the Veterans Affairs Medical Center and the University of California San Francisco School of Medicine (UCSF) after 34 years of service.

Methods: We identified a panel of 25 prostate cancer-related genes through rigorous bioinformatics, computational analyses and genetic testing. These tests, developed by Geneverify Inc. (U.S. patent # 10801072 and 10822661) were utilized and optimized by qRT-PCR to determine the diagnostic and prognostic ability of prostate cancer using blood and tissues.

Results: A total of 419 prostate cancer patients from 9 different hospitals (135 blood and 284 tissue samples) and 130 normal samples were analyzed in this study. The study endpoints were to analyze mRNA genomic profiling and correlate with clinicopathologic parameters of the patients. In the blood, a 25-panel gene-set separated prostate cancer patients from non-cancer, AUC=0.906 [sensitivity=90% and specificity=91%]. Assessment of tissue specimens from benign and cancer patients demonstrated similar results with AUC=0.9514 [sensitivity=95% and specificity=94%]. Interestingly, patients with Gleason grades >7 showed higher expression of these genes compared to Gleason Grades <7, suggesting the prognostic ability of the gene-set. When we compared the gene expression in blood verses tissues, there were similar patterns, suggesting that blood can be used for screening, diagnosis and risk assessment of prostate cancer.

Conclusions: To our knowledge, this is the first study to investigate the role of mRNA-based genomic signatures for screening, early diagnosis and prognosis of prostate cancer using blood samples.

Biography

Professor Vandana Tiwari[1*], Yadvendra Shahi[1], Anumesh Kumar Pathak[1], Akash Agarwal[2]

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Altered expression of endoplasmic reticulum stress response genes and circular RNAs in oral squamous cell carcinoma tumorigenesis

Background: Oral Squamous Cell Carcinoma (OSCC) is a leading malignancy with high mortality and poor prognosis. Dysregulation of Endoplasmic Reticulum Stress Response (ERSR) genes and circular RNAs (circRNAs) contributes to its tumorigenesis and progression.

Aim: This study aimed to evaluate the expression levels of ERSR-related genes and circRNAs in OSCC tissues and their role in disease pathogenesis.

Methods: A case-control study analyzed biopsy-confirmed OSCC tissues (n=25) and adjacent non-tumor tissues (n=25). Patient demographics, tumor location, histopathology, and TNM staging were recorded. Expression levels of ERSR genes (GRP78, CHOP, ATF6) and circ RNAs (CirRNA-CDR1S, CirRNA10036) were quantified using qRT-PCR. Relative fold changes were calculated using the $2^{-}(\Delta CT)$ method, with significance set at $p<0.05$.

Results: The cohort included predominantly males (23:2), mean age 40±10.3 years. Risk factors included tobacco chewing (88%), smoking (64%), and alcohol consumption

Professor Vandana Tiwari studied M.Sc. in Biochemistry at the Allahabad University, Allahabad UP India. She received her PhD degree done her PhD in 1998 from Banaras Hindu University Varanasi, UP India. After three years postdoctoral fellowship form Indian Council of Medical research New Delhi on “Evaluation of Polymerase Chain Reaction in diagnosis of extra pulmonary tuberculosis” she was awarded Women Scientist Fellowship from Department of Science & Technology New Delhi in 2009. In year 2012 she joined as Assistant Professor in Department of Biochemistry at Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow. She has published more than 110 research articles in peer reviewed journals.

(36%). Tumors were located in the buccal mucosa (60%), tongue (28%), alveolus (8%), and lip (4%), with moderate (68%), well (28%), and poorly differentiated (4%) histopathology. Staging included T0 (8%), T1 (4%), T2 (40%), T3 (28%), and T4 (20%), with lymph node involvement in 36% (N+). Significant over expression was observed in OSCC tissues compared to non-tumor tissues: CirRNA-CDR1S (4.14-fold, p=0.048), CirRNA10036 (3.41-fold, p=0.034), ATF6 (4.71-fold, p=0.036), GRP78 (3.8-fold, p=0.03), and CHOP (1.63-fold, p=0.043).

Conclusion: ERSR genes and circRNAs are significantly up regulated in OSCC tissues, underscoring their role in tumorigenesis and progression. These findings warrant further validation in larger cohorts.

Keywords: OSCC, ERSR, Circular RNAs (Circ RNAs), Tumorigenesis, Gene expression.

BOOK OF ABSTRACTS

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ORAL PRESENTATIONS

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Synthesis and characterization of ruthenium(III) complex containing 2-aminomethyl benzimidazole, and its anticancer activity of in vitro and in vivo models

A novel ruthenium(III) complex with 2-Aminomethyl Benzimidazole (AMBI) has been synthesized and characterized by elemental analysis, spectroscopic (FTIR, UV–Vis and XRD), thermal, magnetic and electrochemical techniques. Characterization shown that the ruthenium ion is octahedral coordinated by three H_2O molecules, one chloride ion and a bidentate AMBI. The calf thymus DNA binding activity of complex was studied by absorption spectra and viscosity measurements. The strong interaction between Ru(III) complex and CT-DNA was investigated and the K_b value equals 8.3 × 10−5. The cytotoxicity effect of water soluble $[RuCl(AMBI)(H_2O)_3]Cl_2$ complex on breast adenocarcinoma (MCF-7) and human colon carcinoma (HCT-116) cell lines (in vitro) was investigated by the MTT method. The Ru(III) complex shows good cytotoxic activity and the IC_{50} values are 57.20 and 18.08μg/mL toward MCF-7 and HCT-116, respectively. The antiproliferative effect of Ru(III) complex on HCT-116 cells was nearly as the cisplatin value; that indicates its efficiency as anticancer agents. Apoptosis was studied by AO/EB staining and the Ru(III) complex shows apoptotic effect against the two cancer cell lines by percentage to 23.1 and 33.1%. DNA damage assay by gel electrophoresis was done and the DNA fragmentation that appears in the two treated lanes indicates that there was destruction in the nuclear DNA. The cell cycle arrest by flow cytometry showed that the Ru(III) complex stops the two cancer cell lines proliferation by decreasing the S and G0/G1 phases and increase in G2/M indicates that the interactions of our complex with DNA prevents the entry of cells into the DNA synthesis phase as well as into a new cell cycle. Moreover, the Ru(III) complex stops the proliferation of Ehrlich Ascites Carcinoma (EAC) bearing female mice (in vivo study) with low toxicity to the kidney. In addition, the antioxidant enzymes' activity in the treated and untreated groups' blood samples were also investigated. The decrease in topoisomerase I (Top I) levels in treated mice groups than the EAC group indicates the effect of our compound as topoisomerase I inhibitor.

Biography

Prof. Abdel Ghany F. Shoair and graduated as BSc in 1998 and MSc in 1993 in chemistry (Mansoura university, Egypt). He did his PhD work under supervision of Prof. Bill Griffith (Department of Chemistry, Imperial college, London University, UK) and received his PhD degree in 1998 (Damietta faculty of Science, Mansoura University, Egypt). He obtained two postdoc fellowship one at Tamkang University in 2002 and at Kuwait University in 2004. He was promoted as an Associate Professor in 2007 then as professor in 2015 at Damietta university. He has published more than 50 research articles in international journals.

Amir Ashraf

Homeopathic Physician, Dr. Amir's Family Homoeopathy Clinic, Kannur, Kerala

Effectiveness of homoeopathic medicine for brochogenic carcinoma: Case report with pre & post HRCT report

Introduction: Homoeopathy is an alternative system of medicine discovered by German physician Samuel Hahnemann in 1796. It has been used by several people for various health conditions globally for more than last 200 years. In India, homoeopathy is considered as a major system of alternative medicine. Homoeopathy is found effective in various medical conditions including Cancer. Lung cancer is the most common cancer in the world, and a leading cause of death in men and second most common cause in women. It is responsible for 1.3 million deaths worldwide.

Objective: To reduce the Symptomatology and to prevent the treatment of Chemotherapy.

Case Presentation: Mr Xxx aged 73year old came with complaint of cough, hemoptysis with dyspnea and, with HRCT and contrast CT study of thorax result, as Bronchogenic carcinoma.

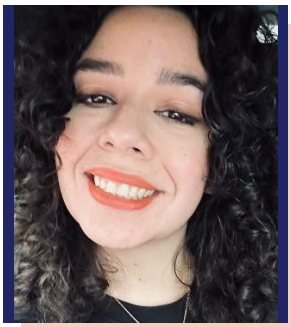
Results: There is significant change noticed since after intake of Homeopathic medicine with the evidence of pre and post HRCT.

Conclusion: Homoeopathic Medicine is effective in treatment of Bronchogenic carcinoma.

Keywords: Homoeopathy, Alternative Medicine, Bronchogenic carcinoma, Medicine.

Biography

Dr. Amir Ashraf has completed the Bachelor of Homoeopathic Medicine and Surgery (BHMS) from the Rajiv Gandhi University of Health Science, Bangalore in 2013 and completed the Internship Training programme for one year at the father Muller Homoeopathic Medical College and Hospital, Mangalore.



Andrea González Báez[1*], Gustavo Hernández Vidal[1], Luis Edgar Rodríguez Tovar[1], Pablo Zapata Benavides[2], Gerardo Méndez Zamora[3], Diana Elisa Zamora Ávila[1]

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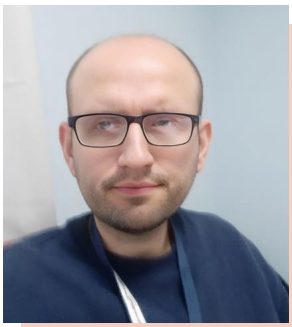
Effect of BRCA silence on the nervous system in a murine model of breast cancer

Cancer is a complex pathology of great relevance for public health due to its high mortality and morbidity. Specifically breast cancer, is one of the main causes of death in women worldwide, demonstrating that its incidence has been exponentially uprising. The multifactorial nature of breast cancer makes it complex to address it; however, understanding its pathophysiological mechanisms and the factors involved in its development and progression is key to enhancing early diagnostic tools, improving treatment alternatives and providing better strategies for managing long-term effects that diminish quality of life for those affected from it in a timely manner. These efforts to elucidate cancer's mechanisms have identified genetics and the nervous system as relevant factors in understanding cancer behavior. Genetic alterations affecting genes crucial for maintaining homeostasis across bodily systems have been associated with an increased risk of certain types of cancer. This is the case of the BRCA gene, which under normal conditions, supports the development and proper functioning of the nervous system, however, when it is mutated, it predisposes to the development of breast, ovarian and pancreatic cancer, among others. In recent years, the nervous system's role as a key factor for cancer development and proliferation has been increasingly recognized, with bidirectional interactions influencing cancer progression, metastasis, and prognosis. While these factors have been studied individually, considering cancer's multifactorial nature, it is essential to investigate the combined effects of genetic and nervous system interactions in cancer patients. Thus, our objective in the present work is to evaluate the effect of BRCA gene silencing in the tumor on the nervous system using a murine model of breast cancer. To achieve this, we will first work on the 4T1 cell line, applying RNA interference to silence the BRCA gene and then evaluating its effect on BRCA gene expression using PCR technique. Subsequently, a murine model of breast cancer will be established and the interference RNA will be administered. After silencing, cognitive damage will be assessed through a battery of standardized ethological tests that will consider the dimensions of learning and memory, exploration, sensory skills, motor skills and anxiety. Following testing, the animals will then be euthanized in compliance with the applicable regulations, and histopathological lesions in the nervous system will be analyzed. Additionally, we will analyze the expression of neurodegeneration and neuroinflammation indicators using the immunohistochemistry technique. With the results obtained, we aim to generate insights that could led to the development of targeted therapies and improved palliative care options for patients with this disease.

Biography

Andrea González, studied Veterinary Medicine at Universidad Autónoma de Nuevo León and Psychology at Universidad del Valle de México, graduate as MSc in 2015 and later got a second Master Degree at Universidad de Zaragoza in the field of applied animal behavior. She is certified as a Behavioral Veterinary Specialist in México by CONEVET as well as an Animal Assisted Therapy specialist by CTAC Barcelona, she is a professor at Veterinary Medicine School at Universidad Autónoma de Nuevo León and today she is doing her phD at the same University in the field of neuroscience, behavior and cancer.



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MicroRNA expression analyses reveal biomarker candidates for improved diagnosis and prognosis of pediatric Burkitt lymphoma

Burkitt lymphoma is the most common non-Hodgkin lymphoma in children. Pediatric Burkitt lymphomas (pBLs) are characterized by fast tumor growth; therefore, rapid diagnosis and initiation of therapy is of utmost significance. MicroRNAs (miRNAs) are small non-coding RNAs that were extensively studied in a variety of cancer types. However, the roles of microRNAs (miRNA) in diagnosis and prognostication were not fully investigated in these lymphomas. In this study, we identified differentially expressed miRNAs in tumor tissues and plasma of pBL cases as circulating cell-free or exosomal miRNAs through microRNA sequencing (miRNA-Seq) so as to discover potential diagnostic and prognostic biomarkers. Through ROC analyses, we identified several overexpressed miRNAs with the potential to improve diagnosis. Analyses of the prognostic potential of 133 overexpressed miRNAs revealed that 7 of them are associated with inferior overall survival. We also identified circulating cell-free and exosomal miRNAs overexpressed in pBL cases which can potentially improve diagnosis through ROC analyses. Analysis of the diagnostic potential of total miRNA concentrations showed that total circulating cell-free miRNAs may have the potential to improve diagnostication. Finally, we

cross-validated expression of selected miRNAs. To sum up, certain tumor tissue and plasma miRNAs may potentially be used as biomarkers for improved diagnosis and prognostication. Validation of the identified plasma miRNAs in independent cohorts will be needed to ensure their non-invasive biomarker potential.

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.

Changjiang Yang

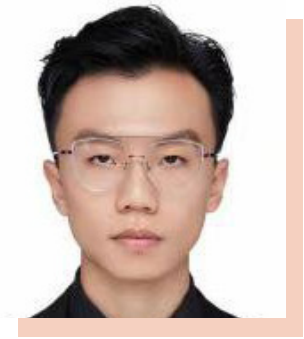
Department of Gastrointestinal Surgery, Peking University People's Hospital, Beijing, China

Importin7 induces M2 macrophage and promotes the progress of colorectal cancer by regulating the nucleus translocation of GRP78

Recent research has linked Importin7 to cancer aggressiveness and a dismal prognosis, but its function in Colorectal Cancer (CRC) is unclear. This study aimed to elucidate the clinical relevance, biological function, and potential molecular mechanisms of IPO7 in CRC. Our results showed that the expression of IPO7 in CRC were higher than normal epithelium. Furthermore, the high expression of IPO7 was related to lymph node metastasis, distant metastasis and stage of CRC patients. The overall survival of patients with high expression of IPO7 was lower than that of patients with low expression. Knockdown of IPO7 significantly inhibited colon cancer cell's proliferation compared with the control group. Significantly, IPO7 expression in CRC was significantly correlated with macrophage. The infiltration of M2 macrophage, which is critical for immunosuppressed microenvironment, was induced by IPO7. Mechanistically, IPO7 might induce macrophage polarization to M2 by regulating the secretion of CCL5. IPO7 mediates the entry of GRP78 into the nucleus to activate the expression of CCL5. In summary, this investigation proved that IPO7 promoted growth in CRC by promoting macrophages into M2 through CCL5, which provided a promising prognostic biomarker and a potential therapeutic target for CRC patients.

Biography

Changjiang Yang received the bachelor of medicine degree in clinical medicine from Central South University, XiangYa School of Medicine, Changsha, China in 2019 and the master degree in Surgery from Peking University Health Science Center, Beijing, China, in 2022. He is currently working toward the Ph.D. degree in Surgery in the Peking University People's Hospital, Beijing, China. His research interests include the mechanism of metastasis and immune microenvironment in gastrointestinal cancer.



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Decoding the universal proliferative engine of pituitary adenomas: From single-cell insights to cross- subtype therapeutic innovation

Objective: Pituitary adenomas (PAs) are clinically heterogeneous tumors with subtype-specific hormonal profiles, yet current therapies remain largely reactive and fail to address their proliferative core. This study challenges the dogma of subtype-driven tumorigenesis by identifying a universal proliferative center across PA subtypes, aiming to redefine therapeutic strategies through mechanism-based targeting.

Methods: Single-cell RNA sequencing was performed on 43 PA samples (125,390 high-quality cells) spanning all major hormonal subtypes. Cell clustering, marker gene analysis, and functional enrichment were employed to characterize subtype-overlapping proliferative populations. Regulatory networks were inferred via transcription factor (TF) prediction (X2K platform) and pathway annotation.

Results: We identified a pan-subtype proliferative center universally present across all PA subtypes, despite their divergent cellular origins. This cluster exhibited robust activation of cell cycle progression (HSA-1640170), DNA replication (ORC1/6,TYMS), and chromatin remodeling (ANP32A/B, PTMA) pathways. A conserved regulatory axis involving CDK1, MYC, and E2F1 emerged as the central driver, orchestrating downstream effectors through transcriptional and post-translational networks. Strikingly, this center's molecular architecture remained consistent across subtypes, suggesting a unified proliferative mechanism independent of lineage-specific origins.

Conclusion: Our findings reveal a previously unrecognized proliferative center that transcends PA subtype classifications, unifying hormonally distinct tumors through a CDK1-MYC-E2F1-centric regulatory program. This discovery implies that divergent PA subtypes may converge on a shared proliferative "core" during tumor progression, offering two key translational hypotheses: First, the core regulators (CDK1, ANP32A/B) could serve as pan-subtype therapeutic targets, potentially disrupting proliferation across all PA variants. Second, quantifying this center's activity may improve risk stratification beyond current subtype-based paradigms. To sum, our data provide a foundational framework for redefining PA biology and guiding future therapeutic development focused on this conserved center.

Biography

Dr. Changqin Pu earned dual Bachelor's degrees in Clinical Medicine (Nanchang University) and Biomedical Science (Queen Mary University of London) with First-Class Honours. Currently training at the Chinese Academy of Medical Sciences, his research integrates single-cell transcriptomics (Institute of Genetics and Developmental Biology, CAS) to decode pituitary adenoma heterogeneity and machine learning-driven prognostic models for glioblastoma (Nanchang University). Awarded the Global 100 "Leaders of Tomorrow in Biomedicine" (GBD, 2022) and Gold Medals at the International "Internet Plus" Innovation Competition (2021, 2022), he has published 6 SCI papers on neuro-oncology and psychiatric informatics, alongside patented innovations in emergency medical systems. His work bridges computational biology with precision oncology.

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Bioinformatics and genomic approaches for assessing gene fusion process in MCF7 breast cancer cell models under early estradiol stimulation for assessing estrogen receptor β (Erβ) tumor suppressor activity

Cancers are genetic and epigenetic diseases in which cells divide and grow in uncontrolled ways due to mutations or molecular alterations. Gene fusion phenomena is recurrent in cancer cells. One of the most common cancers is breast cancer, representing worldwide public health concern. Several studies showed the involvement of estrogens and estrogen nuclear receptors in monitoring breast cancer. Indeed, integration between genomic and bioinformatics tools strongly contribute in improving cancer molecular biology interpretation and comprehension. Herein, we performed a transcriptomic analysis aiming to assess gene fusion events in MCF7 Breast Cancer (BC) models that expressed estrogen nuclear receptors α/β under early (2h) Estradiol (E2) stimulation. We aligned genomic reads sequences of that BC model on the GRCh38 human genome, by using RNA-STAR. Next, we executed gene fusion calling via star-fusion package. Results showed a non-significant variability regarding gene fusion happening events between estradiol-stimulated (MCF7E) and non-stimulated (MCF7noE) MCF7 BC cells lines ($p>0.05$). Common detected genes fusions between analyzed BC models result to be biomarkers of several cancers including BC, and were characterized by the intra-chromosomic interactions. Findings revealed five (5) gene fusion events specific to BC cells lines non-stimulated (MCF7noE), and recognized as BC biomarkers. Results exhibited estrogen nuclear receptor beta (Erβ) as inhibiting the expression of these BC biomarkers in MCF7 BC cells lines under estradiol stimulation (MCF7E). In conclusion, even if early estrogen hormone stimulation by inducing nuclear Erβ has non-significant impact on gene fusion variability between MCF7noE and MCF7E BC cell line models by contrast to the alternative splicing event, present study highlighted onco-suppressor activity of Erβ in breast cancer.

Keyword: MCF7 Breast Cancer, Gene Fusion, Estradiol (E2), Estrogen Nuclear Receptor α and β (Erα and Erβ).

Biography

Dago Dougba Noel, Ph.D., is an Associate Professor at the Biological Sciences Research Unit of Peleforo Gon Coulibaly University in Ivory Coast. He obtained his PhD in Applied Biotechnology from the University of Verona (Department of Biotechnology); specializing in research and innovation in health sciences, particularly in genomics and bioinformatics (Department of Medicine and Surgery at Salerno University-Italy working in Breast

Cancer). Currently, he is an Associate Professor at Peleforo Gon Coulibaly University in Ivory Coast, where he is responsible for the Scientific Council of the Biological Sciences Research Unit. His research areas mainly include molecular biology, genomics, bioinformatics and computational statistics. Number of Published Articles: He has published 45 articles in several reputable journals and more than 20 Conference proceedings.

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Overcoming resistance in ROS1-positive NSCLC: IDX001 as a promising next- generation therapeutic

ROS1 (Receptor Tyrosine Kinase ROS Proto-Oncogene 1) mutations in Non-Small Cell Lung Cancer (NSCLC) lead to the formation of chimeric proteins that activate key oncogenic signaling pathways, contributing to tumorigenesis. These mutations, including ROS1 gene rearrangements such as ROS1-CD74 and ROS1-SLC34A2, are present in 1-2% of NSCLC cases, predominantly affecting younger, non-smoking patients with adenocarcinoma histology. While ROS1 inhibitors like crizotinib and entrectinib are effective, resistance due to secondary mutations in the ROS1 kinase domain, such as L1951R, L2086F, G2032R, and G2032R/L2086F presents a significant clinical challenge. These mutations hinder the binding of existing inhibitors, necessitating the development of next-generation therapies. Repotrectinib, a ROS1/TRK/ALK inhibitor, has shown activity against some resistance mutations, but it faces challenges like residual resistance and CNS-related efficacy limitations. Tumor heterogeneity further complicates treatment, as selective pressure can lead to the emergence of new mutations or activation of parallel pathways. This study explores the potential of a novel small-molecule inhibitor, IDX001, in comparison to repotrectinib, focusing on its effectiveness against various ROS1 mutations. Computational docking revealed that IDX001 exhibits superior or comparable binding energy across various ROS1 mutations, including the clinically relevant G2032R/L2086F combination. Molecular Dynamics simulations conducted over 50 ns demonstrated that IDX001 maintained a stable conformation throughout the simulation period, in contrast to repotrectinib, which showed signs of instability and dissociation from the ROS1 kinase binding pocket. Toxicity predictions for IDX001 revealed no significant toxicity risks. A critical factor in the management of ROS1-positive NSCLC is the ability to address brain metastases, which are commonly observed in ROS1- positive patients. IDX001 demonstrated superior penetration of the Blood-Brain Barrier (BBB), an essential attribute for treating patients with CNS involvement. Toxicity prediction tools confirmed that IDX001 could cross the BBB without inducing toxicity, a key advantage over existing ROS1 inhibitors. Furthermore, IDX001 was identified as a P-glycoprotein I and II inhibitor, though not a P-glycoprotein substrate, ensuring effective CNS penetration without the risk of efflux. It was also found to be a CYP3A4 substrate (but not a CYP2D6 substrate or CYP1A2 inhibitor), which minimizes potential drug-drug interactions and supports its safe use in combination therapies. These findings highlight the promising therapeutic potential of IDX001 as a more effective treatment option for ROS1-positive NSCLC, particularly in cases with brain metastases, and suggest that it could provide a superior alternative to current therapies, overcoming common resistance mechanisms and improving patient outcomes.

Biography

Divyapriya Karthikeyan is a Scientist at InDspireX, with extensive experience in drug development, bioinformatics, and molecular biology. She leads innovative research specializing in miRNA's therapeutic applications and supports clinical trials for solid tumors. Currently, on the verge of receiving her Ph.D. from VIT University, she holds an M. Tech in Industrial Biotechnology from SASTRA University and B. Tech in Biotechnology from SRM University. Divyapriya has contributed to over 10 peer-reviewed publications and has received notable awards, including recognition at international conferences in Australia and South Korea. She also mentors R&D teams and supports regulatory compliance and quality management processes.

Sofia Balafouti, George Zachos, Eleni Petsalaki*

Department of Biology, University of Crete, Heraklion, Greece

A new tension-sensitive signaling pathway involving polymerization of actin prevents chromatin bridge breakage in cytokinesis

Chromatin bridges are strands of incompletely segregated DNA connecting the anaphase poles or daughter nuclei. Chromatin bridges can arise from incompletely replicated DNA, defective resolution of DNA catenates or dicentric chromosomes which are formed by chromosome fusions. If unresolved, chromatin bridges can break in cytokinesis leading to micronuclei formation and accumulation of DNA damage which lead to changes in the DNA sequence and can result in carcinogenesis. To prevent this, human cells activate the abscission checkpoint which delays abscission to prevent chromatin bridge breakage or tetraploidization due to regression of the cleavage furrow. We recently showed that the DNA topoisomerase IIα enzyme binds to catenated DNA on chromatin bridges and Rad17 protein is recruited on DNA “knots”. In turn, Rad17 recruits the Mre11-Rad50-Nbs1 protein complex and activates the ATM-Chk2-INCENP signaling pathway which leads to proper localization of Aurora B at the midbody in order to delay abscission. Furthermore, 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.

Keypoints

- 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 is a Post Docroral Research Scientist in Dr. George Zachos' lab at University of Crete, Greece. She completed her PhD in 2014 in Molecular Biology and Biomedicine at the Department of Biology. Her main interest is mitotic cell division and mechanisms that monitor mitotic progression called the mitotic spindle checkpoint and the abscission checkpoint. She is an author of 17 publications including Journal of Cell Biology, Nature Communications, Journal of Cell Science and others. Her publications have received >500 citations so far. She is currently a member of FEBS, AACR, EACR and Royal Society of Biology.

Dr. Gaurav Vishal
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Behavior of advanced (T3 & T4) squamous cell carcinoma of the oral cavity

Introduction: Squamous Cell Carcinoma (SCC) is the most common malignant neoplasm of the oral cavity. SCC is a major health problem for the world. Based on GLOBOCAN estimates, about 377,713 new cancer patients and 177,757 deaths occurred in 2020 worldwide. The load of oral squamous cell carcinoma is remarkably greater in India when compared to the western countries with 60 to 80% of our patients are documented in locally advanced stages (American Joint Committee on Cancer, Stage III-IV). The purpose of this study was to evaluate the neck node status, patterns of neck metastasis, distribution of patients according to T (T3 & T4) stage and management of squamous cell carcinoma of the oral cavity.

Methodology: 82 histopathologically proven cases of oral squamous cell carcinoma were included in our study. T3 and T4 (advanced) lesions of oral squamous cell carcinoma cases were considered. Recurrent cases and prior treatment of oral cancer by chemotherapy and radiotherapy were excluded. All the patients involved in the study underwent tumor resection with neck dissection.

Results: A total of 82 patients were staged as per TNM criteria (AJCC 8th edition). The percentage of T4 and T3 lesions were 68.30% and 31.70% respectively. The most common oral squamous cell carcinoma site was the buccal mucosa (n=42) followed by the tongue (n=19), lower alveolus (n=16), palate (n=2) and 1 patient each in floor of mouth, lip and RMT region. 53.66% patients were pathologically node-positive (pN+) and 46.34% patients were pathologically node- negative (pN0). In pathologically node-positive patients N3 category was the highest followed by N2 category and N1 category. Nearly 25% patients were pathologically node-positive with extranodal extension (pN+/ENE+) and in this category 10 out of 26 cases were present in buccal mucosa. The lymph node positivity was highest in T4 followed by T3. More than 90% metastases occurred at levels I to III lymph nodes. 62 and 20 patients were treated by surgery with postoperative radiotherapy and surgery with postoperative CTRT respectively.

Conclusion: This study showed that nearly 54% (forty four out of eighty two) of the patients were pathologically node-positive (pN+) and nearly one-fourth (twenty out of eighty two) of the patients were pathologically node-positive with extranodal extension (pN+/ENE+). More than 90% metastases occurred at levels I to III lymph nodes. Histopathology reports demonstrated the most of the patients had well-differentiated squamous cell carcinoma. The dominant site was the buccal mucosa and it is the most common oral squamous cell carcinoma in the Indian subcontinent.

Biography

Dr. Gaurav Vishal is an Oral and Maxillofacial Surgeon (M.D.S), Fellowship in Oral Oncology and Reconstructive Surgery. He completed M.D.S- Oral and Maxillofacial Surgery from Institute of Dental Sciences, Bareilly, India in 2020, Observership in Head and Neck Surgical Oncology from Mahavir Cancer Sansthan, Patna and Fellowship in Oral Oncology and Reconstructive Surgery from Rohilkhand Medical College and hospital, Bareilly, India in 2021. He has received the Emerging Oncosurgeon Award by HPP Cancer Hospital & Research Institute, with collaboration of Indian Medical Association, Lucknow (Oncological CME was organized in Lucknow), India. He has participated in various International conferences as a Speaker and Moderator. He is an expert in the field of Head & Neck Oncology, Reconstructive Surgery, Facial Trauma, Maxillofacial Pathology, Tobacco Cessation and Basal Implantology. He has several International and National Publications to his credit.

Getachew A. Dagne

College of Public Health, University of South Florida, Tampa, FL, USA

Geospatial variation and assessment of factors with incidence of colorectal cancer at small area

Background: Examining spatial distribution of Colorectal Cancer (CRC) incidence or mortality is helpful for developing cancer control and prevention programs, or for generating hypotheses. Such an investigation involves describing the spatial variation of risk factors for colorectal cancer and identifying hotspots. The aim of this study is to identify small area level risk factors that may be associated with the incidence of colorectal cancer and to map hotspots for colorectal cancer.

Methods: Colorectal cancer cases in small areas were recorded in 2018 and measurements on risk factors were also obtained. We used Bayesian spatial models for relative incidence rates and produced posterior predictive that indicates excess risk (hotspots) for colorectal cancer.

Results: The small area level unadjusted incidence rates ranged from 462 to 3.142. After fitting a Bayesian spatial model to the data, the results show that a decreasing risk of colorectal cancer is strongly associated with an increasing median income, higher percentage of Black population, and higher percentage of sedentary life at small area level. Using exceedance probability, it is also observed that there are clustering and hotspots of high colorectal cancer incidence rates in some regions.

Conclusion: Among a few small area level variables that significantly explained the spatial variation of colorectal cancer, income disparity may need more attention for resource allocation and developing preventive intervention in high-risk areas for colorectal cancer.

Biography

Dr. Dagne is a Professor in the College of Public Health, University of South Florida, with a specialization in Bayesian inference and longitudinal data analysis in areas of social behavioral interactions, mental health, and health disparity in populations. Trained as a statistician/biostatistician, Dr. Dagne has more than 27 years of experience in collaborative research in epidemiological studies and health related fields under funding from the National Institute of Health and Department of Defense. He has published more than 110 research articles in peer-reviewed journals.

Professor Dr. Ibrahiim El Bayoumy

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Lecturer of Master of clinical research -Warwick University-UK

Colorectal cancer and associated risk factors: Case control hospital based study in Egypt

Background: Colorectal cancer accounts for approximately 10% of all annually diagnosed cancers and cancer-related deaths worldwide.1 It is the second most common cancer diagnosed in women and third most in men. In women, incidence and mortality are approximately 25% lower than in men. These rates also vary geographically, with the highest rates seen in the most developed countries. With continuing progress in developing countries, the incidence of colorectal cancer worldwide is predicted to increase to 2·5 million new cases in 2035.

Methods: Two hundreds a histopathologically confirmed colorectal cancer cases were recruited from Tanta university oncology department. 150 patients matched with age, sex and social class were recruited as control group.

Results: Multivariable conditional logistic regression model showed that cases were 3.9 times more likely to have had attainted obesity (BMI≥30) in their lifetime compared to controls (OR=3.9; 95% CI:1.8–10.6). Compared to controls, cases rarely consumed fruits and vegetable OR=16.9; 95% CI:6.5–48.5), tended to consume red meat 2-5 times a week (OR=4.7; 95% CI:1.8–8.8) or more than 5 times a week (O=10.8; 95% CI: 4.5–26.9).).

Conclusions: Obesity, excessive red meat consumption and infrequent consumption fruits/vegetables intake were associated with an increased risk of colorectal cancer.

Biography

Professor Dr. Ibrahim El- Bayoumy holds bachelor of medicine and surgery (Tanta faculty of medicine-Egypt,1989), then he earned his master degree in public health, preventive and social medicine (Tanta faculty of medicine-Egypt1996), and an MD,PhD in public health, preventive and social medicine 2003 from Tanta faculty of medicine-Egypt and McGill faculty of medicine–Montreal-Canada in division of clinical epidemiology in Royal Victoria hospital through double channel system as scholarship from ministry of education-Egypt. He has been a Full professor of public health and community medicine in Tanta faculty of medicine-Egypt since November 2016. Dr. El-Bayoumy is currently working as a consultant in Public Health and Preventive Medicine at the Ministry of Health in Kuwait. He has been a lecturer in public health for post graduate Diploma and Master degree of public health in university of South Wales-UK since March 2021. He is also an adjunct professor of public health in School of public health-Texila American university Guyana-South America and He has supervised 24 PhD projects since 2018. He has published many research works in international journals, he is interested in research in epidemiology of infectious diseases like HIV, tuberculosis, brucellosis and infectious hepatitis, he is interested in epidemiology of chronic diseases like diabetes mellitus and its health economics, obesity and cancer and pharmacoepidemiology. He is a reviewer of many national and international journals. He has obtained post-graduate Master degree is diabetes care and education-Dundee faculty of medicine-Scotland–UK October 2015. He has been an invited speaker in many international conferences in China, South Korea, Japan, Hong Kong, Malaysia, Dubai, Kuwait, New Delhi, USA, Paris, France and Roma Italy.

Jian Li*, Zhengqi Cao, Zhouwenli Meng, Ziming Li, Shun Lu

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Cancer-associated fibroblasts derived soluble CADM1 predicts response to neoadjuvant chemoimmunotherapy in lung cancer

Conventional neoadjuvant chemotherapy offers limited benefit for patients with resect able Non-Small-Cell Lung Cancer (NSCLC). Recently, Neoadjuvant Chemoimmunotherapy (NCIT) has transformed perioperative management of NSCLC by priming systemic anti-tumor immunity before surgery, leading to tumor downstaging. However, in real-world clinical settings, more than half of patients fail to respond to NCIT (non-MPR), with non-MPR status typically determined only at surgery via intraoperative pathology. There is a critical need for a fast and accurate method to identify non-MPR patients early in NCIT, which could provide an opportunity to adjust treatment regimens for better outcomes.

In this study, we found that the stromal cell signature is significantly correlated with worse Overall Survival (OS) in NSCLC patients receiving NCIT. To pinpoint the responsible stromal cells subclusters, we utilized Digital Spatial Profiling (DSP) spatial transcriptomics to analyse SMA+ stromal cell-enriched perivascular structures in 16 patients from our NCIT cohort. Ecotyper deconvolution analysis revealed no significant differences in immune cells across different efficacy groups. However, COL11A1+ CAFs and SOX4+ endothelial cells were markedly upregulated in non-MPR patients, findings further confirmed by single-cell data from 24 NCIT specimens. Both CAFs and endothelial cells are key stromal cell types that contribute to tumor microvascular formation.

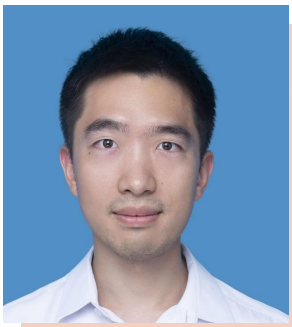
Subsequent multiplex immunofluorescence analysis of NSCLC tissues demonstrated that COL11A1+ CAFs and SOX4+ endothelial cells co-localize around tumor microvasculature. Cell communication analysis suggested that COL11A1+ CAFs interact with SOX4+ endothelial cells via the CADM1-CADM1 axis. To validate these findings, we built a biobank of CAF cell lines from 52 NCIT patients and conducted bulk RNA sequencing. COL11A1 and CADM1 were significantly upregulated in CAFs from non-MPR patients. Alternative Splicing Analysis (AS) further revealed that CAFs produce a soluble form of CADM1 (sCADM1) via AS, whereas endothelial and tumor cells only express membrane-bound CADM1. We then prospectively collected serum samples from 118 patients after their first dose of NCIT. ELISA profiling showed that serum sCADM1 levels could effectively predict non-MPR status (AUC:0.84), which was further validated by western blot analysis in the CAF biobank.

In conclusion, we identified a novel CADM1-CADM1 interaction between COL11A1+ CAFs and SOX4+ endothelial cells as a hallmark of non-MPR in NCIT. Furthermore, soluble CADM1

derived from CAFs can serve as a promising early-stage biomarker for identifying non-MPR patients within 7 days of NCIT initiation. We propose that developing a sCADM1 detection kit could be a powerful tool for monitoring NCIT efficacy in clinical practice.

Biography

Dr. Li has been pursuing a PhD at Shanghai Chest Hospital affiliated with Shanghai Jiao Tong University School of Medicine since 2016 after graduating from undergraduate studies. During doctoral period, she has been deeply engaged in the field of neoadjuvant immunotherapy. She has published 1 research articles in SCI journals.



Jiawei Guo

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Overcoming drug resistance to BET inhibitors by rational combinative strategies

Bromodomain and Extra-Terminal (BET) family members, including BRD2, BRD3, BRD4 and BRDT, function as critical readers of acetylated histones and globally activate oncogene expression. These proteins have emerged as attractive epigenetic targets for cancer therapy, with more than 50 clinical trials over the past two decades targeting lymphoma and solid tumors. Unfortunately, inevitable drug resistance to BET inhibitors (BETi) remains a significant clinical challenge: Castration-Resistant Prostate Cancer (CRPC) and Non- Small Cell Lung Cancer (NSCLC) often exhibit intrinsic response to BET inhibition, showing limited therapeutic response at the outset of treatment; while Triple-Negative Breast Cancer (TNBC) and Acute Myeloid Leukemia (AML) initially respond to BETi but rapidly develop acquired resistance within six months.

To date, significant progress has been made in elucidating the mechanisms underlying resistance to BET inhibitors (BETi). In CRPC, somatic mutations in the SPOP gene impair the ubiquitin degradation system, leading to BRD4 upregulation and conferring inherent resistance to BETi. Our previous work provided further insight into non-mutation-driven inherent BETi resistance in NSCLC, where non-selective inhibition of BRD3 by BET inhibitors unexpectedly activates the oncogene BCL6 to enhance mTOR pathway-mediated cell proliferation. In consistent with the oncogenic roles of BCL6, blocking such stress-related signaling by using BCL6 inhibitors or mTOR inhibitors achieved robust synergistic effects with BET inhibitors. These findings further support an effective combinatorial strategy with potential translatability to sensitize NSCLC to clinical BETi by concurrent inhibition of either BCL6 or mTOR. We believe our findings are of paramount importance, considering that clinical trials of BETi are now being conducted in intractable cancers with limited effectiveness in patients. Additionally, although recent studies revealed that distinguishing BD1 and BD2 of the BET proteins may guide future BET-targeted therapies our findings suggest that development of a BRD4-specific inhibitor may be a pivotal therapeutic option for patients with KRAS-mutant NSCLC, as selective inhibition of BRD4 avoids BCL6 upregulation.

However, in contrast to NSCLCs that exhibit poor response to BET inhibition at the beginning of treatment, TNBC trends to be sensitive to BET inhibition but rapidly develops acquired drug resistance in six months. The underlying mechanisms of acquired BETi resistance is not fully elucidated. In this study, we identified the RNA-binding protein IGF2BP2 as a key driver of acquired BETi resistance in TNBC, primarily through its role in enhancing the translation of c-MYC mRNA. Given that IGF2BP2 is not an ideal target for small-molecular drugs, we

performed RNA immunoprecipitation sequencing (RIP-Seq) and discovered circRNA-BISC as a potent IGF2BP2 repressor. BISC effectively inhibited both c-MYC translation and BETi resistance. Notably, BISC contains a 'CAC-linker-XGGX' motif that specifically binds IGF2BP2 without interactions with IGF2BP1 and IGF2BP3. The efficacy and selectivity of BISC in targeting IGF2BP2 prompted further exploration of BISC-based RNA therapeutics for TNBC. In vitro transcribed and circularized BISC, when combined with the BET inhibitor OTX-015, demonstrated impressive tumor regression in BETi-resistant TNBC models without detectable toxicity. These findings establish BISC as a potent IGF2BP2 repressor and highlight the feasibility of circRNA-based therapeutic strategies to overcome BETi resistance in TNBC.

Biography

Dr. Guo studied Pharmaceutics at the China Pharmaceutic University and graduated as MS in 2012. He then joined the research group of Prof. Mingyao Liu at the School of Life Sciences, East China Normal University (SLS-ECNU). He received his PhD degree in 2017 at the same institution. After five-year postdoctoral fellowship supervised by Prof. Yong Peng at the Center for Molecular Oncology (West China Hospital, Sichuan University), Jiawei he obtained the position of a research associate at the same institution. He has published more than five research articles on the topic of drug resistance.

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The next generation cancer treatments-the role of genomics and AI

The discussion will be on optimum utilization of AI and genomics in delivering personalized treatments in Radiation Oncology for better treatment outcome. Artificial Intellignce, Machine learing and Deep learning will be discussed and the future of artificial intelligence in radiation oncology. AI can be used to predict a patient's response to radiation therapy by analyzing imaging and clinical data. This information can be used to adapt treatment plans in real-time, ensuring that patients receive the most effective and tailored care throughout their treatment course. AI can help enhance quality assurance processes in radiation therapy by automating the verification of treatment plans and the monitoring of treatment delivery. This reduces the risk of errors and ensures that treatments adhere to safety standards. AI-powered image analysis can improve the accuracy of tumor localization and patient positioning during treatment. AI can also assist in real-time tracking of organ motion, enabling more precise and targeted radiation delivery. Other important applications like Dose monitoring and reporting, radiomics, Predictive modeling and automated contouring will be discussed.

The main hurdle for better treatments in radiation oncology is tumour response to the given prescribed dose in each individual due to various factors. Out of so many factors, genetic alterations and treatment resistance tumours are considered the most important ones to consider for better treatment management. Next generation sequence has been developed for detection of DNA-based alterations epigenome, transcriptome or Nome molecular aberrations. Individual human genome can now be sequenced in less than 2 weeks. known oncogenic function can also be identified with NGS. Depending on the molecular profile of their tumors it is possible to prospectively identify patients who are more likely to benefit from radiotherapy. Personalized radiotherapy could be achieved by establishing biomarkers which can classify radio sensitive and resistant tumors before initiation of treatment. Next- generation sequencing (NGS)- Advantages and applications and how NGS can change the radiotherapy will be discussed. Combination of AI and ML with genomics will be discussed. AI and ML is to improve our understanding of hidden patterns in large and complex genomics data sets. AI and genome together can influence the treatments by identifying the genetic disorders, identifying primary kind of cancer etc.

Keywords: Radiation oncology, NGS, Biomarkers, Personalized treatments, AI, ML.

Biography

Dr. K R Muralidhar studied Radiological Physics at the Bhabha Atomic Research Center, India in 1996 and post graduated in MSc Tech in 1995. He then joined the Manipal Hospitals as Medical Physicist. He received his PhD degree in 2008. He worked in various institute like Indo-American Cancer Hospital and American Oncology Institute as Chief Medical Physicist. Now he is working as Director of Physics in Karkinos Healthcare. He also completed Master of Health Administration and Independent Director from Indian Institute of corporate affairs. He has more than 80 publications and presentations including IAEA, AAPM etc.

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The prevention of post-ESD stricture of large early esophageal cancer by periodic triamcinolone injection

Objective: Endoscopic Submucosal Dissection (ESD) is widely used for early esophageal cancer, but the post-ESD stricture rate of patients with a post-ESD mucosal defect of ≥3/4 of the esophageal circumference is extremely high (even reach 90%).Long term repeated endoscopic dilation is required for these patients, whose life quality is significantly reduced. There are many methods and reports for preventing esophageal post-ESD stricture, but the effectiveness is unclear and no standardized prevention method has been established yet. The aim of this study is to investigate the efficacy and safety evaluation of periodic steroid injections in preventing post-ESD stricture for large (diameter ≥ 3/4 circumference) early esophageal cancer, as well as to determine the appropriate number of local injections for preventing stricture.

Method: This study included 45 patients with early-stage esophageal cancer with tumors that had at least 3/4 or the whole circumference. They were divided into an observation group (12 cases with lesions ≥3/4 circumference; 6 cases with circumferential lesions) and the control group (21 cases with lesions ≥3/4 circumference;6 cases with circumferential lesions). ESD was performed for the obove patients. The observation group received 50-100mg of triamcinolone acetonide injection into the submucosal layer of the wound after ESD immediately, followed by endoscopic and injection of triamcinolone acetonide under endoscopy every 7-10 days after ESD. The injection endpoint was complete repair of squamous epithelium ≥1/3 circumference; The control group only received injection of triamcinolone acetonide into the submucosal layer of the wound on the day of ESD. During follow-up, if endoscopy (diameter≥8.9mm) failed, esophageal stricture was determined and endoscopic (balloon/probe) dilation was performed with local injection of triamcinolone acetonide. The observation indicators were the rate of esophageal stricture after injection of triamcinolone acetonide after ESD, the number of endoscopic dilations after stricture, and the incidence of adverse events.

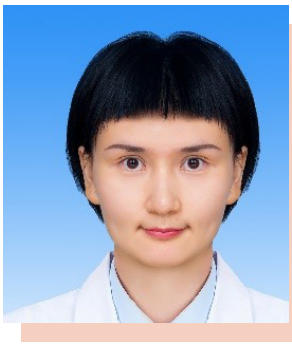
Result: Compared with the control group, the observation group showed a significant reduction in stricture rate (11.1%, 2/18; control group: 48%, 13/27 cases; χ^2=6.667a, P=0.01), and a decrease in the number of dilations after stricture (median 3.5, range 2-5; median 6, range 3-17 in the control group),; The stricture rate in the observation group (16.7%, 1/6) was significantly lower than that in the control group (100%, P=0.015; In the study, the median number of injections of Amphotericin was observed to be 5 times (range 3-9 times); During the follow-up period of the observation group, one case of delayed perforation adverse event occurred, with

a complication rate of 5.6% (1/18).The control group had a complication rate of 11% (3/27), both of which were caused by repeated dilation. One patient developed deep muscle layer tear, and two patients developed esophageal perforation.

Conclusion: The incidence of stricture after ESD surgery for large esophageal cancer is high. Periodic injection of triamcinolone acetonide can prevent post-ESD stricture in large early esophageal cancer, especially in circumferential lesions, Which is safe and easy to implement.

Biography

Dr. LI-Mian Er is a Digestive endoscopist at The Fourth Hospital of Hebei Medical University. She received her M.D degree in 2017 at the same institution. She specializes in endoscopic diagnosis, Minimally invasive treatment for esophageal and Gastrointestinal precancerous lesions. She do some research on Endoscopic diagnosis and treatment in esophageal and Gastrointestinal precancerous lesions, pathogenesis of esophageal and Gastrointestinal tumors. She has published more than 20 research articles in SCI (E) journals.



Liling Jiang

Shapingba Hospital affiliated to Chongqing University (Shapingba District People's Hospital of Chongqing), China

Application of magnetic resonance-diffusion weighted imaging in thyroid nodules

All studies were performed on a 3 T whole-body MRI system (MAGNETOM Prisma, Siemens Healthineers, Erlangen, Germany) using a 16-channel surface coil (Zhongzhi Medical, Jiangsu). Firstly, we evaluated the thyroid nodule image quality between two Diffusion Weighted Imaging (DWI) technique, which were the reduced field of view technique Diffusion-Weighted Imaging (rFOV-DWI) and simultaneous multislice readout segmentation of long variable echo-trains-DWI (SMS-RESOLVE-DWI). This study was performed at b values of 0s/mm2 and 1000s/mm2. Study result suggested that rFOV-DWI exhibited better image quality than SMS-RESOLVE-DWI and similar performance in differential diagnosis. Secondly, the optimal b-value for rFOV-DWI was explored. This study was performed at b values of 0 s/mm2, 500 s/mm2, 1000 s/mm2 and 1500s/mm2.We combined image quality and differential diagnostic performance to determine the optimal b-value for rFOV-DWI detection of benign and malignant nodules. This study suggests that 1500 s/mm2 may be a suitable b-value to differentiate benign and malignant thyroid nodules in ZOOMit-DWI images, which yielded better image quality and comparable diagnostic effectiveness among the different b-value. Lastly, we compared the diagnostic performance of Intravoxel Incoherent Motion (IVIM) and Diffusion Kurtosis Imaging (DKI) in thyroid nodules. IVIM and DKI were performed using the rFOV-DWI technique. Study result suggested that IVIM and DKI were alternative for each other in in differentiating malignant from benign thyroid nodules. The D of IVIM-derived parameters and Dapp of DKI-derived parameter were correlated to the Ki-67 expression.

Biography

Liling Jiang studied Imaging and Nuclear Medicine at the Chongqing Medical University, graduated as MS in 2016. She is an expert in the field of radiology who performs practice in the differential diagnosis of cancer. She is interested in the precise diagnosis of thyroid cancer. She is passionate about reducing suffering and improving life quality of cancer patients. She has published 4 peer-reviewed scientific articles in SCI (E) journals. She serves on the editorial board of Frontiers in Oncology.

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Bioscope: A new instrumental approach for early diagnosis of oncological diseases

Modern medical methods of treatment allow in some cases to prevent the development of pathological processes in the human body. It is known that the effectiveness of such therapeutic effects is largely determined by the possibility of diagnosing the disease at the initial stages of its development. In particular, statistics show that when cancer is detected at an early stage, the probability of cure is 90-95%. In later stages, this figure drops to 50%, and in the third and fourth stages, the probability of cure is only 10-12%.

The foregoing makes it relevant to search for new and more sensitive instrumental approaches to the formation of oncological formations. In this regard, the “Bioscope” hardware complex developed in our laboratory is of particular interest.

The device is simple in design, and the principle of its operation is based on the estimation of the intensity of light scattered in an opaque chamber from a glass plate covered with a thin opaque material (e.g.black rough paper).

Different biological objects located at some distance from the device affect the readings of the "Bioscope" in varying degrees; at the same time, the signals of the equipment also change when the physiological state of the studied system changes. This indicates the possibility of using the "Bioscope" to assess the state of the biological system in various biomedical studies.

In experiments with artificial infection of mice with skin cancer, it was shown that already one day after lethal infection with skin cancer, a pronounced "cancer peak" is formed in the spectral distribution of the "Bioscope" signals, which remains until the death of the mice. In mice that were also infected with skin cancer, but did not die, the "cancer peak" was practically not formed.

At the same time, in experiments with people, an interesting fact was revealed: the patient complained of discomfort in the area of the right mammary gland. Mammographic examination did not reveal any pathology. Nevertheless, the "Bioscope" signals indicated a sharp difference in the states of the right and left mammary glands. After 2 months, using conventional instrumental tools, the onset of neoplasm development in the right mammary gland was confirmed.

The presented results indicate, that after testing on a larger number of patients, the "Bioscope" device create prerequisites for its use as a new diagnostic tool and for early detection of the onset of oncological diseases.

Biography

Luiza Simonyan was born on April 21, 1980, in Vanadzor, Republic of Armenia. She holds Armenian citizenship and currently serves as a researcher and postdoctoral fellow. Luiza obtained her PhD and is affiliated with the Institute of Physiology at the National Academy of Sciences of Armenia. Throughout her career, she has contributed significantly to her field, with 40 published research papers.

Marie Fusella Giuntini[1*], Cyril Voyant[2], Dominique Barbolosi[3], David Taieb[4]

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Developing a digital decision-support tool for personalized treatment of metastatic thyroid cancer

Each year, a high number of new cases of thyroid cancer are identified. Although this disease has a good prognosis, it is often immediately metastatic, which requires removal of the thyroid body, followed by iratherapy (administration of iodine 131) in order to eradicate the various metastatic sites present.

Until now, iodine 131 administration protocols (activities of iodine 131 to be administered, number of iratherapy sessions and interval between two consecutive sessions) are conducted empirically and a large inter-individual variability in responses to treatment has been observed as well as toxicities induced in the more or less long term. In order to optimize the effectiveness-toxicity balance, it is essential to provide clinicians with a decision support tool allowing them to perform in silico simulations of the evolution of thyroglobulin concentrations depending on the chosen diode 131 administration protocol by the therapist.

An in silico simulator will be presented built from a mathematical model managed by a set of parameters whose values are specific to each individual, in particular the parameter (Td) of the doubling time of tumor cells under treatment is identified as a parameter key allowing discrimination from the first weeks of treatment, responding patients and patients refractory to iodine 131. In addition, in the case of a given responding patient, the simulator then makes it possible to propose to the clinician a diagram effective administration while using the minimum amount of iodine 131, so as to minimize the probability of the appearance of possible iatrogenic pathologies induced by excessive irradiation.

The precision of this personalized therapeutic planning simulator is conditioned by a good estimation of the individual parameters of each patient, in particular the Td parameter. In this sense, the use of Artificial Intelligence could help to refine the estimation of these parameters.

Additionally, this work provides proof of concept that the development of powerful digital tools could help enhance the precision of personalized medicine.

Biography

Marie Fusella Giuntini Graduated in mathematics and computer science from the University of Corsica, Corte-France. A Certified teacher in Mathematics, she has been teaching applied mathematics and computer science for 20 years. She was Director of Studies for several university programs for 15 years before enrolling in a thesis

in 2022. Currently a 3rd year doctoral student in the applied mathematics section, her thesis supervision is: Dominique Barbolosi, Professor at the University of Aix-Marseille-France, Laurent Capocchi, Professor at the University of Corsica, Corte-France and Cyril Voyant, Research Director at Mines Paris-PSL (OIE)-France. Her research is centered on the application of mathematics to health.

Maurice T. Agonnoude MD, MPH, PhD[1*], Dalilath Sakpata[1], Luc-Valère Codjobrun[2]

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Associated factors to practice of breast cancer screening among women in Parakou at 2021

Introduction: The integration of breast cancer screening into daily clinical care leads to an early diagnosis and reduces its mortality. However, many women are reluctant and do not get tested. Objective To study the factors associated with the Practice of Breast Cancer Screening (PBCS) among women in Parakou in 2021.

Materials and Methods: This was a cross-sectional observational study with an analytical aim concerning adult women living in Parakou, selected by the random cluster survey. The dependent variable was the PBCS; the independent variables were individual, sociocultural and organizational. The data collection was done by interview using a questionnaire. The prevalence ratio with its confidence interval was used to search for associations.

Results: The prevalence of PBCS among the 375 participants was 55.5% (CI95%=[50,0%-61,0%]). The decision-making power in health care matters, held by the parents (p=0.006) was unfavorable to the PBCS while the level of primary (p=0.036) and secondary education (p=0.001), knowledge of screening methods (p=0.007), information on screening programs and projects (p=0.020) and participation in these programs (p=0.009), satisfactory assessment of the availability of nursing staff (p=0.003) were unfavorable for PBCS.

Conclusion: Actions are needed to promote and increase women's participation in breast cancer screening.

Keywords: Screening, Breast Cancer, Practice, Women, Parakou.

Biography

Maurice T. Agonnoude is a medical specialist in public health, a teaching researcher, and an associate lecturer at CAMES universities in public health (organization and management of health systems). His research interests concern public health and more specifically issues of management, organization and evaluation of health policies and systems. He is also particularly interested in health promotion and frontline health systems.

Dr. Namrata P Awasthi

Professor Department of Pathology, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, UP, India

Flow cytometry for diagnosing malignant carcinomatous effusions

Background: The lecture will introduce the audience to commonly utilized techniques for Cytology of malignant fluids including cytospin, cell block and immunohistochemistry. The advantages and limitations of the techniques will be discussed. Further, basics of flow cytometry (FCM) and application of FCM for detecting malignant epithelial cells will be presented through a research study conducted on 96 serous fluids.

Method: FCM assessment was performed by using epithelial cell adhesion molecule (EpCAM) and MUC-1 (in a subgroup of cases) as epithelial markers and CD45 and CD14 as leucocyte markers. The percentage of EpCAM positivity and MUC-1 positivity was calculated in the CD14 and CD45 dual negative population by selective gating. The findings were then correlated with the defined gold standard criteria. FCM was performed on a 3 laser 10 colour flow cytometer equipped with acquisition and analysis software.

Results: The sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and diagnostic accuracy for EpCAM was determined to be 92.06%, 96.96%, 98.31%, 86.48%, and 93.75%, respectively, while that for MUC-1 the sensitivity and specificity were slightly lower at 79.16% and 93.75%. Combining FCM with cytomorphology significantly enhanced these metrics, resulting in sensitivity, specificity, PPV, NPV, and diagnostic accuracy of 95.3%, 100%, 100%, 91.4%, and 96.8%, respectively.

Conclusions: The study highlighted the importance of multicolored flowcytometric analysis in detecting epithelial malignancies in effusions specially in cases belonging to the atypia of undetermined significance and suspicious for malignancy categories and in cases with strong clinical suspicion of malignancy with negative fluid cytology. We recommend the combined use of FCM and cytology for this specific subgroup of patients in routine clinical practice for fast and accurate reporting.

Biography

Dr. Namrata is presently Professor in Department of Pathology and In-charge Lab Hematology & Flow Cytometry Division at RMLIMS, Lucknow. Research areas of interest include Hemophilia and inhibitors, FNA-Flow cytometry and non-hematological applications of flow cytometry like assessment of DNA damage by Gamma H2AX measurement, Circulating tumor cell detection in Gall bladder Cancer, CD64 and HLADR in Sepsis and COVID 19, detection of malignant epithelial cells in effusion fluids. She has more than 50 publications in national and international peer reviewed pubmed/scopus indexed journals. She is a recipient of 6 awards in international and national conferences.

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Breaking the silence: Breast cancer extracellular vesicles as key players in breast cancer immune suppression

Breast cancer is the most prevalent malignancy in women worldwide and remains a leading cause of cancer-related mortality. Immune system controls tumor progression but is often suppressed in breast cancer. Dendritic Cells (DCs), essential for linking innate and adaptive immunity, frequently exhibit impaired functionality. Emerging evidence highlights the significant role of Extracelular Vesicles (EVs) in shaping the immune microenvironment. These vesicles have the ability to carry many kinds of bioactive molecules from proteins, lipids to microRNAs capable of modulating immune cell functions. Here we explore the participation of EVs as well as a putative mechanism during dendritic cell modulation emphasizing their impact on immune regulation and cancer progression. EVs were isolated from the plasma of breast cancer patients and healthy donors by ultracentrifugation. Characterization of exosomes were performed by western blotting, transmission electron microscopy and nanoparticle tracking analysis. Monocyte-derived DCs (Mo DCs) from healthy donors were differentiated in DC the presence of Breast Cancer Exosomes (BC EVs) or healthy donor exosomes (HD-EVs). Phenotypic and functional analyses were performed using flow cytometry, cytokine quantification, and mixed lymphocyte reactions (MLR). The expression of miR-181b and its target, GSK3β-Interacting Protein (GSKIP), was assessed using qPCR and Western blot. Inhibition of miR-181b was performed to evaluate its role in DC modulation. Mo-DCs exposed to BC-EVs showed maturation arrest with decreased expression of HLA-DR, CD80, CD86, CD11c and increased PD-L1. These cells impaired CD4+ and CD8+ T cell proliferation and expression of pro-inflammatory cytokines. BC-EV/Mo-DCs also induced the differentiation of regulatory T lymphocytes (Tregs), higher expression of IL-10 and PD-1 by T cells. We demonstrated that BC-EVs enriched for miR-181b-5p inhibited expression of GSK3β Interaction Protein (GSKIP) arresting GSK3b inactivation and preventing mTOR activation, switches necessary for DC maturation. Inhibition of miR-181b with specific siRNA restored BC-EV/Mo-DCs pro- inflammatory phenotype highlighting its role in the tolerogenic phenotype. Analysis of BC patient survival revealed that high miR-181b expression correlated with poor prognosis, whereas higher GSKIP levels were associated with better outcomes. BC-EVs enriched with miR-181b induce tolerogenic DCs by modulating the GSK3β/mTOR axis, contributing to immune evasion and cancer progression. Targeting miR-181b represents a

promising strategy to restore DC functionality and improve immunotherapeutic outcomes in breast cancer. Our findings provide important insights into the physiopathology of cancer and immune evasion, which could have implications for cancer prognosis and the development of effective immunotherapy approaches for breast cancer.

Biography

Nayara is associated professor in Federal University of Sao Joao del Rei, Brazil, since 2012 and have expertise in cancer, non-coding RNAs, exosomes, dendritic cells, glycobiology, tumoral evasion and immunotherapy. Nayara studied at the State University of Londrina, where she obtained a degree in Biochemistry and a specialist title in Applied Health Science. The field of oncology was what really motivated her formation in Biochemistry (PhD from FMRP/USP in 2008) and Tumor Immunology (postdoctoral degree from the New University of Lisbon in 2019) fields that allow her to pursue her scientific research career with a special focus on Cancer Immunotherapy.

Oleg Bukhtoyarov*, Denis Samarin

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System error in cancer research

Undoubtedly, cancer refers to the group called “diseases of civilization”. Over the last 100 years, cancer has become a real nightmare for humanity. Despite having the tremendous world scientific and financial resources, successes in the cancer research and treatment, the cancer incidence and mortality are growing worldwide with an extremely unfavourable prognosis. The basis of “diseases of civilization” is chronic stress. A person has found himself in such mental state as a modern civilization model has been developing.

Chronic stress and its extensive damaging effect on the body are well-known: ischemia, inflammation, oxidative-nitrosative stress, etc. They trigger “diseases of civilization”, which in combination with the DNA damages cells and antitumor immunity inhibition are directly related to the pathogenesis of cancer. In a state of chronic stress, the human body is able to develop cancer without exogenous carcinogens.

Cancer is a genetic disease, however currently there are no methods of successful correction of genetic defects, that caused cancer. They are associated with the hope of cancer control. Simultaneously, clinical oncology is focused on the destruction of cancer cells (surgery, chemo-, radiotherapy). Obviously, the modern look of scientific and clinical oncology is concentrated exclusively on genes, molecules, cells. This is precisely the system error in cancer research.

Let us explain. There are 8 biological levels of organization of living matter: 1-molecular-genetic, 2-cellular, 3-tissue, 4-organ, 5-organismal, 6-population, 7-biogenocynotic, 8-biospheric. The main studies of the reason of cancer are carried out on the 1 level, which is a border between living and inanimate, and at the 2 level, where a cell is a structural-functional unit of living. Nevertheless, a holistic vision of a cancer patient is possible only from the 5-organismal level in order to “be treating the person and not the disease” as Hippocrates bequeathed.

It’s impossible to understand the organismal level, if not to see the major integral, control system in the body - the nervous system, specifically, the central nervous system, the higher nervous activity. It is simply impossible to see a cancer patient from the genes, molecules and cells. The same thing is studying broken bricks in detail, while thinking that you are studying a huge building with all life support systems, apartments, residents, etc.

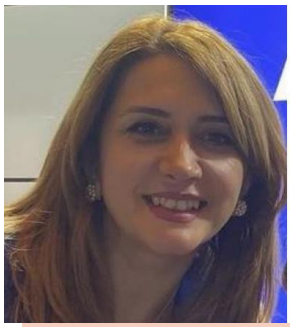
Currently, scientific oncology cannot escape beyond the cell study due to the lack of exact methods for measuring the state of the nervous system. Paradoxically, there is a concept of chronic stress, but there is no method of its accurate measurement (until recently). It is crucial to repeat that the reason of system error in cancer research is the focus on thinking about

molecular-genetic and cellular processes. This explains the past, current and future failures in the creation of seemingly promising antitumor drugs and therapeutic vaccines.

Our clinicals experience shows that serious success in managing the cancer process should be expected from a systemic approach, effective impact on the two main regulatory systems of the body - the nervous and immune, but not only from the use of any cancer drugs.

Biography

Oleg Bukhtoyarov graduated from the Medical Military Academy in Sankt Petersburg (formerly Leningrad, USSR) in 1990 and served as a physician on strategic "Typhoon" class nuclearpowered submarines. He received his PhD degree in psychiatry in 1997 and Doctor of Science degree in Clinical immunology and Medical psychology from the Medical Military Academy in 2012. Founder of the concepts: "stressful carcinogenesis", "cancer reparative trap", "life purpose dominant". Dr. Bukhtoyarov for the past 23 years has been focusing his research on the psychoimmunology of cancer. Owner and chief physician of Medical Center 39 LLC, member of the Russian Society of Immunology.



Özlem Terzi

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Association between respiratory tract viral PCR panel results and clinical outcomes in children with cancer and febrile neutropenia

Febrile Neutropenia (FEN) is the most common life-threatening complication of childhood cancer. Early initiation of broad-spectrum intravenous antibiotics to patients has reduced the mortality of FEN below 5%. Studies have reported viral infections to constitute 35 to 55% of FEN episodes in pediatric patients. In healthy children, these viruses usually cause upper respiratory tract infections, with a self- limited course. However, they can lead to significant morbidity and mortality in immunocompromised patients. It has also been shown that viral respiratory tract infections are more common in cancer patients than in immunocompetent patients. Up to around half of these infections are of viral origin, but viral infections are often difficult to distinguish from bacterial infections. With multiplex real-time Polymerase Chain Reaction (PCR), the diagnosis of respiratory viruses has increased by 30 to 50%. Respiratory tract viruses were found to be positive in between 38 and 76% in children with cancer and FEN when using PCR. In five studies performed on patients with respiratory tract complaints and findings, the PCR positivity rate was between 34 and 76%. These rates ranged from 34 to 93.75% in five studies where the patients had presented with FEN. PCR positivity was reported as 35 and 78% in the two studies including patients with a positive focus of infection but only 22% in the study conducted in patients presenting with fever. PCR positivity ranged from 45 to 68% in three studies with respiratory tract complaints and an episode of FEN. It should be noted that a positive RVP is not necessarily associated with a clinically significant systemic RVI. Previous research has shown that respiratory viral shedding can occur in asymptomatic patients for up to 6 weeks following RVI. In the literature, the rate of viral coinfection varied between 19.5 and 55%. Torres et al reported that the high prevalence of febrile RVIs contributed to the increase in the rate of FEN in pediatric patients with cancer. Krause et al showed that RVIs were associated with treatment delays and increased frequency of hospitalization in pediatric cancer patients. It was reported that the severity of the infection was low in PCR-positive cases. In two different studies, it was reported that patients with only PCR positivity but without blood culture positivity had shorter hospital LOS and lower PICU admission rates. It should be borne in mind that frequency of viral type detected will change seasonally. Another reason for requesting RVP in a patient presenting with FEN would be to aid in selection of drugs to be added to the treatment. However, given that having a positive RVP does not affect LOS or disease severity, the question remains whether RVP testing contributes to clinical management in this population. Future research should also investigate if RVP positivity affects clinician decision-making or parental reassurance at discharge of pediatric patients with cancer and with FEN.

Biography

Dr. Özlem Terzi, after graduating from Uludag University Faculty of Medicine, Prof. Dr. Ozlem Terzi became a pediatric specialist at Prof. Dr. Cemil Tascıoglu City Hospital. She later became a Pediatric Hematology and Oncology Specialist at Basaksehir Cam and Sakura City Hospital. Currently she works at Bakırkoy Dr. Sadi Konuk Training and Research Hospital in the Pediatric Hematology and Oncology Clinic, where she is the founding physician. She is also a lecturer at Demiroglu Bilim University Faculty of Medicine and Halic University Faculty of Medicine, teaching Pediatric Hematology and Oncology courses.

Patricia Tai[1*], Aoife Jones Thachuthara[2], Kurian Joseph[3], Francisco Perera[4], Vimal H Prajapati[5], Edward Yu[4]

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Superficial radiation ulcer of the skin: Comparison of the innovative photodynamic therapy (used in the first documented Canadian case) with other alternatives

Introduction: Photodynamic Therapy (PDT) promotes wound healing while its clinical use is not investigated in human for radiation-induced skin ulcers.

Methods and Materials: We documented the first Canadian in-human case and searched the PubMed literature using “PDT" AND "radiation" AND "ulcer" terms.

Results: PDT has been used on premalignant and malignant skin cancers for years. Our patient had a radiation-induced refractory post-mastectomy radiotherapy chest wall ulcer of 5-year duration, published in the Cureus journal in August 2024. There are 6 laboratory reports (total 95 rats) in the literature with an overall efficacy of 90%. We documented ulcer healing at 3-month follow-up with gradual improvement to total healing by 14 months. The topically applied 5-aminolevulinic acid (5-ALA) was activated by red light of wavelength 630 nm after an incubation time of 5 hours. The median incubation time, as the body tissue takes up 5-ALA, was 4 (range: 2-18) hours after topical application in the literature. We used three 30-minute treatments at month 0, 1, 5 months, which can vary depending on treatment response. No complication developed. Currently large PDT Canadian centers for malignant cutaneous, esophageal and bronchial lesions are located in Calgary, Hamilton, Kingston, Toronto, Ottawa and Montreal.

Comparing with Hyperbaric Oxygen Therapy (HBOT), a well-known standard of care treatment for radiation-induced skin ulcers, PDT is non-invasive and safe with few complications, e.g., skin irritation/swelling (rarely requiring steroid treatment), photosensitivity and retinal damage. PDT has a lower cost: 5-ALA costs only CAN$500/session for Metvix (methyl 5-ALA, currently approved by Health Canada) vs HBOT requiring 30 sessions with a total cost of $15,000 in the Ontario province of Canada. PDT procedure is simple and safe. Hence, PDT providers can be nurse practitioners, family doctors, or specialists (radiation oncologists, general surgeons), unlike HBO requiring subspecialists with special expertise with higher overhead and hence higher billing costs to the health care system. Besides, HBO has risks of hearing damage, pain in the ear/sinus/tooth, claustrophobia, fatigue, temporary near-sightedness and hypoglycemia in diabetics.

Other alternatives for wound healing are (1) pentoxifylline, vitamin E (both non-invasive and easily administered, with track record for safety); (2) mesenchymal stem cells and interferon-gamma, growth factors (still early in development); and (3) surgical debridement/grafting/flap coverage (invasive, labor-intensive, costly, requiring experience, with waiting list due to operation room staff shortage in some countries).

Conclusions: Laboratory publications substantiate the efficacy of PDT on radiation-induced ulcer healing. The first Canadian clinical case was documented by us in Ontario. It is cost-effective for superficial radiation-induced ulcers. Further clinical studies are warranted to evaluate the optimal treatment schema and effects on quality of life. Hopefully it can be widely available with costs covered by the government.

Biography

Professor Patricia Tai graduated with a gold medal from U. of Hong Kong in 1984. Since then she became an experienced clinical oncologist with expertise in skin and urologic cancers. She is one of the international experts on Merkel cell carcinoma, the author in UpToDate since 2000. Being the author of 149 full publications and 167 abstracts, honorary Professor of University of Hong Kong and clinical professor of U. Saskatchewan, Canada, she now works with UpToDate and welcomes collaborations on photodynamic therapy, Merkel cell carcinoma and prostate cancer.

Pauline Picho Keronyai*, Jean Marion Apiny, Namuyanja Rachael

Nama Wellness Community Centre (NAWEC)/Uganda Cancer Society (UCS), Kampala, Uganda

Community health workers' role in advancing cervical cancer prevention and HPV vaccination in Mukono district, Uganda

Cervical cancer remains a significant public health challenge in Africa, with the continent bearing approximately 19% of global cases. The East African region reports particularly concerning statistics, with an age-standardized incidence rate of 40.1 per 100,000 women annually, significantly higher than the global average of 13.3 per 100,000 (Globocan, 2020). In Uganda, cervical cancer is the most common cancer among women of reproductive age, with approximately 6,413 new cases diagnosed annually and an age-standardized incidence rate of 54.3 per 100,000 women (UCI Annual Report, 2021). The high mortality rate is largely attributed to late presentation, with about 80% of cases diagnosed at advanced stages.

In Mukono District, like other rural and peri-urban areas in Uganda, access to screening services remains a significant challenge, with only about 30% of eligible women having ever been screened for cervical cancer (Mukono District Health Report, 2022). These statistics underscore the critical importance of community- based interventions and the role of Community Health Workers in expanding access to essential cancer prevention services, particularly in resource-limited settings.

NAWEC, an NGO in Mukono District, Uganda, transforms community health through 306 professionalized Community Health Workers (proCHWs). Since 2014, it has served over 262,256 clients through comprehensive healthcare programs including integrated Community Case Management (iCCM), Water Sanitation and Hygiene, mental health, and clinical outreaches, working towards equitable healthcare access for all.

In Uganda, where cervical cancer remains a significant public health concern, Community Health Workers (CHWs) have emerged as crucial frontline workers in cancer prevention efforts. This study examines the impact of CHW-supported cervical cancer prevention programs in Mukono District and surrounding areas. Through a comprehensive community-based approach, CHWs facilitated the screening of 7,406 women for cervical cancer, resulting in the identification and treatment of 266 positive cases through thermocoagulation. Notably, the program successfully integrated HIV services in 2024 with cancer screening, reaching 43 HIV- positive clients for cervical cancer screening, demonstrating the effectiveness of service integration. The CHW-led HPV vaccination campaign achieved significant coverage, with 6,962 girls 10 year of age receiving their first dose (HPV1) and 2,222 completing their second dose (HPV2). Additionally, CHWs contributed to increased family planning uptake, serving 28,139 clients, which complemented their cancer prevention efforts. These results highlight the vital

role of professionalized CHWs in expanding access to essential cancer prevention services and underscore their potential in strengthening health systems to achieve comprehensive women's health care in resource-limited settings.

Biography

Pauline Picho Keronyai serves as Executive Director of Nama Wellness Community Centre (NAWEC) in Mukono District, Uganda, since 2015. She holds a Master's in Public Health from Uganda Christian University, is pursuing a Ph.D. in Public Health, and completed the Global Health Delivery Intensive program at Harvard T.H. Chan School of Public Health. With over 15 years of healthcare experience, Pauline has transformed NAWEC, training 100 professional Community Health Workers and establishing a successful Health Centre III. Previously, she managed HIV/TB programs at Reach Out Mbuya Community Health Initiative. She actively participates in the Uganda Cancer Society and Uganda Red Cross Society.

Dr. Phyu Ou Maung[1*], Aung Kyaw Kyaw[2], Thet Thet Mar[2], Bo Bo Aung[3], Khin Maung[4], Sann Win[4], Win Pa Pa Aung[4], Myint Shwe[1]

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Survivin and its splicing isoforms gene expression in oral squamous cell carcinoma patients

Background: Oral Squamous Cell Carcinoma (OSCC) has become a major oncologic problem. Survivin overexpression in OSCC could be associated with increased aggressiveness, poor response to chemotherapy and prediction of patient survival. This study was conducted to analyze the mRNA expression of Survivin and its splicing isoforms in OSCC patients.

Materials and Methods: This was cross-sectional analytical study during 2022-2024. Thirty histologically proven newly diagnosed OSCC patients came to University of Dental Medicine, Mandalay were collected. Clinical tumor and nodal stages were verified by contrast-enchanced computed topography. Histopathological grading were confirmed by Bryne's grading system. Primary tumor tissues and adjacent normal oral mucosa from the same patients were collected, preserved in RNAlater at -80^{0}C freezer and analysed determinination of mRNA level of Survivin and its 4 isoforms (2α, 2B, 3B and ΔEx3) by semi-quantitative Reverse Transcription-Polymerase Chain Reaction (qRT-PCR).

Results: The highest frequency age group for OSCC was 41- 60 years (53%). Male and female ration was 2.3:1. Total 24/30, 80% were low grade and 6/30 cases (20%), high grade. The expression of ΔEx3 was the highest level (mean Ct=34.57) followed by 3B (34.54), Survivin (32.01) and 2α (31.82) in OSCC tissues. The differences of the mRNA expression of Survivin and its splicing isoforms between normal mucosa and OSCC tissues resected from OSCC patients were statistically significant ($p<0.05$). There were statistically significant upregulation of the expression of Survivin, 3B and ΔEx3 in advanced stage ($p<0.05$) and 2α was significantly more upregulated in early staged compared to advanced stage ($p<0.05$). There were not statistically significant difference in the expression of Survivin and its isoforms in relation to Bryne's Grading System among OSCC patients ($p>0.05$). The Survivin, 2α, 3B and ΔEx3 isoform was significantly overexpressed among tumor areas compared to normal tissue in 87%, 57%, 77% and 93% of OSCC with fold changes of 3.01, 1.74, 4.55 and 4.01 ($2^{-\Delta\Delta Ct}$) relative units respectively ($p<0.05$).

Conclusion: Survivin and its splicing isoforms are highly expressed in OSCC and expression of different level of isoforms play an important role in tumor aggressiveness and predict the prognosis of OSCC by implying with clinical staging and histopathological grading in OSCC.

Keywords: OSCC, Oral Squamous Cell Carcinoma, Bryne's Grading, Survivin, RT-PCR.

Biography

Dr. Phyu Ou Maung studied dentistry at University of Dental Medicine, Mandalay, Myanmar (UDMM) and graduated as Bachelor of Dental Surgeon (BDS) in 2010. In 2011, she joined to the cancer research projects with Prof. Dr. Anahid Jewett at Jane and Jerry Weintraub Center and received Master of Science (Oral Biology) from University of California, Los Angeles (UCLA), School of Dentistry in 2014. She attained the highest research grant for "Systemic Bone Density and Clinical Oral Health Status in Menopaused Women" in 2018 awarded by Department of Medical Research, Ministry of Health, Myanmar. In 2021, she started her further degree and achieved Dr. D.Sc (Oral Medicine) from UDMM in 2024. She has contributed her findings as co-auther in Frontiers in Immunology, Journal of OncoImmunology and Cancers. Her recent research in oral cancer dedicates the early diagnosis and determinations of prognosis in the patients with oral cancer in Myanmar.

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Efficient anticancer properties of ashwagandha extracted phytochemicals for lower protein expression on U-251 and clove extracts on LN-229 GBM cell lines

Glioblastoma Multiforme G IV primary malignant Brain tumor medications such as Radiation, Surgery, Gene therapy, Chemotherapy and Ayurveda which are not very much successful with clinical studies. However, the trials are being made towards the various cancers' successful treatment with less side effects. Hence natural phytochemicals extract of Ashwagandha (Withania somnifera) Soya bean (Glycine max) and Clove (Syzygium aromaticum) are also considered for cancer therapeutics having much side effects on Human brain cells and organs.

The Phytochemicals treated GBM cell lines U251 and LN229 subjected for MTT assay, SDS gel electrophoresis, Protein content determination by Lowry and Bradford assays.

The Ashwagandha extracted phytochemicals such as Alkaloids, tannins, saponins, phlobatannins, Flavonoids, Terpenoids were determined. Ashwagandha phytochemicals extract treated U251 cells showed IC50-1.1µg/100 µl and 83.95% cell death for the same concentration.

The LN 229 Cell viability reduced to 40% with clove phytochemicals and cinnamaldehyde.

The protein content reduced to 118.68µg/ml for 138µl/ml Ashwagandha phytochemicals as compared to control protein 257.25µg/ml. Whereas, less protein band intensity displayed for phytochemicals of Ashwagandha, treated U251 cells and less protein expressed for cinnamaldehyde and clove extract phytochemicals treated LN229 cells respectively when compared to their control.

Therefore, GBM U251 and LN 229 cells treated with Phytochemicals of Ashwagandha and clove showed reduction in protein expression directly depicts tumor cell death that acts as anticancer drug conjugate.

Key words: GBM Cells, U 251, LN229, Phytochemicals, Protein Expression, Tumor Cell Death.

Biography

Prabha Muddobalaiah is an accomplished academician with a diverse research background in medical neuroscience, focusing on brain tumors and neurodegeneration, as well as cell/molecular neurochemistry, including neural stem cells and hydrolases. She holds an MSc in Biochemistry from Central College (1996) and a PhD in Biochemistry from Bangalore University and NIMHANS (2005). Prabha also completed a visiting fellowship at NCBS in 2006 and a postdoctoral fellowship at MCBL, IISc from 2007 to 2008. With over 24 years

of research experience, including a PhD and postdoctoral training, she has spent 15 years in academia, with 7 years as an Assistant Professor and currently serving as an Associate Professor since 2017. She has delivered 8 invited talks and received around 30 invitations to speak abroad, published 20 research papers (18 as the first author), and served as an invited reviewer for journals like *Journal of Neurochemistry* and *Cancer Biomarkers*. Prabha is also a life member of several academic organizations, including IAN and SNCI India, and is an editorial member of three journals. Her contributions have earned her several prestigious awards, including the Bharat Shiksha Gaurava Puraskar, the Excellence for Best Educationist Award in 2022, and the Eminent Educationist of India Award by KTK Foundation, New Delhi, in November 2022. She also received the NPTEL Domain Star award in Biotechnology-Biosciences in 2022, earning six silver medals and an Elite certificate, and was recognized with the Women Researcher Award at the International Scientist Awards on Engineering, Science, and Medicine in November 2022.

Ratan Kumar Sarkar

Independent Researcher, Keota, Hooghly, West Bengal, India

Evaluation of mutational values in cancerous point mutation and innovation of values-effective drugs in cancer treatment

The molecular weights of amino acids are time values where integer and decimal part have separate identity but interrelated. Derived from the factor 14.0267 e.g. 119.1197 (Thr)–105.0930 (Ser)=14.0267=131.1736 (Leu)–117.1469(Val), the formula T(time)=0.0019*M(integer) is important with a difference of 0.0001 or 1 associated with directional biology. The values 0.0267=0.0154+0.0113(6) are factors of opposite since 0.1605–0.1451=0.0154 and 777(183)–771(i.e. 357+414)=6 and conversely 177+6=183 (lunar time). To evaluate mutational values, the Core values (Cv) of amino acid have to be measured e.g. 181.1894 (Tyr)=181*0.0019–0.1894=0.1545 or 645 (under suppression)=900–255. The biophysical structure or electro-gravitational structure works within 900 and the reverse domain 109 and Tyr-Val chemistry constitutes an electro-gravitational structure where 289+136=425=938–513(27) and also Tyr-Met chemistry constitutes an electro-gravitational structure where 519+289=808 and conversely 519–289=230 where 808–230=578=575+3. The values are measured from lunar gravity (0.1605 or 705) with decimal part where 0.2124(Met)–0.1605=0.0519 or 519, 0.189(Tyr) –0.1605=0.0289 or 289 and 0.1605–0.1469(Val)=0.0136 or136. The electro-gravitational structure is based on 0.3477(183)–0.2902(i.e.0.1451*2)=0.0575 or 575=0.1605–0.1031(i.e. 900–131=769 that reverse of 967 where 193*0.0019=0.3667 or 967) where 0.1451=0.0938+0.0513. The molar masses of proton 938.28 Mev/C2 and that of electron 0.511Mev/C2 with a significant 0.0002 time difference. The electro-gravitational structure activates if there is a difference of 100 towards structural mutation or blocking of time by ‘9’(0.0171 or 171) where 289 shows tyrosine kinase activity due to blocking of time in directional biology and 28*82=2296 or 496 where 575+496=1071 or 171 and conversely 496–325=171 in 900=575+325 and if 575 is positive then 325 runs negative. There are some crucial structure, 900=360+540=289+611=645+255 =513+387 in the system where the intermediate values 645–255=390 is associated with 109 where 390+109=499(reverse of 994) and 289–255=34(i.e. 0.0646) is significant. The JAK2 G1849T V617F and TP53 G469T V157F would be standard manifestation where core values of Val=754=900-146(complementary values of 469 point) and 705+49(beyond 900)=754 and 109+49=158. The Val754 can be a pathfinder for drug assessment. The EGFR lung cancer mutations L858R, C797S and T790M are interrelated where 858+797=1655 or 755 and 858–109=749 and 797–109*2=579 are the causes of cancer since time is indomitable and cannot be stopped. It is found 790–540=250=575–325 where 360–250=110(suppressed). In BRAF V600E, 754–600=154 and 600–6=594(E) where 154+6=160 causes cancer.

Biography

Ratan Kumar Sarkar graduated with a B.Sc. from the University of Calcutta and has published over twenty research papers in Scientific and Academic Publishing journals.

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Unveiling gaps in cancer care: A qualitative study on the needs of patients from Northeast India

Background: Cancer is the leading cause of death worldwide, with the WHO projecting a 45% increase in global cancer deaths from 2008 to 2030. Beyond health impacts, cancer imposes significant economic strains, particularly on younger, low-income individuals. The extensive economic burden of cancer is well-documented, exacerbated by late-stage diagnoses due to inadequate screening awareness. Rising cancer care costs result in higher out-of-pocket expenses (OOPEs), medical debt, and bankruptcies, with cancer patients facing more significant financial burdens compared to those with other conditions. India experiences the highest OOPEs globally, with 62.6% of health expenditures attributed to this cause. Inadequate health insurance is a primary factor. The Indian government's 2018 launch of the Ayushman Bharat Pradhan Mantri Jan Arogya Yojana (AB PM-JAY) aims to reduce these disparities by providing cashless cancer treatments through over 28,000 hospitals. Meghalaya's Health Insurance Scheme (MHIS), which has been aligned with AB PM-JAY since 2012, seeks to minimise OOPEs and enhance healthcare services. Effective communication with healthcare providers is crucial, influencing decision-making, treatment adherence, and overall patient satisfaction, reducing distress and improving quality of life. Addressing financial aspects through patient-centred plans is essential to mitigate financial toxicity.

Aim: This study aims to explore the financial experiences and needs of cancer patients within a tribal population in Northeast India.

Methods: This qualitative study utilised in-depth face-to-face interviews with cancer patients and their family members, covering head and neck cancer, gastrointestinal cancer, and respiratory cancer from stage I-IV. 19 interviews were conducted with 12 patients and seven caregivers at a hospital Outpatient Department (OPD) Civil Hospital, Meghalaya.

Results: The study identified five key themes: Initial Symptoms and Health-Seeking Behaviour: Participants described delayed healthcare-seeking due to lack of awareness and cultural beliefs. Perception of Cancer and Treatment: Misconceptions about cancer and its treatment influenced patients' decisions and compliance. Financial Burden and Insurance Coverage: High treatment costs, limited health insurance, and significant out-of-pocket expenses created substantial financial stress. Treatment Pathways and Satisfaction: Patients faced challenges navigating treatment options and expressed varying satisfaction levels with care. Support

Systems and Coping Mechanisms: Family and community support and psychological coping strategies were crucial for managing the cancer journey.

Discussion: The study investigates cancer patients' needs and experiences, highlighting the significant impact of cultural beliefs, financial concerns, and healthcare-seeking behaviours. Patients often initially interpret symptoms through artistic lenses, attributing illness to supernatural causes like black magic, which aligns with existing literature on cultural frameworks shaping health behaviors. The transition from traditional to formal healthcare systems is complex, with delays and misdiagnoses common, reflecting barriers such as health literacy and structural inequalities. Culturally sensitive healthcare approaches, integrating traditional healing with biomedical care, may enhance early detection and health outcomes.

Keywords: Cancer, Cost Communication, Financial Burden, Out-of-Pocket Expenditure, Health Care.

Biography

Redolen Rose Dhar completed her BSc in Nursing in 2016 and practised for one year before pursuing her master's in public health at Manipal Academy of Higher Education from 2018 to 2020. During her studies, she gained experience in diverse fields through internships, including Maternal and Child Health (MCH), Non-Communicable Diseases (NCD), and Communicable Diseases. She later joined the Indian Institute of Public Health, Shillong, focusing on NCDs, particularly cancer. In 2022, she enrolled in a PhD program at Manipal Academy of Higher Education, Department of Health Information Management, with her research centred on cancer in the tribal population of Northeastern India.

Dr. Sara Mokbel*, Giuilia Baciarello, Pernelle Lavaud, Aurelius Omlin, Fabio Calabrò, Richard Cathomas, Stefanie Aeppli, Pauline Parent, Patrizia Giannatempo, Kira-Lee Koster, Naara Appel, Philippe Gonnet, Gesuino Angius, Petros Tsantoulis, Hendrick-Tobias Arkenau, Carlo Cattrini, Carlo Messina, Jean Zeghondy, Cristina Morelli, Yohann Loriot, Vincenzo Formica, Anna Patrikidou

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Development and validation of an inflammatory prognostic index to predict outcomes in advanced/metastatic urothelial cancer patients receiving immune checkpoint inhibitors

Background: Immune Checkpoint Inhibitors (ICIs) improve Overall Survival (OS) in advanced/metastatic Urothelial Cancer (a/mUC) patients. Preliminary evidence suggests a prognostic role of inflammatory biomarkers in this setting. We aimed to develop a disease-specific prognostic inflammatory index for a/mUC patients on ICIs.

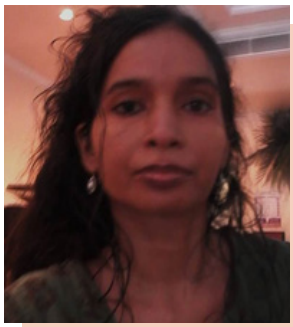
Methods: Fifteen variables were retrospectively correlated with OS and Progression-Free Survival (PFS) in a development (D, n=264) and a validation (V, n=132) cohort of platinum-pretreated a/mUC pts receiving ICIs at L2 or further line. A nomogram and inflammatory prognostic index (U-IPI) were developed. The index was also tested in a control cohort of patients treated with chemotherapy only (C, n=114).

Results: The strongest predictors of OS were baseline Platelet/Lymphocyte (PLR) and Neutrophil/Lymphocyte (NLR) ratios, and Lactate Dehydrogenase (LDH), NLR, and albumin changes at 4 weeks. These were used to build the U-IPI, which can distinctly classify patients into good or poor response groups. The nomogram scoring is significant for PFS and OS ($p<0.001$ in the D, V, and combined cohorts) for the Immunotherapy (IO)cohort, but not for the control cohort.

Conclusions: The lack of a baseline systemic inflammatory profile and the absence of early serum inflammatory biomarker changes are associated with significantly better outcomes on ICIs in a/mUC pts. The U-IPI is an easily applicable dynamic prognostic tool for PFS and OS, allowing for the early identification of a sub-group with dismal outcomes that would not benefit from ICIs, while distinguishing another that draws an important benefit.

Biography

Dr. Sara Mokbel is a resident doctor at Guys and St Thomas' Hospital, London. She graduated from UCL in 2024 during which time she completed a Bsc in Oncology where she undertook research into bladder cancer. Although still in the earlier stages of her career, she is passionate about further research projects particularly within the field of biomarkers.



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Autophagy as an immunomodulatory signalosome 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.

Conclusions: My promising translational research study highlights the emerging immunotherapeutic potential of Autophagy/Toll-like Receptors/Wnt-Frizzled cross-talks/signaling-networks in hepatocellular/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 66 scientific publications in international journals.

Dr. Shivam Singh[1*], Subhash Gupta[1], Ashok Sharma[2], Richa Tripathi[3], V.G. Huddar[4]

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The potential of Piper longum extract on breast cancer cell proliferation by inducing apoptosis

Background: The incidence of breast cancer has increased while the mortality rate has continued to remain high. Effective treatment of this disease is the key to survival. Therefore, this study is a necessity in continuing research into new effective treatments. According to ancient Ayurvedic text, many plants have anticancer effects. They are known to reduce the proliferation of cells and the size of tumour after treatment. Piper longum is an Indian herbal drug that has been shown by Ayurvedic practitioners to be effective in treating cancer with difficult outcomes and have few side effects. Nevertheless, its anti- cancer role in breast cancer is still unknown. The current study investigated the cytotoxic effects of Piper longum (Pippali) aqueous extract on human breast cancer cell line (MCF7) using various in-vitro assays.

Methods: The cytotoxicity of the extract was determined to analyse its inhibitory effects on MCF7 cells growth by MTT assay. DNA cell cycle analysis and apoptosis assays were also performed in pippali aqueous extract treated and untreated MCF-7 cells.

Results: After 24 hours of pippali treatment, the impact of the pippali aqueous extract on MCF-7 proliferation was examined using the MTT test, and the extract's IC50 value was found to be 3.125µg/µl. Additionally, the impact of pippali extract on MCF7 cells' DNA cell cycle and apoptosis was examined using the same IC50 dose. When pippali was applied to cells, apoptosis was induced and there was a greater rate of cell death than in untreated cells. Furthermore, compared to untreated cells, pippali-treated cells showed evidence of G0/G1 arrest.

Conclusion: The studies presented above suggest that Piper longum may have substantial anti- cancerous properties. To confirm the drug's effectiveness against breast cancer, additional functional testing is necessary and will be conducted in our lab shortly.

Keywords: Breast Cancer, Piper Longum, Cytotoxic Assay, Apoptosis, Annexin Assay.

Biography

Dr. Shivam Singh studied Zoological Sciences at the University of Delhi, India and graduated with a Master of Science in 2010. She then joined Dr. Rinu Sharma's research group at Guru Gobind Singh Indraprastha University, India. She received her PhD degree in 2020 at the same institution. After 6 months of industrial experience, she joined the research group of Prof. Subhash Gupta at the All India Institute of Medical Sciences (AIIMS), India as a Postdoctoral fellow. She is currently working on a breast cancer clinical trial project at the same institute. She has published 5 research articles in SCI (E) journals.

Sinem Durmus

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Oxidative stress-fighting miRNAs: Targeting cancer's achilles' heel

Oxidative stress is one of major factors of cancer initiation and progression by inducing damage to cellular macromolecules through the accumulation of Reactive Oxygen Species (ROS). This process contributes to tumor development by triggering events such as DNA mutations, protein modifications, and lipid peroxidation. As small, non-coding RNA molecules that regulate gene expression at the post-transcriptional level, miRNAs play an important role in modulating the cellular effects of oxidative stress. Specific miRNAs target oxidative stress-related signaling pathways and affect cancer cell properties such as proliferation, apoptosis and metastasis. For instance, oncogenic microRNAs such as miR-21 and miR-155 enhance survival of tumor cells by enhancing antioxidant defense mechanisms, whereas tumor suppressor microRNAs such as miR-34a and miR-146a induce cell death triggered by oxidative stress. Such a balance is an important part of cancer cell adaptation to oxidative stress and tumor development. Furthermore, oxidative stress influences microRNA regulation by transcription factors and other signaling pathways. Transcription factors NF-κB, p53, and HIF-1α play a role in miRNA regulation by being activated upon ROS buildup. In addition, oxidative stress-induced DNA damage is able to trigger cellular defense and direct apoptotic or anti-apoptotic activity of cancer cells through miRNAs. A detailed understanding of these mechanisms provides important clues on how to target miRNAs for cancer biology and therapeutic approaches.

This study addresses the critical role of oxidative stress-related miRNAs in cancer biology and their therapeutic potential. Oxidative stress promotes tumor progression by altering the genetic and epigenetic architecture of cancer cells, and miRNAs are important molecules that regulate these processes. Therefore, the use of miRNAs as diagnostic biomarkers and therapeutic targets in cancer is of increasing interest. Current research suggests that miRNA-based therapeutic approaches may open new horizons in cancer treatment by targeting oxidative stress-related pathways. Further studies for clinical applications will help us to better understand the role of miRNAs in cancer treatment and to develop personalized therapeutic strategies.

Biography

Dr. Durmus earned her Ph.D. in Medical Biochemistry from Istanbul University-Cerrahpasa, where she investigated the relationship between disease severity, miRNA-21, and Cathepsin B in patients with Familial Mediterranean Fever. She completed her Master's at Istanbul University, focusing on RAGE gene polymorphisms in endometrial cancer. Dr. Durmus has held research positions at Izmir Katip Çelebi University and Istanbul University-Cerrahpasa. Her research interests include miRNA expression in diseases, gene polymorphisms, and the molecular mechanisms of cancer and other pathologies. She has published extensively in international peer-reviewed journals and has received several awards for her work.

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Histone lactylation drives oncogenesis and function as a prognostic marker in pancreatic ductal adenocarcinoma

Background: Metabolic reprogramming and epigenetic alterations contribute to the aggressiveness of Pancreatic Ductal Adenocarcinoma (PDAC). Lactate-dependent histone modification is a new type of histone mark, which links glycolysis metabolite to the epigenetic process of lactylation. However, the role of histone lactylation in PDAC remains unclear.

Methods: Western blot and immunohistochemistry were applied to identify the expression of histone lactylation in PDAC. The Overall Survival (OS) was evaluated using a Kaplan-Meier survival plot. Inhibition of histone lactylation by glycolysis inhibitors or LDHA knockdown elucidated the pivotal role of histone lactylation in the growth and progression of PDAC both in vitro and in vivo. Western blot and functional experiments were employed to identify potential writers and erasers of histone lactylation in PDAC. CUT & Tag and RNA-seq analyses were utilized to identify potential target genes of H3K18 lactylation. The sequencing results were validated through ChIP-qPCR, RT-qPCR and Western blot analyses. Knockdown or overexpression of TTK protein kinase (TTK) or BUB1 mitotic checkpoint serine/threonine kinase (BUB1B) confirmed the significance of these two genes in PDAC. Co-IP assay was conducted to identify the interaction between TTK and Lactate Dehydrogenase A (LDHA).

Results: Histone lactylation especially H3K18 lactylation (H3K18la) levels are elevated in PDAC, which is associated with poor prognosis. The suppression of glycolytic activity using different kinds of inhibitors or si-LDHA contributes to the anti-tumor effects of PDAC in vitro and in vivo. P300 and HDAC2 are potential writer and eraser respectively of histone lactylation in PDAC cells. Mechanistically, H3K18la is enriched at the promoters and activates the transcription of mitotic checkpoint regulators TTK and BUB1B. Interestingly, TTK and BUB1B could elevate the expression of P300 which in turn increases glycolysis. Moreover, we demonstrate that phosphorylation at tyrosine 239 (Y239) and the activation of LDHA are mediated by TTK, subsequently influencing lactate content and H3K18 lactylation levels.

Conclusions: The glycolysis/H3K18la/TTK&BUB1B positive feedback loop exacerbates dysfunction in PDAC. These findings delivered a new exploration and significant inter-relationship between lactate metabolic reprogramming and epigenetic regulation, which might pave the way toward novel lactylation treatment strategies in PDAC therapy.

Biography

Dr. Wenzhe Si studied molecular biology at the Peking University, she received her PhD degree in 2015 at the same institution. She works in the National Key Laboratory of Vascular Homeostasis and Remodeling, Peking

University, China. She has published 31 research articles, of which 20 are first or corresponding authors (including co-authors), including Cancer Cell, Molecular Cancer (two papers), Signal Transduction and Targeted Therapy, Cell Death & Disease, etc. As a project leader, she has presided over 17 national, provincial, Peking University and horizontal projects, including two National Natural Science Foundation of China (NSFC) projects, one Beijing Nova Program, one Beijing Natural Science Foundation (BNSF) project, and so on.

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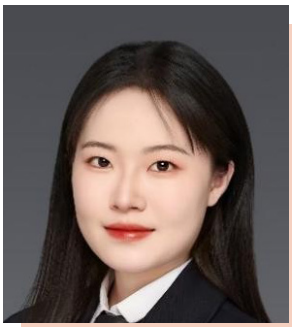
Protein structure maintenance, intracellular signaling and tumor suppression contributed by zinc finger protein ZMYND10

On short arm of human chromosome 3, a region 3p21, which is deleted in a variety of tumors, harbors a cluster of putative Tumor Suppressor Genes (TSGs). One of the TSG candidates, BLU, coding for a zinc protein ZMYND10 has initially recognized as a fragment lost in lung cancers. ZMYND10 has also been reported to be a structural protein whose mutation is responsible for pathogenesis of Primary Ciliary Deficiency (PCD), and data suggests that ZMYND10 plays a role in maintenance of protein structure by serving as a co-chaperone through interacting with heat shock protein 90 (HSP90) and FK 506 Binding Proteins (FKBPs) like FKBP8. Some members of FKBP family has been shown to modulate the activity of NFkappaB pathway by phosphorylation modifying the regulating kinase. In addition to be classified as a member of zinc finger myeloid Nervy deformed autoregulatory containing (ZMYND) known as ZMYND10, it is also classified as a member of the Dynein Assembly Associated Factor (DNAAF), termed DNAAF7. Members of this family of DNAAF have been identified as cancer related with differentially expressed in malignancy as compared with normal controls. At transcript level, the silence of BLU due to hypermethylation on upstream promoter region has been noted in NPC, glioma, Hepatocellular Carcinoma (HCC) and other cancers. We reported that re-expression of BLU reduced the level of IKKalpha and hence the level of p65/NFkappaB3 to downregulate apoptotic inhibitors CFLAR and cIAP to potentiate TRAIL induced apoptosis. It has also been shown that ZMYND10 inhibits signalling of JNK and ERK of Mitotic Activated Protein Kinase (MAPK) family to inhibit colony formation and arrest cell cycle. ZMYND proteins have been known to modify histone marks by regulation of histone methylation. We observed that ZMYND10 downregulates Enhancer of zeste homolog 2 (EZH2), alternatively termed KMT6A to block bi- or trimethylase of a repressive mark lysine 27 on Histone 3 (H3K27) to remove the transcription repression on an inhibitor of cell cycle progression p16/CDKN2A. The effects of ZMYND10 on NFkappaB pathway and histone modification remain to be mechanistic validation. Data obtained from public bioinformatics databases also reveals that ZMYND10 is downregulated in several human tumors, notably Lung Squamous Cell Cancer (LUSC) and adenocarcinoma (LUAD), renal clear cell and papillary cell carcinoma (KIRC and KIRP), Breast Cancer (BRCA), Cervical Squamous Cell Carcinoma (CESC) and several other cancers. To match with the downregulated expression registered in databases TIMER2 and GEPIA (Gene

Expression Profiling Interactive Analysis), Overall (OS) and Relapse Free (RFS) survival in early or late stage LUAD, LUSC, KIRC and KIRP and BRCA were correlated with the reduced level of ZMYND10. The observation remains to be expanded with more clinical specimens of more diversified background, so as to establish ZMYND and DNAAF proteins as antitumor therapeutic targets.

Biography

Dr. Zhang received his MBBS degree in China, and a PhD in tumor and cell biology at Karolinska Institutet in Sweden. Following his dissertation defense in 2005, he joined Chinese University of Hong Kong as a postdoctoral fellow, then the faculty of Guangdong Medical University, China as an associate professor. His research on Tumor Suppressor Gene (TSG) BLU was initiated ever since. His research encompasses the biological activities of BLU encoded zinc finger protein ZMYND10 in terms of its effects on intracellular signaling and malignant proliferation. He has published more than 50 SCI recorded papers in tumor viruses and TSGs.



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Horizontal comparison of the trend of pancreatic cancer in china and abroad: Lower development correlates with reduced pancreatic cancer burden, defying SDI-based predictions

Pancreatic Cancer (PC), characterized by aggressive progression and poor prognosis, poses a critical global health challenge. This multinational study investigates epidemiological disparities in PC incidence and disease burden through a Socio-Demographic Index (SDI)-stratified analysis, with a focus on China's unique patterns. Utilizing data from the Global Burden of Disease Study 2021, we employed DisMod-MR 2.1 for incidence estimation and Cause of Death Ensemble modeling (CODEm) for mortality analysis, integrating geospatial data, cancer registries, and vital registration systems. Age-Standardized Rates (ASRs) for incidence, mortality, and disability-adjusted life years (DALYs) were calculated using WHO population standards, with temporal trends assessed via Estimated Annual Percentage Changes (EAPC).

Our analysis reveals a strong positive correlation between SDI levels and PC incidence: high-SDI countries exhibited significantly higher ASRs compared to low-SDI nation, reflecting developmental and lifestyle influences. China, a middle-SDI country, demonstrated an epidemiological paradox. Despite reporting 118,665 incident cases in 2021 (the highest national count globally), it maintained below-global-average incidence (5.6 vs 6.0 per 100,000) and, while its DALYs were lower than most developed countries. Contrasting previous studies, recent improvements in PC metrics were observed in low-SDI Asian and African nations, attributable to enhanced medical infrastructure (e.g., WHO Global Cancer Initiative) and expanded cancer registries. For instance, African countries strengthened screening capacity through collaborations like the African Organization for Research and Training in Cancer (AORTIC). However, concurrent rises in mortality and DALYs in these regions underscore persistent challenges from delayed diagnoses and resource limitations. In contrast, China's population-scale healthcare framework achieved effective disease control despite demographic pressures, highlighting systemic advantages in managing complex malignancies. This divergence suggests population size alone cannot be considered to explain disease burden variations. Structural analysis indicates China's government-led healthcare system, emphasizing universal screening and centralized prevention policies, likely facilitates earlier detection and control DALYs compared to market-driven systems with fragmented diagnostic access.

These findings challenge SDI-centric disease burden models and emphasize the mediating role of healthcare system architecture. While socioeconomic factors influence PC incidence, centralized health policies and equitable diagnostic access critically modulate outcomes. The study underscores the necessity of integrating health system characteristics into global cancer control strategies, particularly for malignancies requiring complex diagnostic pathways. Policy-driven healthcare organization, as exemplified by China's unified framework, may mitigate mortality disparities even in resource-constrained settings. Further research should prioritize evaluating low-cost screening interventions in under-resourced systems to address the rising burden in transitioning economies.

Biography

Miss. Zhu is a dual-degree candidate (BSc Clinical Medicine, Nanchang University; BSc Biomedicine, Queen Mary University of London, 2021–2026) with interdisciplinary expertise in clinical practice, bioinformatics, and translational research. Her academic work includes pan-cancer studies on KLF transcription factors, IFN mechanisms in viral-associated cancers, and independent research on CYR61 in liver cancer, resulting in peer-reviewed publications (2022, 2024). Clinically, she trained in diverse departments (oncology, neurosurgery, etc.), refining diagnostic and patient management skills. Currently, she applies epidemiological statistics and GBD database analysis to investigate global disease burdens.

Zahra Husain
Government Hospital, Bahrain

Proposal for new diagnostic criteria for sarcopenia based on CT imaging in Saudi population: A novel method in oncology research

Background: Sarcopenia has been shown to be an independent predictor of lower overall survival in oncology patients. Several studies have used Computed Tomography (CT) to measure psoas muscle surface area to define sarcopenia. However, the cut-off values based on CT imaging remain undetermined in Saudi population. The aim of this study is to provide sex and age specific percentiles for Psoas Muscle Area (PMA), Psoas Muscle Index (PMI) and Psoas Muscle Density (PMD) in Saudi population and to establish a formula to calculate the standard PMA based on individual's anthropometric measurement.

Methods: Preoperative CT imaging at the third lumbar vertebra level was used to measure PMA, PMI and PMD in 400 adult donors for Living Donor Kidney Transplantation (LDKT). We determined the age and sex-specific cut-off values of PMA in order to define low skeletal muscle mass. A formula was generated to calculate the standard PMA using body weight as independent variable and further validated on a new dataset involving individuals from the general population.

Results: Males had significantly higher measurements of PMA among females (10.7±2.7 vs. 5.8±1.9, $p<0.0001$). PMA was positively correlated with body weight in both genders. The estimated PMA using the generated formula correlated strongly with the manually traced PMA measurements. The mean differences between estimated and measured PMA values were 0.81±1.70 (95%CI, -1.75 to 0.13) among males and 0.17±1.19 (95%CI, -0.49 to 0.83) among females. These outcomes emphasize the validity of our predictive computations.

Conclusion: PMA can be used in opportunistic screening for sarcopenia and as a radiological marker to predict overall survival in oncology patients.

Biography

Dr. Zahra Husain is a musculoskeletal imaging radiologist from the Kingdom of Bahrain. She obtained her MBBCH degree from Dubai Medical College (UAE), graduating in 2019. Following this, she joined the Diagnostic Radiology Residency Program at Dammam Medical Complex (Saudi Arabia), earning the Saudi Board in Diagnostic Radiology in November 2023. In addition, she completed the Saudi Fellowship in Musculoskeletal Imaging at the Eastern Health Cluster in Dammam, Saudi Arabia, in January 2025.

Zainab Awada[1*], Ikhlak Ahmed[2], Blessing Dason[3], Fathima Abubacker[3], Aisheh Ali[1], Sara Tomei[4], Oleksandr Soloviov[4], Musa Dafallah[5], Ayman Saleh[5], Naima Al Mulla[5], Chiara Cugno[1]

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Genome-wide polymorphisms linked to treatment dosing and outcomes in Qatari pediatric acute lymphoblastic leukemia

Background: Childhood Acute Lymphoblastic Leukemia (ALL) is the most common pediatric cancer, yet significant inter-individual variability exists in response to its therapy. Genetic polymorphisms as biomarkers for treatment efficacy and toxicity in childhood ALL are limited, particularly in Middle Eastern populations whose unique and under-studied genetic variant profiles warrants investigation.

Objective: This study aims to identify the genetic variants associated with treatment dosing and toxicity in pediatric ALL patients of the Qatari population.

Design/Method: We collected clinical data and performed whole-genome sequencing (30X) on blood samples from 77 pediatric ALL patients at Sidra Medicine. Genetic variants within key Drug Metabolizing Enzymes and Transporters (DMETs) of ALL chemotherapy were assessed against 14,000 healthy Qatar Genome Program (QGP) and 1,000 Genomes Project (1kGP) participants using Fisher's exact test. Candidate and whole-genome variants were evaluated for association with 6-Mercaptopurine (6-MP) dosing percentage (administered versus protocol-required dose) using a multivariate mixed linear model, and variants passing genome-wide significance ($P<5x10-8$ and $FDR<0.05$) were reported. Gene-set enrichment analysis was performed using MAGMA, and pathways with $FDR<0.05$ were reported.

Results: Over 300 variants in DMETs were significantly enriched in QGP compared to 1kGP. Among the DMET variants, four including rs10133855 (MTHFD1), rs73379156 (TPMT), rs73142925 (DOK5) and rs597157 (NALCN), were associated ($FDR<0.05$) with reduced 6-MP dosing, with rs73379156 being in proximity to TPMT variants with specific 6-MP prescribing guidance. The homozygous mutant of rs73379156 (TPMT) experienced febrile neutropenia and required >60% reduction in 6-MP dosing, suggesting that the increased toxicity risk resulted in 6-MP dose reduction in mutation carriers. Genome-wide association analysis identified 12 ($P<5x10-8$) and 2,864 ($FDR<0.05$) variants associated with altered 6-MP dosing. Gene-set enrichment analysis showed that genetic variants associated with 6-MP dosing were most significantly enriched in an immune system pathway, highlighting the role of the immune system in 6-MP related complications.

Conclusion: This study revealed candidate and whole-genome genetic variants associated with altered 6-MP dosing in Qatari pediatric ALL patients, with insights into their biological

pathways. Further analysis on other ALL treatment endpoints is in progress, advancing the potential for personalized therapy of pediatric ALL within Middle Eastern populations.

Biography

Dr. Zainab Awada studied Pharmacy at Beirut Arab University, Lebanon and graduated as BS in 2008. She then joined the research group of Prof. Nathalie Zgheib at American University of Beirut and received MS in Pharmacology and Toxicology and PhD in Biomedical Sciences at the same institution. After that, she continued 4 years of postdoctoral fellowship at the International Agency for Research on Cancer, France supervised by Dr. Akram Ghantous. She has published >15 research articles in scientific journals, and her work spans the fields of pharmacogenetics of cancer treatments and epigenetic mechanisms of cancer risk factors, including smoking. She is currently a biostatistician scientist at Sidra Medicine, Qatar.

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Xuyu Gu[1], Chanchan Gao[2*], Xiangyu Su[2], Yaoyao Zhu[3], Qiyu Fang[1], Jia Yu[1], Wentian Zhang[4]

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BATF upregulates RGS2 expression to promote T cell exhaustion in lung cancer by activating the NF-kB pathway

Objective: This study aimed to investigate the role of RGS2 in immune regulation and its impact on the efficacy of PD-1/PD-L1 blockade therapy in Lung Cancer (LC), as well as to explore the regulatory relationship between RGS2 and BATF2 in modulating T cell exhaustion and tumor immune evasion.

Methods: Single-cell transcriptome-based analysis identified $CD8^+$ T-cell profiles as well as regulatory factors in six lung cancer patients receiving neoadjuvant PD-1 blockade therapy. Regulatory factors were validated in vivo via LLC cells and murine tumor organoid models in RGS2 knockout ($RGS2^{-/-}$) and BATF2 knockout ($BATF2^{-/-}$) mice. Functional assays evaluated metastasis, immune infiltration, and T-cell polarization. Mechanistic studies explored the transcriptional regulation of RGS2 by BATF2 using luciferase reporter assays, knockout and co-culture systems. Functional assays assessed metastasis, immune infiltration, and T cell polarization.

Results: RGS2 was highly expressed in $CD8^+$ T-exhausted (Tex) cells and was associated with pro-inflammatory pathways, including TNF-a signaling via NF-κB. High RGS2 expression predicted poor clinical outcomes and limited response to PD-1/PD-L1 blockade therapy. In $RGS2^{-/-}$ mice, metastasis and angiogenesis were suppressed, $CD8^+$ effector T cells were enhanced, and T cell exhaustion markers (PD-1, CTLA4, Galectin-9) were reduced. BATF2 was identified as a key transcriptional regulator of RGS2, promoting T cell exhaustion through inhibition of CXCL13 secretion. Knockdown of BATF2 or RGS2 impaired lung cancer cell proliferation, enhanced sensitivity to NK cell-mediated cytotoxicity, and reduced immune escape. In $BATF2^{-/-}$ mice, stem-like $CD8^+$ T cells were increased, while exhausted T cells were reduced, leading to improved anti-tumor immune responses.

Conclusion: RGS2, regulated by BATF2, plays a critical role in promoting T cell exhaustion and tumor immune evasion in lung cancer. Targeting the BATF2-RGS2 axis may enhance the effectiveness of PD-1/PD-L1 blockade therapy by reversing T cell exhaustion and improving anti-tumor immunity. These findings provide a novel therapeutic strategy for enhancing immunotherapy outcomes in LC.

Biography

Dr. Chanchan Gao is the deputy chief physician, master supervisor and clinical teaching director of the Oncology Department of the Affiliated Zhongda Hospital of Southeast University. She graduated in 1999 with a medical degree and has been working on immunotherapy for solid tumors. In 2021, She was admitted to Southeast University to study for his doctorate. Dr. Chanchan Gao has not only accumulated rich experience in clinical work, but also actively participated in scientific research projects, and promoted the progress in the field of tumor immunotherapy.

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ADGRE5^{+} CD8^{+} T cells overcome resistance to neoadjuvant immunotherapy in lung cancer

Neoadjuvant immunotherapy with anti-Programmed Death-1 (neo-antiPD1) has revolutionized perioperative treatment for improving the Overall Survival (OS) of patients with early-stage lung cancer. Despite its transformative impact, neo-antiPD1 provides limited benefit to nearly 50% of patients, who do not achieve a Major Pathologic Response (non-MPR). Currently, there is an urgent need for methods to predict MPR before surgery and strategies to overcome resistance in non-MPR patients.

In this study, we performed an integrated analysis of publicly available single-cell sequencing data from 55 patients, leading to the identification of a novel subset of ADGRE5+ CD8+ T cells that expand in the Tumor Microenvironment (TME) of lung cancer patients achieving MPR. These ADGRE5+ CD8+ T cells exhibit stem-like properties compared to ADGRE5- CD8+ T cells. Using single-cell sequencing and CYTOF profiling of murine TME, we confirmed the presence of stem-like ADGRE5+ CD8+ T cells in mice that responded to neo-antiPD1 treatment. We further validated the significant expansion of ADGRE5+ CD8+ T cells in MPR patients from our own neo-antiPD1 cohort of 16 patients.

To explore its broader application, we constructed a pan-cancer immunotherapy cohort using RNA-seq data from 782 patients. Leveraging machine learning, we developed an ADGRE5-centered prognostic model (Tsurv), which could accurately distinguish between MPR and non-MPR patients in both TME and Peripheral Blood Mononuclear Cell (PBMC) sequencing data (AUC:0.88). Mechanistically, using a 3D ex vivo system developed by us previously, we revealed that anti-PD1 therapy upregulated ADGRE5 expression in a STAT5-IL32-dependent manner. Moreover, ADGRE5+ CD8+ T cells demonstrated greater stemness, proliferative capacity, and cytotoxicity compared to ADGRE5- CD8+ T cells. Notably, Adoptive Transfer (ADT) of ADGRE5+ CD8+ T cells effectively overcame neo-antiPD1 resistance in murine models.

In summary, our research highlights a novel subset of ADGRE5+ CD8+ T cells with potential utility in non-MPR identification and as a promising candidate for ADT therapy to overcome neo-antiPD1 resistance. Additionally, the robust performance of the Tsurv model in PBMC RNA-seq data suggests its potential application as a liquid biopsy tool for real-time monitoring of neo-antiPD1 response.

Biography

Dr. Li has been pursuing a PhD at Shanghai Chest Hospital affiliated with Shanghai Jiao Tong University School of Medicine since 2016 after graduating from undergraduate studies. During doctoral period, she has been deeply engaged in the field of neoadjuvant immunotherapy. She has published 1 research articles in SCI journals.

Shuaiqi Wang, Hao Sun

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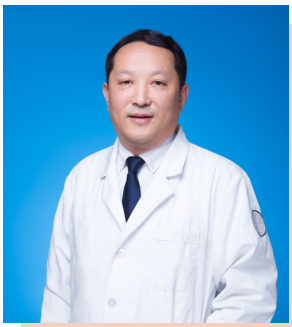

Laparoscopic-endoscopic cooperative non-exposed wall-inversion surgery combined with sentinel node navigation surgery in the treatment of early gastric cancer: A multicenter retrospective study

Background: We developed the Laparoscopic and Endoscopic Cooperative Surgery (LECS) technique, which combines Non-Exposed Wall-Inversion Surgery (NEWS) and Sentinel Node Navigation Surgery (SNNS) to treat Early Gastric Cancer (EGC). It is a technique which preserves organ function and conforms to the concept of super minimally invasive surgery.

Method: We analyzed the data of patients who underwent NEWS-SNNS in five medical institutions from 2019 to 2024. Patients who survive EGC were divided into two groups according to their choice: those who underwent NEWS-SNNS (N-S group) and those who underwent Laparoscopic Radical Gastrectomy (LRG group). Intra-operation situation, postoperative efficacy, safety indicators, quality of life and prognosis were compared between two groups.

Result: Out of the total 51 patients included, 23 underwent NEWS-SNNS and 28 underwent LRG. No statistically difference was noted in baseline characteristics between two groups (all $P>0.05$). No difference was noted in operation time, intraoperative blood loss and lymph node positive rate (all $P>0.05$). The number of lymph nodes dissected in N-S group was less than that in LRG group ($P<0.05$). Moreover, no difference was noted in the serum levels of immune cells on postoperative day 1, 3, and 7 (all $P>0.05$) or in the levels of serum C-Reactive Protein (CRP), Procalcitonin (PCT), and interleukin 6 (IL-6; all $P>0.05$). The time of first feeding, anal exhaust and anal defecation in N-S group were earlier than those in LRG group ($P<0.05$). The postoperative pain score, total incision length and incision infection rate in N-S group were lower that those in LRG group (all $P<0.05$). The scores of EORTC QLQ-C30 and EORTC-QLQ-STO22 in N-S group were lower that those in LRG group ($P<0.05$). No difference was noted in the 2-year PFS rate between two goups ($P<0.05$).

Conclusions: Compared with the standard LRG, NEWS-SNNS can preserve the function of stomach better. It reduced postoperative rehabilitation time and improve postoperative quality of life. While ensuring the sufficient resection margin and lymph node dissection, patients underwent NEWS-SNNS and LRG can obtain similar prognosis. In order to achieve a radical effect, accurate clinical staging of cancer is required before surgery. Double-tacer technique and frozen section are used for lymph navigation and dissection during surgery. Therefore, NEWS-SNNS is effective and safe to cure early gastric cancer. It is worthy of application.

Keywords: Laparoscopic and Endoscopic Cooperative Surgery, Non-Exposed Wall-Inversion

Surgery, Sentinel Node Navigation Surgery, Early Gastric Cancer.

Biography

Deputy Chief Surgeon Wang Shuaiqi has visited in Advent Health Medical Center of Florida in 2019.He has obtained academic titles such as the standing member of the abdominal oncology branch of the Chinese Medical Education Association, the youth standing member of the Oncology and Gastroenterology Special Committee of the Chinese Anti-Cancer Association, et al. His main research direction is minimally invasive treatment of gastrointestinal tumors.

Biography

Chief Surgeon Hao Sun has obtained academic titles such as the standing director of the World Association of Endoscopy Physicians Hepatobiliary Gastrointestinal Minimally Invasive Surgery, the member of the Robotic and Laparoscopic Surgery-Chinese Research Hospital Association Commitee, the member of the Colorectal Tumors Committee of Chinese Medical Doctor Association, the standing member of the ERAS Group of the Colorectal Tumor Committee of the Chinese Medical Doctor Association et al. His main research direction is laparoscopic endoscopic combined surgical treatment of gastrointestinal tumors.

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Development of risk stratification tool to appropriately triage the patients at risk of recurrence in HR+ HER2- early breast cancer

Introduction: HR+/HER2-negative Early Breast Cancer (EBC) is treated with curative intent, but managing locoregional and distant recurrences remains a significant challenge. The lack of a standard risk-identification approach leads to varied assessments across centers. Developing a robust risk assessment tool that incorporates guideline updates, AJCC classification, and clinical trials in a simplified manner is essential to mitigate Recurrence Risk (RoR).

Objective: To develop an effective risk assessment tool to manage and mitigate the RoR in patients with HR+ HER2 –ve early breast cancer incorporating multidisciplinary perspectives and evolving evidence/guidelines and trials.

Methodology: Multiple workshops were conducted across India to enhance the understanding and management of risk in EBC among the multidisciplinary teams involved in EBC management.

The steps to arrive at the algorithm are as follows-

1. Understanding Current Practices: To understand the current clinical practice of the participants, a preliminary poll consisting of 10 questions was conducted. Following this, Participants were divided into three to four groups for case- based discussion. During the group activity, they shared their current risk assessment and treatment practices for node positive and node negative cases.

2. Exploring Evolving Evidence: Following the group activity, a deep dive review of current literature on RoR in both node-positive and node-negative cases, as well as emerging evidence on the use of adjuvant therapy in EBC. Following each topic, a post-polling of relevant questions was conducted to determine any changes in the participants' beliefs regarding risk assessment in node-positive and node-negative EBC.

3. Ideation and Development: Based on the post-polling results, each component of the algorithm was populated. For components with less than 70% agreement, further discussion was conducted to reach a common alignment. Basis this a comprehensive, quick-reference, and user-friendly algorithm for risk assessment was developed.

Results: Five workshops were conducted with 135 multidisciplinary experts: 72 medical oncologists (53.3%), 28 onco- surgeons (20.7%), 26 radiation oncologists (19.3%), and 9 onco-pathologists (6.7%). All N2 patients were unequivocally considered at high ROR by all participants. In addition to nodal status, a tumour size >5cm was considered at high risk of recurrence by 59% versus 93% participants in pre and post polling survey respectively. 94.5% versus 58.8% (post versus pre) participants agreed that patients with N1 status are unequivocally at high risk of recurrence. It was agreed that grading and Ki-67 are key deciding risk stratification factors in patients with N0 tumour with T<5 cm for selection of adjuvant treatments by 74% participants in the post polling. Ki-67 cut off ≥20% was considered as critical to consider your patients at higher risk of progression by 100% post-polling vs 77% pre-polling.

Conclusion: The approach to managing Early Breast Cancer (EBC) is evolving. We implemented a unique methodology using a common questionnaire across different expert group to arrive at a standardized tool for risk assessment in HR+, HER2 -ve EBC patients across India. This tool simplifies the risk stratification approach for EBC patients and can help standardize approach to patient management across healthcare sectors.

Biography

Prof. Dr. Somashekhar S.P., MBBS, MS, MCh (Onco), FRCS Edinburgh, FRCS Glasgow, FARIS (Robotic), is the Chairman of MAB Aster DM Healthcare (GCC & India) and Global Director of Aster International Institute of Oncology. Leads the Department of Surgical and Gynecologic Oncology at Aster International Institute of Oncology, Bangalore, and holds a Professorship at Kings Health Partners, UK. Has received multiple accolades, including gold medals and fellowships, 300 publications, authored several oncology textbooks, and holds editorial positions in key journals. Prof. Dr. Somashekhar is a founding member of the Clinical Robotic Surgeons Association India and a mentor for Intuitive Surgicals and SSI Mantra Robotics.

Yuchun Niu*, Feng Ma

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Advancing cancer care: The impact of MYCNOS-SE on chemoresistance in SCLC

Understanding the Role of Super-enhancer MYCNOS-SE in Chemoresistance of Small Cell Lung Cancer.

Small cell lung cancer (SCLC) is a highly aggressive malignancy characterized by rapid progression and a high propensity for chemotherapy resistance. This presentation will discuss our recent findings on the super-enhancer MYCNOS-SE and its pivotal role in promoting chemoresistance in SCLC.

Super-Enhancers (SEs) are large clusters of transcriptional enhancers that drive the expression of key oncogenes and are crucial in establishing cell identity. In our study, we utilized RNA sequencing to identify highly expressed molecules associated with chemoresistance in SCLC. Through H3K27Ac and ATAC-Seq analyses, we identified MYCNOS-SE as a significant super-enhancer that regulates the expression of its target gene, MYCNOS.

Our findings demonstrate that MYCNOS is upregulated in both in vitro and in vivo models of SCLC, contributing to the development of chemotherapy resistance. We further elucidated the mechanism by which MYCNOS-SE recruits transcription factors CTCF and KLF15, enhancing MYCNOS expression and promoting survival in the presence of chemotherapeutic agents. Importantly, we discovered that MYCNOS, an antisense RNA of MYCN, modulates chemotherapy sensitivity through the NOTCH signaling pathway.

This research highlights the critical role of SE-regulated genes as biomarkers for chemoresistance in SCLC and suggests that MYCNOS could serve as a predictive marker for identifying patients who may benefit from NOTCH inhibitors. By understanding the molecular mechanisms underlying chemoresistance, we aim to provide insights that could lead to the development of novel therapeutic strategies targeting these pathways.

The presentation will also discuss the implications of our findings for future research directions and clinical applications, emphasizing the need for innovative approaches to overcome chemoresistance in SCLC and improve patient outcomes.

Biography

Dr. Yu-Chun Niu obtained her PhD in Oncology from Southern Medical University in Guangdong, China, in 2022. She currently serves as an attending physician at the Oncology Hospital of the First People's Hospital of Foshan. Her research focuses on the role of epigenetics in the occurrence, progression, and chemoresistance of small cell lung cancer. She has published several SCI papers as the first author in renowned international journals, including Cell Death & Differentiation, Oncogene, and Molecular Cancer.

BOOK OF ABSTRACTS

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