4th MSC 2026

MACR INTERNATIONAL SCIENTIFIC CONFERENCE

Advancing Regional Cancer Research for Equitable Cancer Care

PROGRAMME BOOK

Hospital Sultan Abdul Aziz Shah (HSAAS),
Universiti Putra Malaysia, Serdang, Selangor, Malaysia

24th & 25th November 2026
8am-5pm

Organised by:

MACR Malaysian Association for Cancer Research

UPM UNIVERSITI PUTRA MALAYSIA BERILMU BERBAKTI

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4th MSC 2026

MACR INTERNATIONAL SCIENTIFIC CONFERENCE

Advancing Regional Cancer Research for Equitable Cancer Care

Venue



Address:
Auditorium Level 2, Menara Utara, Hospital Sultan Abdul Aziz Shah, UPM
Persiaran Mardi - UPM, 43400 Serdang, Selangor

Google map:
https://maps.app.goo.gl/8yfyx86iRxN2gvnc6

Parking for those coming with cars:
Paid parking space (beside the hospital)

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Platform: Grab, AirAsia Ride, Maxim
Pick-up/Drop-off Point:
Hospital Sultan Abdul Aziz Shah, UPM

4th MSC 2026
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Advancing Regional Cancer Research for Equitable Cancer Care

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Welcome Message



Yang Berbahagia Profesor Dr. Shamsul Bahri Hj. Md Tamrin
Deputy Vice-Chancellor
(Industry and Community Relations)
Universiti Putra Malaysia

It is my great pleasure to welcome all of you to the **MACR International Scientific Conference (4th MSC 2026)**, a remarkable gathering of minds united by a shared vision to advance healthcare innovation through science, technology, and collaboration.

The theme of this year's conference *"Advancing Regional Cancer Research for Equitable Cancer Care"* could not be timelier. Addressing the growing cancer burden across Asia, from early detection and drug discovery to treatment affordability and clinical access, presents complex, multifaceted challenges. No single discipline, institution, or nation can overcome them alone. It is only through regional and global interdisciplinary collaboration that we can truly transform scientific discovery into accessible, real-world solutions that benefit patients.

This conference embodies that spirit. Over the next two days, you will engage in dynamic discussions and explore new frontiers across our core conference tracks, including Asian cancer biology, new cancer drug discoveries, translational cancer medicine, cancer screening and prevention, and clinical trials in precision oncology. I am especially pleased to note the inclusion of dedicated forums on artificial intelligence in cancer care as well as generics and drug accessibility, which are crucial to reshaping both how we develop therapeutics and how we ensure equitable patient access.

Equally important is our focus on sustainable global partnerships, building networks that transcend borders, disciplines, and sectors. By integrating diverse expertise across research, clinical oncology, and policymaking, we can accelerate the translation of research into impactful healthcare outcomes—improving lives not only today, but for generations to come.

I would like to commend the organisers for creating a platform that bridges academia, industry, regulatory bodies, and clinical practice—a platform that nurtures young researchers while celebrating established leaders in the field. Through keynote addresses, expert panel forums, and scientific symposia, I am confident that the 4th MSC 2026 conference will inspire new ideas, collaborations, and innovations that will shape the future of oncology.

As we embark on this exciting journey together, let us reaffirm our shared commitment to drive transformative progress, to make cancer care more equitable, and to build a healthier world for all. Once again, welcome to the 4th MSC 2026 conference. I wish all participants a productive, enlightening, and inspiring conference ahead.

Thank you.

Welcome Message

Dear Esteemed Colleagues, Distinguished Guests, and Friends,

It is with the greatest pleasure and profound honour that we welcome you to the MACR International Scientific Conference **(4th MSC 2026)**, held on 24–25 November 2026 at Hospital Sultan Abdul Aziz Shah (HSAAS), Universiti Putra Malaysia.

This year's theme, *"Advancing Regional Cancer Research for Equitable Cancer Care",* reflects our shared aspiration to bridge disciplines, institutions, and borders in pursuit of meaningful clinical impact. Scientific discoveries reach their fullest potential when nurtured through collaboration, and it is this spirit of partnership that lies at the heart of the 4th MSC 2026.

Over the course of these two days, our programme brings together eminent speakers, innovative researchers, dedicated clinicians, industry leaders, and passionate young scientists. Spanning Asian cancer biology, new cancer drug discoveries, translational cancer medicine, cancer screening and prevention, and clinical trials in precision oncology, alongside dedicated forums on artificial intelligence in cancer care and drug accessibility, these sessions promise to inspire new ways of thinking and open doors to lasting regional and global collaborations.

We extend our deepest gratitude to the Organising Committee, whose dedication and hard work have shaped every detail of this conference, as well as our sponsors whose generous support has made the 4th MSC 2026 possible. Our sincere thanks also go to our distinguished speakers, committed delegates, and tireless volunteers too. Your contributions form the very essence of this gathering.

May the 4th MSC 2026 be a catalyst for knowledge, discovery, and innovation, and may the connections formed here continue to flourish long after the closing session.

Thank you.



Dr. Jonathan Lim Chee Woei

Co-Chair, 4th MSC

Faculty of Medicine and Health Sciences
Universiti Putra Malaysia



Professor Dr. Johnson Stanslas

Advisor, 4th MSC

President, Malaysian Association for Cancer Research

Faculty of Medicine and Health Sciences
Universiti Putra Malaysia



Dr. Ooi Der Jiun

Co-Chair, 4th MSC

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MAHSA University

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PROGRAMME SCHEDULE

24th November 2026 (DAY 1)

0800	**Registration**
0830	**Welcome & Opening Address**
0900	**Cancer Biology**
	Plenary Talk by Prof. Dr. Ian Charles Paterson *Universiti Malaya, Malaysia* **Head and Neck Cancer in South East Asia** *Moderator: AP Dr. Audrey Yong Chee Hui*
0945	**Cancer Drug Discovery**
	Invited Talk by Dr. Leroy Lowe, *The Halifax Institute, Canada* **Engineered Adjunct Nutrition, Molecular Mechanisms and Cancer** *Moderator: AP Dr. Nur Fariesha Md Hashim*
1010	**Tea Break | Poster Viewing/Judging | Visiting Sponsor Booth**
1040	**Special Forum 1 - Artificial Intelligence in Cancer Care**
1140	**Parallel Oral Presentation - Track 1 & 2**
1300	**Lunch Break**
1415	**Sponsored Talk 1 - Yokogawa** *Moderator: Dr. Jonathan Lim Chee Woei*
1500	**Cancer Drug Discovery**
	Special Talk 1 by Dr. Muhammad Asyraf Abduraman *NatureCeuticals Sdn. Bhd.* **Nuvastatic for Cancer-Related Fatigue: From Preclinical Development to Phase III Clinical Evidence** *Moderator: AP Dr. Mohd Razif Shahril*
1525	**Cancer Biology**
	Invited Talk by Assoc. Prof. Dr. Nurul Syakima Ab Mutalib *Universiti Kebangsaan Malaysia, Malaysia* **Mapping the Colorectal Cancer Methylome: Biological Insights and Translational Opportunities** *Moderator: Dr. Chua Eng Wee*
1550	**Invited Talk by Dr. Lim Kue Peng** *Cancer Research Malaysia, Malaysia* ***Therapeutic Cancer Vaccines Reprogram Distinct Tumour Immune States to Overcome Immune Resistance Across Solid Tumours*** *Moderator: Dr. Noraina Muhamad Zakuan*
1615	**Invited Talk by Dr. Florence Wong Yoke Fui** *National Cancer Institute, Malaysia* **Clinical Trials of New Cancer Drugs** *Moderator: Dr. Nurul Akmaryanti Abdullah*
1640	**Tea Break | End of Day 1**
1830	**Gala Dinner**

PROGRAMME SCHEDULE

25th November 2026 (DAY 2)

0800	**Registration**
0900	**Translational Cancer Medicine**
	Plenary Talk by Assoc. Prof. Dr. Sunny Wong *Nanyang Technological University, Singapore* ***The Gut Microbiome in Precision Oncology: From Biology to Cancer Management*** *Moderator: Dr. Lau Su Yin*
0945	**Invited Talk by Dr. Nirmal Rajasekaran** *CURACLE Inc, South Korea* ***From Bench to Bedside: Real-World Lessons in Oncology Drug Development*** *Moderator: Ts Dr. Liew Fong Fong*
1010	**Invited Talk by Prof. Dr. Bassim Irheim Mohammad** *University of Al-Qadisiyah, Iraq* ***Toward Precision Medicine in Chronic Myeloid Leukemia: Integrated Molecular Profiling of BCR-ABL1, ASXL1 and MicroRNAs and Variability in TKI Response in an Iraqi Cohort (Recording)*** *Moderator: Dr. Oh Lixian*
1035	**Tea Break | Poster Viewing/Judging | Visiting Sponsor Booth**
1105	**Parallel Oral Presentation - Track 3 & 4**
1230	**Lunch Break**
1400	**Clinical Trials of New Cancer Drugs / Precision Oncology**
	Plenary Talk by Dr. Yuta Maruki *ATLAS, National Cancer Center, Japan* ***Unlocking the Future of Biliary Tract Cancer Treatment in Asia: Genomics, Precision Medicine, and Beyond*** *Moderator: Dr. Sim Pei Fang*
1445	**Invited Talk by Dr. Noor Shuhada Yop Ahmad** National Cancer Institute (IKN), Malaysia **Evidence-Based Integrative Supportive Care in Oncology: Research, Safety Protocols, and Practice at the National Cancer Institute (IKN)** *Moderator: Dr. Chua Eng Wee*
1535	**Special Forum 2 - Generics and Drug Accesibility**
1635	**Announcement of MACR Grant Awardee**
1645	**Special Sharing Session for MACR Grant Awardee**
1705	**Closing & Award Ceremony**
1830	**Tea Break | End of Conference**

4th MSC 2026

MACR INTERNATIONAL SCIENTIFIC CONFERENCE

Advancing Regional Cancer Research for Equitable Cancer Care

PLENARY SPEAKERS



PROF. DR. IAN PATERSON
(Cancer Biology)
Universiti Malaya
Malaysia



DR. YUTA MARUKI
(Medical Oncology)
ATLAS, National Cancer Center
Japan



AP DR. SUNNY WONG
(Gastroenterology)
Nanyang Technological University
Singapore

INVITED SPEAKERS



PROF. DR. BASSIM IRHEIM MOHAMMAD
(Translational Cancer Medicine)
University of Al-Qadisiyah
Iraq



AP DR. NURUL SYAKIMA AB MUTALIB
(Cancer Biology)
Universiti Kebangsaan Malaysia
Malaysia



DR. LEROY LOWE
(Cancer Biology)
The Halifax Institute
Canada



DR. LIM KUE PENG
(Cancer Immunology & Immunotherapy)
Cancer Research Malaysia
Malaysia



DR. NIRMAL RAJASEKARAN
(Translational Cancer Medicine)
CURACLE Inc
South Korea



DR. NOOR SHUHADA YOP AHMAD
(Traditional & Complementary Medicine)
National Cancer Institute
Malaysia



DR. FLORENCE WONG YOKE FUI
(Clinical Oncology)
National Cancer Institute
Malaysia

SPECIAL TALK SPEAKER



DR. MUHAMMAD ASYRAF ABDURAMAN
(Chief Technical and Regulatory Office)
NatureCeuticals Sdn. Bhd.
Malaysia

FORUM 1

Artificial Intelligence in Cancer Care

Panelists:



AP DR. SUNNY WONG
(Gastroenterology)
Nanyang Technological University
Singapore



AP DR. SUNIZA JAMARIS
(Consultant Breast Oncoplastic Surgeon)
Universiti Malaya
Malaysia



TS. DR. AFZAN ADAM
(AI Technologist)
Universiti Kebangsaan Malaysia
Malaysia

Moderator:



DR. OOI DER JIUN
MAHSA University
Malaysia

FORUM 2

Generics and Drug Accessibility

Panelists:



DR. LOH C-KHAI
(Paediatric Oncology & Haematology)
Universiti Kebangsaan Malaysia
Malaysia



DR. OOI PO LIN
(Clinical Oncology)
Universiti Malaya
Malaysia



MRS. NIK SHAMSIAH NIK SALLEH
(Generic Medicines)
National Pharmaceutical Regulatory Agency
Malaysia

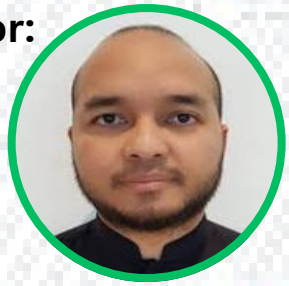


MS. PRIYA DARSHINI ASOKAN
(Pharmaceutical Biotechnology)
Duopharma Biotech Berhad
Malaysia



MR. RAJIS SANTHIRASEGAR
(Vice President, Oral Health and Oncology)
Pharmaniaga Berhad
Malaysia

Moderator:



DR. MOHAMAD SHAFIQ AZANAN
Universiti Malaya
Malaysia

Plenary Speaker

Head and Neck Cancer in South East Asia



PROF. DR. IAN PATERSON

Universiti Malaya, Malaysia

Ian Paterson is a professor of cancer biology at the University of Malaya. After obtaining his PhD in biochemistry and molecular he developed his interest in head and neck cancer at the University of Bristol, UK, where he worked for more than 10 years. For the past 15 years, he has worked at the University of Malaya where he continues to focus on the molecular basis of head and neck cancers, including oral, oropharyngeal and nasopharyngeal carcinomas.

Abstract

Head and neck squamous cell carcinomas (HNSCCs) comprise a biologically and clinically heterogeneous group of malignancies arising from anatomically distinct sites, including the oral cavity, oropharynx, and nasopharynx. Collectively, these cancers represent a substantial global health burden, with considerable geographic variation in incidence, anatomical distribution, and aetiological factors. Despite their anatomical proximity, HNSCCs arising at different sites represent distinct biological entities, characterised by differences in cellular origin, molecular alterations, pathological features, and clinical behaviour.

In South and Southeast Asia, oral cavity cancer and nasopharyngeal carcinoma (NPC) represent particularly important disease burdens, reflecting distinctive patterns of environmental exposure, lifestyle, viral infection, and host susceptibility. Oral cavity cancers are predominantly associated with tobacco and alcohol exposure, whereas oropharyngeal carcinomas are increasingly associated with human papillomavirus (HPV) infection. NPC, in contrast, has a strong association with Epstein–Barr virus (EBV), particularly in endemic populations in China and Southeast Asia.

In this talk, I will examine the diverse aetiologies, cellular origins, histopathological features, and genomic landscapes of HNSCC. I will also explore the tumour microenvironment and the complex interactions between malignant cells and non-malignant cell populations, including fibroblasts, T cells, and macrophages. Finally, I will discuss how these cellular interactions contribute to tumour development and behaviour and may provide opportunities for the development of novel therapeutic strategies.

Plenary Speaker

The Gut Microbiome in Precision Oncology: From Biology to Cancer Management



ASSOCIATE PROFESSOR DR SUNNY WONG

Director, Centre for Microbiome Medicine

Assistant Dean (Academic Medicine),
Lee Kong Chian School of Medicine
Nanyang Technological University, Singapore

Dr Sunny Wong is an Associate Professor at the Lee Kong Chian School of Medicine, Nanyang Technological University Singapore, where he also serves as Assistant Dean in Academic Medicine. He received his MBChB at the Chinese University of Hong Kong and completed his PhD at the University of Oxford. He completed his specialist training in gastroenterology at the Prince of Wales Hospital in Hong Kong. Joining NTU Singapore in 2021, he founded the Centre for Microbiome Medicine in 2022 and has been serving as Director since then. Dr Wong's research focuses on the gut microbiome and host–microbe interactions in digestive diseases and cancers. He has published more than 300 peer-reviewed articles in journals including Gastroenterology, Gut, and Nature Communications. His work has been widely recognized through multiple awards, including the Sir David Todd Lectureship (2020, the Asia Pacific Digestive Week Emerging Leaders Lectureship (2021), Seah Cheng Siang Memorial Lectureship (2025), and Clarivate Analytics Highly Cited Researcher recognitions in 2023, 2024, and 2025.

Abstract

The gut microbiome is emerging as both a diagnostic signal and a therapeutic lever in gastrointestinal cancers. Yet translating correlative microbial associations into clinical tools requires resolving two questions: where cancer-relevant microbes reside, and how they can be measured or modified for patient benefit. This talk will trace a translational pipeline from spatial biomarker discovery to microbial intervention.

First, I will show that microbial location and biology matter in the cancer. The colorectal tumour microbiome is spatially structured — within tumours, across the tumour field, and between right- and left-sided cancers — and the faecal microbiome used in screening is partly a readout of this spatial niche. Multi-compartment sampling (stool, aspirate, mucosal tissue) reveals that specimen choice can dominate the observed signal, with direct implications for the design and interpretation of stool-based biomarkers. Against current screening tests, stool microbial markers — including Fusobacterium nucleatum and multi-marker panels — offer competitive accuracy for colorectal cancer and adenoma detection.

Second, I will discuss modulation of the microbiome as a strategy for prevention and adjunctive treatment. Next-generation probiotics and live biotherapeutics — including Roseburia intestinalis and other candidate strains — can suppress tumourigenesis. In established cancer, the microbiome shapes chemotherapy efficacy and toxicity (5-FU, irinotecan, oxaliplatin) and is a determinant of immune checkpoint inhibitor response, motivating signature-guided approaches such as Gut OncoMicrobiome Signatures.

Finally, I will address the translational challenges — causality, population specificity, and standardization — that must be overcome to deliver microbiome-informed precision oncology for Asian populations.

Plenary Speaker

Unlocking the Future of Biliary Tract Cancer Treatment in Asia: Genomics, Precision Medicine, and Beyond



DR. YUTA MARUKI

Staff Physician
Department of Hepatobiliary and Pancreatic Oncology / Department of Experimental Therapeutics, National Cancer Center Hospital, Tokyo, Japan

Yuta Maruki, MD, PhD, is a staff physician in the Departments of Hepatobiliary and Pancreatic Oncology and Experimental Therapeutics at the National Cancer Center Hospital, Tokyo, Japan. His research focuses on precision oncology for hepatobiliary and pancreatic cancers, integrating cancer genomics, translational research, and trials. He has led multicenter studies, including the Japanese PRELUDE study and the pan-Asian CHOICE study, investigating FGFR2 alterations in cholangiocarcinoma. His work aims to expand biomarker-guided treatment and improve access to genomic medicine across Asia.

Abstract

Biliary tract cancer (BTC) represents a major clinical challenge in Asia, where disease burden, etiological factors, and access to molecular testing vary considerably across countries. Although systemic chemotherapy combined with immune checkpoint inhibition has advanced first-line treatment, outcomes remain unsatisfactory for many patients. The integration of cancer genomics into clinical practice offers an opportunity to reshape BTC management through more precise patient selection and biomarker-directed therapies.

Molecular profiling has revealed substantial heterogeneity across BTC anatomical subtypes and geographic populations. Actionable alterations, including FGFR2 fusions or rearrangements, IDH1 mutations, and HER2 alterations, have created new therapeutic opportunities. Findings from the Japanese PRELUDE study and the multinational Asian CHOICE study illustrate both the value of characterizing FGFR2 alterations in regional patient populations and the practical challenges of implementing comprehensive genomic profiling. Such efforts are essential to understanding the relevance of molecular targets across diverse Asian settings.

Targeted agents against FGFR2 and IDH1, along with emerging HER2-directed approaches, are expanding treatment options beyond conventional chemotherapy. Meanwhile, immunotherapy-based combinations and investigational strategies targeting resistance mechanisms may further transform the treatment landscape. Optimizing the timing and selection of molecular tests, interpreting genomic results, and addressing acquired resistance will be increasingly important as the therapeutic armamentarium grows.

Looking ahead, progress in BTC will depend not only on drug development but also on equitable access to high-quality molecular diagnostics, regional clinical trial networks, and collaboration across Asian countries. Integrating genomic discovery with practical implementation can help translate precision oncology into meaningful benefits for more patients with BTC.

Invited Speaker

Toward Precision Medicine in Chronic Myeloid Leukemia: Integrated Molecular Profiling of BCR-ABL1, ASXL1 and MicroRNAs and Variability in TKI Response in an Iraqi Cohort



PROF. DR. BASSIM IRHEIM MOHAMMAD

Professor
University of Al-Qadisiyah, Iraq

Prof. Dr. Bassim Irheim Mohammad is a Consultant Physician and Professor at the College of Medicine, University of Al-Qadisiyah, Iraq. He holds an MBChB, an MSc and a PhD in Medicine and Clinical Pharmacology. With extensive experience in medicine, clinical pharmacology and precision medicine, Prof. Mohammad's academic and research interests focus on translating molecular and genomic insights into individualized patient care. His research encompasses pharmacogenomics, epigenomics, multi-omics, drug-response variability and the application of emerging molecular approaches to cancer, cardiovascular, and metabolic diseases. He has authored more than 80 scientific publications and has made significant contributions to postgraduate medical education, research supervision and curriculum development. He previously served as the Founder and Dean of the College of Pharmacy at the University of Al-Qadisiyah. Prof. Mohammad is committed to advancing precision medicine through research, education, and clinical practice with ultimate goal of translating scientific discoveries into meaningful improvements in patient care.

Abstract

Background: Resistance or suboptimal response to tyrosine kinase inhibitors (TKIs) remains a clinically important challenge in chronic myeloid leukemia (CML). This study evaluated multiple molecular determinants of treatment outcome in an Iraqi patient cohort.

Methods: Fifty-one Philadelphia chromosome–positive CML patients receiving TKI therapy at Al-Diwaniyah Teaching Hospital, Iraq, were enrolled between September 2024 and February 2025. BCR-ABL1 transcript variants were characterized by RT-PCR. T315I and E255K kinase-domain mutations were screened using allele-specific PCR and ARMS-PCR, respectively. The ASXL1 c.1934dupG mutation was analyzed by PCR and bidirectional Sanger sequencing. miR-21 and miR-451 expression was quantified by RT-qPCR.

Results: The cohort was predominantly male (56.9%), with a mean age of 49.6 ± 14.6 years; 96.1% had chronic-phase disease. The b3a3 transcript was the predominant variant, detected in 78.4% of patients. T315I was identified in one patient (2.0%) and was associated with subsequent relapse, whereas E255K was not detected. ASXL1 c.1934dupG was present in two patients (3.9%). TKI type was significantly associated with treatment response ($p = 0.004$). Approximately 81.5% of imatinib-treated patients achieved a good response, while 83.4% of bosutinib-treated patients had poor outcomes. Neither transcript variant, ASXL1 status, nor miR-21/miR-451 expression significantly correlated with response category. miR-21 expression was significantly higher than miR-451 expression ($p < 0.05$).

Conclusion: In this Iraqi cohort, treatment history was the principal correlate of response, whereas transcript variants, ASXL1 status, and single-time-point microRNA expression were not predictive. The detection of T315I in a relapsing patient supports routine kinase-domain mutation analysis during treatment failure and highlights the need for larger longitudinal studies.

Invited Speaker

From Bench to Bedside: Real-World Lessons in Oncology Drug Development



DR. NIRMAL RAJASEKARAN

Senior Manager, Clinical Operation Team
CURACLE Inc, South Korea

Nirmal Rajasekaran, Ph.D. is an Oncology Drug Development and Translational Therapeutics specialist with more than 10 years of industry experience spanning nonclinical pharmacology, GLP toxicology, and Phase I–IIb clinical development. He currently serves as Senior Manager at Curacle Inc., Seoul, where he leads the clinical development of CU06, a Phase 2b diabetic macular edema program. Previously, Dr. Rajasekaran served as Head of Drug Development, Oncology at Abion Inc., where he led global medical strategy and operations for ABN401, a selective MET inhibitor, through multinational Phase II trials in Korea, the United States, and Taiwan. He also partnered with Johnson & Johnson on a combination study of ABN401 and lazertinib for MET-driven non-small-cell lung cancer. His expertise includes oncology drug development, translational pharmacology, clinical development, regulatory submissions, and emerging therapeutic modalities including oral SERDs, siRNA-LNP platforms, and PROTACs. He has authored more than 19 peer-reviewed publications and is an inventor on two oncology patent filings. Dr. Rajasekaran holds a Ph.D. in Pharmacy from Seoul National University.

Abstract

The translation of promising oncology drug candidates from preclinical research into clinical development is a complex and multidisciplinary process. Successful development requires the integration of pharmacological efficacy, pharmacokinetics and pharmacodynamics (PK/PD), toxicology, biomarkers, clinical strategy, regulatory requirements, and operational execution.

This presentation describes key considerations and practical experiences in advancing oncology drug development programmes from preclinical studies to early-phase clinical trials. Particular emphasis is placed on integrating pharmacological, PK/PD, GLP toxicology, and biomarker data to support critical development decisions, including candidate selection, clinical dose selection, and early clinical study design.

Examples from targeted small-molecule programmes, including the selective MET inhibitor, as well as emerging therapeutic approaches such as oral selective estrogen receptor degraders (SERDs), PROTACs, and siRNA–lipid nanoparticle (LNP) platforms, illustrate the opportunities and challenges associated with translational drug development.

The practical considerations involved in advancing multinational development programmes are also addressed, including CRO management, CMC coordination, regulatory interactions, clinical trial execution, and preparation of IND and IMPD submissions. The MET inhibitor development programme, including its combination strategy with an EGFR tyrosine kinase inhibitor (EGFR-TKI) in MET-driven non-small-cell lung cancer, is presented as a case study demonstrating the integration of scientific and clinical development strategies.

Collectively, these experiences highlight the importance of a coordinated translational development framework that effectively connects preclinical evidence with clinical strategy and execution. Such an integrated approach may facilitate more efficient development of promising oncology therapies and support their advancement toward meaningful clinical benefit for patients.

Invited Speaker

Mapping the Colorectal Cancer Methylome: Biological Insights and Translational Opportunities



DR. NURUL SYAKIMA AB MUTALIB

Associate Professor
Universiti Kebangsaan Malaysia, Malaysia

Dr Nurul Syakima Ab Mutalib is an Associate Professor at the Department of Biological Sciences and Biotechnology, Faculty of Science and Technology, Universiti Kebangsaan Malaysia. Her research focuses on cancer genomics, epigenetics and translational bioinformatics, with particular emphasis on colorectal cancer. She has published over 130 peer-reviewed articles and has an h-index of 42. Her research achievements have been recognised through inclusion among the World's Top 2% Scientists in 2024 and the World's Top 5% Scientists in 2025.

Abstract

Colorectal cancer is a molecularly heterogeneous disease shaped by both genetic and epigenetic alterations. Among these, DNA methylation plays an important role in regulating gene expression, tumour development and disease progression. Characterising the colorectal cancer methylome may therefore provide biological insights and support the discovery of clinically relevant biomarkers. This talk will highlight our research on genome-wide DNA methylation patterns in matched colorectal tumour and adjacent normal tissues from Malaysian patients. Using array-based methylation profiling and reproducible R/Bioconductor analytical workflows, we identified differentially methylated positions and regions associated with colorectal cancer.

Our studies have examined methylation changes involving protein-coding genes, microRNA-associated regions, ATP-binding cassette transporters and pathways linked to tumour development and progression. We have also explored methylation patterns according to clinicopathological characteristics, including tumour stage and anatomical location. The talk will discuss the biological interpretation of these methylation changes and the challenges involved in translating high-dimensional epigenomic findings into meaningful biomarkers. These challenges include tumour heterogeneity, limited subgroup sizes, batch effects, multiple-testing correction and the need for independent validation. Candidate markers are therefore prioritised according to methylation effect size, consistency, genomic context, functional relevance and potential clinical utility.

Our work also extends beyond biomarker discovery through the development of interactive R/Shiny platforms and artificial intelligence-assisted approaches for methylation-data visualisation, interpretation and prioritisation. Collectively, these studies demonstrate how mapping the colorectal cancer methylome can improve understanding of tumour biology and reveal opportunities for disease classification, biomarker development and precision oncology. However, experimental validation and evaluation in larger independent cohorts remain essential before clinical translation.

Invited Speaker

Evidence-Based Integrative Supportive Care in Oncology: Research, Safety Protocols, and Practice at the National Cancer Institute



DR. NOOR SHUHADA YOP AHMAD

Head,
Traditional & Complementary Medicine Unit
National Cancer Institute, Malaysia

Dr. Noor Shuhada Yop Ahmad is a Medical Officer and Head of the Traditional and Complementary Medicine (T&CM) Unit at Institut Kanser Negara (IKN), Malaysia. Graduating with a Medical Degree from Hassanuddin University and an MSc in Acupuncture from the International Medical University (IMU), she brings over a decade of clinical experience in anesthesiology, intensive care, and cardiothoracic anesthesiology across Hospital Tengku Ampuan Afzan (HTAA) Kuantan, and Hospital Putrajaya into supportive oncology care. Her current leadership and research focus on establishing evidence-based integrative supportive care, and clinical safety protocol for cancer patients utilizing T&CM alongside conventional therapies.

Abstract

Background: Traditional and Complementary Medicine (T&CM) utilization is prevalent among cancer patients throughout Asia. Within Ministry of Health Malaysia (KKM) facilities, particularly the National Cancer Institute (IKN), T&CM operates strictly within a regulated supportive care framework. Interventions including herbal therapy and acupuncture are indicated solely for symptom management, toxicity reduction, and enhancing quality of life, rather than as serving a primary anti-cancer treatment or disease-modifying therapies.
Objective & Safety Protocols: To deliver safe, evidence-based integrative supportive oncology while safeguarding patient safety and preserving the integrity of active, conventional oncology protocols, Institut Kanser Negara (IKN) enforces supportive care model and safety monitoring framework. Patients receiving herbal modalities undergo systematic laboratory monitoring—specifically baseline and serial Full Blood Count (FBC), Renal Profile (RP), and Liver Function Test (LFT) evaluations—to proactively screen for potential organ toxicity and adverse events. Concurrently, non-pharmacological modalities such as acupuncture offer valuable, non-systemic supportive avenues that complement pharmacological cancer treatments without introducing systemic drug interference.
Evidence & Clinical Research: Retrospective studies conducted at IKN demonstrate the clinical utility and safety profile of integrated T&CM interventions. Key findings include heavy metal analysis and safety evaluations of adjunct herbal formulations in breast and advanced lung cancer cohorts. Furthermore, clinical trials on Chinese herbal medicine for radiation-induced xerostomia, and acupuncture for Chemotherapy-Induced Nausea and Vomiting (CINV)—highlight effective symptom relief options that preserve treatment quality of life.
Conclusion & Future Directions: Evaluating T&CM modalities like acupuncture and herbal formulations presents unique methodological challenges in standardization, control group selection, and clinical trial design; however, IKN's institutionalized framework successfully mitigates unsafe self-medication by delivering transparent, regulated supportive care. Bridging supportive T&CM with precision oncology opens vital avenues for collaborative research. Systematic clinical trials evaluating standardized symptom control and non-pharmacological interventions can validate T&CM's role in mitigating treatment-related toxicities, ultimately enhancing patient adherence, reducing regimen interruptions, and optimizing overall therapeutic outcomes in modern cancer care.

Invited Speaker

Engineered Adjunct Nutrition, Molecular Mechanisms and Cancer



DR. LEROY LOWE

Executive Director
The Global Research Action Network for Discovery (GRAND)
www.grandscience.org

Dr. Leroy Lowe is a cancer biologist and international research leader with expertise in carcinogenesis, the effects of complex, low dose chemical exposures on cancer development and therapeutic treatment resistance. He co-founded Getting to Know Cancer and directed the Halifax Project, which involved 350 scientists across 31 countries working in 24 interdisciplinary teams. His work received international recognition for cancer-prevention leadership, and he is now focused on how nutrition can be used to support cancer therapy. At the same time, he is currently building the Global Research Action Network for Discovery (GRAND) to enable and accelerate global research collaboration.

Abstract

Clinical guidelines position nutrition in cancer care as supportive, but practice has moved past that description. Ketogenic and fasting-mimicking regimens are given to lower insulin and IGF-1 and attenuate PI3K-AKT-mTOR signaling, substrate-restricted diets are matched to tumor genotype, and high-dose antioxidants are discouraged on mechanistic grounds. These are pharmacological claims about named pathways, made by a field that has not adopted pharmacological methods.

Yet a systematic review of 252 randomized dietary trials in cancer found direct cancer measurements as the primary endpoint in just 8% of trials. The studies carrying the strongest human evidence fare no better. None reports composition at a level permitting reproduction, none declares in advance what magnitude of change would constitute success, and the tumor is sampled in just two trials. Without measured engagement, such trials cannot distinguish a mechanism that is wrong from one that was never delivered. Accordingly, a ten-point specification standard is set out for trials making a mechanistic claim and applied. Single interventions provoke compensation by documented routes. Because dependencies are distributed across subpopulations and signaling networks reroute around single-node inhibition, the operative variable is the number of mechanisms addressed rather than affinity at any one of them, which inverts the potency logic of drug development. A broad spectrum of targets is therefore the indicated response to the distributed dependencies of advanced cancers.

Three constraints that might prevent multi-targeting do not stand in the way. Metabolic load is consumed by concentrated exposure rather than agent count, the in vitro benchmark driving enhanced formulation is the wrong target, and multi-component dietary trials with survival endpoints have run under ethics committee oversight alone. Nonetheless, no registered nutritional trial in cancer combines three or more food-derived agents with a cancer endpoint. A randomized trial is therefore proposed that could be undertaken without delay. It would involve standard of care with and without a specified multi-component regimen at conventional dietary exposure, with overall survival as the primary endpoint.

Invited Speaker

Clinical Trials of New Cancer Drugs



DR. FLORENCE WONG YOKE FUI

Clinical Oncologist
Head of Clinical Research Centre,
National Cancer Institute, Malaysia

Dr Wong obtained her MBBS year 2005 and Master of Clinical Oncology year 2014 from University Malaya. She is currently held the post of Clinical Oncologist and Head of Clinical Research Center for National Cancer Institute, Malaysia. Dr Wong involved in multiple clinical trials and awarded as National Top Recruiter year 2018. She is the developmental group for Stereostatic Body Radiotherapy (SBRT) and trainer for Master of Clinical Oncology. Year 2024, appointed as panel of expert (POE) for First in Human (FIH) study for Ministry of Health. Year 2025, as panel of expert for PAGA ESMO Hepatocellular Cancer Management. Her special interest is in breast, lungs, head and neck, hepatobiliary, neuroendocrine, colorectal cancers.

Abstract

Oncology clinical trials are rapidly evolving from traditional trial-and-error chemotherapy tests into precise, biomarker-driven studies. Recent clinical trials for new cancer drugs focus heavily on precision medicine, targeted therapies, immunotherapies, and antibody-drug conjugates (ADCs). Crucially, this focus now spans every stage of the disease rather than concentrating solely on advanced cancers.

Here, I will share some key breakthroughs and featured clinical trials from this year.

Hard-to-Treat Pancreatic Cancer (RASolute 302 / Abstract LBA5): Daraxonrasib doubled overall survival and progression-free survival compared to chemotherapy in previously treated metastatic pancreatic ductal adenocarcinoma (PDAC), showing a 60% reduction in the risk of death.

Invited Speaker

Therapeutic Cancer Vaccines Reprogram Distinct Tumour Immune States to Overcome Immune Resistance Across Solid Tumours



DR. LIM KUE PENG

Head, Cancer Immunology & Immunotherapy Unit
Cancer Research Malaysia, Malaysia

Dr. Kue Peng Lim is the Head of Cancer Immunology and Immunotherapy at Cancer Research Malaysia, where she leads translational research in cancer vaccine development. She obtained her PhD from the University of Bristol and has over 20 years of experience in cancer biology and immunotherapy. She has secured competitive research funding from national and international agencies and is an inventor on three patents. Her work focuses on developing therapeutic cancer vaccines and advancing them toward clinical translation. She is a recipient of the L'Oréal-UNESCO For Women in Science Award and the Top Research Scientist Malaysia Award.

Abstract

Immune resistance remains a major barrier to effective cancer immunotherapy and can arise through diverse mechanisms, including defective antigen presentation, immunosuppressive tumour microenvironments, and failure to establish durable anti-tumour immunity. We hypothesised that therapeutic cancer vaccination can overcome these distinct immune barriers by reprogramming tumour-immune interactions and restoring effective anti-tumour responses. To investigate this, we evaluated the immunological impact of antigen-specific therapeutic vaccination across multiple solid tumour models representing diverse immune states, including immune-hot but antigen presentation-deficient nasopharyngeal carcinoma (NPC), tolerogenic hepatocellular carcinoma (HCC), and immune checkpoint blockade (ICB)-responsive and resistant lung cancer.

In NPC, we identified downregulation of the transcription factor NLRC5 as a key driver of impaired antigen processing and presentation, resulting in reduced MHC-I expression and immune evasion. Peptide-trained T cells restored the expression of NLRC5, TAP1 and B2M, leading to recovery of antigen presentation capacity and enhanced tumour cell recognition in vitro. In HCC, antigen-specific DNA vaccination expanded effector memory $CD8^+$ T cells while reducing tumour-associated macrophages and regulatory T cells, thereby remodelling the suppressive tumour microenvironment and improving tumour control. Similarly, in lung cancer, DNA vaccination synergised with ICB by promoting tumour-infiltrating $CD4^+$ Th1 responses and enriching stem-like $CD8^+$ T cells associated with sustained anti-tumour immunity, particularly in tumour microenvironments permissive to immune activation.

Collectively, our findings demonstrate that therapeutic cancer vaccines exert broader biological effects beyond T-cell priming by restoring antigen presentation, reversing immune suppression, and promoting durable anti-tumour immunity across biologically distinct tumour types. These results highlight tumour immune reprogramming as a key mechanism underlying vaccine efficacy and support the development of therapeutic cancer vaccines as a versatile strategy to overcome immune resistance and enhance responsiveness to cancer immunotherapy.

Special Talk Speaker

Nuvastatic for Cancer-Related Fatigue: From Preclinical Development to Phase III Clinical Evidence



DR. MUHAMMAD ASYRAF ABDURAMAN

Chief Technical and Regulatory Officer,
NatureCeuticals Sdn. Bhd.
General Manager, Eman Biodiscoveries Sdn. Bhd.

Dr. Muhammad Asyraf Abduraman is the Chief Technical and Regulatory Officer at NatureCeuticals Sdn. Bhd. and General Manager of Eman Biodiscoveries Sdn. Bhd. He holds a PhD in Pharmacology from Universiti Sains Malaysia (USM). He has extensive experience in pharmaceutical and botanical drug development, clinical research, regulatory affairs, and Good Manufacturing Practice (GMP). His areas of expertise include preclinical pharmacology, toxicology, pharmacokinetics, drug–drug and herb–drug interaction studies, particularly cytochrome P450 enzyme and drug transporter assessments.
He is actively involved in translating scientific discoveries into evidence-based therapeutics, bridging preclinical research, clinical development, and regulatory requirements.

Abstract

Cancer-related fatigue is a common and debilitating complication of cancer treatment, adversely affecting physical functioning and quality of life. Limited pharmacological options highlight the need for evidence-based supportive care interventions. This presentation traces the development of Nuvastatic, a standardised, rosmarinic acid-rich extract of Orthosiphon stamineus, from preclinical investigation through Phase I–III clinical evaluation. The development programme integrates botanical authentication, phytochemical standardisation and controlled manufacturing with preclinical efficacy and safety assessment. In a mouse model of paclitaxel-induced fatigue, the extract improved physical endurance and locomotor activity. Its antioxidant and anti-inflammatory properties provide a mechanistic rationale for investigation in cancer-related fatigue. Nonclinical toxicology and safety pharmacology studies supported progression into human trials.

A Phase I programme involving 104 healthy volunteers assessed safety, tolerability and pharmacokinetics. Single oral doses up to 2,000 mg were well tolerated, with no serious adverse events reported. A multicentre, randomised, double-blind, placebo-controlled Phase II trial subsequently enrolled 110 patients with solid tumours receiving chemotherapy. Nuvastatic, administered at 1,000 mg three times daily for nine weeks, significantly improved fatigue and quality-of-life measures and reduced urinary F_2-isoprostanes, supporting a potential contribution from oxidative stress modulation. A multicentre, randomised, double-blind, placebo-controlled Phase III trial evaluated 180 patients with breast cancer receiving chemotherapy.

Nuvastatic demonstrated significantly greater improvement in Brief Fatigue Inventory scores compared with placebo, with supportive responder analyses and improvements in selected quality-of-life domains. Treatment was generally well tolerated. Collectively, these findings illustrate the translation of a standardised botanical preparation from preclinical development to Phase III evaluation and support Nuvastatic's potential as a supportive care intervention for cancer-related fatigue. Further research should establish the durability and clinical relevance of benefit across broader cancer populations.

FORUM 1

Artificial Intelligence in Cancer Care



AP DR. SUNIZA JAMARIS
Universiti Malaya, Malaysia

Dr. Suniza Jamaris is a Consultant Breast Oncoplastic Surgeon and Associate Professor at Universiti Malaya, with expertise in breast cancer surgery, screening, research, and implementation science. She leads the MyBcc breast cancer research cohort and contributes to NavkanMy, an AI-powered, person-centric, gender-responsive digital health initiative developed through the Universiti Malaya AI in Medicine programme to streamline cancer screening, risk assessment, and patient navigation. She is also Course Director of REBUNG, an online training programme for GPs focused on early cancer detection and screening.



TS. DR. AFZAN ADAM
Universiti Kebangsaan Malaysia
Malaysia

Dr. Afzan Adam holds a Ph.D. in Computing from the University of Leeds, UK. She specializes in Machine Learning, Image Processing, and Digital Pathology. She teaches advanced AI skills alongside leading impactful research. Currently serves as the Technical Committee for Malaysian Digital Pathology Guidelines, she was a Reviewer Panel member for the guideline for verification and validation of DP published by KKM. Her research conducted in close collaboration with clinical experts from (HCTM/HUKM) and Pantai Integrated Labs; focuses on screening and classifying breast, blood, prostate, ovarian and cervical cancers, and advancing e-health initiatives across various medical departments. Additionally, her portfolio includes tiny object detection, image encryption, speech recognition, and time-series predictive modeling.



AP DR. SUNNY WONG
Nanyang Technological
University, Singapore

Dr Sunny Wong is an Associate Professor at the Lee Kong Chian School of Medicine, Nanyang Technological University Singapore, where he also serves as Assistant Dean in Academic Medicine. He received his MBChB at the Chinese University of Hong Kong and completed his PhD at the University of Oxford. He completed his specialist training in gastroenterology at the Prince of Wales Hospital in Hong Kong. Joining NTU Singapore in 2021, he founded the Centre for Microbiome Medicine in 2022 and has been serving as Director since then. Dr Wong's research focuses on the gut microbiome and host–microbe interactions in digestive diseases and cancers. He has published more than 300 peer-reviewed articles in journals including Gastroenterology, Gut, and Nature Communications. His work has been widely recognized through multiple awards, including the Sir David Todd Lectureship (2020, the Asia Pacific Digestive Week Emerging Leaders Lectureship (2021), Seah Cheng Siang Memorial Lectureship (2025), and Clarivate Analytics Highly Cited Researcher recognitions in 2023, 2024, and 2025.

FORUM 2

Generics and Drug Accessibility



DR. LOH C-KHAI
Universiti Kebangsaan Malaysia, Malaysia

Dr. Loh C-Khai is a Paediatric Haematologist/Oncologist and Head of the Paediatric Haematology/Oncology Unit, at UKM Children's Speciatlist Hospital, Kuala Lumpur. She obtained her MBBS from the University Malaya in 2002, and later MRCPCH UK in 2006. She had her Haematology/Oncology fellowship at Great Ormond Street, London UK in 2011 and further training in Freiburg, Germany in 2016. Her research interests focus primarily on childhood cancer, with particular experience in the clinical and translational aspects of anticancer drug use in children. She was the principle investigator of the fundamental research grant (FRGS/1/2021), which investigated the efficacy of generic pegylated asparaginase and the impact of anti-asparaginase antibodies among children with acute lymphoblastic leukaemia in Malaysia.



MS PRIYA DARSHINI ASOKAN
Duopharma Biotech Berhad, Malaysia

Priya Darshini A/P Asokan is the Chief Innovation Officer of Duopharma Biotech Berhad, bringing over 17 years of experience in biotechnology, pharmaceuticals, business development, and innovation. She holds an MBA from Monash University and a Bachelor's degree in Medical and Pharmaceutical Biotechnology from the University of South Australia. Since joining Duopharma in 2010, she has played a key role in advancing biosimilars, oncology initiatives, and strategic partnerships. She currently leads the Group's innovation agenda, corporate strategy, research and development, and technology-driven growth initiatives.



Dr. Ooi Po Lin
Universiti Malaya, Malaysia

Dr. Ooi Po Lin is a Clinical Oncologist at University Malaya Medical Centre specializing in breast, GI, and urological cancers. She holds an MBBS and a Masters of Clinical Oncology from the University of Malaya. As a Principal Investigator for international clinical trials, Dr. Ooi advances cancer care while actively advocating for patient access and empowerment. She leads community initiatives and speaks at global forums to ensure patients are informed and supported.

FORUM 2

Generics and Drug Accessibility



MR. RAJIS SANTHIRASEGAR
Pharmaniaga Berhad, Malaysia

Mr. Rajis Santhirasegar is a Malaysian pharmaceutical sales, marketing, and product management professional based in Shah Alam, Selangor, with over two decades of industry experience. He has spent roughly 15 years at Pharmaniaga Berhad, taking on leadership roles across sales, marketing, business development, and specialty therapeutic areas.



PN NIK SHAMSIAH NIK SALLEH
National Pharmaceutical Regulatory Agency, Malaysia

PN Nik Shamsiah Nik Sallehgraduated with a Bachelor's degree in Pharmacy from Universiti Kebangsaan Malaysia and later earned her Master's degree in Analytical Chemistry & Instrumental Analysis from Universiti Malaya. With over 24 years of dedicated service at the National Pharmaceutical Regulatory Agency (NPRA), Ministry of Health, Malaysia, she has held a variety of positions and undertaken diverse responsibilities within the organization. Prior to her current role, she served as the Head of Generic Medicines Section & Head of Cosmetics Section. Her regulatory expertise encompasses the evaluation of both medicinal and cosmetic products. Additionally, she has been actively involved in international and local regulatory dialogues, making significant contributions to discussions on regulatory matters and helping to shape best practices within the field.

Sponsored Talk

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CAYDEN CHOO XUAN HONG
Yokogawa Malaysia

Cayden Choo Xuan Hong works in Regional Business Development at Yokogawa Malaysia, supporting research laboratories with high-content imaging and analysis solutions. Cayden holds a degree in Biomedical Engineering, majoring in medical imaging, from Shanghai Jiao Tong University, and has nearly five years of experience in life science, including a previous role at Thermo Fisher Scientific. Cayden's interest is in helping research groups plan imaging-based assays for 3D cell models and in making image-based results reproducible across laboratories.

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Applications



Live-Cell Imaging | Spheroid Analysis | Cellular Analysis

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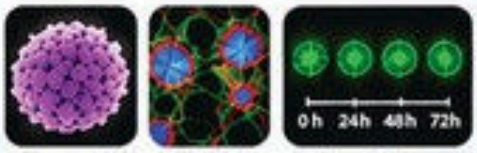
Applications



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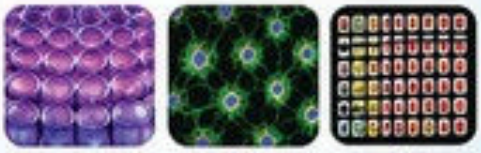
Applications



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Key Applications

Cancer Research | Drug Discovery | Organoids & Spheroids | Cell Biology | Stem Cell Research

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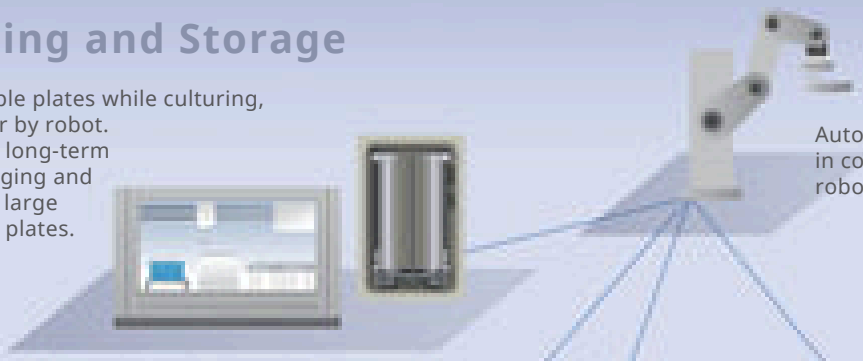

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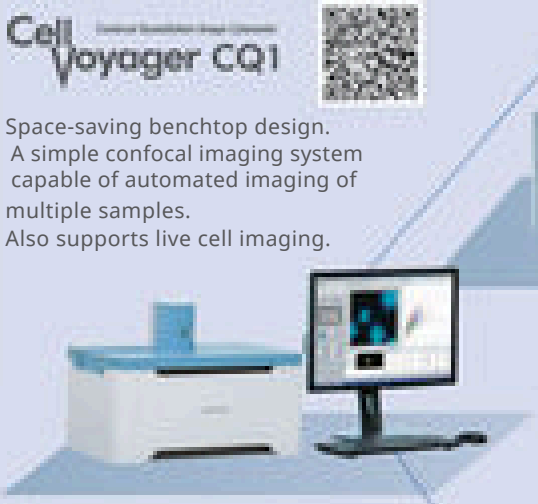

CellVoyager CQ3000



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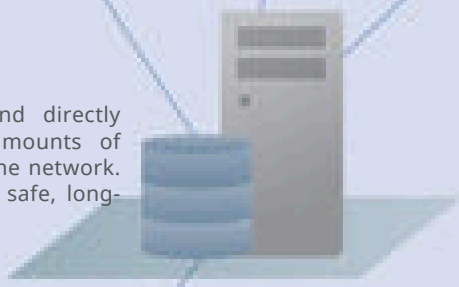
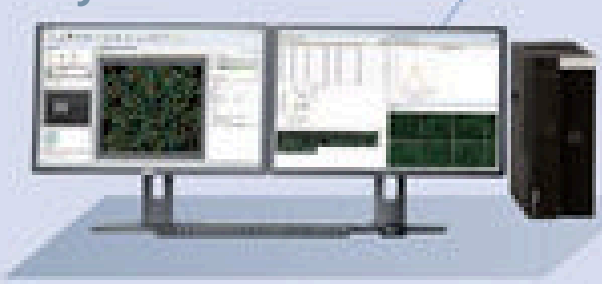


4 Analysis



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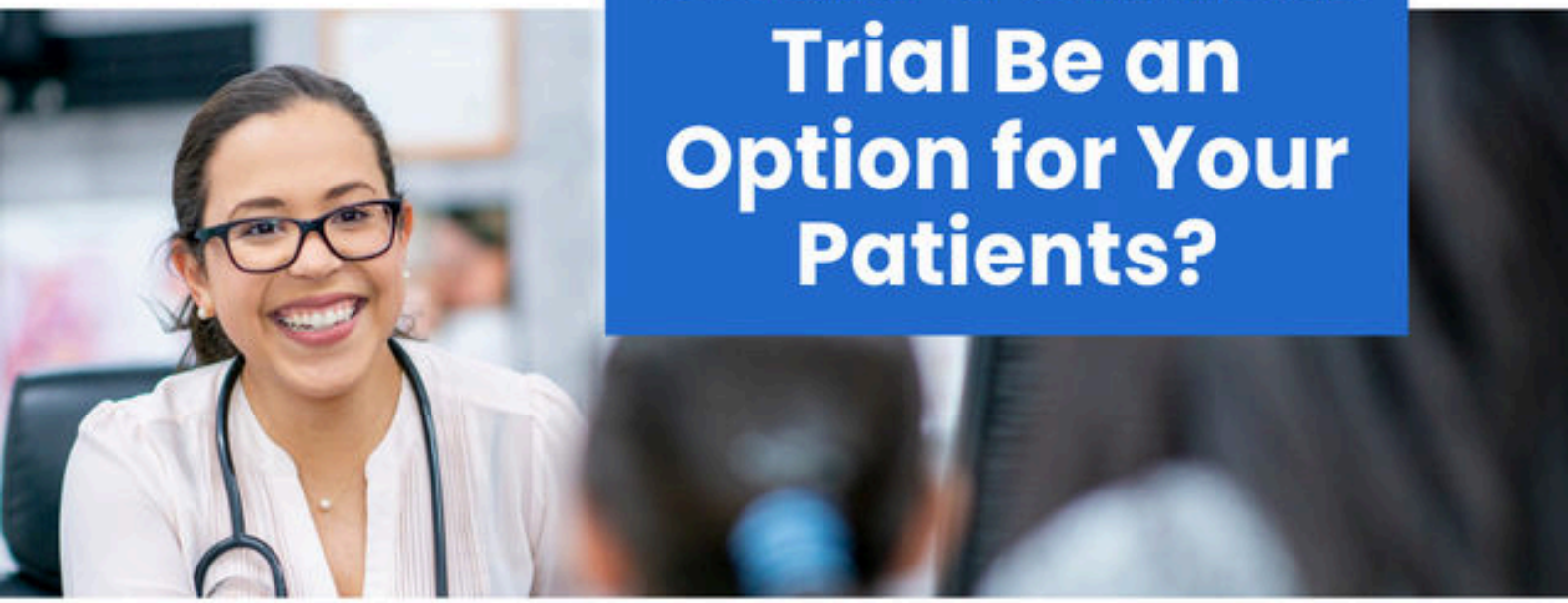

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Day 1 – 24th Nov 2026

ORAL PRESENTATION
Track/Area 1: Cancer Drug Discovery
Time: 11.40am – 1.00 pm
Venue: Auditorium, Level 2

Time	Poster ID	Poster Title	Presenter
11.45 am – 12.00 pm	OA013	Artesunate Exhibits Differential Cytotoxicity and Synergizes with Cisplatin in Nasopharyngeal Carcinoma Cells	Khang Ning Lim
12.00 pm – 12.15 pm	OA017	Cutaneous Sensitisation with Ranunculus Ternatus Thunb. Enhances Dacarbazine Efficacy by Modulating Antitumour Immunity in Melanoma-Bearing Mice	Amy Saik
12.15 pm – 12.30 pm	OA018	Targeting the ADAR1 Zα Domain in Oral Squamous Cell Carcinoma: From Computational Screening to Experimental Evaluation	Annie Wai Yeeng Chai
12.30 pm – 12.45 pm	OA022	Fabrication of OA-MNPs-PLGA-TAM Nanohybrids for Overcoming Tamoxifen Resistance in Breast Cancer via CD24 Downregulation	Norazalina Saad
12.45 pm – 1.00 pm	OA023	Integrated In Silico and In Vitro Evaluation of Quinazolin-3(4H)-One Derivatives Targeting EGFR-Mutant Non-Small Cell Lung Cancer	Md Arif Hossain

ORAL PRESENTATION
Track/Area 2: Cancer Biology
Time: 11.40am – 1.00 pm
Venue: Bilik Seminar 4, Level 1

Time	Poster ID	Poster Title	Presenter
11.45 am – 12.00 pm	OA007	Targeting the SOX13/GPX4 Axis Restores Ferroptosis Vulnerability to Overcome Cisplatin Resistance in Neuroendocrine Prostate Cancer	Tianxiang Wang
12.00 pm – 12.15 pm	OA011	Differential Regulation Of Primary Cilia And KIF3A Expression In Oral Squamous Cell Carcinoma Cells: A Preliminary Study	Misya Lim Binti Adam Lim
12.15 pm – 12.30 pm	OA015	Flightless I as Molecular Target for Inhibition of Radiation-Induced Colorectal Metastasis	Noraina Muhamad Zakuan
12.30 pm – 12.45 pm	OA019	Identification of an EBNA1-Derived NAP1L1-Binding Motif as a Potential Therapeutic Target in Epstein-Barr Virus-Associated Nasopharyngeal Carcinoma	Yap Beow Keat
12.45 pm – 1.00 pm	OA021	miR-146b-5p-Mediated Metabolic Crosstalk Between Tumor Cells and Macrophages in Bladder Cancer	Nur Akmarina Mohd Said

Day 1 – 24th Nov 2026

POSTER PRESENTATION
Time: 10.10am – 10.40am
Venue: Open Area, Level 1

Poster ID	Poster Title	Track/Area	Presenter
PA008	LSD1 as a Mechanistic Bridge in Cardio-Oncology: Epigenetic Regulation in Cardiovascular Disease and Selected Cancers	Cancer Biology	Shadi khadijeh Gholami
PA015	TERT Variations in Exon 15 and the Promoter Region: Potential Genetik Risk Factors for Acute Leukaemia	Cancer Biology	Chia Boon Hock
PA018	Flightless I as Molecular Target for Inhibition of Radiation-Induced Colorectal Metastasis	Cancer Biology	Noraina Muhamad Zakuan
PA020	Association Between Mitochondrial DNA Mutations and Cellular Senescence-Associated Gene Alterations Across Cancer Types	Cancer Biology	Houra Jazayeri
PA024	The pro-metastasis effect of CBFβ in MDA-MB-231 cells	Cancer Biology	Muhammad Muiz Ahmad Jefry
PA031	Establishing an In Vitro Model for Elucidating the Functional Relevance of the Mitochondrial m.3910G>C Mutation	Cancer Biology	Eng Wee Chua
PA009	Exceptionally Selective and Orally Bioavailable p300 Degraders for the treatment of CBP Mutant and p300 Dependent Cancers	Cancer Drug Discovery	Mohamad Fairus Abdul Kadir

Day 2 – 25th Nov 2026

ORAL PRESENTATION
Track/Area 3: Translational Cancer Medicine
Time: 11.05am – 12.30 pm
Venue: Auditorium, Level 2

Time	Poster ID	Poster Title	Presenter
11.10 am – 11.25 am	OA010	Intermittent Dosing with a Second-Generation Tyrosine Kinase Inhibitor Delays T790M Resistance	Wen Tsin Poh
11.25 am – 11.40 am	OA014	Navigating the Disparities Pathway: Multi Model Analysis of Targeted Therapy Access for Metastatic Colorectal Cancer	Gobi Gunasekaran
11.40 am – 11.55 am	OA016	Practices, Barriers, and Enablers to Enhanced Recovery After Surgery (ERAS) Implementation in Upper Gastrointestinal Surgery: A Malaysian Multicentre Qualitative Study	Nur Filzah Binti Aliah
11.55 am – 12.10 pm	OA020	Mapping the Nutritional Phenotypes of Accelerated Biological Ageing in Breast Cancer Survivors: An Explainable Machine Learning Approach	Mohd Razif Shahril

ORAL PRESENTATION
Track/Area 4: Clinical Trials of New Cancer Drugs/Precision Oncology
Time: 11.05am – 12.30 pm
Venue: Bilik Seminar 4, Level 1

Time	Poster ID	Poster Title	Presenter
11.10 am – 11.25 am	OA005	High-Dose Methotrexate Pharmacokinetics in Pediatric Acute Lymphoblastic Leukemia in Indonesia: Correlation with Renal Function and SLCO1B1 Polymorphism	Siti Rahmayanti
11.25 am – 11.40 am	OA006	Population Pharmacokinetic Model-Based Risk Stratification Strategy for High-Dose Methotrexate in Malaysian Adults with Haematological Malignancies	Tuan Fauziah Tuan Roslli
11.40 am – 11.55 am	OA009	Exploration of Factors Influencing the Efficacy of Second-Line Osimertinib in NSCLC Patients	Rui Jing

Day 2 – 25th Nov 2026

POSTER PRESENTATION
Time: 10.35am – 11.05am
Venue: Open Area, Level 1

Poster ID	Poster Title	Track/Area	Presenter
PA004	Virtual Screening of FDA-Approved Drugs Targeting the Allosteric Pocket of Resistant Tertiary C797S Mutant EGFR in Non-Small Cell Lung Cancer	Cancer Drug Discovery	Abdullah Al Galib
PA005	Harnessing Antineoplastic Properties of Halicin against Hepatocellular Carcinoma	Cancer Drug Discovery	Abinithi Samikkannu
PA010	Novel Thioquinazolinone Derivatives: Design, Synthesis and Biological Evaluation Against EGFR-Mutant Non-Small Cell Lung Cancer	Cancer Drug Discovery	Md Swapan Hossain
PA011	Hellebrigenol Suppresses Hepatocellular Carcinoma through Targeting β-Catenin Signalling	Cancer Drug Discovery	Chandra Bhol
PA012	Discovery and characterization of once in a month intravenous and orally bioavailable SMARCA2 degraders for treating SMARCA4 mutant cancers	Cancer Drug Discovery	Pratheev Alagappan
PA025	Drug Repurposing for EGFR-Mutated Non-Small Cell Lung Cancer: Computational and Experimental Evaluation of FDA-Approved Drugs	Cancer Drug Discovery	Nur Aisyah Mohamed
PA033	Liposomal FASN Inhibitor as a Potential Inhalable Drug Delivery System for Non-Small Cell Lung Cancer	Cancer Drug Discovery	Chee Woei Lim

Day 2 – 25th Nov 2026

POSTER PRESENTATION
Time: 1.10pm – 1.35pm
Venue: Open Area, Level 1

Poster ID	Poster Title	Track/Area	Presenter
PA013	Anticancer Effects of a 1,4-Naphthoquinone Derivative in Hepatocellular Carcinoma Mediated by Inhibition of STAT3 Signaling	Cancer Drug Discovery	Ruyi Jin
PA014	In Silico Drug Repurposing Targeting the Allosteric Domain of Mutant EGFR in Non-Small Cell Lung Cancer	Cancer Drug Discovery	Sajeesh Pattammadath
PA016	Modulation of Autophagy in Pancreatic Cancer Cells by a Semisynthetic Andrographolide Derivative	Cancer Drug Discovery	Sajna Purayil
PA017	Anticancer Effects of a Combination of Sotorasib and Osimertinib on A549 Non-Small Lung Cancer Cell Line	Cancer Drug Discovery	Ketharin Tunasamy
PA023	Preliminary in Silico Study on Molecular Docking Analysis of D-Allulose with Glucose Transporters	Cancer Drug Discovery	Nur Afiqah Rosdi
PA032	Combined Effects of Auranofin and Luteolin on Cell Viability and Radiosensitivity in HCT116 Colorectal Cancer Cells	Cancer Drug Discovery	Nurul Akmaryanti Abdullah
PA027	Effects of Zerumbone on Breast Cancer Cell Invasiveness under DMOG-induced Hypoxia	Cancer Drug Discovery	Fariesha Hashim
PA029	Targeting the ADAR1 Zα Domain in Oral Squamous Cell Carcinoma: From Computational Screening to Experimental Evaluation	Cancer Drug Discovery	Annie Wai Yeeng Chai
PA006	From Model to Translation: A Scoping Review of Preclinical Tongue Squamous Cell Carcinoma Models	Translational Cancer Medicine	Fong Fong Liew

Day 2 – 25th Nov 2026

POSTER PRESENTATION
Time: 1.35pm – 2.00pm
Venue: Open Area, Level 1

Poster ID	Poster Title	Track/Area	Presenter
PA019	Practices, Barriers, and Enablers to Enhanced Recovery After Surgery (ERAS) Implementation in Upper Gastrointestinal Surgery: A Malaysian Multicentre Qualitative Study	Translational Cancer Medicine	Nur Filzah Binti Aliah
PA021	Beyond Treatment Availability: Inequities in Timely Access to Targeted Therapy for Metastatic Colorectal Cancer	Translational Cancer Medicine	Fatiha Shabaruddin
PA022	Development and Preliminary User Testing of ColRectCare©: A Bilingual Web Application for Colorectal Cancer Awareness in Malaysia	Translational Cancer Medicine	Ehsan Hussaini
PA026	The Effectiveness And Safety Of 5-Aminolevulinic Acid Photodynamic Therapy In Patients With Persistent HPV Infection With Or Without Cervical Intraepithelial Neoplasia: A Retrospective Cohort Study	Translational Cancer Medicine	Ya Wang
PA030	A Step Towards Healthy Ageing: Does Fat Mass Modulate the Association Between Physical Fitness and Telomere Length in Breast Cancer Survivors?	Translational Cancer Medicine	Nur Afifah Mursyida Binti Zaujan
PA002	High-Dose Methotrexate Pharmacokinetics in Pediatric Acute Lymphoblastic Leukemia in Indonesia: Correlation with Renal Function and SLCO1B1 Polymorphism	Clinical Trials/Precision Oncology	Siti Rahmayanti
PA003	Population Pharmacokinetic Model-Based Risk Stratification Strategy for High-Dose Methotrexate in Malaysian Adults with Haematological Malignancies	Clinical Trials/Precision Oncology	Tuan Fauziah Tuan Roslli
PA007	Mechanism-Guided Strategies for Overcoming Acquired Resistance to Osimertinib in EGFR-Mutated Non-Small-Cell Lung Cancer: A Review	Clinical Trials/Precision Oncology	Remi Johnson
PA028	Integrating Clinical And Transcriptomic Data For Relapse Prediction In Childhood B-Cell Acute Lymphoblastic Leukaemia	Clinical Trials/Precision Oncology	Oh Lixian

4th MSC 2026

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Advancing Regional Cancer Research for Equitable Cancer Care

Oral Presentation

Track/Area 1: Cancer Drug Discovery

Time: 11.40 am – 1.00 pm

Venue: Auditorium, Level 2

Artesunate Exhibits Differential Cytotoxicity and Synergizes with Cisplatin in Nasopharyngeal Carcinoma Cells

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Introduction: Nasopharyngeal carcinoma (NPC) is an Epstein-Barr virus (EBV)-associated cancer. Its management is limited by late presentation, poor survival and treatment toxicity, while immunotherapies and targeted agents remain unaffordable. Drug repurposing offers a cost-effective alternative. Artesunate (ART), a safe, affordable antimalarial drug, has anticancer activity against other tumours but is understudied in NPC. We evaluated the anti-proliferative effect of ART on NPC cells in vitro. **Methods:** The cytotoxicity effect of ART against EBV-positive (C666-1 and NPC43) and EBV-negative (HONE1) NPC cell lines was assessed using Cell Counting Kit-8 viability assays. The potential of ART in sensitizing the effect of conventional chemotherapeutic drug, cisplatin (CIS), was also evaluated using the Chou-Talalay method to determine the combination index (CI). **Results:** ART exerted dose-dependent cytotoxicity across all three NPC cell lines. C666-1 was the most sensitive with an IC50 of 6.79 ± 2.31μM, whereas NPC43 was relatively resistant with an IC50 of 74.93 ± 7.34μM. Interestingly, the isogenic EBV-negative NPC43 cell line showed a similar level of resistance (IC50 of 87.94 ± 7.69μM), suggesting that EBV status alone does not account for differences in ART sensitivity. In parallel, the inactive ART derivative, deoxyartemisinin, had no effect on C666-1 cell viability, confirming the specificity of ART-mediated cytotoxicity. Combination treatment of ART and CIS produced strong synergistic effects against C666-1 cells at ART concentrations below 10μM and CIS concentrations below 5μM, with the strongest synergy observed at 2.5μM ART + 1.25μM CIS. Conversely, ART and CIS exhibited predominantly antagonistic effects across the tested concentration combinations in NPC43 cells. **Conclusion:** ART demonstrates heterogeneous cytotoxic activity across NPC cell lines that is not solely explained by EBV status, while its synergism with CIS against C666-1 provides preclinical support for further investigation of ART as a potential low-cost therapeutic or combination agent for NPC.

Keywords:
Nasopharyngeal carcinoma, Epstein-Barr virus, drug repurposing, artesunate, cisplatin

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OA017

Cutaneous Sensitisation with Ranunculus Ternatus Thunb. Enhances Dacarbazine Efficacy by Modulating Antitumour Immunity in Melanoma-Bearing Mice

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Introduction: Melanoma remains a highly aggressive malignancy, while the clinical efficacy of dacarbazine is limited in part by an immunosuppressive tumour microenvironment (TME) that restricts effective antitumour T-cell responses. Strategies that combine chemotherapy with immune modulation may therefore improve therapeutic efficacy. Controlled cutaneous sensitisation may induce systemic immune activation and enhance antitumour immunity. Ranunculus ternatus Thunb. (R. ternatus), a traditional Chinese herb capable of inducing localised cutaneous inflammation, may serve as a natural source of such immune stimulation. This study investigated whether R. ternatus-induced cutaneous sensitisation could enhance the antitumour efficacy of dacarbazine through modulation of systemic and intratumoral T-cell responses in melanoma-bearing mice. **Methods:** C57BL/6 mice bearing subcutaneous B16F10 melanoma were randomised into four groups: tumour control, topical R. ternatus application (0.5g), dacarbazine (80mg/kg), and combination treatment (n=5 per group). Treatments began when tumour volume reached ≥125 mm^3. Tumour growth, intratumoral $CD8^+$, $CD4^+$ and Foxp3+ regulatory T-cell (Treg) distribution were analysed via immunofluorescence. One-way ANOVA with Dunnett's post hoc test determined statistical significance ($p<0.05$). **Results:** Among all groups, combination treatment showed the most significant reduction in tumour volume (1345±740 mm^3 to 335±161 mm^3, $p<0.001$). As compared to tumour control group, combination treatment caused $CD8^+$ infiltration to increase nearly six-fold (1.98±1.21% to 11.84±2.14%, $p<0.0001$) and $CD4^+$ T-cell recruitment to double (3.41±0.67% to 7.15±1.02%, $p<0.001$). It also optimised the TME, achieving a $CD8^+$/Treg ratio >1, shifting it from an immunosuppressive to a pro-inflammatory state. **Conclusion:** Dacarbazine efficacy for treating melanoma was enhanced by low-dose R. ternatus-induced cutaneous sensitisation, via increasing systemic and intratumoral T-cell responses, and shifting the TME from an immunosuppressive to a pro-inflammatory state. These findings support controlled cutaneous immune priming as a potential adjuvant strategy to improve chemotherapy-associated antitumour immunity and warrant further investigation of its underlying mechanisms and translational potential.

Keywords:
Cutaneous sensitisation, Melanoma, Dacarbazine, Tumour microenvironment, Systemic immune priming

Targeting the ADAR1 Zα Domain in Oral Squamous Cell Carcinoma: From Computational Screening to Experimental Evaluation

Annie Wai Yeeng Chai[1]*, Pei San Yee[1], Jie Ying Teo[1,2], Stephanie Hao Ling Cheah[1,3], Xavier Wezen Chee[3,4], Wei Khang Gan[5], Keng Yoon Yeong[5], Siew Kit Ng[2], Siaw San Hwang[3], Sok Ching Cheong[1,6]

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Introduction: Our genome-wide CRISPR/Cas9 dependency screening in Asian-derived oral squamous cell carcinoma (OSCC) models identified ADAR1, particularly its interferon-inducible p150 isoform, as a recurrent cancer vulnerability. Beyond its RNA-editing activity, ADAR1-p150 contains a Zα domain that recognises Z-DNA and Z-RNA, offering an alternative intervention point for therapeutic development. **Methods:** We applied our proprietary computer-aided drug discovery pipeline, termed the Multi-layer Ultra-large Screening and Exploration (M.U.S.E), to approximately six billion compounds to identify potential ADAR1-p150 Zα-domain binders. Candidate compounds were initially prioritised through complementary biochemical and cellular screening assays, including intrinsic tryptophan fluorescence (ITF), electrophoretic mobility shift assay (EMSA) and MTT-based cytotoxicity assessment. Selected hit was subjected to chemical modification and evaluated using ITF, followed by cellular assessment of prioritised derivatives in OSCC and non-cancerous cell models. **Results:** Of 51 compounds evaluated by ITF, 19 demonstrated favourable binding profiles to recombinant Zα. Integration of biochemical and cellular screening results enabled prioritisation of selected hits, including MUSE-22, for further chemical refinement. Evaluation of MUSE-22 derivatives identified several compounds with improved ITF binding relative to the parent molecule. Among them, MUSE22-C8 was subsequently chosen for cellular evaluation, demonstrating an IC_{50} of 41.72 μM in the ADAR1-p150-dependent OSCC model (ORL-207), compared with no measurable IC_{50} in the tested non-cancerous OKF6/TERT1 and HEK293T models within the concentration range evaluated. **Conclusion:** Our study demonstrates the feasibility of identifying small-molecule compounds that bind the ADAR1-p150's Zα domain and provides preliminary evidence of cellular activity in an ADAR1-p150-dependent OSCC model. Further work is required to improve cellular potency, establish the contribution of ADAR1-p150-targeted activity and address pharmacological properties relevant to therapeutic development. These findings provide a basis for continued exploration of Zα-domain targeting as a potential strategy for ADAR1-p150-dependent OSCC.

Keywords: ADAR1, Zα domain, oral squamous cell carcinoma, virtual screening, drug discovery

Fabrication of OA-MNPs-PLGA-TAM Nanohybrids for Overcoming Tamoxifen Resistance in Breast Cancer via CD24 Downregulation

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Introduction: Tamoxifen resistance remains a major clinical challenge in the treatment of breast cancer. Furthermore, conventional tamoxifen administration is frequently limited by poor bioavailability and rapid systemic clearance, necessitating a sustained-release mechanism to maintain effective dosing. To address this, a nanohybrid system, OA-MNPs-PLGA-TAM, was fabricated to overcome resistance mechanisms through targeted molecular modulation and enhanced intracellular drug delivery. **Methods:** The OA-MNPs-PLGA-TAM nanohybrids were synthesized and further characterized. Structural properties were evaluated using X-ray Diffraction (XRD), Fourier transform infrared (FTIR) spectroscopy, and Transmission Electron Microscopy (TEM). In vitro biological evaluations, including cytotoxicity, immunophenotyping, and Annexin V-FITC apoptosis assays, were conducted on tamoxifen-resistant MCF-7 Tam1 and sensitive MCF-7 breast cancer cell lines upon CD24 downregulation. **Results:** Characterization demonstrated the synthesis and structural integrity of the nanocarriers. XRD revealed overall crystallite sizes ranging from 20 to 60 nm, with a specific wurtzite crystal phase size of 12.952 nm, while FTIR verified the encapsulation of tamoxifen (TAM) within the PLGA matrix. Biologically, the nanohybrids exhibited significant, time-dependent cytotoxicity against MCF-7 Tam1 cells, achieving IC_{50} values of 20.5 ± 0.7 µg/mL and 15.5 ± 4.9 µg/mL at 24 and 48 hours, respectively. Furthermore, immunophenotyping of treated spheroids demonstrated a marked downregulation of CD24 expression in treated spheroids. This targeted CD24 suppression resensitized the resistant cells, leading to a significant induction of early apoptosis in MCF-7 Tam1 cells following 48 hours of treatment. **Conclusion:** The OA-MNPs-PLGA-TAM nanohybrids provide a platform to investigate and overcome tamoxifen resistance. The possible mechanism underlying this therapeutic efficacy may involve the synergistic properties of the nanocarrier. Specifically, the PLGA coating allows for sustained drug release and mitigates P-glycoprotein efflux. Additionally, the nanoscale dimensions enhance cellular endocytosis. Together, these features effectively resensitize resistant cells to apoptosis via CD24 suppression. However, future studies are required to validate this mechanism.

Keywords: Breast cancer; magnetite polymeric nanoparticles; tamoxifen-resistant; nanohybrid; cytotoxic

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OA023

Integrated In Silico and In Vitro Evaluation of Quinazolin-3(4H)-One Derivatives Targeting EGFR-Mutant Non-Small Cell Lung Cancer

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Introduction: Activating mutations in the epidermal growth factor receptor (EGFR) drive the growth and proliferation of non-small cell lung cancer (NSCLC), while treatment with first- and second-generation EGFR tyrosine kinase inhibitors (TKIs) is limited by acquired resistance, frequently mediated by the T790M gatekeeper mutation. This study investigated a series of novel quinazolin-3(4H)-one derivatives designed as mutation-tolerant anticancer agents using an integrated in silico, in vitro, and mechanistic approach. **Methods:** Molecular docking and 100 ns molecular dynamics (MD) simulations were executed against wild-type, L858R-mutant, and T790M-mutant EGFR kinase domains to evaluate binding energies and trajectory stabilities relative to osimertinib. Four synthesised derivatives (CQ1, CQ2, CQ3, and CQ4), were biologically screened via 96-hour MTT cytotoxicity assays across three mutant NSCLC cell lines: PC9 (exon 19 deletion), NCI-H1975 (L858R/T790M), and HCC827 (exon 19 deletion), together with a non-tumorigenic bronchial epithelial line (BEAS-2B) to determine half-maximal inhibitory concentration (IC_{50}) and selectivity index (SI) values. Western blot analysis quantified the concentration-dependent modulation of the MAPK signalling cascade in resistant NCI-H1975 cells. **Results:** While the computational analyses identified CQ2 as the strongest binder against EGFR L858R (−8.2 kcal/mol) and CQ1 as exhibiting the most stable protein–ligand interactions during the 100 ns MD simulation, the biological results showed a different ranking of compound activity. CQ3 emerged as the most potent cellular lead, yielding the lowest IC_{50} values against drug-sensitive PC9 cells (13.7 ± 1.2 μM) and double-mutant resistant NCI-H1975 cells (14 ± 1.7 μM) and inactive against HCC827 cells (IC_{50} > 100 μM). Furthermore, CQ3 exhibited low toxicity against BEAS-2B cells (IC_{50} > 100 μM), resulting in highly favorable selectivity indices of greater than 7. Mechanistic validation in H1975 cells confirmed that CQ3 induced concentration-dependent (7, 14, and 21 μM) suppression of EGFR, c-Raf, and ERK phosphorylation. **Conclusion:** Overall, CQ3 demonstrated the most promising cellular activity among the evaluated quinazolin-3(4H)-one derivatives and was associated with modulation of EGFR-associated MAPK signalling, supporting its further investigation against EGFR-mutant NSCLC.

Keywords:
NSCLC, quinazoline, L858R mutation, T790M mutation, in silico, EGFR.

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Oral Presentation

Track/Area 2: Cancer Biology

Time: 11.40 am – 1.00 pm

Venue: Bilik Seminar 4, Level 1

4th MSC 2026
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OA007

Targeting the SOX13/GPX4 Axis Restores Ferroptosis Vulnerability to Overcome Cisplatin Resistance in Neuroendocrine Prostate Cancer

Tianxiang Wang[1], Bo Liu[1], Weiwei Zhang[1], Ruopeng Su[1], Xinyu Liu[1], Ang Li[1], Xinyu Chai[1], Junyi Wang[1], Jiayan Yi[1], Wenxiao Yang[1], Conghao Wang[1], Jiahua Pan[1], Wei Xue[1], Qi Wang[1]*

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Introduction: Neuroendocrine prostate cancer (NEPC) is an aggressive subtype of prostate cancer with poor clinical outcomes. While cisplatin remains the major treatment option, rapid development of drug resistance substantially restricts its long-term benefit in NEPC. This study aims to identify mechanisms driving cisplatin resistance and develop corresponding intervention strategies for NEPC. **Methods:** High-throughput drug screening was performed to identify compounds that enhance cisplatin efficacy in the NEPC cell line. Candidate compounds were further evaluated using in vitro viability assays and in vivo xenograft models. Treatment-induced ferroptosis was assessed by measuring intracellular labile iron and reactive oxygen species levels. Molecular mechanism was investigated through genetic manipulation, luciferase reporter assay, and chromatin immunoprecipitation. **Results:** Zanamivir was identified as a potent sensitizer that, despite exhibiting limited cytotoxicity itself, synergistically enhanced cisplatin efficacy in NEPC. Ferroptosis inhibition reversed the synergistic effect of the combinatorial treatment, indicating that zanamivir-mediated restoration of ferroptosis vulnerability contributes to cisplatin sensitization. Mechanistically, the transcription factor SOX13 was established to promote ferroptosis resistance by directly activating the expression of GPX4, a key suppressor of ferroptosis. By inhibiting the SOX13/GPX4 axis, zanamivir increased intracellular labile iron accumulation and oxidative stress, ultimately restoring ferroptosis vulnerability and cisplatin sensitivity in NEPC. **Conclusion:** This study identifies the SOX13/GPX4 axis as a critical regulator of ferroptosis vulnerability. Targeting this pathway with zanamivir provides a promising strategy to overcome cisplatin resistance in NEPC.

Keywords
Neuroendocrine prostate cancer, Cisplatin, Zanamivir, Ferroptosis, SOX13/GPX4 axis

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OA011

Differential Regulation Of Primary Cilia And KIF3A Expression In Oral Squamous Cell Carcinoma Cells: A Preliminary Study

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Introduction: Primary cilia are sensory organelles that regulate cellular signalling and have been implicated in cancer. Cilia formation and function depend on ciliary proteins, including KIF3A. However, their role in oral squamous cell carcinoma (OSCC) remains poorly understood. This study investigated the occurrence of primary cilia, ciliary length and KIF3A protein abundance in immortalised normal oral keratinocytes (OKF6-TERT2), HSC-2 (derived from non-metastatic OSCC) and HSC-3 (derived from highly metastatic OSCC) cells following serum starvation. **Methods:** OKF6-TERT2, HSC-2, and HSC-3 cells were serum starved for 24, 48 and 72 hours. Cells were co-stained with Arl13b (axoneme), γ-tubulin (basal body) and DAPI (nuclei) for immunofluorescence microscopy. KIF3A protein abundance was determined by Western blotting. The experiments were independently repeated three times (n=3 biological replicates). **Results:** Primary cilia frequency and length varied according to the duration of serum starvation and cell type. At 24 and 72 hours, cilia frequency was comparable among the three cell lines, with no statistically significant differences observed. At 48 hours, cilia frequency was comparable between HSC-2 and OKF6-TERT2 cells; however, HSC-2 cells exhibited a significantly higher cilia frequency than HSC-3 cells. HSC-3 cells had significantly shorter primary cilia than OKF6-TERT2 cells at 24 hours. No statistically significant differences in cilia length were observed among the three cell lines at 48 or 72 hours. KIF3A protein abundance was significantly lower in HSC-2 cells at 72 hours and in HSC-3 cells at 24 hours compared with OKF6-TERT2 cells. **Conclusion:** KIF3A protein was not involved in primary cilia formation or length in HSC-2 cells, suggesting non-ciliary functions of KIF3A in HSC-2 cells. Shorter cilia in HSC-3 cells at 24 hours were accompanied by reduced KIF3A protein abundance, indicating an association between KIF3A expression and cilia morphology. Further studies are needed to clarify ciliary and non-ciliary roles of KIF3A in OSCC cells.

Keywords:
Primary cilia, KIF3A, Oral squamous cell carcinoma, Ciliogenesis, Serum starvation

Flightless I as Molecular Target for Inhibition of Radiation-Induced Colorectal Metastasis

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Introduction: Colorectal cancer (CRC) is a leading cause of cancer-related mortality worldwide, characterised by uncontrolled cell proliferation, migration, and invasion, which contribute to metastasis. Invadopodia facilitate cancer cell invasion through extracellular matrix (ECM) degradation. Although radiotherapy is an established treatment for CRC, radiation-induced cellular responses may contribute to tumour progression, metastasis, and radioresistance. Ionising radiation has been reported to induce cytoskeletal rearrangements in cancer cells; however, the underlying molecular mechanisms remain poorly understood. Flightless I (FliI), an actin-binding cytoskeletal protein, has been implicated in cancer progression, but its involvement in radiation-induced CRC migration and invasion remains unclear. Therefore, this study investigated the role of FliI in radiation-induced migration, invasion, and invadopodia formation in HCT116 colorectal cancer cells. **Methods:** FliI expression was suppressed using SMARTpool small interfering RNA (siFliI), and knockdown efficiency was validated by Western blot analysis. Cell migration and invasion were assessed using Transwell assays, while invadopodia-mediated ECM degradation was evaluated using a gelatin degradation assay. **Results:** The findings demonstrated that X-ray irradiation significantly increased FliI protein expression in HCT116 cells. siFliI transfection successfully reduced FliI protein expression by approximately 90%. Notably, FliI silencing combined with irradiation significantly suppressed HCT116 cell migration and invasion compared with irradiated control cells. Furthermore, the gelatin degradation assay revealed significantly reduced matrix degradation activity following FliI silencing, suggesting impaired invadopodia-mediated ECM degradation. However, the molecular signalling pathways underlying FliI-mediated migration and invasion remain to be elucidated. **Conclusion:** Collectively, these findings identify FliI as a potential regulator of radiation-associated invasive behaviour in CRC cells. Suppression of FliI may represent a promising strategy to limit radiation-induced migration and invasion, potentially reducing metastatic progression. Further investigations are warranted to clarify the underlying molecular mechanisms and evaluate the therapeutic relevance of FliI inhibition in CRC radiotherapy.

Keywords:
Flightless I; ionizing radiation; migration and invasion; colorectal cancer

Identification of an EBNA1-Derived NAP1L1-Binding Motif as a Potential Therapeutic Target in Epstein-Barr Virus-Associated Nasopharyngeal Carcinoma

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Introduction: Nasopharyngeal carcinoma (NPC) is an Epstein-Barr virus (EBV)-associated malignancy in which Epstein-Barr nuclear antigen 1 (EBNA1) plays a critical role in viral persistence and tumour progression. Despite advances in treatment, therapeutic resistance and toxicity remain major clinical challenges, highlighting the need for targeted therapies. EBNA1 interacts with multiple host proteins to support oncogenic functions, including nucleosome assembly protein 1-like 1 (NAP1L1), a histone chaperone implicated in chromatin remodelling and transcriptional regulation. Elevated NAP1L1 expression has been associated with NPC progression and poor clinical outcome. However, the precise EBNA1 peptide motif responsible for NAP1L1 binding has not been clearly defined. **Methods:** Eight candidate EBNA1-derived peptides were screened for binding to recombinant human NAP1L1 using nuclear magnetic resonance (NMR) spectroscopy and isothermal titration calorimetry (ITC). The antiproliferative effects of binding peptides were subsequently evaluated in EBV-positive NPC (C666-1) and normal nasopharyngeal epithelial (NP69) cell lines using MTT assays. **Results:** A critical NAP1L1-binding motif within EBNA1 was identified (Peptide 3), exhibiting a binding affinity (Kd) of 9.5 μM. Functional studies demonstrated selective inhibition of C666-1 cell proliferation, with an IC_{50} of 192 μM, while minimal cytotoxicity was observed in NP69 cells, indicating preferential activity against EBV-positive NPC cells. **Conclusion:** These findings identify a key EBNA1-derived NAP1L1-binding motif and provide proof-of-concept that disruption of the EBNA1-NAP1L1 interaction represents a potential therapeutic strategy for NPC. Collectively, this work lays the foundation for the development of more potent and selective inhibitors targeting EBNA1-NAP1L1 interactions in EBV-associated malignancies.

Keywords: Nasopharyngeal carcinoma, EBNA1, NAP1L1, Protein-protein interaction, Peptide inhibitor

miR-146b-5p-Mediated Metabolic Crosstalk Between Tumor Cells and Macrophages in Bladder Cancer

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Introduction: Advanced bladder cancer frequently exhibits therapeutic failure with immune checkpoint inhibitors, a limitation driven by an immunosuppressive tumour microenvironment enriched with M2-polarized tumour-associated macrophages (TAMs). We previously identified that oncogenic miR-146b-5p directly represses squalene synthase (FDFT1), redirecting mevalonate flux away from sterol synthesis and promoting chemoresistance. However, whether this tumoral metabolic reprogramming exerts paracrine effects to instruct surrounding immune stroma remains uncharacterized. **Methods:** Here, we investigated the non-cell-autonomous role of the tumoral miR-146b.-5p/FDFT1 axis in governing macrophage phenotype. Utilizing syngeneic murine bladder cancer models, naive RAW264.7 macrophages were exposed to conditioned media (CM) from miR-146b-5p-overexpressing or control bladder cancer cells. The acquisition of M1 and M2 phenotype were assessed via markers such as iNOS, CD86, CD206 and IL10. The miR-146b-5p levels in macrophage were quantified via qRT-PCR. **Results:** Macrophages treated with CM from cancer cells with elevated miR-146b-5p acquired an immunosuppressive M2-like phenotype with elevated pro-tumoral marker expression. Notably, this M2 transition occurred independently of changes in recipient macrophage miR-146b-5p levels, suggesting that immune suppression is driven by cell-extrinsic secretomic cues. In contrast, macrophage treated with CM from control cancer cells sustained an anti-tumoral M1-like phenotype accompanied by endogenous macrophage miR-146b-5p upregulation. **Conclusion:** Together, these findings indicate that tumoral miR-146b-5p/FDFT1 dysregulation functions beyond cell-autonomous resistance, acting upstream to remodel the paracrine immune microenvironment. Interrogating this metabolic crosstalk provides insights into reversing macrophage-mediated immunosuppression and improving immunotherapy efficacy in bladder cancer.

Keywords: Bladder cancer, Tumour- Associated Macrophages (TAMS), miR-146b-5p, Keyword, Cholesterol Metabolism, Immunometabolism

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Poster Presentation

Time: 10.10 am – 10.40 am

Venue: Open Area, Level 1

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PA008

LSD1 as a Mechanistic Bridge in Cardio-Oncology: Epigenetic Regulation in Cardiovascular Disease and Selected Cancers

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Introduction: LSD1 is an enzyme that demethylates H3K4 and H3K9, alongside non-histone substrates including p53, STAT3, and MYPT1, regulating transcription, chromatin remodeling, and cellular differentiation. Emerging evidence positions LSD1 as a molecular bridge in cardio-oncology, with shared mechanistic pathways linking cardiovascular disease (CVD) and cancer. **Methods:** We systematically reviewed PubMed and Embase with focus on LSD1, hypertension and selected cancer. **Results:** In CVD, LSD1 drives context-dependent epigenetic reprogramming. Under hyperglycaemic conditions, LSD1 sustains vascular inflammation and oxidative stress through NF-κB p65 and MMP-9 promoter engagement. LSD1 deficiency disrupts mineralocorticoid receptor signalling, promotes salt-sensitive hypertension, and impairs beige adipocyte differentiation. In ischemic injury, LSD1 is cardioprotective via SOX9 demethylation, whereas in pressure overload, myofibroblast LSD1 drives cardiac fibrosis and systolic dysfunction, underscoring its bidirectional, context-dependent role. In oncology, LSD1 is overexpressed in thyroid, renal, pancreatic, and neuroendocrine cancers, stabilizing HIF-1α, sustaining cancer stemness, facilitating epithelial-mesenchymal transition, and repressing tumour suppressor programs. In neuroendocrine prostate cancer and small cell lung cancer, LSD1 maintains lineage identity through non-catalytic mechanisms, while functioning paradoxically as a tumour suppressor in adrenal tissue. **Conclusion:** LSD1 operates through four shared mechanistic axes in both systems. First, it modulates Wnt/β-catenin, TGF-β/Smad2/3, and Notch developmental pathways implicated in cardiac remodeling and cancer stemness. Second, it regulates NF-κB, HIF-1α, and p53, common mediators of stress, hypoxia, and inflammation across cardiac and tumour microenvironments. Third, LSD1 activates myofibroblasts and promotes fibrosis via Smad2/3 in both heart failure and solid tumours. Fourth, it links androgen and mineralocorticoid receptor signalling, connecting steroid-driven tumour progression with renal and cardiac hormonal dysregulation, a particularly relevant axis in renal cancer and hypertensive CVD.

Keywords:
LSD1, blood pressure, cancer

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PA015

TERT Variations in Exon 15 and the Promoter Region: Potential Genetik Risk Factors for Acute Leukaemia

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Introduction: Telomerase reverse transcriptase (TERT), the catalytic component of telomerase, plays an essential role in telomere maintenance and genomic stability. Genetic variations within TERT, particularly in its promoter region, may influence telomerase regulation and have been reported in various malignancies. This study aimed to identify and characterize geneticvariations in the TERT promoter regionand exon 15 among patients from HPUSM with acute leukaemia and healthy controls. **Methods:** A total of 5 patients diagnosed with acute lymphoblastic leukaemia (ALL) and 14 patients diagnosed with acute myeloid leukaemia (AML) and 20 (control) healthy individuals with no history of malignancy were recruited. Genomic DNA was extracted from peripheral blood. The TERT promoter region and exon 15 were amplified by polymerase chain reaction (PCR) and analysed by Sanger sequencing. Genotype and allele distributions were determined in patients and healthy controls. **Results:** No geneticvariation was detectedin exon 15. In the TERT promoter region, a single variant,NG_009265.1:g.4814T>C (rs2853669), was identified in both groups. The TT genotype was observed in 31.6% of patients and 45.0% of healthy controls, while the TC genotype was observed in 68.4% and 55.0%, respectively. The CC genotype was not detected in either group. The C allele frequency was 34.2% in patients and 27.5% in healthy controls. **Conclusion:** The TERT promoter variant rs2853669 was identified in both acute leukaemia patientsand healthy controls, while no genetic variation was detected in exon 15. These findingsprovide preliminary data on TERT genetic variation in the study population. Larger studies with appropriate statistical analyses are required to determine whether rs2853669 is associated with acute leukaemia.

Keywords:
Acute leukaemia; TERT; rs2853669; telomerase; promotervariant; genetic variation

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PA018

Flightless I as Molecular Target for Inhibition of Radiation-Induced Colorectal Metastasis

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Introduction: Colorectal cancer (CRC) is a leading cause of cancer-related mortality worldwide, characterised by uncontrolled cell proliferation, migration, and invasion, which contribute to metastasis. Invadopodia facilitate cancer cell invasion through extracellular matrix (ECM) degradation. Although radiotherapy is an established treatment for CRC, radiation-induced cellular responses may contribute to tumour progression, metastasis, and radioresistance. Ionising radiation has been reported to induce cytoskeletal rearrangements in cancer cells; however, the underlying molecular mechanisms remain poorly understood. Flightless I (FliI), an actin-binding cytoskeletal protein, has been implicated in cancer progression, but its involvement in radiation-induced CRC migration and invasion remains unclear. Therefore, this study investigated the role of FliI in radiation-induced migration, invasion, and invadopodia formation in HCT116 colorectal cancer cells. **Methods:** FliI expression was suppressed using SMARTpool small interfering RNA (siFliI), and knockdown efficiency was validated by Western blot analysis. Cell migration and invasion were assessed using Transwell assays, while invadopodia-mediated ECM degradation was evaluated using a gelatin degradation assay. **Results:** The findings demonstrated that X-ray irradiation significantly increased FliI protein expression in HCT116 cells. siFliI transfection successfully reduced FliI protein expression by approximately 90%. Notably, FliI silencing combined with irradiation significantly suppressed HCT116 cell migration and invasion compared with irradiated control cells. Furthermore, the gelatin degradation assay revealed significantly reduced matrix degradation activity following FliI silencing, suggesting impaired invadopodia-mediated ECM degradation. However, the molecular signalling pathways underlying FliI-mediated migration and invasion remain to be elucidated. **Conclusion:** Collectively, these findings identify FliI as a potential regulator of radiation-associated invasive behaviour in CRC cells. Suppression of FliI may represent a promising strategy to limit radiation-induced migration and invasion, potentially reducing metastatic progression. Further investigations are warranted to clarify the underlying molecular mechanisms and evaluate the therapeutic relevance of FliI inhibition in CRC radiotherapy.

Keywords:
Flightless I; ionizing radiation; migration and invasion; colorectal cancer

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PA020

Association Between Mitochondrial DNA Mutations and Cellular Senescence-Associated Gene Alterations Across Cancer Types

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Introduction: Mitochondria regulate cellular metabolism and senescence-related signalling. However, links between mitochondrial dysfunction, senescence, and cancer biology remain unclear. This study aimed to investigate associations between mitochondrial DNA (mtDNA) mutations and mutations in cancer-associated cell cycle genes across cancer types. **Methods:** Genomic data from 6,642 patients across nine cancer cohorts were obtained from cBioPortal. Patients were classified by mtDNA mutation status (at least one mutation), and mutations in 20 cancer-associated cell cycle genes were assessed using three approaches: 1) cancer-specific analyses comparing the proportions of patients carrying at least one candidate gene mutation within each cohort; 2) pan-cancer analyses evaluating these associations and gene co-mutation patterns across all patients; and 3) gene-specific analyses comparing the mutation and variant types for each gene according to mtDNA status. All statistical analyses were performed using Fisher's exact test in R. **Results:** Cancer-specific analyses indicated significant associations between mtDNA mutation and TP53 mutations in esophagogastric cancer (73.5% vs. 52.7%, $p = 0.001$), PTEN mutations in hepatobiliary cancer (15.3% vs. 1.5%, $p = 0.01$), NLRX1 mutations in melanoma (33.3% vs. 2.17%, $p = 0.02$), and NLRX1, NAT10, SIRT6 and ATOH8 mutations in myeloid neoplasms (all $p < 0.05$). Overall, mtDNA-mutant patients had considerably higher rates of mutations in these genes. Pan-cancer analyses identified associations between mtDNA mutation and mutations in TP53, KRAS, PTEN, ATM, EPHA2, SIRT6 (all $p < 0.001$) and NLRX1, APAF1, BNIP3 and SMAD3 (all $p < 0.05$). Gene-specific analyses revealed significant differences in mutation and variant types in MYC, TP53 and PTEN according to mtDNA mutation status (all $p < 0.05$). Gene co-mutation patterns were not significantly associated with mtDNA status. **Conclusion:** mtDNA mutation status was statistically associated with mutations in the selected genes, although the biological significance of these associations remains unclear and requires further investigation.

Keywords:
Mitochondrial DNA mutation, Cancer-associated cell cycle genes, Cancer genomics, Pan-cancer analysis

The Pro-Metastasis Effect of CBFβ in MDA-MB-231 Cells

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Introduction: Core-binding factor subunit beta (CBFβ) is a transcription regulator of breast cancer metastasis. Therefore, targeting this protein is promising to suppress the aggressiveness of triple-negative breast cancer (TNBC) cells, especially their migration and invasion. So far, the effect of CBFβ inhibitor (5-ethyl-4-(4-methoxyphenyl)-2-thiazolamine) in TNBC remains unexplored although it has been extensively studied in haematological malignancies. Hence, in this study, we determined the effects of CBFβ inhibitor in impairing the metastatic capacity of MDA-MB-231 cells. **Methods:** First, the MTT assay was performed to detect the viability of MDA-MB-231 cells across a range of concentrations of CBFβ inhibitor from 0 to 213.4 μM. Second, we monitored morphological changes, cellular stress, and detachment via a phase-contrast microscopy. Third, the anti-metastatic effects of CBFβ inhibitor were evaluated by scratch assays followed by the Boyden chamber assays at its sub-lethal concentrations. **Results:** We found that CBFβ inhibitor was not toxic to MDA-MB-231 cells even up to 213.4 μM of concentration. Additionally, the cells maintained their elongated and spindle-shaped, with no evidence of shrinkage, membrane blebbing, or detachment. Functional analyses also indicated that sub-lethal concentrations of CBFβ inhibitor decreased the migration of MDA-MB-231 cells in a time-dependent manner. Specifically, a significant open gap remained after 48 h of treatment with 10 μM (15.50 ± 5.18%) and 100 μM (27.61 ± 3.32%) of CBFβ inhibitor compared to the vehicle control (3.85 ± 1.31%) ($p<0.05$). Furthermore, CBFβ inhibitor suppressed the invasion of MDA-MB-231 cells through the Matrigel matrix in a dose-dependent manner ($p<0.001$). **Conclusion:** CBFβ is required for the migration and invasion of MDA-MB-231 cells. With the data obtained so far, it highlights the potential of CBFβ inhibitor as an anti-metastatic drug, potentially stopping the cancer cells from spreading to distant organs without triggering cell death.

Keywords
Breast cancer, CBFβ inhibitor, Metastasis, MDA-MB-231, TNBC.

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PA031

Establishing an In Vitro Model for Elucidating the Functional Relevance of the Mitochondrial m.3910G>C Mutation

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Introduction: Mitochondrial DNA (mtDNA) mutations impair cellular respiration and have been shown to enable cancer cells to metabolically reprogramme and adapt to resource-scarce environments. Our previous work identified a mutation in the mitochondrial ND1 gene, m.3910G>C, that may influence cancer progression. This study aimed to establish an in vitro model to elucidate the functional impact of m.3910G>C. **Methods:** The mutant gene carrying the C-allele was synthesised and cloned into the PLVX-Puro plasmid vector through sticky-end ligation. The complete plasmid was mass-produced using competent E. coli, sequenced, and lentivirally transduced into HEK293 and MDA-MB-231 cells. Puromycin was then used to isolate transfected cells, and throughout successive subcultures, any morphological changes suggestive of malignant transformation or progression were recorded. To confirm the expression of the mutant gene, total RNA was extracted, converted to cDNA, and used to amplify in quantitative PCR a 142-bp fragment spanning m.3910G>C. **Results and Discussion:** Sanger sequencing confirmed the identity of the cloned plasmid. The transfected cells appeared rounded and stressed after transfection, possibly due to the toxicity of lentiviral particles, but they otherwise showed no signs of acquired or increased malignancy. Melt-curve analysis showed that only the transfected cells expressed the mutant gene, yielding double peaks that indicated a mixture of amplicons originating from both the wildtype and alternate alleles. In contrast, untransfected cells gave a single melt peak, suggesting that only a single allele was present, and the mutant gene was not expressed. **Conclusion:** An in vitro model expressing m.3910G>C was successfully established and will be used in further investigations of mitochondrial dysfunction, drug resistance, and cellular senescence. Given the technical challenge of delivering CRISPR systems effectively into mitochondria, plasmid-based, extramitochondrial expression of mutant genes may be a sensible alternative for investigating the functional relevance of mtDNA mutations.

Keywords
Mitochondrial DNA mutation, cancer development, plasmid transfection, cellular senescence

Exceptionally Selective and Orally Bioavailable p300 Degraders for the treatment of CBP Mutant and p300 Dependent Cancers

Mohamad Fairus A.K.[1], Pratheev A.[1], Krishna Chaitanya T[2], Iram Khan Iqbal[2], Kishore Narayanan[2], Naveen Kumar R[2], Aravind A B[2], Avinash Kumar[2], Adithya Madhukar S[2], Dabbeeru Madhu Babu[2], Pathur Obanna[2], Ankita Manna[2], Rabin Madhaiyan[2], Damodhran B[2], Shraddha B Shirsat[2], Shilpa S Nayak[2], Farhana M. Z.[1], Lim Y. J.[1], Dharani P[2], Suraj T Gore[2], Girish Daginakatte[2], Rajesh Eswarappa[2], Chandrasekhar Abbineni[2], Saravanan Thiyagarajan[2], Murali Ramachandra[2] and Susanta Samajdar[2]*

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Introduction: EP300 (p300) is a histone acetyltransferase and transcriptional co-activator that regulates transcription via chromatin remodelling, acetylating both histone and non-histone proteins and scaffolding transcription factors including p53, c-Myc, ER, and AR. Selective targeting of p300 is expected to yield therapeutic efficacy in CBP-mutant and p300-dependent malignancies while sparing the paralog CBP in normal cells. CBP-mutant cancers span solid and haematological tumours (bladder, lung, colorectal, melanoma, DLBCL, MM), while p300-dependent malignancies include prostate cancer and MYCN-amplified neuroblastoma. Conventional CBP/p300 bromodomain inhibitors lack paralog selectivity due to high sequence homology, causing severe thrombocytopenia clinically. **Methods:** A targeted protein degrader approach was adopted, leveraging a differentiated ternary complex for paralog-selective p300 degradation. Degraders were assessed for potency, selectivity over CBP, BRD4, and the CRBN neo-substrate GSPT1, and antiproliferative activity across CBP-mutant and p300-dependent cell models. Oral bioavailability and tolerability were evaluated in rodents, and efficacy was tested in KARPAS422 (CBP-null) and VCaP (AR-positive) CDX models. **Results:** Potent, first-in-class oral p300 degraders were identified, demonstrating high paralog selectivity over CBP, BRD4, and GSPT1. They induced synthetic lethality in CBP loss-of-function mutant lines, p300-dependent models, including AR-positive prostate cancers with GI50 of 1 – 50 nM. Furthermore, combination with enzalutamide in LNCaP-C42B4 (CRPC) cells showed strong synergy, with a Combo score > 300. The degraders showed good oral bioavailability up to 120 mg/kg (MTD not reached) and were well tolerated in rodents, with no thrombocytopenia. Excellent efficacy was demonstrated in KARPAS422 and VCaP CDX models with > 70% TGI at 3 and 10 mg/kg (QD). **Conclusion:** Highly selective p300 degraders with a desirable profile have been identified. Efforts are in progress toward nominating a development candidate by the end of 2026.

Keywords:
p300 degrader, Synthetic lethality, CBP-mutant

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Oral Presentation

Track/Area 3: Translational Cancer Medicine

Time: 11.05 am – 12.30 pm

Venue: Auditorium, Level 2

Intermittent Dosing with a Second-Generation Tyrosine Kinase Inhibitor Delays T790M Resistance

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Introduction: First-line treatment for patients with advanced EGFR mutation-positive NSCLC is often limited by pre-existing (de novo) EGFR T790M mutation-bearing resistant cells. This study aimed to determine whether intermittent afatinib dosing with a 72-hour treatment-free interval is more effective than daily dosing in delaying resistance development. **Methods:** 3D spheroids were established by co-culturing HCC827 (afatinib-sensitive; EGFR exon19 deletion) and 0.1% NCI H1975 (afatinib-resistant; EGFR T790M) cells using the hanging drop method (6,000 cells in 30 µL per droplet). Spheroids were treated with either daily or intermittent dosing (500 pM afatinib followed by 72-hour treatment-free intervals) for 8 days. In an in vivo xenograft model, tumours were established in NCR-bearing mice, which received either daily or intermittent afatinib treatment for 30 days. Tumour growth was monitored throughout the study, after which the tumours were harvested and analysed by western blot to evaluate the MAPK signalling and by immunofluorescence with EGFR exon19 deletion mutant-specific antibody to assess the enrichment of afatinib-sensitive cells. **Results:** Continuous afatinib dosing produced the greatest size reduction in spheroids, whereas intermittent treatment dosing resulted in larger spheroids than continuous dosing but smaller than control. In vivo, continuous afatinib treatment produced an initial antitumour response; however, the subsequent increase in tumour volume approximately after Day 20 is consistent with the possibility of expansion of pre-existing de novo resistant cell populations. Immunofluorescence analysis of xenograft tumours demonstrated greater enrichment of afatinib-sensitive cells in the intermittent treatment group than in the continuous treatment group. Western blot analysis further indicated that afatinib treatment suppressed activity of the EGFR/MAPK signalling pathway, as evidenced by alterations in EGFR and downstream MAPK pathway proteins in both the continuous and intermittent treatment groups. **Conclusion:** Intermittent afatinib dosing may offer a potential strategy for modulating tumour growth and delaying resistance development in heterogeneous NSCLC.

Keywords:
Non-small lung cancer (NSCLC), 3D spheroid, NCI-N1975 cell, HCC827 cell, afatinib-sensitive cells

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OA014

Navigating the Disparities Pathway: Multi Model Analysis of Targeted Therapy Access for Metastatic Colorectal Cancer

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Introduction: Targeted therapy remains inaccessible to many patients with metastatic colorectal cancer (mCRC), with care pathway breakdowns perpetuated by structural limitations of publicly funded healthcare systems. This study examined socio-demographic and clinical factors in the receipt of targeted therapy in such systems. **Methods:** A retrospective cohort study was conducted across three public institutions (Hospital Seri Manjung, Hospital Raja Permaisuri Bainun Ipoh, and the National Cancer Institute, Malaysia), enrolling patients diagnosed with mCRC between 2017 and 2021 with follow-up through 31 December 2025. Using Andersen's Behavioural Model as a conceptual frame, targeted therapy receipt was modelled with hierarchical logistic regression (adjusted odds ratio, aOR), time to initiation with Cox regression (adjusted hazard ratio, aHR), and initiation net of competing mortality risk with a Fine-Gray model (subdistribution hazard ratio, sHR). A piecewise structural equation model mapped the stepwise route to receipt. **Results:** Of 889 patients enrolled, 247 (27.8%) received targeted therapy, with a 3.1-month survival gain relative to non-recipients ($p < .001$). KRAS mutation testing emerged as the single strongest predictor of receipt throughout every model (aOR 25.92; aHR 6.12; sHR 9.67). Ethnicity showed no direct effect on receipt, but an indirect pathway was evident where Malay and Indian patients from civil-service households raised the likelihood of both being tested for KRAS (estimate 0.52) and ultimately receiving therapy (estimate 0.57). However, diagnosed at a public hospital carried no direct effect on receipt, yet it lowered the probability of KRAS testing (estimate -1.69, $p < .001$). **Conclusion:** This study found that civil-service household status emerged as a structural determinant of therapy access by increasing the likelihood of KRAS testing and subsequent targeted therapy receipt. This system-level advantage translated into survival differences, underscoring how non-clinical levers within the public healthcare system can perpetuate inequities in cancer care

Keywords
Metastatic colorectal cancer, targeted therapy, KRAS testing, disparities, public healthcare

Practices, Barriers, and Enablers to Enhanced Recovery After Surgery (ERAS) Implementation in Upper Gastrointestinal Surgery: A Malaysian Multicentre Qualitative Study

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Introduction: Surgery is a cornerstone of curative treatment for benign and malignant upper gastrointestinal (UGI) conditions, including resectable cancers, yet carries substantial risks of complications, malnutrition and delayed recovery. Enhanced recovery after surgery (ERAS) protocols, encompassing preoperative counselling, nutritional optimisation, and early feeding and mobilisation, have been shown to attenuate the surgical stress response and expedite recovery, linking curative treatment with supportive care, including for patients with cancer. However, ERAS adoption across Malaysia remains inconsistent, which may limit equitable access to evidence-based perioperative care. This study explored current ERAS practices, barriers and enablers perceived by multidisciplinary teams implementing ERAS for UGI surgery across Malaysian centres. **Methods:** A qualitative study was conducted using semi-structured, in-depth online interviews with surgeons, anaesthetists, dietitians and nurses, recruited through purposive and snowball sampling from six Malaysian UGI centres. Interviews were audio/video-recorded, transcribed verbatim and analysed thematically using a six-step framework in Atlas.ti software. **Results:** Twenty-four participants were interviewed. ERAS practices clustered into two themes: (1) partial and inconsistent implementation, and (2) the absence of a unified, written protocol across centres. Barriers clustered into four themes: (1) workforce instability, (2) professional hierarchy and team dynamics, (3) fragmented standardisation and training, and (4) resource and patient-related constraints. Enablers centred on three themes: (1) multidisciplinary teamwork, (2) workforce capability, and (3) institutional support through protocol standardisation and dedicated funding. **Conclusion:** ERAS implementation in Malaysian UGI surgery remains constrained by workforce, hierarchical and structural barriers, which may perpetuate variation in perioperative care between centres. Multidisciplinary collaboration, targeted training and institutional funding can strengthen implementation. These findings support a standardised, contextually appropriate ERAS implementation protocol for Malaysian UGI centres and offer transferable insights for advancing equitable perioperative and supportive care, including cancer care, across the region.

Keywords
Enhanced recovery after surgery, Upper gastrointestinal surgery, Perioperative care, Nutrition support, Qualitative

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OA020

Mapping the Nutritional Phenotypes of Accelerated Biological Ageing in Breast Cancer Survivors: An Explainable Machine Learning Approach

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Introduction: The interconnected variables driving accelerated biological ageing in breast cancer survivorship remain poorly mapped. This study deployed explainable machine learning architectures to untangle the non-linear interplay between nutrition, lifestyle, and telomere attrition. **Methods:** As part of the C*STAR study, a cross-sectional analysis was conducted among 262 breast cancer survivors and 262 cancer-free controls. The study assessed four multidimensional variable groups, including ageing phenotypes, behavioural lifestyle factors, sociodemographic profiles, and clinical characteristics. Absolute telomere length, utilised as the primary ageing biomarker, was quantified via quantitative real-time polymerase chain reaction (qPCR). To map non-linear associations, an Extreme Gradient Boosting (XGBoost) model integrated with the Synthetic Minority Over-sampling Technique for Regression with Gaussian Noise (SMOGN) was optimized via Particle Swarm Optimisation. SHapley Additive exPlanations (SHAP) evaluated feature importance. **Results:** The XGBoost-SMOGN pipeline substantially reduced the mean absolute error compared to native predictive models. SHAP analysis showed that accelerated biological ageing (shortened telomeres) in survivors was uniquely associated with higher processed meat consumption, lower potassium intake, radiotherapy, and impaired functional status. This distinct post-treatment profile contrasted fundamentally with the dietary drivers of telomere attrition observed in cancer-free controls. **Conclusion:** Explainable machine learning provides a robust exploratory framework to decipher the complex, interrelated variables associated with telomere attrition. Isolating true nutritional signals from clinical noise helps identify highly vulnerable survivors, paving the way for targeted, personalised survivorship care to preserve telomere health.

Keywords:
Age Acceleration, Ageing Phenotypes, Behaviour, Telomere Length, Machine Learning

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Oral Presentation

Track/Area 4: Clinical Trials of New Cancer Drugs/Precision Oncology

Time: 11.05 am – 12.30 pm

Venue: Bilik Seminar 4, Level 1

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OA005

High-Dose Methotrexate Pharmacokinetics in Pediatric Acute Lymphoblastic Leukemia in Indonesia: Correlation with Renal Function and SLCO1B1 Polymorphism

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Introduction: Safe use of high-dose methotrexate (HD-MTX) requires close drug monitoring (TDM), but this is often unavailable in resource-limited settings like Indonesia. As a result, methotrexate pharmacokinetic (PK) profiles in these populations remain poorly characterized, as does the extent to which variation in drug levels arises from clinical or genetic factors. This study describes the correlation between MTX blood levels, kidney function, toxicity, and SLCO1B1 gene polymorphism in Indonesian children with acute lymphoblastic leukemia (ALL) receiving HD-MTX. **Methods:** Seventy-nine pediatric ALL patients were included. MTX levels were measured at 24 and 42 hours using LC-MS/MS, and clearance was estimated with a one-compartment model. Two SLCO1B1 variants (rs2306283, rs4149056) were genotyped, and toxicity was graded using CTCAE v5.0. Regression analysis tested whether clinical factors, genotype, and kidney function explained variation in drug levels and clearance. **Results:** Doses were already reduced in practice, averaging 76% of the standard BSA-based dose. Even so, 22.8% of patients showed delayed drug clearance (C42 ≥1 µmol/L), meaning dose reduction alone was not enough to prevent risk. Kidney function was linked to C42 levels (SCr: $r = 0.358$, $p = 0.001$; eGFR: $r = -0.252$, $p = 0.025$). SLCO1B1 variants affected early drug levels (C24) and clearance, but not later elimination (C42), suggesting that genetics drives early exposure while kidney function governs later elimination. However, a model combining dose, BSA, creatinine, and each genotype explained only 17.4% of C24 variation ($R^2 = 0.174$), while a model combining log(C24) with SCr explained 35% of C42 variation ($R^2 = 0.350$). **Conclusion:** Delayed MTX clearance still occurs even with reduced doses, and neither clinical nor genetic factors reliably predict individual drug exposure. This underscores an urgent need for accessible TDM, ideally with monitoring at multiple time points, to make HD-MTX therapy safer and more personalized in resource-limited like Indonesia.

Keywords:
Methotrexate, Acute Lymphoblastic Leukemia, Therapeutic Drug Monitoring, Toxicity, Personalized Medicine

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OA006

Population Pharmacokinetic Model-Based Risk Stratification Strategy for High-Dose Methotrexate in Malaysian Adults with Haematological Malignancies

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Introduction: High-dose methotrexate (HDMTX) demonstrates substantial interindividual pharmacokinetic variability, which may influence the risk of delayed elimination and toxicity. Population pharmacokinetic (PopPK) modelling can characterize sources of this variability and support simulation of clinically relevant exposure outcomes. This study aimed to develop a PopPK model of HDMTX using routine clinical data from Malaysian adults with haematological malignancies and to evaluate the influence of patient characteristics on delayed MTX elimination through model-based simulations. **Methods:** Plasma methotrexate (MTX) concentrations at 48, 72, and 96 h were retrospectively collected from adult patient receiving >500 mg/m^2 MTX between August 2024 to Jun 2025. PopPK model was developed using Monolix, including demographic characteristics, renal and liver function parameters, haematocrit, treatment protocol, and infusion duration as covariates. The final model was implemented in Simulx to simulate MTX exposure across albumin concentrations of 14–39 g/L and HDMTX doses of 750–7000 mg, at different infusion duration. **Results:** A total of 91 HDMTX administrations, comprising 206 concentration measurements were included. A two-compartment model with first-order elimination and combined residual error adequately described the observed data, with albumin as a significant predictor of MTX clearance. The final clearance model was described as CL (L/h) = 11.13 × (Albumin/39)$^{0.7}$. Model-based simulations demonstrated a greater probability of MTX concentrations remaining ≥0.2 µmol/L at 48 h and a longer time to reach 1.0 µmol/L at lower albumin concentrations, with the effect becoming more pronounced at higher HDMTX doses administered over longer duration of infusion. **Conclusion:** A PopPK model describing HDMTX disposition in Malaysian adults with haematological malignancies was successfully developed using routinely collected clinical data. Albumin was identified as an important determinant of MTX clearance. Model-based simulations further demonstrated how albumin plays role in early risk stratification and individualized monitoring of patients receiving HDMTX.

Keywords: popPK, pharmacokinetic, methotrexate, MTX, HDMTX

Exploration of Factors Influencing the Efficacy of Second-Line Osimertinib in NSCLC Patients

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Introduction: Epidermal growth factor receptor (EGFR) mutations are among the most common oncogenic drivers in non-small cell lung cancer (NSCLC). Osimertinib, a third-generation EGFR-TKI, is widely used as a second-line treatment after progression on prior therapies. This study primarily aimed to determine the impact of concurrent gene mutations on osimertinib efficacy, while also accounting for other common clinical factors, including age, sex, smoking status, alcohol consumption, and the neutrophil-to-lymphocyte ratio (NLR). Furthermore, predictive models were established to facilitate individualised therapeutic decision-making. **Methods:** A multicentre observational study of 198 EGFR-mutant NSCLC patients receiving second-line osimertinib was conducted. Concurrent genomic alterations were detected by next-generation sequencing (NGS) or polymerase chain reaction (PCR). Median progression-free survival (mPFS) was evaluated using survival analyses and Cox regression, and a prognostic nomogram was developed and internally validated. **Results:** The overall mPFS was 11 months (95% CI: 10.057-11.943). Concurrent gene mutations were identified in 12% of patients, the most common being TP53 (6.06%), PIK3CA (1.52%), and MET (1.01%). In non-parametric tests, patients with concurrent mutations did not show a statistically significant difference in the mPFS compared to those without (10.0 months, 95% CI: 8.042-11.958 vs 11.0 months, 95% CI: 9.932-12.068; p = 0.324). Other subgroup analyses also showed no significant differences. The Kaplan-Meier survival analysis indicated a trend towards shorter PFS in the concurrent mutation group, although this did not reach statistical significance (log-rank test, p = 0.131). Patients with EGFR mutations harbouring only concurrent TP53 mutations had the shortest PFS (6.0 months, 95% CI: 1.684-10.316; p = 0.059). Based on the multivariable Cox regression analysis, age, NLR, and concurrent gene mutation status were included in the prognostic model. Internal validation demonstrated acceptable discrimination and calibration, with an area under the ROC curve (AUC) of 0.649 in the training set and 0.638 in the validation set. **Conclusions:** Concurrent gene mutations were closely associated with shorter PFS in patients with EGFR-mutant NSCLC receiving second-line osimertinib. A predictive model incorporating concurrent genomic status, age, and NLR was developed to indicate potential clinical applicability.

Keywords:
NSCLC, EGFR mutation, Concurrent gene mutations, Osimertinib, PFS, Prediction model

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Poster Presentation

Time: 10.35 am – 11.05 am

Venue: Open Area, Level 1

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PA004

Virtual Screening of FDA-Approved Drugs Targeting the Allosteric Pocket of Resistant Tertiary C797S Mutant EGFR in Non-Small Cell Lung Cancer

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Introduction: Lung cancer is the leading cause of cancer death worldwide, with non-small cell lung cancer (NSCLC) accounting for most cases. Although targetable EGFR mutations initially sensitize tumours to tyrosine kinase inhibitors (TKIs), secondary and tertiary mutations such as T790M and C797S drive resistance across TKI generations. Third-generation EGFR-TKIs, such as osimertinib, improve outcomes; however, acquired resistance remains a major clinical challenge. Effective therapeutic options to overcome late-stage TKI resistance remain limited. Targeting non-ATP allosteric sites may bypass orthosteric site resistance. Drug repurposing provides a cost-effective approach to identify allosteric modulators. In this study, an in silico virtual screening protocol was applied to identify FDA-approved drugs targeting the allosteric site of mutant EGFR. **Methods:** FDA-approved drugs were virtually screened against the allosteric pocket of tertiary C797S mutant EGFR using the DrugRep server. Top candidates underwent CDOCKER molecular docking in Discovery Studio. Their binding interactions and energy scores were compared between four allosteric protein structures [7LTX (T790M/V948R), 5D41, 7JXQ (T790M/V948R), 7K1I (L858R/V948R)] and four ATP-binding structures [5UGC (L858R/T790M/V948R), 6LUD (L858R/T790M/C797S), 5UG9 (L858R/T790M/V948R), 6VHN]. Independent scoring functions were utilized to rank candidates and assess binding affinity and stability. **Results:** Virtual screening and docking identified one candidate drug that showed favourable interaction energies at the allosteric site (ranging from -58.5 to -66.5 kcal/mol) compared to the ATP-binding site (-45.7 to -50.2 kcal/mol). This drug consistently ranked first across three independent scoring protocols, Normalizing Scoring Analysis, Z-Score Normalization Analysis, and Consensus Scoring Analysis, showing strong preference for the allosteric site and binding stability over all other screened candidates. **Conclusions:** In silico screening and docking identified a potential FDA-approved drug candidate with high affinity and favourable predicted binding to the tertiary C797S mutant EGFR allosteric site. Drug repurposing represents a viable strategy to overcome TKI resistance in NSCLC, which warrants further functional validation.

Keywords: Drug Repurposing, EGFR Allosteric Site, Virtual Screening, Non-Small Cell Lung Cancer, Molecular Docking

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PA005

Harnessing Antineoplastic Properties of Halicin against Hepatocellular Carcinoma

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Introduction: Hepatocellular carcinoma (HCC) remains a major global health problem, contributing to over 80% of primary liver cancers. Without effective therapeutic interventions, HCC incidence is projected to rise to 1.52 million, with 1.37 million deaths by 2050 globally. Stemness and drug resistance persist as key barriers to treatment, demanding new therapeutic approaches. Drug repurposing offers a promising alternative in anticancer drug discovery, owing to its established safety, cost effectiveness and reduced development timeline. Here, we repurposed Halicin; a JNK inhibitor, and a broad-spectrum antibiotic, as a neoplastic agent against HCC. **Methods:** The anti-cancer effects of Halicin were evaluated against HCC cell lines (HepG2 and HCCLM3), assessing its impact on cell proliferation, apoptosis, autophagy, epithelial-mesenchymal transition (EMT) and stemness. Furthermore, autophagy-apoptosis crosstalk was studied to evaluate the underlying nature of Halicin-induced autophagy. **Results:** We provide the first evidence that Halicin exerts potent anticancer effects against HCC by inhibiting cell proliferation and inducing apoptosis. It also triggered autophagy and enhanced lysosomal function. Ongoing studies are investigating its effects on EMT and stemness, along with the intricate interplay between autophagy and apoptosis, to elucidate key mechanisms underlying Halicin-induced HCC cell death. **Conclusion:** Together, these findings support that Halicin may act as a promising candidate for HCC treatment, offering renewed hope for improved therapeutic outcomes. Further preclinical evaluation of its safety and efficacy may advance it clinical translation.

Keywords:
Hepatocellular carcinoma, Halicin, Apoptosis, Autophagy

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PA010

Novel Thioquinazolinone Derivatives: Design, Synthesis and Biological Evaluation Against EGFR-Mutant Non-Small Cell Lung Cancer

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Introduction: The emergence of resistance to epidermal growth factor receptor (EGFR)-targeted therapies remains a major challenge in the treatment of non-small cell lung cancer (NSCLC). The quinazoline scaffold is a well-established pharmacophore in EGFR-targeted anticancer agents, and incorporation of a thione functionality may provide an opportunity to modulate the biological and physicochemical properties of the scaffold. Therefore, thioquinazolinone derivatives were investigated as potential candidates against EGFR-mutant NSCLC. **Methods:** Four novel thioquinazolinone derivatives MSH1-MSH4 were synthesized through a multistep synthetic route. Four novel thioquinazolinone derivatives were synthesized through a multistep synthetic approach. The synthesized compounds were characterized by thin-layer chromatography (TLC), melting point determination, ^{1}H NMR, ^{13}C NMR, and mass spectrometry.The synthesized compounds were characterized by TLC, melting point determination, ^{1}H NMR, ^{13}C NMR, and mass spectrometry. Their in vitro antiproliferative activities were evaluated against H1975 cells harboring EGFR L858R/T790M mutations, PC9 and HCC827 cells harboring EGFR exon 19 deletion, and normal human bronchial epithelial BEAS-2B cells using the MTT assay. **Results:** All target compounds were successfully synthesized and structurally characterized. MSH1 exhibited the greatest antiproliferative activity, with IC_{50} values of 14.0 ± 1.7 μM against H1975 and 13.7 ± 1.2 μM against PC9, while showing minimal cytotoxicity toward BEAS-2B cells (IC_{50} >100 μM). MSH2 showed moderate activity against H1975 (15.0 ± 2.0 μM) and PC9 (28.3 ± 4.0 μM), whereas MSH3 displayed reduced activity against H1975 (21.7 ± 3.1 μM) and was inactive against PC9 (IC_{50} >100 μM). MSH4 exhibited moderate antiproliferative activity against H1975 (31.3 ± 2.5 μM) and PC9 (28.7 ± 2.5 μM), with low cytotoxicity toward normal BEAS-2B cells (IC_{50} >100 μM). SAR demonstrated that the unsubstituted thioquinazolinone scaffold (MSH1) was optimal for antiproliferative activity. Progressive N-3 functionalization from the parent compound to the ester (MSH2), hydrazide (MSH3), and cyclized thiazolidinone derivative (MSH4) resulted in a gradual reduction in biological activity, indicating that increasing steric and structural complexity at the N-3 position was unfavorable for EGFR-mutant NSCLC inhibition. **Conclusion:** A series of novel thioquinazolinone derivatives was successfully synthesized and biologically evaluated against EGFR-mutant NSCLC cell lines. Preliminary SAR revealed that maintaining the parent thioquinazolinone framework was more favorable than N-3 structural modification, with MSH1 emerging as the most potent and selective analogue. These findings provide valuable guidance for the rational optimization of thioquinazolinone-based EGFR-targeted anticancer agents.

Keywords:
Thioquinazolinone derivatives; EGFR-mutant NSCLC; Antiproliferative activity; Structure–activity relationship (SAR); MTT assay.

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PA011

Hellebrigenol Suppresses Hepatocellular Carcinoma through Targeting β-Catenin Signalling

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Introduction: Hepatocellular carcinoma (HCC) is a highly metastatic and aggressive malignancy that is frequently diagnosed at advanced stages and is linked with poor prognosis. Aberrant activation of β-catenin signalling promotes stemness, therapeutic resistance leading to HCC aggressiveness. Here, we investigated the antineoplastic effects of Hellebrigenol (HBGL), a metabolite extracted from Bufadienolides for targeting β-catenin signalling in HCC. **Methods:** The effects of HBGL on HCC cell proliferation and viability were evaluated using MTT, colony formation and Calcein AM/ethidium homodimer-1 staining assays. Western blotting, Annexin V/PI staining, and Caspase-3/7 activity were performed to analyse the HBGL-induced apoptosis in HCC. Wound-healing, migration, invasion, and sphere-formation assays were used to measure the effects of HBGL on EMT and stemness. Acridine orange, LysoTracker staining and Western blotting were performed to analyse HBGL induced autophagy. Moreover, MTT, colony-formation, Caspase-3/7 and Western blotting techniques were carried out in the presence of autophagy-blockers to determine the nature of HBGL-induced autophagy in determining HCC cell fate. **Results:** HBGL exhibited potent antiproliferative activity against HCC, as evidenced by MTT and colony-formation assays. Dose-dependent treatment with HBGL significantly triggered apoptosis and autophagy while suppressing EMT and stemness in HCC. Mechanistically, these anti-tumour effects of HBGL were associated with the suppression of β-catenin signalling in HCC and can be considered as a critical molecular target for HCC regression. **Conclusion:** Overall, our findings suggest that HBGL exerts a potential antitumor effect in HCC through targeting β-catenin signalling and may offer a promising therapeutic approach for HCC treatment.

Keywords:
Hepatocellular carcinoma, Hellebrigenol, Apoptosis, Stemness, β-Catenin

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PA012

Discovery and Characterization of Once in a Month Intravenous and Orally Bioavailable SMARCA2 Degraders for Treating SMARCA4 Mutant Cancers

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Introduction: The BAF (SWI/SNF) chromatin remodelling complex regulates chromatin accessibility through ATP-dependent nucleosome repositioning, modulating transcription, DNA recombination and repair, and mitotic chromosome segregation. Its catalytic activity is mediated by two mutually exclusive ATPases, SMARCA2 (BRM) and SMARCA4 (BRG1). In SMARCA4-deficient tumours, oncogenic dependency shifts to the residual SMARCA2-containing SWI/SNF complex, establishing a synthetic lethal vulnerability validated through genetic silencing studies. This positions SMARCA2 as a compelling therapeutic target in SMARCA4-mutant cancers. **Methods:** We designed highly potent and selective SMARCA2 degraders by optimizing assemblies of SMARCA2/4 bromodomain binders, degron-linking moieties, and E3 ligase ligands. Design prioritization was guided by our proprietary ternary complex modelling algorithm, ALMOND (ALgorithm for MOdeling Neosubstrate Degraders). Selected compounds were synthesized and evaluated across multiple cellular assays to assess mechanistic and functional outcomes. Subsequent ADME optimization and long-acting injectable (LAI) formulation development were also performed. **Results:** Several compounds demonstrated potent and selective SMARCA2 degradation, eliciting distinct phenotypic responses in a panel of SMARCA4-mutant cell lines. The lead IV compound showed robust anti-tumour efficacy in multiple SMARCA4-mutant xenograft models with confirmed target degradation at well-tolerated doses; its IND application was accepted by the US FDA. ADME optimization yielded compounds with favourable oral bioavailability across tested species. The lead degrader exhibited robust antitumor efficacy in multiple SMARCA4-deficient CDX models at well-tolerated doses. An LAI formulation for the IV candidate demonstrated tolerability and efficacy with once-monthly dosing. **Conclusion:** Rationally designed SMARCA2 degraders deliver potent, selective target degradation and robust anti-tumour efficacy in SMARCA4-deficient preclinical models at well-tolerated doses. With an FDA-accepted IND, favourable oral bioavailability, and an LAI formulation enabling an infrequent dosing regimen, these degraders represent a promising therapeutic strategy for SMARCA4-mutant cancers.

Keywords:
SMARCA2 degrader, PROTAC, SWI/SNF, Synthetic lethality, SMARCA4-mutant

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PA025

Drug Repurposing for EGFR-Mutated Non-Small Cell Lung Cancer: Computational and Experimental Evaluation of FDA-Approved Drugs

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Introduction: Non-small cell lung cancer (NSCLC) has statistically contributed to 85% of lung cancer cases and remains a leading cause of cancer-related mortality worldwide. The mutations in epidermal growth factor receptor (EGFR), including exon 19 deletion and T790M, play significant roles in therapeutic response studies involving tyrosine kinase inhibitors (TKIs). As resistant mutations occur, first- and second-generation TKIs no longer work effectively, leading to the use of third-generation TKIs such as osimertinib. However, the affordability of osimertinib has become a major challenge in the healthcare industry, especially in low- and middle-income countries (LMICs). Drug repurposing has emerged as a promising strategy to accelerate the discovery of novel anticancer agents by identifying new therapeutic applications for existing drugs. This study aims to evaluate the anticancer potential of FDA-approved drugs against EGFR-mutated NSCLC through computational and experimental approaches, including in silico, in vitro, in ovo, and in vivo. **Methods:** 3,067 FDA-approved drugs were screened using DrugRep, identifying 99 and 204 compounds to show binding affinity toward EGFR T790M and exon 19 deletion mutations, respectively. Drugs with prior anticancer reports, serious adverse effects, CNS activity, or limited suitability for long-term use were excluded, leaving Drugs A and B for further evaluation. The molecular docking was performed by preparing mutant EGFR protein using BIOVIA Discovery Studio, and docking was executed using PyRx. The growth inhibitory effects of the drugs were assessed using the MTT assay in HCC827 (EGFR Del19) and NCI-H1975 (EGFR L858R/T790M) cell lines. Western blot assay was used to investigate the effects of the selected drug on key signalling pathways. **Results:** The molecular docking results showed that Drug A has higher binding affinity towards the allosteric site of mutated EGFR at -8.6 kcal/mol, and inhibited the growth of HCC827 and NCI-H1975 cell lines, with mean IC_{50} values of 11.6 µM and 16.0 µM, respectively. Western blot assay demonstrated that Drug A suppressed EGFR signalling by reducing the activation of EGFR. **Conclusion:** This study highlights Drug A to have anticancer effects on EGFR-mutated NSCLC cells, suggesting its potential as a therapeutic candidate for this cancer. Further testing of the drug involving Western blot assay of the EGFR signalling downstream pathway, in ovo chorioallantoic membrane (CAM), and in vivo xenograft studies are required to validate the antitumour activity of the drug.

Keywords: NSCLC, drug repurposing, EGFR, mutation, tyrosine kinase inhibitors

Liposomal FASN Inhibitor as a Potential Inhalable Drug Delivery System for Non-Small Cell Lung Cancer

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Lung cancer remains one of the most commonly diagnosed cancers worldwide and is the leading cause of cancer-related mortality, accounting for more than 1.8 million deaths annually. Despite advances in treatment, its management remains challenging due to limited therapeutic selectivity and adverse effects associated with conventional therapies. Therefore, targeted drug delivery strategies that enhance drug availability at the tumour site while minimising systemic exposure are highly desirable. Fatty acid synthase (FASN), a key enzyme involved in de novo fatty acid synthesis, is frequently overexpressed in various cancers, including lung cancer, and is associated with tumour progression, poor prognosis and therapeutic resistance. We utilized an FDA-approved anti-obesity drug, exhibits inhibitory activity against FASN, highlighting its potential for cancer treatment. However, this FASN inhibitor is also known of poor oral bioavailability, limiting its therapeutic application. Pulmonary administration of liposomal FASN inhibitor via inhalation may provide an alternative approach to improve local drug delivery and pulmonary drug availability. Our study aimed to evaluate the potential of a liposomal formulation to improve the delivery characteristics of this FASN inhibitor for potential application in non-small cell lung cancer (NSCLC) treatment. In vitro findings demonstrated that liposomal FASN inhibitor exhibited sustained release compared with free FASN inhibitor. Cellular uptake studies using Nile Red as a fluorescent marker demonstrated differences in the release and uptake characteristics between liposomal and free forms. Furthermore, Franz diffusion studies showed negligible release of free FASN inhibitor in phosphate-buffered saline (pH 7.4) containing 3% sodium lauryl sulfate (SLS), whereas liposomal FASN inhibitor demonstrated sustained release for up to 10 hours. Similar sustained-release characteristics were observed in acetate buffer (pH 5.6) containing 3% SLS. In vivo biodistribution analysis in BALB/c mice following nose-only inhalation showed significantly higher maximum lung concentration (C_{max}) and shorter time to reach maximum concentration (T_{max}) for liposomal FASN inhibitor compared to its free counterpart. A biphasic retention profile was observed, indicating efficient initial deposition and prolonged residence in lung tissue. Overall, these preliminary findings support further investigation of formulated FASN inhibitor as a potential inhalable drug delivery system for NSCLC treatment.

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Poster Presentation

Time: 1.10 pm – 1.35 pm

Venue: Open Area, Level 1

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PA013

Anticancer Effects of a 1,4-Naphthoquinone Derivative in Hepatocellular Carcinoma Mediated by Inhibition of STAT3 Signaling

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Introduction: Hepatocellular carcinoma (HCC) is one of the most prevalent and lethal malignancies worldwide, and its incidence and mortality continue to impose a substantial global health burden. Although considerable progress has been made in targeted therapies and immunotherapies, their clinical efficacy is frequently limited by intrinsic or acquired drug resistance. Therefore, identifying novel compounds with potent antitumor activity and elucidating their mechanisms of action remain urgent priorities. In this study, we synthesized a novel 1,4-naphthoquinone derivative and systematically evaluated its therapeutic potential against HCC. **Methods:** A novel 1,4-naphthoquinones derivative weas synthesized starting from 2-hydroxynaphthalene-1,4-dione. HepG2 and HCCLM3 cells were used to assess the effects of the compound in vitro. The key molecular pathways responsible for compound-induced HCC cell death are also being investigated, with a particular focus on STAT3 signal pathway. **Results:** The structure of this compound was confirmed by ^{1}H NMR, ^{13}C NMR and LC-MS analyses. The compound exhibited potent anti-HCC activity, with IC_{50} values ranging from 1 to 2 μM. The compound effectively inhibited IL-6-induced STAT3 phosphorylation at Tyr705 in HCC cells. **Conclusion:** These preliminary findings demonstrate that the synthesized 1,4-naphthoquinone derivative possesses promising anti-HCC activity and may represent a potential lead compound for HCC treatment. Nevertheless, more comprehensive mechanistic studies, pharmacological evaluations, and in vivo validation are required to establish its therapeutic efficacy, safety, and translational potential.

Keywords:
Hepatocellular carcinoma, Therapeutics, 1,4-Naphthoquinone derivative, STAT3

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PA014

In Silico Drug Repurposing Targeting the Allosteric Domain of Mutant EGFR in Non-Small Cell Lung Cancer

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Introduction: Non-small cell lung cancer (NSCLC) has the highest mortality rate among major cancers. A significant clinical challenge in the treatment of NSCLC is abnormal activation through mutant EGFR. Due to sustained conformational changes in mutant EGFR receptors, current first, and second generation inhibitors often lose therapeutic efficacy, leading to uncontrolled cell proliferation. To bypass the mutated active sites that cause resistance, targeting the allosteric domain of EGFR has emerged as a critical therapeutic strategy. Allosteric inhibition blocks kinase activity and stabilizes the entire kinase domain without displacing ATP. Because traditional drug discovery can be slow and expensive, drug repurposing has proven to be a highly efficient technique to identify new treatment options with minimal capital investment. Therefore, this study aimed to accelerate the drug development process cost-effectively by repurposing existing Food and Drug Administration (FDA)-approved drugs into potential allosteric EGFR inhibitors. **Methods:** Molecular docking was employed to screen a curated library of FDA -approved drugs against the allosteric pocket of the EGFR L858R/T790M double mutant (PDB ID: 5D41) and the active ATP-binding pocket of the EGFR exon-19 (del-747-749) mutant (PDB ID: 7TVD) proteins. To evaluate the binding affinity and interaction potential of the compounds, molecular docking was performed using AutoDock Vina software. Virtual screening was performed to prioritize candidate compounds for further evaluation. Five hit drugs were identified, and from there, two proceeded for this study. **Results:** Virtual screening successfully identified the top candidate compounds, which demonstrated the highest stability and optimal interactions with the receptor. These compounds at the EGFR L858R/T790M allosteric binding site (PDB: 5D41) revealed that Drug 1 (-8.2 kcal/mol) and Drug 2 (-8.1 kcal/mol) exhibited stronger binding affinities than the reference allosteric inhibitor EAI045 (-7.4 kcal/mol). Against 7TVD, Drug 2 (−8.2 kcal/mol) and Drug 1 (−6.7 kcal/mol) were compared with the control osimertinib (−7.2 kcal/mol) Both drugs formed consistent hydrogen bonds (Glu1004), and hydrophobic interactions with (Met1002, Pro741 Lys846, Lys745 and Ala743) within the EAI045 allosteric and 7TVD ATP binding pocket. **Conclusion:** By utilizing advanced computational drug screening, this study demonstrated that several existing FDA-approved drugs hold significant potential to be repurposed as allosteric and ATP-binding EGFR inhibitors. These findings outline a rapid and cost-effective avenue for discovering new pharmacotherapies to overcome life-threatening drug resistance in mutant EGFR in NSCLC and highlight promising candidates that warrant immediate in vitro and in vivo evaluation.

Keywords:
non-small cell lung cancer (NSCLC); Epidermal Growth Factor Receptor (EGFR) mutations; drug repurposing; molecular docking; and in silico screening.

Modulation of Autophagy in Pancreatic Cancer Cells by a Semisynthetic Andrographolide Derivative

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Introduction: Pancreatic cancer is an aggressive disease with limited effective treatment options worldwide. Andrographolide has been reported to possess various pharmacological effects, including anticancer activity. SRJ23, a semisynthetic derivative of andrographolide, also exhibits anticancer properties. Because autophagy is an intracellular self-degradation mechanism that may contribute to tumour growth, this study aimed to investigate the regulation of SRJ23-induced autophagy in pancreatic cancer cells. **Methods:** The effect of SRJ23 on cell viability and autophagy was evaluated using the pancreatic cancer PANC-1 cell line. Cell viability was determined by measuring formazan absorbance at 550 nm using the MTT assay. Activation and expression of LC3-II were evaluated to assess autophagic activity. The autophagy inhibitor 3-methyladenine (3-MA) was combined with SRJ23 to compare its activity with SRJ23 alone. Morphological changes were examined, and RT-PCR was used to analyze LC3-II gene expression. **Results:** SRJ23 reduced the viability of PANC-1 cells, with an IC_{50} value of 4.03 ± 0.09 µM. Inhibition of autophagy enhances SRJ23 cytotoxicity: Combining SRJ23 with 3-MA, an autophagy inhibitor, reduced the IC_{50} from 4.03 to 2.57 ± 0.20 µM, suggesting that autophagy inhibition makes PANC-1 cells more sensitive to SRJ23. Morphological examination showed a brighter green signal in cells treated with SRJ23 alone compared with combination treatment. RT-PCR analysis showed the highest expression of the LC3-II gene in cells treated with 2.5 µM SRJ23. However, a decreasing trend in LC3-II expression was observed from 5 µM onwards, indicating reduced expression at concentrations above the IC_{50} value. **Conclusion:** SRJ23 induces cytotoxicity and protective autophagy in PANC-1 cells. Enhanced cytotoxicity following autophagy inhibition indicates that SRJ23-induced autophagy represents a protective cellular response. At concentrations above the IC_{50}, the decline in LC3-II expression reflects attenuation of autophagy, potentially reflecting increased cellular damage.

Keywords:
Andrographolide, SRJ23, Autophagy, Pancreatic cancer cell line (PANC-1), LC3-II protein

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PA017

Anticancer Effects of a Combination of Sotorasib and Osimertinib on A549 Non-Small Lung Cancer Cell Line

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Introduction: Non-small cell lung cancer (NSCLC) was identified as one of the common types of lung cancer, which comprises of squamous cell lung cancer, adenocarcinoma, and large cell lung carcinoma. Mutations in the tyrosine kinase receptors EGFR and KRAS genes lead to uncontrolled cell division linked to NSCLC. Sotorasib and Osimertinib are drugs used for the treatment of NSCLC mutations. Osimertinib, a mono-aniline-pyrimidine drug, selectively targets a variety of EGFR mutations acting as a covalent EGFR tyrosine kinase inhibitor. Sotorasib, a covalent inhibitor, selectively targets and inhibits the KRAS G12C mutations that promotes cellular proliferation. Combination therapy is a form of treatment combining two or more therapeutic drugs, a crucial part of cancer treatment since it generally targets crucial pathways in an additive or synergistic way; the combination of anticancer drugs is expected to have higher efficacy than monotherapy. This study evaluated the combined anticancer effects of Sotorasib and Osimertinib in A549 NSCLC cells harbouring wild-type EGFR and a KRAS G12C mutation. **Methods:** The A549 cells were seeded in 96-well plate, incubated overnight and treated with test compounds at concentrations ranging from 0.1mM to 100mM. The growth-inhibitory effects of the compounds were evaluated by an MTT cell viability assay. Two designs of combination study were prepared using IC_{50} values of both compounds. First design had Osimertinib at IC_{50} mixed with Sotorasib at varying concentration (0.00001 mM to 100 mM) and second design had Sotorasib at IC50 mixed with Osimertinib at varying concentration (0.00001 mM to 100mM). The drug combination effects (combination index, CI) were assessed using the Chou-Talalay method. **Results:** The results showed that the IC_{50} value of Osimertinib was lower (8.9mM) compared to Sotorasib (60mM). The drug combination designs showed lower IC_{50} values. The first design combination study containing Osimertinib with 60mM Sotorasib had IC_{50} value of 9 mM, whereas the second design combination study containing Sotorasib with 8.9 mM Osimertinib has IC_{50} value of 0.00001mM. The calculated CI value (0.5) showed a synergistic effect for these drugs interaction. **Conclusion:** The results indicate that the combination of Sotorasib and Osimertinib produces a synergistic anti-cancer effect in A549 NSCLC cells harbouring wild-type EGFR and a KRAS G12C mutation.

Keywords: Sotorasib, Osimertinib, EGFR, KRAS, A549.

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PA023

Preliminary in Silico Study on Molecular Docking Analysis of D-Allulose with Glucose Transporters

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Introduction: Cancer cells exhibit the Warburg effect, characterised by elevated glucose uptake and glycolysis to sustain rapid proliferation. D-allulose, a rare sugar with anti-inflammatory, antioxidant and anti-glycation properties, has gained attention as a metabolic regulator in cancer metabolism. This preliminary in silico study investigated its interaction with key glucose transporters using molecular docking. **Methods:** Structures of glucose transporters (GLUT) 1, 2, 4 and 5 and sodium-glucose cotransporter 1 (SGLT1) were obtained from the Research Collaboratory for Structural Bioinformatics (RCSB) Protein Data Bank and AlphaFold, and D-allulose from PubChem. Receptors were prepared in UCSF Chimera 1.19 version, and docking was performed using HDOCKlite. **Results:** D-allulose showed the strongest predicted binding to SGLT1 (HDOCK score: −302.16), followed by GLUT1 (−139.83), GLUT2 (−136.46), GLUT4 (−134.49) and GLUT5 (−126.63). For comparison of molecular dynamics, SGLT1 was also docked with D-glucose (−152.48), D-allose (−152.58) and D-gulose (−153.34), all of which scored markedly less favourably than D-allulose, suggesting a potentially high affinity of D-allulose for SGLT1. **Conclusion:** D-allulose may interact with glucose transporters, particularly SGLT1. Further studies, including molecular dynamics simulations and in vitro glucose uptake assays in cancer cell lines, are warranted to validate these predictions and to evaluate its translational potential as a metabolic therapeutic in cancer.

Keywords
D-allulose, Cancer metabolism, Molecular docking, Glucose transporter, Rare sugars

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PA032

Combined Effects of Auranofin and Luteolin on Cell Viability and Radiosensitivity in HCT116 Colorectal Cancer Cells

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Introduction: Colorectal cancer (CRC) is the third most common cancer and the second leading cause of cancer-related death worldwide. Enhanced antioxidant defence in cancer cells may reduce the effectiveness of radiotherapy. Auranofin inhibits thioredoxin reductase (TrxR), but this inhibition may activate nuclear factor erythroid 2-related factor 2 (Nrf2) as a compensatory antioxidant response. Luteolin inhibits Nrf2 and may enhance the effects of auranofin. This study evaluated the cytotoxic interaction and radiosensitising effects of auranofin and luteolin in HCT116 CRC cells. **Methodology:** Cells were treated with different concentrations of auranofin and luteolin for 24 hours. Cell viability was assessed using the MTT assay, followed by determination of the IC_{20}, IC_{50} and combination index. For clonogenic analysis, cells were treated with 2 µM auranofin and 5 µM luteolin, individually or in combination, for 24 hours before irradiation with 2 Gy using a Caesium-137 source. Intracellular reactive oxygen species (ROS) levels were measured using the 2′,7′-dichlorodihydrofluorescein diacetate assay. **Results:** Auranofin and luteolin reduced HCT116 cell viability, while combination-index analysis demonstrated a synergistic interaction between the two agents. Auranofin alone and in combination with luteolin significantly reduced colony formation without irradiation. Irradiation alone significantly decreased clonogenic survival. Auranofin plus irradiation and the combination plus irradiation further reduced clonogenic survival compared with irradiation alone. However, luteolin did not significantly enhance radiosensitivity. The absence of a corresponding increase in intracellular ROS suggests that auranofin-induced radiosensitisation may occur through an ROS-independent pathway, although further validation is required. **Conclusion:** Auranofin demonstrated cytotoxic and radiosensitising effects in HCT116 cells. Its combination with luteolin produced synergistic cytotoxicity but did not further improve radiosensitisation compared with auranofin alone.

Keywords: radiosensitivity, combination treatment, auranofin, luteolin

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PA027

Effects of Zerumbone on Breast Cancer Cell Invasiveness under DMOG-induced HypoxiaFrom Computational Screening to Experimental Evaluation

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Introduction: The hypoxic tumour microenvironment, regulated by hypoxia-inducible factor 1-alpha (HIF-1α), plays a critical role in breast cancer progression. Zerumbone (ZER)has demonstrated anti-cancer properties, yet its activity under hypoxia remains poorly understood. This study explored the effects of ZER on the invasive behaviours of breast cancer cells under normoxia and dimethyloxalylglycine (DMOG)-induced hypoxia. **Methods:** IC20 values of ZER towards MCF-7 and MDA-MB-231 breast cancer cells under normoxia and DMOG-induced hypoxia were determined using MTT assay. Cell invasion following ZER treatment was assessed using Transwell invasion assay, in which the cells traversed Matrigel-coated cell culture inserts. **Results:** MCF-7 cells exhibited comparable sensitivity to ZER under normoxia (IC20: 48 μM) and DMOG-induced hypoxia (IC20: 50 μM), whereas MDA-MB-231 cells displayed enhanced sensitivity towards ZER under DMOG-induced hypoxia (IC20: 29 μM) compared to normoxia (IC20: 55 μM). In MCF-7 cells, ZER at 25 μM ($p<0.0001$) and 50 μM ($p<0.0001$) significantly reduced invasion under both normoxia and DMOG-induced hypoxia. In MDA-MB-231 cells, ZER at 12.5 μM ($p<0.05$), 25 μM ($p<0.05$), and 50 μM ($p<0.0001$) significantly suppressed invasion under normoxia, but significant inhibition was observed only at 25 μM ($p<0.05$) and 50 μM ($p<0.0001$) under DMOG-induced hypoxia. **Conclusion:** Overall, these findings suggest that ZER effectively attenuated the invasive capabilities of MCF-7and MDA-MB-231 cells under normoxia and DMOG-induced hypoxia. Future studies should incorporate hypoxic 3D models to better define the therapeutic potential of ZER in breast cancer management.

Keywords:
Breast cancer, DMOG-induced hypoxia, Zerumbone, Invasion

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PA029

Targeting the ADAR1 Zα Domain in Oral Squamous Cell Carcinoma: From Computational Screening to Experimental Evaluation

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Introduction: Our genome-wide CRISPR/Cas9 dependency screening in Asian-derived oral squamous cell carcinoma (OSCC) models identified ADAR1, particularly its interferon-inducible p150 isoform, as a recurrent cancer vulnerability. Beyond its RNA-editing activity, ADAR1-p150 contains a Zα domain that recognises Z-DNA and Z-RNA, offering an alternative intervention point for therapeutic development. **Methods:** We applied our proprietary computer-aided drug discovery pipeline, termed the Multi-layer Ultra-large Screening and Exploration (M.U.S.E), to approximately six billion compounds to identify potential ADAR1-p150 Zα-domain binders. Candidate compounds were initially prioritised through complementary biochemical and cellular screening assays, including intrinsic tryptophan fluorescence (ITF), electrophoretic mobility shift assay (EMSA) and MTT-based cytotoxicity assessment. Selected hit was subjected to chemical modification and evaluated using ITF, followed by cellular assessment of prioritised derivatives in OSCC and non-cancerous cell models. **Results:** Of 51 compounds evaluated by ITF, 19 demonstrated favourable binding profiles to recombinant Zα. Integration of biochemical and cellular screening results enabled prioritisation of selected hits, including MUSE-22, for further chemical refinement. Evaluation of MUSE-22 derivatives identified several compounds with improved ITF binding relative to the parent molecule. Among them, MUSE22-C8 was subsequently chosen for cellular evaluation, demonstrating an IC_{50} of 41.72 μM in the ADAR1-p150-dependent OSCC model (ORL-207), compared with no measurable IC_{50} in the tested non-cancerous OKF6/TERT1 and HEK293T models within the concentration range evaluated. **Conclusion:** Our study demonstrates the feasibility of identifying small-molecule compounds that bind the ADAR1-p150's Zα domain and provides preliminary evidence of cellular activity in an ADAR1-p150-dependent OSCC model. Further work is required to improve cellular potency, establish the contribution of ADAR1-p150-targeted activity and address pharmacological properties relevant to therapeutic development. These findings provide a basis for continued exploration of Zα-domain targeting as a potential strategy for ADAR1-p150-dependent OSCC.

Keywords: ADAR1, Zα domain, oral squamous cell carcinoma, virtual screening, drug discovery

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PA006

From Model to Translation: A Scoping Review of Preclinical Tongue Squamous Cell Carcinoma Models

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Introduction: Tongue squamous cell carcinoma (TSCC) is characterised by aggressive local invasion, early perineural involvement, and a high risk of cervical lymph node metastasis. Translation of preclinical findings into effective therapies remains limited, partly because no single model fully reproduces the biological and clinical complexity of TSCC. This scoping review aimed to characterise current preclinical TSCC models and evaluate their applications, strengths, limitations, and clinical relevance. **Methods:** A scoping review was conducted in accordance with PRISMA-ScR. PubMed, Embase, and Web of Science were searched for studies published from 1 January 2019 to 31 December 2024 using terms related to TSCC animal models, chemical carcinogenesis, orthotopic transplantation, genetically engineered mouse models, and patient-derived models. Original research and methodological studies describing preclinical TSCC models were included. Two independent reviewers screened records and charted data on model characteristics, applications, limitations, and translational relevance. **Results:** Of 1,063 records identified, 183 studies met the inclusion criteria. Four major model categories were identified: chemical induction models (n=25), mainly using 4-nitroquinoline 1-oxide (4NQO), which reproduce multistep carcinogenesis but are less suitable for metastatic studies; orthotopic transplantation models (n=147), which enable investigation of tumour growth, invasion, metastasis, and the native tongue microenvironment; genetically engineered mouse models (n=6), used to investigate molecular drivers and tumour initiation; and patient-derived xenograft/organoid models (n=5), which preserve clinically relevant tumour characteristics and support personalised therapeutic evaluation. Methodological heterogeneity, inconsistent outcome measures, and variable ethical reporting limited comparability. **Conclusion:** Current TSCC models provide complementary strengths, and model selection should be guided by the specific research question. Standardisation of clinically relevant endpoints, including depth of invasion and cervical lymph node metastasis, together with adherence to the 3R principles, may improve reproducibility and translational relevance.

Keywords: Tongue Squamous Cell Carcinoma (TSCC), Animal Models, Humanised Mice, Tumour Microenvironment, Immunotherapy

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Poster Presentation

Time: 1.35 pm – 2.00 pm

Venue: Open Area, Level 1

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PA019

Practices, Barriers, and Enablers to Enhanced Recovery After Surgery (ERAS) Implementation in Upper Gastrointestinal Surgery: A Malaysian Multicentre Qualitative Study

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Introduction: Surgery is a cornerstone of curative treatment for benign and malignant upper gastrointestinal (UGI) conditions, including resectable cancers, yet carries substantial risks of complications, malnutrition and delayed recovery. Enhanced recovery after surgery (ERAS) protocols, encompassing preoperative counselling, nutritional optimisation, and early feeding and mobilisation, have been shown to attenuate the surgical stress response and expedite recovery, linking curative treatment with supportive care, including for patients with cancer. However, ERAS adoption across Malaysia remains inconsistent, which may limit equitable access to evidence-based perioperative care. This study explored current ERAS practices, barriers and enablers perceived by multidisciplinary teams implementing ERAS for UGI surgery across Malaysian centres. **Methods:** A qualitative study was conducted using semi-structured, in-depth online interviews with surgeons, anaesthetists, dietitians and nurses, recruited through purposive and snowball sampling from six Malaysian UGI centres. Interviews were audio/video-recorded, transcribed verbatim and analysed thematically using a six-step framework in Atlas.ti software. **Results:** Twenty-four participants were interviewed. ERAS practices clustered into two themes: (1) partial and inconsistent implementation, and (2) the absence of a unified, written protocol across centres. Barriers clustered into four themes: (1) workforce instability, (2) professional hierarchy and team dynamics, (3) fragmented standardisation and training, and (4) resource and patient-related constraints. Enablers centred on three themes: (1) multidisciplinary teamwork, (2) workforce capability, and (3) institutional support through protocol standardisation and dedicated funding. **Conclusion:** ERAS implementation in Malaysian UGI surgery remains constrained by workforce, hierarchical and structural barriers, which may perpetuate variation in perioperative care between centres. Multidisciplinary collaboration, targeted training and institutional funding can strengthen implementation. These findings support a standardised, contextually appropriate ERAS implementation protocol for Malaysian UGI centres and offer transferable insights for advancing equitable perioperative and supportive care, including cancer care, across the region.

Keywords
Enhanced recovery after surgery, Upper gastrointestinal surgery, Perioperative care, Nutrition support, Qualitative

Beyond Treatment Availability: Inequities in Access to Targeted Therapy for Metastatic Colorectal Cancer

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Background: Access to targeted therapy for metastatic colorectal cancer (mCRC) depends on treatment availability and also on patients' ability to navigate molecular testing, financing and clinical care pathways. This study examined factors associated with time to targeted therapy initiation among patients with mCRC in the Malaysian public healthcare system. **Methods:** This multicentre retrospective cohort study included patients diagnosed with mCRC and meeting Ministry of Health indication for targeted therapy treatment at three Malaysian public hospitals between 2017 and 2021, with follow-up through 2025. Time from metastatic disease diagnosis to targeted therapy initiation was analysed using Cox proportional-hazards regression. A Fine-Gray competing-risk model with a competing-risk cumulative incidence function (CIF) accounted for death before treatment initiation. **Results:** Among 889 eligible patients, only 247 (27.8%) initiated targeted therapy and the median from diagnosis to targeted therapy initiation was 38.6 weeks, (Inter Quartile Range 11.5–90.0). KRAS testing was the strongest factor associated with treatment initiation (adjusted hazard ratio [aHR] 6.12, 95% CI 4.47–8.39, even when adjusted for death before treatment initiation [sHR] 9.67, 95% CI 6.79–13.79; p<0.001). Patients from civil-service households (aHR 1.64, 95% CI 1.17–2.31) and diagnosed at private facilities (aHR 1.86, p<0.001) initiated treatment more rapidly. Overall, 566 (63.7%) died before treatment initiation and the CIF at 1 year showed 33% of patients had died before initiating therapy versus 17% who had initiated it, and the CIF at 2 years showed that 50% had died versus 25% who had initiated. **Conclusions:** Timely access to targeted therapy in public healthcare system was associated with access to molecular testing and financing-related status. Patients who died before treatment initiation highlights gap between treatment availability and access to cancer therapy. Expanding molecular testing and reducing financial barriers may represent health-system strategies to improve equity in access to precision oncology.

Keywords:
Targeted therapy , Metastatic , Colorectal Cancer , time-to-treatment , Molecular Testing

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PA022

Development and Preliminary User Testing of ColRectCare©: A Bilingual Web Application for Colorectal Cancer Awareness in Malaysia

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Introduction: Colorectal cancer is a major public health concern in Malaysia, where late-stage diagnosis contributes to poor survival. ColRectCare© was developed as a free, bilingual (English and Bahasa Malaysia) web application to promote awareness of colorectal cancer risk factors, symptoms, and screening. **Methods:** The browser-based application uses a five-step questionnaire to collect information on personal characteristics, family history, lifestyle, symptoms, previous investigations, and screening history. A provisional weighted scoring algorithm, informed by colorectal cancer risk factors and symptoms, categorises responses as Low, Worth Checking, or Higher Concern and provides corresponding advice. Seven respondents completed the questionnaire in a preliminary pilot test. Descriptive statistics were used to summarise score categories and screening-related responses. The scoring algorithm has not been clinically validated. **Results:** All seven respondents completed the five-step questionnaire. Six (85.7%) were classified as Low and one (14.3%) as Worth Checking; none was classified as Higher Concern. Six respondents (85.7%) reported never having undergone colorectal cancer screening, while five (71.4%) identified the absence of symptoms as a barrier to screening. These findings describe the pilot participants and the application's provisional scoring outputs; they do not establish the accuracy of the scoring algorithm or the representativeness of the sample. Usability and acceptability outcomes were not available for reporting. **Conclusion:** ColRectCare© provides an accessible digital platform for colorectal cancer awareness. The findings highlight the need to address symptom-based misconceptions about screening. Further evaluation, which aims to assess usability and acceptability in a larger, more diverse sample, will be designed to include clinical expert review and validation of the scoring.

Keywords:
Colorectal cancer, Self-awareness, Web app, Risk assessment, Early detection

The Effectiveness And Safety Of 5-Aminolevulinic Acid Photodynamic Therapy In Patients With Persistent HPV Infection With Or Without Cervical Intraepithelial Neoplasia: A Retrospective Cohort Study

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Introduction: Persistent human papillomavirus (HPV) infection is a major cause of cervical precancer and cancer. 5-Aminolevulinic acid photodynamic therapy (5-ALA-PDT) is a non-invasive treatment option for cervical lesions. This study evaluated its effectiveness and safety and explored factors associated with HPV clearance. **Methods:** Clinical records of 158 patients with persistent cervical HPV infection, with or without cervical intraepithelial neoplasia (CIN), who received 5-ALA-PDT were retrospectively reviewed. HPV genotyping, cytology, colposcopy and colposcopy-guided biopsy were performed. The primary outcome was HPV clearance at 3–6 months after treatment. Factors associated with clearance were analysed using multivariate logistic regression. **Results:** Among 153 patients with follow-up data, 92 (60.13%) achieved HPV clearance. Clearance was higher in patients aged ≤35 years than in those aged >35 years (67.44% vs 50.75%, P=0.036), in single-type than multiple-type HPV infection (72.04% vs 41.67%, P<0.001), and in low-grade squamous intraepithelial lesion (LSIL) than high-grade squamous intraepithelial lesion (HSIL) (64.71% vs 52.63%, P=0.004). Multivariate analysis identified age >35 years (OR 2.785, 95% CI 1.320–5.878), multiple-type HPV infection (OR 4.027, 95% CI 1.951–8.313), and HSIL (OR 2.716, 95% CI 1.279–5.760) as independent factors associated with reduced HPV clearance. Lesion regression occurred in 81.16% of LSIL and 84.31% of HSIL cases. **Conclusion:** 5-ALA-PDT showed promising preliminary effectiveness for persistent HPV infection. Age, HPV infection pattern and lesion grade were associated with HPV clearance. Larger studies with longer follow-up are warranted to confirm these findings.

Keywords
5-Aminolevulinic acid, Photodynamic therapy, Human papillomavirus, Cervical intraepithelial neoplasia, HPV clearance

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PA030

A Step Towards Healthy Ageing: Does Fat Mass Modulate the Association Between Physical Fitness and Telomere Length in Breast Cancer Survivors?

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Introduction: While physical fitness fosters healthy ageing, evidence linking it to telomere length in Malaysian breast cancer survivors remains underexplored, prompting this evaluation of the association within this population. **Method:** A cross-sectional study of 262 breast cancer survivors was conducted via purposive sampling at Hospital Canselor Tuanku Muhriz, Universiti Kebangsaan Malaysia, as part of the Cancer Survivors' Trajectories of Ageing Research (C*STAR) project. Physical fitness was evaluated using the two-minute step test (number of steps), handgrip strength test (kg), 30-second stand test (number of stands), chair sit-and-reach test (cm), back-scratch test (cm), and Timed Up-and-Go test (seconds). Absolute telomere length (kb/genome) was measured via quantitative real-time PCR (qPCR), and the fat mass (%) was measured using the Tanita SC-330 (TANITA Corporation, Tokyo, Japan) platform. The analyses were performed using SPSS version 29. **Results:** The 30-second sit-to-stand test was positively associated with telomere length (B = 12.019 kb/genome, 95% CI: 1.094 – 22.944, p = 0.031) after adjustment for working status, family history of cancer and heart disease, age at cancer diagnosis, cancer stage, survival duration (years), cancer treatments (surgery, chemotherapy, and radiotherapy), chemotherapy drugs, and receptor status. This indicates that each additional repetition completed in the 30-second sit-to-stand test was associated with a 12.019 kb/genome increase in telomere length. The association remained significant following additional adjustment for fat mass (B = 11.909 kb/genome, 95% CI: 0.936 – 22.882, p = 0.034). Other physical fitness measures of the two-minute step test, handgrip strength test, chair sit-and-reach test, back-scratch test, and Timed Up-and-Go test were not significantly associated with telomere length (p > 0.05). The minimal change in the regression coefficient following fat mass adjustment suggests that the association between lower-body physical function and telomere length was largely independent of fat mass. **Conclusion:** Lower-body functional fitness was positively associated with telomere length, independent of fat mass adjustment, suggesting its potential relevance to biological ageing among breast cancer survivors.

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PA002

High-Dose Methotrexate Pharmacokinetics in Pediatric Acute Lymphoblastic Leukemia in Indonesia: Correlation with Renal Function and SLCO1B1 Polymorphism

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Introduction: Safe use of high-dose methotrexate (HD-MTX) requires close drug monitoring (TDM), but this is often unavailable in resource-limited settings like Indonesia. As a result, methotrexate pharmacokinetic (PK) profiles in these populations remain poorly characterized, as does the extent to which variation in drug levels arises from clinical or genetic factors. This study describes the correlation between MTX blood levels, kidney function, toxicity, and SLCO1B1 gene polymorphism in Indonesian children with acute lymphoblastic leukemia (ALL) receiving HD-MTX. **Methods:** Seventy-nine pediatric ALL patients were included. MTX levels were measured at 24 and 42 hours using LC-MS/MS, and clearance was estimated with a one-compartment model. Two SLCO1B1 variants (rs2306283, rs4149056) were genotyped, and toxicity was graded using CTCAE v5.0. Regression analysis tested whether clinical factors, genotype, and kidney function explained variation in drug levels and clearance. **Results:** Doses were already reduced in practice, averaging 76% of the standard BSA-based dose. Even so, 22.8% of patients showed delayed drug clearance (C42 ≥1 µmol/L), meaning dose reduction alone was not enough to prevent risk. Kidney function was linked to C42 levels (SCr: $r = 0.358$, $p = 0.001$; eGFR: $r = -0.252$, $p = 0.025$). SLCO1B1 variants affected early drug levels (C24) and clearance, but not later elimination (C42), suggesting that genetics drives early exposure while kidney function governs later elimination. However, a model combining dose, BSA, creatinine, and each genotype explained only 17.4% of C24 variation ($R^2 = 0.174$), while a model combining log(C24) with SCr explained 35% of C42 variation ($R^2 = 0.350$). **Conclusion:** Delayed MTX clearance still occurs even with reduced doses, and neither clinical nor genetic factors reliably predict individual drug exposure. This underscores an urgent need for accessible TDM, ideally with monitoring at multiple time points, to make HD-MTX therapy safer and more personalized in resource-limited like Indonesia.

Keywords:
Methotrexate, Acute Lymphoblastic Leukemia, Therapeutic Drug Monitoring, Toxicity, Personalized Medicine

Population Pharmacokinetic Model-Based Risk Stratification Strategy for High-Dose Methotrexate in Malaysian Adults with Haematological Malignancies

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Introduction: High-dose methotrexate (HDMTX) demonstrates substantial interindividual pharmacokinetic variability, which may influence the risk of delayed elimination and toxicity. Population pharmacokinetic (PopPK) modelling can characterize sources of this variability and support simulation of clinically relevant exposure outcomes. This study aimed to develop a PopPK model of HDMTX using routine clinical data from Malaysian adults with haematological malignancies and to evaluate the influence of patient characteristics on delayed MTX elimination through model-based simulations. **Methods:** Plasma methotrexate (MTX) concentrations at 48, 72, and 96 h were retrospectively collected from adult patient receiving >500 mg/m^2 MTX between August 2024 to Jun 2025. PopPK model was developed using Monolix, including demographic characteristics, renal and liver function parameters, haematocrit, treatment protocol, and infusion duration as covariates. The final model was implemented in Simulx to simulate MTX exposure across albumin concentrations of 14–39 g/L and HDMTX doses of 750–7000 mg, at different infusion duration. **Results:** A total of 91 HDMTX administrations, comprising 206 concentration measurements were included. A two-compartment model with first-order elimination and combined residual error adequately described the observed data, with albumin as a significant predictor of MTX clearance. The final clearance model was described as CL (L/h) = 11.13 × (Albumin/39)$^{0.7}$. Model-based simulations demonstrated a greater probability of MTX concentrations remaining ≥0.2 µmol/L at 48 h and a longer time to reach 1.0 µmol/L at lower albumin concentrations, with the effect becoming more pronounced at higher HDMTX doses administered over longer duration of infusion. **Conclusion:** A PopPK model describing HDMTX disposition in Malaysian adults with haematological malignancies was successfully developed using routinely collected clinical data. Albumin was identified as an important determinant of MTX clearance. Model-based simulations further demonstrated how albumin plays role in early risk stratification and individualized monitoring of patients receiving HDMTX.

Keywords: popPK, pharmacokinetic, methotrexate, MTX, HDMTX

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Mechanism-Guided Strategies for Overcoming Acquired Resistance to Osimertinib in EGFR-Mutated Non-Small-Cell Lung Cancer: A Review

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Introduction: Osimertinib is a preferred first-line treatment for advanced EGFR-mutated non-small-cell lung cancer (NSCLC) and demonstrates significant improvements in progression-free survival (PFS) and overall survival (OS) over previous-generation EGFR tyrosine kinase inhibitors (TKIs). However, the clinical utility of this agent is inevitably limited by the development of acquired drug resistance in nearly all patients. Consequently, rather than relying on a uniform next-line standard of care, the therapeutic landscape must transition toward a paradigm stratified by specific acquired molecular mechanisms of resistance. Objective: This review aimed to organize the post-osimertinib resistance landscape by underlying molecular mechanism rather than chronological drug generation; map each mechanism to the evidence supporting newly developed countermeasures; and clarify which strategies are approved or in late-phase clinical or pre-clinical development. **Methods:** This review is a narrative, mechanism-organized synthesis of the literature (not a PRISMA-adherent systematic review or meta-analysis) identified through PubMed/MEDLINE, Embase, and ClinicalTrials.gov, as well as ASCO, ESMO, WCLC, and ELCC conference proceedings, from January 2016 to July 2026. Single-arm early-phase and case-level evidence were retained only when no higher-quality evidence existed for that particular resistance subtype, and evidence was identified by developmental stage throughout Phase 2/3 trials; peer-reviewed translational studies were prioritized. **Results:** Acquired osimertinib mechanisms were grouped into four categories: 1. EGFR-dependent (on-target, mainly C797S and cis/trans configuration of C797S relative to T790M); 2. Bypass-pathway mediated (off-target, mainly MET amplification, with HER2, BRAF, RET and PIK3CA alterations reported less frequently); 3. Histological or phenotypic transformation (principally small-cell lung cancer (SCLC) transformation); 4. Unidentified mechanisms (tumour progression occurring in the absence of identifiable genomic drivers despite exhaustive genomic sequencing). For each category, current investigational and approved therapeutic options were evaluated, including EGFR-dependent inhibitors, MET-directed target therapies, platinum-etoposide-based regimens for histological transformation, and antibody-drug conjugates (ADCs) as a mechanism-agnostic salvage option. **Conclusion:** These results suggest that efficacy in the post-osimertinib setting is fundamentally dependent on the underlying resistance mechanisms, validating a paradigm shift away from empirical next-line treatments toward mechanism-guided, personalized oncology. Future research is needed to solidify preclinical and clinical evidence supporting transformation-directed and mechanism-agnostic strategies and to resolve currently undefined resistance mechanisms to optimize patient stratification

Keywords: EGFR mutation; osimertinib; acquired resistance; C797S; MET amplification; small-cell transformation; antibody-drug conjugate; non-small-cell lung cancer

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Integrating Clinical And Transcriptomic Data For Relapse Prediction In Childhood B-Cell Acute Lymphoblastic Leukaemia

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Introduction: Despite advances in childhood B-cell acute lymphoblastic leukaemia (B-ALL) treatment, relapse remains a major cause of treatment failure and mortality. Conventional clinical risk stratification has limited predictive accuracy, highlighting the need for more precise approaches. This study aims to establish a machine learning framework integrating clinical and transcriptomic data to improve relapse prediction and provide biological insights into relapse susceptibility. **Methods:** A well-annotated, multicentre Malaysia–Singapore B-ALL cohort comprising 942 patients, including 474 with matched diagnostic RNA sequencing (RNA-seq) data. Univariate regression analyses identified clinically relevant relapse predictors. Feature reduction was incorporated into the transcriptomic analytical strategy to address RNA-seq high dimensionality and minimise overfitting. Differential gene expression and pathway-based approaches will be used to prioritise biologically relevant transcriptomic features. A literature review of diagnostic transcriptomic studies on relapse prediction was also conducted to synthesise reported relapse-associated signatures and inform biological feature selection. The modelling workflow integrates clinical and transcriptomic features using time-to-event modelling, with 10-fold cross-validation and class-balancing strategies to address limited relapse events. **Results:** Preliminary regression-based analyses supported the relevance of age at diagnosis, BCR-ABL1 positivity and day 33 measurable residual disease (MRD) status. The literature review synthesised reported relapse-associated transcriptomic signatures into five biological domains: 1) cell death and stress-adaptive survival, 2) immune modulation and metabolic adaptation, 3) dysregulated developmental and stemness, 4) drug resistance, and 5) post-transcriptional regulation. These domains provide a framework for prioritising pathway-level features. A structured modelling workflow incorporating time-to-event analysis, cross-validation and class imbalance management has been established, with model training ongoing. **Conclusion:** This study establishes a clinically grounded and biologically informed framework for machine learning-based relapse prediction in childhood B-ALL, leveraging a well-annotated Malaysia-Singapore multicentre cohort. Integrating clinical predictors with pathway-level transcriptomic features may improve relapse risk stratification and support more personalised treatment strategies.

Keywords: B-cell acute lymphoblastic leukaemia, relapse, machine learning, transcriptomics, risk stratification



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