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2nd Edition of

International Cancer & Immuno-Oncology Conference

"Transforming Cancer Care: Innovations, Integrations, and Impact"

March 19-21, 2026
Singapore

IN-PERSON:

Village Hotel
Changi 1 Netheravon Rd,
Singapore 508502

VIRTUAL:

Greenwich Mean
Time (GMT)

2ND EDITION OF

International

CANCER & IMMUNO-ONCOLOGY CONFERENCE

HYBRID EVENT

19-21
MARCH 2026

CIOC
2026

BOOK OF ABSTRACTS

Index

Keynote Speakers

Keynote Speakers

Anyou Wang
DIFIBER LLC, United States

Atif A Ahmed
Seattle Children's Hospital, United States

George Zachos
University of Crete, Greece

Michael Thompson
University of Toronto, Canada

Patricia Tai
University of Saskatchewan, Canada

Paulo Cesar De Morais
Catholic University of Brasilia, Brazil

Keynote Speakers

Pietro Salvatori

Formerly, Humanitas San Pio X Hospital, Italy

Rajvir Dahiya

University of California San Francisco, United States

Sandrine Lacombe

University Paris Saclay-CNRS, France

Sergey Suchkov

N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Russian Federation

Stephan Bodis

University of Zurich, Switzerland

Thomas Jay Webster

Hebei University of Technology, United States

Welcome Message

Atif A. Ahmed, MD

Seattle Children's Hospital, United States

Dear Conference Attendees,

It is my great pleasure to welcome you to the "2nd Edition of International Cancer & Immuno-Oncology Conference" held in Singapore in a hybrid format. On behalf of the conference organizers, I 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 basic, preclinical and clinical research topics on cancer including clinicopathologic presentations, prognostic features, cancer immunology, 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.

Welcome Message

Patricia Tai

University of Saskatchewan, Canada

Dear Conference Attendees,

The Cancer Research conference is a well-organized event that every researcher and layperson can enjoy. With new cancer research emerging every day, the topic remains highly relevant to people from all walks of life. Various factors such as pollution, viruses, sun damage, genetics, and cigarette smoke increase cancer risk, making this subject crucial for everyone.

We had a highly successful conference in March 2025, and the organizing committee is pleased to invite you to join us in 2026! Take advantage of networking opportunities through in-person or virtual meetings, and gain valuable insights from the latest research.

In line with this, my keynote speech will highlight new updates on prostate cancer, focusing on its detection and management at initial diagnosis and subsequent recurrence. I will cover the latest publications and provide a summary of the conference, as much as the 40-minute time frame allows! This will be a valuable opportunity for all participants to gain insights into these recent advances. I promise to make the presentation as comprehensible as possible using layman's terms, though the slides will include abstracts, diagrams, and titles with some medical jargons. So much to share in just 40 minutes!

The other speakers are exceptional as well, hailing from renowned international universities and institutes, offering participants a truly enriching experience. The venue, or the city hosting this conference, is a delightful place to visit, featuring reasonably priced hotels that I always enjoy staying at. The shopping experience is fantastic—you can always find a great bargain or the perfect gift to take home to your loved ones.

Welkom [Afrikaans], مرحبا[Arabic], 欢迎 [Chinese], welcome [English], bienvenue [French], willkommen [German], स्वागत [Hindi], benvenuta [Italian], تمالس دايتغ[Malay Arabic], witamy [Polish], boas-vindas [Portuguese], bienvenida [Spanish], **ยินดีต้อนรับ** [Thai], hoş geldin [Turkish], chào mừng [Vietnamese]! We apologize that not all languages can be included due to space limitations; however, this conference will undoubtedly serve as an international hub for scientists from all nations!

Welcome **Message**

Rajvir Dahiya

University of California San Francisco, United States

As a keynote speaker and a member of the organizing committee, it is my great honor and responsibility to address you today at this 2nd Edition of International Cancer & Immuno-Oncology Conference held on March 19-21, 2026, Singapore. We are united by a single, powerful purpose—that is to treat the most deadly and complex diseases facing humanity, and to change its course through science, innovation, and compassion.

Cancer remains a global crisis. Despite remarkable advances, it continues to take millions of lives each year, often diagnosed too late, treated too late, and understood too little. Behind every statistic is a patient—a mother, a father, a child—whose life is altered forever. As physicians and scientists, we are not merely observers of this reality; we are entrusted with the responsibility to change it.

The future of cancer care lies in early detection and precise prognosis. Detecting cancer before symptoms appear, when disease is still curable, represents one of the greatest opportunities to save lives. Advances in molecular biology, genomics, liquid biopsies, and immune profiling are transforming how we identify cancer risk, stratify patients, and monitor treatment response. These tools are shifting oncology from reactive treatment to proactive prevention.

Equally transformative is the revolution in immuno-oncology and gene-based therapies. By harnessing the power of the immune system and targeting cancer at its genetic roots, we are moving beyond conventional therapies toward highly specific, durable, and potentially curative treatments. Gene therapies, RNA-based technologies, and immune modulation are no longer distant dreams—they are becoming clinical realities, offering new hope to patients who once had none.

To the young doctors and scientists in this audience: The future of cancer medicine belongs to you. You are entering this field at a moment of unprecedented opportunity. Your role is not only to build upon existing knowledge, but to challenge assumptions, innovate boldly, and push the boundaries of what is possible. Never forget that every experiment you design, every trial

you conduct, and every discovery you make has the potential to change a patient's life. Let this conference serve as a catalyst: for new ideas, new partnerships, and renewed determination.

Today, my presentation will be on "A novel Blood-based mRNA genomics technology for cancer diagnosis and treatment"

Together, let us transform fear into hope, science into cures, and vision into reality for the millions of patients who depend on us. Thank you for your dedication, passion, and commitment to advancing cancer care. I wish you a truly inspiring and impactful conference.

Keynote Address: Distinguished Colleagues, Respected Scientists, Young Investigators, and Honored Guests.

Welcome Message

Sergey Suchkov

N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Russian Federation

Distinguished colleagues, Esteemed oncology professionals, Global healthcare leaders, Dear Colleagues, Partners and Friends, We take great pride in welcoming all the attendees of the 2nd Edition of International Cancer & Immuno-Oncology Conference (CIOC 2026) which is being scheduled to be held in Singaporefrom March 19-21, 2026. This vital platform brings together leading minds dedicated to accelerating progress in cancer research, enhancing therapeutic approaches, and fostering cross-disciplinary collaboration under the aegis of Personalized and Precision Oncology (PPO) as a trend of new generation in the fight against cancer.

The Event has experienced remarkable growth over the years, firmly establishing itself as the premier event for the Oncology community. As the field of PPO continues to evolve, this Grand Event aims to bridge the gap between scientific discovery, design-driven translational research and clinical application to improve patient outcomes worldwide. Bringing together cancer surgeons, oncologists, researchers, biodesigners and bioengineers, medical administrators, entrepreneurs and insurers, and healthcare professionals from many countries of the Globe, the Conference serves as a forum for sharing knowledge, fostering collaboration, and advancing oncology practices. Over the course of this Conference, attendees will have the opportunity to engage in insightful discussions, participate in interactive workshops, and explore cutting-edge research through keynote speeches, oral and poster presentations, and networking sessions.

Our prognostic grand success is built on our state-of-the-art program that covers a wide range of new treatments, and technologies, unlocking the secrecy of PPO as a model of cancer-related healthcare services of the next-step generation. In this context, this conference would bring together relevant field experts, professors, clinicians, industry representatives, postdoctoral fellows, and research students from around the world, providing them with opportunity to report, present, share, and discuss scientific questions, achievements, issues and challenges in the PPO-driven and guided field. Numerous presentations as well as poster exhibition presented during this Conference will highlight some of the exciting developments in the fields of cancer

biology, cancer etiology, cancer screening and diagnosis, cancer imaging technologies, and cancer treatment and prevention, whilst illustrating the latest achievements in the area of PPO.

This Conference is a unique opportunity to confront peers from different disciplines, to set the current standards in oncology and up-to-date knowledge of the management of tumors, to deep dive into the complexity of PPO, and to foresee future challenges and opportunities in this rapidly changing and evolving field.

This Event is a fruitful platform to meet fellow key decision makers in universities, healthcare institution, pharmaceutical and biotech industries, to share experience, foster collaborations through the research talks and presentation to put many thoughts provoking strategies for discovering new ideas and new skills, in addition to expose your capabilities and discoveries to many interested colleagues.

We warmly encourage you to take an active role in this collaborative environment, share your expertise, and contribute to advancing the global fight against cancer. Finally, we would like to thank each of you for attending this prestigious conference and bringing your expertise to our gathering.

We hope you will have an academically productive time at the 2026 conference and a fun-filled time in a tiny but the Grand Singapore. We invite you save the date for this Conference, and look forward to seeing you there.

About
Magnus Group

About

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
CPD Accreditation

Continuing Professional Development (CPD) credits are valuable for CIOC 2026 attendees as they provide recognition and validation of their ongoing learning and professional development. The number of CPD credits that can be earned is typically based on the number of sessions attended. All the participants have an opportunity to avail 1 CPD credit for each hour of Attendance.

Some benefits of CPD credits include:

Career advancement: CPD credits demonstrate a commitment to ongoing learning and professional development, which can enhance one's reputation and increase chances of career advancement.

Maintenance of professional credentials: Many professions require a minimum number of CPD credits to maintain their certification or license.

Increased knowledge: Attending CIOC 2026 and earning CPD credits can help attendees stay current with the latest developments and advancements in their field.

Networking opportunities: This Conference provide opportunities for attendees to network with peers and experts, expanding their professional network and building relationships with potential collaborators.

About CIOC 2026

The **2nd International Cancer & Immuno-Oncology Conference (CIOC 2026)** will be held from **March 19–21, 2026**, as a **hybrid event** in **Singapore and online.** The conference aims to promote innovation and global collaboration in oncology, bringing together leading minds to share the latest advances in cancer research under the theme "**Transforming Cancer Care: Innovations, Integrations, and Impact.**" Emphasizing the translation of scientific discoveries into real-world clinical applications, **CIOC 2026** will provide a dynamic platform for discussing breakthroughs in cancer biology, prevention, and therapeutic strategies.

This global forum will bring together a diverse audience, including oncologists, immunologists, radiologists, clinical and translational researchers, geneticists, pharmaceutical experts, industry professionals, and healthcare policymakers. By connecting research insights with patient-centered care, the conference seeks to accelerate the adoption of novel technologies and treatment approaches that enhance cancer outcomes. The event will also support early-career researchers and students by offering valuable educational and networking opportunities.

The scientific agenda will encompass a wide range of topics such as early cancer detection, diagnostic innovations, biomarker discovery, targeted and immune-based therapies, palliative care, pediatric and organ-specific cancers, and the growing role of artificial intelligence in oncology. Key sessions will address clinical trials, survivorship, comorbid conditions, and emerging challenges in personalized medicine, immunotherapy, and drug resistance.

CIOC 2026 will feature **plenary lectures, oral and poster sessions, panel discussions, and interactive networking events,** creating an engaging environment for knowledge exchange and collaboration. Participants will have the opportunity to showcase their research, engage with experts, and explore new directions in cancer science.

Be part of this influential oncology conference—**join us in Singapore or virtually** to help shape the future of cancer research and care.

Table of Contents

Table of Contents

Table of Contents

Table of Contents

Table of Contents

Table of Contents

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KEYNOTE PRESENTATIONS

Anyou Wang
DIFIBER LLC, United States

Biography: Dr. Wang graduated from the University of California, Riverside, and is passionate about computational biology, big data, and AI (combai.org). He develops advanced algorithms to extract fundamental insights from large-scale biological data. As a pioneer in mining the world's largest biological database, he discovered that noncoding RNAs (ncRNAs) operate through a functional system distinct from proteins. This breakthrough helps explain how ncRNAs play a central role in various disease mechanisms and immune responses to environmental fluctuations. Dr. Wang further identified ncRNAs as the primary drivers in all cancers and key determinants of lifespan across animal species, challenging the long-standing protein-centric paradigm. He also developed novel computational tools to trace the evolutionary trajectory and origin of SARS-CoV-2, contributing to the understanding of the COVID-19 pandemic.

Deep systems insights into the conceptual breakthrough of the noncoding RNA functional network and its role in cancer

Noncoding RNAs (ncRNAs) transcribed from noncoding regions of the human genome are highly active and functionally significant; however, the nature of their functional system remains a subject of debate. Recent advances in big data and artificial intelligence have enabled a conceptual breakthrough in understanding this system. Unlike traditional models derived from protein studies and evolutionary conservation patterns, ncRNAs operate through distinct functional mechanisms and serve as critical endogenous regulators across all cancers. This presentation will provide deep systems-level insights into the ncRNA functional network and its universal role in cancer biology, and clarify major misconceptions in the field.

Atif A Ahmed[1*], Leonard Yermakov[2], Terrie Flatt[3]

[1]Seattle Children's Hospital, Seattle, USA
[2]University of Washington, Seattle, USA
[3]Children's Mercy Hospital, Kansas City, USA

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. 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. Member of the Society for Pediatric Pathology, Children's Oncology Group, International Society of Pediatric Oncology, and the College of American Pathologists.

MicroRNA expression in pediatric sarcoma

MicroRNAs are small non-coding RNA molecules that play a significant role in many physiological processes in the body, including posttranscriptional regulation of gene expression. Their expression in cancer cells can become dysregulated to influence the development of cancer. Although they have been extensively studied in adult cancers, their roles in pediatric sarcomas remain poorly defined. In order to define the microRNA profile associated with three common pediatric sarcomas, we used multiple tissue samples from different sources to detect microRNA differential expression in 26 Ewing's sarcoma, 50 rhabdomyosarcoma, and 32 osteosarcoma cases. We have used the NanoString multiplex nCounter platform to identify the expression of 827 human miRNAs and confirmed our findings with microRNA *in situ* hybridization (miRNA-ISH) of specific probes on tissue sections. The differential expression analysis of nCounter data identified 23 miRNAs enriched in RMS, 33 in EWS, and 45 in OS. MiR-206 was most strongly associated with RMS and demonstrated the highest sensitivity and specificity in distinguishing RMS from EWS and OS; this finding was also confirmed by miRNA-ISH. A combined signature of differentially expressed miRNAs reliably separated alveolar from embryonal RMS. The expression of miR-9-5p in EWS and miR-140-5p in OS discriminated among the different tumors and correlated with adverse patient outcome. The NanoString nCounter profiling method exhibited higher

sensitivity in detecting profiles and differential expression of microRNAs compared with microRNAscope, which identified the in situ hybridization of specific microRNA molecules. MicroRNA expression correlated with adverse patient outcome. Our findings demonstrate that distinct miRNA profiles can differentiate pediatric sarcoma types and provide clinically relevant insights into potential diagnostic and prognostic applications.

Eleni Petsalaki, Sofia Balafouti, George Zachos*

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

Biography: George Zachos completed his PhD at the University of Crete and received postdoctoral training at the Beatson Institute for Cancer Research, Glasgow, U.K. investigating DNA damage checkpoint mechanisms. In 2008, he moved to the Department of Biology of the University of Crete in Greece as an Assistant Professor in Cell Biology, became Associate Professor in 2015 and continues to hold this position today. Discoveries from the Zachos lab have identified novel mechanisms of the mitotic spindle and abscission checkpoints during cell division in human cells. He has published >40 papers in leading scientific journals and has received >3000 citations.

Building bridges: Mechanisms that prevent chromatin bridge breakage in cytokinesis

Chromatin bridges are strings of mis-segregated chromatin connecting the anaphase poles or daughter nuclei in mitotic cell division and can arise from incomplete DNA replication or decatenation, or from dicentric chromosomes generated by end-to-end chromosome fusions. Chromatin bridges pose a major threat for genome integrity because, if unsupported, they can lead to chromatin bridge breakage-fusion-bridge cycles or to chromothripsis, which can cause burst-like accumulation of genomic alterations that can drive carcinogenesis. As a result, preventing chromatin bridges from breaking is essential for cells to maintain genome stability. For this purpose, human cells use at least two major mechanisms to stabilize chromatin bridges: Firstly, they impose an abscission-delay, called “the abscission checkpoint”, to prevent chromatin breakage or tetraploidization by regression of the cleavage furrow. Recent findings from our lab shed light into how chromatin bridges are sensed by the cell and into the molecular mechanisms involved. We show that Topoisomerase IIα, an enzyme that can untangle catenated DNA molecules, recognizes “knotted” DNA on chromatin bridges and triggers a downstream MRN-ATM-Chk2-INCENP/Aurora B signaling pathway to delay abscission and prevent chromatin breakage. Secondly, cells with chromatin bridges generate accumulations of polymerized actin, called “actin patches”, at the base of the intercellular canal to maintain chromatin bridges and prevent them from breaking. We show

that daughter nuclei connected by chromatin bridges are under mechanical tension that requires interaction of the nuclear membrane Sun1/2-Nesprin-2 Linker of Nucleoskeleton and Cytoskeleton (LINC) complex with the actin cytoskeleton. This nuclear tension promotes accumulation of Sun1/2-Nesprin-2 proteins at the base of chromatin bridges, local activation of the small GTPase RhoA, and downstream ROCK-LIMK-Cofilin and mDia1 signaling to generate actin patches and prevent chromatin bridge breakage in cytokinesis. Together, this presentation will describe basic mechanisms that maintain genome stability in human cells and can protect against tumorigenesis.

Michael Thompson*, Soha Ahmaadi, Kathariana Davoudian, Navina Lotay, Lidia Nemtsov

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

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. His research is centered on the surface chemistry of proteins, cells and bacteria. Professor Michael 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 Bioelectronics 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.

Mutliplexed biosensor 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. In the present paper we discuss the application of biosensor technology for the early-stage detection of ovarian cancer. 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 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.

Patricia Tai[1*], Patrick Martineau[2], Kelvin Wong[3], Evgeny Sadikov[2], Glenn Ollenberger[1], Kurian Joseph[4], Edward Yu[5], Derek Liu[4], Aoife Jones Thachuthara[6], Arbind Dubey[7], Rashmi Koul[7]

[1]U. Saskatchewan, Canada
[2]U. British Columbia, Canada
[3]Astellas Pharma Canada, Canada
[4]U. Alberta, Canada
[5]Western U., Canada
[6]Cork University Hospital, Ireland
[7]U. Manitoba, Canada

Biography: Professor Patricia Tai graduated with a gold medal from U. of Hong Kong (ranked 11 globally) in 1984. Since then, she became an experienced clinical oncologist with expertise in skin and urologic cancers. Being an international expert on Merkel cell carcinoma, she is the author for 5 chapters in UpToDate since 2000. Professor Patricia has authored 155 full publications and 196 abstracts. She was the honorary Professor of University of Hong Kong. Currently she is a clinical professor of U. Saskatchewan, Canada, and welcomes collaborations on photodynamic therapy, skin cancers, Merkel cell carcinoma and prostate cancer.

Updates on controversies surrounding the staging and management of newly diagnosed localized prostate cancer using Prostate-Specific Membrane Antigen (PSMA) positron emission tomography

Prostate cancer is a very common malignancy in men, particularly in the Western world, although its incidence is lower among Asians. It can be identified using the relatively new Prostate-Specific Membrane Antigen (PSMA) Positron Emission Tomography (PET), a highly sensitive and specific imaging modality for prostate cancer. For disease localized to the prostate, several management options are available. This talk highlights significant updates on controversial issues relevant to cancer patients, caregivers, and researchers.

One major controversy concerns the appropriate timing of PSMA PET. Conventional imaging such as Computerized Tomography (CT) or bone scans are not required before ordering PSMA PET; in other words, CT or bone scans are not prerequisites. PSMA PET is a modern, more effective technique that can be used as a frontline imaging tool. However, studies from different countries have reported conflicting findings regarding its cost effectiveness, raising questions about whether its routine use is economically justified.

Following accurate staging with PSMA PET, subsequent management will be discussed, incorporating the latest updates in a manner comprehensible to patients and researchers across disciplines.

Key Topics Include:

(1) A brief global overview addressing the sustainability and cost effectiveness of routine PET, as well as treatment sequencing for hormonal therapy before, during, or after radiotherapy—an area requiring further research.

(2) Various forms of hormonal therapy, collectively known as Androgen Deprivation Therapy (ADT). Among these, gonadotropin releasing hormone antagonists have demonstrated better response rates, lower recurrence rates, and fewer complications compared with agonists.

(3) Management of the unfavorable intermediate risk group, which may include prostatectomy or radiotherapy combined with 4–6 months of ADT. Radiotherapy alone may be considered for patients with co morbidities, Gleason score 7 (3+4), and <50% positive biopsy cores, provided an escalated radiation dose is delivered.

(4) Results from the three Prostate Advances in Comparative Evidence (PACE) studies, which show that highly focused stereotactic radiotherapy—an approach that relies heavily on PSMA PET—is as effective as surgery or conventional radiotherapy.

(5) Clinical trial evidence indicating that pelvic nodal radiotherapy provides a survival benefit, suggesting that regional lymph nodes should be treated electively even before radiologic evidence of spread.

(6) Brachytherapy, a radiotherapy technique that delivers radiation very close to the tumor, thereby sparing adjacent normal tissues. A brachytherapy boost offers superior outcomes compared with an external beam boost, eliminating the need for ADT in intermediate risk cancers and reducing ADT duration to six months in high risk cancers. Notably, even short term use (4–6 months) of gonadotropin releasing hormone agonists can contribute to cardiac morbidity.

The keynote speech will introduce foundational concepts and modern treatment approaches in a stepwise manner, enabling the audience to understand the latest advances in prostate cancer management clearly and effectively.

Paulo C. De Morais[1,2]

[1]Genomic Sciences and Biotechnology, Catholic University of Brasilia, Brasilia, Brazil

[2]Institute of Physics, University of Brasilia, Brasilia, Brazil

Biography: Paulo C. DE MORAIS (H-61), PhD, was full Professor of Physics at the University of Brasilia (UnB)–Brazil up to 2013, Appointed as UnB's Emeritus Professor (2014), Appointed as Guest Professor of Huazhong University of Science and Technology–China (2011), Visiting Professor at Huazhong University of Science and Technology (HUST)–China (2012-2015), Appointed as Distinguished Professor at Anhui University (AHU)–China (2016-2019), Appointed as Full Professor at Catholic University of Brasília (UCB)–Brazil (2018), Appointed as CNPq-1A Research Fellowship since 2010. 2007 Master Research Prize from UnB, 2008-member of the European ERA NET Nanoscience Committee, Member of the IEEE-Magnetic Society Technical Committee, Senior Member of the IEEE Society, 2012 China's 1000 Foreign Expert Recipient, and 2012 Academic Excellence Award from Brazilian Professor's Union. 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–Brazil. He graduated in both Chemistry (1976) and Physics (1977) at UnB. Professor Morais is member of the Brazilian Physical Society and the Institute of Electrical and Electronics Engineers–IEEE. He has served as referee for more than 50 technical journals, takes part of the Editorial Board of more than 15 technical journals and has conducted research on nanomaterials for over 40 years. Professor Morais is known for his research in preparation, characterization and applications of nanosized materials (magnetic fluid, magnetoliposome, magnetic nanoemulsion, magnetic nanocapsule, magnetic nanofilm, magnetic nanocomposite, nanosized semiconductors, polymeric dots, carbon dots, and graphene quantum dots). With more than 500 published papers in peer reviewed journals, more than 14,000 citations, more than 250 international invited talks (35 countries), and 16 filed patents. He has appeared in recent World ranking of top scientists, such as 2020-Stanford, 2022-Research.com, 2023-AD Scientific Index, 2023-Research.com, 2024-Elsevier, 2025-AD Scientific Index, 2025-Research.com, and ONE Research Community.

Unveiling the synergism of radiofrequency therapy and graphene nanocomposite in tumor cell viability assay

In this keynote talk, the use of the Hill model to assess the Benchmark Dose (BMD), the Lethal Dose 50 (LD50), the Cooperativity (E), and the dissociation Constant (K) while analyzing cell viability data using nanomaterials will be explored. The presentation is addressed to discuss the antitumor potential while combining Radiofrequency (RF) therapy and engineered and selected nanomaterials. In particular, it will be discussed the use of nanocomposites, for instance those comprising Graphene Oxide (GO) surface functionalized with Polyethyleneimine (PEI) and decorated with gold nanoparticles (GO-PEI-Au). Data

collected from the cell viability assays using different tumor cell lines (e.g. LLC-WRC-256 and B16-F10) will be presented and discussed. The findings will demonstrate that while the tested nanocomposite (e.g. GO-PEI-Au) may be biocompatible against different cancer cell lines in the absence of Radiofrequency (nRF), the application of Radiofrequency (RF) enhances the cell toxicity by orders of magnitude, pointing to prospective studies with the tested cell lines using tumor animal models.

Pietro Salvatori

Formerly, Head of ENT Dept, Humanitas San Pio X Hospital, Milan, Italy

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 formed at the National Cancer Institute of Milan, Italy. Then, he acted as 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. He published more than 70 papers. During recent pandemic, he made research with international colleagues and published on ethanol inhalation to treat SARS-CoV-2 infection and Covid-19. During recent pandemic, he made research with international colleagues and published on ethanol inhalation to treat SARS-CoV-2 infection and Covid-19.

Nasopharyngeal carcinoma: A “different” head and neck tumour

As in some other sites of the head and neck, a broad range of tumours can arise in the nasopharynx: Epithelial, mesenchymal, lymphoid, and neuro-ectodermal. However, Naso-Pharyngeal Carcinoma (NPC) is the most interesting and intriguing type, because it is a “peculiar” malignancy, and “different” from almost all other head and neck tumours according to several points of view. In fact, NPC is a unique disease whose aetiology, clinical behaviour, epidemiology, and histopathology are different from those of all other squamous cell carcinomas of the head and neck. The most distinguishing characteristics of this malignancy are the consistent association with Epstein-Barr virus and the striking geographical differences in its incidence. The aim of this narrative review is to analyse the very large number of studies (sometimes contradictory) on NPC.

In the first part, the histopathology, aetiology, epidemiology, clinical behaviour, natural history, diagnostic work up, and staging will be examined.

The second part deals with treatment. Given that Radiotherapy (RT) is the treatment of choice for NPC, Chemotherapy (CT) has been added to standard RT to improve outcome in high-risk patients, either as an adjuvant, neoadjuvant or concurrent treatment modality with radiation.

Surgery plays an important role in rescuing recurrent or persistent disease after primary (CT) RT. A critical analysis of various treatment described in the literature, both for primary cancer and for regional and distant metastases, is presented. The prognostic factors and the final results of the various treatments will also be analysed.

Rajvir Dahiya M.S., Ph.D, M.D, D.Sc

Professor Emeritus, University of California San Francisco School of Medicine (UCSF) San Francisco, CA 94143, USA

Biography: Rajvir Dahiya holds Ph.D. in Experimental Medicine from Post Graduate Institute of Medical Education and Research Chandigarh, India, post-doctoral fellowship in medical oncology research from the University of Chicago Pritzker School of Medicine, M.D. from the Kagoshima University Faculty of Medicine, Kagoshima, Japan and D.Sc. from the Osaka University Graduate School of Medicine, Osaka, Japan. He became director of Oncology Urology Oncology Research Center at the UCSF/VAMC in 1991. After 34 years of service, he retired as a Professor Emeritus and Director of Urology Research Center and also, published more than 550 original research manuscripts. Dahiya's world ranking in medicine is 4759 and USA ranking is 2644 with more than 35,500 research citations and D-index of 107 in 2024. He has written books and holds multiple patents in oncology. Based on the NIH and VA data base NIH Reporter and Grantome, Dahiya's research programs were supported (99 times awarded) by the NIH and VA. Currently, he is an associate editor of "Clinical Cancer Research" journal.

A novel blood-based mRNA genomics technology for cancer diagnosis and treatment

Current screening methods—radiological imaging, DNA-based tests, and pathological assessments—often fail to detect malignancies at early stages. Despite technological advances, gene-based diagnostics specifically designed for early cancer detection remain limited. To bridge this gap, we evaluated the clinical utility and diagnostic accuracy of Geneverify test, a plasma cell-free mRNA-based test for prostate cancer. This novel, non-invasive approach has the potential to deliver faster, more precise diagnoses while eliminating the risks associated with surgical biopsies. In our study, we analyzed 500 prostate cancer samples and 150 normal samples, collected from nine hospitals. Blood and surgical specimens were obtained based on defined eligibility criteria. The study aimed to correlate mRNA genomic profiling with clinical and pathologic parameters. In blood samples, a 25-gene panel effectively distinguished prostate cancer patients from non-cancer individuals, achieving an AUC of 0.906 (sensitivity 90%, specificity 91%). Similar diagnostic performance was observed in tissue samples (AUC 0.9514, sensitivity 95%, specificity 94%). Notably, patients with Gleason scores >7 showed significantly higher expression of the gene panel compared to those with GS<7, underscoring the test's prognostic potential. Comparable

gene expression patterns between blood and tissue samples support the use of blood-based testing for screening, diagnosis, and risk assessment. These findings were further validated in a prospective study. Geneverify test demonstrated high accuracy in detecting early-stage prostate cancer with strong concordance to biopsy results. To our knowledge, this is the first real-time clinical validation of a blood-based, cell-free mRNA genomic test for prostate cancer screening. Our results indicate that mRNA genomic profiling from blood can accurately diagnose prostate cancer and help stratify patients into prognostic groups. This non-invasive method offers a promising alternative to traditional biopsy delivering faster, safer, and more accessible early detection, and paving the way for personalized treatment strategies. RNA-based cancer vaccine trials are currently underway, targeting a wide range of cancers. With promising results, many of these vaccines could receive FDA approval within the next five years or even sooner. These ground breaking therapies have the potential to revolutionize cancer treatment, offering new hope to every patient and significantly improving the lives of millions of patients.

Foutina Feghali[1], Erika Porcel[1], Pauline Maury[3], Ryoichi Hirayama[2], Sandrine Lacombe[1*]

[1]Université Paris-Saclay-CNRS, France
[2]QST-HIMAC, Japan
[3]Gustave Roussy, France

Biography: Sandrine Lacombe completed her PhD in chemical-physics, she conducted early research at the Fritz Haber Institute of the Max Planck Society under Nobel Laureate Professor G. Ertl. She later served as invited professor at the Sherbrooke University Hospital (Canada) and received an International Research Collaboration Award from the University of Sydney (Australia). Her research focuses on advancing particle therapy using theranostic nanoparticles. She contributed to 93 publications, supervised 11 PhDs. She currently direct the interdisciplinary center for innovative radiation therapies, iNanoTheRad. Former Vice-President for International Affairs of U. Paris-Saclay, she also offer her expertise to other institutions for the development of international and European strategies.

Theranostic nanoparticles combined particle therapy

The challenge of radiotherapy is to increase radiation damage on tumor whilst preserving healthy tissue. Particle therapy offers an alternative method whose ballistic properties improve tumor targeting. However, the development of this radiation molality is limited by the impact on the healthy tissues irradiated at the entrance of beam in the body. This work aims to enhance particle therapy's performance by adding Radioenhancing Nanoparticles (REN) that amplify ionizing radiation effects at the target where they concentrate. After a state-of -the-art of the current developments on NP-radiation combined protocols, the first results obtained by the team on human cells spheroids will be presented. It includes internalization and localization of the REN in the cells and their effect on cell survival treated by medical Carbon ion beam (290MeV/uma) with or without REN.

To understand how PtNPs interact with biomolecules to induce radio-enhancement, this study investigates the combined effect of PtNPs and ion irradiation on cell proliferation and DNA Double-Strand Break (DSB) repair in tumor spheroids.

Sergey Suchkov[1-12*], Hiroyuki Abe[6,13], David Smith[14], Shawn Murphy[15,16], William Thilly[17]

[1]N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia
[2]China Hong Kong Innovation International Business Association, Hong Kong
[3]New York Academy of Sciences, USA
[4]Russian Academy of Natural Sciences, Moscow, Russia
[5]EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU, Belgium
[6]ISPM (International Society for Personalized Medicine), Japan
[7]PMC (Personalized Medicine Coalition), Washington, DC, USA
[8]AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland
[9]ACS (American Chemical Society), Washington, DC, USA
[10]AHA (American Heart Association), Dallas, TX, USA
[11]ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA
[12]ISER (International Society for Eye Research), Anchorage, AK USA
[13]Abe Cancer Clinic, Tokyo, Japan
[14]Center for Cancer Genomics, Mayo Clinic, Rochester, MN, USA
[15]MGH, USA
[16]Harvard Medical School, Boston, MA, USA
[17]MIT, Cambridge, MA, USA

Biography: Sergey Suchkov was born in the City of Astrakhan, Russia, in a family of dynasty medical doctors. In 1980, gradu-ated from Astrakhan State Medical University and awarded with MD. In 1985, Suchkov maintained his PhD as a PhD student of Sechenov University and Institute of Medical En-zymology. In 2001, Suchkov maintained his Doc-tor Degree at the National Institute of Immunology, Russia. From 1989 through 1995, a Head of the Lab of Clinical Immunology, Helmholtz Eye Re-Search Institute in Moscow. From 1995 through 2004 - a Chair of the Dept for Clini-cal Immunology, Moscow Clinical Research Institute (MONIKI). In 1993-1996. At present, Dr Sergey Suchkov, MD, PhD, is: Professor in Medicine & Immunology, Director for Center for Biodesign of N.D. Zelinskii Institute for Organ-ic Chemistry of the Russian Academy of Sciences, Moscow, Russia. R&D Director, InMedStar, Russia-UAE. Sen-ior Scientific Advisor of China Hong Kong Innovation International Business Association, Hong Kong. Member, New York Academy of Sciences, USA. Member: EPMA (European Association for Predictive, Preventive and Per-sonalized Medicine), Brussels, EU. Member, ISPM (International Society for Personalized Medicine), Japan. Member, PMC (Personalized Medicine Coalition), Washington, USA. Member, AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland. Member, ACS (American Chemical Socie-ty), Washington, DC, USA. Member, AHA (American Heart Association), Dallas, TX, USA. Member, ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA. ISER (International Society for Eye Re-search), Anchorage, AK USA. Secretary General, United Cultural Convention (UCC), Cambridge, UK.

Personalized and Precision Medicine (PPM) through the view of biodesign-inspired translational research: An option for clinical oncologists, caregiv-ers and consumers to realize the potential of genomics-informed care to secure the human biosafety

A new systems approach to diseased states and wellness result in a new branch in the healthcare services, namely, Personalized and Precision Medicine (PPM). Meanwhile, the era of genomics-based medicine and thus genomics biomarkers promises to provide molec-ular tests that will permit PPM as applicable to Per-Sonalized & Precision Oncology (PPO). To achieve the implementation of PPM-guided oncology concept, it is necessary to create a fundamentally new strategy based upon the subclinical recognition of biopredictors (includ-ing genomics ones) of hidden abnormalities (pre-cancer conditions) long before the disease clinically manifests itself.

Every human has a unique genetic makeup that causes them to respond differently to can-cer. In this context, the genomic profiling can be done using genomic, transcriptomic, epige-nomic or metagenomic information, and will look at the genetic structure of the tumor. This helps us discover "actionable" mutations that can be targeted with therapy. These discover-ies can lead to new treatment recommendations that may effectively treat your cancer on a personalized level. Through those analyses, we can not only diagnose and classify cancer patients and/or pre-cancer persons-at-risk based on their comparative risk, but also monitor their response to emerging canonical, preventive or prophylactic therapies. Continued pro-gress using these methods will transform how we approach treatment modalities for cancer patients.

The advent of Next Generation Sequencing (NGS) and GWAS technologies has advanced our understanding of the intrinsic biology of different tumor types. Prospective randomized clinical trials will determine whether matching actionable aberration with targeted therapy will contribute to improve survival in patients with malignancies.

PPM globally holds great promise, especially in cancer therapy and control, where PPO would allow practitioners to use this information to optimize the targeted treatment of a pa-tient. PPO for groups of individuals would also allow for the use of population group specific diagnostic or prognostic cancer biomarkers. The integration of PPM-guided genomics into clinical practice is transforming treatment paradigms. Identification of oncogenes and tumor suppressor genes can become the stimulus for rational design of novel, selec-tive drugs that execute specific activity directed at underlying genetic aberrations. This information can be used to track the progress of cancer, and to establish the molecular basis for drug resistance and allow the targeting of the genes or pathways responsible for drug resistance.

The enormous development of biodesign-driven genomics research has raised great expec-tations concerning its impact on PPM aiming to customize medical practice with a focus on the individual, based on the use of genetic tests, identification of genomic biomarkers, and development of targeted drugs. In this sense, the impact of precision cancer pathology al-lows a modular approach, as its various aspects are under development in sometimes

unre-lated areas of PPM. Integration of the concepts will provide a true challenge for the future, re-quiring collaboration between clinicians, physiologists, pathologists, biodesigners and bio-engineers and remaining a real challenge to bioindustry.

Meanwhile, each decision-maker values the impact of their decision to use PPM and PPO on their own budget and well-being, which may not necessarily be optimal for society as a whole. It would be extremely useful to integrate data harvesting from different databanks for applications such as prediction and personalization of further treatment to thus provide more tailored measures for cancer patients and/or pre-cancer persons-at-risk resulting in improved outcomes, reduced adverse events, and more cost effective use of health care resources. A lack of medical guidelines has been identified by the majority of responders as the predominant barrier for adoption, indicating a need for the development of best practices and guidelines to support the implementation of PPM in clinical practice!

Stephan Bodis MD

University Zurich, IT'IS Foundation Board, Ethics Committee

Zurich, Switzerland

Biography: Stephan Bodis began his medical studies in 1978 at the University of Fribourg in Switzerland and completed them in 1984 at the University of Basel. He then worked in the Department of Internal Medicine at the Cantonal Hospital of Baden until 1987 and at the University Hospital of Zurich until 1989. From 1989 to 1991, he was a Fellow of the European Society for Medical Oncology (ESMO) and also a Research Fellow in the Department of Hematology/Oncology and Molecular Pharmacology at the Gustave Roussy Institute in Villejuif near Paris. This was followed by a clinical and research residency and fellowship in radiation oncology at the Joint Centre for Radiotherapy at Harvard Medical School in Boston and at the Massachusetts Institute of Technology, Department of Biology, Cambridge, until 1995. From 1995 to 2003, Bodis was a senior physician and head of the Laboratory of Molecular Radiobiology at the University Hospital of Zurich. In March 1998 he was appointed Privatdozent at the University of Zurich, and in 2004 he was appointed Titular professor. From 2003 to 2021, Stephan Bodis was Director of the Institute of Radiation Oncology at the Aarau Cantonal Hospital, and from 2015 to 2021, he was Head of the Radiation Oncology Centres at the Aarau and Baden Cantonal Hospitals. In September 2012, he became a member of the Faculty of Medicine at the University of Zurich (Associate Professor).

The art of patient conversation in oncology

A special effort is needed to improve patient information and patient conversation in radiation oncology. This focus does not only improve patient satisfaction but also contributes to overall quality care and outcome of radiation oncology therapies. With tight resources high quality time spent with patients is even more relevant. There is no reason for more random time and words. But we need to focus expertise, skills, empathy and novel tools like AI on our patients need.

Material/Methods: Beside systematic teaching, feed-back and outcome measurements on patient conversations we need complementary perspectives and tools. Fictitious "Narrative Oncology" based on patient health care staff interactions can provide such a different perspective while preserving the privacy. Microstories are maximally condensed as a story-tool and go to the core of a human interaction with minimal words. In this abstract two original microstories based on patient observations are depicted as an example. Further microstories within relevant background information's will be provided.

Results:

(1) The Consultation (Microstory)

The X-ray treatment was safely delivered today. The staff person explains the irradiation course for next week. The patient breathes calmly. The pulse is regular. Pearls of sweat and anxiety on the dry skin are dropping to the floor. A shy smile. A long handshake. Thank you so much. Who's next? Two minutes later an anxious voice in the door. The next patient is here. She needs you - now.

(2) Eternal Moment (FLASH Microstory)

Eight steps, 4 eyes, 2 hands, one hug, an eternal moment, for today. Patient and spouse can go home, for now, together. And come back tomorrow.

Summary: This presentation will depict selected microstories based on patient observations in an oncology environment. Then in a broader view the importance of how, why and when we share or words, our sentences, our information's with patients will be shared and discussed.

Thomas J. Webster

School of Health Sciences and Biomedical Engineering, Hebei University of Technology, Tianjin, China and Division of Pre-College and Undergraduate Studies, Brown University, Providence, RI, USA

Biography: Thomas J. Webster's (H index: 136; Google Scholar) degrees are in chemical engineering from the University of Pittsburgh (B.S., 1995; USA) and in biomedical engineering from RPI (Ph.D., 2000; USA). He has served as a professor at Purdue (2000-2005), Brown (2005-2012; 2021-present), and Northeastern (2012-2021; serving as Chemical Engineering Department Chair from 2012-2019) Universities and has formed over a dozen companies (with some acquired by Medtronic) who have numerous FDA approved medical products currently improving human health in over 30,000 patients with no failures. He is currently helping those companies and serves as a professor at Brown University, Saveetha University, Hebei University of Technology, UFPI, and others. Dr. Webster has numerous awards including: 2020, World Top 2% Scientist by Citations (PLOS); 2020, SCOPUS Highly Cited Research (Top 1% Materials Science and Mixed Fields); 2021, Clarivate Top 0.1% Most Influential Researchers (Pharmacology and Toxicology); 2022, Best Materials Science Scientist by Citations (Research.com); and is a fellow of over 8 societies. Prof. Webster is a former President of the U.S. Society for Biomaterials and has over 1,350 publications to his credit with over 70,000 citations. He was recently nominated for the Nobel Prize in Chemistry. Prof. Webster also recently formed a fund to support Nigerian student research opportunities in the U.S.

Controlling cells: One nanoparticle at a time

This presentation will cover a close to 30 year journey researching and commercializing nanotechnology for improving disease prevention, diagnosis, and treatment (including cancer) which has led to numerous products including nano spinal implants now in over 30,000 patients to date showing no signs of failure according to the FDA MAUDE database. Traditional orthopedic implants face a failure rate of 5–10% and sometimes as high as 60% for bone cancer patients. The talk will cover not only human clinical evidence of the unprecedented efficacy of nanotechnology in medicine but also fundamental evidence of how nanotechnology can be used clinically to kill cancer cells, bacteria, inhibit inflammation, and promote tissue growth (if needed) without drugs. This talk will also describe the future of nanotechnology and how it will in the not too distant future combat traditional failures in our global healthcare system including reversing the current decrease in global average life expectancy, creating a reactive compared to predictive healthcare system, transforming a healthcare system that relies too much on drugs and pharmaceutical agents to treat ailments,

facilitating a non-personalized healthcare system, combating increasing costs, treating a growing global population, and more through the future use of implantable nano sensors, 4D printed nano materials, smart nano materials, environmentally-friendly nanomaterials, and AI as well as other predictive models in medicine and more.

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ORAL PRESENTATIONS

Alessandra Storm Mauricio[1*], Jack Mc Williams[2], Leina Singh MD[3], Snehal Jadhav BDS, MPH[4]

[1]Christian Brothers High School, Sacramento, CA, USA
[2]Jesuit High School, Sacramento, CA, USA
[3]Kaiser Permanente, Sacramento, CA USA
[4]Verily Life Sciences, Raleigh, NC, USA

Cancer awareness in teens: A hybrid cross-sectional survey and informational campaign

Introduction: Cancer in Adolescents and Young Adults (AYAs) is a worldwide devastating disease. In the US, it is among the top four causes of AYA deaths. Cancer signs/symptoms are similar to common ailments, delaying diagnosis, thus life-threatening. Less than one-third of the adult population reports confidence in cancer signs/symptoms recognition, and less than half are aware of certain risk factors linked to cancer. We seek to define the perception of and awareness of cancer specifically in AYAs. These young people are the "lost tribe" in oncology.

The study describes the awareness of AYAs on cancer risk factors they can modify. We also investigate their knowledge on cancer signs/symptoms. Measurement of confidence in sharing said symptoms with their families and clinicians was made. Included in this research were opportunities for information dissemination regarding cancer care awareness.

Methods: Cross-sectional survey was administered to 1,155 AYAs aged 13–18 at a high school in Sacramento, CA for two weeks in August 2025. School-level institutional board review, student consent/assent and parental consents were obtained. Jot form instrument (San Francisco, CA) was used to collect data on demographics, cancer knowledge, signs/symptoms recognition, risk factor prevention, help-seeking attitudes, and preferred educational resources. Descriptive statistics used to summarize overall trends. Non-parametric tests (Mann-Whitney U, Kruskal-Wallis H) used to analyse differences by gender, grade level, ethnicity, and socioeconomic data.

Results: The statistic of 1 in 285 people being diagnosed with cancer before age 20 was at least moderately surprising to 70.9% of respondents. Change in appearance in a mole (14.7%), blood in urine or stools (13.1%), and seizures (10.5%) were least reported as cancer signs/symptoms. Numerous birthmarks (30.1%), close contact with cancer patients (21.2%), frequent common cold (14.4%) were identified as cancer risks.

Respondents preferred to learn about cancer through educational websites (93.8%), online videos (89.9%), instructor-led lectures (85.9%), and talking to cancer patients (84.3%). The two most common barriers for seeking medical evaluation for cancer were uncertainty if symptoms were serious (64.7%) and lack of confidence in discussing their symptoms (60.1%). 56.2% reported seeking help first from a doctor when suspecting cancer.

Females reported they think significantly about cancer (p=0.0041) and have higher surprise levels to the cancer statistic (p=0.0042) compared to males. Older students reported higher levels of thinking about cancer (p=0.0230) and confidence in making choices to lower cancer risk (p=0.02529) compared to younger students. Knowing someone with cancer correlates with high levels of thinking about cancer (p=0.0197).

Discussion and Conclusions: This is the first-ever hybrid activity of a cancer awareness survey and information campaign.

Male and younger populations may benefit from additional cancer education. Participants demonstrated high levels of recognition for several key cancer signs/symptoms and risk factors. However, more awareness is needed for less common signs/symptoms and cancer risk factor misconceptions. Respondents prefer digital and interactive resources and talking to cancer patients to increase their knowledge. Participants knowing people with cancer reported higher cancer awareness.

Therefore, we believe cancer patients and their families may potentially serve as important sources of cancer information for AYAs.

Biography

Alessandra Storm Mauricio loves being an analogical thinker–seeking connections between unrelated concepts and bridging disparate ideas in surprising ways. To Alessandra, it's delightfully mind-blowing! She believes fresh insights serve as the cornerstone of innovation and the development of solutions. Combining two different goals, research and information dissemination, she uncovered AYA's cancer awareness gaps and how cancer experts can bridge them. Equally important to her is to communicate scientific findings effectively with fellow investigators and the world. Alessandra aspires to grow as an inspired scientist promoting STEM literacy for everyone, especially to her future patients as a physician. She will champion them and their health.

Arthi Roy[1*], Arpita Kundu Riya[2], Chad Sultana[3], Abir Biswas[4], Moontasir Ahmed[5], Jannatara Tina[5]

[1]Pabna Medical College, Pabna, Bangladesh
[2]Dinajpur Medical College, Dinajpur, Bangladesh
[3]Holy Family Red Crescent Medical College, Dhaka, Bangladesh
[4]Sher-E- Bangla Medical College, Barishal, Bangladesh
[5]Tangail Medical College, Tangail, Bangladesh

Effect of Posaconazole compared to other antifungal drugs in survival & prevention of infection in patients with haematological malignancy: A systematic review

Introduction: Patients with acute leukemia who experience neutropenia after intensive chemotherapy have a high risk of developing Invasive Fungal Infections (IFIs), which can be life threatening. Although diagnostics and supportive care have been enhanced, IFIs continue to be a major problem. Therefore, prophylaxis with an antifungal agent is a vital part of management. This Systematic Review examined the effectiveness of Posaconazole vs. other antifungal agents for the prevention of IFIs and improved survival in haematological malignancy patients.

Methods: A Systematic Review that followed PRISMA guidelines, was completed using randomized controlled trials, cohort studies and observational studies that evaluated antifungal prophylaxis in acute leukemia and associated haematologic malignancies. The extracted data included the type of antifungal agent(s) used; the number of proven/probable IFIs; the number of breakthrough infections; overall survival; adverse events and treatment discontinuation. Since there was considerable heterogeneity among study design and outcome measures, findings were synthesized narratively.

Results: Posaconazole consistently demonstrated greater efficacy than both Fluconazole and Itraconazole for the prevention of IFIs, specifically invasive mold infections with a significant reduction in invasive aspergillosis. A number of studies demonstrated that patients receiving Posaconazole experienced improved overall survival, fewer breakthrough infections and less use of empirical antifungal therapy. Echinocandins, most notably Micafungin, were well-tolerated and demonstrated efficacy in certain clinical scenarios. Voriconazole continued

to be a viable option; however, due to toxicity and pharmacokinetic variability, it was often discontinued. Fluconazole was effective in preventing candida infections; however, it offered limited coverage for mold infections. Newer formulations of Posaconazole resulted in enhanced drug exposure and clinical usefulness.

Conclusion: The current evidence suggests that Posaconazole is a very effective prophylactic antifungal agent for high-risk patients with acute leukemia. It has broad-spectrum activity, predictable pharmacokinetics and demonstrates efficacy against invasive mold infections making it the first-line option for these patients; however, alternatives will need to be available for azole-intolerant patients. Multicenter studies will be required to define optimal patient selection criteria and monitor antifungal resistance.

Biography

Arthi Roy is a Medical Intern at Pabna Medical College Hospital, Bangladesh. She earned her Bachelor of Medicine and Bachelor of Surgery (MBBS) from Pabna Medical College, Bangladesh in November 2025. She is at the early stage of her research career with a developing interest in Oncology, focusing on supportive cancer care. Her work includes research on the prevention of infections in patients with hematological malignancies, aiming to improve patient outcomes through evidence-based clinical practices.

Can Küçük[1*], Burcu Akman[2,3], Xiaozhou Hu[4], Tevfik Hatipoğlu[2,3], Ahmet Şeyhanlı[5,6], Arda Ceylan[2,3], Bircan Yılmaz[1], Osman Can Öztürk[5], Taner Kemal Erdağ[7], Güner Hayri Özsan[5]

[1]Department of Medical Biology, Faculty of Medicine, Dokuz Eylül University, 35340 İzmir, Türkiye
[2]İzmir International Biomedicine and Genome Institute, Dokuz Eylül University, 35340 İzmir, Türkiye
[3]İzmir Biomedicine and Genome Center, 35340 İzmir, Türkiye
[4]Department of Medical Biochemistry, Faculty of Medicine, Dokuz Eylül University, 35340 İzmir, Türkiye
[5]Department of Hematology, Faculty of Medicine, Dokuz Eylül University, 35340 İzmir, Türkiye
[6]Department of Hematology, Faculty of Medicine, Gazi University, 06560 Ankara, Türkiye
[7]Department of Otorhinolaryngology, Faculty of Medicine, Dokuz Eylül University, 35340 İzmir, Türkiye

Genetic and epigenetic alterations of *SOX7* in multiple myeloma and allied neoplasms

Multiple Myeloma (MM) is one of the most frequent hematological malignancies. Its incidence and mortality rate is globally increasing. Most MM patients experience relapse, which leads to poor prognosis. The genes associated with MM development or relapse have not been totally elucidated yet. The recurrently deleted 8p23.1 locus in MM includes tumor suppressor gene candidates. As a transcription factor, *SOX7* was shown to be downregulated through genetic or epigenetic alterations in different cancer types. However, the aberrations of *SOX7* were not evaluated in multiple myeloma or allied plasma cell neoplasms such as Smoldering MM (SMM) or Plasma Cell Leukemia (PCL). In this study, we reanalyzed the publicly available datasets to evaluate *SOX7* copy number, promoter methylation, and transcript expression levels in MM or related neoplasms. Furthermore, we performed qPCR and qRT-PCR analyses with the in-house MM cohort to cross-validate *SOX7* copy number and transcript level estimates. We observed frequent *SOX7* deletions in newly diagnosed and relapsed MM cases. The *SOX7* promoter was hypermethylated in most MM cell lines as well as many MM and PCL patient tumor samples. Consistent with these aberrations, *SOX7* was transcriptionally silent in MM cell lines and under expressed in MM and high-risk SMM cases. When we analyzed patient-matched MM cases, we observed moderate levels of positive correlation between *SOX7* copy numbers in tumor tissues obtained at diagnosis and relapse. In these patient-matched samples, *SOX7* deletion and promoter methylation levels had a tendency to be mutually exclusive. As a noteworthy

observation, *SOX7* promoter methylation levels were significantly higher in relapsed cases compared to the diagnostic ones. Given that small *SOX7* mutations were very rare, deletion and promoter hyper methylation may be the main mechanisms for *SOX7* under expression in MM and allied neoplasms. These genetic and epigenetic aberrations may be pathologically and clinically significant in these plasma cell neoplasms.

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.

Dr. Chockalingam S

Cell Signaling Research Laboratory, National Institute of Technology Warangal, India

Discovery of an R-spondin–ALK–Wnt axis: Expanding the oncogenic landscape of anaplastic lymphoma kinase

Anaplastic Lymphoma Kinase (ALK), a receptor tyrosine kinase, has emerged as a critical driver of malignancy in diverse cancers such as anaplastic large cell lymphoma, non-small cell lung cancer, and neuroblastoma. Although ALK is normally activated by its ligands ALKAL1 and ALKAL2, cancer cells frequently exploit ligand-independent mechanisms, most notably by forming oncogenic fusions with other proteins that preserve the kinase activity of ALK. Aberrant ALK signaling stimulates well-characterized signaling cascades including Ras/Raf/MEK/ERK, JAK/STAT3, and PI3K/AKT. More recently, evidence has hinted at a possible connection between ALK and the Wnt pathway, though the molecular basis remains unclear. To study the role of ALK in modulating Wnt signaling, we employed a network-driven gene association approach with GeneMANIA, which identified R-spondins, the known amplifiers of Wnt signaling, as a putative ALK partners. Protein–protein interaction analysis predicted that R-spondins engage the TNF-like and EGF-like domains of ALK, analogous to the known ALKAL2 recognition regions. Docking and molecular dynamics simulations further supported the stable and energetically favorable binding of these complexes. We validated our *in-silico* results in neuroblastoma cell lines IMR32 and SH-SY5Y where full-length ALK is implicated in carcinogenesis. The results showed that when these cells are treated with a combination of Wnt3a and Rspo2, the Wnt pathway activity was enhanced. However, upon inhibition of ALK, the Wnt signaling activity reduced markedly. Our findings indicate the existence of R-spondin-ALK-Wnt signaling axis as an alternate to the already known R- spondin-LGR-Wnt signaling. This crosstalk broadens the biological scope of ALK beyond its canonical oncogenic role and suggests opportunities for therapeutic strategies that co-target ALK and Wnt signaling in ALK-driven tumors.

Biography

Dr. Chockalingam is an Assistant Professor in the Department of Biotechnology at the National Institute of Technology, Warangal, India, where he leads the Cell Signaling Research Laboratory. He earned his PhD from the Indian Institute of Technology Guwahati under the supervision of Prof. Sidhartha Sankar Ghosh. Dr. Chockalingam subsequently carried out postdoctoral research in Prof. Gopinath's laboratory at the Indian Institute of Technology Roorkee and in Prof. Karim Malik's laboratory at the University of Bristol, United Kingdom. His research focuses on cancer cell signaling and nanobiotechnology.

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

A new sensing mechanism for chromatin bridges by the abscission checkpoint in human cancer cells

Chromatin bridges are DNA strings that link anaphase poles or daughter nuclei and are associated with carcinogenesis. DNA bridges occur from the separation of twisted, linked chromatin that is caused by problems in DNA replication or incomplete DNA decatenation.

In response to chromatin bridges, cells delay abscission by activating the abscission checkpoint which delays completion of cytokinesis in order to prevent chromatin breakage or tetraploidization by furrow regression. In mammalian cells, the abscission checkpoint is dependent on Aurora B kinase activation. We recently showed that in cytokinesis with chromatin bridges a biochemical pathway including MRN complex, ATM and Chk2 promotes INCENP localization to the midbody to delay abscission and prevent chromatin breakage.

In this study we used site directed mutagenesis, RNA silencing, time lapse and confocal microscopy, FISH, BrdU labeling, TUNEL assay and differential retention assay.

Here, we show that spontaneous or replication stress-induced chromatin bridges derived from catenated DNA exhibit “knots” of tangled, overtwisted DNA next to the midbody. Topoisomerase IIα (Top2a), an enzyme that relaxes supercoils and untangles catenated DNA forms irreversible Top2-DNA cleavage complexes (Top2ccs) on DNA knots. Inhibition of Top2a results in diminished localization of Rad17, MRN, ATM, Chk2 and CPC complex to the DNA bridges and induces chromatin bridge breakage. Furthermore, proteolytic degradation of Top2ccs is required for localization of Rad17 to the bridge DNA. In turn, Rad17 promotes recruitment of the MRN complex to DNA knots and downstream abscission checkpoint signaling to delay abscission and prevent chromatin bridge breakage in cytokinesis. In

contrast, chromatin bridges generated by dicentric chromosomes do not exhibit DNA knots or Top2ccs next to the midbody, and fail to recruit Top2a, Rad17 and other downstream proteins and are unable to induce an abscission delay.

Our results describe a novel mechanism by which the abscission checkpoint detects chromatin bridges in human cells, through generation of irreversible Top2ccs on DNA knots. Because chromosomal instability is a major cause of cancer, identifying new genes that protect genome integrity is very important for cancer research.

The research project was supported by Worldwide Cancer Research (Project 25-0103), Fondation Santé, the Hellenic Foundation for Research and Innovation (H.F.R.I.) under the "2nd Call for H.F.R.I. Research Projects to support Post-Doctoral Researchers" (Project Number: 629) and the "2nd Call for H.F.R.I. Research Projects to support Faculty Members and Researchers" (Project Number: 2486).

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 has published 18 publications including Nature Communications, EMBO Journal, Journal of Cell Biology, Journal of Cell Science and others. Her publications have received >600 citations so far. Dr. Eleni is currently a member of FEBS, AACR, EACR and Royal Society of Biology.

Feng Yan*, Xun Tang

Department of Clinical Laboratory, the Affiliated Cancer Hospital of Nanjing Medical University and Jiangsu Cancer Hospital and Jiangsu Institute of Cancer Research, Nanjing 210009, China

tRNA-derived fragment *3'tRF-AlaAGC* modulates cell *Chemoresistance* and M2 macrophage polarization via binding to TRADD in breast cancer

Background: Drug resistance, including Adriamycin-based therapeutic resistance, remains a challenge in Breast Cancer (BC) treatment. Studies have revealed that macrophages could play a pivotal role in mediating the *Chemoresistance* of cancer cells. Accumulating evidence suggests that *tRNA*-Derived small RNAs (tDRs) are associated the physiological and pathological processes in multiple cancers. However, the underlying mechanisms of tDRs on *Chemoresistance* of BC in tumor-associated macrophages remain largely unknown.

Methods: The high-throughput sequencing technique was used to screen tDRs expression profile in BC cells. Gain-and loss-of-function experiments and xenograft models were performed to verify the biological function of *3'tRF-Ala-AGC* in BC cells. The CIBERSORT algorithm was used to investigate immune cell infiltration in BC tissues. To explore the role of *3'tRF-Ala-AGC* in macrophages, M2 macrophages transfected with *3'tRF-Ala-AGC* mimic or inhibitor were co-cultured with BC cells. Effects on Nuclear factor-κb (NF-κb) pathway were investigated by NF-κb nuclear translocation assay and western blot analysis. RNA pull-down assay was performed to identify *3'tRF-Ala-AGC* interacting proteins.

Results: A *3'tRF* fragment of *3'tRF-AlaAGC* was screened, which is significantly overexpressed in BC specimens and Adriamycin-resistant cells. *3'tRF-AlaAGC* could promote cell malignant activity and facilitate M2 polarization of macrophages in vitro and *in vivo*. Higher expression of M2 macrophages were more likely to have lymph node metastasis and deeper invasion in BC patients. Mechanistically, *3'tRF-Ala-AGC* binds Type 1-associated death domain protein (TRADD) in BC cells, and suppression of TRADD partially abolished the enhanced effect of

3'tRF-Ala-AGC mimic on phenotype of M2. The NF-κb signaling pathway was activated in BC cells co-cultured with M2 macrophages transfected with *3'tRF-AlaAGC* mimic.

Biography

Feng Yan is currently a professor and doctor's supervisor at Department of Clinical Laboratory, the Affiliated Cancer Hospital of Nanjing Medical University. She has long been engaged in research on the mechanisms of malignant tumor development and related biomarkers. Feng Yan has published 67 papers as first author or corresponding author (51 of which are SCI-indexed), obtained 6 authorized national invention patents, co-authored one English monograph (Elsevier: *Immunosensing for Detection of Protein Biomarkers*, 2017), edited two Chinese monographs, and contributed to one additional Chinese monograph.

Han Sun[1*], Xiangyin Meng[2], Yanni Zhang[1], Ye Yuan[3], Hujian Hong[1], Yanli Qu[1]

[1]Department of Radiotherapy, Cancer Hospital of China Medical University, Liaoning Cancer Hospital & Institute, Cancer Hospital of Dalian University of Technology, Shenyang, China

[2]The Fourth Affiliated Hospital of China Medical University, China

[3]Department of Pharmaceutical Machinery, General Hospital of Shenyang Coal Industry Group of Liaoning Health Industry Group, Shenyang, China

A prospective study of tislelizumab combined with radiotherapy in unresectable or recurrent advanced hepatocellular carcinoma: Efficacy and immunological safety analysis

Background: Radiotherapy (RT) can induce immunogenic cell death and enhance tumor antigen release and presentation, thereby augmenting the host anti-tumor immune response. The combination of PD-1 inhibitor tislelizumab with RT may produce synergistic effects and improve outcomes in advanced or recurrent Hepatocellular Carcinoma (HCC). This study aimed to evaluate the efficacy and immunological safety of tislelizumab combined with RT in patients with unresectable or progressive recurrent HCC.

Methods: This was a single-center, single-arm, prospective clinical study. From march 2023 to march 2025, 32 patients with unresectable or recurrent progressive HCC were enrolled. All patients received local RT (30–50Gy) combined with tislelizumab (200mg every 3 weeks) administered concurrently or sequentially for at least one cycle. The primary endpoints were immune-related adverse events (irAEs) and Treatment-Related Adverse Events (TRAEs). Secondary endpoints included Objective Response Rate (ORR), Disease Control Rate (DCR), Progression-Free Survival (PFS), Overall Survival (OS), and Duration of Response (DOR).

Results: A total of 32 patients were enrolled, with a median age of 59 years (range 43–75); 28 were male and 4 were female. The clinical stages included IIIA (n=3), IIIB (n=17), and IVA (n=12), with 29 patients presenting with Portal Vein Tumor Thrombus (PVTT). The incidence of any-grade TRAEs was 96.9% (31/32), mainly fatigue (65.6%), decreased appetite (75.0%), hepatic

dysfunction (87.5%), and hematologic toxicity (anemia 6.3%, thrombocytopenia 18.8%). Grade ≥3 TRAEs occurred in 15.9% of patients, with no treatment discontinuation or death due to adverse events. The incidence of irAEs was 96.9%, including hepatotoxicity (50.0%), endocrine disorders (31.2%), mild diarrhea/colitis (34.4%), and skin reactions (9.4%), with grade ≥3 irAEs in 21.9% of patients.

The Overall Response Rate (ORR) was 37.5%, and the Disease Control Rate (DCR) was 65.6%. The median PFS was 7.3 months, with a 6-month PFS rate of 47.1%. The 6-month OS rate was 86.7%, and the median OS had not yet been reached at data cutoff.

Conclusions: Tislelizumab combined with radiotherapy demonstrated promising efficacy and manageable immunological safety in patients with unresectable or recurrent advanced HCC. This combined approach provides a potential therapeutic option and warrants further validation in multicenter randomized controlled trials.

Biography

Dr. Han Sun is an attending radiation oncologist at Liaoning Cancer Hospital, specializing in the clinical management and research of hepatocellular carcinoma. Her work focuses on optimizing radiotherapy techniques and developing multidisciplinary treatment approaches to improve therapeutic efficacy and patient outcomes. Dr. Sun has been actively involved in clinical studies exploring precision radiotherapy and personalized treatment strategies for liver cancer.

Hoang Minh Quan Pham[1,2*], Po-Hao Feng[3], Chia-Ling Chen[3], Kang-Yun Lee[3], Chiou-Feng Lin[3]

[1]International Ph.D. Program in Medicine, College of Medicine, Taipei Medical University, Taipei 11031, Taiwan
[2]Department of Oncology, Faculty of Medicine, Can Tho University of Medicine and Pharmacy, Can Tho, Vietnam
[3]Taipei Medical University, Taipei 11031, Taiwan

BCL2A1 expression in CD8⁺ T cells is associated with survival and immune checkpoint blockade response in lung adenocarcinoma

Background: Biomarkers associated with response to Immune Checkpoint Blockade (ICB) in Lung Adenocarcinoma (LUAD) remain incompletely defined. This study examined the transcriptional landscape of BCL2A1 in CD8⁺ T cells and evaluated its association with clinical outcomes in ICB-treated patients.

Results: Single-cell analysis revealed a distinct enrichment of BCL2A1 in tissue-resident memory and proliferating CD8⁺ T cell subsets, which appeared preferentially expanded in ICB responders. Cell-cell communication inference indicated that these BCL2A1-high CD8⁺ T cells possess high outgoing signaling potential ($p=0.0278$), particularly involving the Macrophage Migration Inhibitory Factor (MIF) pathway. Clinically, high BCL2A1 expression was significantly associated with improved survival in ICB-treated patients ($HR=0.43$, $p< 0.05$), an association not observed in non-ICB cohorts. Furthermore, we developed a "tri-marker" model combining BCL2A1, Programmed death-ligand 1 (PD-L1), and a 27-gene HOT score. This model demonstrated robust predictive performance (Discovery AUC=0.826; Validation macro-AUC=0.774), outperforming PD-L1 alone and established signatures such as Tumor Immune Dysfunction and Exclusion (TIDE), Immunophenoscore (IPS), Tumor Inflammation Signature (TIS), and Interferon-Gamma (IFNG). Cross-platform simulations suggested high reproducibility ($\rho=0.982–0.993$).

Conclusions: BCL2A1 marks a transcriptionally distinct subset of CD8⁺ T cells associated with survival outcomes and inferred intercellular signaling activity in LUAD treated with ICB. Integration of BCL2A1 into a multi-marker framework may support improved stratification of patients receiving immunotherapy, warranting further prospective validation.

Biography

Quan Pham is a third-year PhD student at Taipei Medical University and a practicing clinical physician treating patients with lung cancer in Vietnam. His research focuses on cancer immunology and translational bioinformatics, integrating bulk and single-cell transcriptomic analyses to investigate immune biomarkers associated with response to immune checkpoint blockade. Through his dual roles in clinical oncology and computational research, he aims to bridge biological insights with clinically relevant stratification strategies for cancer immunotherapy.

Jiazhou Ye[1,2,3*], Rongyun Mai[1,2], Rong Liang[2,3,4]

[1]Department of Hepatobiliary Surgery, Guangxi Medical University Cancer Hospital, Nanning 530021, China
[2]Guangxi Liver Cancer Diagnosis and Treatment Project Technology Research Center, Nanning 530021, China
[3]Guangxi Key Laboratory of Basic and Translational Research for Colorectal Cancer, Nanning 530021, China
[4]Department of Digestive Oncology, Guangxi Medical University Cancer Hospital, Nanning 530021, China

Integrating single-cell and spatial transcriptomics to uncover and elucidate GP73-mediated pro-angiogenic regulatory networks in hepatocellular carcinoma

In this study we are the first worldwide to reveal a GP73-mediated regulatory network that promotes tumor angiogenesis and progression in Hepatocellular Carcinoma (HCC) under hyperxia stress. Our findings demonstrated that GP73 plays crucial roles in tumor angiogenesis niche and exhibits favourable anti-angiogenic potential, highlighting its therapeutic relevance for HCC treatment.

HCC was characterized as being hyper vascular. In the present study, we generated a single-cell spatial transcriptomic landscape of the vasculogenic etiology of HCC and illustrated overexpressed Golgi Phosphoprotein 73 (GP73) HCC cells exerting cellular communication with vascular endothelial cells with high pro-angiogenesis potential via multiple receptor–ligand interactions in the process of tumor vascular development. Specifically, we uncovered an interactive GP73-mediated regulatory network coordinated with c-Myc, lactate, Janus Kinase 2/Signal Transducer and Activator of Transcription 3 (JAK2/STAT3) pathway, and Endoplasmic Reticulum Stress (ERS) signals in HCC cells and elucidated its proangiogenic roles *in vitro* and *in vivo*. Mechanistically, we found that GP73, the pivotal hub gene, was activated by histone lactylation and c-Myc, which stimulated the phosphorylation of downstream STAT3 by directly binding STAT3 and simultaneously enhancing Glucose-Regulated Protein 78 (GRP78)-induced ERS. STAT3 potentiates GP73-mediated pro-angiogenic functions. Clinically, serum GP73 levels were positively correlated with HCC response to anti-angiogenic regimens and were essential for a prognostic nomogram showing good predictive performance for determining 6-month and 1-year survival in patients with HCC treated with anti-angiogenic

therapy. Taken together, the aforementioned data characterized the pro-angiogenic roles and mechanisms of a GP73-mediated network and proved that GP73 is a crucial tumor angiogenesis niche gene with favorable anti-angiogenic potential in the treatment of HCC.

Biography

Jiazhou Ye M.D., is a Professor of Hepatobiliary Surgery at the Guangxi Medical University Cancer Hospital, China (also known as Guangxi tumor institute/Guangxi cancer center, China). He has been awarded multiple Chinese national science grants for liver cancer research projects. Jiazhou Ye serves as the chief secretary of Integrated Prevention and Screening Committee for Liver Cancer, China Anti-Cancer Association. With over 40 publications as first or corresponding author, his works has been cited by the European Association for the Study of the Liver (EASL) Clinical Practice Guidelines and other four clinical guidelines or expertise consensus on the HCC management.

Chengbing Jin*, Yonghong Tang
The Second Affiliated Hospital of Chongqing Medical University, Chongqing, 400010, China

Ultrasound-guided high-intensity focused ultrasound ablation for pancreatic cancer: Follow-up and clinical analysis

Objective: To evaluate the efficacy and safety of high-intensity focused ultrasound ablation in the treatment of pancreatic cancer and to analyze relevant prognostic factors.

Methods: A retrospective analysis was conducted on 68 patients with pancreatic cancer who underwent high-intensity focused ultrasound treatment. Ablation effects and adverse reactions related to HIFU were evaluated after the procedure. The Kaplan-Meier method was used for survival analysis. Univariate analysis of prognostic factors was performed using the log-rank test, and multivariate analysis was conducted using the COX regression model.

Results: The adverse events related to HIFU were mostly mild to moderate (CTCAE grades 1–2), with no severe adverse events (CTCAE grades 4–5) observed. The overall tumor response rate was 54.4%, and the disease control rate was 88.3%. The median progression-free survival was 4 months. The 6-month, 1-year, and 2-year progression-free survival rates were 36.8%, 11.8%, and 1.5%. The median overall survival was 13.1 months, with 6-month, 1-year, and 2-year overall survival rates of 81.0%, 62.3%, and 15.2%, respectively. Univariate analysis of prognostic factors of PFS showed that tumor location, tumor size of the pancreas, T stage, N stage, M stage, TNM staging, vascular invasion, liver metastasis, extrahepatic metastasis, tumor response, and combination therapy had significant difference ($P<0.05$). Multivariate analysis showed that TNM stage, liver metastasis, extrahepatic metastasis, tumor response, and combination therapy were independent prognostic factors for PFS ($P <0.05$). Multivariate analysis of risk factors influencing OS showed that TNM stage, liver metastases, extrahepatic metastases, prior antitumor therapy, HIFU sessions and combination therapy were independent prognostic factors ($P<0.05$).

Conclusion: High-Intensity focused ultrasound demonstrates favorable safety and efficacy in the treatment of pancreatic cancer. Liver metastases, extrahepatic metastases, TNM staging, combination therapy, prior antitumor treatment, tumor response, and HIFU sessions were identified as independent prognostic factors. Among these, combination therapy and repeated HIFU sessions were protective prognostic factors.

Keywords: High-Intensity Focused Ultrasound, Pancreatic Cancer, Prognostic Factors, Safety, Efficacy.

Biography

Dr. Jin Chengbing is an associate professor, MD, and a dedicated High Intensity Focused Ultrasound ablation (HIFU) specialist with over 20 years of experience, especially with a rich clinical experience and active research in HIFU for malignant solid tumors such as liver cancer and pancreatic cancer. As first author or corresponding author, several HIFU-related research papers were published including at Ultrasound in Medicine and Biology, European Journal of Radiology and Frontiers in Oncology.

Jinyan Feng*, Xianfu Wei, Long Chen, Houzhi Yang, Yiqin Li, Yongheng Liu, Xiuxin Han, Guowen Wang

Department of Bone and Soft Tissue Tumors, Tianjin Medical University Cancer Institute and Hospital, Tianjin, China

Integrative multi-omics reveals metabolic–stemness coupling and novel therapeutic targets in osteosarcoma chemoresistance

Chemoresistance remains the predominant cause of treatment failure in Osteosarcoma (OS), yet its underlying molecular circuitry remains poorly defined. To address this gap, we conducted a series of mechanistic and clinically informed studies integrating molecular biology, epitranscriptomics, and single-cell transcriptomics to systematically decode resistance plasticity in OS. Our work demonstrates that dysregulation of ubiquitin-mediated proteostasis plays a central role in chemotherapy resistance. We identified the E3 ubiquitin ligase SOCS1 as a key suppressor of resistance, which is frequently downregulated in chemoresistant OS tissues and cisplatin-resistant cell models. Mechanistically, SOCS1 promotes K63-linked ubiquitination and proteasomal degradation of ACTN4. Loss of SOCS1 stabilizes ACTN4, thereby enhancing tumor stemness, self-renewal capacity, and tolerance to cisplatin. Restoration of SOCS1 expression or inhibition of ACTN4 reverses these phenotypes, establishing the SOCS1–ACTN4 axis as a druggable resistance driver with clinical relevance. In parallel, we uncovered an epitranscriptomic mechanism of metabolic adaptation in OS chemoresistance. Our findings demonstrate that METTL3-mediated m^6A modification stabilizes the oncogenic long noncoding RNA LINC00520 in a YTHDF2-dependent manner. LINC00520 binds and stabilizes ENO1 by preventing FBXW7-mediated ubiquitination, leading to enhanced glycolytic flux and supporting cisplatin resistance under metabolic stress. Targeting this METTL3/LINC00520/ENO1 glycolytic axis suppresses tumor growth and restores drug sensitivity in vivo, highlighting an RNA modification–driven resistance pathway. To characterize resistance heterogeneity in the clinical setting, we performed single-cell RNA sequencing on chemotherapy-resistant OS patient samples collected at our centre. We identified highly mutated malignant subpopulations of pre-osteoblast origin

that emerged under chemotherapy selection. These resistant cell states demonstrated strong cell–cell communication with Inflammatory Cancer-Associated Fibroblasts (iCAFs) and exhausted $CD8^+$ T cells, particularly through MIF–CD74 and CXCL signaling, promoting immune evasion and survival under chemotherapeutic stress. These findings suggest that resistance maintenance is not solely tumor-intrinsic but also reinforced by tumor–stroma–immune crosstalk within the OS microenvironment. Collectively, these works establish a multi-dimensional model of chemoresistance in osteosarcoma driven by ubiquitination failure, epitranscriptomic reprogramming, metabolic adaptation, and immune remodelling. By decoding resistance as a coordinated and adaptive network rather than a single-pathway event, our findings provide a mechanistic foundation for multi-axis therapeutic strategies targeting protein stability, RNA modification, and micro environmental signaling to overcome drug resistance in OS.

Biography

Jinyan Feng Ph.D. in Oncology, is an Assistant Research Fellow at Tianjin Medical University Cancer Institute and Hospital. She mainly focus on tumor chemoresistance, osteosarcoma biology, and translational oncology. Her work integrates multi-omics analysis with mechanistic validation to investigate ubiquitination signaling, RNA modification, and tumor metabolic reprogramming. Jinyan Feng has published over ten SCI papers as first or corresponding author in journals including Hepatology, Cancer Letters, Theranostics, Acta Pharmacologica Sinica, Frontiers in Immunology, and International Journal of Cancer. Her research work was funded by the National Natural Science Foundation of China and provincial programs.

Jinzhao Song

Hangzhou Institute of Medicine, Chinese Academy of Sciences, Zhejiang Cancer Hospital, Hangzhou, Zhejiang 310022, China

Programmable enzyme-enabled ultra-sensitive liquid biopsy for early detection of tumor MRD

Minimal Residual Disease (MRD) can persist in cancer patients even after potential cure, resulting in cancer recurrence and contributing to the low five-year survival rate of cancer patients. Currently, circulating tumor DNA (ctDNA) is the preferred biomarker for MRD detection. However, due to the extremely similar nucleic acid sequences, wild type nucleic acid existing in body fluids in large quantities poses a big challenge to the highly sensitive detection of ctDNA.

We developed a new ultra-sensitive ctDNA detection method using programmable nuclease and dubbed it PASEA (Programmable Enzyme-Assisted Selective Exponential Amplification), which, in the process of nucleic acid amplification, wildtype nucleic acid was specifically cleaved by programmable endonuclease, including Argonaute and CRISPR-Cas systems, to achieve the specific exponential enrichment of ctDNA, so that the detection sensitivity could be lower than 0.01%MAF. We conducted a clinical evaluation of PASEA for tumor efficacy monitoring and MRD detection through follow-up monitoring of patients who received chemotherapy and surgery. Our analysis revealed that PASEA is a powerful tool for predicting cancer recurrence and guiding personalized treatment, offering significant clinical benefits. We also observed that the detection rate of ctDNA in pancreatic cancer was closely linked to treatment status, efficacy and metastatic site.

Biography

Jinzhao Song is a Professor at the Hangzhou Institute of Medicine (HIM), Chinese Academy of Sciences, Zhejiang Cancer Hospital. Prior to this role, he served as a Research Assistant Professor at the University of Pennsylvania, School of Engineering and Applied Science. Dr. Song received his Ph.D. from the Institute of Chemistry, Chinese Academy of Sciences (ICCAS). With over 15 years of experience, he has focused on developing next generation molecular diagnostic systems based on isothermal nucleic acid amplification; microfluidic chip that utilize smartphones for detection, analysis, result recording and sharing; and programmable enzymes such as Argonaute and CRISPR-Cas proteins. Dr. Song has co-authored 51 peer-reviewed papers with H-index 23, 15 of which he was the lead author, and was named as co-inventor in 9 granted patents and 10 patent applications. He is a recipient of the NIH K01 Research Scientist Development Award, as well as a PI/co-PI of an R21 NIH grant and two venture incubation programs. His contributions to the field of molecular diagnostics earned him the AACC's "2019 Young Investigator Award for Outstanding Research in Personalized Medicine." Additionally, Dr. Song founded EzDx Technology Inc., an enterprise dedicated to the commercialization and widespread adoption of these cutting-edge diagnostic tools, thereby setting a new standard in healthcare diagnostics and personalized medicine.

Qiang Yang[1,2], Ya Ma[2], Menghang Zu[2,3], Xinghong Cai[2], Qiang Gao[2], Yingui Cao[2], Ga Liu[2], Yajun Wang[3], Bo Xiao[2,3], Jun Zhang[1*]

[1]Clinical Center for Tumor Therapy, The Second Affiliated Hospital of Chongqing Medical University, Chongqing 400010, China

[2]State Key Laboratory of Resource Insects, College of Sericulture, Textile, and Biomass Sciences, Southwest University, Chongqing 400715, China

[3]Department of Pharmacy, Personalized Drug Therapy Key Laboratory of Sichuan Province, Sichuan Provincial People's Hospital, School of Medicine, University of Electronic Science and Technology of China, Chengdu 610054, China

Platinum micromotors for mucosal penetration and colorectal cancer therapy

The therapeutic outcomes of medications were restricted by the colonic mucosal barrier during the treatment of Colorectal Cancer (CRC). Micro/nanomotors can overcome the mucus barriers to reach deep colorectal tumors. In this study, we constructed a novel microsized PLGA-Pt Micromotor (MM) driven by hydrogen peroxide (H_2O_2) to enhance drug delivery to the CRC tissues and achieve effective antitumor therapy. The PLGA-Pt MMs actively traversed the colonic mucosal barrier with the assist of gas propulsion, while continuously releasing Pt 2^+ ions within the tumor microenvironment. *In vitro* studies revealed that the PLGA-Pt MMs exhibited rapid movement in the presence of H_2O_2, achieving superior colonic mucosal penetration. It effectively delivered Pt 2^+ ions to the nuclei, forming DNA-Pt adducts that induced significant DNA damage and apoptosis of CRC cells. *In vivo* studies showed the PLGA-Pt MM significantly suppressed orthotopic tumor growth and activated antitumor immunity, enhancing the therapeutic effect against CRC. This study presents a micromotor capable of overcoming mucosal barriers for efficient treatment of orthotopic CRC.

Keywords: Colonic Mucosal Penetration, PLGA-Pt Micromotor, H_2O_2-Driven, Colorectal Cancer.

Biography

Dr. Zhang is a medical doctor with a Ph.D., specializing in individualized treatment strategies for digestive system tumors. Clinically, his core focus lies in establishing a comprehensive diagnosis and treatment system that integrates chemotherapy, targeted immunotherapy, and minimally invasive interventional therapies. He has published multiple SCI papers in journals such as International Journal of Oncology, Shock, Cancer Medicine, and International Journal of Pharmaceutics. Through these works, Dr. Zhang has advanced understanding of liver cancer stem cell biology, identified critical illness biomarkers, and developed image guided HIFU ablation strategies, demonstrating a strong commitment to translational research that bridges basic science and clinical oncology.

K R Muralidhar
Karkinos Healthcare Private Limited, India

Clinical interpretation of whole genome sequencing to assess radiation treatment effects

Introduction and Objective: Radiation therapy is a cornerstone of cancer treatment, yet its efficacy is often limited by inherent or acquired tumour radio resistance and the potential for long-term adverse effects. Understanding the genomic alterations induced by radiation and their impact on tumour evolution is crucial for optimizing therapeutic strategies and predicting patient outcomes. These abstract outlines a framework for the clinical interpretation of Whole Genomic Sequencing (WGS) data obtained from tumours post-radiation treatment, with the primary objective of elucidating the effects of radiation on the tumour genome.

Materials and Methods: WGS offers an unparalleled resolution to identify diverse genomic changes, including Single Nucleotide Variants (SNVs), small insertions/deletions (indels), Copy Number Variations (CNVs), and Structural Variants (SVs), as well as their spatial and temporal distribution within the tumour. Post-radiation WGS analysis will focus on identifying radiation-induced mutational signatures, which are distinct patterns of DNA damage and repair errors characteristic of ionizing radiation exposure.

Results and Discussions: Furthermore, we will assess changes in driver gene mutations, pathways associated with DNA Damage Response and Repair (DDR), immune evasion, and cell cycle regulation. Comparative analysis of pre-and post-treatment WGS data, where available, will enable the tracking of clonal evolution, selection of resistant subclones, and the emergence of novel therapeutic targets. The integration of WGS findings with clinical parameters, pathological responses, and radiological imaging will facilitate a comprehensive understanding of treatment response and inform personalized therapeutic interventions.

Conclusion: Ultimately, this approach aims to identify genomic biomarkers predictive of radiation sensitivity or resistance, guide the rational selection of targeted therapies or immunotherapies in combination with radiation, and monitor the long-term genomic landscape of irradiated tumours to improve patient management and outcomes.

Biography

Dr. K R Muralidhar studied Radiological Physics at the Bhabha Atomic Research Centre, India in 1996 and post graduated in MSc Tech in 1995. He then joined the Manipal Hospitals as Medical Physicist. Dr. Muralidhar 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 and also completed Master of Health Administration and Independent Director from Indian Institute of corporate affairs. Dr. Muralidhar has more than 90 publications and presentations including IAEA, AAPM etc.

Keerthi Teja*, Bimal Prasad Jit, Ashok Sharma
All India Institute of Medical Sciences New Delhi, India

Impact of diclofenac on expression of POTE family genes POTE E and POTE B in epithelial ovarian cancer cell lines

Ovarian Cancer (OC) is the 3rd most common gynecological cancer worldwide with high morbidity and mortality rate and Epithelial Ovarian Cancer (EOC) is the most common subtype (90%). Despite advances in treatment, late diagnosis and inadequate therapeutic options continue to be the major challenges. Studies have shown that Non-Steroidal Anti Inflammatory Drugs (NSAIDS) like diclofenac and sulindac induce apoptosis in OC cells, inhibit tumorigenesis and metastasis. Their potential as effective chemotherapeutic agents is being studied.

POTE (prostate, ovary, testis and placenta expressed) family is a recently discovered primate specific multigene family whose expression is restricted to certain tissues and but highly expressed in certain cancers including OC. Though the exact functions of POTE genes is unknown yet, higher expression of POTE genes in germ cells of testis is found to be associated with apoptosis during spermatogenesis. We thus hypothesized that NSAIDs induced apoptosis can be linked to varied expression of POTE genes OC.

Our study investigates variation in expression of two POTE genes POTE E and POTE B against diclofenac, a potent NSAID in EOC cell lines, A2780 for POTEE and OVCAR4 for POTEB due to their higher expression in the respective EOC cell lines. The cell lines were treated with diclofenac of different concentrations and changes in gene expression were analyzed using qRTPCR and western blot techniques. Cell cycle and apoptosis assays were performed to assess the effects. Results showed that diclofenac treatment significantly induces apoptosis ($p<0.001$). There is a marked upregulation of POTEB in OVCAR4 ($p<0.001$) but a decrease in expression of POTEE gene in A2780 ($p<0.001$) compared to respective untreated controls.

The results suggest that diclofenac induced apoptosis of EOC cells can be mediated through the modulation of POTE genes expression especially POTEB gene. This further implies underlying biochemical processes that regulate tumorigenesis and cancer progression through altered expression POTE genes. But most importantly this paves way for further research on immunotherapy of OC where the POTE proteins are targeted and also the potential of NSAIDs as not only chemotherapeutic agents for OC but also to enhance the efficacy of immunotherapy for OC.

Keywords: Ovarian Cancer, NSAIDs, Diclofenac, POTE Family Genes, Apoptosis.

Biography

Keerthi Teja is a 3rd year medical undergraduate at All India Institute of Medical Sciences, New Delhi, India. He passionate about understanding tumor biology and carrying out research for development of chemo and immunotherapeutics. His current work focuses on POTE family genes and their role in tumorigenesis. He is also interested in understanding AI, ML and its application and implications in medical field.

Kylie Dickerson MD, MS[1,2*], Kimberly Powers MSN-L, RN[2], Brittany Murphy MD, MS[1,2], Rajesh Ramanathan MD[1,2], Amanda Dean Martin DNP[2], Zeynep Aydi MD[1,2]

[1]Department of Surgery, University of Arizona College of Medicine Phoenix, Phoenix, AZ 85004, USA

[2]Banner MD Anderson Cancer Centre, Banner Health, Gilbert, AZ 85234, USA

AI guided navigation of high risk incidental findings on imaging studies leads to cancer diagnoses

Introduction: Artificial Intelligence (AI) is a burgeoning tool in the field of surgery. AI technology is vastly unexplored with few applications currently in use. High risk incidental findings in radiologic studies are recognized as a critical source of missed or delayed cancer diagnoses due to gaps in communication, manual tracking burdens, and limited resources. AI has the potential to fundamentally improve recognition of high risk incidental findings on imaging studies and aid with navigation to timely oncologic care.

Methods: We utilized a commercial end-to-end oncology focused Natural Language Processing (NLP) solution that employed machine learning to identify and track high risk incidental findings in real-time. The software reviewed imaging studies performed at emergency and outpatient imaging facilities between 6/1/2024 and 6/30/2025. The software model was provided with keywords and parameters to identify high risk lung, liver and pancreas lesions for malignancy. The software identified hits among the reports and then a High Risk Incidental Findings Team (HRIFT) manually reviewed the radiology reports for validation. Patients with new high risk findings without established oncologic care were navigated.

Results: Over 13 months, 961,298 imaging studies were reviewed by the software and 48,605 (5%) reports had high risk lesions identified. The HRIFT reviewed all 48,605 imaging reports and confirmed 43,708 clinically significant high risk findings. AI software demonstrated a rate of 4.5% high risk incidental findings among all scans reviewed, and a 0.5% false positive rate. Among the confirmed high risk incidental findings group, 4,833 patients did not have an established specialty provider. 416 of these patients were successfully navigated for multidisciplinary care. After additional work up, 59 patients were ultimately diagnosed with cancer, including 13 (22%)

liver, 39 (66%) lung and 7 (12%) pancreatic cancers. This demonstrates an incidence of 0.1% new cancer diagnoses among all validated reports and 14% incidence among navigated patients.

Conclusion: AI technology identified high risk lesions in approximately 5% of patients reviewed, which would have taken significantly more time to be worked up by traditional means or might have been missed altogether. The impact on patients may include decreased time to diagnosis and increased rates of follow up with specialty care. The machine learning algorithms and validation will enable improved model performance by modifying the variables entered. This project highlights that AI technology has vast potential for streamlined patient care and helps support multidisciplinary teams in ensuring that actionable findings are appropriately identified and managed. AI technology may fundamentally change the ability of patients to receive prompt and efficacious cancer care.

Biography

Kylie Dickerson is a general surgery resident at the University of Arizona, in Phoenix. She is interested in pursuing surgical oncology and is currently doing a year of dedicated research after completing 3 years of clinical training. Kylie Dickerson is from California originally and is passionate about international travel and global medicine.

Li-Shuang Wei[1,2], Wen-Xiang Wang[3], Hong-Xia Li[3], Qi Wang[1,2], Ming-Ming Hu[3], Yin Wang3, Kan Zhai[1,2], Li Ma[3,4*]

[1]Department of Respiratory and Critical Care Medicine, Beijing Institute of Respiratory Medicine and Beijing Chao-Yang Hospital, Capital Medical University, Beijing, 100020, China

[2]Clinical Center for Pleural Diseases, Capital Medical University, Beijing, 100020, China

[3]Department of oncology, Capital Medical University, Beijing Chest Hospital, Beijing, 101149, China

[4]Clinical Research Institute, Institute of Advanced Clinical Medicine, Peking University, Beijing, 100191, China

Pleural fluid C-C motif chemokine ligand 2: An effective biomarker for diagnosing malignant pleural effusion

Background: Accurate diagnosis of Malignant Pleural Effusion (MPE) using biomarkers remains challenging. C-C motif chemokine ligand 2 (CCL2), a chemokine markedly overexpressed under pathological conditions, has emerged as a pivotal biomarker across various diseases and plays a key role in the immune response associated with MPE. This study aimed to evaluate the diagnostic accuracy of CCL2 for MPE.

Methods: From June 2019 to April 2022, 170 patients with exudative pleural effusion admitted to our hospital were enrolled and randomly divided into a training cohort (71.8%, n=122) and an internal validation cohort (28.2%, n=48). Clinical indicators, including pleural fluid Carcinoembryonic Antigen (CEA), were collected. Pleural fluid CCL2 concentrations were determined using enzyme-linked immunosorbent assay. Analyses included Receiver Operating Characteristic (ROC) curves, univariate logistic regression, and nomogram construction. Model performance was evaluated by the Area Under the ROC Curve (AUC), concordance index (c-index), and calibration plots.

Results: Pleural fluid CCL2 levels were significantly higher in MPE than in non-MPE cases (cut-off value=234.73 pg/mL, AUC=0.754, sensitivity=95.6%), whereas Adenosine Deaminase (ADA) levels were lower. Compared with other pleural fluid biochemical indicators, pleural fluid CCL2 showed competitive diagnostic accuracy, particularly for exclusion diagnosis (negative predictive value=94.4%). In addition, CCL2 exerts a significant stratification effect

on the diagnosis of MPE subgroups, especially in the ADA ≥26.5 U/L group, chloride ≥109.25 mmol/L group, and CEA ≥2.62 ng/mL group, where its diagnostic efficacy is more prominent. A nomogram incorporating pleural fluid CCL2, age, and ADA achieved AUCs exceeding 0.9 in both the training and validation cohorts (c-index=0.974). Calibration curves demonstrated strong agreement between predicted and observed probabilities of MPE.

Conclusion: Pleural fluid CCL2 is an effective biomarker for diagnosing MPE. The CCL2-based nomogram represents a robust, minimally invasive tool for accurate identification of MPE.

Keywords: CCL2, MCP-1, Malignant Pleural Effusion.

Biography

Dr. Li Ma is an Associate Chief Physician, Associate Professor, and Master's Supervisor at the Cancer Center of Beijing Chest Hospital, Capital Medical University. Her clinical and research interests focus on the early diagnosis and treatment of lung cancer, as well as biomarkers for predicting immunotherapy efficacy and patient prognosis. She is actively engaged in translational and clinical research aimed at improving personalized treatment strategies for lung cancer patients.

Dr. Maria S. Shmelkova*, Prof. Natalia K. Serova, Prof. Natalia M. Eliseeva, Dr. Olga F. Tropinskaya, Dr. Artem A. Poddubsky, Prof. Grigory L. Kobyakov, Dr. Elena R. Vetlova, Dr. Svetlana V. Zolotova

National Medical Research Centre for Neurosurgery named after N.N. Burdenko, Moscow, Russian Federation

Intraocular lymphomas associated to primary CNS lymphomas: Great mystification. Clinical landscape and diagnostic challenges

Primary Intraocular Lymphomas (PIOL) or Vitreoretinal Lymphomas (VRL) are a type of Primary Central Nervous System Lymphoma (PCNSL), accounting for approximately 2–3% of all non-Hodgkin lymphomas and developing in about 11–25% of patients with PCNSL. Morphologically, PIOLs are diffuse large B-cell lymphomas in more than 90% of cases. PIOLs are characterized in the vast majority of cases by involvement of the vitreous, retina, and/or, less rarely, the optic nerve.

The diagnosis of PIOL is very challenging and in the vast majority of cases patients get the right diagnosis months or years after the onset of clinical manifestations as clinical picture is non-specific and PIOL can mimic other ocular diseases. Patients present to an ophthalmologist with complaints of blurred vision (40-50%), decreased visual acuity (25-30%), and floaters (20-25%). Given the low awareness of possible clinical manifestations of the disease, ophthalmologists most often mistake PIOL symptoms for uveitis or posterior vitreous detachment. Consequently, establishing the correct diagnosis takes an average of 6 to 40 months from the onset of clinical symptoms. For comparison, the average time to diagnosis for PCNSL is 35 days.

The process is often binocular and is accompanied by decreased visual acuity, corneal precipitates, vitreous lymphoid infiltration and subretinal infiltrates. In the advanced stage PIOL can lead to tractional retinal detachment.

The pathogenesis of intraocular involvement in PCNSL is not fully understood. Specific somatic mutations play an important role, determining tumor biology and serving as potential diagnostic markers. The most common mutations are found in the MYD88 and CD79B genes.

The main principles of therapy include intravitreal administration of chemotherapeutic agents (methotrexate, rituximab) and Stereotactic Radiotherapy (SRT) to the area of the affected eye.

We hereby present a clinical case of a patient with PIOL.

Clinical Case: Patient 1. Female, 55y/o. Presented to Burdenko National Medical Research Centre for Neurosurgery with walking difficulties and memory troubles in May 2022. After total examination and biopsy, she was diagnosed with PCNSL, diffuse large B-cell type. The ophthalmic examination revealed decreased vision of the left eye, accompanied with severe redness, corneal (mutton–fat) precipitates and vitreal cloud-like haze. Two years ago the patient the first time experienced these symptoms and was diagnosed with uveitis. Local steroids and immunosuppressive therapy had very slight positive effect. She was tested for HLA-B27, ACE, IGRA, Toxoplasma gondii IgM, HLA-B51, ANA with all negative results. She had radiotherapy at a dose of 20–25Gy fractionated in 15 sessions for whole brain and left eye with use of lens shielding. Since then she comes for control visits every 3 month revealing no intraocular or brain relapse.

Biography

Dr. Maria S. Shmelkova was born in 1987, Rotov, Russia. In 2004 she entered Moscow State University the faculty of Fundamental Medicine which she successfully graduated in 2009. In 2009 she entered Russian National Research Medical Institute (Moscow Russia), faculty of General Medicine and successfully graduated it in 2017. In 2017 she entered PhD program in the National Research Institute for Eye Diseases (Moscow, Russia). The theme of PhD study was Hereditary Optic Neuropathies: Clinical, biological and genetic aspects. In 2018 she was attendee in the course of Eye Genetics in University of Bologna, Italy. In 2021-2023 Dr. Maria Shmelkova had a fellowship in the Retina and Optic Nerve Department of University Clinic of Nice, France. In 2024 she successfully accomplish Harvard Medical School Course of Human Genetics. Now she is an ophthalmologist and research fellow in National Medical Research Centre for Neurosurgery named after N.N. Burdenko, Moscow, Russia. Intraocular lymphomas compound the part of her scientific interests. Dr. Maria Shmelkova fluently speaks English, French, Italian and Russian.

Megadeepan Senthil kumar*, Prakruthi S Kaushik, Arun Kumar AR, T. Avinash, Sridevi Ravichandran, Neha Da Rocha, Anusha Rachapudi

Kidwai Memorial Institute of Oncology, Bengaluru, India

Unmasking the cytokine storm: A spectrum of paediatric hemophagocytic lymphohistiocytosis across immuno-oncologic, infectious, and genetic landscapes - insights from a tertiary oncology centre in South India

Background: Hemophagocytic Lymphohistiocytosis (HLH) is a hyperinflammatory syndrome with heterogeneous triggers, ranging from genetic predispositions and immunodeficiencies to malignancies and infections. We report four paediatric cases highlighting the clinical spectrum, diagnostic dilemmas, and therapeutic challenges of HLH.

Case 1: 14-year girl presented with fever, rash, generalized lymphadenopathy, cytopenias, and hepatosplenomegaly. Investigations revealed elevated ferritin and triglycerides; lymph node biopsy confirmed ALK-negative anaplastic large cell lymphoma, and bone marrow showed hemophagocytosis, consistent with malignancy-associated HLH. An EBV viral load of 12,340 copies/mL posed a diagnostic dilemma—HLH triggered by malignancy or EBV. As HLH in ALK-negative ALCL with EBV is rare, early HLH-2004 therapy controlled cytokine storm. Subsequently, ALCL-directed therapy initiated, but patient defaulted and succumbed, likely due to recurrent cytokine storm or progressive disease.

Case 2: 12-year old boy with AML developed high-grade fever, cytopenias, and extreme hyperferritinemia during induction therapy. He progressed to sepsis, acute liver failure, coagulopathy, upper GI bleeding, DCT-positive autoimmune hemolytic anaemia, and ARDS requiring non-invasive ventilation. A risk-adapted, aggressive HLH approach—steroids despite infection, doseadjusted etoposide despite liver failure, IVIg, and diuretics despite risk of hypotension—controlled cytokine storm, stabilized organ dysfunction.

Case 3: 4-month-old female, third child of a non-consanguineous family, presented with fever, hepatosplenomegaly, pancytopenia, and hyperferritinemia. Male sibling had similar features and died undiagnosed at 11 months. Bone marrow examination showed hemophagocytosis. During hospitalization, she developed seizures, hypoglycemia, acidosis, respiratory failure, and septic shock, succumbed despite steroid therapy, genetic testing could not be performed.

Case 4: 3-month-old female, born to third-degree consanguineous marriage, presented with fever, pallor, silvery hair, and hepatosplenomegaly had pancytopenia, hyperferritinemia, hypertriglyceridemia, and bone marrow hemophagocytosis. Hair shaft examination showed irregular pigment clumps, suggestive of Griscelli syndrome. HLH-directed therapy initiated, child developed hypertensive crisis (?steroid/etoposide induced), required five antihypertensives, altered sensorium with normal brain imaging, anasarca, LFT derangement, and respiratory distress, ultimately succumbed despite intensive care.

Conclusion: These cases highlight broad spectrum of paediatric HLH and offer critical lessons for clinical practice and future research:

Case 1: Early recognition and prompt HLH-directed therapy can control cytokine storm in malignancy-associated HLH, but strict adherence and timely diseasespecific treatment are crucial to prevent recurrence and death.

Case 2: Early aggressive, risk-adapted HLH therapy should be instituted. Multidisciplinary care and timely immunosuppression can reverse cytokinemediated organ damage even in critically ill oncology patients.

Case 3: Familial clustering highlights need for high suspicion of primary HLH in infants with cytopenias and hepatosplenomegaly; rapid diagnosis and early intervention remain crucial despite limited genetic confirmation.

Case 4: Genetic predisposition can cause severe multisystem HLH, with therapy related risks; intensive monitoring, individualized treatment, and early supportive measures are essential to prevent complications.

Future Prospects: These cases highlight HLH's evolving immuno-oncology spectrum. Future strategies should focus on early molecular diagnostics, targeted &less toxic therapies, and multidisciplinary care.

Biography

Dr. Megadeepan Senthil Kumar is a Paediatric Oncology Resident at Kidwai Memorial Institute of Oncology, Bengaluru, where he is immersed in the comprehensive management of childhood cancers at one of India's leading oncology centers. His medical training spans two of India's most respected institutions: his foundational MBBS was completed at Coimbatore Medical College, followed by postgraduation in Paediatrics from Swami Dayanand Hospital, Delhi, where he developed robust clinical expertise in managing complex pediatric conditions. Driven by a passion for tackling the most challenging pediatric malignancies, Dr. Megadeepan

Senthil Kumar is carving a niche in the specialized fields of Paediatric Neuro-Oncology and Histiocytic Disorders. His academic focus lies at the crossroads of these disciplines, particularly exploring the burgeoning potential of paediatric immuno-oncology. He is actively involved in clinical research aimed at understanding the tumor microenvironment of CNS malignancies and applying novel immunotherapeutic approaches to improve survival and quality of life for his patients. His ultimate goal is to contribute to the development of targeted, less toxic therapies for children with brain tumors and refractory histiocytic diseases.

Mostafa Mesbah Mahmoud Sabbah

Gaza Oncology Center, Israel

Cancer care challenges in Gaza under crisis conditions: Field experience from an oncology nurse

Gaza faces unique challenges in providing cancer care due to ongoing conflict, resource limitations, and disruptions in healthcare infrastructure. As an oncology nurse, I have firsthand experience managing these challenges, particularly in delivering chemotherapy safely, monitoring treatment side effects, and supporting patients and their families under crisis conditions. This presentation will share practical strategies, field insights, and lessons learned in maintaining continuity of care despite shortages of medications, limited access to advanced therapies, and the emotional impact on patients. It aims to inform international colleagues about effective approaches to cancer care in conflict-affected regions and to foster discussion on innovative solutions for similar settings worldwide.

Biography

Mostafa Mesbah Mahmoud Sabbah is a registered nurse with over 20 years of professional experience in critical care, medical-surgical, and oncology nursing. He holds an M.Sc. in Public Health (Epidemiology) from Al-Quds University and has led oncology units in major hospitals across Gaza. His expertise includes chemotherapy administration, infection prevention and control, emergency response, and the development and implementation of national oncology care protocols. He is deeply committed to humanitarian health initiatives, patient education, and the advancement of cancer care standards. Through leadership, collaboration, and continuous professional development, he strives to contribute to global health equity and promote compassionate, evidence-based nursing practice.

Dr. Mudalsha Ravina[1*], Dr. Shobhana Raju[1], Dr. Amit Kumar Agrawal[2], Dr. Saroj Bala[3], Dr. Rutuja Kote[1], Dr. Sonali Thakur[1], Dr. Pratheek Prasanth[1]

[1]Department of Nuclear Medicine, All India Institute of Medical Sciences, Raipur, Chhattisgarh, India
[2]Department of Medical Oncology, All India Institute of Medical Sciences, Raipur, Chhattisgarh, India
[3]Department of Clinical Hematology, All India Institute of Medical Sciences, Raipur, Chhattisgarh, India

A case-based approach using IMPeTUs criteria in FDG PET/CT reporting of multiple myeloma

[18F]FDG PET/CT has become an integral component in the evaluation of Multiple Myeloma (MM), offering significant advantages in disease detection, staging, treatment response assessment, and prognostication. The Italian Myeloma criteria for PET Use (IMPeTUs) provide a standardized framework for interpreting PET/CT in MM, incorporating key parameters such as metabolic activity, number and location of focal lesions, bone marrow uptake, and the presence of Extramedullary Disease (EMD). This talk will present a case-based overview of the clinical relevance and practical application of IMPeTUs in three distinct patient scenarios.

The first case highlights a 62-year-old female presenting with back pain and weight loss. MRI revealed multiple lumbosacral lesions, and subsequent FDG PET/CT showed more than ten focal FDG-avid lesions involving the spine, skull, and long bones, along with extensive lytic lesions and extramedullary involvement of lymph nodes, spleen, and lungs. This case illustrates the prognostic significance of high disease burden and EMD, both of which are associated with adverse outcomes.

The second case involves a 55-year-old male with known MM who showed FDG-avid lytic lesions at baseline. Post-therapy PET/CT demonstrated a complete metabolic response, underscoring the utility of FDG PET/CT in assessing treatment efficacy and guiding further management. Achieving a complete metabolic response is associated with improved progression-free and overall survival.

The third case discusses a 48-year-old female with MM and a pathological fracture of the right humerus. Pre-therapy PET/CT revealed widespread skeletal and soft tissue involvement.

Post-treatment imaging showed resolution of disease elsewhere, with residual uptake limited to the fracture site, likely reflecting healing rather than active disease. This emphasizes PET/CT's role in distinguishing post-therapeutic changes from residual disease, particularly in the context of bone lesions and fractures.

These cases collectively demonstrate the value of FDG PET/CT and IMPeTUs criteria in the comprehensive management of MM. In alignment with findings from the National Oncologic PET Registry and NCCN guidelines, PET/CT not only influences therapeutic decisions in a significant proportion of cases but also serves as an independent prognostic tool. Imaging parameters such as the number of focal lesions, SUVmax values, and the presence of EMD correlate strongly with treatment outcomes. As we look to the future, the development and application of novel PET tracers may further refine imaging strategies, particularly in non-secretory or hypometabolic disease variants.

This talk aims to highlight the practical application of standardized PET/CT reporting through IMPeTUs, and how it supports more informed, individualized care pathways in patients with multiple myeloma.

Biography

Dr. Mudalsha Ravina is an Associate Professor and Head of the Department of Nuclear Medicine at the All India Institute of Medical Sciences (AIIMS), Raipur, a premier tertiary care government centre in India. She earned her medical degree from the prestigious Mysore Medical College, Karnataka, in 2008, and went on to complete her postgraduate training in Nuclear Medicine at the renowned Army Hospital Research and Referral, New Delhi, in 2012. She further honed her clinical and academic expertise during her senior residency at Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS), Lucknow. Dr. Ravina began her academic career as an Assistant Professor at AIIMS Raipur, where she was later promoted to Associate Professor and continues to serve. She has had the opportunity to work on an International Atomic Energy Agency (IAEA)-supported project under the mentorship of Dr. Sanjay Gambhir at SGPGIMS. In recognition of her academic contributions, she was awarded the DST travel grant to attend the prestigious European Association of Nuclear Medicine (EANM) conference in Hamburg, 2015. She again represented the country in EANM 2023 at Vienna, Austria. She has been serving as an Executive Committee Member of the Society of Nuclear Medicine India (SNMI) for the past two consecutive terms, playing a key role in advancing the field at the national level. With a strong commitment to research and education, Dr. Ravina has authored over 50 publications in PubMed-indexed journals, reflecting her significant contributions to the field of Nuclear Medicine.

N D Vaswani[1*], Seema Lekhwani[2]
[1]Department of Pediatrics, PGIMS Rohtak, India;
[2]Department of Biochemistry, PGIMS Rohtak, India

Emerging dynamics of pediatric oncology in India: A comprehensive systematic review protocol

Background: Despite the WHO Global Initiative for Childhood Cancer (GICC) target of 60% survival by 2030, India's outcomes remain suboptimal at 45% vs. the 80% global benchmark. This disparity is driven by a 50–60% malnutrition prevalence, which synergistically fuels a 22% therapeutic attrition rate.

Research Gap & Hypothesis: India lacks integrated data quantifying the multiplier effect of particulate matter (PM 2.5), pesticides, and heavy metals on children with nutritional "debt." We hypothesize that the Pooled Synergistic Risk exceeds the sum of isolated risks; specifically, "sub-toxic" pesticide runoff in the Indo-Gangetic plain becomes highly carcinogenic when DNA repair enzymes are inhibited by chronic micronutrient deficiency.

Rationale: The National Cancer Registry Programme (NCRP) forecasts a 12.8% escalation in Indianpediatriconcologydiagnosesbylate2026.Thisprotocolinvestigatesthetoxiconutritional synergy behind the "Double Hit" phenomenon: where Zinc and Folate deficiencies impair cellular DNA maintenance, lowering the oncogenic threshold for environmental xenobiotics (the "3 P's": Particulate matter, Pesticides, Pollutants).

Methodology: Adhering to PRISMA-P guidelines, a 2014–2026 multi-database search (PubMed, Cochrane) is conducted. Procedural integrity is maintained via a PICOS framework, filtering records into a cohort of all eligible high-quality clinical studies. Analytical robustness is ensured through a Random-Effects Meta-Analysis to address regional heterogeneity (τ^2) across diverse Indian cohorts. Quality is assessed using the Newcastle-Ottawa Scale (NOS). This protocol is registered with Open Science Framework (https://doi.org/10.17605/OSF.IO/NHQVX).

Conclusion: We advocate for Integrated Bio-Shielding via mandatory toxico-nutritional profiling and micronutrient fortification to bolster genomic stability against anthropogenic stressors in low-resource settings.

Keywords: Pediatric Oncology; Toxico-Nutritional Synergy; PRISMA-P; Particulate Matter; NCRP; India.

Biography

Dr. N D Vaswani has done MD in Pediatrics from King George Medical College Lucknow India in 2001. Currently He is working as Professor in the Department of Pediatrics, PGIMS, Rohtak, Haryana, India. His areas of interest are Growth and Development, Nutrition and Cancer.

Ujjawal Khurana[2], Ashwin Khandelwal[1], Swagata Brahamachari[3], Neel Kamal Kapoor[2], Deepti Joshi[2], Neetu Kalra[1*]

[1]School of Arts and Sciences, Azim Premji University, Bhopal, India

[2]Department of Pathology and Lab Medicine, All India Institute of Medical Sciences, Bhopal, India

[3]Department of General surgery, All India Institute of Medical Sciences, Bhopal, India

Lifestyle, reproductive factors, and gene expression patterns in breast cancer among rural Indian women

Breast cancer is one of the most common cancers among women, accounting for over 23% of all cancer-related fatalities in India. This study aimed to identify the risk factors associated with breast cancer among women living in rural regions of Madhya Pradesh. A hospital-based retrospective case-control survey was conducted to investigate the association between lifestyle factors including anthropometric, demographic, and reproductive characteristics—and the odds of developing breast cancer. Age-matched data from 105 healthy controls and breast cancer patients without a prior family history were analyzed using univariate and multivariate methods, with risk estimated using Odds Ratio and 95% Confidence Intervals. The molecular subtypes were distributed as 12% Luminal A, 28% Luminal B, 36% Her2neu-enriched, and 25% TNBC. Early-onset breast cancer was observed in 19% of cases, with 8% classified as TNBC. Moderate risk factors included early age at marriage, with 40% of cases married before 18 years (OR=2.1, 95% CI: 1.3–3.5), multiple pregnancies reported in 22% of cases (OR=1.7, 95% CI: 1.0–2.9), and early onset of menopause observed in 60% of cases (OR=1.8, 95% CI: 1.1–3.0). Breastfeeding was not associated with a suppressed protective effect (OR=0.7, 95% CI: 0.4–1.2). Elevated Body Mass Index (BMI) also contributed to risk, with 30.8% of cases classified as overweight (OR=1.5, 95% CI: 1.0–2.3) and 13% as obese (OR=2.4, 95% CI: 1.3–4.5). Transcriptomic analysis using data from 107 breast cancer patients and Weighted Gene Co-expression Network Analysis identified elevated expression of genes involved in cell–cell adhesion including, lymphocyte proliferation, and T cell activation pathways. This study is the first report from a rural population in Bhopal, and further research with larger cohorts is warranted.

Biography

Neetu Kalra has completed her post-doctoral training at the National Cancer Institute (NCI), National Institute of Health (NIH), Bethesda, USA. Her research primarily focused on conducting preclinical investigations of drugs aimed at understanding the mechanisms involved in improving the prognosis of rare types of cancer. During her time at the Indian Institute of Science Education and Research (IISER), Bhopal as a visiting faculty, she made significant contributions to both academic teachings and research endeavours. Notably, she actively participated in a team dedicated to developing antibody-drug conjugates targeting Breast Cancer, resulting in numerous publications in prestigious international journals. Before her current position, she worked at the Vellore Institute of Technology (VIT), Bhopal as an Assistant Professor.

Omar Alqaisi[1*], Patricia Tai[2], Suhair Al-Ghabeesh[1], Guy Storme[3]

[1]Nursing Department, Al-Zaytoonah University, Airport Street, Amman, Jordan

[2]Department of Oncology, University of Saskatchewan, 105 Administration Place, Saskatoon, SK, S7N 5A2, Canada

[3]Department Radiation Oncology, UZ Brussel, Laarbeeklaan 101,1090 Jette, Belgium

Early nursing recognition and management of oncologic emergencies: A scoping review of emergency and supportive/palliative care approaches

This scoping review systematically maps the evidence on nursing interventions for early recognition and management of oncologic emergencies in adults with cancer. Oncologic emergencies represent time-critical conditions including neutropenic sepsis, malignant spinal cord compression, tumor lysis syndrome, hypercalcemia, acute airway obstruction, superior vena cava syndrome, and catastrophic bleeding complications of cancer disease progression or antineoplastic treatment toxicity that frequently trigger unplanned emergency department visits, intensive care admissions, and rapid clinical deterioration. Early nursing recognition, prompt triage, coordinated supportive management, and palliative care integration are essential to prevent avoidable morbidity and mortality, yet robust evidence on nursing-led approaches remains fragmented and incompletely synthesized across healthcare settings, cancer populations, and care delivery models. Following PRISMA-ScR guidelines, we conducted a systematic search of PubMed, Scopus, CINAHL, and ScienceDirect databases to identify primary research describing nurse-led assessment protocols, clinical recognition tools, rapid response pathways, patient/caregiver education, telehealth triage, and collaborative care models for oncologic emergencies in emergency departments, acute care units, and inpatient oncology settings. Two independent reviewers screened records, extracted data, and synthesized findings narratively and through evidence mapping. The review identified critical gaps in standardized nursing protocols for emergency detection and acute symptom stabilization, particularly in resource-limited settings and community care environments. Integration of supportive and palliative care alongside acute management is inconsistently documented across the literature. The findings demonstrate that current nursing interventions provide insufficient emergency-specific frameworks for

time-critical oncologic complications. There exists an absence of evidence-based acute care pathways, standardized nursing recognition algorithms, and sustainable digital health tools for remote patient monitoring and early symptom escalation. This scoping review provides an evidence-based foundation to inform the development of standardized nursing protocols, educational curricula, and resource-appropriate interventions for emergency and supportive/palliative oncology nursing care, directly addressing the Special Issue's focus on emergency and supportive approaches in oncological healthcare.

Keywords: Nursing Interventions, Oncologic Emergencies, Emergency Care, Early Recognition, Triage Protocols, Supportive Care, Palliative Care, Nursing Management.

Biography

Mr. Omar Al-Qaisi from Al-Zaytoonah University is a nursing expert in oncology and emergency medicine. He holds a master's degree in emergency and disaster medicine from Al-Zaytoonah University. He currently works as a part-time clinical instructor at Al-Zaytoonah University and also at the Military Oncology Center. Mr. Omar has experience using Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) and the Mixed Methods Appraisal Tool (MMAT) for research. His recent research focuses on sexual healthcare, selenium, orthopaedics, sleep quality, pain management and patient satisfaction in oncology patients.

Patricia Tai[1*], Arbind Dubey[2], Rashmi Koul[2], Kelvin Wong[3], Avi Assouline[4], Kurian Joseph[5], Edward Yu[6], Aoife Jones Thachuthara[7], Evgeny Sadikov[8]

[1]Division of Oncology, U. Saskatchewan, 105 Administration Place, Saskatoon, Saskatchewan S7N 5A2, Canada

[2]Department of Oncology, U. Manitoba, 205 Tier Building, Fort Garry Campus, Winnipeg, Manitoba, R3T 2N2, Canada

[3]Astellas Pharma Canada. 675 Cochrane Drive, Suite 650 West Tower, Markham, Ontario L3R 0B8, Canada

[4]Oncologue Radiothérapeute, Centre de Cancérologie de la Porte de Saint-Cloud (CCPSC), Président de la CME et Coordonnateur médical du CCPSC, 30 rue de Paris - 92100 Boulogne-Billancourt, France

[5]Department of Radiation Oncology, Division of Oncology, Cross Center; U. Alberta, 11011 - 88 Avenue, Edmonton, Alberta T6G 2G5, Canada

[6]Department of Oncology, Western U., 1151 Richmond Street, London, Ontario, N6A 3K7, Canada

[7]Department of Medical Oncology, Cork University Hospital, Cork, T12 DC4A, Ireland

[8]Department of Radiation Oncology, U. British Columbia, 6200 University Boulevard, Vancouver, British Columbia, V6T 1Z4, Canada

A pioneering, first-of-its-kind Canadian off-site program designed to reduce the burden on hospital staff and optimize the use of space

Aim: Degarelix, a GnRH antagonist for ADT, showed remarkable results in clinical trials. We launched Canada's first off-site injection program to decentralize services from overloaded acute care settings. This is the first reported Canadian community experience with degarelix.

Methods: Pharmacy records identified patients on degarelix, and additional data were retrieved from paper and electronic charts. A prospective database was built to track follow-ups. Primary outcome: Absolute PSA decline.

Results: From Jan 2011 to Apr 2015, 176 men were advised to use degarelix; 169 proceeded. Breakdown according to treatment purpose, first line response rate and second line response rates are: Adjuvant (27 patients, response rates not applicable), biochemical failure (49 patients, 80%, 40%) metastatic (74 patients, 76%, 28%). When used as the sole primary treatment in 19 patients, overall response was 38%. Maximal androgen blockade decreased PSA on the addition of bicalutamide in 81.4% (22/27) men. Side effects included local pain (13), fever/chills (8), rashes (5), hot flashes (5), swelling (3), and pulmonary embolism (1). Compliance: 18% stopped due to pain/swelling, but only 4.5% quit when given supportive medications. The impacts of this off-site program were reduced clinic crowding and allowed home visits for mobility-limited patients. Nurses could reallocate time to other duties. Patients better adhered when informed about degarelix's superiority over GnRH agonists.

Conclusions: Off-site injection proved feasible and well-received, relieving overburdened cancer centers and providing better care options for patients.

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.

Pietro Salvatori
Formerly, Head of ENT Dept, Humanitas San Pio X Hospital, Milan, Italy

Reconstruction after salvage surgery for laryngo-pharyngo-esophageal tumors: The experience of the national cancer institute of milan, Italy (1970-2010)

The Head and Neck Oncology Division (HNOD) of the National Cancer Institute of Milan (NCI), once equipped with 66 beds, operating rooms, and daily outpatient clinics, has been a leading center for elective and salvage surgery for head and neck tumors since the 1960s. Approximately 100 Italian and international physicians have attended the OCC to begin or further their studies in head and neck oncology.

Efficient reconstruction of the alimentary canal (primary or secondary) has been, and is, a significant challenge, often made more difficult by the patient's general (malnutrition, reduced immunocompetence, etc.) and local (reduced post-radiation healing capacity) conditions. In chronological order, but also with overlaps and pluralities, the fundamental stages in achieving this goal were: Local grafts and flaps, the deltopectoral flap (Bakamjian, 1965), the tubulized gastric pull-up (Akiyama, 1978), the pectoralis major musculocutaneous flap (Ariyan, 1979), and free flaps (jejunal loop first described by Seidenberg in 1957).

A brief overview of the aforementioned techniques is provided, along with some technical details and precautions adopted by the HNOD.

This presentation aims to document a broad and consolidated path in pharyngolaryngoesophageal reconstruction after salvage surgery, a path that is largely historical but still relevant and evolving. Above all, the Authors propose it in memory of the Masters who traced this itinerary and of the Colleagues who shared it.

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 50 papers. During recent pandemic, he made research with international colleagues and published on ethanol inhalation to treat SARS-CoV-2 infection and Covid-19.

Prabha M*, Sangeetha S
Ramaiah Institute of Technology, India

Investigation of new synthetic compounds: 4- (methyl sulfanyl) phenyl-1, 3, 4-thiadiazol-2-amine and 1-methyl-[4-(methyl sulfanyl) phenyl]-1, 3, 4-thiadiazol-2-amine, for anti-cancerous activity and for p53 anti-cancer gene expression on Glioblastoma cell cultures (LN-229)

Glioblastoma Multiforme (GBM) is a stage IV brain tumour is complex and deadly diseases. There is no promising therapeutic applications for GBM, and so many drugs are developed, and screened in pre-clinical/clinical tests. To understand the mechanism of GBM cancer causes the cell lines models were considered, of which LN-229 is used in the present study. There exists newly synthesized thiadiazole derivatives, namely, 4-(methyl sulfanyl) phenyl-1, 3, 4-thiadiazol-2 amine and 1-methyl- [4-(methyl sulfanyl) phenyl]-1,3,4-thiadiazol-2-amine, that needs to be tested for their anti-cancer activity. The research gap of not using GBM cell lines extensively for drug screening are examined for current studies. Hence, LN-229 is used as in vitro model to assess the anti-cancer activity of the given thiadiazole derivatives. It has to be noted that this is a small-scale study involving few samples, trying to determine only the IC50 value in case of LN-229 cells. The feasibility of the study outcomes is notable enough to consider for further analysis. Hence, there is cancer gene expression carried out to determine upregulation/downregulation of genes such as TP53, and EGFR, mutated in cancer cells. So the MTT assay performed for cell ability testing and RT-qPCR for gene expression analysis were employed for the current study. Overall, the aim is to integrate drug screening with disease of interest. The present studies confirm for new synthetic thiadiazole compounds are proven to have potential anti-cancer activity against GBM cell line LN-229, with upregulation of TP53 gene and downregulation of EGFR gene which promises the experimental design for anticancer chemotherapeutics.

Keywords: Glioblastoma Multiforme, LN228 Cell Lines, P53, EGFR, Thiadiazol-Derivatives.

Biography

Dr. Prabha M is an Associate Professor at RIT, located in Bengaluru, India. She has published 20 research articles and 8 conference proceedings publications. She was Post-Doctoral Fellow for CSIR RA fellowship 2008 at IISc. She has received the Distinguished woman in health and medical Sciences-Biochemistry—From Venus International foundation, Chennai. 2019. She has been awarded as Bharat Shiksha Gaurava Puraskar–2022 and Excellence for Best Educationist Award-2022 by KTK foundation, New Delhi. She got certificate for NPTEL Domain star–Biotechnology—Biosciences 2022. She has received the certificate for winner for Women Researcher Award–In the International Scientist Awards on Engineering, Science and Medicine, held on 04-Nov-2022, Organized by VDGOOD® Professional Association. She is an invited editor for Journal of Clinical Science & Translational Medicine, Journal of Biomedicine and Biosensor and Biochemical Engineering & Bioprocess Technology. She is invited as reviewer constantly for. Clinical and Translational oncology Springer publications, Cancer Biomarker, Journal of Neurochemistry and for Indian journal of Neurosciences. She is guiding PhD and many MTech /BE project students.

Ratan Kumar Sarkar

Independent Researcher, India

The role of biophysics in cancer

The proliferative biophysical structure is associated with Earth's or Lunar time (183 or 777), Earth's gravity (734) and Lunar gravity (0.1605 or 705) in context of 900-109 (reverse) fundamental mathematical complex with a directional difference of 1 that would be responsible for cell signaling.

The Histidine (155.1552)-Tyrosine (181.1894) structural complex is associated with cancer biology where Core values (C_v) of tyrosine=181*0.0019–0.1894=0.1545 or 645=900–255 according to the formula Time = M*0.0019 derived from 14.0267, an inter-amino acid factor somewhere and M is the integer mass can be transformed into time. Towards evaluation of mutational values in cancerous point mutations core values are important.

The Osimertinib (499.619g/mol), a tyrosine kinase inhibitor can be derived from tyrosine core values where 645–255=390 (neutral point) and 390+109=499 (reverse of 994) and 390–109 (downstream) = 281= 900–619 (i.e. 705*2+109 are also chemical components) causing the drug in the structure.

Considering histidine structure, 559–155=404 or 319 where 31*13=403 with a directional difference of 1. Histidine possesses a bisectional infrastructure where 0.1552 or 652*2=1304 or 404 and 248*2= 496 (i.e. 289) linked to tyrosine. It is seen 559+496=1055 or 155 and 403–319=84=652–568 (i.e 734*2) and conversely 403+319=722=900–178 where 568+510 (i.e. 705*2) =1078 or 178 under suppression.

A blocking of oxy-time (329 or 239) has been found in the system towards mutation. Oxy-therapy might be a process in cancer treatment needed investigations. Structurally, 155+84=239 and correspondingly 652+84=736 (i.e. 32*23) =900–164 (i.e.955) and 248–84=164 (i.e. 32=164*2+1)

and also 107 (would be influx of electro-gravitational wave) +48(reverse of 84)=155 in the structure.

In JAK2 G1849T V617F or TP53 G469T V157F, the mutational values 151(G)–126(T)=25 or 0.0475 and 1235(phe C_v)–754(val C_v)=481=900–419 where 41*14=574 (reverse of 475) avoiding decimals and negative mark. It is seen 481+93=574 where 652–93=559 and 248–93=155. Mathematically, 107(influx of electro-gravitational wave in the system)+93=200=481–281 and 619–200=419 that related to tyrosine.

In EGFR lung cancer mutations, L858R and C797S are two anti-parallel mutations that shows 1289(R C_v)–753(L C_v)=536 and 1065(S Cv)–709(C C_v)=356 where 536+3=539=734+705=900-361 and 356+3= 359 and where 539–359=180=183–3 in the structure. Now, 53*35=1855 or 955 where 55+109= 164 (reverse of 361)=107+57(3) in the structure.

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. He also participated in the International Cancer Research Conference 2025, contributing to global discussions on cutting-edge developments in cancer research.

Dr. Sabura Banu U

Department of Electrical and Electronics Engineering, Saveetha Engineering College, Saveetha Nagar, Thandalam, Chennai–602105, Tamil Nadu, India

Bio-inspired optimization and AI interpretability for melanoma classification

The early and precise diagnosis of melanoma plays a crucial role in improving survival rates and enabling timely intervention. However, automated melanoma detection systems face challenges due to image artifacts, illumination variation, and inter-class similarities. This study introduces a generalized bio-inspired optimization and deep learning framework that integrates multiple metaheuristic algorithms to enhance model learning and interpretability for melanoma classification. The proposed approach optimizes critical parameters in deep Convolutional Neural Networks (CNNs), improving convergence speed, feature selection, and generalization. Optimization algorithms dynamically tune hyperparameters such as learning rate, dropout, and dense layer configuration to achieve an optimal exploration–exploitation balance.

For interpretability, Gradient-weighted Class Activation Mapping (Grad-CAM) is applied to visualize the discriminative regions influencing the model's decisions, enhancing clinical transparency and reliability. The experimental analysis utilizes publicly available dermoscopic datasets for melanoma detection, with comprehensive comparisons across models optimized by different heuristic algorithms. The bio-inspired optimized deep models demonstrate significant improvements in accuracy, precision, recall, and AUC–ROC, with the best-performing configuration achieving over 99% classification accuracy.

Furthermore, statistical and visual analyses confirm the robustness, stability, and interpretability of the optimized models. The proposed framework not only enhances melanoma detection performance but also contributes to explainable AI (XAI) development in dermatology, bridging the gap between computational intelligence and clinical trust. This synergy between

bio-inspired optimization and AI-driven interpretability demonstrates potential for integration into real-time diagnostic systems and teledermatology applications, advancing intelligent healthcare automation.

Biography

Dr. Sabura Banu U is a Professor in the Department of Electrical and Electronics Engineering at Saveetha Engineering College, Chennai, India. Her research focuses on artificial intelligence, bio-inspired optimization, medical image processing, and intelligent healthcare systems. She has published numerous articles in reputed SCI and Scopus-indexed journals and actively contributes to developing AI-driven solutions for medical diagnostics and energy systems. She also serves as a reviewer and technical committee member for international conferences and journals in the field of soft computing and machine learning.

Saumya Pandey (M.Sc. Biochemistry, Ph.D. Life Science)

Department of Clinical Research, Indira-IVF Hospital, Udaipur-Lucknow, India

University of Texas Medical Branch, Galveston, Texas, USA

Toll-Like receptors-autophagy-PI3Akt "immune-inflammatory metabolic signalosome" in prostate cancer in the robotic prostatectomy golden era: Damage associated molecular patterns in immunogenic cell-death?

Objective: I aimed to investigate the potential immunomodulatory role of complex Autophagy, Toll-like Receptors and PI3-Akt signaling networks/cross-talks in single cell immunobiology by targeting circulating tumor cells in benign prostate hyperplasia and prostate cancer for eventual design of promising patient-friendly cost-effective predictive and prognostic biomarkers and/or pharmacological scaffolds for future immunotherapeutically potent combinatorial drugs with minimal adverse effects in the robotic prostatectomy era.

Material and Methods: Cell-viability MTT-proliferation and cytotoxicity assays were performed under basal and Glucose-deprived metabolic/physiological conditions in TLR-4 agonist Lipopolysaccharide (LPS/endotoxin)-treated AR+/-prostate cancer cells in culture, primarily LNCaP and PC3 cells.

Autophagy-flux was monitored by assessing the 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 LPS followed by protein isolation by RIPA-method, protein estimation by Bradford's assay, Western blotting (primary antibodies: LC3, Beclin1, Bcl-2, Atg 2/5, HMGB1 from Cell Signaling Tech./Santa Cruz Biotech., CA, USA). Immunoblots (primary antibody 1:10) were subjected to densitometric scans and relative protein expressions/fold-changes determined (Kodak Imaging software); Glyceraldehyde-3-Phosphate-Dehydrogenase (GAPDH) and/or beta-actin were used as internal controls.

Tumor biopsies were collected from clinically diagnosed/confirmed patients of prostate cancer (early vs advanced as per Prostate Specific Antigen (PSA)-Gleason grade/TNM stage) of North American ethnicity (American Whites, African Americans, Caucasians, Hispanics) from New York State, USA undergoing radical robotic prostatectomy and Circulating Tumor Cells (CTCs) were evaluated for phenotypic differential expression of target genes involved in utophagy/apoptosis/proliferati/necrosis using high-throughput precision-based Single Cell Analysis (SCA). Further, clinical follow-ups of robotic prostatectomy patients (tobacco users/non-users) post-surgery was planned for future bio-bank development in prostate cancer.

Results: LNCaP and PC3 prostate cancer 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 (14 kDa) vs LC3-I (16 kDa) in 0-12-24-48 hours' time-course. TLR4 agonist LPS-modulated autophagic flux was significant in LNCaP cells in 48 hours with differential LC3-II vs LC3-I expression levels; protein expression patterns of LC3-II isoform were significantly higher than LC3-1 over 0-12-24-48 hours in AR+/-prostate cancer cells ($p \leq 0.05$).

Hypoxic, vascular insufficient, necrotic tumor cores were assessed for CTCs and frozen for prostate cancer/oncofertility biobank/biorepository. Robotic prostatectomy CTCs-SCA clinical research data-sets in North American patients of New York State, USA yielded promising outcomes for predictive and prognostic biomarkers-development (Autophagy-TLR-PI3Akt) for subgroup-stratification of susceptible early vs advanced prostate cancer patients (tobacco users vs non-users) with differential survival trends (Risk Ratios/Hazard Ratios/Odds Ratios) post-surgery. Moreover, necrotopic marker High-Mobility-Group-Box-1 protein, apoptotic marker Bcl-2, and autophagy-signature: Atg2-Atg5, Atg7, Atg-10 and Beclin1 protein expression levels were relatively higher in TLR agonist/GD-triggered prostate cancer cells.

Conclusions: My promising translational research study strongly highlights the emerging immunotherapeutic potential of Autophagy/Toll-like Receptors/PI3-Akt cross-talks/signaling-networks in DAMPs-mediated AR+/-Prostate Cancer for future large sample size-based pharmacogenetics/genomics/transcriptomics/metabolomics-based CTCs-single cell biology-public health oriented meaningful multicentric large sample-size based epidemiology studies (prospective/retrospective) in ethnically disparate population-subsets (tobacco users/non-users) of States of New York/Texas, USA as well as Asia-Pacific region (North+South India). Further, bio-banking in prostate cancer and development of oncofertility-biorepositories may prove to be a boon in DAMPs-mediated "prostate cancer-infertility" men's health/urology-oncology-reproductive medicine research globally.

Acknowledgements: Dr. Pandey acknowledges NIH, Bethesda, Maryland, 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. Saumya 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–ChhatrapatiShahujiMaharaj 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.

Sergey Suchkov[1–12*], Hiroyuki Abe[6,13], David Smith[14], Shawn Murphy[15,16], William Thilly[17]

[1]N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia
[2]China Hong Kong Innovation International Business Association, Hong Kong
[3]New York Academy of Sciences, USA
[4]Russian Academy of Natural Sciences, Moscow, Russia
[5]EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU, Belgium
[6]ISPM (International Society for Personalized Medicine), Japan
[7]PMC (Personalized Medicine Coalition), Washington, DC, USA
[8]AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland
[9]ACS (American Chemical Society), Washington, DC, USA
[10]AHA (American Heart Association), Dallas, TX, USA
[11]ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA
[12]ISER (International Society for Eye Research), Anchorage, AK USA
[13]Abe Cancer Clinic, Tokyo, Japan
[14]Center for Cancer Genomics, Mayo Clinic, Rochester, MN, USA
[15]Wayne State University, School of Medicine, Detroit, MI, USA
[16]Jefferson College of Population Health, Professor Sigmund Freud University, Austria
[17]MIT, Cambridge, MA, USA

Personalized and precision oncology via integrating genomics, pathology-related modeling, and IT-assisted approaches to prevent, treat, and cure cancer and its complications

Individualizing patient treatment is a core objective of the medical field. Meanwhile, the in-herent variability of cancer illustrating the molecular differences between tumors, securing the linkages of those differences to an effective drug and resulting in immense patient bene-fits, lends itself to the growing field of PPM. Personalized cancer treatment in particular stands to highly benefit from PPM therapies, since extensive variability between tumors pre-sents a need to target each case in a personalized manner.

At this point, personalized cancer therapy is considered to be a treatment strategy cantered on the ability to predict which patients are more likely to respond to specific cancer therapies. This approach is founded upon the idea that cancer biomarkers are associated with patient prognosis and tumor response to therapy. And personalized tumor molecular profiles (tumor biomarkers can be OMICS-profiles that predict therapy response.), tumor disease site and other patient characteristics are then potentially used for determining optimum individualized therapy options.

Recent advances in systems biology and cancer pathology have tremendously affected the practice of pathology, gradually transforming it from a morphology-based into a precise mo-lecular-based cancer-related discipline. The improvement of methodology for genomic test-ing has made it one of the cornerstones of PPM-related cancer medicine (PPO). Various ge-nomic analyses of human cancers are being incorporated into diagnostic and decision-making algorithms of the precision cancer pathology

In this context, most of advances in PPM-guided cancer management are associated with patient care and treatment, including development of new or more precise individual therapies and genome-driven diagnostics, which had implicated in better outcomes and extended survivals, mostly due to personalized approaches for each tumor, cancer patient and pre-cancer person-at-risk into the PPM era. In order to be effective and successful, PPM-guided approach as applicable to clinical oncology practice assumes the integration of several areas of interdisciplinary knowledge and advanced technologies focused on patient's characteristics and specific healthy needs, including OMICS sciences, bioinformatics, biomarkers, digital health, data science & sharing, and data bioanalytics. In this context, the implementation of translational studies based on liquid biopsy and organoids or xenografts to evaluate molecular changes due to clonal pressure generated due to the use of target agents or tumor heterogeneity would help in the detection of mechanisms of resistance, suggesting the possibility for novel combinations. Precision pathology has therefore become fundamental not only to inform on tumor diagnosis and prognosis but also to drive therapeutic decisions in daily practice.

Providing functional PPM to cancer patients in real life is very challenging. Biodesign-driven translational research has revolutionized how we develop new treatments for cancer pa-tients. This shift in perspective, in which attention is focused on the specific molecular altera-tions of the tumor, has opened the door to personalized treatment. This situation is reflected in the increasing number of basket trials selecting specific molecular targets. But the com-plexity of cancer cells enriched with concomitant molecular alterations complicates identifi-cation of the driver. Moreover, tumor heterogeneity could be responsible for the lack of benefit when targeted agents are used. And thus the fusion of the above-mentioned strategies has created a new dimension for PPM-guided cancer therapy. This entails the development of next generation cancer targeted drugs (for therapeutic applications) and individualized can-cer vaccines (for preventive purposes). The latter is becoming crucial for personalized & pre-cision cancer therapy since the molecular heterogeneity of cancer, and the complex interac-tion of cancer, tumor microenvironment and immune cells, require sophisticated combinato-rial genotypic and phenotypic testing in order to answer a broad scope of important questions for new cancer-related targeted agent discovery, preclinical and clinical development. So, PPM

calls for a transdisciplinary approach, and considerations for how best to develop inno-vation frameworks to support safe and effective deployment of the new enabling diagnostic and therapeutic technologies in clinical oncology!

Biography

Sergey Suchkov was born in the City of Astrakhan, Russia, in a family of dynasty medical doctors. In 1980, gradu-ated from Astrakhan State Medical University and awarded with MD. In 1985, Suchkov maintained his PhD as a PhD student of Sechenov University and Institute of Medical En-zymology. In 2001, Suchkov maintained his Doc-tor Degree at the National Institute of Immunology, Russia. From 1989 through 1995, a Head of the Lab of Clinical Immunology, Helmholtz Eye Re-Search Institute in Moscow. From 1995 through 2004-a Chair of the Dept for Clini-cal Immunology, Moscow Clinical Research Institute (MONIKI). In 1993-1996. At present, Dr Sergey Suchkov, MD, PhD, is: Professor in Medicine & Immunology, Director for Center for Biodesign of N.D. Zelinskii Institute for Organ-ic Chemistry of the Russian Academy of Sciences, Moscow, Russia. R&D Director, InMedStar, Russia-UAE. Sen-ior Scientific Advisor of China Hong Kong Innovation International Business Association, Hong Kong. Member, New York Academy of Sciences, USA. Member: EPMA (European Association for Predictive, Preventive and Per-sonalized Medicine), Brussels, EU. Member, ISPM (International Society for Personalized Medicine), Japan. Member, PMC (Personalized Medicine Coalition), Washington, USA. Member, AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland. Member, ACS (American Chemical Socie-ty), Washington, DC, USA. Member, AHA (American Heart Association), Dallas, TX, USA. Member, ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA. ISER (International Society for Eye Re-search), Anchorage, AK USA. Secretary General, United Cultural Convention (UCC), Cambridge, UK.

Zou Gengyi[1], Fang CaoCao[1], Calena-Abel Brown[1], Yibo Fan[1], Aryana Bhati[1], Bansi Vanpria[1], Melissa P Pizzi[1], Sivareddy Kotla[2], Arati Sharma[3], Feng Yin[1], Yuan-Hung Lo[4], Jaffer A Ajani[1], Shilpa S Dhar[1*]

[1]GI Medical Oncology department, UT MD Anderson Cancer Center, Houston TX, 77030, USA
[2]Cardiology department, UT MD Anderson Cancer Center, Houston TX, 77030, USA
[3]Department of Molecular and Precision Medicine, Pennstate Cancer Institute, Hershey, PA 17033, USA
[4]Molecular and Cellular Oncology department, UT MD Anderson Cancer Center, Houston TX, 77030, USA

Uncovering tumor-suppressive roles of histone modifiers in gastric cancer through spatial transcriptomics and epigenomics

Gastric Adenocarcinoma (GAC) remains one of the leading causes of cancer-related mortality worldwide, largely due to its late-stage diagnosis, limited treatment options, and poorly understood molecular drivers. Recent studies have implicated epigenetic dysregulation in GAC pathogenesis, yet the functional role of specific modifiers such as the histone H3K4 methyltransferase KMT2D (also known as MLL4/MLL2) remains controversial. Here, we integrate functional genomics, spatial transcriptomics, and epigenomics to define the tumor-suppressive role of KMT2D in GAC.

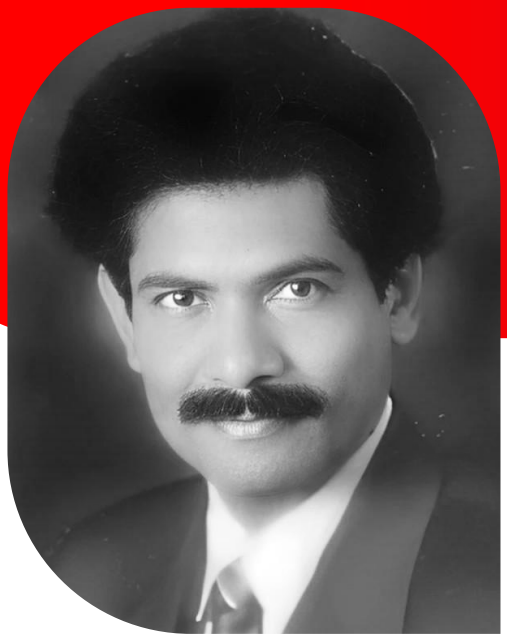
Our analyses of The Cancer Genome Atlas (TCGA) revealed that KMT2D is one of the most frequently mutated epigenetic regulators in GAC (14%), with low expression or mutation correlating with poor overall survival. Immunohistochemical (IHC) analysis further showed significantly lower KMT2D protein expression in GAC tissues compared to normal gastric mucosa. Consistent with these findings, KMT2D knockdown in AGS and GA0518 GAC cell lines promoted proliferation and invasion, while in vivo deletion accelerated tumor growth in xenograft and gastric organoid models derived from Kmt2dfl/fl mice. RNA-sequencing revealed that KMT2D loss upregulated oncogenic pathways, including those related to cytokine signaling, GTPase activation, and immune response—particularly regulatory T cell (Treg) signatures. Conversely, KMT2D positively regulated tumor-suppressive genes such as NCOR2, RCOR3, TOB2, ZNF136, SIRT1, and PER2. To better characterize the tumor microenvironment (TME), spatial transcriptomic profiling of human GAC tissues revealed regional enrichment of Tregs and immunosuppressive cytokines (e.g., IL10, TGFB1) in KMT2D-

low regions. Furthermore, spatial epigenomic profiling (spatial CUT&Tag for H3K4me1/2/3 marks) indicated widespread loss of active enhancer marks in immune-regulatory and tumor suppressor genes upon KMT2D depletion. Our single- cell RNA-seq data from GAC primaries, Peritoneal Carcinomatosis (PC), and matched normals showed that KMT2D-low tumor cells were associated with increased immune infiltration, suggesting a context- specific immunoregulatory role. Interestingly, a subset of KMT2D-high tumors exhibited higher PD-L1 expression, pointing to potential therapeutic stratification.

Together, these findings identify KMT2D as a key tumor suppressor in GAC that shapes both tumor- intrinsic transcriptional programs and the immune TME. Our data provide a rationale for further investigation of KMT2D as a prognostic biomarker and therapeutic target, particularly in the context of immunotherapy responsiveness.

Biography

Dr. Shilpa S. Dhar is an Assistant Professor in GI Medical Oncology at MD Anderson Cancer Center and a faculty member at UTHealth Graduate School of Biomedical Sciences. Her research focuses on epigenetic regulation in gastrointestinal cancers, particularly gastric adenocarcinoma (GAC), with key discoveries on histone modifiers, such as KMT2D. Driven personally by her family's experience with GAC, she integrates molecular biology and translational research, utilizing mouse models, spatial transcriptomics, and epigenomics. Dr. Dhar has authored several high-impact publications and is a dedicated mentor to trainees at all levels.



Dr. Surya Prakash Tadepalli[1,2,3]

[1]Jai Surya Global Healthcare Center–International Lobby for Critical Health Solutions, India

[2]Jai Surya Multiple Knowledge Development Organization, India

[3]SPT Oncocure Research Division–Vedichoice, Visakhapatnam, Andhra Pradesh, India

Unveiling etiological mechanisms and a targeted, non-toxic integrative approach for cancer management

Cancer has emerged as a major global public health challenge, affecting populations across all age groups. Once considered a rare and inexplicable disease, advances in biomedical sciences have established that cancer arises from identifiable biological, environmental, nutritional, lifestyle, and psychosocial factors. Understanding cancer etiology is fundamental for effective prevention, early diagnosis, and development of targeted therapeutic strategies. Traditional Indian medical systems emphasize causation-based diagnosis and root-level correction, offering valuable complementary perspectives to modern oncology.

The objectives of this study are to analyze the multifactorial origins of cancer, explore the therapeutic and preventive potential of medicinal plant–derived natural alkaloids, and propose a targeted remedial approach that selectively acts on cancerous tissues while preserving healthy organs. Emphasis is placed on non-toxic, patient-centric strategies that prioritize prevention, early intervention, and quality of life.

This conceptual and narrative review integrates evidence from classical Indian medical literature, contemporary oncology research, phytochemical studies, and relevant clinical observations. Particular attention is given to naturally occurring alkaloids, their biological mechanisms, safety profiles, and selective anticancer properties.

The analysis indicates that most cancers have identifiable etiological triggers, enabling rational prevention and targeted intervention. Numerous medicinal plants contain bioactive alkaloids exhibiting selective anticancer activity with minimal systemic toxicity. Therapeutic strategies that preserve healthy tissues demonstrate clear scientific and ethical advantages over non-selective destructive treatments. Integrative medical models combining traditional knowledge with modern science show promise in improving cancer management outcomes.

Cancer should no longer be regarded as an inevitable or universally incurable disease. Identification of root causes enables effective prevention and targeted therapeutic intervention. Nature provides biologically active compounds capable of selectively modulating cancer pathology without harming healthy tissues. A non-toxic, etiology-based, and integrative treatment paradigm represents a sustainable and humane direction for future cancer care.

Biography

Dr. Surya Prakash Tadepalli is a multidisciplinary healthcare professional and integrative medicine researcher with extensive academic training, including BAMS, MS in Clinical Microbiology, MS in Organic Chemistry, MS in Dietetics, MS in Psychology, MBA, and LLB, along with multiple postgraduate diplomas in healthcare sciences, bioinformatics, psychotherapy, anatomy and physiology, nutrition, and sports injury management. He is the Founder President of the Jai Surya Multiple Knowledge Development Organization and heads the SPT Oncocure Research Division under Vedichoice. His professional expertise spans preventive medicine, integrative oncology, chronic and incurable disease management, epidemiology, health education, and non-toxic therapeutic strategies. His research focuses on etiology-based Indian medical interventions for cancer, cardiac disorders, viral infections, metabolic diseases, and self-diagnosis–oriented, cost-effective healthcare models aimed at preserving health and quality of life.

Svetlana Baziliansky*, Tal Jean Ben-Artzi, Miri Cohen
School of Social Work, University of Haifa, Mount Carmel, Haifa, Israel

The role of emotion regulation, self-compassion, and perceived lifestyle discrepancy in maintaining a healthy lifestyle among breast cancer survivors

Aims: An unhealthy lifestyle elevates the risk of comorbidities, cancer recurrence, and diminished quality of life among breast cancer survivors. Yet, the influence of psychosocial factors on sustaining healthy lifestyle behaviors (physical activity and healthy diet) in this population remains underexplored. The present study investigated healthy lifestyle practices and the associations of perceived lifestyle discrepancy, self-compassion, and emotional distress with lifestyle maintenance among breast cancer survivors, as well as the mediating role of emotion regulation strategies (cognitive reappraisal and expressive suppression) in these associations.

Method: A total of 145 female breast cancer survivors, aged 31 to 77, completed self-report questionnaires assessing healthy lifestyle maintenance, perceived lifestyle discrepancies, self- compassion, and emotion regulation patterns. Data were analyzed using structural equation modeling.

Results: Mean scores for physical activity and healthy diet maintenance were at a moderate level. Only 30% of the survivors met the recommended guidelines for physical activity, and nearly 41% reported primarily sedentary work. Dietary patterns for most nutritional components were below the recommendations of the World Cancer Research Fund/American Institute for Cancer Research. For instance, just over half of the participants adhered to the daily recommended servings of fruits, vegetables, or whole grains. The average Body Mass Index (BMI) was 25.92(SD=4.74), slightly exceeding the recommended range.

Structural equation modeling indicated good model fit. Lower perceived lifestyle discrepancy was directly associated with greater engagement in physical activity and healthier dietary patterns. Self-compassion was positively associated with physical activity but not with diet. Cognitive reappraisal was linked to higher expressive suppression and lower physical activity, and both constructs mediated the association between self-compassion and physical activity.

Discussion: The findings underscore the significance of psychosocial factors—specifically self-compassion, perceived lifestyle discrepancy, and emotion regulation patterns—in supporting the maintenance of a healthy lifestyle among breast cancer survivors. Providing lifestyle recommendations alone may not be sufficient to ensure adherence. We suggest that health professionals assess and address survivors' perceived discrepancies and their emotional and behavioral implications. Moreover, fostering adaptive emotion regulation patterns and cultivating self-compassion may promote both well-being and long-term healthy lifestyle maintenance.

Biography

Dr. Svetlana Baziliansky is a researcher and lecturer specializing in emotion regulation, self-compassion, personal resilience, psychological distress, quality of life, fatigue, and end- of-life care. She previously worked for over 16 years as a senior oncology social worker, an experience that deeply shaped her research interests and clinical perspective. Currently, she is based at the School of Social Work, University of Haifa, where her work focuses on the psychosocial aspects of cancer survivorship and palliative care, aiming to enhance patients' well-being and adaptive coping. Dr. Baziliansky has presented her research at national and international conferences and continues to integrate research with clinical insights.

Venketesh K. Panda[1*], Kavita Kumari[1], Gopal C. Kundu[1,2]

[1]School of Biotechnology, KIIT Deemed to be University, Bhubaneswar–751024, India
[2]Kalinga Institute of Medical Sciences (KIMS), KIIT Deemed to be University, Bhubaneswar–751024, India

Osteopontin-induced M2 macrophage polarization via Pi3K/AKT/mTOR signaling in melanoma and its inhibition by andrographolide

Melanoma progression is critically shaped by the tumor microenvironment, where tumorassociated macrophages predominantly acquire an immunosuppressive M2-like phenotype. Osteopontin (OPN), a secreted glycoprotein overexpressed in melanoma, has been implicated in immune modulation; however, its role in macrophage polarization and therapeutic targeting remains inadequately defined. In this study, we investigated OPNdriven macrophage polarization and evaluated the effect of andrographolide as a potential inhibitor of this process.

THP-1–derived macrophages were treated with recombinant OPN (rOPN, 15 nM), A375 melanoma-Conditioned Medium (CM), and OPN-neutralized A375 CM, with or without andrographolide (20 μM). Macrophage polarization was assessed by flow cytometry using single and dual staining of M2 markers CD163 and CD206. rOPN and A375 CM significantly increased CD163 and CD206 expression, including a distinct $CD163^+$ $CD206^+$ doublepositive macrophage population. Neutralization of OPN in A375 CM markedly reduced M2 marker expression, confirming OPN dependency. Notably, andrographolide treatment significantly suppressed CD163 and CD206 expression both individually and in the dualpositive population across all treatment conditions.

Mechanistic investigations revealed that OPN-induced macrophage polarization was mediated through activation of the PI3K/AKT/mTOR signaling pathway, as evidenced by increased expression of PI3K p85, phosphorylated AKT, and phosphorylated mTOR. Andrographolide treatment effectively attenuated activation of this pathway, correlating with reduced M2 marker expression.

Collectively, these findings identify OPN as a key mediator of melanoma-induced macrophage polarization via the PI3K/AKT/mTOR axis and demonstrate that andrographolide effectively inhibits this process. Targeting OPN-driven macrophage reprogramming using andrographolide or related compounds may represent a promising therapeutic strategy to remodel the melanoma tumor microenvironment and limit disease progression.

Biography

Venketesh K. Panda is a graduate student and a DST-INSPIRE Senior Research Fellow working in the field of cancer immunology and tumor microenvironment. He is a University Gold Medalist with an h-index of 6 and an i10-index of 6, with more than 350 citations. He has published research articles in reputed international journals including Molecular Cancer (Impact Factor 37), Biomedicines, Cancers, and other peer-reviewed journals. His research is carried out under the supervision of Prof. Gopal C. Kundu, a renowned cancer biologist and Shanti Swarup Bhatnagar Awardee.

Vladan Radosavljevic[1*], Vladimir Bancevic[2], Branko Kosevic[2]

[1]Institute of epidemiology, Military Medical Academy, Belgrade, Serbia,

[2]Urology Clinic, Military Medical Academy, Belgrade, Serbia

Radosavljevic's urinary screening biomarkers of the key chemical carcinogens for bladder cancer

The aim of the study was to examine the possibility of using the urinary concentration of 1-hydroxypyrene for screening high-risk male individuals for bladder cancer. Radosavljević's urinary screening protocol for the key chemical carcinogens used. Numerical data were arithmetic mean with standard deviation or median with interquartile range, while categorical data shown as percentages. Two independent samples compared by Student t-test or Mann-Whitney U test, depending on the data distribution. Normality evaluated by Shapiro-Wilk test and box plots. The analysis performed in statistical software IBMSPSS ver. 29. This case-control study comprised 117 urinary bladder male cancer patients and 110 healthy male controls. The average age of patients was 67.86±9.94 years, while for controls was an average of 82.95±7.62 years. The mean urinary values of 1-hydroxypyrene at 1st measurement–in patients 205.0pg/ml (IQR=116.5-456.5) and in controls 136.5pg/ml (IQR=76-374.5) with statistically higher values in urinary bladder male cancer patients than in controls (p=0.020). At 2nd measurement–in patients 183.0pg/ml (IQR=141.8-407.3) and 137.0pg/ml (IQR=73-353.0) with statistically higher values in urinary bladder male cancer patients than in controls (p=0.013). 1-Hydroxypyrene results were higher in bladder cancer patients group, but determination of 1-hydroxypyrene alone in urine is not sufficient for urinary screening of bladder cancer in men. We suggest that HPLC and ICP analysis urinary biomarkers from other chemical carcinogens incriminated as causes of bladder cancer, from Group 1 carcinogens according to IARC.

Keywords: Human Exposure, Chemical Carcinogens, Screening, Bladder Cancer.

Biography

Vladan Radosavljevi graduated from the Medical Faculty of the University of Belgrade, Serbia, in 1991. He specialized (May 1995) and received his doctorate (November 1999) in epidemiology at the Medical Faculty of the University of Belgrade. Dr. Radosavljevi was the head of the Department of Epidemiology and deputy director of the Military Institute for Preventive Medicine in Belgrade from 2003 to 2010. He was the head of military preventive medicine from 2010 to 2020 in the Ministry of Defence of Serbia, and in 2020 he moved to the Institute of Epidemiology of the Military Medical Academy, Belgrade, where he works as an expert epidemiologist. He was a professor at the Biological Weapons course at the Military Academy of the University of Defence in Belgrade and a research associate at the Epidemiology course. Since 2015, Dr. Radosavljevi is a United Nations expert on biological weapons within the mechanism of the United Nations Secretary General.

Yanni Zhang[1*], Han Sun[1], Ye Yuan[2], Hujian Hong[1], Yanli Qu[1]

[1]Department of Radiotherapy, Cancer Hospital of China Medical University, Liaoning Cancer Hospital & Institute, Cancer Hospital of Dalian University of Technology, Shenyang, China

[2]Department of Pharmaceutical Machinery, General Hospital of Shenyang Coal Industry Group of Liaoning Hal Industry Group, Shenyang, China

Evaluation of survival prognostic factors in different treatment modalities for locally advanced unresectable esophageal squamous cell carcinoma

Background: Esophageal cancer, particularly Esophageal Squamous Cell Carcinoma (ESCC), remains a leading cause of cancer-related mortality worldwide. This study investigates the impact of different treatment modalities, namely Concurrent Chemoradiotherapy (CCRT), Chemoimmunotherapy (CIT), and induction Chemoimmunotherapy followed by chemoradiotherapy or radiotherapy (CIT+CRT/RT), on survival outcomes in patients with locally advanced unresectable ESCC.

Methods: We conducted a retrospective cohort analysis of 213 ESCC patients treated at the Fourth Affiliated Hospital of China Medical University and Liaoning Cancer Hospital from April 2015 to September 2023. Patients were categorized into three groups: CCRT, CIT, and CIT+CRT/RT. The study aimed to compare Overall Survival (OS), Progression-Free Survival (PFS), and other treatment efficacy indicators. Data were analyzed using Kaplan-Meier and Cox regression models.

Results: The median OS for the CIT+CRT/RT group (28 months) was significantly higher than that of the CCRT group (20 months) and the CIT group (24 months), with the difference between CIT+CRT/RT and CCRT groups reaching statistical significance (P=0.032). However, the 1-year OS rates did not show significant differences between the groups (P=0.082). The 2-year OS rate was significantly better in the CIT+CRT/RT group (33.3%) compared to the CIT group (9.0%) (P< 0.001). PFS at 2 years was also significantly better for the CIT+CRT/RT group compared to CIT (P= 0.001). Factors such as age and performance status were found to be independent prognostic factors.

Conclusion: Our study demonstrates that the CIT+CRT/RT regimen provides a significant survival advantage in patients with locally advanced unresectable ESCC compared to CCRT and CIT. These findings highlight the importance of combined treatment modalities in improving patient outcomes, although the influence of radiotherapy dose remains minimal. Further research is needed to refine treatment strategies and assess long-term survival benefits.

Biography

Dr. Yanni Zhang is an attending radiation oncologist at Liaoning Cancer Hospital, specializing in the clinical diagnosis and treatment of esophageal and abdominal malignancies. Her research focuses on optimizing radiotherapy strategies and improving treatment outcomes for gastrointestinal cancers. Dr. Zhang has been actively involved in multidisciplinary cancer management and clinical research, contributing to advancements in precision radiotherapy and patient care.

Yanyu Qi
Chengdu Third People's Hospital, China

Bevacizumab versus anlotinib respectively combined with chemotherapy drug in the treatment of EGFR-TKI acquired resistant advanced lung adenocarcinoma

Objective: To compare the short-term therapeutic efficacy and safety of bevacizumab versus anlotinib, each in combination with chemotherapy, for the treatment of advanced lung adenocarcinoma with acquired resistance to Epidermal Growth Factor Receptor Tyrosine Kinase Inhibitors (EGFR-TKIs).

Methods: A retrospective analysis was conducted on 84 patients with EGFR-TKI-resistant advanced lung adenocarcinoma treated at the Third People's Hospital of Chengdu between June 2019 and October 2021. Patients were divided into three groups: chemotherapy alone (32 cases), anlotinib plus chemotherapy (24 cases), and bevacizumab plus chemotherapy (28 cases). The chemotherapy group received pemetrexed disodium and carboplatin, with symptomatic management for adverse events. The anlotinib combination group received oral anlotinib hydrochloride 10 mg once daily (14 days on, 7 days off) starting on the first day of chemotherapy, in addition to the chemotherapy regimen. The bevacizumab combination group received intravenous bevacizumab 15 mg/kg one day before chemotherapy, in addition to the chemotherapy regimen. All groups completed four 3-week cycles. Outcomes included Overall Response Rate (ORR), Disease Control Rate (DCR), median Progression-Free Survival (mPFS), changes in serum tumor markers, incidence of adverse events, and 1-year survival rate.

Results: After four cycles, the ORR and DCR were significantly higher in both combination therapy groups than in the chemotherapy alone group ($P<0.05$). The mPFS was also significantly longer in both combination groups than in the chemotherapy alone group, and the DCR was

significantly higher in the anlotinib combination group than in the bevacizumab combination group ($P<0.05$). Serum tumor marker levels decreased significantly in all three groups after treatment, with greater reductions observed in the combination therapy groups ($P<0.05$). There were no significant differences in the incidence of adverse events (including nausea, vomiting, and bone marrow suppression) or 1-year survival rate among the three groups ($P>0.05$).

Conclusions: Both bevacizumab and anlotinib, when combined with chemotherapy, are effective and well-tolerated treatments for advanced lung adenocarcinoma with acquired EGFR-TKI resistance.

Biography

Yanyu Qi is an Associate Chief Physician in the Department of Oncology at the Affiliated Hospital of Southwest Jiaotong University. His primary research focus is on mechanisms of resistance to chemotherapy, targeted therapy, and immunotherapy in malignancies. Clinically, he specializes in optimizing radiotherapy and interventional techniques, as well as developing multidisciplinary diagnosis and treatment protocols, with the goal of improving therapeutic efficacy and patient outcomes. He has published multiple peer-reviewed papers in high-impact journals, including Phytomedicine, Frontiers in Cell and Developmental Biology and China Pharmacy.

Yongke Wu[1*], Yuanhao Su[1], Nassuf Mourdi[1], Yiyuan Zhao[1], Mingwang Shen[2], Yuanyuan Ji[3], Zhidong Wang[1]

[1]Department of Geriatric General Surgery, The Second Affiliated Hospital, Xi'an Jiaotong University, Xi'an 710004, Shaanxi, China

[2]School of Public Health, Xi'an Jiaotong University Health Science Center, Xi'an, Shaanxi, 710061, China

[3]Scientific Research Center and Precision Medical Institute, The Second Affiliated Hospital, Xi'an Jiaotong University, Xi'an 710004, Shaanxi, China

Development and validation of a clinical decision-support model for preoperative individualized assessment of high-volume central lymph node metastasis in older adults (over 55 years) with papillary thyroid carcinoma

Background: Elderly patients with Papillary Thyroid Carcinoma (PTC) constitute a unique subgroup in clinical practice, often exhibiting more aggressive tumor characteristics. Although current guidelines offer few specific recommendations for patients aged ≥55 years, it is vital to identify High-Volume Central Lymph Node Metastasis (HVCLNM) before surgery to guide personalized treatment. This study focused on creating and validating a Machine Learning (ML) model based on real-world clinical data to predict HVCLNM preoperatively in this understudied group, thereby aiding clinical decision-making.

Methods: We retrospectively examined electronic medical records from a real-world cohort of 644 elderly PTC patients. To address class imbalance, we applied the Synthetic Minority Oversampling Technique for Nominal and Continuous features (SMOTE-NC) during data preprocessing. Eleven critical risk factors were identified by combining univariate logistic regression, LASSO, Boruta, mRMR, and Random Forest selection methods. We then developed and systematically compared ten machine learning algorithms. Model performance was assessed using the Area Under the Curve (AUC), and SHAP analysis was used to interpret and visualize feature importance. The final model was integrated into an interactive web-based application.

Results: Among the ten ML models evaluated, the CatBoost classifier achieved the highest discriminative performance, with AUCs of 0.864 (95% CI: 0.834–0.894) in the training set, 0.721 (95% CI: 0.627–0.815) in the internal test set, and 0.737 (95% CI: 0.646– 0.828) in the external validation set. SHAP analysis identified the most influential preoperative predictors and their interactions, improving model interpretability. A clinical heatmap further demonstrated the model's ability to stratify patients effectively. The web tool allows clinicians to input patient variables for personalized risk assessment.

Conclusions: Using real-world data, this study developed and validated an interpretable machine learning framework to predict preoperative HVCLNM in elderly PTC patients. The model showed stable performance across internal and external cohorts and was converted into an easy-to-use web application for clinical use. This tool provides a valuable resource for preoperative assessment, supporting personalized clinical decisions in a population with limited evidence-based guidance.

Biography

Yongke Wu is pursuing a master's degree in oncology at the Second Affiliated Hospital of Xi'an Jiaotong University. He has contributed to multiple SCI-indexed publications. His research focuses on real-world studies of thyroid tumors and lymph node metastasis, including the development of clinical prediction models. Additionally, Yongke Wu integrates ultrasound, imaging, and histopathology data with multi-omics techniques using artificial intelligence.

Yuanhao Su[1*], Yongke Wu[1], Cheng Li[1], Nassuf Mourdi[1], Yiyuan Zhao[1], Yuanyuan Ji[2], Zhidong Wang[1]

[1]Department of Geriatric General Surgery, The Second Affiliated Hospital, Xi'an Jiaotong University, Xi'an 710004, Shaanxi, China

[2]Scientific Research Center and Precision Medical Institute, The Second Affiliated Hospital, Xi'an Jiaotong University, Xi'an 710004, Shaanxi, China

Phosphate analog PPF inhibits papillary thyroid cancer by disrupting phosphate balance and reprogramming tumor metabolism

Background: The global incidence of Papillary Thyroid Cancer (PTC) has been increasing in recent years, placing greater demands on healthcare systems worldwide. While standard treatments such as surgery combined with radioactive iodine therapy and chemoradiotherapy are common, many patients still experience refractory or progressing disease due to the large number affected. Additionally, some patients are unable to tolerate surgery or the side effects of radiotherapy and chemotherapy, which can lead to treatment delays and poor outcomes. This highlights an urgent need for new, safe, and effective therapies for PTC. Our earlier research indicated that the inorganic phosphate (Pi) transporter XPR1 promotes PTC growth and progression, suggesting that disrupted phosphate regulation could be a key factor in the metabolic abnormalities and malignancy of thyroid cancer. Based on these findings, this study investigates an innovative strategy targeting tumor phosphate metabolism to develop metabolism-based treatments for PTC.

Methods: By examining the three-dimensional structure of the phosphate transporter XPR1, we used molecular dynamics simulations and structure-based screening to find small-molecule compounds that can specifically bind to the XPR1-mediated inorganic Pi transport channel. Importantly, this study is the first to suggest repurposing an FDA-approved phosphate analog, PPF, as a possible therapeutic for PTC. To thoroughly investigate the antitumor effects and mechanisms of the phosphate analog PPF, various experimental approaches were employed. These included CCK-8 cell viability tests, EdU proliferation assays, colony formation assays, wound healing tests, transwell invasion studies, transcriptomic sequencing, metabolomic profiling, and assessment of intracellular free Pi levels. Together, these methods aimed to uncover the molecular basis of PPF's effects and evaluate its potential for clinical use.

Results: CCK-8 assays demonstrated that treating TPC-1 cells with PPF at concentrations of 0.5mM, 1mM, 2mM, 3mM, and 4mM for 48 hours significantly reduced cell viability (all $p<0.0001$). Colony formation tests showed that 48-hour exposure to 1mM and 2mM PPF substantially decreased the clonogenic ability of TPC-1 cells (1mM: $p=0.0042$; 2mM: $p=0.0002$). Similarly, EdU incorporation assays indicated that 1mM and 2mM PPF treatments markedly inhibited DNA synthesis and cell proliferation (1mM: $p=0.0072$; 2mM: $p<0.0001$). Wound healing assays also revealed a significant reduction in cell migration after 48 hours of PPF treatment (1mM: $p=0.0006$; 2mM: $p<0.0001$). Transwell assays confirmed PPF's dose-dependent inhibition of TPC-1 cell invasion. Transcriptomic analysis showed that PPF caused significant alterations in genes involved in phosphate homeostasis, including SLC20A1, SLC25A3, and XPR1. Additionally, genes related to nucleoside, purine, and pyrimidine transport (SLC29A3 and SLC29A4) were notably downregulated, while those associated with ribosomal protein synthesis, such as RPS27L, exhibited significant dysregulation. Metabolomic profiling revealed that differentially expressed metabolites were mainly linked to nucleoside, purine, pyrimidine transport, amino acid metabolism, and aminoacyl-tRNA biosynthesis, consistent with transcriptomic findings. Furthermore, intracellular Pi measurements showed a significant decrease in phosphate levels in TPC-1 cells following PPF treatment ($p=0.0055$).

Conclusions: PPF exerts antitumor effects by inhibiting proliferation, invasion, and metastasis in TPC-1 cells. Moreover, this study presents an innovative antitumor strategy that targets tumor metabolism to treat PTC. More importantly, the findings indicate that phosphate analogs, such as PPF, can modulate intracellular Pi levels and alter Pi-dependent metabolic pathways, particularly those related to nucleotide and amino acid synthesis. These metabolic shifts interfere with the production of essential cellular proteins, resulting in antitumor effects of PPF.

Biography

Yuanhao Su is a doctoral candidate in Surgery at the Second Affiliated Hospital of Xi'an Jiaotong University, with multiple SCI-indexed publications. His research focuses on the molecular mechanisms underlying thyroid cancer, particularly tumor initiation, progression, and metastasis. Additionally, he explores precision therapy approaches, such as identifying and validating new therapeutic targets and creating innovative treatments for clinical application.

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Alec N.A. Jotte* MD, Natalie B. Otto MD

RUSH MD Anderson Cancer Center, 1653 W Congress Pkwy #12, Chicago IL 60612

PD-1 inhibitor-induced refractory Acute Inflammatory Demyelinating Polyneuropathy (AIDP): Case report

Immune checkpoint inhibitors have marked a new era in contemporary cancer therapy. However, they are associated with a spectrum of immune-related Adverse Events (irAEs), including rare but severe neurologic toxicities (nirARs). Here, we report a case of Acute Inflammatory Demyelinating Polyneuropathy (AIDP) which developed following neoadjuvant pembrolizumab for triple negative breast cancer. The patient experienced progressive ascending weakness, autonomic dysfunction, and ultimately respiratory failure requiring mechanical ventilation. Despite recognition and prompt treatment with immunosuppressive therapies, the patient's course was complicated by progression of neurologic symptoms and paralysis necessitating tracheostomy and Percutaneous Endoscopic Gastrostomy (PEG) tube placement. This case highlights a rare but life-threatening neurologic toxicity of immune checkpoint inhibitors and demonstrates the importance of multidisciplinary management, careful neurologic monitoring, and the degree of ambiguity regarding long-term neurologic recovery from nirARs. It also highlights the need for the development of models that can identify which patients may be at highest risk of developing serious (grade 3 or greater) immune-related adverse events from immune checkpoint inhibitor therapies.

Biography

Alec Jotte, MD is an internal medicine resident physician at Rush University Medical Center in Chicago, IL. He completed an undergraduate degree at Vanderbilt University in Nashville, TN in Biochemistry and Chemical Biology after which he worked in social services for two years before returning to complete his medical degree at Northwestern University in Chicago, IL. His professional interests include immunology, oncology, health equity, health systems financing, and patient-centered care with a particular focus on non-English speakers living in the United States.

Fu Dian*, Li Xinijian, Fu An, Wu Ding, Yi Xiaoming, He Haowei, Cheng Wen
Jinling Hospital, China

Temporal changes in HER2 and Nectin4 expression in urothelial carcinoma: Implications for antibody-drug conjugate therapy

Background: Urothelial carcinoma is frequently managed with chemotherapy combined with immunotherapy or Antibody-Drug Conjugates (ADCs) targeting HER2 and Nectin4, which have shown promising efficacy in clinical practice. However, treatment response often diminishes over time, potentially due to alterations in target expression. Previous studies indicate that target heterogeneity exists across tumor sites, but dynamic changes in expression at the same site over time remain underexplored.

Methods: We conducted a retrospective analysis of 129 urothelial carcinoma patients treated at our center over 5 years, all with at least two pathological evaluations. Changes in HER2 and Nectin4 expression levels were assessed between initial and follow-up biopsies.

Results: HER2 expression was downregulated in 78 cases (e.g., from 3+ to 2+ or 1+), and Nectin4 expression was downregulated in 64 cases. This suggests a high incidence of target loss during treatment.

Conclusion: The observed downregulation of HER2 and Nectin4 may contribute to the reduced efficacy of ADC therapies over time. Monitoring these expression changes could inform optimal treatment duration and sequencing strategies, potentially enhancing personalized therapy approaches.

Keywords: Urothelial Carcinoma, HER2, Nectin4, Antibody-Drug Conjugates, Expression Downregulation, Treatment Resistance.

Biography

Dr. Fu Dian is a clinical oncologist and researcher specializing in genitourinary malignancies in Jinling Hospital which is a major cancer centre. With a focus on translational research, Dr. Fu's work investigates the molecular evolution of urothelial carcinoma and the mechanisms underlying response and resistance to novel therapies, including Antibody-Drug Conjugates (ADCs) and targeted agents. This research aims to identify predictive biomarkers and optimize treatment sequencing, as explored in recent literature on the topic. Dr. Fu has contributed to multiple clinical trials and publications in the field of precision oncology for bladder cancer.

He Haowei*, Shen Tianyi, Shen Hongwei, Sun Jinlu, Li Shiyang, Chen Junjie

Department of Urology, Jinling Hospital, Affiliated Hospital of Medical School, Nanjing University, Nanjing, China

Targeting HSD11B2-PD-L1 axis: A novel strategy to reverse immune escape and spontaneous metastasis in renal cell carcinoma

Background: Metastatic Renal Cell Carcinoma (mRCC) remains a therapeutic challenge with poor prognosis, as current immunotherapy combinations often encounter resistance. Immune escape is a key driver of spontaneous metastasis; however, the underlying regulatory mechanisms in mRCC remain to be fully elucidated.

Purpose: This study aims to investigate the role of 11β-Hydroxysteroid Dehydrogenase 2 (HSD11B2) in RCC spontaneous metastasis, clarify its regulatory network regarding immune escape, and evaluate the translational potential of HSD11B2-targeted therapy.

Methods: A spontaneous lung metastasis model of RCC was established via subcapsular injection of RENCA cells. Transcriptome sequencing identified HSD11B2 as a key downregulated molecule in metastatic foci. *In vitro* (MTT, Transwell, flow cytometry) and *in vivo* (subcutaneous xenograft, tail vein metastasis) experiments verified the function of HSD11B2. Co-IP, mass spectrometry, and ChIP assays explored interactions with PD-L1 and upstream regulation by the NR3C1/SP1/p300 complex. The therapeutic efficacy of the HSD11B2 activator, Isomaltotetraose (alone or combined with the PD-L1 inhibitor Lesabelimab), was evaluated in preclinical models.

Results: HSD11B2 was significantly downregulated in RCC metastatic tissues and cell lines ($p<0.01$). Knocking down HSD11B2 enhanced RCC cell invasion (2.3-fold vs. control, $p<0.05$) and lung metastasis (4.1-fold vs. control, $p<0.01$), while overexpression reversed these effects. Mechanistically, HSD11B2 bound to PD-L1 and promoted its ubiquitination and degradation

via the ERAD pathway; consequently, downregulated HSD11B2 stabilized PD-L1, leading to reduced $CD8^+$ T cell infiltration (38% decrease, $p<0.05$). Additionally, the NR3C1/SP1/p300 complex was found to suppress HSD11B2 transcription by inhibiting H3K27ac modification. Isomaltotetraose treatment reduced lung metastases by 52% ($p<0.01$), and its combination with Lesabelimab achieved a 76% reduction ($p<0.001$).

Conclusions: HSD11B2 downregulation promotes RCC spontaneous metastasis via PD-L1-mediated immune escape. The HSD11B2 activator Isomaltotetraose, particularly in combination with PD-L1 inhibitors, offers a novel therapeutic strategy for mRCC, aligning with the conference theme of transforming cancer care through innovative integrations.

Biography

Dr. Haowei He is a researcher at the Department of Urology, Jinling Hospital, Affiliated to Nanjing University School of Medicine, and a core member of the Nanjing University Institute of Urology and Andrology. His research focuses on the mechanisms of renal cell carcinoma metastasis and the identification of novel immunotherapeutic targets. Dr. He has published over 20 peer-reviewed papers in high-impact journals, including European Urology and Cell Communication and Signaling.

Maojie Xue[1,2], Leyi Chen[1], Yuening Ding[1], Jiacheng Zhou[1], Li Su[3*], Yu Chen Zhao[3], Xuan Wang[1], Ning Xie[1], Mengsheng Zhao[1], Manel Esteller[4,5,6,7], Rayjean J. Hung[8], Christopher I. Amos[9], Yi Li[10], Lei Li[2], Feng Chen[1,11,12], David C Christiani[3,13], Ruyang Zhang[1,3,14,15,16]

[1]Department of Biostatistics, Center for Global Health, School of Public Health, Nanjing Medical University, Nanjing, Jiangsu 211166, China

[2]Department of Health Inspection and Quarantine, Center for Global Health, School of Public Health, Nanjing Medical University, Nanjing, Jiangsu, 211166, China

[3]Department of Environmental Health, Harvard T.H. Chan School of Public Health, Boston, MA 02115, USA

[4]Josep Carreras Leukaemia Research Institute, Barcelona, Catalonia 08021, Spain

[5]Centro de Investigacion Biomedica en Red Cancer, Madrid 28029, Spain

[6]Institucio Catalana de Recerca i Estudis Avanc, ats, Barcelona, Catalonia 08010, Spain

[7]Physiological Sciences Department, School of Medicine and Health Sciences, University of Barcelona, Barcelona, Catalonia 08007, Spain

[8]Lunenfeld-Tanenbaum Research Institute, Sinai Health, and University of Toronto, Toronto, ON M5G 1X5, Canada

[9]The Institute for Clinical and Translational Research, Baylor College of Medicine, Houston, TX 77030, USA

[10]Department of Biostatistics, University of Michigan, Ann Arbor, MI 48109, USA

[11]China International Cooperation Center for Environment and Human Health, Nanjing Medical University, Nanjing, Jiangsu 211166, China

[12]Jiangsu Key Lab of Cancer Biomarkers, Prevention and Treatment, Cancer Center, Collaborative Innovation Center for Cancer Personalized Medicine, Nanjing Medical University, Nanjing, Jiangsu 211166, China

[13]Pulmonary and Critical Care Division, Department of Medicine, Massachusetts General Hospital and Harvard Medical School, Boston, MA 02114, USA

[14]Changzhou Medical Center, Nanjing Medical University, Changzhou, Jiangsu 213164, China

[15]Information Center, The Second People's Hospital of Changzhou, the Third Affiliated Hospital of Nanjing Medical University, Changzhou, Jiangsu, 213164, China

[16]Laboratory for Digital Intelligence & Health Governance, Nanjing Medical University, Nanjing, Jiangsu 211166, China

Identification of prognostic biomarkers and potential therapeutic targets for lung cancer by metabolomics analysis

Rationale: Metabolic reprogramming is a hallmark of lung cancer and a source of druggable vulnerabilities. Bridging mechanistic metabolic signatures with clinical prognostication may reveal targetable pathways that inform precision therapy and biomarker guided trials. However, there is no metabolic study of lung cancer prognosis up to date.

Objectives: We aim to identify serum metabolites with prognostic value that are embedded in targetable mechanism pathways, quantify the additional prognostic value of serum metabolites, and explore potential pathways through mediation analysis and multi-omics integrative analysis.

Methods: We applied a two-phase analytic strategy to identify metabolites associated with lung cancer Overall Survival (OS) time, which having either main effects or metabolite-age interactions. In the discovery phase, we performed a Metabolome-Wide Association Study (MWAS) of lung cancer OS for 2,134 non-targeted serum metabolites on 327 patients from the Boston Lung Cancer Study (BLCS). Significant metabolites whose false discovery rate ≤5% were screened out. In the validation phase, they were again tested in 218 patients from BLCS.

Network and mediation analyses revealed cross-regulatory mechanisms between exogenous and endogenous metabolites. Using UK Biobank omics data and summary-level molecular Quantitative Trait Loci (xQTL) data, we performed multi-omics integration to identify potential metabolite-based prognostic regulatory pathways.

Measurements and Main Results: Totally, 13 metabolites with main effects and 6 ones with metabolite-age interactions were reliably validated, demonstrating strong prognostic predictive value. The anti-inflammatory lipid mediator prostaglandin J2 served as a core metabolic hub, mediating the effects of specific exogenous metabolites. Multi-omics analysis further revealed trans-omics regulatory axis, e.g., carnosine metabolite - CNDP1 protein–AGAP3 gene expression–CDK5 DNA methylation.

Conclusions: This study identified metabolic biomarkers associated with lung cancer prognosis and their underlying pathways, highlighting the anti-inflammatory PGJ2 axis and CNDP1-carnosine pathway as potential therapeutic targets. These findings provide a mechanistic foundation for precision metabolic interventions and drug development in lung cancer.

Biography

Li Su is an accomplished molecular epidemiologist and Lab Director at the Harvard T.H. Chan School of Public Health. With expertise in lung cancer susceptibility, she has authored over 160 publications. Her first-author research includes investigating matrix metalloproteinase-1 promoter polymorphisms and the association of p21 and p53 genotypes with lung cancer risk. Li received the Harvard ACE Award. She currently leads laboratory operations, training doctoral students in advanced molecular techniques for international oncology research.

Yi Xiaoming*, He Haowei, Fudian, Cui yankang, Zhou wenqun

Department of Urology, Jinling Hospital, Affiliated to Nanjing University School of Medicine, Nanjing, China

The oncogenic role of KCNN4 in renal clear cell carcinoma: A mechanism involving immune regulation via Tregs and resting mast cells

Background: Renal Cell Carcinoma (RCC) is a common malignant tumor of the urinary system, with clear cell RCC (ccRCC) representing its predominant subtype. The pathogenesis of ccRCC remains incompletely understood. KCNN4 (potassium calcium-activated channel subfamily N member 4) has recently emerged as a potential oncogene implicated in multiple malignancies. This study aimed to investigate the expression and functional role of KCNN4 in ccRCC and to elucidate its mechanism in modulating tumor immune responses through regulatory T cells (Tregs) and resting mast cells, with the goal of identifying novel therapeutic targets.

Methods: The GSE53757 dataset was obtained from the GEO database to analyze KCNN4 expression in ccRCC. Tissue samples were collected from 30 ccRCC patients (study group) and 30 patients with benign renal tumors (control group) at Jinling Hospital, Nanjing Medical University (April 2022–April 2023). KCNN4 mRNA and protein expression were validated using quantitative PCR and Immunohistochemistry (IHC) in clinical samples and ccRCC cell lines (786-O and OS-RC-2). Functional assays, including CCK-8 proliferation, scratch wound healing, and Transwell migration/invasion, were performed following KCNN4 inhibition or siRNA knockdown. A xenograft model was established in BALB/c-nu mice using 786-O cells with or without KCNN4 knockdown to monitor tumor growth. Immune cell profiling was conducted via flow cytometry to assess CD4+Foxp3+ Tregs and resting mast cells. The expressions of KCNN4, Foxp3, TLR4, and GSK3β were detected by qPCR and IHC. Cytokine levels (IL-6, TNF-α, VEGF, TGF-β) in tumor tissues were measured by Western blot, and IL-4 and TGF-β in cell culture supernatants were quantified by ELISA.

Results: KCNN4 was significantly overexpressed in ccRCC tissues compared with normal adjacent tissues and benign controls ($p<0.05$). Similarly, 786-O and OS-RC-2 cells showed higher KCNN4 levels than the normal renal cell line HK-2. KCNN4 knockdown significantly suppressed cell proliferation, migration, and invasion *in vitro* ($p<0.05$). *In vivo*, KCNN4-silenced cells exhibited delayed tumor formation and reduced tumor volume compared with controls. Flow cytometry revealed increased Treg infiltration and decreased resting mast cells in ccRCC tissues. KCNN4 expression positively correlated with Foxp3 and negatively with TLR4 and GSK3β. Moreover, elevated levels of IL-6, TNF-α, VEGF, and TGF-β were observed in KCNN4-high tumors and were positively associated with KCNN4 expression. ELISA confirmed increased IL-4 and TGF-β in supernatants from KCNN4-expressing cells.

Conclusions: KCNN4 is highly expressed in ccRCC and promotes tumor progression, potentially through impairing antitumor immunity by enhancing Treg recruitment and suppressing resting mast cells, thereby facilitating immune escape. These findings identify KCNN4 as a novel oncogenic regulator and a promising therapeutic target in ccRCC.

Keywords: Renal Cell Carcinoma, KCNN4, Tumor Microenvironment, Regulatory T Cells, Resting Mast Cells, Immune Response.

Biography

Dr. Xiaoming Yi is a researcher at Department of Urology, Jinling Hospital, Affiliated to Nanjing University School of Medicine, and a core member of the Nanjing University Institute of Urology and Andrology. His research focuses on clinical and basic research in renal cancer and andrological disorders. Dr. Xiaoming Yi has contributed to multiple clinical trials and publications in the field of precision oncology for renal cancer and andrological disorders.

Xingyue Chen[1*], Tingting Liu[2], Shah Nawaz Khan[3], Xiaoxu Li[4], Bo Kong[5], Yingjun Zhou[2], Yangyan Li[3], Yuhua Ge[1], Gang Chen[3], Xu Deng[2]

[1]Southeast University, China
[2]Central South University, China
[3]Shanghai Jiaotong University, China
[4]China Tobacco Hunan Industrial, China
[5]Beijing Life Science Academy, China

Phytohormone strigolactams overcomes oxaliplatin-resistance in colorectal cancer by blocking the late-stage autophagy to tip the metabolic balance toward ferroptosis

My presentation addresses one of the most pressing challenges in oncology: Treatment Resistance. I will focus on a novel strategy to overcome oxaliplatin resistance in colorectal cancer, titled " Phytohormone strigolactams overcomes oxaliplatin-resistance in colorectal cancer by blocking the late-stage autophagy to tip the metabolic balance toward ferroptosis".

The clinical efficacy of oxaliplatin, a first-line chemotherapy for colorectal cancer, is severely limited by the development of intrinsic and acquired resistance. A key mechanism enabling this resistance is the upregulation of protective autophagy, which allows cancer cells to survive chemotherapy-induced stress by recycling damaged components. Simply blocking autophagy with non-specific agents has proven challenging due to toxicity.

Our research introduces a new approach. We developed a novel synthetic strigolactam analog, SL 39, which directly targets this resistance mechanism. SL 39 acts not as a broad autophagy poison, but as a precision inhibitor of late-stage autophagy, specifically blocking autophagosome-lysosome fusion. This unique action traps oxaliplatin-resistant cells with their own accumulated metabolic damage.

Crucially, we demonstrate that SL 39 does more than just inhibit autophagy—it actively reprograms the cell's fate. By shutting down this critical survival pathway, SL 39 tips the metabolic balance within resistant cells, converting the protective process into a lethal vulnerability. This leads to overwhelming oxidative stress, mitochondrial dysfunction, and, most importantly, the induction of ferroptosis—an iron-dependent cell death pathway to which these resistant cells are now uniquely sensitive.

Our data show that combining SL 39 with oxaliplatin delivers a powerful synergistic effect, effectively resensitizing resistant colorectal cancer cells both *in vitro* and *in vivo*. This combination strategy exploits the weakness created by the resistant cells' own dependency on autophagy.

In conclusion, our work moves beyond merely inhibiting a resistance mechanism. We present a paradigm-shifting strategy: Hijacking the very pathway that confers resistance (autophagy) to trigger a potent alternative cell death (ferroptosis). This approach offers a promising and targeted therapeutic avenue to break the cycle of treatment failure in colorectal cancer and potentially other resistant malignancies.

Biography

Xingyue Chen is currently a Ph.D. candidate in Chemistry at Southeast University, under the joint supervision of Prof. Yuhua Ge (SEU) and Prof. Gang Chen (SJTU). She earned her M.S. from Southeast University in 2021 and her B.S. from Chongqing Normal University in 2018. Xingyue Chen research focuses on the synthesis and functional exploration of bioactive natural products, aiming to develop novel therapeutic agents against pressing global health challenges, including chemotherapy resistance and microbial drug resistance.

Yutong Zhou[1*], Yangyan Li[2,4], Dazhi Zhou[3], Yujie Jiang[1], Yingchuan Que[2], Zhiming Li[3], Gang Chen[2,4]

[1]Southeast University, China
[2]Shanghai Jiao Tong University, China
[3]Fudan University, China
[4]Qinghai Institute of Salt Lakes, China

Overcoming therapeutic resistance in oncology: A regioselective borylation platform for resurrecting and evolving nucleoside therapeutics

The emergence of treatment resistance remains a central challenge in oncology, often stemming from the limited chemical diversity and predictable mechanisms of existing drugs. We present a transformative chemical strategy to combat this by creating novel nucleoside-boron hybrids capable of bypassing common resistance pathways. Our method enables the direct, regioselective installation of a versatile boron handle specifically at the C2-position of unprotected adenosine and its therapeutic derivatives—including drugs like Tenofovir and GS-441524—via a mild Minisci-type reaction.

This C2-Boryl Group Serves A Dual Purpose: As a synthetic linchpin for diversification and as a direct modulator of bioactivity. Computational studies reveal that magnesium chloride guides this exquisite selectivity by pre-organizing the substrate. The boron handle allows for rapid conversion into diverse functionalities, creating a library of next-generation analogues. Crucially, this single modification can "resurrect" inactive nucleoside scaffolds, converting them into potent agents against resistant cancers. For instance, a boron-modified derivative (3u) of an inert precursor exhibited potent, low-micromolar IC_{50} values across several cancer cell lines.

This platform provides a powerful tool for drug sequencing and combination strategies. It allows for the systematic remodelling of approved nucleoside drugs to overcome resistance and for the creation of hybrid molecules with novel mechanisms. By integrating synthetic innovation with direct biological impact, our work offers a new paradigm for developing adaptive therapeutics designed to address the evolving challenge of treatment resistance.

Biography

Yutong Zhou is a Ph.D. student in Chemistry at Southeast University, under the joint supervision of Prof. Yuhua Ge (SEU) and Prof. Gang Chen (SJTU). She earned her M.S. from Zunyi Medical University in 2023 and her B.S. from Hainan Medical University in 2020. Yutong Zhou research focuses on nucleoside medicinal chemistry and radical chemistry, committed to developing novel nucleoside and oligonucleotide therapeutics to address global health challenges, including targeting and drug resistance.

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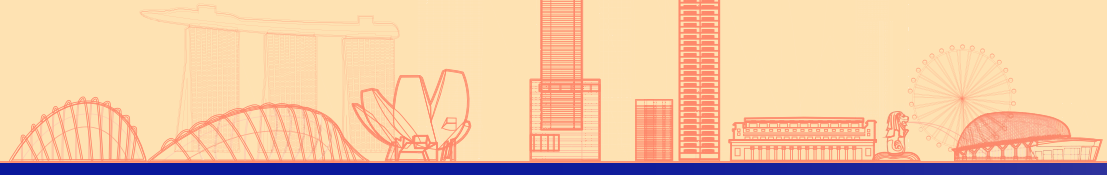

Questions? Contact

Phone: +1 (702) 988 2320 | WhatsApp: +1 (640) 666-9566

E-mail: cancer-research@magnusconference.com