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ABSTRACT BOOK

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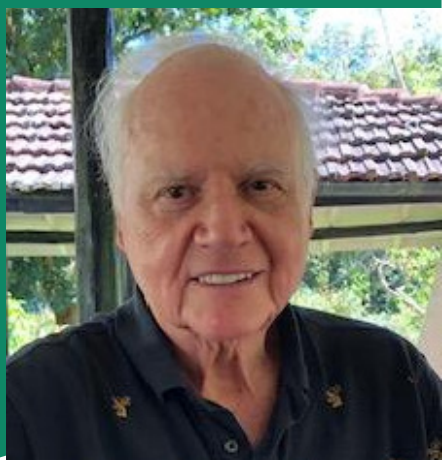


Angus B Gordon
Imperial College Healthcare Trust, London W6 8RF

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.

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.

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.

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.

Janusz Rak
McGill University, Canada

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

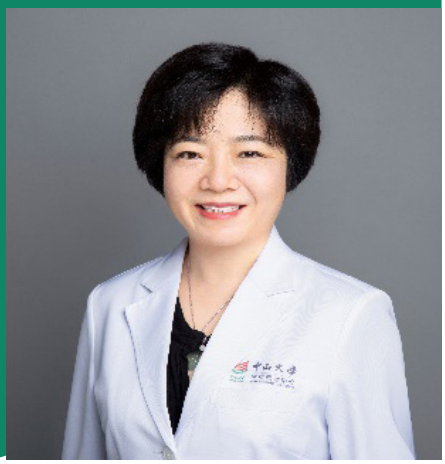
Abstract

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.



Jiang Li
Sun Yat-sen University Cancer Center, China

Modulation of Anti-Tumor Immunity: Lactate-Driven FerroptosisofCD8+TCellsintheTumorMicroenvironment 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.

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.

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.

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.

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

I am MELLITI Rihab, a medical oncologist trained in Tunisia and currently practicing in France. I completed my medical degree at the Faculty of medicine of Monastir and specilized in medical oncology through national residency training across leading tunisian oncology centers. I subsequently pursued multiple postgraduate diplomas in breast cancer, thoracic oncology, oncogenetics, cervicofacial an gynecologic oncology. I am currently a medical oncologist associate practitioner at Aix en Provence general Hospital. I have presented scientific work at the NCCN, ESMO and MASCC congresses and am a member of several international oncology societies.

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

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.

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

chalcone 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.