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ABSTRACT BOOK

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Dr.M. Dougul Regis

Assistant Professor ,Institute of child health and hospital for children ,Madras medical college ,India

Cancer Stem Cells and Metastasis

Cancer stem cells (CSCs), which comprise a small fraction of cancer cells, are believed to constitute the origin of most human tumors. Considerable effort has been focused on identifying CSCs in multiple tumor types and identifying genetic signatures that distinguish CSCs from normal tissue stem cells. Many studies also suggest that CSCs serve as the basis of metastases. Yet, experimental evidence that CSCs are the basis of disseminated metastases has lagged behind the conceptual construct of CSCs. Recent work, however, has demonstrated that CSCs may directly or indirectly contribute to the generation of metastasis. Moreover, CSC heterogeneity may be largely responsible for the considerable complexity and organ specificity of metastases. In this review, we discuss the role of CSCs in metastasis and their potential as therapeutic targets.

Genetic signatures in CSCs are thought to predict tumor recurrence and metastases, providing some support for the concept that CSCs may be metastatic precursors . For example, expression of the CSC marker CD133 in glioblastoma and lung adenocarcinoma is correlated with both the proliferation marker Ki67 and poorer clinical outcomes . CD133 antigen expression has also been shown to correlate with patient survival in high-grade oligodendroglial tumors , rectal cancer , gastric adenocarcinoma , and non-small cell lung cancer. Additionally, in patients with colorectal carcinoma, the combination of CD133, CD44, and CD166 can successfully identify patients at low-, intermediate-, and high-risk of recurrence and

metastasis . Likewise, methylation of Wnt-target-gene promoters are also strong predictors for recurrence in colorectal cancer, suggesting that CSC gene signatures, rather than reflecting CSC numbers, may reflect differentiation status of the malignant tissue and the risk for dissemination.

Biography:

I am DR.M.Dougul Regis ,assistant professor of pathology at institute of child health and hospital for children.I completed my MBBS degree in stanley medical college in 2005 .After completing my ug I did my Diploma in clinical pathology in the prestigious madras medical college and received my graduation in the year 2011 .From 2014 to 2017 I pursued my M.D. pathology course at madras medical college after which I was appointed as assistant professor in stanley medical college .I worked for four years in stanley and currently I work at institute of child health and hospital for children in chennai .I do enjoy working for pediatric patients .I have published 9 articles in various international and national journals .To add on I received the prestigious ASIA RESEARCH AWARDS for excellence in pathology for my publication on pancytopenia in IJRMS .I have a great passion and enthusiasm in teaching undergraduates and post graduate students in the institute I currently work.

Parisa Aziminezhadan

Deparment of General Surgery, School of Medicine, Tehran, Iran

Determining the Upgrade Rate of De novo Breast Intraductal Papillomas to Malignancy, its Related Risk Factors, and 6 Month Outcome After Treatment

Intraductal papillomas (IDPs) are benign breast lesions with potential for malignant transformation. This study aimed to determine the upgrade rate of de-novo IDPs to malignancy, identify associated risk factors, and assess the 6-month outcome after treatment. This retrospective cohort study included 320 patients diagnosed with de-novo IDP at a breast surgery clinic in Tehran, Iran, between March 2011 and March 2022. Patients were divided into upgraded (malignant) and non-upgraded (benign) groups based on pathology results from core needle biopsy (CNB) or vacuum-assisted excision (VAE). Baseline characteristics, pathology outcomes, and follow-up outcomes were analyzed. Multivariable logistic regression identified risk factors for malignant upgrade. Of the 320 participants, 16 (5.0%) had upgraded (malignant) IDPs, and 304 (95.0%) had non-upgraded (benign) IDPs. The median age was significantly higher in the upgraded group (53 years) compared to the non-upgraded group (43 years) ($p<0.001$) Age≥50 years was a significant risk factor for malignant upgrade (, $p<0.001$) The most common malignant pathology was ductal carcinoma in situ (DCIS) (68.8%). Age was identified as a significant risk factor for malignancy, with older age increasing the likelihood of an upgrade (OR=1.249, $p=0.02$). After 6 months follow-up, three patients with IDP were detected by sonography. Older age was the sole significant risk factor for malignant

transformation of IDPs. Continuous follow-up is recommended, especially for older patients, to promptly detect potential recurrence or malignant progression.

Shirin Tavakoli

Department of Applied Cell Sciences, School of Advanced Technologies in Medicine, Tehran University of Medical Sciences, Tehran, Iran

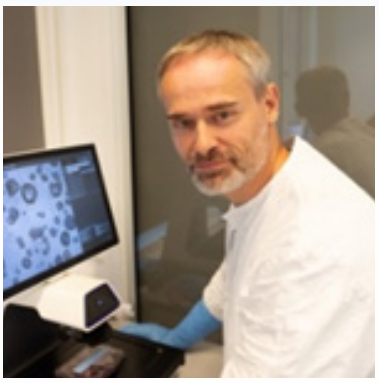
Evaluating the Safety and Feasibility of Allogeneic NK Cell Infusion in High-Risk Lymphoma Patients Post-Autologous Stem Cell Transplantation

Lymphoma, a cancer with a poor prognosis, is a growing global health challenge that encompasses two primary types, Hodgkin (HL) and non-Hodgkin lymphoma (NHL), each further divided into various subtypes with distinct biological behaviors. Conventional therapeutic strategies include chemotherapy, radiation, surgery, and autologous hematopoietic stem cell transplantation (auto-HSCT). Natural killer (NK) cells exhibit intrinsic cytotoxicity against tumor cells without the need for prior immunization or activation. In this prospective clinical trial, we evaluated the feasibility of allogeneic NK cell therapy in patients with high-risk lymphoma who had a poor prognosis. Each patient received a 1 × 10^7 NK cells/kg infusion without interleukin-2 (IL-2) supplementation. Therapy was tolerated without graft-versus-host disease, cytokine release syndrome, or neurotoxicity. During the follow-up period, 7 had complete responses (CR) (87.5%), and one case exhibited stable disease (SD) (12.5%). In summary, our investigations support the development of allogeneic NK cellular therapies for advanced lymphoma to overcome chemoresistance. Therapeutic efficacy may be further improved by disrupting the immunosuppressive environment and infusion of exogenous IL-15. This approach presents a promising and pragmatic strategy for managing high-risk lymphoma post-HSCT. Future research should focus on optimizing NK cell dosages and infu-

sion frequency to maximize treatment effectiveness.

Biography:

She completed her PhD from Tehran University of Medicine at the age of 36. She has published more than 13 articles in reputable journals.



Jarle Bruun
Oncosyne

Targeting multiresistant colorectal tumors with combination therapies using patient-derived tumoroids

In advanced cancer patients receiving chemotherapy, fewer than 40% achieve clinical benefit, and multidrug resistance is common. For cancer drugs entering phase I trials, fewer than 15% ultimately gain approval. Emerging technologies in 3D patient-derived cell culture, automated drug dispensing, high-content screening, and machine learning offer new opportunities to improve drug development and increase patient response rates through individualized combination therapies.

We applied iCAN, a clinical-grade drug profiling platform based on predictive phenotyping of patient-derived tumoroids (PDTs), to screen a representative biobank of 56 unique PDTs from primary colorectal cancers against 35 clinically relevant drugs. Comparative dose–response profiling identified a set of multidrug-resistant tumors, of which 8 plus 3 controls were subsequently tested with rational drug combinations based on their individual pharmacogenomic profiles comprising approved, off-label, and investigational agents. We focused on models with trippel wild-type RAS and KRAS G12D, KRAS G13D and NRAS Q61K mutations. In particular, synergistic and additive effects were observed for inhibitors of the MAPK signaling pathway. In addition, a novel strong synergistic relationship was uncovered between two off-label drugs.

The iCAN tumoroid platform enables identification of novel and potentially effective combination therapies, supporting individualized treatment strategies for patients with multidrug-resistant colorectal cancer.

Biography:

Jarle Bruun earned his PhD in cell biology, genomics, and pathology from the University of Oslo in 2014, followed by postdoctoral research at the Institute for Cancer Research, Oslo University Hospital. He is the CEO and co-founder of Oncosyne, a biotechnology company developing clinical-grade tools for next-generation functional precision medicine.

Dr. Bruun has extensive hands-on experience in building end-to-end technology platforms, including next-generation sequencing, high-throughput biomarker discovery through digital image analysis of fluorescence-based immunohistochemistry on large tissue microarrays, and 3D patient-derived organoid systems for translational drug screening and pharmacogenomics.

He has authored more than 35 peer-reviewed publications and continues to bridge cutting-edge translational research with clinical application in oncology drug development.

Dr. Rana Jahanban Esfahlan

Department of Medical Biotechnology, Faculty of Advanced Medical Sciences, Tabriz University of Medical Sciences, Tabriz, Iran

Dual-Loaded Niosome-Dendrimer Nanoplatform Enhances Tirapazamine Delivery To Hypoxic Breast Cancer Cells

Breast cancer (BC) remains one of the most prevalent cancers among women, necessitating effective and comprehensive treatment methods to reduce both its impact and associated healthcare costs. In this study, we developed a novel dual nanoparticle system—niosomes encapsulating PAMAM and Tirapazamine (N@P/T)—and evaluated its therapeutic potential using both computational and experimental approaches.

Through molecular docking and protein–protein interaction network analysis, HIF1A was identified as a key target for Tirapazamine (TPZ), with several strong binding sites and interactions linked to critical cancer pathways. The N@P/T nanoparticles, created via the thin-film hydration technique, displayed an average size of ~200 nm, a zeta potential of −4 mV, and a spherical shape. MTT assay revealed that N@P/T significantly outperformed both PAMAM/TPZ (P/T) and free TPZ in terms of cytotoxicity, showing the lowest IC50 value at 14.14 µM, compared to 71.37 µM for P/T and 143.3 µM for free TPZ. Annexin-V FITC/PI staining confirmed stronger pro-apoptotic effects with N@P/T (65.33%) and P/T (44.28%), likely due to modulation of BCL2, caspase-3, and BAX gene expression. Cellular uptake studies showed over 90% internalization of N@P/T within 4 hours. Additionally, real-time PCR confirmed HIF1A as a functional target of TPZ

under hypoxic conditions. In 3D tumor spheroid models, N@P/T demonstrated superior penetration and significantly disrupted spheroid structure and size.

Overall, this combined computational and laboratory investigation highlights N@P/T as a promising and targeted treatment strategy for hypoxic breast cancer cells, offering enhanced efficacy through precise molecular targeting.

Biography:

Dr Rana Jahanban is an assisstant professor of Medical Biotechnology. With an H-index of 46, she ranks among 2% highly cited young scientists in the world. Her research focuses on innovative strategies for breast cancer therapy, emphasizing the development of advanced drug delivery systems. By exploring targeted delivery methods, she aim to enhance the efficacy of therapeutic agents while minimizing side effects. This involves the use of nanotechnology, biomaterials, and personalized medicine approaches to improve the precision of treatment. Her goal is to create more effective, patient-centered therapies that can significantly improve outcomes for individuals diagnosed with breast cancer.

Marina Patysheva

Laboratory of Cancer Progression Biology, Cancer Research Institute, Tomsk National Research Medical Center

Monocyte-Related Markers as Predictors of Neoadjuvant Chemotherapy Efficacy In Breast Cancer Patients

The immune system plays a direct role in cancer development and response to therapy. Neoadjuvant chemotherapy (NAC) is a standard treatment for locally advanced breast cancer, and its efficacy is increasingly recognized to depend on immune modulation. Monocytes, as key components of the innate immune system, may serve as potential predictive biomarkers of NAC response.

We aimed to evaluate the predictive value of monocyte subsets in patients with luminal B and triple-negative breast cancer (TNBC) undergoing NAC. Peripheral blood monocytes were analyzed by flow cytometry, bulk RNA sequencing, and 3' single-cell RNA sequencing in a cohort of NAC responder and non-responder breast cancer patients. We found that the frequencies of CD14lowCD16highCD163+ and CD14highCD16highCD163+ monocytes were significantly higher in patients compared to healthy women ($p=0.039$ and $p=0.046$, respectively). Responders to NAC exhibited a significantly higher proportion of CD14lowCD16highHLA-DR+ monocytes than non-responders (84.62% vs. 55.12%, $p=0.005$). Notably, we identified, for the first time, a myeloid-derived suppressor cell (MDSC)-like signature within the S100AhighMHC-IIlow monocyte subset and developed an MDSC score that differentiated responders from non-responders in triple-negative breast cancer patients. Our data underscore the functional relevance of monocyte subsets

in determining NAC response and highlight their potential as non-invasive predictive biomarkers, particularly in breast cancer.

Biography:

In 2023, Ms. Patysheva completed her PhD on "Characteristics of Monocytes in Breast Cancer Under Chemotherapy" at Tomsk State University and the Tomsk National Research Medical Center, under the supervision of Professor J.G. Kzhyshkowska. She is currently conducting postdoctoral research at the Tomsk National Research Medical Center. Dr. Patysheva (PhD, MD) is a researcher in the Laboratory of Cancer Progression Biology at the Cancer Research Institute, Tomsk National Research Medical Center, Tomsk, Russia. Her expertise lies in immuno-oncology, with a focus on high-precision single-cell transcriptomic analysis. Her research interests include the immunology of breast, colorectal, and oral cancers, as well as the biology of the innate immune system—particularly monocytes and tumor-associated macrophages. She also investigates the effects of cancer therapies on immune responses.

Eun-Min Cho
Seokyeong University, Republic of Korea

Utilizing the CompTox Chemicals Dashboard for Toxicity Prediction of Hazardous Chemicals: Case-Based Approach

In the past, various experimental-based toxicity data, including epidemiological studies and animal experiments, were collected for human health risk assessment. However, with the recent rise of international attention to ethical concerns in animal testing and the drive to maximize efficiency, the 3R principles, along with shifts in human risk assessment paradigms, have highlighted the need for and spurred active research on NAMs (New Approach Methodologies), a novel non-animal experimental approach. In particular, obtaining data that provide scientific evidence on the human toxicity and risk of chemicals is of great importance. The U.S. EPA provides a web-based database for chemical toxicity and risk prediction, systematically collecting and offering data on chemicals. In this study, we aimed to provide foundational data for computational toxicology assessment by utilizing the CompTox Chemicals Dashboard for chemicals exhibiting reproductive toxicity, including chemical properties, in vitro bioactivity, high-throughput screening (HTS) results, concentration-response data, and conducting read-across analysis to assess human health risk.

Biography:

I obtained my Ph.D. in Applied Biotechnology from the University of Tokyo and conducted my postdoctoral research at the University of Florida. I am currently serving as a professor in the Department of Nano Chemical and Biological Engineering at Seokyeong University in Korea. Recently, as part of my research on the effects of environmental hazardous substances on human health, I have been establishing fundamental datasets to assess their toxic impacts using public data.

Mats Ljungman

Department of Radiation Oncology, Rogel Cancer Center and Center for RNA Biomedicine, University of Michigan, USA

KLIPP: Precision targeting of fusions and amplified oncogenes using CRISPR

Oncogenic fusion genes and amplified oncogenes are common drivers of cancer but there are currently no therapeutic approaches available that directly target these cancer-specific genomic features. Amplified oncogenes in cancer can be found on chromosomes as homologous staining regions (HSRs) or on extrachromosomal DNA (ecDNA). Presence of oncogenes on ecDNA poses a particular challenge because they are under strong positive selection in response to anti-cancer therapy leading to treatment resistance as their copy number can rapidly change due to uncontrolled segregation during cell division. To specifically target cells with fusions or amplified oncogenes, we have developed a CRISPR-based therapeutic approach, "KLIPP," which is designed to target structural variants junctions (SVJs) specific to cancer genomes, with few, or no off-target effects expected in normal cell. KLIPP uses a "split enzyme" approach consisting of a dead Cas9 endonuclease (dCas9) fused to the endonuclease Fok1 where two Fok1 endonucleases need to homodimerize to become active. To "nucleate" and activate these complexes, sgRNAs are designed to bind sequences flanking cancer-specific SVJs, bringing two Fok1-dCas9 complexes together to induce double-strand breaks (DSBs). Using the KLIPP approach, we have obtained strong proof-of-concept that it can specifically target cancer-unique SVJs and induce DSBs leading to cell death in cancer cells. We have shown this for simple SVJs in cancer genomes as well as

in oncogenic fusion genes such as EWS-FLI1 in Ewing sarcoma. Furthermore, we show that we can use KLIPP to target unique SVJs on amplified oncogenes on ecDNA resulting in strong induction of γH2AX and loss of cell survival. We believe that KLIPP could be a safe and cancer-specific precision therapy for targeting cancer driven by fusions and amplified oncogenes.

Biography:

Mats Ljungman, PhD

Professor of Radiation Oncology and Environmental Health Sciences, University of Michigan

I grew up in Stockholm, Sweden. After attending 4 years of college studies in the USA, I landed a graduate student position in the lab of Professor Gunnar Ahnström at Stockholm University in Sweden. My thesis was entitled "The role of chromatin in the induction and repair of DNA damage". I performed my postdoctoral work, in the laboratory of Dr. Phil Hanawalt at Stanford University, where I developed a new technique that uses the ability of psoralen to "sense" torsional tension in DNA to probe for "unconstrained DNA torsional tension" in the genomes of living cells. In 1994, my family and I moved to Ann Arbor where I became Assistant Professor in the Department of Radiation Oncology at the University of Michigan Medical School where I have been ever since. My early work identified blockage of transcription as a major trigger of p53 and apoptosis after DNA damage. To map transcription genome-wide and to investigate the effect of DNA damage on ongoing transcription we developed Bru-seq, which is based on bromouridine labeling of nascent RNA followed by immunocapturing and deep sequencing of the Bru-labeled RNA. We were members of the ENCODE consortium for 10 years contributing with Bru-seq. We recently developed Precision KLIPP Therapy as a personalized and specific cancer-targeting approach using CRISPR.

Nilabja Sikdar[1,2], Akash Bararia[2], Ankita Chatterjee[3], Deepyman Das[4], Arunima Maiti[5], Gourav Ghosh[6], Sumit Mukherjee7, Paramita Roy[8], Shibajyati Ghosh[9], Supriyo Ghatak[10], Bitan K Chatterjee[11], Debabrata Ghosh Dastidar[6], and Sudeep Banerjee[8]

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Identification of Novel Epigenetically Regulated Genes With Poor Prognosis in Indian Pancreatic Cancer Patients Cohort

Background

Pancreatic ductal adenocarcinoma (PanCa) is noted for its high lethality, with a survival rate of approximately 12%. The study emphasizes the need for a deeper understanding of the disease's pathophysiology, particularly the role of ion channels in cancer progression.

Methods

Methylome data, generated using 450 K bead array, was compared between paired PDAC and normal samples in the TCGA cohort (n=9) and our Indian cohort (n=7). The total Indian Cohort consisted of n = 75. Validation of differential methylation (6 selected CpG loci) and associated gene expression for differentially methylated genes (10 selected gDMs) were carried out in separate validation cohorts, using MSP, RT-PCR and IHC correlations between methylation and gene expression were observed in TCGA, GTEx cohorts and in validation cohorts. Metascape and Enrich R were used for pathway analysis.

Results

We identified 156 DMPs, mapped to 91 genes (gDMs), in PDAC; 68 (43.5%) DMPs were found to be differentially methylated both in TCGA cohort and our cohort, with significant concordance at hypo- and hyper-methylated loci. Enrichments of "regulation of ion transport", "Interferon alpha/beta signalling", "morphogenesis and development" and "transcriptional dysregulation" pathways were observed among 91 gDMs. Hyper-methylation of NPY and FAIM2 genes with down-regulated expression in PDAC, were significantly associated with poor prognosis in the Indian patient cohort. KCNJ5, CACNB2, CLIC5, RASA3, GABBR2, KCNA3 and KCNA6 were identified as key ion transport genes. KCNJ5 was found to be hypomethylated and overexpressed in this cohort, contrasting with its hypermethylated and underexpressed status in the TCGA cohort. High expression levels of KCNJ5 were associated with poor survival outcomes in patients, as demonstrated by Kaplan-Meier survival analysis. This suggests that KCNJ5 could serve as

a potential prognostic biomarker for PanCa. KCNJ5 was shown to be part of a highly clustered protein-protein interaction network, indicating its significant role in various biological pathways related to ion transport. The analysis revealed that KCNJ5 interacts with other potassium channels and is involved in critical cellular functions. The study also performed molecular dynamics simulations to assess the stability of the Kcnj5 protein complex with protodioscin, suggesting its potential as a therapeutic target.

Conclusion

Ethnic variations in Indian PDAC patients may influence their epigenetic landscape. The study identified novel differentially methylated genes, mainly NPY and FAIM2, associated with poor survival and advanced tumor stages. KCNJ5 could be a promising therapeutic target for Indian PanCa patients, but further validation is needed.

Hany Nabil Mazloum Azzam

Department of Pharmacology and Toxicology, Faculty of Pharmacy, Heliopolis University, Cairo, Egypt

Metabolic/hypoxial axis predicts Tamoxifen resistance in Breast Cancer

We sought in our cross-sectional study to investigate the role of metabolic/hypoxial axis in the development of tamoxifen (TMX) resistance in BC patients. Quantification of plasma LncRNA Taurine upregulated-1 (TUG-1), miRNA 186-5p (miR-186), serum Sirtuin-3 (SIRT3), Peroxisome Proliferator Activator Receptor alpha (PPAR-1 α) and Hypoxia Inducible Factor-1 (HIF-1α) was done in a cohort of patients divided into TMX-sensitive and TMX-resistant candidates. Multiple logistic regression and Receiver Operating Characteristic curve were developed for significant predictors. Plasma TUG-1 and miR-186 were significantly elevated in TMX resistant patients. Serum proteins SIRT3, PPAR-1 α and HIF-1α were deficient in TMX resistant patients compared to TMX sensitive patients respectively. miR-186 was associated with respiratory symptoms, while, HIF-1α was associated with metastases in TMX resistant patients. Strong correlations were found between all parameters. A predictive model was constructed with TUG-1 and HIF-1α to estimate TMX resistance in BC patients with 88.3% sensitivity and 91.6% specificity. Hypoxia and metabolic dysregulations play important role in the development of TMX resistance in BC patients. Correlation between hypoxia, carcinogenesis and patient's mortality have led to more aggressive phenotypes, increased risk of metastasis and resistance to TMX.

Biography:

I'm Hany Nabil. I'm a Senior Clinical Pharmacist at Dr Bakhsh Hospital, Jeddah, KSA. I worked as a Former Lecturer in the Clinical Pharmacy department at Heliopolis University. I had my PhD in pharmacology at Ain Shams University and my master's at the German University in Cairo. I'm an American Board-Certified Pharmacotherapy Specialist and a Good Clinical Practice certificate holder. Additionally, I earned lately Healthcare Excellence Diploma from the American University in Cairo.

As per my experience as a Lecturer, I think I'm capable of teaching in lectures, ensuring content, methods of delivery and learning materials meet the defined learning objectives for individual teaching sessions.

As per my experience as a Clinical Pharmacist. When I worked as a Clinical Pharmacist, I assisted medical staff in selection of treatment plans, in patient counseling and in therapeutic interventions.

KWAN Hiu Yee

Centre for Cancer & Inflammation Research, Institute of Systems Medicine and Health Science, School of Chinese Medicine, Hong Kong Baptist University, Hong Kong

Obesity promotes breast cancer growth: Role of circulating small extracellular vesicles

The cargo content in small extracellular vesicles (sEVs) changes under pathological conditions. Our data shows that in obesity, extracellular matrix protein 1 (ECM1) protein levels are significantly increased in circulating sEVs, which is dependent on integrin-β2. Knockdown of integrin-β2 does not affect cellular ECM1 protein levels but significantly reduces ECM1 protein levels in the sEVs released by these cells. In breast cancer (BC), overexpressing ECM1 increases matrix metalloproteinase 3 (MMP3) and S100A/B protein levels. Interestingly, sEVs purified from high-fat diet-induced obesity mice (D-sEVs) delivermore ECM1 protein to BC cells compared to sEVs from control diet-fed mice. Consequently, BC cells secrete more ECM1 protein, which promotes cancer cell invasion and migration. D-sEVs treatment also significantly enhances ECM1-mediated BC metastasis and growth in mouse models, as evidenced by the elevated tumor levels of MMP3 and S100A/B. Our study reveals a mechanism and suggests sEV-based strategies for treating obesityassociated BC.

Biography:

Prof. Kwan hold a PhD in Physiology from the Faculty of Medicine at The Chinese University of Hong Kong, followed by postdoctoral training in the Department of Nutritional Sciences & Toxicology at the University of California, Berkeley, USA.

She has published over 120 research papers, review articles, editorials, and book chapter in the areas of oncology, obesity and nutrition in prestigious journals. She is actively involved in developing multiple research platforms dedicated to investigating the roles of small extracellular vesicles (sEVs) in disease development, as well as exploring the intricate relationship between obesity and cancer. Additionally, she has made significant contributions in discovering innovative sEV-based therapeutics for the treatment of cancer, obesity, and related comorbid conditions..

Karim Khaled

Department of Ophthalmology, Gilbert and Rose-Marie Chagoury School of Medicine, Lebanese American University, Beirut, Lebanon

Prostate Cancer and Dietary Sugar Intake: A Systematic Review

Prostate cancer is a leading malignancy among men worldwide, and its incidence is projected to increase due to aging populations and lifestyle changes. While established risk factors include age, ethnicity, and genetics, the role of modifiable dietary factors, particularly sugar intake, remains unclear. This systematic review aimed to evaluate the current evidence on the association between dietary sugar intake and prostate cancer risk. A comprehensive search of six databases identified observational studies published between January 2005 and April 2025. Eligible studies quantitatively assessed sugar intake (total sugars, added sugars, or sugar-sweetened beverages) in relation to prostate cancer outcomes. Screening, data extraction, and risk of bias assessment (ROBINS-E) were conducted independently by multiple reviewers. Six studies met the inclusion criteria, comprising four prospective cohorts, one case–control study, and one cross-sectional study, with a combined sample of 11,583 men from the USA, Canada, Sweden, and France. Three studies reported a significant positive association between high sugar intake—particularly from added sugars and sugar-sweetened beverages—and increased prostate cancer risk, while two found no association and one reported mixed results depending on sugar type. Methodological heterogeneity, differences in exposure assessment, and varied confounder adjustments limited comparability across studies. This review suggests a possible link between high dietary sugar intake and increased prostate cancer risk, but inconsistent findings and study limitations highlight the need for robust, standardized prospective research to clarify this relationship.

Biography:

Dr. Karim Khaled holds a PhD in Epidemiology and serves as an Assistant Professor with over four years of academic teaching experience, nine years of epidemiological research, and three years of clinical practice. His research includes epidemiology, cancer, systematic reviews and meta-analyses, nutrition, mental health, reproductive health, surveillance, and ethical considerations in public health. He has published extensively in high-impact journals, including MDPI and BMC journals, with recent works addressing ethical consideration of using mental health questionnaires, dietary patterns, prostate cancer, stress, and mental health outcomes. Dr. Khaled is a Registered Nutritionist with the Association for Nutrition (UK) and a Licensed Dietitian with the Ministry of Public Health (Lebanon). He is an active member of The Nutrition Society, an organizing member of the International Conference on Nutritional Health and Food Science & Technology, and a certified reviewer for MDPI, Frontier, Springer Nature journals, and others. In academia, he leads and co-leads modules in epidemiology, research methods, microbiology, and nutrition, fostering evidence-based public health practice.

Areeba Ahmed

Aga Khan University Hospital, Karachi Stadium Road, P.O. Box 3500 Karachi 74800

Primary Urothelial Carcinoma of an Ileal Conduit; Six Decades After Childhood Bladder Exstrophy Surgery: A Rare and Late Complication

Background:

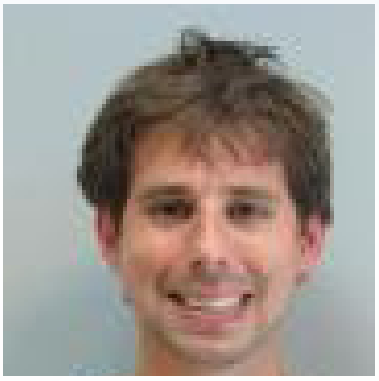
Bladder exstrophy is a rare congenital anomaly that requires surgical reconstruction or urinary diversion early in life. While adenocarcinoma is the most commonly associated malignancy, primary urothelial carcinoma arising within an ileal conduit without any evidence of disease in the entire urinary tract is exceedingly rare and has never been reported before.

Case presentation:

We report a case of a 64-year-old male with a history of bladder exstrophy managed with an ileal conduit in early childhood. He presented with intermittent bleeding from his urinary stoma, and subsequent evaluation revealed a high-grade invasive urothelial carcinoma arising within the ileal conduit, without involvement of the ureteric orifices or native urinary tract. Metastatic spread to the regional lymph nodes and liver underscored the aggressive disease course. Despite prompt initiation of chemotherapy and later immunotherapy, the disease progressed rapidly, leading to severe complications, including bilateral hydronephrosis requiring percutaneous nephrostomy. The patient was ultimately transitioned to palliative care.

Conclusion:

Primary urothelial carcinoma in an ileal conduit of bladder exstrophy patient is a rare condition. The latency period for the onset of this aggressive cancer in urinary diversions can be long but mainly occurs before the age of 65. This reinforces the need for long-term follow-up of patients with urinary diversions, even in the absence of symptoms. We advocate for routine screening of these patients, initiating before the age of 30 as previously recommended for bladder exstrophy patients.



Yarden Ariav
Weizmann Institute of science, Rehovot, Israel

Elevating cGMP levels through PDE5A inhibition restricts cancer metastasis by impeding NPC1-mediated cholesterol trafficking

Background: Cholesterol metabolism is integral to cancer progression, particularly in supporting the energy-intensive processes of tumor cell migration and metastasis. Although metastatic cells often exhibit increased cholesterol synthesis, disruptions in cholesterol trafficking can limit their migration and metastatic capability. PDE5A, a cyclic GMP phosphodiesterase, is critical in regulating cGMP levels, influencing various cellular processes. We hypothesized that sildenafil citrate (Viagra), a well-known PDE5A inhibitor, could impact cancer progression by reducing GMP and altering nucleotide pools. We discovered that sildenafil's primary effect on tumors is the disruption of cholesterol trafficking, therefore impairing metastatic progression. Sildenafil's established clinical use highlights its potential as a therapeutic to target cholesterol metabolism and metastasis.

Methods: We used RNA-seq, proteomics, and lipidomics to define sildenafil's molecular effects across multiple cancer models. Cell proliferation and migration were assessed via live imaging. Genetic knockdown of PDE5A confirmed specificity. Lysosomal cholesterol trafficking was evaluated through immunofluorescence. Binding assays were performed to confirm direct cGMP interaction with the NPC1 transporter. In vivo metastasis was quantified in murine models.
Clinical relevance was supported by retrospective analysis of cancer patients treat-

ed with PDE5A inhibitors and statins.

Results: Sildenafil treatment elevated intracellular cGMP and impaired cholesterol binding to the lysosomal transporter NPC1, leading to lysosomal cholesterol accumulation and reduced mitochondrial ATP production. This triggered compensatory cholesterol gene expression and disrupted membrane lipid raft organization. Importantly, the effect was specific to tumor cells, with minimal impact on untransformed cells, underscoring a cancer-selective vulnerability. These alterations significantly reduced cell proliferation and migration in vitro. In vivo, sildenafil lowered metastatic burden, with enhanced efficacy when combined with statins. Retrospective analysis of patient medical records revealed improved survival with PDE5A inhibitor treatment, with additive benefits observed when combined with statin therapy.

Conclusion: This study uncovers a novel anti-metastatic mechanism of sildenafil, showing that PDE5A inhibition disrupts cholesterol trafficking specifically in tumor cells via direct cGMP- mediated inhibition of NPC1, a previously undescribed interaction with major translational implications. By targeting a cancer-specific metabolic weakness with a well-characterized drug, our findings support repurposing sildenafil as a clinically accessible strategy to inhibit metastasis.

Biography:

I recently completed my PhD in cancer metabolism at the Weizmann Institute of Science in the lab of Prof. Ayelet Erez and am now a postdoctoral researcher in the same lab, studying how lipid metabolism influences cancer progression and metastasis. I also hold an MBA from Tel Aviv University, which I combine with my biomedical research background to approach science with strategic thinking and a focus on real-world impact. Coming from a family of agronomists, I have a lifelong interest in connecting scientific discovery to practical applications. My work has included both independent and collaborative projects under tight timelines, and I have presented it at national and international conferences. Fluent in English and Spanish, I value cross-cultural collaboration and am passionate about building bridges between academia and industry to advance innovative solutions in health and beyond.

Ashleigh Rosson
ECS Health Centers of America

Women, trauma and alcohol dependency: Connections and disconnections in alcohol treatment for women

Statement of the Problem: The public's interest in plant-based medicine has grown substantially, yet many individuals still face challenges with understanding how to properly dose and utilize cannabinoids for optimal wellness. A significant barrier exists in public perception—most believe plant-based medicine, especially cannabis, requires a psychoactive experience to be effective. This misconception prevents many from exploring or benefiting from natural therapeutic options. Furthermore, mainstream health systems often lack the tools and education necessary to support this alternative healing modality.

Methodology & Theoretical Orientation: This presentation combines personal narrative, clinical observation, and audience education to explore the practical use of cannabinoid therapy. It is grounded in applied holistic wellness and supported by current research into the endocannabinoid system and homeostasis. Drawing from years of practice and anecdotal evidence, Ashleigh Rosson integrates story, science, and strategy to deliver a dynamic, empowering session.

Findings: Over the course of her work, Ashleigh has helped over 23 individuals eradicate cancer from their bodies and guided hundreds off opioid dependency—all without the use of high-inducing cannabis products. These outcomes highlight

the transformative potential of properly dosed cannabinoid therapy and the critical role of individualized care and education.

Conclusion & Significance: Plant-based medicine—when understood and applied properly—can be a safe, effective solution for chronic conditions, pain management, and nervous system regulation. This presentation underscores the importance of educating the public and medical communities on cannabinoid therapy to remove fear, reduce stigma, and improve health outcomes. Attendees will leave with actionable insights and a renewed perspective on the science and soul of plant healing.

Biography:

Ashleigh Rosson is a wellness practitioner, speaker, and founder of ECS Health Centers and Earth Island Lotion. With a background in cannabinoid therapy and a deep passion for holistic health, Ashleigh brings eight years of experience transforming lives through plant-based medicine. Her mission was born from personal tragedy—losing a loved one to an epileptic episode—which ignited her dedication to understanding why cannabis oil stops seizures and how plant medicine can bring people back into balance. Ashleigh has helped over 23 individuals overcome cancer and guided countless others off opioids through customized dosing programs. She is the creator of Plant the Love TV, a speaker at institutions like the Mayo Clinic, and an advocate for empowering people to take control of their health—without fear and without the high. Her work bridges science, spirituality, and community care for real and lasting impact.

Issam Jamil Atiya AbuQeis, PhD, MPH, BSc (MedImg)

Research Assistant
Oncology laboratory, Institute of Neuroscience, School of Basic Medicine, Kunming Medical University, China

The study on the role of ATAD2 from glioma to brain ischemia: A primary exploration of cancer for neuroscience

Background: Gliomas demonstrate significant heterogeneity in aggressiveness, ranging from slow- growing low-grade gliomas (LGG) to highly malignant high-grade gliomas (HGG). Cerebral ischemia-reperfusion (CIR) injury is a significant cause of long-term neurological dysfunction, with the molecular mechanisms underlying impaired cognitive recovery remaining largely undefined. This study introduces a novel perspective, "Cancer for Neuroscience," reframing gliomas not merely as pathological entities but as potential mediators of central nervous system (CNS) repair.

Methods: Bioinformatics analysis of glioma datasets identified key hub genes. Their functional impact was assessed in vitro via siRNA silencing in C6 glioma cells (morphology, viability, apoptosis). Candidate gene expression was profiled in rodent models of CIR, spinal cord injury (SCI), and traumatic brain injury (TBI). The role of the lead candidate, ATAD2, was validated in vivo using an AAV-mediated gain- and loss-of-function approach in a rat model of middle cerebral artery occlusion (MCAO), followed by comprehensive behavioral, histological, and molecular analyses.

Results: Our results demonstrated that ATAD2 overexpression markedly enhanced

spatial learning, memory consolidation, and exploratory behavior. Conversely, ATAD2 knockout exacerbated cognitive deficits. Histological and molecular analyses further corroborated these findings, revealing that ATAD2 overexpression effectively alleviated neuronal apoptosis and improved neuroplasticity following CIR. Collectively, these results provide the first compelling evidence that ATAD2 plays a protective role in cognitive recovery post cerebral ischemia.

Conclusion: This study validates the "Cancer for Neuroscience" approach, thereby challenging traditional paradigms and demonstrating that the oncogenic factor ATAD2 can be repurposed to promote neuroprotection and functional recovery after cerebral ischemia. Our work bridges glioma biology and neural repair, offering a novel therapeutic strategy and a transformative perspective for treating CNS injuries.

Keywords: Cancer, Neuroscience, Glioma, Brain Ischemia, Spinal Cord Injury, Traumatic Brain Injury.

Biography:

Issam Jamil Atiya AbuQeis, PhD, MPH, BSc (MedImg)
Research Assistant
Oncology laboratory, Institute of Neuroscience, School of Basic Medicine, Kunming Medical University.

Dr. Sami Salahia
Dr Batoul Reffai Medical Center, Dubai

Systematic Review and Network Meta Analysis Training Course

To guide participants through a step-by-step process of conducting systematic reviews and meta-analysis. To create a collaborative atmosphere that inspires meaningful, high-quality research outputs. To enhance the research capabilities of medical professionals through practical, hands-on learning.

Biography:

Master's Student in Manufacturing of Advanced Therapy Medicinal Products, UOG, Spain.
Medical Residency in Dermatology from Ain Shams University Hospital, Cairo, Egypt
Doctor of Medicine (MBBCh/MD) from the School of Medicine, Ain Shams University, Cairo, Egypt (2013-2020).

Clinical Experience
Practicing as General Practitioner at Dr. Batoul Reffai Medical Center in Dubai, UAE- DHA License
Specializes in Regenerative, Anti-Aging, and Laser Dermatology.
Completed Internship & Observership in Dermatology at Ain Shams University Hospitals, Cairo.

Research & Leadership
Founder of Research Pioneer, a hybrid platform for evidence-based medical innovation.
Co-founder of the MRSA Online Group for Oncology Databases. Published Researcher with multiple peer-reviewed articles and a Google Scholar H-Index of 5.
Presented award-winning research competition at the international IMCAS Conference in Paris (2022).

Fatemeh Sanjabi
Prostate Cancer Research Australian Centre

Testing the efficacy of Neuropilin-1 inhibition in pre-clinical models of claudin- low breast cancer

Claudin-low breast cancers (CLDNlow BrCa) are aggressive malignancies characterised by low expression of cell-cell adhesion proteins and enrichment of mesenchymal and stem-like properties. Since limited therapeutic targets are available, chemotherapy is the mainstay of treatment. Neuropilin-1 (NRP1), a cell surface co-receptor, has been implicated in multiple oncogenic processes, including angiogenesis and cell migration, and identified as a regulator of CLDN low BrCa progression. This research phenotypically characterises the impact of NRP1-inhibition on CLDNlow BrCa cells and assess their responsiveness to standard chemotherapy agents through in- vitro assays and bioinformatic analysis. NRP1 was suppressed in claudin-low cells by three approaches: CRISPR-Cas9 knockdown (KD), the anti-NRP1 antibody Vesencumab, and siRNA. NRP1 CRISPR KD models were assessed by in-vitro functional assays and related driving pathways explored by NRP1-KD RNA-seq data. Also, NRP1 KD models were tested in combination with chemotherapy drugs such as doxorubicin, docetaxel, and paclitaxel. Cell-viability assays were used to measure changes in IC_{50} after NRP1 inhibition. NRP1 CRISPR KD models demonstrated a decrease in cancer-stem/tumour-initiating capacity, migration, and invasion, without affecting proliferation compared to control cells. RNA-seq data of NRP1-KD cells revealed differential expression of stemness- and EMT-associated genes, and 14 candidates were prioritised for

subsequent molecular analysis. In 2D cultures the IC_{50} of the three chemotherapy drugs were unaffected by either Vesencumab or CRISPR knockdown; only siRNA sequence 5 produced an increase in chemosensitivity. In the next step, we will explore EMT plasticity in NRP1-KD RNA- seq data to uncover pathways potentially linking anti-invasive phenotypes to persistent chemoresistance observed in-vitro.

Biography:

Fatemeh Sanjabi is a third-year PhD student, working under the supervision of Dr. Brett Hollier at Queensland University of Technology. Her project focuses on investigating the role of Neuropilin-1 (NRP1) in preclinical models of claudin-low breast cancer and evaluating the impact of NRP1 inhibition on tumour progression and chemoresistance using in vitro and transcriptomic approaches.

Sayuri Herath
Prostate Cancer Research Centre-Queensland

CBL0137 as a Promising Therapeutic Strategy for Anaplastic, Treatment-Emergent Neuroendocrine Prostate Cancer

Treatment-emergent neuroendocrine prostate cancer (tNEPC) is a highly aggressive, anaplastic form of prostate cancer with no effective curative treatments. Accordingly, there is an urgent clinical need to find novel therapeutic targets and/or strategies to improve the survival outcomes of men harbouring tNEPC. Recent studies have identified overexpression of the facilitates chromatin transcription (FACT) complex in anaplastic tumours, and in particular those possessing a neuroendocrine phenotype. Recently, a curaxin-based molecule, CBL0137, has been shown to be an effective targeted-agent for inhibiting FACT function. Therefore, this study aims to assess the effect of CBL0137 as a novel targeted treatment for tNEPC, both as a monotherapy and as a co-therapy alongside the standard platinum-based chemotherapy, cisplatin and characterise the molecular consequences of CBL0137 by multi-omic profiling. By taking advantage of several clinically relevant ex-vivo patient-derived xenograft organoid (PDXO) models, anti-tumour activity of CBL0137 was evaluated and has further probed its therapeutic potential as a companion therapy with cisplatin. This project also conducts a comprehensive multi-omic investigation into the molecular mechanisms enacted by CBL0137. CBL0137 exhibits potent inhibitory effects across all the PDXO models tested, with calculated IC50 values in low micromolar ranges confirming its strong anti-tumour activity against tNEPC. Western blot analysis revealed a marked re-

duction in tumour proliferation and enhanced apoptotic mechanisms following CBL0137 treatment. Notably, combination treatment with cisplatin and CBL0137 resulted in improved therapeutic efficacy compared to either drug alone, indicating a potential beneficial effect that enhances overall treatment response. Mechanistically, CBL0137 upregulated p53-mediated tumour suppression, and induced cell cycle arrest via p21 in NEPC models. In conclusion, our findings confirmed that CBL0137 demonstrates robust anti-tumour activity across diverse NEPC PDXO models, significantly suppressing tumour proliferation and enhancing therapeutic efficacy when combined with cisplatin. The co-treatment consistently outperformed either drug alone, highlighting its potential as a more effective strategy for aggressive NEPC. Ongoing multi-omic profiling will further unravel the molecular consequences of CBL0137 treatment and support the development of improved therapeutic approaches for patients with anaplastic and treatment-resistant prostate cancer phenotypes.

Biography:

Sayuri Herath is a PhD candidate at Queensland University of Technology, Australia, investigating novel therapeutic targets and strategies to improve outcomes in poorly differentiated, highly aggressive anaplastic prostate cancer. Her research focuses on investigating the anti-tumour response of novel treatment strategies using ex-vivo organoids and co-targeting approaches to enhance platinum-based chemotherapy. She also explores the functional mechanisms and molecular consequences of novel treatment to better understand and manage advanced prostate cancer pathogenesis.

Xia Hu

Department of Emergency Surgery, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

ATRA upregulates OTUD6B to recruit CD8+ T cells to suppress colorectal liver metastasis

Introduction: Colorectal cancer (CRC) is the third most common cancer globally, with liver metastasis being the primary driver of poor prognosis. All-trans-Retinoic acid (ATRA), a first-line drug for hematological malignancies like acute promyelocytic leukemia (APL), exhibits potent immune-regulatory properties. Herein, we investigate how ATRA suppresses CRC liver metastasis by upregulating OTUD6B, focusing on its targeted regulatory mechanisms.

Methods: OTUD6B expression was analyzed via UALCAN and GSE datasets, with protein levels quantified by western blot in CRC cells and liver metastasis patient tissues. Stable OTUD6B- overexpressing/knockdown CRC lines were constructed with CCK-8, scratch, and Transwell assays evaluating proliferation, migration, and invasion. Liver metastasis models were established via splenic injection in nude mice (immune-deficient), OT-1 transgenic mice, and C57BL/6J mice (immune-competent). Flow cytometry, immunofluorescence, qPCR, ELISA, co- immunoprecipitation, and ubiquitination assays were used to validate cellular, immune, and molecular mechanisms.

Results: OTUD6B is upregulated in colorectal cancer (CRC). In CRC cells, it exerts no effect on proliferation but promotes cell migration and invasion. While

OTUD6B overexpression accelerates CRC liver metastasis in immunodeficient nude mice, it exerts an opposing effect in immunocompetent C57BL/6J mice by facilitating $CD8^+$ T cell infiltration. Mechanistically, OTUD6B exerts antitumor effects by upregulating CXCL11 to recruit $CD8^+$ T cells. It deubiquitinates and stabilizes DEAD-box helicase 5 (DDX5), which in turn resolves the RNA G- quadruplex (rG4) structure of signal transducer and activator of transcription 3 (STAT3), leading to STAT3 upregulation, enhanced CXCL11 transcription, and increased tumor-infiltrating $CD8^+$ T cells. Notably, all-trans retinoic acid (ATRA) enhances the protein expression of OTUD6B and DDX5 in CRC cells and suppresses CRC liver metastasis by upregulating OTUD6B, highlighting ATRA's potential as a targeted agent for precision CRC therapy.

Conclusions: ATRA suppresses CRC liver metastasis via OTUD6B-mediated stabilization of the DDX5/STAT3/CXCL11 axis and $CD8^+$ T cell recruitment, supporting its potential as a precision- targeted agent for CRC, with potential to stratify patients by OTUD6B expression for personalized therapy—combining immune modulation and targeted metastasis suppression.

Biography:

Xia Hu received her Bachelor's degree from Tongji Medical College, Huazhong University of Science and Technology in 2024. She is currently a second-year graduate student at Union Hospital, Wuhan. Her research during the master's program focuses on oncology, with specific dedication to investigating the role and mechanism of all-trans retinoic acid (ATRA) in colorectal cancer liver metastasis, aiming to contribute to the development of personalized therapeutic strategies for metastatic colorectal cancer.

Monavvar Andarva
Western Sydney University Australia, Australia

Innate immune mediator Group IIA secreted phospholipase A_2 regulates lipid droplets in prostate cancer

Inflammation plays a central role in tumour growth and metastasis. The molecular mediators of these pro-tumorigenic effects and their therapeutic potential remain under investigation. This study examines the role of human secreted phospholipase A2 (PLA2G2A, Group IIA sPLA2, hGIIA) in lipid droplet (LD) formation in prostate cancer (PCa) cells. hGIIA, aberrantly expressed in inflammatory diseases including cancer, hydrolyses phospholipids extracellularly and binds intracellular vimentin—an intermediate filament that scaffolds LDs. LDs actively regulate inflammation and support PCa progression by providing energy and fatty acids for membrane synthesis, with elevated LDs linked to therapy resistance and poor prognosis. We hypothesized that hGIIA influences LD formation via vimentin interaction, impacting energy metabolism in PCa.

We assessed LD formation using immunofluorescence microscopy in live and fixed DU145 cells treated with hGIIA. Vimentin's role was evaluated by CRISPR/Cas9-mediated knockout. LD markers (PLIN2, PLIN3), lipid metabolism markers (DGAT-1, FASN), and apoptosis markers (procaspase-3, cytochrome c) were also analysed.

Our findings show that Oleic acid strongly induced LD formation in wild-type DU145 cells but not in vimentin-knockout cells, indicating a blunted response

without vimentin. hGIIA induced LD accumulation at lower doses in wild-type cells, whereas knockout cells required higher doses. PLIN2 regulation followed similar patterns, while PLIN3 levels remained stable. These findings reveal that vimentin enhances cellular sensitivity to fatty acid and hGIIA-driven LD formation by modulating PLIN2, positioning vimentin as a key regulator of lipid storage dynamics and identifying hGIIA-mediated lipid metabolism as a potential therapeutic target in PCa.

Biography:

I am currently a third-year PhD candidate in the School of Medicine, Western Sydney University, Sydney, Australia, where my research focuses on novel therapeutic strategies for Secreted Phospholipase A2 type IIA enzyme (hGIIA) dependent diseases, including prostate cancer. My thesis project is conducted in collaboration with Filamon Ltd, a biotechnology start-up, and is partially supported by the NSW Government Office of Health and Medical Research Partnership Scholarship program. This partnership aims to identify methods of treating or preventing hGIIA- dependent conditions by targeting the non-catalytic activity of hGIIA—an innovative approach that may open new avenues for disease intervention.

My work involves confocal microscopy and AI dependant image analyses, High throughput cytokine assessment using Isolight technology and in vivo and in vitro evaluation of prostate cancer Tumour Microenvironment, combining both translational and basic science components to progress toward clinical applications.

Luo Jingyi

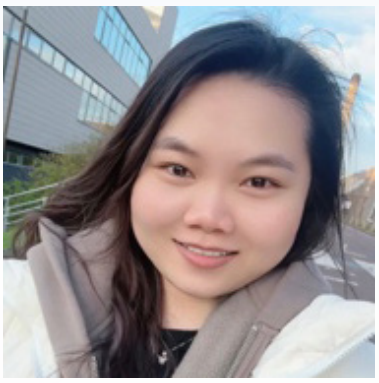
School of Biomedical Sciences, Faculty of Medicine, The University of Hong Kong, Hong Kong

Tubulin acetyltransferases target microtubule lumen via taxane-site anchors

The acetylation of α-tubulin Lys 40 residue by acetyltransferases is a critical post-translational modification that occurs within the microtubule lumen. Acetylated tubulin serves as a marker for micrometer-long stable microtubules and plays a critical role in mechanosensation and neurogenesis. However, how tubulin acetyltransferases target the luminal K40 in micrometer-long microtubules remains elusive. Here, we present the first high-resolution cryo-electron microscopy reconstructions and single-molecule characterization of the full-length tubulin acetyltransferase bound to the inner surface of microtubules with physiologically relevant substrates that mimic different stages in the tubulin acetylation reaction. Our structures reveal multivalent microtubule luminal recognition that one acetyltransferase catalytic core docks two α-tubulins, and its C-terminal domain (CTD) extends into the taxane-binding pockets of two β-tubulins. The anchors at the taxane-binding pockets facilitate tubulin acetyltransferases to access the microtubule lumen for the formation of a ternary complex with acetyl-CoA and unacetylated α-tubulin. After Lys 40 acetylation, the dissociation of CoA reduces the catalytic core's affinity for the luminalsurface, and the β-tubulin-anchored CTD allows the apo enzyme to remain near theluminal surface to load another acetyl-CoA for the subsequent enzyme turnover. Our model elucidates the multivalent interactions involving α- and β-tubulins for enzymes to access and acetylate the confined microtubule lumen.

Biography:

She is a fourth-year Ph.D. candidate at the University of Hong Kong researching the role of tubulin isotypes and post-translational modifications in regulating microtubule structure and function. Her innovative work in this area has been recognized through her selection for the prestigious NSFC Young Student Basic Research Program (PhD candidate) 2024, awarded by the National Natural Science Foundation of China.



Manting Wang
University of Oxford , UK

Develop A Preventative Vaccine for Ovarian Cancer

Introduction: Ovarian cancer (OC) is the leading cause of gynecologic cancer deaths, and the most common type is high-grade serous ovarian carcinomas (HGSOC). The fallopian tube is considered the most probable origin of most HGSOC. The treatments for OC are limited, and no preventative treatment for OC currently, so it's urgent to have a vaccine for ovarian cancer.

Methods: We used an in-silico analysis combining The Cancer Genome Atlas Program (TCGA) data and immunopeptidomic datasets to identify ovarian cancer-associated mutations and tumour-associated antigens (TAAs) peptides that cover most population. Immunogenicity tests of selected peptides were conducted to identify a potential vaccine for OC. Quality controls, including fluorescence-activated cell sorting (FACS) for dendritic cells (DCs) and naïve T cells, immunofluorescence (IF) and IL-12 ELISA for DCs, were used to validate the immunogenicity test result.

Results: 58 mutations peptides and 42 TAA peptides that achieve over 80% HLA class I population coverage were identified, and 20 peptide pools were generated. A high purity percentage of DC (98.7%) and naïve T cells (96%) were obtained from FACS for the immunogenicity test, and DCs were confirmed to be mature by FACS, IF and IL-12 ELISA. Peptide pools 5, 8, 9, 10, and 19 had the strongest

T-cell response and peptide pools 18, 14, 17, 20 and 2 induced a moderate T-cell response among all 20 peptide pools.

Conclusion and impact: The quality controls made the immunogenicity results convincing. The findings of the peptide pools' response showed promising peptide candidates for further evaluation.

Biography:

I am a second-year DPhil student in the University of Oxford in the UK, based on MRC Weatherall Institute of Molecular Medicine (WIMM). My project is to develop a vaccine for ovarian cancer on cancer immunology. Prior to my DPhil, I completed my MSc Cancer at University College London (UCL), and MBBS at Guangzhou Medical University.

Somin Woo

College of Pharmacy, Kyungpook National University, Daegu, Republic of Korea

Inhibition of VGCC and BKCa Channels by Trimebutine Maleate Suppresses Stemness and Drug Resistance in Ovarian Cancer Stem Cells

Ovarian cancer is the most lethal gynecologic malignancy, with a five-year survival rate of only 29% at stage IV. Despite initial responsiveness to platinum- and taxane-based chemotherapy, recurrence and drug resistance remain major clinical hurdles. Cancer stem cells (CSCs), which possess self-renewal and differentiation capabilities, contribute to these therapeutic failures via pathways such as Wnt/β-catenin and Notch. Hence, targeting CSCs is a critical strategy for durable therapeutic response. In this study, Trimebutine maleate (TM)—an FDA-approved drug for gastrointestinal disorders—was identified through high-throughput screening as a potential anti-CSC agent.

Sphere-forming CSC-like populations were enriched from A2780, SKOV3, and OVCAR3 ovarian cancer cells. TM's anti-proliferative effects were evaluated using CellTiter-Glo assays, while apoptosis and cell cycle distribution were analyzed via Annexin V/PI and PI staining followed by flow cytometry. Whole-cell patch clamp recordings were used to assess ion channel activity. Expression of CSC markers and key signaling components was measured using qRT-PCR and western blot. In vivo efficacy was assessed in A2780-SP xenograft mouse models.
TM selectively inhibited CSC viability while sparing bulk parental cancer cells.

It induced G0/G1 cell cycle arrest and significantly enhanced apoptosis in CSC populations. TM treatment also suppressed tumor growth in vivo without inducing systemic toxicity. Mechanistically, TM inhibited VGCC, sodium, and BKCa channel activity in CSCs, leading to disrupted calcium signaling. TM downregulated the expression of stemness-associated transcription factors OCT4 and SOX2 and attenuated Wnt/β-catenin signaling. Dual blockade of calcium and BKCa channels was crucial for TM's CSC-specific effects.

These findings indicate that TM effectively reduces ovarian CSC proliferation, stemness, and drug resistance through combined inhibition of VGCC and BKCa channels. TM holds promise as a repositioned therapeutic agent for overcoming recurrence and resistance in ovarian cancer treatment.

Biography:

Somin Woo is a Master's student in the Laboratory of Innovative Drug Development at the College of Pharmacy, Kyungpook National University, Republic of Korea. Her research focuses on discovering small-molecule inhibitors targeting cancer stem cells (CSCs) and therapeutic targets in cancer. She is also interested in AI-integrated drug discovery approaches, such as virtual screening and de novo molecular design. She aims to combine computational and experimental strategies to develop next-generation anticancer therapies.