The ICONIC MEETINGS
.....Meet Influential People.....

ONCOLOGY SUMMIT-2026

International Experts Summit on
Oncology & Cancer Care

April 13-15, 2026 I Tokyo, Japan

ABSTRACT BOOK

Website: https://www.cancer.theiconicmeetings.com/

Table Of Contents

PLENARY SPEAKERS

7 Title: Spatial Profiling of Protein Kinase A Subunits Identifies Aggressive Prostate Cancer in MRI-Targeted Biopsies

Ronit Ilouz, Bar-Ilan University, Israel

8 Title: HOW PALLIATIVE IS COUNSELLING TO CANCER PATIENTS

NABWIRE SUSAN, Uganda Women Cancer Support Organisation, Uganda

9 Title: Epigenetic mechanisms of hepatocellular carcinoma post HCV cure

Meital Gal-Tanamy, Bar-Ilan University, Israel

KEYNOTE SPEAKERS

10 Title: The role of curcumin in apoptosis in breast cancer cell and normal cell line

Hassan Dawud Jidda, Sharda University, India

11 Title: Evaluating the Efficacy of Peptide-Based Vaccines in Cancer Treatment: A Systematic Review

Arezoo Esmaeili, Islamic Azad University, Damghan, Iran

12 Title: New insights of YAP activity in brain metastases from colorectal cancer

Inès GARROUCHE, University of Poitiers, France

13 Title: Glutamate dehydrogenase (GDH) Promotes Survival of Triple-Negative Breast Cancer Persister Cells Through Regulation of EMT and Anaplerotic Metabolism

Biswajit Dey, National Institute of Pharmaceutical Education and Research, Telangana, India

14 Title: The glucocorticoid receptor suppresses lung inflammation induced by nicotine-derived nitrosamine ketone by suppressing cyclooxygenase-2 in A549 cells.

Wonchung Lim, Cheongju University, Cheongju, South Korea.

15 Title: Human Adipose-Derived Stem Cells Promote Migration of Papillary Thyroid Cancer Cells via the Leptin Pathway

Botao Zhang, The Affiliated Hospital of Southwest Medical University, China

16 Title: Harnessing extracellular vesicle pathways to target vascular peculiarities and immune cell exclusion in glioblastoma

Janusz Rak, McGill University, Montreal, Canada

17 Title: Identification of Histological and Molecular characteristics associated with high-risk Oral leukoplakia

Rinal Chavda, Cancer Research Institute, ACTREC, TMC, Navi Mumbai, Maharashtra, India.

18 Title: High Tumor Mutation Burden in Triple-Negative Breast Cancer among Young Ethiopian Women: Insights from Targeted Sequencing for Improved Diagnosis and Care

Esmael Besufikad, Cancer and Other non-communicable Disease Research Divisison, AHRI, Ethiopia

19 Title: Modulation of Anti-Tumor Immunity: Lactate-Driven Ferroptosis of CD8+ T Cells in the Tumor Microenvironment and Its Therapeutic Implications

Jiang Li, Sun Yat-sen University Cancer Center, China

20	Title: YAP nuclear translocation induced by HIF-1α under hypoxic conditions in 3D model
	Hsueh-Chun Wang, National Cheng Kung University Tainan 70101, Taiwan.
21	Title: Development of breast cancer survivorship program within a community-based hospital in New York.
	Julie Black, University of New York, USA
22	Title: The Role of Vimentin in the Survival of Triple Negative Breast Cancer Patients : A Retrospective Cohort Study
	Karl Heinz Leonhardt Rowika, Universitas Indonesia – Cipto Mangunkusumo Hospital, Indonesia
23	Title: Exploring the Impact of Taxane-Based Chemotherapy on the Physical Function of Breast Cancer Patients Using Markerless Motion Capture
	Mahtab Azhdar, University of Alberta, Canada
24	Title: Toward a New Era in Palliative Care in Cancer
	Kunihiko Ishitani, President, SCPSC; President, Higashi Sapporo Hospital, Japan
25	Title: Adult Lymphoma-Associated Hemophagocytic Lymphohistiocytosis: A 24-Year Single-Center Retrospective Analysis of Clinical Features, Diagnostic Hurdles, and Treatment Outcomes
	Mitchell Boshkos, Baylor College of Medicine, USA

26	Title: Clinical Relevance of BCL2A1-High $CD8^+$ T Cells in Predicting Outcomes of Immune Checkpoint Blockade in Lung Adenocarcinoma
	Hoang Minh Quan Pham, Taipei Medical University, Taipei 11031, Taiwan.
27	Title: High-Sensitivity Detection of Invasive Ductal Carcinoma via Domain-Specific SimCLR Pre-Training
	Mufakir Qamar Ansari, The University of Toledo, USA
28	Title: Cancer des voies biliaires à l'Hôpital Principal de Dakar : Aspects épidémiologiques, cliniques, morphologiques et histologiques
	Omar TOURE, Hôpital Principal de Dakar, Senegal
29	Title: Reprogramming the Tumor Microenvironment: Novel Combination Strategies to Overcome Immune Exclusion
	Patrycja Nowak-Sliwinska, University of Geneva, Switzerland
30	Title: Phytochemical-Mediated Synthesis of Silver Nanoparticles Using Tridax procumbens and Their Therapeutic Efficacy Against Lung and Skin Cancer Cell Lines
	Nilesh Makwana, Jawaharlal Nehru University, New Delhi, India
31	Title: Seroprevalence and risk factors associated with influenza D virus infection in cattle in peri-urban areas of Dakar and Thiès, Senegal
	Mireille Catherine KADJA, School of Veterinary Medicine (EISMV), Senegal

32 Title: Integrative Systems Profiling Reveals a Circulating Stemness–Exhaustion Axis Driving Metastatic Prostate Cancer

Poornima Verma, Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS), Lucknow, India

33 Title: Immune checkpoint inhibitor–induced sarcoidosis is a rare but increasingly reported adverse event, particularly with anti-PD-1 therapies. We report a case of systemic sarcoidosis in a patient treated with pembrolizumab for bilateral breast cancer.

Rihab MELLITI KHALIL, General Hospital Of Aix en Provence, France

34 Title: Machine Learning-Based Prediction of Nodal Status in Oral Squamous Cell Carcinoma and Development of a Prognostic Nomogram

Vasantha MN, Manipal Academy of Higher Education, Manipal, Udupi, Karnataka-576102, India.

35 Title: Breast cancer screening in Lebanon: Understanding knowledge, attitudes barriers. Practices during economic crisis/ Covid 19 Pandemics.

Tamina, ELIAS-RIZK, Lebanese American University, Lebanon

FEATURED SPEAKERS

36 Title: Targeted protein degradation for cancer therapy

CAPDEVILLE Manon, Institut Pasteur, Paris, France.

37 Title: Targeted inhibition of NBCn1 (SLC4A7) by antibodies prevents net acid extrusion, causing reduced proliferation and apoptosis in breast cancer in a pH- dependent manner

Ali Mohammadi, Aarhus University, Aarhus, Denmark

38 Title: Triple negative and hormone-dependent human breast cancer cells are responsive to pro-apoptotic stimule from organometallic compounds of new generation

Dafne Bahena Salmerón, Universidad Nacional Autónoma de México (UNAM), Mexico

39 Title: Organitin compounds induce regulated cell death in mouse breast cancer cells 4T1 and JC.

Del Moral-Santillan Ehecatl , Universidad Nacional Autónoma de México (UNAM), Mexico

40 Title: Breastfeeding is associated with cancer prevalence: A nationwide population study

Eyun Song, Korea University College of Medicine

41 Title: The role of curcumin in apoptosis in breast cancer cell and normal cell line

Hassan Dawud Jidda, Sharda University, India

42 Title: Assessment of changes in the expression profile of PIP-dependent genes according to the molecular subtype of breast cancer in the context of chemotherapy response

Karolina Jablonska, Wroclaw Medical University, Poland.

43 Title: Germline defects of familial haemophagocytic lymphohistiocytosis–related genes may represent a predisposing factor for mature T- and natural killer-cell lymphoma.

Chong Wei, Peking Union Medical College, Beijing, China

44 Title: Unraveling inflammation reprogramming roles of TNFα-NFκB signaling during fetal liver development and Hepatocellular Carcinoma progression.

Ita Novita Sari, Institute of Molecular and Cell Biology (IMCB), Singapore.

45 Title: The dual effect of immune checkpoint inhibitor and tyrosine kinase inhibitor on thyroid function and its impact on survival in patients with advanced renal cell carcinoma from The Cancer Clinical Library Database

Jwa Hoon Kim, Korea University College of Medicine, Korea

46 Title: Dissecting ERK5 Functional Domains in Regulating Epithelial-to-Mesenchymal Transition (EMT) and Migration in Triple-Negative Breast Cancer (TNBC)

Kan Kar Mern, University of Manchester, Manchester, United Kingdom.

47 Title: In vitro and in silico evaluation of talinum fruticosum tumor cell co-culture derived molecules as predicted precision biotherapy for inflammatory breast cancer.

NDONG MENGOME CHRISTINE, Independent Researcher, Gabon

48 Title: A hydroxylated chalcone derivative induces reactive oxygen species-mediated Bax activation and apoptosis in CD133+ lung cancer organoids

Seo Lyn Choi, Sungkyunkwan University, Korea

49 Title: The MORC2/CREB Axis Promotes Stemness and Aggressive Phenotypes in CD133+ Hepatocellular Carcinoma Cells

Seohee Park, Sungkyunkwan University, Korea

50 Title: Delayed Metabolic Toxicity in a Patient with Advanced ALK-Rearranged NSCLC Treated with Lorlatinib: Clinical Response and Safety Profile

Mònica Aguiló Domingo, Spain

51 Title: An evolutionarily conserved telomerase–cGAS–STING axis shapes inflammatory microenvironments in colitis and colitis-associated colorectal cancer.

Prativa Majee, Institute of Molecular and Cell Biology (IMCB), Agency for Science, Singapore,

52 Title: Association Between Age and Changes in Left Ventricular Ejection Fraction in Breast Cancer Patients Receiving Doxorubicin: A Cross-Sectional Study.

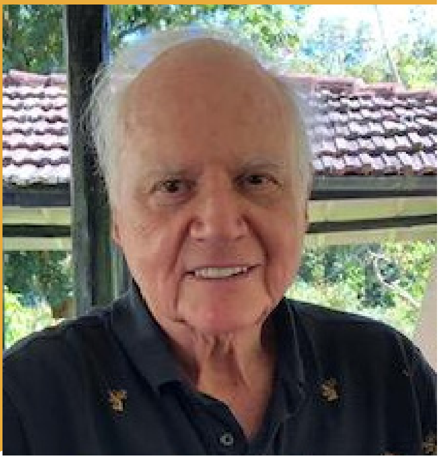
Setiani Silvi Nurhidayah, Universitas Gadjah Mada, Indonesia

53 Title: Engineering Robust SOPs for Clinical Data Management: A Strategic Framework for Quality, Compliance, and Operational Excellence

Shovan Saha, United States

54 Title: Ovary histological characterization treated with steroidal saponin (25R)-spirost-5-en-3β-yl O-α-L-rhamnopyranosyl-(1→4)-β-D-glucopyranoside (3GRD) in a murine breast cancer model

Ximena Yazmin García Bustamante, Universidad Nacional Autónoma de México (UNAM), Mexico



Angus B Gordon

Imperial College Healthcare Trust, London W6 8RF

Tumour Volume Analysis applied to Breast cancer

Abstract

This study evaluates the limitations of the Response Evaluation Criteria in Solid Tumours (RECIST), which assesses tumour response based on changes in the longest diameter (LD), and explores the application of Tumour Volume Analysis (TVA) as a more accurate alternative. RECIST defines partial response (PR) as a 30% reduction and progressive disease (PD) as a 20% increase in tumour diameter. In contrast, TVA incorporates three diametric measurements to calculate tumour volume, potentially providing a more precise assessment of tumour response.

A pilot investigation was conducted on breast cancer patients undergoing neoadjuvant chemotherapy with evaluation through magnetic resonance imaging (MRI). Tumour response was analysed using both RECIST and TVA parameters. Among the evaluated cases, RECIST misclassified a significant proportion of patients compared to TVA results. The application of TVA increased the number of patients classified as having a partial response and reduced the number categorized as stable disease.
These findings demonstrate that reliance on a single linear measurement may lead to inaccurate tumour response categorisation. TVA, by incorporating volumetric measurements, improves the accuracy of tumour assessment and may have important implications for treatment planning and patient management in breast cancer.

Biography

Formerly Consultant Breast Surgeon at Imperial College Healthcare Trust (at Charing Cross Hospital 2006 -2014) London SW6 .Previously Consultant Breast Surgeon at Royal Marsden

Hospital ,Fulham Road, London SW3.

Presentations and Posters. Tumour Volume Analysis.

San Antonio Breast Cancer Symposium 2007,2008,2009

Chicago ASCO 2015

Vienna ECR European Congress of Radiology 2016.

European Journal of Surgical Oncology. EJSO: 2025 ; 51, 109578.

Andrew L. Wolfe

Hunter College, City University of New York (CUNY), USA

Modulating sensitivity of cancers to Ras inhibitors

Abstract

KRAS mutations, which induce proliferative signaling driving many human cancers, are detectable in many non-small cell lung cancers, pancreatic ductal adenocarcinomas, colorectal cancers, and a small subset of osteosarcoma (OS) patients. The recently developed pan-KRAS inhibitor daraxonrasib, also known as RMC-6236, is capable of targeting a wide array of KRAS mutations and shows promise against pancreatic and lung cancers. However, the efficacy and mechanisms of action of daraxonrasib in osteosarcoma remain unclear. We evaluated the effects of daraxonrasib on the viability, proliferation, and metastatic potential of wild-type and KRAS mutant OS cells. We assayed the effects of treatment on downstream targets using qPCR, immunoblotting, and activity assays to explore the underlying mechanism by which daraxonrasib selectively suppresses the metastatic potential of KRAS mutant osteosarcoma. We investigated how the increased prevalence of GTP-bound KRAS enhanced the sensitivity of KRAS wild-type osteosarcoma cells to daraxonrasib using siRNA targeting RASA1. Daraxonrasib selectively attenuated the proliferation and migratory ability of KRAS mutant KRAS mutant cells without affecting KRAS wild-type controls. Additionally, daraxonrasib suppressed the expression of the matrix metalloproteases MMP9 and MMP1, which promote cell motility and metastasis. Daraxonrasib selectively inhibited the AKT/ETS1 pathway in KRAS mutant cells, whereas no such effect was observed in WT cells. KRAS WT cells were sensitized to daraxonrasib by knocking down the GTPase-activating protein RASA1. KRAS inhibition decreased MMP1, MMP9, and AKT/ETS1 signaling. Daraxonrasib is a promising agent for treating osteosarcoma with activation of KRAS signaling, and exploration of genotype sensitivities and combination therapeutics may improve patient outcomes.

Biography

Andrew Wolfe completed his doctoral research at Weill Cornell Medicine in a laboratory at Memorial Sloan Kettering Cancer Center, studying how protein translation influences T-cell leukemia. Following postdocs at Mount Sinai and the University of California San Francisco, where he was the recipient of a fellowship from the Damon Runyon Cancer Research Foundation and a K99/R00 award from the National Cancer Institute, he launched the Wolfe lab at Hunter College. The lab focuses on Ras biology, including mechanisms of regulation, approaches toward inhibition, and strategies for overcoming therapeutic resistance.

Moi Sankar Chandra

National Institute of Technology Durgapur, Durgapur 713209 WB, India

A route map to Pt(II) based anticancer drug design and development

Abstract

Recently, cancer is going to be a challenging global threat and the second leading cause of unnatural human death. This sobering reality has encouraged to develop safer and more effective anticancer drugs, to prevent ultimately loss of human life. The serendipitous discovery of anticancer activity of cisplatin[1], [Pt(NH3)2Cl2] by B. Rosenberg, which was first recognized in 1978 by FDA, USA for the treatment of several types of cancer. However, cisplatin's notable side effects posed significant challenges.

To reduce the side effect and toxicity[2,3], next generation drugs like carboplatin and oxaliplatin were recognized, but not fully devoid of toxicity. We have designed and synthesized heterocyclic diamine based Pt(II) complex [Pt(diamine)Cl2], C1 and its hydrolysed product [Pt(diamine)(OH2)2](NO3)2, C2. The thiol containing L-cysteine (L-cys) and Glutathione (GSH) chelated Pt(II) complex [Pt(diamine)(L-cys)]+, C3 and [Pt(diamine)(GSH)], C4 were synthesized from complex C2 and were characterized for the same. The binding aptitude of the Pt(II) complexes (C1- C4) with bio-macromolecule BSA and DNA were performed to evaluate their binding mode and binding constants. The drug likeness property was evaluated by PASS prediction and molecular docking of the complexes were executed with DNA and BSA. The complexes have better anticancer activity against MCF-7 and MDAMB231 cancer cell lines and less toxic on human normal embryonic kidney cell line HEK293T.

The complexes are less toxic on normal cells, whereas more effective on cancer cells compared to recognized anticancer drug cisplatin, carboplatin and oxaliplatin. The cells

(MCF-7) undergo necrosis when treated with the complexes (C1 – C4). Their lower level of ROS generation in MCF-7 cell lines are comparable with the recognized anticancer drugs. Their cell death mechanism(Cell cycle arrest) was executed to confirm the drug activity on cancer cell lines.

Xiaomei Wang
Founder & CEO, PathoAI, Canada

From Pixels to Precision: How Multimodal AI is Redefining Pathology Diagnostics and Drug Discovery

Abstract

The integration of multimodal artificial intelligence (AI) into pathology is revolutionizing both diagnostics and pharmaceutical research. At PathoAI, we have developed medical device license granted multimodal pathology foundation model capable of recognizing 57 tumor subtypes across 9 organs. This breakthrough exemplifies how AI can transform static pathology images into dynamic, clinically actionable insights.

Our approach combines three pioneering innovations:

1. Organ-Specific Feature Pyramid Networks, enabling hierarchical analysis from cellular to whole-slide features;
2. Pathology Chain-of-Thought framework, which replicates and visualizes diagnostic reasoning paths with human-interpretable logic;
3. Lightweight deployment architecture, making precision pathology accessible even in resource-limited settings.

Beyond diagnostics, our multimodal AI serves as the intelligent engine for our digital pathology CRO platform, where it accelerates drug and medical device development through automated, quantitative analysis of therapeutic effects across species. By integrating pathology images with clinical, genomic, and imaging data, we are creating a new paradigm where AI bridges traditional diagnostic boundaries.

This presentation will demonstrate:

- Real-world cases where our model achieved 98.7% concordance with expert

pathologists in tumor subtyping;

- How pharmaceutical partners reduced preclinical pathology analysis time by 70%;
- The roadmap toward multimodal precision oncology, where AI synthesizes pathology, EHRs, and multi-omics data for MDT decision support.

The future of pathology lies not in replacing human expertise, but in amplifying it through AI that understands both pixels and clinical context. Join us to explore how this convergence is reshaping global healthcare standards.

Biography

Xiaomei Wang is the Founder and CEO of PathoAI, a pioneering company at the forefront of AI-driven diagnostics and biopharmaceutical research. With over 20 years of expertise in AI and data analytics, she previously served as IBM's Global Leader of Big Data & Analytics, spearheading transformative projects worldwide. At PathoAI, she leads the development of cutting-edge multimodal AI models that enable highly accurate, multi-organ cancer analysis to advance patient care and accelerate medical research.

A recognized thought leader, Xiaomei is also a guest professor at CSIRO (Australia) and Zhejiang University of Finance & Economics. She is the best-selling author of influential works such as "Industry Innovation in the Era of Artificial Intelligence – The AI Compass" and "AI 3.0," cementing her role as a prominent voice in technology and healthcare innovation.

Dave SB Hoon

Saint Johns Cancer Institute, USA

Multiomics cfDNA Fragmentomics Monitoring Methylation Variants and SNPs Of Melanoma Patients Receiving Checkpoint Inhibitor Immunotherapy

Abstract

Checkpoint inhibitor immunotherapy (CII) has significantly prolonged AJCC stage III/IV melanoma patients' survival in the past several years through multiple human monoclonal antibodies targeting checkpoint proteins on both melanoma and immune cells. However, there are no efficient blood biomarkers to determine the efficiency of CII in realtime and determining when to switch therapy to improve patient overall survival. We have developed various forms of cfDNA blood melanoma biomarkers in the past. Traditional approaches assessing specific cfDNA genes for mutations and metthylation changes which we pioneered used realtime PCR assays then developed into multiple gene-based probe assays. More recent we developed a novel multiomic whole genome sequence (WGS) cfDNA fragmentomic platform based on 6 base sequences that covered methylation associated variants (MAV) of both 5-methyl cystine(5-mC) and 5-hydroxy mC(5-hmC) of gene promoters, bodies and non-coding regions of the genome. The MAVs fragmentomics analysis using machine learning were performed on the same WGS platform involving NGS 30x paired end reads. Through this platform we carried out analysis on longitudinal clinically well annotated CII patients blood categorized with progressive disease. death, partial response, stable disease on specific patients's bleeds during treatments between 3- 24 mos. Results of patients demonstrated both significant 5-mC and 5-hmC were detected whereby, levels of quantitative changes were related to patient responses during treatment. In addition, we detected WGS SNVs changes during CII treatment which demonstrated

significant changes parallel to clinical changes. Specific melanoma-related gene SNVs such as in BRAF, EGFR, ARIDIA, JAK, and TERT genes, etc were monitored for changes during treatment. These studies demonstrated through WGS genomics/ epigenomic multiomics analysis of CII patients cfDNA have clinical utility.

Biography

Is Professor and Director Depts Translational Molecular Medicine and NGS Center, Saint Johns Cancer Institute, Providence Health System. His Google Scholar H-score is 123 with > 450 peer-reviewed publicatiion mostly in molecular oncology as related to solid human tumor clinical studies of which many in high impact journals. He is a pioneer of cfDNA and has published and patented assays in mutation, methylation, amplification, and more recently methylation associated variants and SNP fragmentomics in clinical studies since the 90s. He has been involved in multiple clinical phase CDx clinical studies that include immunotherapies and sentinel lymph node multicenter clinical trials. His focus is in melanoma but also as well in other solid tumors. He also researchs on ubiquitin proteomic cancer regulatory events in tumor progression and resistance. He is a senior reviewer on multiple NCI/DOD grant study sections for >25 yrs; also founding member of NCI Cancer Biomarker study section.

Qingjie Li

The University of Texas Medical Branch at Galveston, USA

Erlotinib Suppresses Tumorigenesis in a Mouse Model of Colitis-Associated

Abstract

Colorectal cancer is the third most diagnosed cancer and second most common cause of cancer mortality worldwide. Colitis-associated cancer (CAC) in inflammatory bowel diseases exhibits more aggressive behavior than sporadic colorectal cancer; however, the molecular mechanisms remain unclear. No definitive preventative agent against CAC is currently established in the clinical setting. We investigated the molecular mechanisms of CAC in the azoxymethane/dextran sulfate sodium (AOM/DSS) mouse model and assessed the antitumor efficacy of erlotinib, a small molecule inhibitor of the epidermal growth factor receptor (EGFR). Erlotinib premixed with AIN-93G diet at 70 or 140 parts per million (ppm) inhibited tumor multiplicity significantly by 96%, with ~60% of the treated mice exhibiting zero polyps at 12 weeks. Bulk RNA-sequencing revealed more than a thousand significant gene alterations in the colons of AOM/DSS-treated mice, with KEGG enrichment analysis highlighting 46 signaling pathways in CAC development. Erlotinib altered several signaling pathways and rescued 40 key genes dysregulated in CAC, including those involved in the Hippo and Wnt signaling. These findings suggest that the clinically-used antitumor agent erlotinib might be repurposed for suppression of CAC, and that further studies are warranted on the crosstalk between dysregulated Wnt and EGFR signaling in the corresponding patient population..

Biography

Dr. Qingjie Li is a Professor in the Division of Gastroenterology and Hepatology, Department of Internal Medicine at the University of Texas Medical Branch (UTMB) at Galveston and the President/Founder of ClearLi Biomedicines LLC. He received his PhD from Central South University in China and completed postdoctoral training at Oregon State University. Dr. Li's research centers on colorectal cancer, the gut microbiome, inflammatory bowel disease (IBD), and its extraintestinal manifestations, as well as the development of novel therapeutics for digestive diseases and aging-related conditions. He serves as a reviewer for several NIH study sections, Digestive Disease Week, and multiple scientific journals. Dr. Li has published more than 50 peer-reviewed articles in leading journals, including Gastroenterology.

Ronit Ilouz
Bar-Ilan University, Israel

Spatial Profiling of Protein Kinase A Subunits Identifies Aggressive Prostate Cancer in MRI-Targeted Biopsies

Abstract

Prostate cancer is a multifocal disease with substantial spatial and biological heterogeneity that challenges accurate risk assessment. Most molecular biomarker studies rely on bulk or cross-sectional analyses that average signals across patients and lesions, often obscuring spatially localized molecular alterations relevant to tumor aggressiveness.

To address this limitation, we performed lesion-matched molecular profiling of MRI-targeted prostate biopsies obtained from both suspicious and non-suspicious regions within the same prostate. Protein Kinase A (PKA) subunit expression and pathway activity were analyzed using quantitative immunofluorescence with a custom image-analysis pipeline, Western blotting of paired biopsy samples, and re-analysis of publicly available proteomics datasets from diagnostic (n=116) and recurrence (n=306) prostate cancer cohorts.

We identified a grade-dependent shift in PKA signaling characterized by progressive loss of the regulatory subunit RIβ, redistribution of the catalytic subunit PKAC into proliferating tumor cells, and increased global phosphorylation of PKA substrates in suspicious lesions. These spatial alterations were consistently observed within individual patients and correlated with tumor Grade Group. Analysis of bulk proteomics datasets revealed high inter-patient variability of RIβ and enrichment of catalytic subunits in recurrence-associated tumors, highlighting the limitations of non-spatial analyses. Notably, nearly half of recurrence-associated proteins contained predicted

PKA phosphorylation motifs, supporting increased pathway activity in aggressive disease.

In conclusion, spatial profiling of PKA subunits in MRI-targeted biopsies reveals a reproducible molecular signature associated with prostate cancer aggressiveness. These findings establish the PKAC:RIβ imbalance as a potential biomarker and demonstrate the value of intra-patient, spatially resolved molecular analysis for improving prostate cancer diagnostics and risk stratification.

Biography

Dr. Ronit Ilouz is an Assistent Professor at the Azrieli Faculty of Medicine, Bar-Ilan University. Her research focuses on spatial signaling mechanisms in cancer and neurodegeneration, combining quantitative imaging, proteomics, and structural biology to identify clinically relevant biomarkers. She has contributed to defining Protein Kinase A (PKA) dysregulation in human disease, including lesion-specific biomarkers in prostate cancer. Dr. Ilouz is supported by the Israel Science Foundation, Israel Cancer Research Fund, and the U.S.–Israel Binational Science Foundation.

NABWIRE SUSAN

Uganda Women Cancer Support Organisation, Uganda

How Palliative Is Counselling To Cancer Patients

Abstract

Globally the number of newly diagnosed cancer cases increased by 33% between 2010 and 2017, and a progressive increase in cancer incidence was expected, according to a feasibility study conducted at Uganda Cancer Institute (UCI) about 3500 new case surge in Uganda every year from 2021 with only 30% surviving and 70% succumbing to the disease. Therefore, this study aimed at finding out how palliative is counselling to cancer patients. In particular the study sought to; examine the contribution of counselors in instilling hope among the cancer patients, assess relevance of Hope among the cancer patients and examine challenges that affect the provision of counseling services to the counselors. The study adopted exploratory research design. The exploratory research focuses on words more than numbers. This does not mean that qualitative data could not also be presented in table format the data is usually just expressed in words rather than numbers. Qualitative research is more concerned with defining and explaining concepts which arrive at meaningful and subjective conclusions. It helped the researcher to obtain information in the shortest period of time.

The results revealed that a huge number of cancer patients diagnosed with cancer know very limited information about it. It is the diagnosis that quickly shatters a person's feelings of invulnerability and immortality. The fact that the biggest number of patients succumb to death most of the patients upon diagnosis go into depression. The results further indicated cancer takes one's strength, destroys organs and bones, and weakens the body's defenses against other illnesses and it is so expensive to treat. It is not surprising that people diagnosed with cancer are marginalized and feel isolated as they are removed from healthy world, this is the effect of such shock and devastation

the diagnosis brings to the system

The study concludes that cancer has a significant impact on individuals and their families, resulting in unique stresses that arise during the course of treatment and the need for effective coping patterns to deal with these strains. The study recommends that counseling interventions should be immensely incorporated in medical centers and should be provided by professionals in order to create positiveness in the minds of the cancer patients as well as family care givers.

Biography

Formerly Consultant Breast Surgeon at Imperial College Healthcare Trust (at Charing Cross Hospital 2006 -2014) London SW6 .Previously Consultant Breast Surgeon at Royal Marsden Hospital ,Fulham Road, London SW3.

Meital Gal-Tanamy
Bar-Ilan University, Israel

Epigenetic mechanisms of hepatocellular carcinoma post HCV cure

Abstract

Hepatitis C virus (HCV) is a major cause of death and morbidity globally and the leading cause of hepatocellular carcinoma (HCC). Although now, with new direct-acting antivirals (DAAs) therapy available, HCV is a curable cancer-associated infectious agent, HCC prevalence is expected to continue to rise because HCC risk still persists after HCV cure. Understanding the factors that lead from HCV infection to HCC pre- and post-cure may open-up opportunities to novel strategies for HCC prevention. We recently reported the induction of alterations in the transcriptome of host cells via epigenetic dysregulation by HCV that persist after cure by DAAs as an epigenetic signature. This epigenetic signature is associated with hepatocarcinogenesis. Different treatment regimes show range of persistence of the epigenetic signature that correlate with treatment efficiency. Moreover, HCV induce the epigenetic and oncogenic signatures by perturbation of host signaling pathway, such as EGFR. We also identified correlation between HCV-induced changes in epigenetic marks associated with chromatin decompaction and mutation loads in HCV-related HCC. Inhibitors for epigenetic modifiers showed promising results as means for reversion of HCV-related epigenetic signature and oncogenic phenotypes. These studies have important contribution for understanding of the mechanisms of HCV-induced cancer pre and post cure..

Biography

Associate Professor at the Azrieli Faculty of Medicine, Bar-Ilan University, I lead the Molecular Virology Lab and serve as President of the Israel Society of Microbiology (ISM). With 22 years *of experience in virology, my work centers on Hepatitis C virus (HCV) pathogenesis and virus–host interactions that contribute to liver disease and hepatocellular carcinoma (HCC). My research dissects the interplay between HCV-driven inflammation, chromatin and epigenetic remodeling, tumor-associated mutations, viral evolution, and immune responses to explain mechanisms linking infection to HCC risk. Our lab generated the first comprehensive map of HCV-induced epigenetic alterations and was first to show persistence of an HCV-induced "epigenetic signature" after viral clearance with direct-acting antivirals (DAAs). I have been invited to speak at national and international conferences, including keynote presentations at the HCV 2021 international meeting and the 2024 APASL meeting in Taiwan on post-cure epigenetic signatures.*

Hassan Dawud Jidda

Sharda University, India

The role of curcumin in apoptosis in breast cancer cell and normal cell line

Abstract

Apoptosis is a Programmed cell death that is important determination to response to chemotherapy among the factors controlling this process a significant role played by p53,caspase 3,Bcl2 and p21,The expression of which, together with estrogen receptors content and tumor proliferative activity was investigated in breast cancer cell line, the aim of my study is to evaluate how curcumin can influence the growth of normal and breast cancer cells, and also to evaluate how curcumin can regulate Apoptosis related genes expression in normal and breast cancer cells . The Curcumin inhibited cell growth in cancer cell MCF-7, however in normal cells curcumin does not affect the cell growth, does amply that it is not harmful to normal cell but its toxic to cancer cells and also it is anti-carcinogenic. Curcumin can regulate apoptosis by inducing Caspase 3, p53, p21 and Bcl2 in MCF-7 cancer cells. Does imply that curcumin specifically target breast cancer cell and not normal cells gene expression. Hence curcumin induces apoptosis in cancer cells.

Keyword: apoptosis, Curcumin, cell death, caspases.

Arezoo Esmaeili

Islamic Azad University, Damghan, Iran

Evaluating the Efficacy of Peptide-Based Vaccines in Cancer Treatment: A Systematic Review

Abstract

Background and Aim: Peptide-based vaccines have emerged as a promising approach in cancer immunotherapy, harnessing the immune system to target tumor-associated antigens. However, the efficacy and utility of these vaccines in different types of cancer require comprehensive evaluation. This systematic review aimed to investigate the impact of peptide- based vaccines on different types of cancer, assessing their efficacy, safety, and potential for integration into existing treatment regimens.

Methods: A systematic search of electronic databases including Web of Science, PubMed, and Scopus was conducted to identify relevant studies from 2015 to 2025. The quality of studies was assessed using the Critical Appraisal Skills Program (CASP) tool. Moreover, data extraction and synthesis were performed in accordance with the PRISMA guidelines.

Results: A total of 15 studies met the inclusion criteria, covering a wide range of cancers including melanoma, breast, prostate and lung cancer. The findings showed that peptide- based vaccines elicited significant immune responses in many cases, with some studies reporting improvements in overall survival and progression-free survival. However, variability in efficacy was observed across cancer types, with melanoma showing the most promising results. Adverse events were generally mild to moderate, mainly consisting of local reactions at the injection site.

Conclusion: Peptide-based vaccines have shown potential as a valuable addition to cancer treatment strategies, particularly in certain cancer types such as melanoma.

Despite the encouraging results, further large-scale clinical trials are necessary to establish standardized protocols and identify optimal patient populations for these vaccines.

Biography

virology and cancer research, particularly in the study of Epstein-Barr Virus (EBV) and its role in oncogenesis. including my recent review on EBV's modulation of programmed cell death, which provides crucial insights into viral oncogenesis and potential therapeutic interventions. my expertise in molecular biology, immunology, and vaccine development directly aligns with interest in combating infectious diseases and virus-associated cancers.

Inès GARROUCHE
Université de Poitiers, France

New insights of YAP activity in brain metastases from colorectal cancer

Abstract

Brain metastases (BM) represent the majority of malignant intracranial tumors and a life-threatening complication for patient with colorectal cancers (CRC). Currently, YAP and TAZ, belonging to the Hippo signaling pathway, are considered as crucial malignancy factors in many solid tumors. In this work, we studied the impact of the transcriptional coactivator YAP in two different cohorts of CRC patients (PETACC8 cohort including 327 patients with grade III and a local cohort from Poitiers with 79 grade IV patients with BM) as well as its rôles in brain metastasis stem cells derived from CRC patients (BM-SC-CRC). First, we found that YAP expression was significantly higher in the BM cohort and associated with the tumor stage at diagnosis. However, we did not find a significant association with patient prognosis in both cohorts. In vitro, we showed that YAP was involved in proliferation and survival as its selective inhibition by verteporfin reduced the viability of BM-SC-CRC cultures. To get insight into the role of YAP in brain metastasis, we found using spatial transcriptomic approach that this coactivator was strongly expressed in tumor area with metabolic changes. Notably, YAP inhibition induced a decrease of mitochondrial activity with reduced NUAK2 expression, suggesting a role of YAP in regulation of cellular respiration. Altogether, our results highlight a potential role of YAP in CRC progression, particularly in BM stem cells.

Biswajit Dey
National Institute of Pharmaceutical Education and Research, Telangana, India

Glutamate dehydrogenase (GDH) Promotes Survival of Triple-Negative Breast Cancer Persister Cells Through Regulation of EMT and Anaplerotic Metabolism

Abstract

Chemotherapeutic treatment destroys the majority of cancer cells, but a small percentage survives through genetic and non-genetic pathways. These surviving cells, known as drug-tolerant persister (DTP) cells, are major causes of tumour recurrence. This study aimed to identify key factors regulating DTP cells in triple-negative breast cancer (TNBC) and to elucidate the underlying mechanisms conferring their survival advantage. In this study, we found that glutamate dehydrogenase (GDH) plays a critical role in DTP cell survival by regulating epithelial-to-mesenchymal transition (EMT), mitochondrial metabolism. Bioinformatic analyses revealed that elevated GDH expression in breast cancer patients correlates with poor prognosis. Western, qRT-PCR, and immunofluorescence analyses confirmed GDH upregulation in DTP cells, which was further supported by mass spectrometry and extracellular flux assay. Cell viability and wound healing assays demonstrated that GDH upregulation enhanced DTP cell viability and migratory capacity, whereas suppression produced the opposite result. Moreover, stable GDH knockdown impaired EMT and DTP formation. Notably, GDH expression declined after 30 days of drug withdrawal, suggesting a transient regulatory role during the DTP state. In vivo xenograft experiments further demonstrated, chemotherapeutic treatment in GDH knockdown xenografts significantly enhances chemosensitivity. Collectively, our data suggest that GDH is a crucial regulator of DTP cell survival and targeting GDH1 may represent a promising therapeutic strategy to overcome chemoresistance in TNBC

Biography

Biswajit Dey is currently pursuing his Ph.D. at the National Institute of Pharmaceutical Education and Research (NIPER), Hyderabad, where he enrolled in March 2023, and serves as a Senior Research Fellow (SRF). His research focuses on elucidating the molecular mechanisms driving cancer progression and therapeutic resistance, with an emphasis on identifying novel molecular targets for treatment. He has co-authored several peer-reviewed publications, demonstrating his active involvement in collaborative research. Biswajit possesses strong expertise in molecular biology and cell culture techniques, including Western blotting, qRT-PCR, immunofluorescence microscopy, and gene expression analysis. In addition to his technical proficiency, he is known for his effective communication and presentation skills, often delivering scientific talks with confidence and clarity. His analytical mindset and dedication to translational cancer research position him as a promising young scientist in the field of molecular oncology.

Wonchung Lim

Cheongju University, Cheongju, South Korea

The glucocorticoid receptor suppresses lung inflammation induced by nicotine-derived nitrosamine ketone by suppressing cyclooxygenase-2 in A549 cells

Abstract

It is evident that smoking cigarettes constitutes a considerable risk factor for the development of lung cancer. The role of glucocorticoid receptors (GR) in the context of the cyclooxygenase-2 (COX-2)/prostaglandin E2 (PGE2) pathway induced by nicotine-derived nitrosamine ketones (NNK) remains to be elucidated. The NNK-induced increase in COX-2 expression and PGE2 production was stopped by glucocorticoids, and this effect was blocked by the GR antagonist, RU38486. What's more, dexamethasone stopped NNK-induced cell invasion. In short, the data show that a substance called NNK produces a chemical reaction that causes the growth of lung cancer cells. The findings of the study provide a valuable insight into the pathophysiological connection between NNK, GR, inflammatory responses, and cell metastasis.

Biography

Lim finished his PhD at 31 years old from Sejong University and postdoctoral investigations from Harvard Medical School. He is an associate professor at Cheongju University. He has published more than 30 scientific papers on the subject of inflammation.

Botao Zhang

The Affiliated Hospital of Southwest Medical University, China

Human Adipose-Derived Stem Cells Promote Migration of Papillary Thyroid Cancer Cells via the Leptin Pathway

Abstract

Objective:
Obesity is associated with an increased risk and poor prognosis of thyroid cancer, but the underlying mechanisms remain unclear. This study aimed to investigate the role of human adipose-derived stem cells (ADSCs) in promoting the migration and invasion of papillary thyroid cancer (PTC) cells via the leptin pathway.

Methods:
In Vitro Experiments: ADSCs-conditioned medium (ADSC-CM) was collected and used to culture PTC cell lines (TPC-1 and BCPAP). Cell migration and invasion were assessed using Trans well and wound healing assays. Cell proliferation and viability were evaluated via EdU incorporation and CCK-8 assays. Leptin levels in ADSC-CM and PTC cells were measured by ELISA. The role of leptin was further examined by adding leptin-neutralizing antibodies (NAB) to ADSC-CM. Western blotting was performed to analyze the expression of leptin receptor (Ob-R), matrix metalloproteinase-2 (MMP-2), and MMP-9.

In Vivo Experiments: A nude mouse xenograft model was established by injecting TPC-1 cells into the tail vein. The leptin receptor antagonist Allo-aca (1 mg/kg/day) was administered subcutaneously to block the leptin pathway. After 8 weeks, lung metastatic nodules were counted, and their diameters were measured. Serum levels of T3, T4, and TSH were determined by ELISA.

Results:

ADSC-CM Promotes PTC Cell Migration and Invasion: ADSC-CM significantly enhanced the migration (wound healing assay: 50% increase in scratch closure rate, $p < 0.01$) and invasion (Trans well assay: 2.5-fold increase in migrated cells, $p < 0.001$) of TPC-1 and BCPAP cells. ADSC-CM upregulated MMP-2 expression ($p < 0.01$) but had no effect on MMP-9. Leptin Mediates the Pro-Migratory Effects of ADSCs: ADSC-CM contained ~50 pg/mL leptin, 3-fold higher than control medium ($p < 0.001$). Leptin NAB attenuated ADSC-CM-induced migration (30% reduction, $p < 0.05$) and invasion (40% reduction, $p < 0.01$), and downregulated MMP-2 expression ($p < 0.05$).

In Vivo Validation: Allo-aca treatment reduced the number of lung metastatic nodules by 60% ($p < 0.001$) and decreased their diameter ($p < 0.05$). Serum T3 levels were elevated in Allo-aca-treated mice ($p < 0.05$), while T4 and TSH remained unchanged.

Conclusion:

ADSCs promote the migration and invasion of PTC cells through paracrine leptin, which upregulates MMP-2 expression via the leptin/Ob-R pathway. These findings highlight a key mechanism linking obesity to thyroid cancer progression and suggest that targeting the leptin pathway may offer a therapeutic strategy to mitigate PTC metastasis.

Biography

BoTao Zhang finished his MD at 29 years old years from Southwest Medical University. Over the past three years, he has authored more than 10 research publications, with a cumulative impact factor exceeding 46.

Janusz Rak
McGill University, Canada

Harnessing extracellular vesicle pathways to target vascular peculiarities and immune cell exclusion in glioblastoma

Abstract

Glioblastoma (GBM) is an incurable brain cancer, where inevitable recurrence follows initial intervention involving surgery and radio-chemotherapy. GBM relapse is driven by tumour-initiating glioma stem cells (GSCs) 1, which survive and expand in the brain after tumour depopulation. GBM progression unfold amidst the expansion of the abnormal tumour vasculature and scarcity of infiltrating cytotoxic CD8+ T cells and natural killer (NK) cells 2. Although the nature of this immune cell exclusion is poorly understood, it occurs on the background of intercellular communication within the vascular tumour microenvironment, involving physical contacts, soluble factors and extracellular vesicles (EVs). EVs are cellular fragments enveloped in the plasma membrane, and capable of transferring bioactive cargo, including mutant oncogenes and their effectors between cellular populations 3. EVs are also known to be released by two distinct subsets of GSCs, with either proneural (PN) or mesenchymal (MES) molecular profiles, each exhibiting unique vascular activities 4,5. Thus, while PN-GSC form highly angiogenic and slow growing tumours in immune deficient mice, their MES-GSC counterparts drive rapid tumour expansion associated with non-angiogenic dilated vasculature, formed through a process termed ‘vasectasia’. This peculiar vascular growth is associated with overexpression of the oncogenic epidermal growth factor receptor (EGFR), and its mutant variant III (EGFRvIII) and their EV-mediated transfer between cancer cells and endothelium5. Conversely, endothelial derived (angiocrine) EVs modulate PN-GSCs to adopt a more MES-GSC-like and invasive phenotype 6. Targeting Rab27a/b pathway of EV biogenesis in tumour recipient mice results formation of dilated and leaky vascular channels permissive of immune cell infiltration into the tumour a process

leading to improved responses to systemic adoptive T cell immunotherapy 7. Of note, human GSCs exhibit subtype specific sensitivity to immune effectors. For example, MES-GSCs (unlike PN-GSCs) are highly sensitive to NK cell mediated killing, which if properly timed with temozolomide chemotherapy in vivo, can lead to curative effects in mice8. These effects can be recapitulated by intracranial delivery of NK cell-derived cytotoxic EVs 9. Thus, EV-mediated interactions between different subsets of cancer cells, endothelial cells and immune effectors profoundly impact the biology of GBM and may inspire new therapeutic approaches.

Biography

Janusz Rak, MD, PhD is a Professor of Pediatrics and Jack Cole Chair in Pediatric Hematology/Oncology at McGill University and Investigator at the Research Institute of the McGill University Health Centre (RIMUHC). His laboratory investigates how oncogenic events deregulate tumour microenvironment, orchestrate intercellular communications and trigger vascular alterations and systemic vascular paraneoplastic syndromes in cancer. The focal point of these studies are processes mediated by the exchange of extracellular vesicles (EVs), including exosomes carrying oncogenic, diagnostic and therapeutic cargo. He published over 220 research papers (43,000 citations, h-index 87). He currently directs the CFI funded program – Centre for Applied Nanomedicine (CAN) and the NET program sponsored by Fondation Charles Bruneau and CIBC to investigate EV-based liquid biopsy approaches in pediatric cancer. He also leads projects supported by Canadian Institutes of Health Research, Cancer Research Society and other sources. He is a Fellow of the Royal Society of Canada

Rinal Chavda

Cancer Research Institute, ACTREC, TMC, Navi Mumbai, Maharashtra, India.

Identification of Histological and Molecular characteristics associated with high-risk Oral leukoplakia

Abstract

Oral leukoplakia (OL) is the most common oral potentially malignant disorder with 30% to 50% lifetime risk to develop oral squamous cell carcinoma (OSCC). In clinic, histopathological grading remains the gold standard to identify lesions at risk of progression into OSCC. However, the significance of individual histological characteristics and underlying molecular alterations associated with progressive OL remains unexplored. Furthermore, prospective studies with comprehensive understanding are lacking. The Institutional Ethics Committee approved the study protocol and signed informed consent was obtained from all the participants. This study included 498 OL tissue and blood samples. Pathologists SR and NM designed an assessment form featuring the architectural and cellular changes in OL cases. Haematological parameters were noted at the time of enrolment. DNA was extracted from OL tissues, and the presence of HPV was detected. The copy number was evaluated for 15 genes using DD-PCR. Follow-up at regular intervals of 6 months was done, and clinical outcomes were recorded as free of disease, persistence of lesion, recurrence of lesion, and malignant transformation (MT). The mean follow-up period was 31.73 months. In the histopathological assessment of 25 cellular and architectural features, we observed a significant association of dyskeratosis, nuclear hyperchromatism, enlarged nucleoli, and irregular epithelial stratification in both recurrence of lesion and MT. Additionally, an abnormal form of mitosis was observed in MT. Interestingly, inflammation in tissue and low platelet to lymphocyte ratio in blood were strongly associated with MT. All cases were negative for HPV. Using DD-PCR, we identified novel prognostic biomarkers to stratify high-risk leukoplakia

patients. Through this information, we provide a model to identify progressive lesions. Collectively, this prospective study provides a comprehensive understanding of individual histopathological features, along with CNAs associated with the disease progression. Our findings may aid to stratify high-risk OL patients and determine the prognosis of precancerous lesions, thus helping in clinical management.

Biography

Ms. Rinal Pala Chavda is a Ph.D. scholar at the Advanced Centre for Treatment, Research and Education in Cancer (ACTREC), Tata Memorial Centre, where she is currently in the fifth year of her doctoral program. Her research focuses on the molecular characterization of oral precancer and oral squamous cell carcinoma, with a particular interest in studying copy number alterations and their prognostic significance. She has published her work in peer-reviewed journals, including a recent article titled "Comparison of Real-time PCR and nCounter NanoString Techniques to Validate Copy Number Alterations in Oral Cancer" in Scientific Reports (PMID: 40595185). She has also submitted an additional manuscript based on her thesis findings. Her broader research goals include improving early detection, risk stratification, and biomarker-guided management of oral potentially malignant disorders. She aims to contribute to translational cancer research by integrating molecular insights with clinical applications to improve patient outcomes.

Esmael Besufikad

Cancer and Other non-communicable Disease Research Divisison, AHRI, Ethiopia

High Tumor Mutation Burden in Triple-Negative Breast Cancer among Young Ethiopian Women: Insights from Targeted Sequencing for Improved Diagnosis and Care

Abstract

Breast cancer remains a major public health concern in Ethiopia, characterized by considerable molecular and clinical diversity. Genomic alterations, including somatic mutations, play a crucial role in breast cancer diagnosis, prognosis, and treatment. However, genomic data on Ethiopian breast cancer patients remain limited. Therefore, this study aimed to explore somatic mutations with potential clinical significance among Ethiopian breast cancer patients to enhance understanding of the disease's molecular landscape. Targeted sequencing of 294 cancer-related genes was performed on 39 formalin-fixed paraffin-embedded breast cancer samples. Sequence data were analyzed using bioinformatics tools, including Qiagen GeneGlobe and smCounter2 for variant detection. VCF files were processed into MAF format using the Genome Analysis Toolkit (GATK) with the GRCh38 human genome reference. MAF files were further analyzed and visualized using the maftools package in R for mutation summarization, annotation, and visualization. A high number of somatic mutations were identified across the analyzed samples. Clinically relevant genes, including BRCA1, BRCA2, TP53, PIK3CA, and ESR1, exhibited a elevated mutation burden. Furthermore, key oncogenic signaling pathways such as NOTCH, RTK-RAS, TP53, and WNT were frequently affected by somatic alterations. The tumor mutation burden (TMB) was notably higher in triple-negative breast cancer (TNBC) cases and in samples derived from Jimma compared to other subgroups. The observed high tumor mutation burden, particularly in clinically relevant genes and TNBC cases, provides significant insights

that may inform the development of future targeted and immunotherapeutic strategies.

Biography

I am an aspiring biomedical researcher with a focus on breast cancer genomics and molecular biology. I am building my experience in immunohistochemistry, genomic sequencing, and bioinformatics through hands-on laboratory and clinical research. My work explores somatic and germline mutations, breast cancer subtyping, and clinicopathological characteristics to better understand genetic alterations and subtype-specific factors that contribute to breast cancer development, progression, and treatment response. I am passionate about generating genomic data that can have a meaningful impact on the development of future precision therapies in Ethiopia, aiming to advance personalized approaches to breast and other cancer care.

Jiang Li

Sun Yat-sen University Cancer Center, China

Modulation of Anti-Tumor Immunity: Lactate-Driven Ferroptosis of CD8+ T Cells in the Tumor Microenvironment and Its Therapeutic Implications

Abstract

The abundance of CD8$^+$ T cells within the tumor microenvironment (TME) is a critical determinant of immunotherapy response. In this study, we demonstrated that STING- or TOX-deficient CD8$^+$ T cells exhibit enhanced antitumor activity, as evidenced by increased tumor infiltration and elevated production of IFN-γ and granzyme B. Transcriptomic analysis revealed that these cells upregulate stem-like gene signatures and ferroptosis-inhibitory pathways, while downregulating genes associated with lipid peroxidation and ferroptosis. Concurrent activation of mitochondrial biogenesis was also observed. Mechanistically, STING and TOX form a positive feedback loop that suppresses HO-1 expression in tumor-infiltrating CD8$^+$ T cells, leading to ferrous ion overload, mitochondrial ROS accumulation, lipid peroxidation, and ultimately ferroptosis. Lactate was identified as a key microenvironmental factor activating the STING/TOX signaling axis in CD8$^+$ T cells. In TC1 tumor-bearing mice, the combination of STING/TOX-deficient CD8$^+$ T cells with TIM-3/PD-1 blockade, STING agonists, or cisplatin therapy resulted in significantly enhanced tumor clearance. Clinically, high levels of TOX and HO-1 expression in intratumoral T cells were correlated with poor outcomes in cervical cancer patients. Together, these findings define the STING–TOX–HO-1 axis as a central regulator of CD8$^+$ T cell ferroptosis in the TME and suggest promising combinatorial immunotherapeutic strategies, including the adoptive transfer of STING/TOX-engineered T cells, to overcome resistance and improve treatment efficacy.

Biography

Prof. Jiang Li is a Professor and Doctoral Supervisor at the Biotherapy Research Center, Cancer Center of Sun Yat-sen University. She received her medical degree from Chongqing University of Medicine in 1992 and her Ph.D. from Sun Yat-sen University in 2002. She subsequently conducted postdoctoral research at the Karolinska Institute in Sweden, focusing on microorganisms and cancer therapy, and at Baylor University in the U.S., where she worked in the Cell and Gene Therapy Center. Prof. Li has led six major research projects funded by the National Natural Science Foundation of China as well as provincial and ministerial programs. She has also played a key role in several national R&D initiatives. Her work has been published in high-impact SCI journals, including JCI, JAMA Network Open, Cell Death & Differentiation, and JITC, as first or corresponding author. Additionally, she holds memberships in several academic societies dedicated to tumor biotherapy, tumor exosome research, and cancer prevention.

Hsueh-Chun Wang

National Cheng Kung University Tainan 70101, Taiwan

YAP nuclear translocation induced by HIF-1α under hypoxic conditions in 3D model

Abstract

Background: Hypoxia is commonly characterized by malignant tumors that promote the aggressiveness and metastatic potential of cancer. Triple-negative breast cancer (TNBC) is the most aggressive subtype of breast cancer, with approximately 46% capacity related to distant metastasis. Transcriptional factor yes-associated protein (YAP), a core component of the Hippo pathway, is associated with poor prognosis and outcome in cancer metastasis. We previously demonstrated YAP is novel factor and positively responsible for hypoxia-promoting mesenchymal TNBC cell migration in 2D model. However, YAP activation in TNBC cells under hypoxia conditions in 3D model has not been investigated. In this study, we demonstrated that YAP promoted mesenchymal TNBC cell migration under hypoxia conditions in 3D cell culture.

Methods: We characterized the effect of hypoxia on YAP in different breast cancer cell lines using a hypoxia chamber and CoCl2 in 3D model. Cell spheroids will use light-sheet microscope for imaging and 3D image construction will be processed by the Imaris software to further evaluate the effect of hypoxia-mediated YAP activation n mesenchymal TNBC cell migration.

Results: We have established spheroids generated by forced aggregation of the cells in micro-U- wells with uniform size (Fig.1). Under clearing process, cell spheroids will be evaluated by light- sheet microscopy and Imaris image analysis (Fig.2). However, our findings provide further evidence and outcomes to help prevent the progression of TNBC using this strategy and further achieve the goals of 3R in replacement and reduction of animal experiments.

Biography

Hsueh-Chun Wang finished her PhD at 37 years old years and postdoctoral investigations from National Cheng Kung University in Taiwan. She is the manager of bioimage core, a head optical iamge facility in Tainan, Taiwan. She has Published in excess of 10 papers in rumored diaries.

Julie Black

Dominican University of New York, USA

Development of breast cancer survivorship program within a community-based hospital in New York

Abstract

As breast cancer survivorship continues to rise due to advances in early detection and treatment, there is a growing need for comprehensive, community-based survivorship care. This Doctor of Nursing Practice (DNP) capstone project proposes the establishment of a formalized breast cancer survivorship program within a community hospital in the Greater New York area. Guided by the Corbin-Strauss Chronic Illness Trajectory Model, as its theoretical framework, the project developed a protocol to establish a structured breast cancer survivorship program tailored to the needs of post-treatment patients.

The initiative included a detailed literature review,and stakeholder engagement to assess feasibility and support. The proposed program features individualized survivorship care plans (SCPs), multidisciplinary collaboration with ancillary services, coordination with primary care providers, and integration of community resources. Key components include symptom management, lifestyle counseling, family support services, and education on recurrence surveillance.

A patient survey conducted to assess interest and feasibility revealed strong support for the program, validating the demand for structured survivorship care. The implementation protocol outlines team roles, care workflows, educational materials, and metrics for evaluation. The program aims to improve clinical outcomes, reduce recurrence risk, and enhance patient quality of life.

By enhancing care continuity and addressing physical, emotional, and socioeconomic

challenges, this program aims to improve patient outcomes and quality of life. The model aligns with national guidelines and DNP competencies, offering an accessible solution for other community-based cancer centers.

Biography

Julie Black, FNP-C, is a dedicated Family Nurse Practitioner with extensive experience in both hospitalist and emergency/urgent care settings. Fluent in English and Creole, she brings over a decade of clinical expertise from respected institutions including, CrystalRun Healthcare, Montefiore St. Luke's, The Valley Hospital, and Good Samaritan Hospital. Julie is recognized for her calm, organized approach to high-acuity situations, strong collaboration skills, and compassionate patient care. She holds a Master of Science in Nursing from Dominican College, where she also completed her BSN, and currently working on her Doctorate. Julie is certified by the AANP and licensed in both New York and New Jersey. Beyond the bedside, she served over a decade as an EMT with Spring Hill Ambulance Corps, where she held several leadership roles. Julie remains committed to advancing healthcare access and safety for diverse populations across the Hudson Valley and beyond.

Karl Heinz Leonhardt Rowika

Universitas Indonesia, – Cipto Mangunkusumo Hospital, Indonesia

The Role of Vimentin in the Survival of Triple Negative Breast Cancer Patients : A Retrospective Cohort Study

Abstract

Triple negative breast cancer (TNBC) is a subtype of breast cancer with the poorest prognosis and aggressive biological behavior. In Indonesia, approximately 15–25% of breast cancer cases fall into the TNBC category. One of the biological markers involved in the epithelial–mesenchymal transition (EMT) process and tumor progression of TNBC is vimentin. Positive vimentin expression has been reported to correlate with reduced survival; however, data in the Indonesian population remain limited.

This retrospective study included 29 TNBC patients who received treatment at Dr. Cipto Mangunkusumo National General Hospital between 2017 and 2022, with a three-year follow-up period. Clinicopathological data and vimentin expression were analyzed using bivariate analysis and Kaplan–Meier survival analysis.

Most patients were aged ≥40 years (82.8%), had a normal body mass index (82.8%), presented with locally advanced disease (72.4%), high histopathological grade (62.1%), and high Ki-67 expression (72.4%). A significant association was observed between vimentin expression and overall survival (p=0.019), where increased vimentin expression was associated with reduced survival (mean survival 30.42 months in the negative group vs. 20.86 months in the positive group). Vimentin expression was also significantly associated with Ki-67 expression (p=0.045), indicating a link between cellular proliferation and activation of EMT. Further analysis in patients with TNBC demonstrated that positive vimentin expression significantly increased the risk of mortality (p=0.015; HR 5.64; 95% CI 1.39–22.7), while inadequate treatment was also associated with poorer overall survival (p=0.011; HR 4.05; 95% CI 1.37–11.9).

Vimentin expression is significantly associated with reduced overall survival in patients with TNBC and correlates with high Ki-67 expression, reflecting tumor aggressiveness and activation of EMT. Inadequate treatment also contributes to poorer survival outcomes. These findings highlight the potential role of vimentin as a prognostic molecular marker in TNBC within the Indonesian population.

Biography

The writer is a chief resident in general surgery at Universitas Indonesia, enrolled at Cipto Mangunkusumo Hospital, Jakarta, Indonesia. The writer collaborates with the head of the general surgery residential program, a surgical oncology consultant, and a consultant from the clinical pathology department, subspecialized in reproductive tissue. We have a strong interest in the oncology field, especially in the outcomes we have described in our research.

Mahtab Azhdar
University of Alberta, Canada

Exploring the Impact of Taxane-Based Chemotherapy on the Physical Function of Breast Cancer Patients Using Markerless Motion Capture

Abstract

Breast cancer survivors often experience declines in physical function during and after chemotherapy, particularly with taxane-based regimens known to cause neuropathy, fatigue, and reduced endurance. Monitoring these changes is critical for timely rehabilitation planning, yet conventional clinical measures may lack sensitivity to subtle impairments. Markerless motion capture (MMC) technology offers an innovative, non-invasive approach to quantify body functions such as balance, and sit-to-stand performance.

This study will longitudinally evaluate the effects of taxane-based chemotherapy on the physical function of women with breast cancer stage I–III using clinic-based 3D MMC and home-based 2D MMC systems. A single-group longitudinal cohort (n=32) will be assessed at baseline, mid- treatment, end-of-treatment, and at 1.5 and 3 months post-treatment. Objective measures will include the Short Physical Performance Battery and 1-Minute Sit-to-Stand Test, and motion capture-derived metrics of sway, gait, and sit-to-stand performance. Patient-reported outcomes will be collected using the Functional Assessment of Cancer Therapy/Gynecologic Oncology Group- Neurotoxicity and the Edmonton Symptom Assessment System. Data will be analyzed using linear mixed-effects models to evaluate longitudinal changes in body functions over time. In addition, correlations and regression analyses will be conducted to examine associations between objective MMC-derived metrics and patient-reported outcomes.

This protocol is designed to highlight the potential of MMC to capture treatment-related functional decline and recovery in breast cancer patients, while also serving as a bridge between lab-based technologies and practical clinical assessment tools. Findings from this work are expected to inform survivorship care planning and support the integration of advanced technologies into oncology rehabilitation

Biography

Mahtab Azhdar is a PhD candidate in Rehabilitation Science at the University of Alberta whose work integrates markerless motion capture (MMC) into oncology and rehabilitation practice. An internationally trained occupational therapist with four years of clinical experience, she completed an eight-month internship in Canada successfully implementing MMC workflows in a physical therapy clinic, translating lab-based methods into practical assessment pathways. Her research focuses on bridging engineering innovations with clinician-friendly tools to quantify functional performance and, ultimately, to improve quality of life for clients. She has authored three publications on balance and physical function, secured competitive grants and scholarships, and has been recognized as the top-ranked graduate with the highest GPA in both her bachelor's and master's programs. Through her doctoral studies, Mahtab collaborates to advance the bench-to- bedside translation of advanced technologies.

Kunihiko Ishitani

President, SCPSC; President, Higashi Sapporo Hospital, Japan

Toward a New Era in Palliative Care in Cancer

Abstract

Over the past few years, I have spoken at various forums about how both research and practice in palliative care have been approaching a turning point. One contributing factor is the emergence of facilities and researchers dedicated to disease-specific palliative care—such as for cancer, intractable neurological diseases, chronic heart failure, and, more recently, COVID-19—as well as their pursuit of original scientific research. Another factor is the presence of institutions and healthcare professionals who intentionally provide non-disease-specific palliative care for all illnesses, tailored to the circumstances of each country or region and aligned with the philosophy of the institution. This latter approach holds significant implications for both global health and healthcare systems.

The 5th Sapporo Conference for Palliative and Supportive Care in Cancer (SCPSC), which we sponsor, has aimed to deliver innovative programs in palliative oncology and psycho-oncology, informed by the current state of each field. Furthermore, as anticipated, we have successfully attracted pioneering researchers from around the world. In other words, we have moved beyond the turning point and entered a new era of research and practice in palliative care. The 5th SCPSC, scheduled for July 2026, will be held with the support of BMJ Supportive & Palliative Care as its official journal. Held every three years, the SCPSC provides a unique forum where global experts in cancer palliative care engage in passionate, non-competitive discussions on the latest topics. The conference has even been affectionately dubbed "the Olympics of Cancer Palliative Care."

Among the many topics addressed at the upcoming conference, the intersection between cancer immunotherapy and palliative care is of particular interest. One of the symposia, entitled “The Era of Personalized Palliative and Supportive Care for Patients with Cancer: Progress and Innovation,” will include discussions on recent advances in immunotherapy. It is increasingly important to approach this field not only from the standpoint of clinical oncology but also from that of cancer palliative care.

Philosophically speaking, palliative care aligns closely with the functions of the immune system. It addresses the universal human experience of birth, aging, illness, and death—the four sufferings of Buddhist philosophy—and strives to uphold human dignity. The immune system, as a biological defense mechanism, eliminates non-self-elements; however, cancer cells exploit immunosuppressive mechanisms to evade immune detection and proliferate as if part of the self. Even when immunotherapy proves effective, the immune system remains an active participant throughout the patient’s journey and plays a vital role in preserving dignity in life and death.

Mitchell Boshkos
Baylor College of Medicine, USA

Adult Lymphoma-Associated Hemophagocytic Lymphohistiocytosis: A 24-Year Single-Center Retrospective Analysis of Clinical Features, Diagnostic Hurdles, and Treatment Outcomes

Abstract

Lymphomas can often trigger immune dysregulation, leading to hemophagocytic lymphohistiocytosis (HLH), a systemic disorder characterized by excessive inflammatory cytokine production and widespread tissue damage. The co-occurrence of lymphoma, various infections (especially EBV), and HLH presents significant challenges due to overlapping clinical features and complex treatment decisions. Currently, no established treatment guidelines exist for lymphoma-associated HLH (LA-HLH).

This study aimed to investigate the clinical characteristics, diagnostic associations/difficulties, and treatment outcomes in adult lymphoma patients with concurrent HLH. We conducted a retrospective study identifying adult patients diagnosed with both lymphoma and HLH between January 2000 and January 2024.

Of 78 total HLH cases identified over the 24-year period, 39.7% were malignancy-associated, 25.6% linked to infections, and 20.5% associated with autoimmune diseases. Among malignancy-associated cases, 20 patients (16 males, 4 females; median age 45.5 years) had LA-HLH, with the majority being Hispanic (75%). The most common lymphoma subtype was Hodgkin lymphoma (60%), followed by T-cell lymphoma (20%) (including cutaneous, peripheral, NK-/T-cell), diffuse large B-cell lymphoma (10%), marginal zone lymphoma (10%), and EBV-positive CNS lymphoma

(5%). Additionally, 30% of patients were HIV-positive, with 66.7% receiving antiretroviral therapy.

Regarding patients with HLH linked to infection (26% of total HLH cases), we sought to determine if active EBV infection or the malignancy itself drove the hyperinflammatory response. While EBV-HLH is well-described, only 5 of 19 patients with infectious processes and concomitant HLH had detectable EBV viral loads at HLH diagnosis. Five additional patients lacked EBV viral load measurements, and 9 had undetectable viral loads. Notably, all 19 patients had positive IgG against EBV, consistent with widespread prior infection.

Median IL2R level was 26,915.5 pg/mL (IQR: 9,734.5–39,871). Hemophagocytosis was observed in 47.4% of bone marrow samples (median HLH score 253). Elevated EBV levels were seen in 72.2% of patients. Clinical features included hepatomegaly (94.4%) and splenomegaly (60%). Median laboratory values were: max temperature 101.4°F, hemoglobin 6.25 g/dL, platelet count 18.5 × 10^3/µL, ANC 0.45 × 10^3/µL. Ferritin was elevated (median 7,500 ng/mL), LDH 743 U/L. Other notable findings: median triglycerides 307 mg/dL, fibrinogen 257 mg/dL, AST 439.5 U/L, median INR 1.7. Furthermore, 55% of patients experienced acute liver failure.

For treatment, 40% received AVD/ABVD chemotherapy, 20% ICE, 26.7% R-CEOP/R-CHOP/DA-CHOP, and 6.7% SMILE. The median number of chemotherapy cycles was 3. HLH-directed therapies included steroids (90%) and etoposide (70%), with 50% of etoposide recipients requiring dose reductions. Concurrent HLH and lymphoma therapies were administered to 72.2% of patients.

Regarding outcomes, 45% of patients died, 45% achieved clinical response (CR), and 10% had refractory LA-HLH entering hospice. Notably, 71.4% of CR patients received concurrent etoposide/steroids and lymphoma treatment. Among HIV-positive patients, 33.3% achieved CR, and 25% of those with EBV infections also achieved CR. For patients with liver failure, 18.2% achieved CR; both had Stage 4 Hodgkin lymphoma and received etoposide with ICE and AVD. This study provides a comprehensive 24-year analysis of LA-HLH. Our findings suggest that the lymphomatous malignancy itself, rather than active EBV infection, was the primary driver of HLH, based on temporal symptom onset and undetectable viral loads in most patients. The study further highlights the complexity of managing g HLH in lymphoma, especially involving T-cell lymphomas, viral infections, and liver failure.

Hoang Minh Quan Pham
Taipei Medical University, Taipei 11031, Taiwan

Clinical Relevance of BCL2A1-High CD8$^+$ T Cells in Predicting Outcomes of Immune Checkpoint Blockade in Lung Adenocarcinoma

Abstract

Background:
Despite advances in immune checkpoint blockade (ICB), reliable biomarkers to guide treatment decisions in lung adenocarcinoma (LUAD) remain limited. Immune cell–specific transcriptional features may provide clinically relevant information beyond tumor-centered markers. This study evaluated the association between BCL2A1 expression in CD8$^+$ T cells and clinical outcomes following ICB.

Methods:
We performed an integrative analysis of publicly available bulk and single-cell RNA sequencing datasets, including one discovery cohort (n = 60) and five independent validation cohorts (n = 126). Analyses included cell-type–resolved expression profiling, survival association analyses in ICB-treated and non-ICB cohorts, and inference-based cell–cell communication modeling. A composite biomarker model was constructed and evaluated across platforms.

Results:
Single-cell analyses showed that BCL2A1 expression was enriched in tissue-resident memory and proliferating CD8$^+$ T-cell subsets, which appeared more prevalent in ICB responders. Inferred cell–cell communication suggested that BCL2A1high CD8$^+$ T cells exhibit increased outgoing signaling activity, particularly involving the macrophage

migration inhibitory factor (MIF) pathway ($p = 0.0278$). In bulk transcriptomic cohorts, higher BCL2A1 expression was associated with improved survival in ICB-treated patients ($HR = 0.43$, $p < 0.05$), while no survival association was observed in non-ICB cohorts. A tri-marker model integrating BCL2A1, programmed death-ligand 1 (PD-L1), and a 27-gene immune-hot score demonstrated consistent predictive performance across cohorts (discovery AUC = 0.826; validation macro-AUC = 0.774) and high cross-platform reproducibility ($\rho = 0.982$–0.993).

Conclusions:

BCL2A1 identifies a $CD8^+$ T-cell subset associated with favorable survival outcomes in LUAD patients receiving ICB. Incorporation of immune cell–specific markers such as BCL2A1 into composite models may support more informed patient stratification in clinical oncology practice, warranting further prospective evaluation

Biography

Quan Pham is a third-year PhD student at Taipei Medical University and a clinician involved in the treatment of lung cancer patients in Vietnam. His research interests lie at the interface of cancer immunology and translational data science, with an emphasis on integrating bulk and single-cell transcriptomic data to identify immune biomarkers associated with immune checkpoint blockade outcomes. By combining clinical experience with computational analysis, he seeks to develop clinically meaningful biomarker frameworks to support decision-making in cancer immunotherapy.

Mufakir Qamar Ansari
The University of Toledo, Toledo, USA

High-Sensitivity Detection of Invasive Ductal Carcinoma via Domain-Specific SimCLR Pre-Training

Abstract

Automated detection of Invasive Ductal Carcinoma (IDC) in digital histopathology images remains challenging due to the domain gap between natural image pre-training and tissue-specific features, as well as significant class imbalance in patch-level datasets. We investigate domain-specific self-supervised learning (SSL) using the SimCLR contrastive framework to pre-train a ResNet50 encoder on 277,524 unlabeled 50 × 50-pixel IDC patches from 162 patients, employing a rigorous patient-stratified split (90% train, 5% validation, 5% test) to prevent data leakage. Following pre-training, we fine-tuned the encoder for binary classification with weighted cross-entropy loss to address the 71.6%/28.4% class imbalance. The SimCLR-pretrained model achieved an AUC-ROC of 0.950 and AUC-PR of 0.886 on the held-out test set, improvements of +0.032 and +0.044 over ImageNet transfer learning, while maintaining high sensitivity (Recall 0.932) and a low false-negative rate (47/13,876 patches). t-SNE and UMAP visualizations demonstrate superior class separation in SSL-derived embeddings, and Grad-CAM heatmaps confirm focus on histologically relevant features. These findings underscore the methodological rigor of large-scale, stratified evaluation and highlight the efficacy of domain-specific SSL for robust, interpretable computational pathology.

Biography

Mufakir Qamar Ansari finished his MS at The University of Toledo in computer science and engineering.

Omar TOURE
Hospital Principal de Dakar, Senegal

Biliary Tract Cancer at the Principal Hospital of Dakar: Epidemiological, Clinical, Morphological, and Histological Aspects

Abstract

Introduction: Cholangiocarcinomas represent the second primary malignant liver tumor after Hepatocellular carcinoma. Epidemiological studies have shown an increase in its incidence. Histological confirmation is sometimes difficult because of their location. It is a tumor whose diagnosis is often delayed and whose prognosis is poor. The aim of our study was to analyze the epidemiological, clinical, morphological, and histological characteristics of patients with cholangiocarcinoma and to determine the associated risk factors.

Patients and Methods: This is a retrospective study including all patients treated in the gastroenterology department for cholangiocarcinoma between January 2023 and December 2024.

Results: Twenty-seven patients were included with an average age of 60.25 years (range 27–93 years) and a male-to-female ratio of 2:1. The average duration of symptoms was 3 months (0.5–8 months). Jaundice was the most common presenting symptom (70.37%), followed by pruritus and general health deterioration (62.9% and 62.96% respectively), and abdominal pain in 55.6% of cases. One patient consumed alcohol (3.7%), and four were smokers (14.8%). Hypertension was found in six patients (22.27%), Hepatitis B in six patients (22.2%), and diabetes in 11.7%. Five patients were overweight, and one patient had Crohn's disease. Three patients had a family history of chronic viral hepatitis B (11.1%), and one patient reported a

maternal history of Stomach cancer. Physical examination showed hepatomegaly in six patients (22.2%). Abdominal ultrasound was performed in 37% of patients, Computed Tomography (CT) in 100%, Magnetic Resonance Imaging (MRI) in 33.3%, and Endoscopic Retrograde Cholangiopancreatography in 22.2% of patients. Imaging classified these cholangiocarcinomas as intrahepatic (33.33%), hilar (37.03%), distal (11.1%), Ampulla of Vater adenocarcinoma (7.4%), and Gallbladder cancer (11.11%). Dilation of the intrahepatic bile ducts was observed in 62.96% of patients. Vascular invasion was present in 14.5% of patients and metastases in 63%, including lymph node metastases (57%), liver metastases (22.22%), and lung metastases (7.4%). Peritoneal carcinomatosis was observed in 11.11% of patients. Histology was obtained in fourteen patients, and the type identified was adenocarcinoma. None of the patients underwent genetic analysis of their tumor.

Treatment was mainly symptomatic for most patients. Only five patients were referred for surgery, six received palliative chemotherapy, and six underwent endoscopic biliary drainage. Eleven patients died with an average survival of 4.4 months, and the remaining sixteen patients were lost to follow-up.

Conclusion: Cholangiocarcinomas have a poor prognosis. Only early diagnosis allowing detection of the tumor at a localized stage can improve prognosis and survival. It is necessary to develop molecular biology approaches to detect mutations, especially in cases of cholangiocarcinoma in young patients.

Patrycja Nowak-Sliwinska
University of Geneva, Switzerland

Reprogramming the Tumor Microenvironment: Novel Combination Strategies to Overcome Immune Exclusion

Abstract

The immunosuppressive and immune-excluded tumor microenvironment (TME) of microsatellite-stable (MSS) colorectal cancer (CRC) is a fundamental barrier to immunotherapy efficacy. Here, we directly targeted this niche using optimized drug combinations (ODCs) rationally designed via the Therapeutically Guided Multidrug Optimization platform. We systematically evaluated four low-dose ODCs in murine AKP organoids, complex 3D co-cultures with endothelial and immune components, and immunocompetent syngeneic models.

The lead ODC containing regorafenib, vemurafenib, erlotinib, and selumetinib profoundly remodeled the TME. In co-cultures, it induced a pro-inflammatory endothelial state, marked by significant upregulation of ICAM-1, VCAM-1, and E-selectin. In vivo, ODC suppressed tumor growth comparably to oxaliplatin but with a superior safety profile. Critically, while both agents reduced cancer cell proliferation, immunofluorescence revealed a distinct mechanism: ODC uniquely promoted vascular normalization and fostered an immune-permissive TME, facilitating the recruitment and perivascular positioning of cytotoxic CD8+ T cells. In contrast, oxaliplatin exhibited broader immunosuppressive effects. This TME reprogramming was underpinned by synergistic inhibition of overlapping oncogenic and survival pathways within stromal and tumor compartments, as predicted by computational modeling.

Our findings demonstrate that a rational, low-dose multidrug strategy can directly dismantle the immune-excluded architecture of the MSS CRC TME, offering a compelling combinatorial approach to unlock immunotherapy responses

Biography

Patrycja completed her PhD from Jagiellonian University and the Swiss Federal Institute of Technology. She conducted her postdoctoral investigations at the UMC Amsterdam (The Netherlands) and at the University Hospital in Lausanne (Switzerland). She was awarded aprestigious Marie Curie Intra-European Fellowship for Career Development and the ERC Starting grant. She is associete professor and vice-president of the School of Pharmaceutical Sciences at the University of Geneva, Switzerland. She published over 100 scientific publications in high-impact journals and co-authored 4 international patents.

Nilesh Makwana
Jawaharlal Nehru University, New Delhi, India

Phytochemical-Mediated Synthesis of Silver Nanoparticles Using Tridax procumbens and Their Therapeutic Efficacy Against Lung and Skin Cancer Cell Lines

Abstract

The development of targeted and biocompatible nanotherapeutics is critical to overcoming the systemic toxicity associated with conventional chemotherapy. This study explores the biogenic synthesis of silver nanoparticles (TNPs) using Tridax procumbens leaf extract and evaluates their specific anti-cancer potential. TNPs were synthesized via an eco-friendly green route and characterized as stable, spherical particles with an average size of 10 nm. HR LC-MS analysis revealed a complex corona of 38 phytochemicals, including geijerone and brassilexin, which contribute to the particles' biological activity.

The cytotoxic effects of TNPs were evaluated against A549 (human lung cancer), B16F10 (mouse melanoma), and A431 (human epidermal melanoma) cell lines. MTT assays demonstrated significant dose-dependent inhibition of cell viability, with IC50 values as low as 4 μg/ml in skin cancer models. These results were further validated through clonogenic and wound-healing assays, which confirmed that TNPs significantly impair the proliferative and migratory capacity of cancer cells. Confocal microscopy imaging verified the successful internalization of TNPs and their subsequent accumulation within the cell nucleus.

To understand the molecular mechanisms driving this cytotoxicity, high-throughput transcriptomic and proteomic analyses were conducted. The findings revealed that TNPs induce cell death by modulating key signaling pathways, including the mTOR pathway, autophagy, and mineral absorption. Specifically, TNPs were found to

trigger apoptosis and oxidative stress within the tumor microenvironment. These results suggest that T. procumbens-mediated TNPs serve as a potent, multi-targeted nanotherapeutic candidate for the treatment of lung and skin cancers.

Biography

Dr. Nilesh Makwana is a recently awarded Ph.D. graduate from the School of Life Sciences at Jawaharlal Nehru University (JNU), New Delhi. His doctoral research specialized in the green synthesis of silver nanoparticles and their applications in cancer therapeutics and molecular oncology. He possesses expertise in high-throughput transcriptomics, proteomics, and advanced in vitro bioassays, including confocal microscopy and flow cytometry. Throughout his doctoral tenure, his work was supported by prominent funding agencies such as DST-SEED and SERB. He was also the recipient of the International Travel Award from the Science and Engineering Research Board (SERB) to present his findings at the 14th International Nanomedicine Conference at UNSW, Sydney, Australia. He has contributed significantly to the field of nanomedicine with multiple publications in PubMed-indexed journals, focusing on the intersection of nanotechnology and cancer biology.

Mireille Catherine KADJA

School of Veterinary Medicine (EISMV), Senegal

Seroprevalence and risk factors associated with influenza D virus infection in cattle in peri-urban areas of Dakar and Thiès, Senegal

Abstract

Influenza D virus infections generally cause mild disease in cattle, but can also affect various species such as sheep, goats, pigs, camelids, poultry, and humans. Since its discovery, the influenza D virus has been circulating in several regions of the world and is increasingly recognized for its pathogenic role in respiratory diseases in cattle, especially in Europe and the USA. In Africa, very few studies have been conducted on the virus and the prevalences observed are relatively low compared to other continents. The lack of studies on the virus in Senegal, combined with the high mutation rate of influenza viruses, motivated the present study. Thus, 168 bovine sera were collected from 18 farms in the peri-urban areas of Dakar and Thiès using simple random sampling. The hemagglutination inhibition test was used for laboratory analyses with the viral strain D/bovine/France/59 as the antigen. The analyses were performed with R Studio, including chi-square or Fisher's exact tests and logistic regressions. Serological status was the dependent variable. Variables with a p-value ≤ 0.20 in univariate analysis were included in multivariate analysis. The significance threshold was 0.05 for all analyses.

The results revealed an overall prevalence of 31.5%, broken down as follows: 32.2% in the Dakar region and 30.8% in the Thiès region, with a herd prevalence of 94.4%. The highest prevalence rates were observed in large herds (>50 cattle) (41.1%), as well as in local, exotic, and crossbred animals born in Senegal (53.3%). Herd size, animal origin, and breed were identified as potential factors associated with influenza D virus infection

Biography

finished his PhD at 25 years old years from Andhra University and postdoctoral investigations from Stanford University School of Medicine. He is the chief of XXXX, a head Bio-Soft administration association. He has Published in excess of 25 papers in rumored diaries and has been filling in as a publication board individual from notoriety.

Poornima Verma

Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS), Lucknow, India

Integrative Systems Profiling Reveals a Circulating Stemness–Exhaustion Axis Driving Metastatic Prostate Cancer

Abstract

Metastatic prostate cancer (mPCa) progression is driven by the interplay between cancer stem-like cells (CSCs) and T-cell exhaustion, promoting immune evasion and metastatic adaptation. This study delineates a circulating stemness–exhaustion axis through integrative computational and clinical approaches.

Transcriptomic datasets from GEO (GSE21034, GSE70768) and TCGA-PRAD were analyzed to identify stemness (SOX2, OCT4, NANOG, ALDH1A1) and epithelial–mesenchymal transition (EMT; SNAI1, ZEB1, TWIST1) programs co-enriched with PDCD1/CD274 signatures in metastatic disease. Peripheral blood from mPCa patients ($n = 28$) and healthy controls ($n = 20$) was analyzed for EpCAM^{+}/CD45^{-} circulating tumor cells (CTCs) and CSCs (CD44^{+}/CD133^{+}) by flow cytometry. PBMCs were assessed for PD-1 expression in CD3^{+}CD4^{+} lymphocytes, and serum cfRNA was quantified for validation via RT-qPCR.

CTCs were detected in 82% of mPCa versus 10% of controls ($p < 0.001$), with 6.8-fold enrichment of CD44^{+}/CD133^{+} CSCs. PD-1^{+} CD4^{+} T cells were expanded in mPCa (18.4% vs 6.5%, $p = 0.002$), correlating with CTC burden ($\rho = 0.46$, $p = 0.01$). cfRNA confirmed 2.8–5.1× upregulation of stemness and 2.2–3.7× elevation of EMT transcripts ($q < 0.05$). The composite biomarker panel (CSC%, PD-1^{+} CD4^{+}%, SOX2/ZEB1 cfRNA) discriminated mPCa with AUC = 0.84 (95% CI 0.74–0.94).

Integrative systems profiling thus reveals a circulating stemness–exhaustion axis

defining the metastatic immune landscape in prostate cancer and highlights non-invasive biomarkers for CSC–immune–targeted therapy design.

Biography

Dr. Poornima Verma is a translational cancer biologist at the Department of Urology and Renal Transplantation, SGPGIMS, Lucknow, India. Her research integrates molecular oncology, bioinformatics, and immunogenomics to elucidate tumor progression mechanisms and identify non-invasive biomarkers. She has contributed to several studies on cancer stem cells, immune modulation, and circulating RNA-based diagnostics in urogenital malignancies. Her interdisciplinary expertise bridges computational discovery and clinical validation, focusing on prostate and bladder cancer metastasis, liquid biopsy development, and immune-stemness interaction. Dr. Verma has published multiple peer-reviewed articles in reputed journals, including ESMO Open, DNA and Cell Biology, and Journal of Cellular and Molecular Medicine.

Rihab MELLITI KHALIL

General Hospital of Aix en Provence, France

Immune checkpoint inhibitor–induced sarcoidosis is a rare but increasingly reported adverse event, particularly with anti-PD-1 therapies. We report a case of systemic sarcoidosis in a patient treated with pembrolizumab

Abstract

Case report

A 52-year-old woman with a past medical history of myocardial infarction, type II diabetes, hypertension, hypercholesterolemia and L5–S1 vertebral compression fracture was diagnosed in January 2024 with bilateral invasive ductal carcinomas (NST): Right breast: triple-negative tumor, grade II, Ki-67 40% and Left breast: hormone receptor-positive tumor (ER 40%, PR 100%), HER2-negative, Ki-67 30%. After discussion at the multidisciplinary tumor board, a neoadjuvant chemotherapy regimen combined with pembrolizumab was initiated and completed on June 26, 2024, achieving a complete metabolic response on PET–CT and marked tumor regression on breast MRI. At the time of the last neoadjuvant cycle, the patient developed painless, non-inflammatory subcutaneous nodules on the limbs. PET–CT revealed intense hypermetabolism of bilateral mediastinal and hilar lymph nodes, along with pulmonary lesions suspicious for sarcoidosis. Skin biopsy confirmed non-caseating epithelioid and giant cell granulomas, consistent with pembrolizumab-induced sarcoidosis. Pulmonary, cardiac and ophthalmologic evaluations were normal. The patient underwent bilateral mastectomy, and histopathology showed an RCB-I response. Pembrolizumab was continued in the adjuvant setting (9 doses) without respiratory symptoms. Cutaneous lesions improved with low-dose corticosteroid therapy, discontinued one month after the last dose of immunotherapy. Outcome: One year after discontinuing pembrolizumab, the patient

remains asymptomatic, with complete resolution of mediastino-hilar lymphadenopathy and pulmonary abnormalities, and no recurrence of cutaneous lesions.

Conclusion: PD-1 inhibitor-induced sarcoidosis, although rare, should be recognized by clinicians to avoid misdiagnosis as tumor progression. This case highlights the importance of histological confirmation and multidisciplinary management, allowing continuation of immunotherapy in paucisymptomatic forms.

Biography

Formerly Consultant Breast Surgeon at Imperial College Healthcare Trust (at Charing Cross Hospital 2006 -2014) London SW6 .Previously Consultant Breast Surgeon at Royal Marsden Hospital ,Fulham Road, London SW3.

Vasantha MN

Manipal Academy of Higher Education, Manipal, Udupi, Karnataka-576102, India

Machine Learning-Based Prediction of Nodal Status in Oral Squamous Cell Carcinoma and Development of a Prognostic Nomogram

Abstract

Objective: Inflammatory and nutritional parameters have been identified as prognostic factors in diverse malignancies. This study aimed to evaluate potential predictors of Nodal Stage (N-Stage) and Extra Nodal Extension (ENE) among preoperative patients diagnosed with oral squamous cell carcinoma (OSCC).

Methods: The retrospective data of 411 OSCC patients were charted from the Kasturba Medical College and Hospital, Manipal between 2018 and 2023. To assess statistical significance, we carried out univariate analysis of the predictors. Significant predictors—T-stage, DOI, NLR, PLR, PMR, and SII—were statistically significant, whereas Gender, Site, PMR and LMR were not significant for either N-stage. We then conducted multivariate analysis to evaluate the association between predictors and the outcome. Subsequently we compared the results of logistic regression with machine learning models. Model evaluation will be performed using ROC analysis and AUC. Overall accuracy will be calculated to determine the model's predictive performance, including class accuracy, which evaluates how correctly instances are classified by assessing sensitivity and specificity. The goodness of fit model will be assessed using the likelihood ratio test. All statistical analyses will employ two-sided tests. A nomogram will be developed using R software (version 4.4.2) with the rms package. Nomogram will be initially validated using bootstraps with 1000 resamples.

Results: The AUC for the model is 0.816, indicating good discriminatory ability between patients with N0 and N+ status. C – Index = 0.6318343. The above-mentioned statistical methods used for development of nomogram for N-status.

Conclusions: Clinicopathological, inflammation-, and nutrition-related indicators may enhance TNM-based prognosis in OSCC; Random Forest proved most reliable for clinical decision support.

Biography

Is doing 2nd year PhD scholar at 39 years from Manipal University, Manipal, Karnataka, India. I had published 6 articles among that 4 are scopus indexed and 2 are non-indexed. Planning to file patent and copy right with present work.

Tamina, ELIAS-RIZK

Lebanese American University, Lebanon

Breast cancer screening in Lebanon: Understanding knowledge, attitudes barriers. Practices during economic crisis/Covid 19 Pandemics

Abstract

Breast cancer (BC) has been increasing in both prevalence and incidence in Lebanon. Knowing the positive impact mammographic screening has on reducing mortality rates, we sought to investigate the knowledge, attitudes and barriers towards BC screening amongst Lebanese women across all districts. In addition, public health efforts towards breast cancer (BC) prevention have been largely absent from healthcare planning in modern-day Lebanon. Mammography screening campaigns have been present since 2002, but their implementation has been inconsistent in terms of pricing, locations, and the centers involved. In 2020, Lebanon was caught in the whirlwind of the Covid pandemic while facing a brewing economic crisis and a direct hit to the capital’s center of commerce. We wanted to evaluate the impact of the complex situation created by these crises on BC screening. We conducted a cross-sectional study with 400 Lebanese women aged 35–75, with no prior or current diagnosis of BC, employing an online questionnaire filled face-to-face with participants to gather sociodemographic data and assess BC history and screening practices. We utilized the Breast Cancer Screening Beliefs Questionnaire (BCSBQ) and Champion Health Belief Model Scale (CHBMS) to evaluate knowledge, attitudes, and barriers. Andwe assessed the BC screening practices of these 400 women.

Findings revealed inadequate attitudes towards general health check-ups (77.5 %) and insufficient BC screening knowledge (56.4 %). Furthermore, 38.5 % encountered obstacles to mammography screening. Education significantly affected BC knowledge.

Interestingly, increased knowledge of BC reduced barriers to mammographic screening. Participants with healthcare connections or background exhibited better attitudes towards health check-ups and encountered fewer screening obstacles. One tenth of participants halted mammography screening during the multifaceted crisis, while more than half of participants had continued or improved their BC screening practices after 2020. Women with an unfavorable attitude towards general health check-ups and single participants were more vulnerable to experience change in their BC screenings. Contrarily, women with relatives affected by BC and those financially stable to cover basic needs and more had higher proclivities to undergo BC screening. Our data highlight the crucial role of education in advocating for early BC screening and the necessity to reevaluate national campaigns, particularly in communication methods, to ensure equitable access to screening across the country. Future campaigns should nurture a culture that promotes general health check-ups, clearly advertised and communicated to the general public, especially in terms of cost and centers involved, while still offering financial support.

Biography

Chief of Breast diagnosis and interventional at LAU Medical Centers, Lebanon since 2011. Earned medical Degree (MD) at the Faculty of Medicine from Saint-Joseph University, Beirut, Lebanon. After residency training in Medical radiology at Hotel Dieu de France Beirut, pursued a specialized training in Radiology in breast and body MR imaging at Henri Mondor CHU-Creteil- Paris12, France, Pierre et Marie Curie University Paris 6 and Descartes Medical Faculty, Paris 5.

Obtained a Master in Biological and Medical Sciences Saint-Joseph University Beirut with certificates of Genetics and molecular bases with M2 in breast cancer diagnosis at Cancer and Metabolism Laboratory Saint-Joseph University.

hold many diplomas and certificates in Clinical Simulation, Healthcare Leadership and Medical Education.

Member of Lebanese and international societies of Radiology/ Breast Imaging. Have given many lectures/co-Organized symposiums in breast cancer.

Involved in many research projects related to Breast Cancer.

CAPDEVILLE Manon
PASTEUR INSTITUTE, PARIS

Targeted protein degradation for cancer therapy

Abstract

Mycolactone, a toxin secreted by Mycobacterium ulcerans, inhibits the Sec61 translocon, a channel responsible for importing most secreted and transmembrane proteins into the endoplasmic reticulum. Based on this mechanism, we hypothesized that Sec61 inhibition could represent a novel strategy to eliminate secretory cancer cells such as multiple myeloma (MM) cells. MM is a plasma cell malignancy characterized by excessive immunoglobulin secretion. Blocking Sec61 leads to intracellular protein accumulation, triggering cellular stress and ultimately cell death.

Our study evaluates the anti-MM potential of novel synthetic Sec61 inhibitors. We assessed their cytotoxic effects on MM and healthy cells to determine their therapeutic window. For the most promising candidates, we investigated the mechanisms underlying their toxicity and analyzed their impact on the expression of key survival proteins in MM cells. Proteomic analyses were also performed to examine their global effects on MM cellular pathways.

Our findings demonstrate that synthetic Sec61 inhibitors selectively induce MM cell death, reduce immunoglobulin production, and decrease transmembrane protein expression. Interestingly, these inhibitors increase the expression of a therapeutic antigen, enhancing the effectiveness of immunotherapies targeting this protein. Combination treatments with immunotherapies and proteasome inhibitors further improved MM cell killing.

Collectively, these results identify Sec61 inhibitors as promising therapeutic agents for multiple myeloma and suggest their potential application in other cancers characterized by abnormal protein secretion.

Biography

3rd year PhD Student Pasteur Institute Paris

Ali Mohammadi
Aarhus University, Aarhus, Denmark

Targeted inhibition of NBCn1 (SLC4A7) by antibodies prevents net acid extrusion, causing reduced proliferation and apoptosis in breast cancer in a pH- dependent manner

Abstract

Na+,HCO3–-cotransporter NBCn1/Slc4a7 accelerates murine breast carcinogenesis. Lack of specific pharmacological tools previously restricted therapeutic targeting of NBCn1 and identification of NBCn1-dependent functions in human breast cancer.
We develop extracellularly-targeted anti-NBCn1 antibodies, screen for functional activity on cells, and evaluate (a) mechanisms of intracellular pH regulation in human primary breast carcinomas, (b) proliferation, cell death, and tumor growth consequences of NBCn1 in triple-negative breast cancer, and (c) association of NBCn1-mediated Na+,HCO3–-cotransport with human breast cancer metastasis.

We identify high-affinity (KD ≈ 0.14 nM) anti-NBCn1 antibodies that block human NBCn1- mediated Na+,HCO3–-cotransport in cells, without cross-reactivity towards human NBCe1 or murine NBCn1. These anti-NBCn1 antibodies abolish Na+,HCO3–-cotransport activity in freshly isolated primary organoids from human breast carcinomas and lower net acid extrusion effectively in primary breast cancer tissue from patients with macrometastases in axillary lymph nodes. Inhibitory anti-NBCn1 antibodies decelerate tumor growth in vivo by ~50% in a patient-derived xenograft model of triple-negative breast cancer and pH-dependently reduce colony formation, cause G2/M-phase cell cycle accumulation, and increase apoptosis of metastatic triple-negative breast cancer cells in vitro.

Inhibitory anti-NBCn1 antibodies block net acid extrusion in human breast cancer tissue, particularly from patients with disseminated disease, and pH-dependently limit triple-negative breast cancer growth.

Biography

I earned my PhD in 2022 from the University of Southern Denmark. Since then, my research has focused on advancing our understanding of cancer, resulting in over 100 publications spanning cancer biology, immuno-oncology, and cancer genetics. Through this work, I strive to uncover insights that can lead to more effective treatments and improved patient outcomes.

Dafne Bahena Salmerón

Universidad Nacional Autónoma de México (UNAM), Mexico

Triple negative and hormone-dependent human breast cancer cells are responsive to pro-apoptotic stimule from organometallic compounds of new generation

Abstract

Triple negative breast cancer (TNBC) carries the worst prognosis and current chemotherapy are not efficient treatment, making an important target of effort in the development of new strategies to control the disease. In this study, the response to a new generation Organotin (IV) compound was evaluated in two human breast cancer cell lines, MDA-MB-231 (TNBC) and MCF-7 (hormone- dependent). The effect on the proliferative potential and the cytotoxicity of the organotin compounds 4a, 4b, and 4f was evaluated by means the violet crystal and by means the LDH technique, respectively. Additionally, the apoptotic effect was identified in both breast cancer cell lines by the active caspase-3 immunodetection. The results showed that the compounds exerted an antiproliferative effect on both cell lines, with a better antiproliferative response in the MCF-7 cell line. The necrotic effect caused was not significative, suggesting a non-necrotic effect. The immunodetection of active caspase-3, evidencing the apoptotic activation. These results evidence that organotin compounds 4a,4b, and 4f exert an antiproliferative effect, induce to a programmed cell death elimination, with low necrotic (cytotoxic) effect to MDA-MB-231 and MCF7 breast cancer cells.

Biography

Biologist, Electron Microscopy Specialist and Master´s student in biological sciences at UNAM, at 25 years old years. Currently studying the processes involved in cell death in cancer cell lines.

Eyun Song

Korea University College of Medicine

Breastfeeding is associated with cancer prevalence: A nationwide population study

Abstract

Breastfeeding is well known to reduce the risk of breast cancer. However, studies of the association between breastfeeding and other cancers are limited. Using the data from 2010–2019 Korea National Health and Nutrition Examination Survey, this study included 18,332 women aged ≥20 years with available data on breastfeeding and diagnosed cancer types. Cancer types included in the survey were gastric, colon, hepatic, breast, cervix, lung, thyroid, and others. Breastfeeding was categorized into none or less than 12 months (group A), 12–35 months (group B), and ≥36 months (group C). There were 1,082 women (5.9%) with diagnosed cancer; 199 (18.4%) in group A, 460 (42.5%) in group B, and 423 (39.1%) in group C. The distribution was significantly different ($p<0.001$) from those without cancer; 4,221 (24.5%) in group A, 7,286 (42.2%) in group B, and 5.742 (33.3%) in group C. After adjustment for multiple confounders, group C had significantly lower odds of cancer with an odds ratio (OR) of 0.74 (95% confidence interval [CI] 0.56–0.98, p=0.018) compared with group A. In specific, female cancers including breast, cervix, and thyroid cancers are least prevalent in group C with OR of 0.63 (95% CI 0.45–0.89, p=0.007) in reference to group A. Notably, gastrointestinal cancers including gastric, colon, and liver cancers showed increased odds in both group B (OR 3.85, 95% CI 1.56–9.48, p=0.003) and group C (OR 2.76, 95% CI 1.10–6.91 p=0.030). Breastfeeding is associated with a lower prevalence of diagnosed cancers in Korean women. While female cancers showed lower odds in women who breastfed longer, the odds of gastrointestinal cancers were increased. Further longitudinal studies are needed to conclude a causal relationship.

Biography

Eyun Song, MD, PhD, is an Assistant Professor in the Division of Endocrinology and Metabolism, Department of Internal Medicine at Korea University Guro Hospital in Seoul, Korea. She received her MD degree from Chung-Ang University School of Medicine and completed her MS and PhD at the University of Ulsan College of Medicine. Dr. Song completed her clinical training and served as a Clinical Instructor at Asan Medical Center, followed by appointments as Clinical Assistant Professor and Assistant Professor at Korea University Guro Hospital. Her clinical and research interests focus on diabetes mellitus, diabetic complications, and metabolic disorders. She has participated in multiple clinical trials involving type 2 diabetes, diabetic neuropathy, and dyslipidemia. Dr. Song is a board-certified physician licensed in Korea and is actively involved in patient care, clinical research, and medical education.

Hassan Dawud Jidda
Sharda University, India

The role of curcumin in apoptosis in breast cancer cell and normal cell line

Abstract

Apoptosis is a Programmed cell death that is important determination to response to chemotherapy among the factors controlling this process a significant role played by p53,caspase 3,Bcl2 and p21,The expression of which, together with estrogen receptors content and tumor proliferative activity was investigated in breast cancer cell line, the aim of my study is to evaluate how curcumin can influence the growth of normal and breast cancer cells, and also to evaluate how curcumin can regulate Apoptosis related genes expression in normal and breast cancer cells . The Curcumin inhibited cell growth in cancer cell MCF-7, however in normal cells curcumin does not affect the cell growth, does amply that it is not harmful to normal cell but its toxic to cancer cells and also it is anti-carcinogenic. Curcumin can regulate apoptosis by inducing Caspase 3, p53, p21 and Bcl2 in MCF-7 cancer cells. Does imply that curcumin specifically target breast cancer cell and not normal cells gene expression. Hence curcumin induces apoptosis in cancer cells.

Keyword: apoptosis, Curcumin, cell death, caspases

Karolina Jablonska

Wroclaw Medical University, Poland

Assessment of changes in the expression profile of PIP-dependent genes according to the molecular subtype of breast cancer in the context of chemotherapy response

Abstract

Breast cancer is one of the most commonly diagnosed cancers worldwide, with 2.3 million new cases and 670,000 deaths reported in 2022. Despite medical progress, many patients remain resistant to standard treatments. Our previous studies indicate that Prolactin-Induced Protein (PIP) may serve as a potential biomarker of chemotherapy response. High PIP expression has been shown to enhance drug-induced apoptosis by upregulating key pro-apoptotic genes and increasing tumor sensitivity to agents like doxorubicin. These findings suggest that PIP contributes to modulation of cell death pathways, reinforcing its potential relevance in predicting and improving therapeutic responses. Its variable expression across breast cancer molecular subtypes suggests distinct, subtype-specific biological functions. However, the mechanisms underlying this association are still poorly understood. In this study, we used single-cell and spatial transcriptomic techniques, including 10x Chromium Flex Gene Expression and the Xenium In Situ platform, to investigate the molecular network associated with PIP expression and treatment response. The analyses were performed on representative FFPE breast cancer samples selected based on molecular subtype, differential response to adjuvant chemotherapy, and varying levels of PIP expression. Our findings highlight distinct PIP-associated molecular signatures across breast cancer subtypes and identify gene networks that may contribute to differential therapeutic outcomes. These results provide novel insights into the biological mechanisms underlying chemotherapy response and support the further exploration of PIP-related pathways as potential predictive biomarkers in breast cancer.

Chong Wei

Peking Union Medical College, China

Germline defects of familial haemophagocytic lymphohistiocytosis–related genes may represent a predisposing factor for mature T- and natural killer-cell lymphoma

Abstract

Peripheral T-cell lymphoma (PTCL) is relatively prevalent in Asian populations. Previous studies suggest that germline mutations in familial haemophagocytic lymphohistiocytosis (FHL)-related genes may predispose individuals to lymphoproliferative disorders. To investigate the underlying molecular mechanisms, we analysed paired tumour and germline deoxyribonucleic acid from 74 patients with T- and natural killer-cell lymphomas. Germline variants in FHL-related genes (UNC13D, PRF1, STXBP2, STX11, SH2D1A and XIAP) were assessed by whole-exome sequencing, while somatic mutations were analysed by targeted sequencing. A total of 21 germline mutations in FHL-related genes were detected in 14 of 74 patients (18.9%), including mutations in UNC13D (N = 11), STXBP2 (N = 6), PRF1 (N = 3) and STX11 (N = 1). The most frequent mutation was UNC13D c.2588G>A (p.G863D), which was significantly enriched in PTCL patients compared to the general Chinese Han population (allele frequency: 4.7% vs. 0.7%, OR = 6.785, p = 0.002). In line with estab- lished PTCL mutation profiles, somatic mutations were frequently detected in TET2, RHOA, DNMT3A and IDH2. Patients with FHL-related germline mutations exhib- ited a trend towards better overall survival. In conclusion, germline mutations in FHL-related genes, particularly UNC13D, may contribute to PTCL susceptibility in Chinese patients and are associated with clonal somatic mutations.

Ita Novita Sari

Institute of Molecular and Cell Biology (IMCB, Singapore

Unraveling inflammation reprogramming roles of TNFα-NFκB signaling during fetal liver development and Hepatocellular Carcinoma progression

Abstract

The TNFα-NFκB signaling axis is a critical driver of chronic inflammation, a well-established hallmark of cancer. Pathological amplification of this pathway is frequently implicated in tumorigenesis, yet the precise mechanisms governing TNFα hyper-production remain elusive. While TNFα-NFκB signaling is essential for fetal liver development, its dysregulation can be lethal, as seen in Rela mice which die perinatally from massive, TNFα-driven liver degeneration. Conversely, the upregulation of NFκB (p65) in human hepatocellular carcinomas (HCC) often promotes resistance to apoptosis and induces an "immune-cold" tumor phenotype, characterized by a scarcity of immune cells. Here, we used Rela-deficient embryos to uncover the cellular and molecular drivers of paradoxical TNFα amplification, aiming to identify novel therapeutic targets applicable to inflammation-driven cancer. To investigate this, we performed single-cell RNA sequencing (scRNA-seq) and spatial transcriptomics on fetal livers isolated from Rela-/- and Rela-/-Tnfr1-/- mouse embryos. Our investigation highlights liver-resident macrophages (Kupffer cells) as the source of excessive TNFα production. We uncovered a pathological feedback loop initiated by the loss of Rela in hepatocytes, which triggers initial cell death and the release of Macrophage Migration Inhibitory Factor (MIF). This MIF, in turn, acts on Kupffer cells, dramatically amplifying TNFα production. This creates a vicious cycle of reciprocal signaling between dying hepatocytes and macrophages, leading to massive inflammatory cell death—a phenomenon reminiscent of the tumor microenvironment. Deletion of the

TNFα receptor (Tnfr1) completely rescued this phenotype, confirming the essentiality of this amplification loop. Together, we have identified a novel MIF-TNFα signaling axis between hepatocytes and macrophages that drives embryonic lethality in Rela-/- mice. Our findings suggest that the MIF signaling pathway is a key vulnerability in conditions of TNFα-NFκB hyperactivation. Therefore, therapeutically targeting MIF could represent a promising strategy to disrupt this inflammatory feedback loop and treat inflammation-driven malignancies, such as HCC.

Biography

*I got my PhD from Soonchunhyang Institute of Medi-Bio Science, South Korea at 2021 where I continued my postdoctoral journey for a year before I moved to IMCB, A*STAR Singapore. During PhD study, I carried out my research in the field of cancer, especially colon cancer, lung cancer, liver cancer, and leukemia. During my postdoc period in IMCB, I expand my expertise in single cells RNA-Sequencing (scRNA-Seq) for spatial transcriptomics assay to study embryonic liver development and HCC progression. I am also currently involved in the molecular identification of new components in NFκB/TNFα signaling pathway. I have published 17 papers, with h-index of 13.*

Jwa Hoon Kim
Korea University College of Medicine, Korea

The dual effect of immune checkpoint inhibitor and tyrosine kinase inhibitor on thyroid function and its impact on survival in patients with advanced renal cell carcinoma from The Cancer Clinical Library Database

Abstract

Immune checkpoint inhibitor (ICI) plus tyrosine kinase inhibitor (TKI) therapy has become a standard first-line treatment for patients with advanced renal cell carcinoma (aRCC). Both ICIs and TKIs are well-established risk factors for thyroid dysfunction; however, data regarding the incidence and clinical impact of thyroid dysfunction associated with their combined use remain limited. We evaluated the incidence, clinical characteristics, and prognostic implications of thyroid dysfunction in patients with aRCC treated with first-line ICI plus TKI. This nationwide retrospective study included 254 patients with aRCC who received first-line ICI plus TKI between 2020 and 2024, using data from the Korea–Clinical Data Utilization Network for Research Excellence, a Korean Ministry of Health and Welfare–funded common data model. Thyroid dysfunction was assessed in terms of incidence, time to onset, severity, need for thyroid hormone replacement, and association with survival outcomes. With a median follow-up of 22.8 months, thyroid dysfunction occurred in 63.8% of patients, including hyperthyroidism (6.3%), subclinical hypothyroidism (37.8%), and overt hypothyroidism (19.7%). The median time to thyroid dysfunction was 2.8 months from treatment initiation. All cases of hyperthyroidism resolved spontaneously to hypothyroidism or normal thyroid-stimulating hormone levels, consistent with thyroiditis. Overt hypothyroidism required thyroid hormone replacement, and approximately half of patients with subclinical hypothyroidism required treatment when thyroid-stimulating hormone levels showed

a progressive increase and exceeded 10 mU/L. Median progression-free survival and overall survival were 17.3 months and 49.2 months, respectively. On multivariate analysis, both overt hypothyroidism and subclinical hypothyroidism were independently associated with improved progression-free and overall survival. Thyroid dysfunction is a common treatment-related adverse event in patients with aRCC receiving first-line ICI plus TKI and is associated with favorable survival outcomes. Close monitoring of thyroid function and multidisciplinary management with endocrinology are warranted to enable early detection and appropriate thyroid hormone replacement, thereby supporting the continuation of ICI plus TKI therapy..

Biography

Jwa Hoon Kim, MD, PhD, graduated from Kyung Hee University College of Medicine in Korea and completed his PhD training at Korea University College of Medicine. He currently serves as the Chief of Medical Oncology at Korea University Anam Hospital. Dr. Kim's clinical and research interests focus on solid tumors, with particular expertise in genitourinary malignancies and immuno-oncology. He has published more than 20 peer-reviewed articles in international journals, contributing to research on treatment outcomes, biomarkers, and real-world evidence in oncology. He is actively involved in multidisciplinary cancer care, nationwide collaborative research projects, and academic activities aimed at advancing precision oncology.

Kan Kar Mern
University of Manchester, Manchester, United Kingdom

Dissecting ERK5 Functional Domains in Regulating Epithelial-to-Mesenchymal Transition (EMT) and Migration in Triple-Negative Breast Cancer (TNBC)

Abstract

Triple-negative breast cancer (TNBC) is an aggressive subtype characterized by enhanced migration, invasion, and limited therapeutic options. ERK5, a unique member of the MAP kinase family, possesses both a kinase domain and a large C-terminal transcriptional regulatory region, yet its domain-specific roles in TNBC remain poorly defined. Using CRISPR-engineered SUM159 TNBC cell lines, we generated ERK5 knockout, kinase-inactive (AEF), and C-terminal truncated (ΔC) mutants to dissect functional contributions of each domain. ERK5 knockout cells displayed the strongest reduction in migration, while both AEF and ΔC mutants exhibited intermediate defects. Quantitative PCR analysis revealed decreased EMT-associated markers, including CD44, ZEB1, and FRA1, with the ΔC mutant showing the greatest reduction. Interestingly, the AEF mutant selectively downregulated Slug and Vimentin, suggesting that ERK5 kinase activity may drive cytoskeletal remodeling and mesenchymal signaling. Despite lowered CD44 expression, all mutants retained spheroid-forming ability, indicating distinct pathways for adhesion and motility regulation. Collectively, these findings highlight domain-specific ERK5 functions in TNBC and shift the perspective from whether ERK5 is important to how its kinase and non-kinase activities can be targeted for therapeutic intervention.

Biography

*Kar Mern is a third-year PhD candidate at the University of Manchester, United Kingdom, under the A*STAR Research Attachment Programme (ARAP) with Singapore. Her research focuses on breast cancer biology, particularly understanding the signaling mechanisms that drive tumor progression and metastasis. She is interested in dissecting key molecular pathways and identifying potential therapeutic targets that could inform future treatment strategies for aggressive breast cancer subtypes.*

Ndong Mengome Christine
Independent Researcher, Cancer Biology, Gabon

In vitro and in silico evaluation of talinum fruticosum tumor cell co-culture derived molecules as predicted precision biotherapy for inflammatory breast cancer

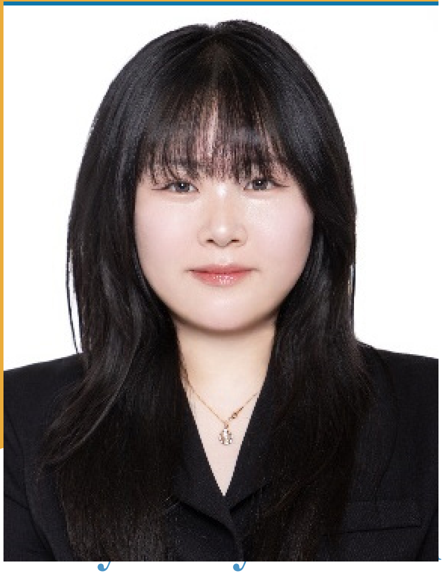
Abstract

Current cancer therapies face major limitations, including lack of specificity, drug resistance, and systemic side effects. These challenges are particularly pronounced in inflammatory breast cancer (IBC), an aggressive and highly prevalent subtype worldwide. Understanding the metabolic and molecular pathways through which therapeutic agents act is essential for developing more targeted and effective interventions. Aim: This proposed study aims to investigate the autologous molecular modulation of Talinum fruticosum (waterleaf) when co-cultured with patient- derived breast tumor cells, as part of a precision biotherapy development framework. The objective is to validate predicted drug candidates in vitro by integrating plant-based molecular modification with computational drug-target prediction. Methodology: We plan to screen fifty breast cancer related genes from samples obtained from consented participants using proteomic profiling techniques, followed by RT-qPCR to assess the diagnostic and prognostic relevance of candidate targets. T. fruticosum tissue cultures will be established in tumor-cell enriched media to enable plant tissues to acquire potential tumor-specific molecular characteristics. Subsequent analyses including genetic profiling, mass spectrometry, and in silico molecular docking will be performed to evaluate the potential interactions between plant derived compounds and key tumor-associated proteins such as HER2. Through computational modeling, we aim to identify strong mRNA-protein interaction signatures and predict binding affinities that may indicate therapeutic potential. Expected results: The anticipated outcome

is to determine whether tumor-modulated T. fruticosum extracts can yield bioactive derivatives with enhanced tumor-targeting capacity. Conclusion: To our knowledge, this will be the first study to explore T. fruticosum derived molecular conjugates in the context of human tumor biology. This work is expected to support the feasibility of combining plant-based molecular modulation with AI-guided drug design to develop novel, patient-specific therapeutic candidates for aggressive cancers such as IBC.

Biography

Christine Ndong Mengome is a biomedical scientist with a strong background in molecular biology, immunology, and bioinformatics. She studied Biotechnology and completed her Master's degree in Biology in 2024 at Catholic University of Central Africa, School of Health Sciences. Since 2019, she has been part of the Centre de Recherches Médicales de Lambaréné (CERMEL) in Gabon, where she also manages laboratory operations and archives. Christine has contributed to several research projects focused on infection biology, vaccine development, and aim to specialize in precision therapies. Significantly, she played a key role in establishing the production of Necator americanus for Africa's first controlled human hookworm infection study. Passionate about innovation and personalized medicine, Christine is committed to advancing biomedical research and improving health outcomes in sub-Saharan Africa.



Seo Lyn Choi
Sungkyunkwan University, Korea

alcone derivative induces reactive oxygen species- mediated Bax activation and apoptosis in CD133+ lung cancer organoids

Abstract

Backgrounds ung cancer remains a leading cause of cancer-related mortality worldwide primarily due to therapeutic resistance and tumor recurrence. Accumulating evidence indicates that CD133$^+$ cancer stem–like cells (CSCs) play a critical role in tumor initiation, maintenance and resistance to chemotherapy. These cells exhibit enhanced survival signaling reduced apoptotic sensitivity and increased tumorigenic potential. Reactive oxygen species (ROS)–dependent redox homeostasis is essential for CSC survival. Disruption of intracellular redox balance can sensitize CSCs to mitochondrial apoptotic signaling particularly through Bax activation. However, effective therapeutic strategies to selectively eliminate CD133$^+$ lung cancer stem–like cells remain limited. UR-2, a hydroxylated chalcone derivative, has demonstrated anticancer potential but its molecular mechanism in lung CSCs has not been fully elucidated.

Methods: CD133$^+$ and CD133$^-$ lung cancer organoids were established and characterized. Proteomic profiling was performed to identify signaling pathways affected by UR-2 treatment. Cell viability and clonogenic assays were conducted to assess growth suppression. Intracellular ROS levels and mitochondrial membrane potential were analyzed to evaluate redox and mitochondrial responses. Western blot analysis was performed to examine death receptor signaling, Bax activation, cytochrome c release and caspase-3 cleavage. Chemoresistance was evaluated using 5-fluorouracil and cisplatin treatment models. In vivo validation was conducted using xenograft

models derived from CD133$^+$ organoids..

Results: Proteomic analysis revealed that UR-2 significantly modulated apoptosis-related signaling networks involving FAS, Bax, and ROS-associated pathways. UR-2 selectively suppressed the growth and clonogenic capacity of CD133$^+$ lung cancer organoids while exerting minimal effects on CD133$^-$ counterparts. Mechanistically, UR-2 induced intracellular ROS accumulation leading to activation of death receptor signaling mitochondrial membrane depolarization, cytochrome c release, enhanced Bax activation and caspase-3–mediated apoptotic cell death. UR-2 effectively overcame chemoresistance to 5-fluorouracil and cisplatin in CD133$^+$ organoid models. Consistently in CD133$^+$ organoid-derived xenografts UR-2 treatment significantly reduced tumor growth decreased CD133 expression and shifted the BCL-2/Bax ratio toward a pro-apoptotic state..

Conclusion: Our findings demonstrate that UR-2 induces ROS-mediated Bax activation and mitochondrial apoptotic signaling in CD133$^+$ lung cancer stem–like cells. By disrupting redox homeostasis and enhancing apoptotic susceptibility, UR-2 selectively targets chemoresistant CSC populations in both organoid and in vivo models. These findings suggest that modulation of ROS–Bax–dependent apoptotic vulnerability represents a promising therapeutic strategy for overcoming lung cancer chemoresistance.

Biography

SeoLyn Choi is an integrated Master's program student in the Department of Meta-BioHealth at Sungkyunkwan University. She is interested in cancer stem-like cells (CSCs) and therapeutic resistance and is currently conducting research in this field. In particular, she explores how changes in gene expression are associated with tumor aggressiveness and metastasis, focusing on CD44$^+$ and CD133$^+$ CSC populations. She currently utilizes three-dimensional spheroid and organoid models, as well as organoid-derived xenograft models, to analyze CSC-related characteristics and tumor progression.

Seohee Park
Sungkyunkwan University, Korea

The MORC2/CREB Axis Promotes Stemness and Aggressive Phenotypes in CD133+ Hepatocellular Carcinoma Cells

Abstract

Background: Hepatocellular carcinoma (HCC) is a highly heterogeneous malignancy driven in part by populations of cancer stem-like cells (CSCs), which are associated with poor prognosis, metastasis, and resistance to sorafenib. While chromatin remodeling proteins have emerged as key regulators of CSC plasticity, the role of microrchidia family CW-type zinc finger protein 2 (MORC2), a multifunctional epigenetic modulator, in CD133$^+$ HCC remains unclear.

Methods: We investigated the expression and function of MORC2 in HCC using CD133$^+$ spheroid and organoid cultures, CD133-based cell sorting, shRNA-mediated knockdown, and xenograft and metastasis mouse models. Molecular assays, immunohistochemistry, and drug sensitivity analyses were employed to evaluate stemness, EMT, and anti-cancer drug sorafenib response.

Results: MORC2 was overexpressed in HCC tissues and cell lines and positively correlated with CD133 and expression. It was enriched in spheroid cultures and CD133$^+$ cells, where it supported spheroid formation, stemness marker expression (Sox2), and epithelial–mesenchymal transition (EMT) features (Snail, Slug, Vimentin). MORC2 knockdown impaired these CSC-associated traits, significantly reduced invasiveness in vitro, and suppressed tumor growth and lung metastasis in vivo. In CD133$^+$ organoids, MORC2 knockdown reduced proliferation and sensitized cells to sorafenib, with combined treatment showing enhanced suppression of viability and increased apoptosis.

Conclusions: Our findings identify MORC2 as a key regulator of stemness, EMT, and drug resistance in $CD133^+$ HCC. MORC2 may serve as both a prognostic biomarker and a therapeutic target to overcome CSC-driven tumor progression and sorafenib resistance in HCC.

Biography

Seohee Park is a Ph.D. student in the Department of MetaBioHealth at Sungkyunkwan University. She earned her Master's degree from the Samsung Advanced Institute for Health Sciences & Technology (SAIHST) at the same university. Her research focuses on the molecular mechanisms of tumor development, specifically investigating the role of the gene MORC2 in the DNA damage response. Her recent work examines how dysregulated MORC2 expression drives oncogenic processes using cancer stem-like cell (CSLC) models. By integrating molecular genetics with oncology, she aims to identify novel therapeutic targets and advance the understanding of cancer progression to improve patient care.

Mònica Aguiló Domingo
Spain

Delayed Metabolic Toxicity in a Patient with Advanced ALK-Rearranged NSCLC Treated with Lorlatinib: Clinical Response and Safety Profile

Abstract

Background:
Anaplastic lymphoma kinase (ALK) rearrangements are identified in approximately 3–5% of advanced non-small cell lung cancer (NSCLC) and define a molecular subgroup highly responsive to ALK tyrosine kinase inhibitors (TKIs). Lorlatinib, a third-generation ALK inhibitor with enhanced central nervous system penetration, has demonstrated superior systemic and intracranial efficacy compared with crizotinib in the phase III CROWN trial [2,6,8]. Metabolic adverse events, particularly hypercholesterolemia and hypertriglyceridemia, are reported in up to 70–80% of patients and typically occur early during treatment [1,3].

Case Presentation:
We report a 71-year-old male diagnosed with stage IVB EML4-ALK–rearranged lung adenocarcinoma with visceral and brain metastases. Due to symptomatic disease progression at presentation and clinical urgency, systemic platinum-based chemotherapy combined with pembrolizumab was initiated as first-line treatment while awaiting next-generation sequencing (NGS) molecular results. After confirmation of ALK rearrangement, treatment was switched to lorlatinib 100 mg daily.

At 15 months of therapy, the patient achieved a partial visceral response with >50% tumor reduction and complete intracranial response, maintaining preserved performance

status. The observed systemic and CNS response is consistent with the durable efficacy reported in the CROWN study and long-term follow-up analyses [2,6,8,11].

Delayed Metabolic Toxicity and Management:

Contrary to the early-onset dyslipidemia described in clinical trials, the patient developed clinically significant hypercholesterolemia and hypertriglyceridemia approximately 12 months after initiating lorlatinib. Management was guided by the expert consensus on lorlatinib-associated adverse event management by Arriola et al. [3], which served as the primary framework for therapeutic decisions and treatment escalation.

Therapeutic intervention included lipid-lowering therapy with a moderate-intensity statin as first- line treatment for LDL-C control. Due to persistent hypertriglyceridemia >200 mg/dL despite initial intervention, treatment escalation with an additional hypolipidemic agent from a complementary pharmacologic class was required to achieve metabolic control. Arterial hypertension was managed with antihypertensive therapy. No interruption, dose reduction, or discontinuation of lorlatinib was necessary.

Conclusion:

This case confirms the durable systemic and intracranial efficacy of lorlatinib consistent with results from the CROWN trial [2,6,8], while highlighting that metabolic toxicity may present as a delayed

event rather than exclusively early-onset. Long-term surveillance for dyslipidemia is essential to optimize safety without compromising oncologic benefit in ALK-positive NSCLC.

Prativa Majee

Institute of Molecular and Cell Biology (IMCB), Agency for Science, Singapore

An evolutionarily conserved telomerase–cGAS–STING axis shapes inflammatory microenvironments in colitis and colitis -associated colorectal cancer

Abstract

Reactivation of Telomerase is considered an hallmark step in most human cancers, allowing the maintenance of the telomere length that enables unlimited proliferative capacity in tumour cells. Beyond this canonical function, telomerase has been implicated in non-telomeric processes relevant to tumor biology, including inflammation and immune regulation; however, the underlying molecular mechanisms remain poorly defined. In our study, we have uncovered a previously unrecognized role for the telomerase reverse transcriptase subunit, TERT, in directly regulating inflammation through the cytoplasmic cGAS–STING nucleic acid–sensing pathway, using murine and zebrafish models of intestinal inflammation. Employing a knock-in TERTVAA mouse model, in which a reverse-transcriptase–inactive TERT is expressed from its endogenous locus, combined with molecular, pharmacological, and single-cell transcriptomic approaches, we identify a distinct myeloid subpopulation, termed T-MAC, in which TERT potentiates STING activation and induces type I interferon responses. Notably, this inflammatory function of TERT is independent of telomere length and reverse transcriptase activity, establishing a direct immunoregulatory role for TERT in immune signaling in colitis and colitis -associated colorectal cancer. Collectively, our findings redefine telomerase as a cell-type-specific regulator of inflammation with direct relevance to tumor-associated immune responses. By linking TERT activity to cGAS–STING signaling in myeloid cells, this work provides mechanistic insight into how telomerase may shape the inflammatory tumor microenvironment and highlights a therapeutic rationale for targeting TERT in cancer beyond its role in telomere maintenance.

Biography

*Dr. Prativa Majee is a Research Scientist at the Institute of Molecular and Cell Biology (IMCB), A*STAR, Singapore. She obtained her PhD from the Indian Institute of Technology (IIT-Indore), India where her doctoral research focused on antiviral drug discovery and viral genome targeting. Her current research centers on understanding the molecular mechanisms of telomerase reactivation and iron metabolism in cancer, with a particular interest in colorectal and liver cancers. Dr. Majee integrates biochemical, molecular, and cell-based approaches to study cancer progression and to identify novel therapeutic vulnerabilities. She has published twenty papers in leading peer-reviewed journals, including Cancer Discovery, Nature Cell Biology, Molecular Therapy – Nucleic Acids, etc Her work spans cancer biology, telomerase regulation, and RNA structure–based therapeutic targeting. Dr. Majee is a recipient of the A*STAR Open Fund Young Investigator Research Grant, supporting her independent research program in cancer biology.*

Setiani Silvi Nurhidayah
Universitas Gadjah Mada, Indonesia

Association Between Age and Changes in Left Ventricular Ejection Fraction in Breast Cancer Patients Receiving Doxorubicin: A Cross-Sectional Study.

Abstract

Breast cancer is one of the leading causes of cancer-related mortality among women worldwide. Anthracycline-based chemotherapy, particularly doxorubicin, remains a cornerstone of systemic treatment due to its effectiveness. However, its use is associated with cardiotoxicity, commonly manifested as a reduction in left ventricular ejection fraction (EF). Age has been proposed as a potential risk factor for chemotherapy-induced cardiac dysfunction, yet available evidence remains inconsistent. This study aimed to evaluate the effect of age on EF changes in breast cancer patients treated with doxorubicin.

A cross-sectional study was conducted involving 90 female breast cancer patients who received doxorubicin-based chemotherapy at Dr. Sardjito General Hospital between January and December 2023. Patients were categorized into two age groups: <65 years and ≥65 years. EF values were obtained through echocardiographic examination before chemotherapy initiation and after completion of treatment. Comparative analysis between groups was performed using independent t-tests, while regression analysis was used to assess the influence of age and comorbidities on EF changes.

The overall mean EF among participants was 67.82 ± 7.62%. Patients aged ≥65 years showed a slightly higher mean EF compared to those aged <65 years (72.11 ± 5.67% vs. 67.35 ± 7.69%), but the difference was not statistically significant ($p = 0.075$).

Regression analysis demonstrated that age and comorbidities were not significant predictors of EF changes ($R^2 = 0.026$, $p = 0.322$).

In conclusion, age was not significantly associated with changes in ejection fraction among breast cancer patients undergoing doxorubicin chemotherapy. These findings highlight the importance of cardiac monitoring in all patients receiving anthracycline-based therapy, regardless of age. Larger studies with longer follow-up periods are required to better define risk factors for chemotherapy-related cardiotoxicity.

Biography

Setiani Silvi Nurhidayah, MD, is a medical doctor with a strong interest in oncology, women's health, and clinical research. She obtained her medical degree from Universitas Gadjah Mada, Indonesia, and has been actively involved in research related to breast cancer prognosis, maternal health, and nutritional status in gynecologic oncology patients. She has participated in several scientific projects, including studies on prognostic indices in breast cancer and quality of life outcomes in ovarian cancer survivors. Her academic interests focus on evidence-based medicine, cancer outcomes, and improving patient-centered care through clinical research. Silvi is also engaged in academic writing, conference presentations, and multidisciplinary collaborations. She aspires to pursue further training in surgical oncology and contribute to advancements in cancer care and women's health in Indonesia and beyond.

Shovan Saha
United States

Engineering Robust SOPs for Clinical Data Management: A Strategic Framework for Quality, Compliance, and Operational Excellence

Abstract

Clinical trial delays and failures often stem from data quality issues and regulatory compliance gaps, making robust Standard Operating Procedures (SOPs) essential for clinical data management excellence. This presentation explores the strategic engineering of SOPs as the cornerstone for ensuring data integrity, regulatory compliance, and operational efficiency in modern clinical trials.

Drawing from ICH E6(R2), 21 CFR Part 11, and CDISC standards, we examine how robust SOP frameworks address the complexity of contemporary clinical trials, where organizations manage data across multiple global sites in decentralized and hybrid trial models. The presentation showcases how quality-by-design (QbD) principles integrated into SOP development can significantly improve first-pass data quality while reducing data cleaning cycles and inspection preparation time.

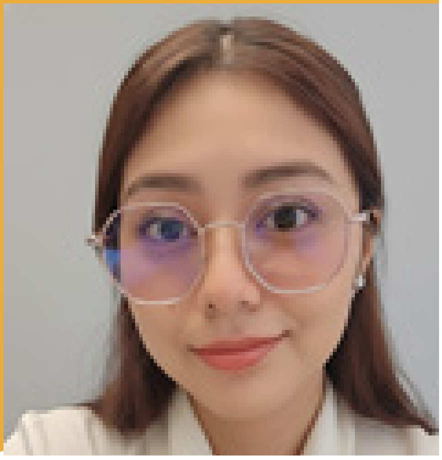
Key topics include SOP engineering for electronic data capture (EDC) systems, implementing risk-based monitoring strategies that optimize resource allocation, establishing data governance frameworks ensuring data integrity across multinational trials, and developing audit-ready documentation. The session covers practical approaches to protocol deviation management, medical coding workflows, statistical programming standards, and final data lock procedures.

Real-world case studies demonstrate how organizations leveraging strategic SOP development have achieved measurable improvements in protocol deviation reduction, data lock timeline optimization, and regulatory inspection success rates. The presentation provides actionable frameworks for developing scalable, technology-agnostic SOPs that adapt to emerging trends including real-world evidence integration, AI-driven data validation, and advanced audit trail technologies.

Attendees will gain practical methodologies for transforming SOPs from compliance documents into strategic enablers of operational excellence, ensuring clinical trial data remains accurate, consistent, and submission-ready throughout its lifecycle while meeting evolving regulatory expectations.

Biography

Shovan Saha serves as Associate Director of Clinical Data Management at Daiichi Sankyo with over 18 years of comprehensive clinical trial experience spanning the complete lifecycle from study startup through database lock activities. Specializing in oncology for 14+ years, he has served as Lead Data Manager and Program Clinical Data Manager for five FDA-submitted and approved cancer treatments across breast cancer, hepatocellular carcinoma, melanoma, pancreatic neuroendocrine tumors, and tuberous sclerosis complex. His expertise encompasses Phase I-IV studies with particular strength in Phase III pivotal trials that achieved regulatory approval. Shovan demonstrates technical proficiency across multiple Clinical Data Management Systems including Oracle Clinical, Oracle RDC, Inform, Rave, Clintrail, and PHOSCO, complemented by expertise in data visualization platforms such as Spotfire, ICRS, and J-Review. With 12+ years successfully managing international CRO relationships, he currently leads multidisciplinary teams of 4-6 staff while providing strategic oversight for complex global initiatives including electronic SAE reporting system implementation and safety database integration. He holds the prestigious Certified Clinical Data Manager (CCDM) designation from SCDM and earned his Master of Science in Biotechnology from Bangalore University. Currently, Shovan is advancing multiple antibody-drug conjugate (ADC) treatment projects across non-small cell lung, gastric, prostate, and breast cancers toward regulatory approval, maintaining the highest standards of data integrity and regulatory compliance throughout the drug development process.



Ximena Yazmin García Bustamante
Universidad Nacional Autónoma de México (UNAM), Mexico

Ovary histological characterization treated with steroidal saponin (25R)-spirost-5-en-3β-yl O-α-L-rhamnopyranosyl-(1 → 4)-β-D-glucopyranoside (3GRD) in a murine breast cancer model

Abstract

Breast cancer is the most common malignant neoplasm in women, with a globally increasing incidence. Current chemotherapy treatments for this disease often induce severe gonadotoxicity. Diosgenin, a naturally occurring steroidal saponin, has been studied as a potential chemopreventive or chemotherapeutic agent. It has demonstrated significant antiproliferative and apoptosis-inducing effects in various cancer types. This study evaluates its glycoside, (25R)-spirost-5-en-3β-yl O-α-L-rhamnopyranosyl-(1 → 4)-β-D-glucopyranoside (3GRD), as a potential antitumor alternative with a favorable ovarian safety profile. An in vivo model using female Balb-C mice was employed by inducing tumor growing via inoculation with 4T1 breast cancer cells. Once the tumor mass developed, mice received antitumor treatment with 3GRD at doses of 2.5, 5, and 10 mg. Cisplatin was included as a positive control; it is a relevant agent for treating aggressive subtypes such as triple-negative breast cancer, but is known for its high systemic toxicity. To analyze the effect of the treatments on the ovary, samples underwent histological characterization via Hematoxylin and Eosin (H&E) staining.

The results evidenced an important inflammatory data and increased atresia process in ovaries without treatment as well in those treated with Cisplatin. The damage observed in non-treated and Cisplatin ovaries is recovered after treatment with 3GRD, showing that there is a better response to higher dosis of 3GRD. The data obtained showed

that the compound significantly improves the preservation of ovarian histoarchitecture and decreases the rate of follicular atresia present during the tumorigenesis process.

Biography

Formerly Consultant Breast Surgeon at Imperial College Healthcare Trust (at Charing Cross Hospital 2006 -2014) London SW6 .Previously Consultant Breast Surgeon at Royal Marsden Hospital ,Fulham Road, London SW3.

Thank You