IO HK 2026
IMMUNO-ONCOLOGY

香港中文大學
The Chinese University of Hong Kong

CU Medicine HONG KONG
香港中文大學醫學院
Faculty of Medicine
The Chinese University of Hong Kong

IMMUNO - ONCOLOGY HONG KONG 2026

7 - 8 November 2026



Organised by:

Department of Clinical Oncology &
Hong Kong Cancer Institute

In conjunction with State Key Laboratory
of Translational Oncology

The Chinese University of Hong Kong

AstraZeneca 阿斯利康 Johnson&Johnson MSD Pfizer

Address The **UNSEEN RISK** Before Surgery



Occult **micrometastatic disease** may **drive relapse** — even after **curative-intent surgery** in GC/GEJC[1-6]

Discuss **high-risk resectable** cases in **MDT** before surgery[7,8]

Abbreviations: GC = gastric cancer; **GEJC** = gastroesophageal junction cancer; **MDT** = multidisciplinary team.

References: 1. Deng Q, et al. *Clin Exp Med.* 2023;23(8):4385-4399. **2.** Heeren PA, et al. *Eur J Surg Oncol.* 2005;31(3):270-276. **3.** Lee MW, et al. *Gut Liver.* 2019;13(4):394-401. **4.** Liu D, et al. *World J Surg Oncol.* 2016;14(1):305. **5.** Spolverato G, et al. *J Am Coll Surg.* 2014;219(4):664-675. **6.** Okemoto D, et al. *Gastroenterol Rep (Oxf).* 2026;14:goag010. **7.** Basta YL, et al. *Ann Surg Oncol.* 2017;24(9):2669-2678. **8.** Joshi SS, et al. *CA Cancer J Clin.* 2021;71(3):264-279.

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Abbreviation: 1L: First line; Ami+Laz: Amivantamab and Lazertinib; cEGFR: common EGFR mutation, MET: Mesenchymal-epidermal transition factor gene; NCCN: National Comprehensive Cancer Network

Reference: 1. NCCN Clinical Practice Guidelines in Oncology for Non-Small Cell Lung Cancer V.5.2026. **2.** Hayashi H, et al. Long-Term Impact of First-Line Amivantamab Plus Lazertinib Versus Osimertinib on Mechanisms of Acquired Resistance in MARIPOSA: A Brief Report, Journal of Thoracic Oncology. 10.894, April 28,2026

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Welcome

On behalf of the organizing and scientific committees, it is a great pleasure to welcome you to the 31st Annual Scientific Symposium of the Hong Kong Cancer Institute: Immuno-Oncology Hong Kong 2026. This biennial symposium is co-organized by the Department of Clinical Oncology and the Hong Kong Cancer Institute in conjunction with State Key Laboratory of Translational Oncology at The Chinese University of Hong Kong.

Continuing the success of our last IOHK 2024, we are delighted to announce that IOHK 2026 takes place on 7 - 8 November 2026 and accommodates delegates to join the conference physically and virtually. This year we have the wealth of information for you with the recent developments of immuno-therapy across multi-tumour sites, including lung cancer, liver cancer, GI cancer and GU Malignancy, Small Cell Lung Cancer, Head and Neck Cancer, Hepatocellular and Pancreatic Cancer, Lung Cancer (NSCLC) and Phase 1 Development. So, we have speakers all over the world, Asia, Europe and USA, to provide you with the best information.

We expect to attract over 400 attendees joining the physical event from across the world and over 500 attendees joining virtually. Your participation is pivotal to our cause. We hope this meeting will bring you new insights into the development of immunotherapies for cancer patients.

So, welcome you and please come to enjoy the cuisine in Hong Kong and our hospitality.



Professor Anthony CHAN
Director, Hong Kong Cancer Institute
The Chinese University of Hong Kong



Professor Tony MOK
Chairman, Department of Clinical Oncology
The Chinese University of Hong Kong

Symposium Advisory, Organising & Scientific Committees

Symposium Advisory Committee

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The Chinese University of Hong Kong

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Hong Kong Sanatorium Hospital, Hong Kong

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The Chinese University of Hong Kong

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Members, Symposium Scientific Committee

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The Chinese University of Hong Kong

Stephen CHAN
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Molly LI
The Chinese University of Hong Kong

Herbert LOONG
The Chinese University of Hong Kong

Darren POON
The Chinese University of Hong Kong

Kelvin YAN
The Chinese University of Hong Kong

Joyce SUEN
Prince of Wales Hospital, Hong Kong

Edwin HUI
Prince of Wales Hospital, Hong Kong

Opening Ceremony

Opening Ceremony of
The 31st Annual Scientific Symposium

Immuno-Oncology Hong Kong 2026

7th November 2026 (Saturday) 9:00 a.m.
Grand Ballroom, Kerry Hotel, Hong Kong

*

Opening Speech by
Professor Dennis LO, GBS, SBS, JP
Vice-Chancellor and President
The Chinese University of Hong Kong

*

Vote of Thanks by
Professor Tony MOK, BBS
Chairman, Department of Clinical Oncology
The Chinese University of Hong Kong

*

Group Photos

Scientific Programme

Day 1 – Saturday, 7 November 2026

Time	Session	Chairman/ Speaker
08:20 - 08:50	Registration	
09:00 - 09:30	Opening Ceremony	Prof. Dennis LO *The Chinese University of Hong Kong, HKSAR, China*
		Chairman/ Speaker
09:30 - 10:15	Keynote Lecture Supported by Centre for Novostics	Prof. Allen CHAN *The Chinese University of Hong Kong, HKSAR, China* Prof. Tony MOK *The Chinese University of Hong Kong, HKSAR, China*
09:30 - 10:05	Economy of Global Drug Development	Prof. Pietro VERONESI *The University of Chicago, USA*
10:05 - 10:15	Discussion	All
10:15 - 11:25	GU Malignancy	Prof. Peter CHIU *The Chinese University of Hong Kong, HKSAR, China* Dr. Darren POON *The Chinese University of Hong Kong, HKSAR, China*
10:15 - 10:35	Overcoming the Immune-cold Microenvironment: Emerging Novel Strategies in Metastatic Prostate Cancer	Prof. Aaron HANSEN *The University of Queensland, Australia*
10:35 - 10:55	The Evolving Role of Antibody Drug Conjugates and Immunotherapy in Localized and Metastatic Urothelial Carcinoma	Prof. Terence FRIEDLANDER *University of California San Francisco, USA*
10:55 - 11:15	The Optimal Sequencing and Practical Consideration of Combination Therapies in Resected and Metastatic Renal Cell Carcinoma	Prof. Viktor GRÜNWALD *University of Essen, Germany*
11:15 - 11:25	Discussion	All
11:25 - 11:55	Coffee Break and Poster Session	
11:55 - 13:20	Small Cell Lung Cancer	Prof. Rina HUI *The University of Hong Kong, HKSAR, China* Dr. Molly LI *The Chinese University of Hong Kong, HKSAR, China*
11:55 - 12:20	Development of Treatment Targeting DLL3	Dr. Giannis MOUNTZIOS *Henry Dunant Hospital Center, Greece*

Time	Session	Chairman/ Speaker
12:20 - 12:45	State-of-Art Therapy for ES-SCLC	Prof. Yilong WU *Guangdong Provincial People's Hospital, China*
12:45 - 13:10	Biomarker Subtyping of SCLC: Is It Clinically Relevant?	Dr. Yang XIA *Second Affiliated Hospital of Zhejiang University, China*
13:10 - 13:20	Discussion	All
13:20 - 14:20	Lunch Symposia *(Boxed lunches will be provided at the symposium venues)* *Diamond – Pfizer (Ballroom 1-3)* *Platinum – Daiichi Sankyo (Hung Hom 1)*	
14:20 - 15:45	Head and Neck Cancer	Prof. Anthony CHAN *The Chinese University of Hong Kong, HKSAR, China* Dr. David JOHNSON *Prince of Wales Hospital, HKSAR, China*
14:20 - 14:45	Advances in Immunotherapy for the Treatment of Locally Advanced NPC	Prof. Haiqiang MAI *Sun Yat-sen University Cancer Center, China*
14:45 - 15:10	Changing Landscape in Perioperative & Neoadjuvant Immunotherapy for Locally Advanced Head and Neck Squamous Cell Cancer	Prof. Robert HADDAD *Dana-Farber Cancer Institute, USA*
15:10 - 15:35	Novel Biomarkers for Individualizing Immunotherapy in Patients with Recurrent/Metastatic Head and Neck Cancer	Prof. Bhumsuk KEAM *Seoul National University Hospital, Republic of Korea*
15:35 - 15:45	Discussion	All
15:45 - 16:45	Satellite Symposium *Diamond – Johnson & Johnson (Ballroom 1-3)*	
16:45 - 17:45	Pre-Dinner Cocktail Reception at Ballroom 4	
18:00 - 22:00	Conference Dinner (Ballroom 1–3)	

Scientific Programme

Day 2 – Sunday, 8 November 2026

Time	Session	Chairman/ Speaker
09:00 - 10:25	Hepatocellular and Pancreatic Cancers	Prof. Stephen CHAN *The Chinese University of Hong Kong, HKSAR, China* Dr. Janet KUNG *The Chinese University of Hong Kong, HKSAR, China*
09:00 - 09:25	Neoadjuvant Therapy for HCC	Prof. David PINATO *Imperial College London, United Kingdom*
09:25 - 09:50	Novel Therapy for Pancreatic Cancer	Dr. Teresa MACARULLA *Hospital Clinic Barcelona, Spain*
09:50 - 10:15	The Role of Local Treatment for Oligoprogression in HCC	Dr. Landon CHAN *The Chinese University of Hong Kong, HKSAR, China*
10:15 - 10:25	Discussion	All
10:25 - 10:45	Coffee Break	
10:45 - 12:15	Lung Cancer (NSCLC)	Prof. Tony MOK *The Chinese University of Hong Kong, HKSAR, China* Dr. Oscar CHAN *Hong Kong Integrated Oncology Centre, HKSAR, China*
10:45 - 11:05	Peri-Operative Therapy for Early-Stage NSCLC and the Role of MRD	Prof. Solange PETERS *Lausanne University Hospital, Switzerland*
11:05 - 11:25	Management of Resistance to Immunotherapy	Prof. Marina Chiara GARASSINO *The University of Chicago, USA*
11:25 - 11:45	Novel Therapy on the Horizon for AGA Driven NSCLC	Prof. Pasi JÄNNE *Dana-Farber Cancer Institute, USA*
11:45 - 12:05	Novel Therapy on the Horizon for Non-AGA Driven NSCLC	Prof. Daniel TAN *National Cancer Centre Singapore, Singapore*
12:05 - 12:15	Discussion	All
12:15 - 13:15	Lunch Symposia *(Boxed lunches will be provided at the symposium venues)* *Diamond – AstraZeneca (Ballroom 1 – 3), MSD (Ballroom 4),* *Platinum - BMS (Hung Hom 1)*	
13:15 - 14:40	Phase I Development	Dr. Herbert LOONG *The Chinese University of Hong Kong, HKSAR, China* Dr. Aya El HELALI *The University of Hong Kong, HKSAR, China*
13:15 - 13:40	Interfacing with the Future: Artificial Intelligence in Drug Development for Immuno-Oncology	Dr. Arsela PRELAJ *Fondazione IRCCS Istituto Nazionale dei Tumori of Milan, Italy*
13:40 - 14:05	Adoptive Cell Therapy in Solid Tumours: From Early-Phase Trials to Real-World Implementation	Dr. Jonathan LIM *The University of Manchester, United Kingdom*
14:05 - 14:30	Incorporating Patient Reported Outcome Measures (PROMs) in Early Phase Oncology Drug Development	Dr. Herbert LOONG *The Chinese University of Hong Kong, HKSAR, China*
14:30 - 14:40	Discussion	All
14:40 - 16:25	CTONG - Young Session on IIT*	Dr. Molly LI *The Chinese University of Hong Kong, HKSAR, China* Dr. Kezhong CHEN *Peking University People's Hospital, China* Dr. Siyang LIU *Guangdong Provincial People's Hospital, China* Dr. Fang WU *The Second Xiangya Hospital of Central South University, China* Dr. Jiatao ZHANG *Guangdong Provincial People's Hospital, China*
14:40 - 14:50	How to Design an Impactful Investigator Initiated Trial (IIT)	Dr. Yongchang ZHANG *Hunan Cancer Hospital, China*
14:50 - 15:05	From Participating Site to Trial Initiator: Building IIT Capacity in Inner Mongolia, China	Dr. Rila SU *Inner Mongolia People's Hospital, China*
15:05 - 15:25	IIT #1 and Discussion *Panellists from CTONG YOUNG*	Dr. Heming LI (Presenter) *The First Hospital of China Medical University, China*
15:25 - 15:45	IIT #2 and Discussion *Panellists from CTONG YOUNG*	Dr. Xue YANG (Presenter) *Peking University Cancer Hospital & Institute, China*
15:45 - 16:05	IIT #3 and Discussion *Panellists from CTONG YOUNG*	Dr. Yichen ZHANG (Presenter) *Guangdong Provincial People's Hospital, China*
16:05 - 16:25	IIT #4 and Discussion *Panellists from CTONG YOUNG*	Dr. Jiefei HAN (Presenter) *Beijing Chest Hospital, Capital Medical University , China*

*refer to P.69 About CTONG and CTONG YOUNG

Invited Faculty



Dr. Landon Chan
The Chinese University of Hong Kong
HKSAR, China

Dr. Landon Chan is a clinical oncologist by training. He joined the Department of Clinical Oncology at the Chinese University of Hong Kong as an Assistant Professor (clinical) in 2023. Prior to his career in medicine, he was trained as a Mathematician and received his Bachelor of Arts from Carleton College in Minnesota, United States. After working at the Mayo Clinic for one year as a statistical analysis programmer, he returned to Hong Kong to pursue his career in clinical medicine. He obtained his medical degree from the Chinese University of Hong Kong in 2017 and became a fellow of the Royal College of Radiologists in 2022. He also received a MSc degree in Precision Cancer Medicine from Oxford University in 2022.

Dr. Landon Chan has a long history of interest in personalized medicine. Fully convinced of the potentials of using sequencing technology to understand tumour biology and hence tailor-made personalized treatment, he has participated in multiple bioinformatics projects in renowned international research groups, including Prof. Dennis Lo's and Prof. Kevin Yip's group at the Chinese University of Hong Kong, Prof. Mark Gerstein's group at the Yale University, and Prof. Francesca Buffa and Dr. Simon Lord's group at the Oxford University, with hands-on experience on DNA-seq and RNA-seq data.

His current research interest includes clinical and translational studies on hepatopancreatico-biliary, and how to combine radiotherapy and immunotherapy as part of a personalized therapeutic treatment strategy.



Prof. Terence Friedlander
University of California San Francisco
USA

I am a medical oncologist and clinical investigator specializing in urologic cancers, specifically bladder cancer, with a focus on designing and conducting clinical trials and exploring biomarkers of treatment response and resistance. I have helped develop multiple novel therapies in urothelial cancer, including multiple PD-1 immune checkpoint inhibitors, FGFR3-targeted small molecules, and antibody–drug conjugates targeting Nectin-4, HER2, and TROP-2. I co-lead the UCSF Bladder Cancer Program. Our program aims to identify biomarkers of response and resistance to therapy and explore novel therapeutic targets. Since 2017, I have also served as Chief of the Division of Hematology and Oncology at Zuckerberg San Francisco General Hospital and Trauma Center, where I have built a clinical research program focused on expanding access to cancer clinical trials.



Prof. Marina Chiara Garassino
The University of Chicago
USA

Marina Chiara Garassino, MD, is a Professor of Medicine and Director of the Thoracic Program in the Section of Hematology/Oncology at the University of Chicago. An internationally recognized leader in thoracic oncology, she has authored more than 350 peer-reviewed publications and has helped redefine standards of care in lung cancer through pivotal clinical trials, including serving as Principal Investigator for KEYNOTE-189, as a steering committee member for KEYNOTE-671, and as an active principal investigator for PACIFIC, as well as leading investigator-initiated studies such as TAILOR and PEOPLE. She has also conducted landmark translational research on LKB1 and hyperprogressive disease, advancing biologic understanding of resistance and immune dynamics in lung cancer. Dr. Garassino is at the forefront of artificial intelligence–driven oncology, In parallel, she is expanding her AI agenda into AI-enabled drug discovery and repurposing, building an integrated bench-to-bedside strategy that links tumor profiling, advanced analytics, and clinical trial execution to accelerate the identification and testing of new therapeutic hypotheses in lung cancer. During the COVID-19 emergency, she founded TERAVOLT, a global thoracic cancer registry with broad international participation, with results published in leading journals. She is also the founder and president of Women for Oncology Italy and an active advocate for equity and leadership development in oncology. She has actively participated in multiple ESMO committees and Taskforces (Scientific Committees, Public Policy extended Committee, Press Committee, and Women for Oncology). She has been the ESMO Chair of National Societies.



Prof. Viktor Grünwald
University of Essen
Germany

Viktor Grünwald is a dedicated expert in the field of GU cancers. He holds a position as Director of the Carolus Institute for Urologic Oncology at the University Hospital of the University of Duisburg-Essen. His primary focus are conservative treatments in patients with urogenital cancers at the Essen University Hospital.

Professor Grünwald completed his medical studies at the Hannover Medical School in 1998, after which he was appointed as a Private Lecturer and subsequently Professor of Haematology and Oncology at the same institution. Since 2018, he has held the Carolus-endowed full Professorship in Interdisciplinary GU Oncology and received in 2024 a call as Professor for Urologic Oncology at the University Hospital Essen. He chairs the patient-centric Carolus Institute for Urologic Oncology that harmonizes efforts across multiple disciplines including medical oncology, urology, and nuclear medicine. This interdisciplinary collaboration teams the expertise from various fields to optimize patient outcomes.

Professor Grünwald has served as a scientific member for a number of guideline committees, symposia and congresses and has chaired different working groups in academic organizations. He lately became the president elect of the German Studies Group in Medical Oncology (AIO). He furthermore actively engaged with international committees within ESMO and EORTC and held faculty positions in various forums.



Prof. Robert Haddad
Dana-Farber Cancer Institute
USA

Robert I. Haddad, MD, is the McGraw Chair in Head and Neck Oncology and Professor of Medicine at Harvard Medical School. He serves as Division Chief and Institute Physician in the Center for Head and Neck Oncology at Dana-Farber Cancer Institute, where he is also a member of the Department of Adult Oncology. Dr Haddad earned his medical degree from Saint Joseph University, French Faculty of Medicine. He completed his internship and residency at St. Luke's–Roosevelt Hospital Center in New York, followed by a fellowship in hematology/oncology at the Center. He is an active member of leading professional societies, including the American Society of Clinical Oncology, American Association for Cancer Research, and American Society for Radiation Oncology. His research focuses on advancing innovative therapeutic strategies in head and neck cancer, with particular emphasis on induction chemotherapy and the development of novel immunotherapeutic approaches for locally advanced disease. Dr. Haddad has played a central role in the design, conduct, and publication of numerous pivotal phase II and III clinical trials that have shaped modern standards of care in head and neck oncology. He has been a key contributor to landmark studies including KEYNOTE-689, CheckMate 141, Keynote 0-12 and TAX 324, which collectively supported the regulatory approval and integration into practice of pembrolizumab, nivolumab, and TPF-based chemotherapy in head and neck cancer. A dedicated educator and mentor, Dr. Haddad is actively involved in the training of oncology fellows, medical residents, and otolaryngology trainees through both outpatient and inpatient programs at Dana-Farber and Brigham and Women's Hospital. He is a sought-after speaker who lectures widely at national and international meetings. He has authored more than 300 peer-reviewed publications and edited two textbooks in head and neck oncology, reflecting his sustained contributions to research, education, and clinical innovation in the field.



Prof. Aaron Hansen
University of Queensland
Australia

Aaron Hansen is the Medical Director of Cancer Services at the Princess Alexandra Hospital (PAH), the Director of Clinical Trials at the Translational Research Institute (TRI), Professor at the Mayne School of Medicine, University of Queensland and Consultant Medical Oncologist at Greenslopes Private Hospital in Brisbane Australia. Prior to this, he was a staff medical oncologist at Princess Margaret Cancer Centre in Toronto, Canada, where he worked in the drug development phase I program and was lead for genitourinary (GU) cancers. He serves on multiple committees for ANZUP and is a member of the AP Genitourinary Cancer Consortium (APGUCC). His career focuses on clinical trials and developing new therapies for cancer patients. Prof Hansen has led numerous clinical trials, published over 220 papers, and received multiple research awards. He is recognized internationally for his expertise in drug development and GU tumors.



Prof. Pasi Jänne
Dana-Farber Cancer Institute
USA

Senior Vice President for Translational Medicine, Dana-Farber Cancer Institute
Professor of Medicine, Harvard Medical School
American Cancer Society Research Professor
David M. Livingston, M.D. Chair
Director, Belfer Center for Applied Cancer Science
Director, Chen-Huang Center for EGFR Mutant Lung Cancers

Dr. Jänne is the Senior Vice President for Translational Medicine at Dana-Farber Cancer Institute, a Professor of Medicine at Harvard Medical School, and the David M. Livingston, MD Chair at Dana-Farber Cancer Institute. He also serves as the Director of the Belfer Center for Applied Cancer Science at the Dana-Farber Cancer Institute. He is a thoracic medical oncologist and led the Thoracic Medical Oncology Program at Dana- Farber Cancer Institute from 2013 to 2024. After earning his MD and PhD from the School of Medicine at the University of Pennsylvania, Dr. Jänne completed his internship and residency in Medicine at Brigham and Women's Hospital, Boston. He subsequently completed fellowship training at the Dana-Farber Cancer Institute/Massachusetts General Hospital combined program in medical oncology in 2001. In 2002, he earned a Master's Degree in clinical investigation from Harvard University.

Dr. Jänne's research combines laboratory-based studies with translational research and clinical trials of novel therapeutic agents in patients with lung cancer. His main research interests center around understanding and translating the therapeutic importance of oncogenic alterations in lung cancer. He has made seminal therapeutic discoveries, including being one of the co-discoverers of EGFR mutations, and findings from his work have led to the development of several clinical trials. Dr. Jänne has received several awards for his work, including from the American Association for Cancer Research, the European Society for Medical Oncology, the American Society of Clinical Oncology, and the Medal of Honor from the American Cancer Society.



Prof. Bhumsuk Keam
Seoul National University Hospital
Republic of Korea

Bhumsuk Keam, MD, PhD, is a medical oncologist at Seoul National University Hospital (SNUH) and a Professor in the Department of Internal Medicine at Seoul National University College of Medicine. He earned his Medical Degree and his Ph.D. in Internal Medicine from Seoul National University.

Dr. Keam's primary research focuses on head and neck cancer, early-phase (Phase I) clinical trials, immuno-oncology, and translational research. As a principal investigator, he leads numerous clinical trials investigating novel targeted therapies and immune checkpoint inhibitors for head and neck squamous cell carcinoma, salivary gland cancers, and non- small cell lung cancer.

In the realm of translational research, Dr. Keam is highly active in precision oncology. He is currently exploring the complexities of the tumor microenvironment, notably utilizing artificial intelligence-powered spatial analysis of tumor-infiltrating lymphocytes to discover predictive biomarkers for treatment efficacy. Beyond medical interventions, Dr. Keam is deeply committed to the holistic treatment of cancer patients, conducting significant research into palliative medicine, clinical ethics, advance care planning, and improving the quality of end- of-life care.

Dr. Keam is an internationally recognized leader in oncology, having published over 300 peer-reviewed papers with an H-index of 70 and over 22,000 citations. His leadership roles span prominent domestic and international organizations. He serves as the Chair of the Head and Neck Cancer Division for the Korean Cancer Study Group (KCSG), is a member of the ESMO Public Policy Committee, and serves on the Japanese Society of Medical Oncology (JSMO) International Committee. He has also authored several guidebooks to help cancer patients navigate their diagnosis and treatment, and is authoring a scientific book on chemotherapy and tumor biology.

Looking to the future, Dr. Keam is focused on advancing precision oncology through a highly multi-disciplinary approach to head and neck cancers. His upcoming trajectory centers on integrating genomic medicine and spatial transcriptomics to overcome resistance to immunotherapies and perfectly tailor treatments for recurrent, rare, and hard-to-treat cancers. Furthermore, he aims to seamlessly bridge the gap between rigorous scientific innovation and compassionate patient care, expanding global clinical trial networks to elevate the standard of cancer treatment.



Dr. Jonathan Lim
The University of Manchester
United Kingdom

Clinical Senior Lecturer in Cancer Biology
Division of Cancer Sciences, The University of Manchester

Honorary Consultant Medical Oncologist in Advanced Immunotherapy and Cell Therapy
Department of Medical Oncology, The Christie NHS Foundation Trust

Dr Jonathan Lim is a Clinical Senior Lecturer and Honorary Consultant Medical Oncologist in Advanced Immunotherapy and Cell Therapy for solid cancers at The University of Manchester and The Christie NHS Foundation Trust. He is passionate about translational research and in advancing novel advanced therapies, especially in the settings of early-phase clinical trials and first-in-human studies.

His translational research programme focuses on the tumour microenvironment, immune biomarkers, and ex vivo modelling to accelerate development of novel therapies. He completed his PhD on dendritic cell biology in cancer at The Francis Crick Institute and Imperial College London, earning the ACP McElwain Prize for Translational Research and AACR-Pezcoller Scholar-in-Training Award. He subsequently undertook a Crick Postdoctoral Clinical Fellowship in London, and ICF-ESMO Visiting Fellowship in cell therapy at the Netherlands Cancer Institute in Amsterdam.

Dr Lim leads the Manchester-based RISE consortium and is deputy lead of the UK- wide MANIFEST programme, both focused on immunotherapy biomarker research; and serves as Lead Coordinator of the ESMO Translational Research Faculty.



Dr. Herbert Loong
The Chinese University of Hong Kong
HKSAR, China

Associate Professor (Clinical), Department of Clinical Oncology
Medical Director (Oncology), Phase 1 Clinical Trial Centre
The Chinese University of Hong Kong

Consultant (Honorary), Department of Clinical Oncology, Prince of Wales Hospital, Hong Kong

Dr. Herbert H. Loong holds conjoint appointments of Clinical Associate Professor in the Department of Clinical Oncology and Medical Director (Oncology) of the Phase 1 Clinical Trials Centre of The Chinese University of Hong Kong. Dr. Loong obtained his medical degree with a Distinction in Surgery from The University of Hong Kong in 2003. He has completed a Fellowship in Drug Development at Princess Margaret Cancer Centre in Toronto, Canada with a focus on Experimental Therapeutics. His clinical and research interests include Thoracic Oncology and Sarcoma Medical Oncology. He was elected as a Fellow of ASCO (FASCO) in 2024.

Dr. Loong is a recipient of the European Cancer Congress Fellowship Grant (2013), the American Society of Clinical Oncology (ASCO) Annual Meeting Merit Award (2014), the Hong Kong College of Physicians Young Investigators' Award (2014). Dr. Loong led the Lung Cancer Team at CUHK to be bestowed the IASLC Foundation Cancer Care Team Award in recognition for providing the best thoracic oncology care in "Asia & Rest of the World" in 2018.

In recent years, Dr. Loong has co-founded the Asia Pacific Oncology Drug Development Consortium (APODDC) and the Asia Pacific Coalition Against Lung Cancer (APCLC).

Dr. Loong has served or continues to serve in various capacities in various professional international oncology bodies, including the International Affairs Committee (IAC) and the Asia-Pacific Regional Council of ASCO. After completing his role as the Chair of the IAC of ASCO (2024-25), He currently serves as the Immediate Past Chair (2025-2026). He has also served in the Membership, Education and Communications Committees of the IASLC. He has been elected as a Board Member of the Connective Tissue Oncology Society (CTOS) for 2022-2024. Dr. Loong is a current Steering Committee member of the Lung Cancer Policy Network (LCPN). In his prior appointment as a member of Pharmacy and Poisons Board of Hong Kong, he oversaw the registration of medicinal products and clinical trials in the territory.



Dr. Teresa Macarulla
Hospital Clinic Barcelona
Spain

Dr. Teresa Macarulla, as the Head of the Medical Oncology Department at Hospital Clínic de Barcelona, dedicates her professional journey to the clinical and translational investigation of hepatobiliopancreatic tumors. As the Principal Investigator for Phase I, II,and III clinical studies, she actively shapes trial designs within national and international cooperative groups and pharmaceutical-sponsored initiatives. Driven by a specific focus on developing molecular target therapies and defining optimal biological doses, her work has yielded numerous high-profile publications and regulatory approvals from bodies such as the FDA and EMA. Presently, Dr. Macarulla leads a dynamic, multidisciplinary team at the forefront of cancer research, seamlessly integrating clinical expertise with cutting-edge translational approaches to enhance patient care and survival. The team strategically combines clinical and biomarker research breakthroughs, conducting innovative studies on drug effectiveness for tailored treatment approaches in upper GI cancers. Dr. Macarulla's extensive research portfolio spans diverse areas, including the design and development of investigator-initiated clinical trials, participation in collaborative research programs, coordination of late-stage clinical studies, molecular characterization of biliary tract and pancreatic tumors, implementation of liquid biopsy for disease monitoring, and the discovery and validation of novel prognostic and predictive biomarkers for pancreatic and biliary tract tumors. Her leadership extends globally as the Associate Editor of ESMO Gastrointestinal Oncology, co-chair of the ESMO Gastrointestinal Cancers Congress, and contributor to SEOM guidelines, solidifying her status as an internationally renowned clinical researcher with over 160 peer-reviewed publications.



Prof. Haiqiang Mai
Sun Yat-sen University Cancer Center
China

Assistant to the Dean, Director of the Nasopharyngeal Carcinoma Department, Secretary of the General Party Branch for Faculty and Students of the Nasopharyngeal Carcinoma Department, Professor and Doctoral Supervisor. Dr. Mai is a recipient of the National Science Fund for Distinguished Young Scholars, a selected expert of the National High-level Talent Support Program, a Young and Middle-aged Leading Scientist in Science and Technology Innovation recognized by the Ministry of Science and Technology, a Young Oncologist of China, a New Century Excellent Talent of the Ministry of Education, a Guangdong Provincial Leading Talent in Science and Technology Innovation, a Guangdong Provincial Leading Medical Talent, and a winner of the Fulang Award of Sun Yat-sen University.He currently holds multiple academic posts: Chair of the Integrated Rehabilitation Committee for Nasopharyngeal Carcinoma under the Chinese Anti-Cancer Association; Member of the Academic Committee of the Proton Therapy Center at the University of Texas MD Anderson Cancer Center; Vice Chair of the Nasopharyngeal Carcinoma Quality Control Expert Committee of the National Cancer Center; Standing Committee Member of the Nasopharyngeal Carcinoma Committee of the Chinese Anti-Cancer Association; Standing Committee Member of the Nasopharyngeal Carcinoma Expert Committee of the Chinese Society of Clinical Oncology (CSCO); and Chair of the Precision Therapy Committee for Nasopharyngeal Carcinoma under the Guangdong Clinical Medicine Association.His primary research focuses on individualized multidisciplinary therapy for nasopharyngeal carcinoma. As the first or corresponding author, he has published over 100 SCI papers in top-tier medical and oncology journals, including JAMA, Nature Medicine, The Lancet Oncology, Journal of Clinical Oncology, and JAMA Oncology. Six of his research findings have been incorporated into international clinical guidelines such as the NCCN and ASCO guidelines, advancing the development of individualized multidisciplinary treatment for nasopharyngeal carcinoma. As a key contributor, he has been awarded two Second Prizes of the National Science and Technology Progress Award and one First Prize of the Science and Technology Award from the Chinese Anti-Cancer Association.



Dr. Giannis Mountzios
Henry Dunant Hospital Center
Greece

Giannis Mountzios is the Director of the 4th Oncology Department and Clinical Trials Unit at the Henry Dunant Hospital Center in Athens, Greece and a Scientific Instructor in medical oncology at the University of Athens School of Medicine, Athens. Dr Mountzios obtained his MD from the Aristotle University of Thessaloniki in 1998 and graduated from the Hellenic Military Medical Academy in the same year. He completed his residency in internal medicine at the Airforce General Hospital of Athens and in medical oncology at the University of Athens School of Medicine, “Alexandra” University Hospital. He then obtained an MSc in translational and clinical research in oncology from the Institut Gustave-Roussy and the University Paris XI (Paris-Sud), France in 2005 and became board-certified in medical oncology in 2009. He obtained his PhD in thoracic oncology from the University of Athens School of Medicine. Dr Mountzios has served as a member of the Young Medical Oncologists’ Committee and Chair of the Adolescents and Young Adults (AYA) working group of the Educational Committee of the European Society of Medical Oncology (ESMO). He has also served as a member of the board of directors of the Hellenic Society for Medical Oncology (HeSMO) where he was also Chair of the Young Medical Oncologist’s Association. He currently serves as member of the Educational Publications Working Group of ESMO and member of the ESMO Lung Cancer Faculty, as well as member of the Career Development working group of the International Association for the Study of Lung Cancer (IASLC). He was the main editor of the Guide for AYA with Cancer, the Handbook of Supportive and Palliative Care and the Thoracic Essentials of ESMO publications. and has published more than 140 articles in peer-reviewed journals as the first or participating author, including The New England Journal of Medicine, Lancet, Cell, Nature Medicine, Nature Reviews Clinical Oncology, Journal of Clinical Oncology, Annals of Oncology, and Clinical Cancer Research.



Prof. Solange Peters
Lausanne University Hospital
Switzerland

Solange Peters, MD, PhD, is a full professor and director of medical oncology, as well as the thoracic malignancies program in the Department of Oncology at the University Hospital of Lausanne in Lausanne, Switzerland. She received both her doctorate in medicine and PhD from the University Hospital of Lausanne. After completing her clinical education in medical oncology and molecular biology in Switzerland and Italy, Prof. Peters has specialized in thoracic tumors, lung cancer, and pleural tumors.

Her main fields of interest are new biomarkers discovery and validation in preclinical and clinical settings, multimodality strategies for locally advanced non-small cell lung cancer (NSCLC), as well as cancer immunotherapy. Parallel, she acts as the scientific committee chair and Foundation Council member of the European Thoracic Oncology Platform (ETOP). She was recently nominated as the Strategic Advisory Board President of Paris Saclay Cancer Cluster, as well as President of Oncosuisse, the Swiss Cancer umbrella organization. She also acts as President of the Swiss Academy of Multidisciplinary Oncology (SAMO) and is Vice Director of the Swiss National Cancer League. Prof. Peters is active in the educational programs of the International Association for the Study of Lung Cancer (IASLC) and European Society for Medical Oncology (ESMO) - where she created the Women for Oncology Committee and for which she was the youngest President ever for an extended time of 3 years 2020-2022. She is part of the Board of Directors of Galenica SA.

Professor Peters has authored more than 500 peer-reviewed manuscripts and book chapters, acts as Associate Editor of the Annals of Oncology, and of ESMO Open, past Editor in Chief of Lung Cancer, and serves on the editorial board of several other oncology journals.



Prof. David Pinato
Imperial College London
United Kingdom

Dr David J. Pinato is a Clinician Scientist and Consultant Medical Oncologist working within the Department of Surgery and Cancer at Imperial College in London.

He is Director of Developmental Cancer Therapeutics at Imperial College, where he leads a translational research program focusing on the early clinical implementation of novel experimental anticancer therapies to the clinic, with particular emphasis on anti-cancer immunotherapy. He has led the inception of a portfolio of first-in-class studies of immune checkpoint inhibitors in liver cancer, which has represented David's focus of research since graduation with highest honours at the University of Piemonte Orientale "A. Avogadro" in Novara, Italy.

David's group has made significant contributions to the understanding of host mechanisms underpinning response to anti-cancer immunotherapy. David completed his research training being awarded a Masters of Research in Translational Medicine and a Ph.D. in Clinical and Experimental Medicine. David's Ph.D. research focused on inflammation as a pathogenic and prognostic mechanism in liver cancer.

Since his graduation, David matured his clinical skills attending several academic institutions in Europe including the University Hospital in Zürich, the Medical University in Vienna and the University of Ioannina in Greece. He completed his core medical training across some of the busiest acute hospitals in London, being elected member of the Royal College of Physicians (MRCP) in 2014. He has trained in Medical Oncology as part of the Royal Marsden Hospital rotation, having migrated to Imperial College in April 2015 to take up a Clinical Lectureship funded by the National Institute for Health Research (NIHR).

In 2018 David was awarded a Clinician Scientist fellowship funded by the Wellcome Trust Strategic Fund centered on the development of immunotherapy in liver cancer. Hepatocellular Carcinoma (HCC) is the most frequently occurring primary liver tumour that currently ranks 5th by incidence and 3rd by mortality on a global scale. David's studies, supported by international collaborations, have led to the qualification of novel prognostic and predictive biomarkers in HCC, which have been published in leading journals in the field including the Journal of Clinical Oncology, Annals of Oncology, Hepatology and many others.

In 2022 David was promoted Clinical Reader in Medical Oncology.

David's research efforts in liver cancer have been recognized with the prestigious EASL Emerging Leader Award in 2023. David was three time recipient of a Merit Award from the American Society of Clinical Oncology (ASCO) in 2016, 2017, 2019 as well as a fourth Merit Award jointly awarded by ASCO and by the Society for Immunotherapy of Cancer (SITC) in 2019.

David is also a recipient of the ASCO Global Oncology Young Investigator Award 2019 in recognition of his research on HIV-associated hepatocellular cancer. David was also awarded a prize by the British Society of Pharmacology in 2018, the Sylvia Lawler Prize in Oncology from the Royal Society of Medicine in 2016, the European School of Oncology fellowship in 2008 and the Fulbright Visiting Research Scholar Fellowship for 2010/2011.

David lectures internationally in the field of molecular oncology with a specific interest in HCC and acts as a reviewer for a number of peer-reviewed journals including The Lancet, Cancer Discovery, Hepatology, Journal of Hepatology, Oncogene, Gut and many others. He has acted as peer reviewer for a number of grant awarding bodies including the MRC, Breast Cancer Now and Cancer Research UK.



Dr. Arsela Prelaj
Fondazione IRCCS Istituto Nazionale Tumori di Milano
Italy

She is a Consultant Medical Oncologist specializing in thoracic malignancies at the Thoracic Unit of Istituto Nazionale Tumori di Milano in Italy since 2018. She completed her residency in Medical Oncology at Sapienza University, Policlinico Umberto I Hospital in Rome, Italy. During her training, she was awarded the ESMO Clinical Unit Visit in 2017 and spent time at The Christie Hospital in Manchester in the Lung Cancer Division.

Over the past seven years, her research has primarily focused on thoracic malignancies, particularly non-small cell lung cancer (NSCLC) and translational immunotherapy. She recently earned a Ph.D. in Bioengineering and Artificial Intelligence from Politecnico di Milano. She serves as the principal investigator for multiple international clinical trials and is actively involved in coordinating various national and international grant applications in the field of thoracic malignancies and immunotherapy. As the head of the AI-ON-Lab (Artificial Intelligence for Oncology Lab) at Istituto Nazionale Tumori of Milano, she is responsible for leading research and development in the application of AI in oncology. Furthermore, she has been appointed as the Responsible for INT's Research Line 4 program, which focuses on big data, clinical research, healthcare, and outcome research. She is an esteemed member of the ESMO Working Group on Real-World Data and Digital Health, and her expertise in AI has led to collaborations with ESMO starting in 2021. She is leading many EU and National projects: I3LUNG, a 10-million project, APOLLO 11, BuonMarrow (4-million, Bone Marrow-on-Chip). She is PI of the SHAIPED EU project focused on health data body access. Finally, she is the President of the newly founded European Interdisciplinary Society of AI in Cancer Research (ESAC).

She is an ESMO, ASCO, AACR, IASLC and AIOM member and faculty member at many congresses on AI.



Dr. Rila Su
Inner Mongolia People's Hospital
China

Dr. Surila is a distinguished Oncologist and the Director of the Clinical Medical Research Center at a leading hospital in Hohhot, China. Her clinical expertise is in oncology, with a profound commitment to advancing patient-centered care and translational research.

Dr. Surila's academic journey reflects a dedication to global health and rigorous scientific inquiry. She earned her DrPH from the prestigious Johns Hopkins University Bloomberg School of Public Health, where her research focused on healthcare and patient-reported outcome measures. This was preceded by a Ph.D. from Southern Medical University of China, completed in a joint program with Michigan State University, where she investigated the tumorigenesis of hepatocellular carcinoma. Her foundational medical training began with an M.D. from Inner Mongolia Medical University.

Dr. Surila's research is at the intersection of clinical oncology, health outcomes, and molecular biology. Her recent work has centered on improving the quality of life for cancer patients, notably through the validation of patient-reported outcome measures for Mongolian-speaking breast cancer patients on adjuvant endocrine therapy, a project she has presented at the ISOQOL conference. Concurrently, her laboratory has made significant contributions to understanding the molecular mechanisms of lung cancer, with a particular focus on the role of the NCOA5 gene in tumor progression. She has published extensively in high-impact journals, including Cell Death & Disease, Quality of Life Research, and the British Journal of Cancer.

As a principal investigator and sub-investigator, she has led and contributed to numerous clinical trials, including Phase I-III studies on novel immunotherapies like anti-PD-1 and anti-CTLA-4 antibodies, as well as biosimilars for breast and lung cancer. Her grant portfolio, funded by organizations such as the Inner Mongolia Science and Technology Institute and the National Natural Science Foundation, underscores her leadership in investigating the mechanisms of cancer development.

Dr. Surila is an active member of the global oncology community, holding memberships in the Chinese Thoracic Oncology Group (CTONG YOUNG) and the Chinese Society of Clinical Oncology (CSCO). She also serves as Vice Chairman of the Precision Imaging Diagnosis and Treatment Committee for the Beijing Cancer Prevention and Treatment Society. Her contributions have been recognized with multiple awards, including the Inner Mongolia Medical Association Science and Technology First Prize.



Prof. Daniel Tan
National Cancer Centre Singapore
Singapore

Professor Tan is the head of the Division of Clinical Trials and Epidemiological Sciences and a senior consultant in the Division of Medical Oncology at the National Cancer Centre Singapore (NCCS). He also serves as a professor at Duke-NUS Medical School and a senior clinician-scientist at the Genome Institute of Singapore. He leads the Experimental Cancer Therapeutics Unit (ECRU) at the NCCS, one of Asia's largest Phase I units. He is also the Principal Investigator of the Cancer Therapeutics Research Laboratory at NCCS focused on developing representative patient-derived preclinical models to study drug response and resistance. He currently serves as the principal investigator of the National Medical Research Council ("NMRC") Lung Cancer Large Collaborative Grant in Singapore (2025-2029 and previously 2019–2023) and chairs the Asian Thoracic Oncology Research Group, a regional platform for translational research and clinical trials. He is also currently a director of Epoch Biosciences Pte. Ltd. He has contributed to over 200 peer-reviewed articles in journals such as Nature, Nature Medicine, and The Lancet Oncology.

Professor Tan holds a Bachelor of Science with first-class honors in tumor biology from University College London, a Bachelor of Medicine and Surgery from St Bartholomew's and the Royal London School of Medicine and Dentistry, and a PhD from the National University of Singapore. He is a member of the Royal College of Physicians of the United Kingdom and serves in leadership roles in global oncology, including Chair of the IASLC Education Committee.



Prof. Pietro Veronesi
The University of Chicago
USA

Pietro Veronesi is the Sherman and Vivian Chao Distinguished Service Professor of Finance at the University of Chicago, Booth School of Business. He is also a research associate of the National Bureau of Economic Research and a research fellow of the Center for Economic and Policy Research. Additionally, he is a former director of the American Finance Association and co-editor of the Review of Financial Studies.

Veronesi conducts research that focuses on asset pricing, stock and bond valuation under uncertainty, bubbles and crashes, return predictability and stochastic volatility. Most recently, he has been interested in studying, both theoretically and empirically, the interaction between government interventions and the behavior of asset prices. His work has appeared in numerous publications, including the Journal of Political Economy, American Economic Review, Quarterly Journal of Economics, Journal of Finance, Journal of Financial Economics, and Review of Financial Studies. He is the recipient of several awards, including the 2015 AQR Insight award, the 2012 and 2003 Smith Breeden prizes from the Journal of Finance; the 2008 WFA award; the 2006 Barclays Global Investors Prize from the EFA; the 2006 Fama/DFA prizes from the Journal of Financial Economics; and the 1999 Barclays Global Investors/Michael Brennan First Prize from the Review of Financial Studies.

In summer 2020, Professor Veronesi co-founded PREDOC (Pathways to Research and Doctoral Careers, https://predoc.org), a consortium of universities and research institutions that aims to foster a talented and diverse pipeline of students in doctoral programs. PREDOC members work together to expand the talent pool, and to inform, engage, mentor, and educate undergraduates from any background to help them be competitive in the market of pre-doctoral research assistants ("pre-docs"), and in doctoral programs.

Professor Veronesi teaches both masters- and PhD-level courses. He is the recipient of the 2009 McKinsey Award for Excellence in Teaching.

His undergraduate work was in economics at Bocconi University where he received a laurea magna cum laude with honor in 1992. He earned a master's degree with distinction in 1993 from the London School of Economics. He joined the Chicago Booth faculty upon obtaining his PhD in Economics from Harvard University in 1997.



Prof Yi-Long Wu
Guangdong Provincial People's Hospital
China

Prof. Yi-Long Wu is a tenured professor of Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences and Guangdong Lung Cancer Institute. He is the Past President of Chinese Society of Clinical Oncology (CSCO, 2013-2017), the President of Chinese Thoracic Oncology Group(C-TONG). He served as a Member of Board of Directors of the International Association Study of Lung Cancer (IASLC) from 2013-2017 and a past Member of staging committee of the IASLC (2009-2017), conference Chair of the 2020 World Conference on Lung Cancer, Chair of the Joint IASLC-CSCO-CAALC Session at WCLC (2013-2023).

He graduated from Sun Yat-sen University of Medical Sciences in 1982 and completed his thoracic surgery training in Germany in 1989. His main research interests are the multidisciplinary therapy on lung cancer in translation medicine and evidence-based medicine in oncology. He plays a leadership role in Chinese lung cancer clinical research and has been the Principal Investigator or Co-PI of more than 50 international or national multicenter clinical trials. He has contributed 20 books on cancer and has published more than 500 articles in peer-reviewed journals including New Engl J Med, Nat Med, J Clin Oncol, Lancet Oncol, Ann Oncol, Cancer Cell and J Thorac Oncol. He played an important role in adjuvant target treatment for resected NSCLC. ALINA, ADAURA, ADJUVANT/CTONG1104 and EMERGING/CTONG1103 were the most important papers in this field. He also serves on the editorial boards of Nature Review Clinical Oncology, Annual of Oncology, Lung Cancer, Annals of Surgical Oncology.

He received the IASLC Science/Paul A. Bunn, Jr. MD awards in 2015 and Awards of Highly Cited Research in the field of Clinical Medicine 2018- 2023.



Dr. Yang Xia
Second Affiliated Hospital of
Zhejiang University
China

Dr. Xia is an associate professor and deputy director of the Department of Respiratory and Critical Care Medicine at the Second Affiliated Hospital of Zhejiang University School of Medicine. He specializes in treating lung cancer, with a focus on precision medicine and translational research. He has served as first or corresponding author on over 60 peer-reviewed articles, including the top tier journals such as Nature Reviews Clinical Oncology, Nature Communications, The Lancet Respiratory Medicine, Annals of Oncology, Journal of Thoracic Oncology, et.al. Dr. Xia is a member of numerous academic societies, including the Multidisciplinary Clinical Science Committee of International Association for the Study of Lung Cancer (IASLC), American Thoracic Society, European Respiratory Society and Chinese Society of Clinical Oncology.



Dr. Yongchang Zhang
Hunan Cancer Hospital
China

M.D., Chief Physician, Professor, Doctoral Supervisor

Director, Oncology Investigational New Drug Research Ward

Director, Hunan Engineering Research Center for Novel Drug Development in Lung Cancer

2025 National "New Era Youth Pioneer Award"

2025 Hunan Provincial "Advanced Worker"

Invited Abstracts

Day 1 – Saturday, 7 November 2026

GU Malignancy

Overcoming the Immune-cold Microenvironment: Emerging Novel Strategies in Metastatic Prostate Cancer P30
Prof. Aaron HANSEN
The University of Queensland, Australia

The Optimal Sequencing and Practical Consideration of Combination Therapies in Resected and Metastatic Renal Cell Carcinoma P31
Prof. Viktor GRÜNWALD
University of Essen, Germany

Small Cell Lung Cancer

Development of Treatment Targeting DLL3 P32
Dr. Giannis MOUNTZIOS
Henry Dunant Hospital Center, Greece

State-of-Art Therapy for ES-SCLC P33
Prof. Yilong WU
Guangdong Provincial People's Hospital, China

Head and Neck Cancer

Advances in Immunotherapy for the Treatment of Locally Advanced NPC P34
Prof. Haiqiang MAI
Sun Yat-sen University Cancer Center, China

Novel Biomarkers for Individualizing Immunotherapy in Patients with Recurrent/Metastatic Head and Neck Cancer P35
Prof. Bhumsuk KEAM
Seoul National University Hospital, Republic of Korea

Day 2 – Sunday, 8 November 2026

Hepatocellular and Pancreatic Cancer

Neoadjuvant Therapy for HCC P36
Prof. David PINATO
Imperial College London, United Kingdom

Lung Cancer (NSCLC)

Novel Therapy on the Horizon for AGA Driven NSCLC P37
Prof. Pasi JÄNNE
Dana-Farber Cancer Institute, USA

CTONG - Young Session on IIT

From Participating Site to Trial Initiator: Building IIT Capacity in Inner Mongolia, China P38
Dr. Rila SU
Inner Mongolia People's Hospital, China

Overcoming the Immune-cold Microenvironment: Emerging Novel Strategies in Metastatic Prostate Cancer

Aaron Hansen
University of Queensland, Australia

Metastatic prostate cancer remains largely refractory to immunotherapy, reflecting a profoundly "immune-cold" tumour microenvironment characterised by low T-cell infiltration, immunosuppressive myeloid populations, heterogeneous antigen expression and multiple mechanisms of T-cell dysfunction. However, a new generation of immune-redirection strategies is challenging this paradigm. This presentation will review emerging approaches designed to recruit, activate and sustain antitumour T-cell responses in metastatic prostate cancer, with particular emphasis on bispecific T-cell engagers targeting prostate-associated surface antigens including PSMA, STEAP1 and KLK2. Early clinical experience, including encouraging activity with STEAP1-directed Xaluritamig, provides proof-of-concept that pharmacologic T-cell redirection can generate meaningful responses even in heavily pretreated metastatic castration-resistant disease. The evolving role of cellular therapies, including CAR-T and other adoptive T-cell platforms targeting PSMA, PSCA and related antigens, will also be discussed alongside novel vaccines, cytokine-based approaches and rational combinations. Key barriers—including cytokine release syndrome, on-target/off-tumour toxicity, antigen heterogeneity and escape, T-cell exhaustion and limited durability, will be considered. Finally, the presentation will explore next-generation engineering, biomarker-guided patient selection and combination strategies aimed at converting immune exclusion into clinically effective and durable antitumour immunity.

The Optimal Sequencing and Practical Consideration of Combination Therapies in Resected and Metastatic Renal Cell Carcinoma

Viktor Grünwald
University of Essen, Germany

Immune checkpoint inhibitors (ICIs) based therapies have reshaped the management of renal cell carcinoma (RCC) across disease stages. Adjuvant pembrolizumab is the first systemic therapy to improve overall survival after nephrectomy in patients at increased risk of recurrence, whereas other adjuvant ICI strategies have failed. In the metastatic setting, ICI–VEGFR tyrosine kinase inhibitor (TKI) doublets and dual checkpoint blockade with nivolumab plus ipilimumab are standard first-line options, yet no head-to-head data defined an optimal regimen.

We will examine evidence-based sequencing across the disease continuum, including treatment selection by IMDC risk, histologic features and disease burden. We address the growing population relapsing after adjuvant ICI, the lack of benefit from ICI rechallenge in the second line (CONTACT-03, TiNivo-2), and the emerging role of HIF-2α inhibition with belzutifan. Practical aspects include patient seöection and managing overlapping toxicities of ICI–TKI combinations, attributing adverse events, dose modification or treatment breaks.
Rational sequencing demands individualized, biology- and toxicity-informed decisions; prospective trials and predictive biomarkers remain urgently needed.

Development of Treatment Targeting DLL3

Giannis Mountzios
Henry Dunant Hospital Center, Greece

Advances in Molecular Biology have enabled the identification of novel, clinically relevant biomarkers of drug efficacy in SCLC. Delta-like Ligand 3 (DLL3) is a neuroendocrine transcription factor that synergizes with upregulation of the ASCL pathway to mediate neuroendocrine differentiation and down regulates the NOTCH pathway, leading to a more biologically aggressive phenotype. Biotechnological advances led to the development of T-cell engagers (Tarlatamab, Gocatamig, Obrixtamig, Alveltamig), that act as “molecular bridges” that engage DLL3-expressing SCLC cells with T-cells, enabling thus immunogenic cell-death. Tarlatamab was the first T-cell engager approved globally as second-line treatment in ES-SCLC, by improving overall survival when compared with second-line standard chemotherapy (topotecan, amrubicin, lurbinectedin). More recently, ADCs targeting DLL3, like ZL-1310, utilize a Topo-1 payload linked to an antibody targeting DLL3-expressing cells via a cleavable linker and are currently being tested in clinical trials, Important questions to be answered in the near future include the optimal implementation of T-cell engagers and antibody-drug conjugates to the current therapeutic algorithm, including duration of treatment, and how to best combine those two agents in order to enhance efficacy while minimizing synergistic or additive toxicity.

State-of-Art Therapy for ES-SCLC

Yilong Wu
Guangdong Provincial People's Hospital, China

First-line ES-SCLC remains platinum–etoposide plus a PD-(L)1 inhibitor (atezolizumab or durvalumab). The biggest advance is **DeLLphi-305**: after durvalumab–chemo induction, adding **tarlatamab** (DLL3×CD3 BiTE) to durvalumab maintenance gave statistically significant and clinically meaningful gains in **OS, PFS, and ORR** vs durvalumab alone, with no new safety signals. This moves T-cell engager therapy from 2L relapse into **1L maintenance**, reframing maintenance as active immune redirection rather than just IO continuation. Tarlatamab is already approved in 2L+ ES-SCLC (DeLLphi-304: mOS 13.6 vs 8.3 mo).

For **B7-H3**, the clinical reality in SCLC is ADCs. HS-20093 and YL201 show strong response and overall survival benefits in relapsed disease. Taishan 302 and ARTEMIS 008 both achieved HR 0.46 (ARTEMIS 008 mOS 18.5 vs 10.3mo, Taishan 302 mOS 13.3 vs 9.4mo), both vs topotean in relapsed ES-SCLC.
Whereas **B7-H3×CD3 TCEs remain preclinical/early-phase** because B7-H3 is broadly expressed on normal tissues, causing higher off-tumor toxicity. DLL3 remains the cleaner TCE target; B7-H3 fits antibody–drug conjugates.

PCI in ES-SCLC is essentially de-adopted. In the IO/ADC era, prophylactic cranial irradiation improves intracranial relapse control but worsens cognition without clear OS benefit versus MRI surveillance. Standard is contrast **MRI brain surveillance** (q2–3 mo year 1, q3–6 mo year 2). PCI is reserved for poor MRI access, noncompliance, or patient preference after shared decision-making; if used, 20–25 Gy in 5–10 fractions, avoid >30 Gy, consider memantine/hippocampal avoidance.

Net paradigm: **stronger systemic deepening** (tarlatamab maintenance, B7-H3 ADCs) plus **MRI-driven CNS management**, not prophylactic whole-brain radiotherapy. Limited-stage SCLC PCI is still debatable; extensive-stage PCI is not. Trials remain preferred at relapse.

Advances in Immunotherapy for the Treatment of Locally Advanced NPC

Haiqiang Mai
Sun Yat-sen University Cancer Center, China

Nasopharyngeal carcinoma (NPC) is an EBV-related malignancy highly prevalent in southern China, accounting for 48% of global cases. Induction chemotherapy plus concurrent chemoradiotherapy (IC+CCRT) is the standard curative regimen for locally regionally advanced NPC (LA-NPC), yet approximately 20% of patients develop recurrence or distant metastasis. Rich lymphocyte infiltration and high PD-L1 expression make NPC suitable for anti-PD-1 immunotherapy, which has been established as first-line treatment for recurrent/metastatic NPC.

The optimal timing of immune checkpoint inhibitors for LA-NPC remains debated. The CONTINUUM trial revealed improved event-free survival with full-course sintilimab combined with IC+CCRT, but no overall-survival benefit and increased toxicities. Radiotherapy-phase immunotherapy yields unsatisfactory outcomes partly because radiation damages anti-tumour lymphocytes and draining-lymph-node immunity. The DIPPER trial confirmed survival benefits of post-CCRT camrelizumab adjuvant therapy. BEACON and other trials demonstrated that neoadjuvant PD-1 plus GP chemotherapy raises complete-response rates with good tolerability.

Preclinical and cross-cancer data support neoadjuvant immunotherapy for activating systemic anti-tumour T-cell immunity. To date, ICI-based regimens have not consistently achieved overall-survival gains in LA-NPC. Adjuvant immunotherapy is a practical alternative, whereas neoadjuvant chemo-immunotherapy and the sandwich model await further prospective validation. Future studies should compare different immunotherapy strategies against standard IC+CCRT for risk-stratified patients.

Novel Biomarkers for Individualizing Immunotherapy in Patients with Recurrent/Metastatic Head and Neck Cancer

Bhumsuk Keam
Seoul National University Hospital, Republic of Korea

Immune checkpoint inhibitors targeting the PD-1/PD-L1 axis are foundational systemic therapy for R/M HNSCC, yet most patients derive no durable benefit. In routine practice, tumour PD-L1 expression reported as CPS remains the only broadly implemented treatment-selection biomarker: in KEYNOTE-048, pembrolizumab monotherapy improved overall survival versus cetuximab-chemotherapy in CPS≥20 and CPS≥1 populations, while CPS<1 patients did not benefit. Its predictive performance is nonetheless imperfect, limited by intratumoural heterogeneity, assay variability and modest negative predictive value.

This lecture reviews the biomarkers most likely to improve patient-level decisions beyond CPS, graded by evidence level. Tumour mutational burden and the gene expression profile are independently associated with response in KEYNOTE correlative analyses and perform best in combination; MSI-H/dMMR is highly predictive but rare in HNSCC. Spatial immune contexture- multiplex immunofluorescence, stromal B cells and tertiary lymphoid structures - can refine or outperform CPS. Tumour-informed circulating tumour DNA kinetics and clearance predict response and progression-free survival early on treatment.

Because response is governed by partially independent dimensions - antigenicity, inflammation, immune exclusion and immune escape - no single marker suffices. The most credible near-term path is a composite, multi-layer framework combining antigenicity, tumour-inflammation state, spatial architecture and early on-treatment kinetics, validated in trials that lock assay and cutoff prospectively.

Neoadjuvant Therapy for HCC

David Pinato
Imperial College London, United Kingdom

Evidence for the efficacy of neoadjuvant immunotherapy is accumulating in various solid tumor types, such as melanoma, breast cancer, and lung cancer. In the clinical setting of early-stage, operable HCC, the optimal liver function reserve, good performance status, greater potential to tolerate immune-related toxicities, and increased chances of cure in this patient population provide support for the neoadjuvant use of immunotherapy.

Neoadjuvant immunotherapy in HCC may improve surgical outcomes, treat micrometastatic disease early in the disease trajectory, as well as provide an in vivo test of the sensitivity of the tumor to further therapy that may be given following surgery or ablation.

Novel Therapy on the Horizon for AGA Driven NSCLC

Pasi Jänne
Dana-Farber Cancer Institute, USA

Despite the efficacy of next generation targeted therapies including osimertinib for EGFR mutant NSCLC and lorlatinib for ALK positive NSCLC, there continues to be a need to develop new therapeutic strategies especially when cancers develop on target secondary mutations. For EGFR, that includes the development of novel kinase inhibitors, such as BH-30643 and VRN110755, capable of inhibiting EGFR despite the EGFR C797S mutation. Allosteric EGFR inhibitors, which bind a distinct site within EGFR, are efficacious in preclinical models and shortly entering clinical development.

The next generation ALK inhibitor, Neladalkib, has shown promising clinical activity in patients treated with prior Alk inhibitors including those treated with lorlatinib and those whose tumors harbor the ALK G1202R resistance mutation. Another novel ALK targeted therapy is the ALK degrader TRI-611 which binds a region outside of the kinase domain and has preclinically demonstrated activity across a broad range of ALK secondary resistance mutations.

Another new therapeutic approach for AGA driven NSCLC is to target cancers harboring a concurrent MTAP loss with PRMT5 inhibitors. Homozygous MTAP loss is found in ~15% of all cancers including NSCLCs harboring AGAs. PRMT5 inhibitors, such as BMS-986504 have demonstrated single agent efficacy across AGA subset including, EGFR, ALK, and KRAS mutant NSCLC. In addition, preclinical studies suggest potential combination effects when combined with AGA targeted therapies.

From Participating Site to Trial Initiator: Building IIT Capacity in Inner Mongolia, China

Rila Su
Inner Mongolia People's Hospital, China

Clinical trial capacity in China remains unevenly distributed, with investigator-initiated trials (IITs) concentrated in major academic centers. Regional referral hospitals may serve large cancer populations and possess substantial clinical and research infrastructure, yet have limited capacity to independently initiate and lead prospective trials. Inner Mongolia People's Hospital illustrates this gap. As a major regional referral center, it serves cancer patients from across Inner Mongolia and has extensive experience as a participating site in multicenter trials. The hospital has also established GCP trial infrastructure, biobanking, next-generation sequencing, flow cytometry, and bioinformatics platforms. However, most trials remain externally initiated, with limited experience in locally led IITs.

This experience highlights a "missing middle" between having patients and technology and developing sustainable trial leadership. Key gaps include protocol development, biostatistics, prospective data systems, research coordination, regulatory support, quality assurance, funding, mentorship, and multicenter collaboration. Geographic dispersion and unequal access to research networks further challenge regional centers.

A pragmatic pathway may begin with prospective registries and biobank-linked translational cohorts, progress to feasible single-center investigator-led studies, and ultimately develop multicenter IITs through established collaborative networks such as CTONG. Moving from participating site to trial initiator requires not simply more infrastructure, but an integrated research ecosystem **connecting patients, clinical questions, data, biospecimens, investigators, and collaborative networks.**

Submitted Abstracts

Abstract No.	Name	TITLE
P01	Li ZHANG	SCRNA+TCR+BCR-SEQ IDENTIFIED THE PROPORTIONS AND GENE EXPRESSION PROFILES OF DUAL-RECEPTOR T AND B LYMPHOCYTES IN NPC AND NLH
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P01 SCRNA+TCR+BCR-SEQ IDENTIFIED THE PROPORTIONS AND GENE EXPRESSION PROFILES OF DUAL-RECEPTOR T AND B LYMPHOCYTES IN NPC AND NLH

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Although the relationship between single-receptor lymphocytes and cancer has been thoroughly investigated, the origins and biological roles of dual-receptor lymphocytes in the tumor microenvironment (TME) remain poorly understood. Nasopharyngeal carcinoma (NPC), a malignancy strongly associated with immune cell infiltration, presents a unique opportunity to study these enigmatic cells in the context of cancer. In this study, we employed single-cell RNA sequencing, together with T cell receptor (TCR) and B cell receptor (BCR) sequencing (scRNA + qTCR + BCR-seq), to analyze samples obtained from 7 patients with NPC and 3 with nasopharyngeal lymphatic hyperplasia (NLH), which serves as a non-malignant control representing an inflammatory microenvironment.

Our findings demonstrate, for the first time, the presence of dual-receptor lymphocytes in both the TME of NPC and the inflammatory milieu of NLH. The detection of these cells in both malignant and non-malignant tissues suggests that they may play fundamental roles in immune surveillance and response. We further observed clonal expansion of dual-receptor lymphocytes, implying their potential involvement in antigen-driven immune activity. Detailed lineage analysis revealed that these cells predominantly derive from memory lymphocyte populations. Regarding receptor pairing, β+α1+α2 was identified as the most common form for dual TCRs, while heavy+κ+λ was the leading configuration for dual BCRs.

Comprehensive gene expression profiling enabled us to compare the function of dual-receptor lymphocytes with that of their single-receptor counterparts, revealing significant differences that may underlie unique immunological functions. This study advances our understanding of dual-receptor lymphocytes and their role in shaping the immunological landscape of NPC. By contrasting findings with NLH, we also provide new insights into the immunological shifts that occur during malignant transformation, suggesting new avenues for diagnostic and therapeutic advances."

P02 EFFICACY AND SAFETY OF AURICULAR VAGUS NERVE STIMULATION (AVNS) ON CANCER RELATED SYMPTOMS: A SYSTEMATIC REVIEW

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Background: Auricular Vagus Nerve Stimulation (AVNS) is an emerging neuromodulation technique with promising potential in alleviating cancer-related symptoms (CRS). Given its growing clinical relevance and implications for cancer patient care, we conducted a systematic review to evaluate the efficacy, safety, and commonly reported treatment parameters of AVNS in managing CRS.

Methods: We systematically searched for randomised controlled trials (RCTs) from six databases (MEDLINE, Embase, the Cochrane Library, CINAHL, China National Knowledge Infrastructure, and WanFang Database) were searched from inception until 21st January 2026. RCTs in either English or Chinese reporting the effect of AVNS on cancer-related symptoms in cancer patients were included. Methodological quality was evaluated using the revised Cochrane risk-of-bias tool. Meta-analysis was not performed due to high heterogeneity among studies.

Findings: Seven RCTs were included in this study (596 subjects). AVNS significantly improved cancer-related symptoms on chemotherapy-induced nausea and vomiting, cancer therapy-related neuropathic pain, and cancer-related fatigue when compared with control or sham stimulation. There were certain concerns on risk-of-bias for most studies. Commonly used parameters were identified. Adverse events were low, with no serious complications.

Interpretation: This systematic review suggested that AVNS may be a safe and effective treatment to relieve a range of cancer-related symptoms for cancer patients. More RCTs with larger sample size and robust methodology are warranted in future research.

P03 GENOTYPE-DIRECTED DE-ESCALATION OF IMMUNOTHERAPY IN LYNCH SYNDROME COLORECTAL CANCER: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: Lynch syndrome (LS)-associated colorectal cancer (CRC) exhibits higher neoantigen loads than sporadic dMMR/MSI-H tumors, yet whether LS genotype confers superior immunotherapy benefit remains unresolved.

Methods: Systematic search of PubMed, Web of Science, and Embase (Dec 2015–Dec 2025) identified studies with genetically confirmed LS-CRC receiving immune checkpoint inhibitors (ICI). Primary outcomes were progression-free survival (PFS), complete response (CR), and objective response (OR). Random-effects meta-analyses with two-sided significance tests compared LS vs sporadic dMMR/MSI-H cohorts. Effect estimates were reported as point estimates with 95% confidence intervals or standard deviations.

Results: Nineteen studies (1,663 patients) were included. LS patients demonstrated significantly superior PFS (HR=0.51; 95% CI, 0.30–0.88; P=0.016) and higher CR rates (RR=1.15; 95% CI, 1.02–1.29; P=0.026) versus sporadic dMMR/MSI-H. Notably, neoadjuvant PD-1 monotherapy achieved equivalent pathological CR to dual-ICI (77% vs 76%; P=0.991), whereas metastatic disease favored dual blockade (ORR 64% vs 43%; P=0.024).

Conclusions: LS-CRC derives superior immunotherapy benefit, supporting genotype-directed stratification. Neoadjuvant monotherapy adequacy suggests de-escalation potential in early-stage LS, while metastatic disease requires intensified dual blockade.

P04 EVALUATING A SOCIAL INTERDEPENDENCE THEORY-BASED INTERVENTION FOR ENHANCING DIGITAL HEALTH LITERACY IN OLDER ADULTS WITH CANCER: A TWO-ARM QUASI-EXPERIMENTAL DESIGN

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Objective: To evaluate the effectiveness of a nurse-led, group-based educational intervention grounded in Social Interdependence Theory in improving digital health literacy, self-efficacy, health self-management behaviors, and reducing technology-related anxiety in older adults with cancer.

Methods: A two-arm quasi-experimental design with a historical control was employed. 84 older adults with cancer were allocated chronologically by hospital admission to either the control group (July–October 2024) or the intervention group (October 2024–January 2025). Ultimately, N = 80 participants (control: n = 41; intervention: n = 39) completed the study. Outcomes were evaluated using traditional pre-post and between-group statistics, alongside network analysis to explore structural reconfigurations in the interaction among these variables.

Results: Following the intervention, the intervention group demonstrated significant improvements in digital health literacy ($P < 0.05$), self-efficacy ($P < 0.001$), and health self-management behaviors ($P < 0.05$), alongside a marked reduction in technology-related anxiety ($P < 0.001$), compared to the control group, which showed no significant changes. Crucially, network analysis revealed profound structural optimization. Self-efficacy transitioned remarkably from an isolated node to a functionally integrated component of the network. Furthermore, Health Mutual Assistance remained the most central node (rs = 1.288 post-intervention), functioning as the key intervention target. Although Technophobia emerged as a secondary core node, its expected negative influence within the system was substantially mitigated (EI = 0.583).

Conclusions: The nurse-led intervention not only improved individual clinical indicators but fundamentally reshaped how these variables interact. Routine oncology care should actively incorporate structured peer-driven learning to help older patients overcome digital barriers and manage their illness more effectively.

Keywords Social interdependence theory, Older adults with cancer, Digital health literacy, Self-efficacy, Health self-management behaviors, Technology-related anxiety

P05 FAP+ FIBROBLASTS INDUCE METS VIA IL-33 TO PROMOTE THYROID CANCER DEDIFFERENTIATION

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Background: Anaplastic thyroid carcinoma (ATC) is a highly malignant tumor characterized by complete dedifferentiation and poor prognosis, yet the role of the interaction between FAP^+ cancer-associated fibroblasts (CAFs) and macrophages—specifically the formation of macrophage extracellular traps (METs) induced via IL-33—in thyroid cancer dedifferentiation remains poorly understood. This study aims to elucidate the molecular mechanism of the FAP-STAT3-IL-33-METs signaling axis underlying this process.

Methods: FAP^+ and FAP^- CAFs were isolated from ATC tissues. Transcriptomic sequencing, ChIP-qPCR, and luciferase reporter assays were performed to identify and validate the key transcription factor regulating IL-33 expression. Macrophages were stimulated with recombinant IL-33, and METs formation was assessed by immunofluorescence and quantification of dsDNA/MPO release. A macrophage-thyroid cancer cell co-culture model was established to evaluate the effect of METs on thyroid cancer dedifferentiation. Clinical relevance of the signaling axis was analyzed using multiplex immunofluorescence and FAPI PET/CT in patient cohorts.

Results: FAP^+ CAFs were significantly enriched in dedifferentiated regions of thyroid cancer, and their abundance correlated with METs formation and poor patient prognosis. Mechanistically, STAT3 was activated in FAP^+ CAFs and directly bound to the IL-33 promoter to enhance its transcription. IL-33 induced METs formation in macrophages via the ST2 receptor and histone citrullination-dependent chromatin decondensation. METs promoted the downregulation of differentiation markers (NIS, TG, TSHR) and the acquisition of stem-like properties in thyroid cancer cells. In vivo experiments confirmed that targeting the FAP-IL-33-METs axis suppressed tumor dedifferentiation and progression. Clinical analysis revealed spatial co-localization of FAP^+ CAFs, IL-33 expression, and METs in ATC regions, which was associated with worse patient survival.

Conclusion: The FAP-STAT3-IL-33-METs signaling axis drives thyroid cancer dedifferentiation and represents a potential therapeutic target for intervening in ATC progression.

P06 BLOCKADE OF PD-1 REVERSES PANCREATIC TUMOR PROGRESSION TRIGGERED BY LOW-INTENSITY ELECTRICAL STIMULATION

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Background: Irreversible electroporation (IRE) represents a potential therapeutic approach for pancreatic ductal adenocarcinoma (PDAC). However, differences in tumor size and shape can lead to an uneven distribution of the electric field, causing certain cells to receive only low-intensity electric stimulation (LIES). This study aimed to explore the tumor immune microenvironment following LIES and evaluate the synergistic antitumor effects of LIES combined with anti-PD-1 blockade.

Methods: Orthotopic PDAC models in mice were treated with LIES with or without anti-PD-1 blockade. In vitro assays under varying electric fields assessed tumor cell viability, migration, and epithelial-mesenchymal transition (EMT). Tumor immune microenvironment remodeling was comprehensively profiled using flow cytometry, multicolor immunofluorescence, and single-cell RNA sequencing (scRNA-seq). The underlying mechanism was further investigated via bulk RNA sequencing and validated in vitro.

Results: Orthotopic PDAC models were established and utilized to evaluate, for the first time, the effect of immune modulation and macrophage polarization of LIES. Cell-based assays were conducted to assess the impact of varying field strengths on cell proliferation, migration, and EMT. Anti-PD-1 blockade reversed the tumor growth and systemic antitumor responses following the LIES. Flow cytometry, multicolor immunofluorescence, and scRNA-seq revealed that combination therapy amplified CD8+ T cell activation and M1 macrophage polarization. Further bulk RNA-seq analysis revealed that LIES activated the JAK2-STAT3 pathway, inducing expression of PD-L1 in PDAC cell.

Conclusions: Anti-PD-1 blockade can reverse LIES-driven tumor progression by upregulating CD8+ T cell expression and enhancing M1 polarization. A combination strategy involving anti-PD-1 blockade along with LIES could emerge as a therapeutic approach for PDAC.

P07 SPATIAL ARCHITECTURE OF THE IMMUNE-TUMOR BATTLEFIELD DETERMINES THERAPEUTIC RESPONSE AND PROGNOSIS IN NASOPHARYNGEAL CARCINOMA

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Unraveling the spatial organization of the tumor microenvironment (TME) is essential for precision therapy but remains largely unexplored in nasopharyngeal carcinoma (NPC). Here, we applied imaging mass cytometry to generate a single-cell spatial atlas of 159 NPC patients, mapping cellular heterogeneity and developing the boundary nest ratio to quantify tumor nest topologies that were associated with lymphocyte-rich niches. These niches, orchestrated by B cells, formed reticulated lymphoid networks extending beyond tertiary lymphoid structures, and represented novel prognostic architectures independent of cell abundance. Moreover, the tumor leading edge acted as a dynamic epicenter that predicted global cross-talk. By integrating these spatial features, we defined three NPC subtypes with distinct prognosis and responses to induction chemotherapy and immunotherapy, which were validated in two independent cohorts (n=265) using a clinically applicable classifier. Together, our findings delineate the spatial landscape of immune-tumor battlefield and offers a framework for leveraging spatial insights to inform precision oncology.

P08 COMPLEX DETERMINANTS OF DISCHARGE READINESS IN COLORECTAL CANCER PATIENTS WITH STOMA: A FUZZY-SET QUALITATIVE COMPARATIVE ANALYSIS

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Importance: Discharge readiness is a key predictor of post-discharge outcomes for colorectal cancer patients with a stoma. Although known to be influenced by multiple interacting factors, most existing studies use linear approaches that overlook configurational pathways.

Objective: To identify the necessary and sufficient combinations of individual stoma acceptance, family support, and medical resource accessibility that lead to high discharge readiness.

Design, Setting, and Participants: This cross-sectional study used fuzzy-set qualitative comparative analysis (fsQCA). A total of 195 colorectal cancer patients with a stoma were enrolled from a tertiary hospital in Guangzhou, China, between May 2024 and May 2025. Data were collected via validated scales and a demographic questionnaire.

Interventions: None.

Main Outcomes and Measures: The outcome was high discharge readiness, assessed by the Readiness for Hospital Discharge Scale. Antecedent conditions included stoma acceptance, family support, and medical accessibility. fsQCA examined which condition combinations were sufficient for the outcome.

Results: Participants had a mean age of 50.9 years (SD=17.9); 111 (56.9%) were male. Necessity analysis indicated no single condition was required for high readiness. Sufficiency analysis revealed five configurations (overall consistency: 0.889; overall coverage: 0.497), synthesized into four patient prototypes: (1) Social-Psychological Adaptation-Driven (high acceptance plus strong family support); (2) Family Support-Healthcare Resource Driven (strong family support and good accessibility compensating for moderate acceptance); (3) Individual-Family-Resource Synergistically Driven (high on all three); and (4) Psychological Resilience-Driven (high acceptance even without good accessibility or strong support). A compensatory effect between family support and medical accessibility was noted.

Conclusions and Relevance: High discharge readiness arises via multiple equivalent pathways, underscoring causal complexity. Instead of single-factor interventions, clinical efforts should adopt tailored strategies that harness the integration and compensation among individual, family, and medical resources to improve hospital-to-home transitions.

Keywords: Discharge readiness; Colorectal cancer; Enterostomy; fsQCA; Health services research; Transitional care

P09 LAG3 EXPRESSION IN SOLITARY FIBROUS TUMORS SHAPES AN IMMUNOSUPPRESSIVE MICROENVIRONMENT AND PREDICTS POOR PROGNOSIS

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Solitary fibrous tumor (SFT) is a rare mesenchymal neoplasm whose tumor immune microenvironment (TIME) remains poorly defined. Whether the immune checkpoint lymphocyte activation gene 3 (LAG3) shapes this microenvironment and influences clinical outcome in SFT is unknown. Here we combined single-cell and spatial transcriptomics with multiplex imaging of four specimens, a machine learning prognostic model of 347 patients across three cohorts, and primary SFT cell lines. Single-cell profiling resolved four tumor cell subclusters, with the LAG3 subcluster exhibiting the most pronounced malignant phenotype. High LAG3 expression independently predicted poor prognosis and correlated with mitotic activity, cellular pleomorphism, and Ki-67 index. LAG3-high tumor cells were enriched in EMT, MAPK, KRAS, and NOTCH pathways and engaged in prominent crosstalk with M2 macrophages. LAG3-high tissues were infiltrated by HLA-DPB1 /CD163 macrophages and regulatory T cells. In vitro, LAG3 drove macrophage M2 polarization, which was abolished by the LAG3-blocking antibody relatlimab. These findings establish high LAG3 expression as an independent predictor of poor prognosis in SFT and implicate LAG3-mediated tumor cell–macrophage crosstalk as a druggable axis of immune evasion.

P10 GLIS3 RESTRICTS ANTITUMOR IMMUNITY BY SILENCING TYPE I INTERFERON SIGNALING VIA SETD7-MEDIATED CHROMATIN REMODELING

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Transcriptional repression of type I interferon (IFN-I) signaling is a key mechanism of immune evasion, but the epigenetic regulators involved remain incompletely understood. Here we show that PRMT5-mediated methylation stabilizes transcription factor GLIS3, enabling it to function as a master epigenetic repressor of tumor cell intrinsic type I interferon (IFN-I) signaling in nasopharyngeal carcinoma (NPC). Mechanistically, GLIS3 directly represses SETD7, limiting H3K4me1-mediated chromatin accessibility and silencing key IFN-I transcription factors. PRMT5 methylates GLIS3 at R918, protecting it from K48-linked ubiquitination and degradation. Genetic or pharmacological disruption of this axis destabilizes GLIS3, restores chemoradiotherapy (CRT)-induced IFN-I activation, and reinvigorates $CD8^+$ T cell-dependent antitumor immunity. Among 112 NPC patients receiving CRT, high PRMT5 expression independently predicts poor survival. These findings establish the PRMT5-GLIS3-SETD7 axis as a druggable epigenetic checkpoint linking protein methylation to chromatin regulation, providing a mechanistic rationale for combining PRMT5 inhibitors with CRT in NPC.

P11 FIRST-LINE IPILIMUMAB PLUS NIVOLUMAB IN CHINESE PATIENTS WITH ADVANCED RENAL CELL CARCINOMA: A REAL-WORLD STUDY ON THE EFFICACY, SAFETY, AND LONG-TERM OUTCOMES

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Purpose: Ipilimumab plus nivolumab (IPI-NIVO) is a standard first line regimen for intermediate- and poor-risk metastatic renal cell carcinoma (mRCC), but real-world data in Chinese patients are scarce. We report, to our knowledge, the first and largest Chinese cohort receiving first-line IPI-NIVO.

Methods: In this retrospective study of 60 consecutive Chinese patients with mRCC, the efficacy, long-term and safety outcomes were analysed. Objective response was assessed as per RECIST 1.1. Progression-free (PFS) and overall survival (OS) were estimated by Kaplan–Meier analysis; a pre-specified six-month landmark analysis with Cox adjustment was undertaken to evaluate the association between early disease control and survival.

Results: Median age was 63 years (IQR 57–67); 33% had poor-risk IMDC. The objective response rate was 20.0% (95% CI 10.8–32.3) in the intention-to-treat population and 22.6% (95% CI 12.3–36.2) among evaluable patients, with no complete responses observed; the disease control rate was 50.0%. Median PFS and OS were 10.7 (95% CI 4.1–28.2) and 19.6 (95% CI 12.7–34.1) months; 5-year OS was 19.5%. Disease control at the 6-month landmark was strongly associated with OS (adjusted hazard ratio 0.16; 95% CI 0.05–0.49; p = 0.0014). Any-grade and grade ≥ 3 immune-related adverse events occurred in 58.3% and 5.0%, respectively.

Conclusions: First-line IPI-NIVO achieved PFS comparable to CheckMate 214 and other non-Chinese Asian series in this cohort. OS was much lower than in the randomised trial, likely reflecting limited access to subsequent-line therapies, as only 47% of first-line failures received any second-line treatment, highlighting the healthcare inequality and importance of health policy in resource allocation in underprivileged settings especially in the Asia-Pacific. Durable disease control, rather than objective response, characterised long-term survival, further suggesting that surrogacy for immunotherapy in real-world clinical practice may differ from non-immune therapies and well-controlled randomised controlled trials.

P12 REAL-WORLD EXPERIENCE OF WHOLE-GENOME-INFORMED WHOLE-GENOME CFDNA MINIMAL RESIDUAL DISEASE TRACKING IN ROUTINE PRACTICE

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Background: Tumor-informed cell-free DNA (cfDNA) minimal residual disease (MRD) testing is clinically available across the Asia-Pacific region, but little is published on its clinical impact.

Methods: We reviewed the results of whole-genome-informed whole-genome cfDNA MRD assays (MRDVision(TM)), wherein baseline tumor profiling establishes a somatic fingerprint tracked in cell-free DNA. Data were collected between November 2024 and July 2026. The cohort included 50 patients (81 timepoints) from 19 institutions across Hong Kong, Taiwan, and the United States. Stage was extracted from clinical records; serial imaging, tumor markers, and histopathology were available for three patients.

Results: The median age was 57 years; 26 were male. Sixteen tumor types were represented, primarily colorectal (16/50). Thirty of 45 patients with recorded stage had stage IV disease. Circulating tumor DNA (ctDNA) was detected in 32 of 50 patients (64%) and at 50 of 81 timepoints (median tumor fraction among detected samples, 1,931 ppm; limit of detection, 1-786 ppm). Sixteen had serial testing (median interval, 84 days). Three cases illustrate distinct applications.

Case 1: Stage III sigmoid colon cancer (pT3N1b, LVI+; TP53-mutated, MSS): Undetectable ctDNA four weeks post-colectomy supported the abbreviation of adjuvant XELOX to four cycles; six-month CT showed no recurrence.

Case 2: Recurrent esophageal squamous cell carcinoma (CPS 1, pMMR): ctDNA dropped from 3,749 ppm to undetectable post-salvage chemoradiotherapy, matching complete metabolic response on PET/CT.

Case 3: Gastric signet-ring cell carcinoma with peritoneal metastases: After six cycles of zolbetuximab and platinum-based regimen, ctDNA cleared from 9,269 ppm, and CA 19-9 fell from 55.7 to 21.6 U/mL, matching near-complete pathological response at gastrectomy.

Conclusion: In routine practice, whole-genome-informed whole-genome cfDNA tracking detected disease across diverse solid tumors, reflecting therapeutic response and surgical clearance to inform adjuvant de-escalation and residual disease evaluation. Sensitivity varied with the trackable variant count, which should inform interpretation of negative results.

P13 MACROCARPAL I INDUCES IMMUNOGENIC CELL DEATH AND SYNERGIZES WITH IMMUNE CHECKPOINT INHIBITION BY TARGETING TUBULIN AND PARP1 IN COLORECTAL CANCER

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Colorectal cancer (CRC) presents an obstacle to immunotherapy, primarily because most cases are microsatellite stable (MSS) tumors, which are often described as"cold tumors" with limited immunogenicity. Recent studies have indicated that several therapeutic approaches, such as chemotherapy and targeted therapies, can elicit immunogenic cell death (ICD) and stimulate immune responses. However, challenges such as target affinity and in vivo pharmacokinetics limit the efficacy and immune response of current targeted therapies. In this study, we demonstrate that Macrocarpal I is a potent inducer of ICD by activating the PERK/eIF2A/ATF4/CHOP signaling pathway. Furthermore, Macrocarpal I induces apoptosis and ferroptosis, both of which act as triggers for ICD. Mechanistically, Macrocarpal I directly targets TUBB2B and PARP1, disrupting microtubule polymerization and DNA repair processes. Importantly, treatment with Macrocarpal I enhances the anti-tumor immune response and augments responsiveness to anti-PD-1 therapy in an MC38 syngeneic mouse model of CRC.

P14 ONCOGENIC KRAS DRIVES IMMUNOSUPPRESSION OF COLORECTAL CANCER BY IMPAIRING DDX60-MEDIATED DSRNA ACCUMULATION AND VIRAL MIMICRY

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Immune checkpoint inhibitors (ICIs) have transformed the treatment of microsatellite instability-high (MSI-H) or mismatch repair-deficient (dMMR) colorectal cancer (CRC), yet most patients with microsatellite-stable (MSS) or mismatch repair-proficient (pMMR) tumours derive limited benefit, and acquired resistance also occurs in initially responsive patients. Impaired interferon (IFN) responses represent a major barrier to effective antitumour immunity and ICI therapy. Emerging evidence indicates that endogenous double-stranded RNAs (dsRNAs) can activate RIG-I-like receptors (RLRs), including RIG-I and MDA5, triggering a "viral mimicry" response that promotes IFN signalling, tumour immunogenicity and antitumour T-cell responses. However, how oncogenic signalling regulates endogenous dsRNA homeostasis and thereby restrains viral mimicry in CRC remains poorly understood. Here, we identify oncogenic KRAS as a critical regulator of dsRNA homeostasis through suppression of the RNA helicase DDX60. Oncogenic KRAS promotes the degradation of endogenous dsRNAs, thereby limiting dsRNA sensing and suppressing IFN responses. Conversely, ablation of oncogenic KRAS induces dsRNA accumulation and activates RIG-I/MDA5-dependent viral mimicry. Mechanistically, DDX60 binds endogenous dsRNAs and protects them from RNA-induced silencing complex (RISC)-mediated degradation, thereby maintaining an immunostimulatory dsRNA pool available for innate immune sensing. Restoration of DDX60 expression promotes dsRNA accumulation, reactivates IFN signalling, enhances antitumour immunity and increases CRC sensitivity to ICI therapy. We further demonstrate that oncogenic KRAS suppresses DDX60 through the AKT-GSK3β-STAT3 signalling cascade by inhibiting STAT3 phosphorylation and consequently reducing STAT3-mediated transcription of DDX60. Together, our findings uncover a previously unrecognized mechanism by which oncogenic KRAS suppresses tumour-intrinsic antiviral immunity by disrupting endogenous dsRNA homeostasis. The KRAS-DDX60-dsRNA axis provides a molecular link between oncogenic signalling, viral mimicry and immune evasion, and suggests that restoring dsRNA-mediated innate immune sensing may convert immunologically "cold" CRCs into more immune-responsive tumours and improve the efficacy of ICI therapy, particularly in KRAS-mutant CRC.

P15 HBSAG SEROSTATUS MODULATES ANTI-PD-1 IMMUNOTHERAPY EFFICACY IN NASOPHARYNGEAL CARCINOMA: CLINICAL OUTCOMES AND TREG-MEDIATED IMMUNE MICROENVIRONMENT REMODELING

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Background: Hepatitis B surface antigen (HBsAg) serostatus has been implicated in modulating treatment outcomes in nasopharyngeal carcinoma (NPC), yet its impact on anti-PD-1 immunotherapy efficacy and the underlying immune mechanisms remain poorly understood.

Methods: A total of 1,419 patients with NPC were enrolled, stratified by HBsAg status and treatment modality (CCRT alone, IC+CCRT, or aPD-1+IC+CCRT). Progression-free survival (PFS), and objective response rate (ORR) were compared. Multivariable Cox and logistic regression analyses were performed to identify independent predictors and the HBsAg–ICI interaction. Single-cell RNA sequencing and multiplex immunohistochemistry were employed to characterize tumor immune microenvironment differences between HBV-positive and HBV-negative tumors.

Results: HBsAg serostatus did not independently predict PFS but significantly modified the therapeutic benefit of anti-PD-1 immunotherapy. HBsAg-positive patients derived a markedly greater PFS benefit from aPD-1+IC+CCRT than HBsAg-negative patients (absolute gain: 26.4 vs. 14.9 percentage points) and achieved a significantly higher ORR under anti-PD-1 treatment (97.5% vs. 85.3%; P = 0.030). A significant HBsAg–ICI interaction was confirmed by multivariable logistic regression (sOR = 10.36, 95% CI: 1.85–195.96, P = 0.030). Mechanistically, single-cell analysis revealed a massive, selective expansion of terminal effector-like FOXP3 Tregs in HBV-positive tumors, accompanied by diminished CD8 T-cell differentiation toward effector/exhausted states, indicating that HBsAg seropositivity is associated with a Treg-dominant, CD8 effector-low immune microenvironment that may be particularly susceptible to immune checkpoint blockade.

Conclusions: HBsAg serostatus is a critical modifier of anti-PD-1 immunotherapy efficacy in NPC. HBsAg-positive patients, despite exhibiting a less proliferative tumor phenotype and a more immunosuppressive microenvironment, derive a pronounced survival advantage from ICI-based regimens. These findings support HBsAg serostatus as a clinically actionable biomarker for immunotherapeutic stratification in NPC.

P16 INTERACTING BACTERIAL COMMUNITIES DEFINE NASOPHARYNGEAL MICROBIOME ECOTYPES WITH DISTINCT FUNCTIONAL TRAITS

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Background: The nasopharynx represents a critical ecological niche in the upper respiratory tract, harboring a complex microbial community intricately linked to nasopharyngeal carcinoma (NPC). However, a comprehensive framework defining the structural and functional organization of the nasopharyngeal microbiota across populations remains to be established.

Methods: To systematically characterize this community framework, we integrated nasopharyngeal swab microbiome profiles from our prospective cohort with a large-scale public 16S rRNA gene sequencing dataset. Utilizing non-negative matrix factorization (NMF), we identified six distinct nasopharyngeal microbiome ecotypes that define the major nasopharyngeal microbial structures.

Results: These ecotypes successfully captured discrete patterns of microbial composition and taxonomic co-occurrence. Notably, an oral-like ecotype primarily enriched with Streptococcus and Prevotella was prominently enriched in NPC samples. This specific ecotype exhibited profound alterations in its microbial network, displaying significantly tighter co-occurrence patterns and enhanced microbial cross-feeding, which indicates a collaborative and interdependent microbial consortium. Furthermore, metatranscriptomic profiling of representative samples was conducted to elucidate the functional activities modulating these distinct ecotypes.

Conclusions: Collectively, our findings establish a robust classification of nasopharyngeal microbiome ecotypes, providing a clinically relevant framework for deciphering how microbial structural organization drives NPC pathogenesis and progression.

P17 SINGLE-CELL SPATIAL TRANSCRIPTOMICS UNCOVER THE FORMATION OF INTRATUMORAL BACTERIA-DRIVEN IMMUNOSUPPRESSIVE NICHE THROUGH TUMOR-MACROPHAGE CROSSTALK

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Background: The important role of intratumoral bacteria in the initiation and progression of nasopharyngeal carcinoma (NPC) has been gradually recognized. However, the spatial colonization patterns of intratumoral bacteria and their complex interactions with cellular components of the tumor microenvironment (TME) remain poorly understood.

Methods: Single-cell spatial transcriptomics with customized 16S rRNA probes on NPC tissues were analyzed to reveal the intricate interactions between intratumoral bacteria and TME cells. Based on the bioinformatic analysis results, in vitro and in vivo experiments were performed to investigate the underlying molecular mechanisms.

Results: Spatial analysis reveals a unique colonization pattern of intratumoral bacteria and orchestrate the formation of an immunosuppressive spatial niche. Mechanistically, intratumoral bacteria remodel tumor cells into a distinct functional state termed as bacteria-related malignant epithelium (BRME), which subsequently promotes the regional accumulation of immunosuppressive macrophages and leads to the exhaustion of CD8+ T cells. Clinically, the distinct colonization pattern of intratumoral bacteria and increased abundance of this immunosuppressive niche are significantly associated with inferior clinical outcomes of patients with NPC.

Conclusions: Our study reveals a unique spatial colonization pattern of intratumoral bacteria and uncovers a bacteria-mediated mechanism that drives the formation of an immunosuppressive tumor niche. These findings highlight the active role of intratumoral bacteria in the remodulation of TME and provides potential therapeutic targets for future antitumor interventions.

P18 Validation of the Chinese Version of the MDASI-Immunotherapy in a Multicenter, Multicancer Cohort

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Objective: The M.D. Anderson Symptom Inventory-Immunotherapy for Early-Phase Trials (MDASI-Immunotherapy EPT) was developed to assess symptom severity in cancer patients receiving immunotherapy. However, the original module did not fully capture the range of symptoms reported by patients. This study aimed to revise and validate the Chinese version of the MDASI-Immunotherapy in a multicenter, multicancer cohort.

Methods: A two-phase sequential mixed-methods design was employed. In Phase I, a systematic literature review, semi-structured interviews with patients (n=20) and clinicians (n=10), a two-round Delphi expert consensus (n=11), and cognitive interviews (n=10) were conducted to identify and refine immunotherapy-specific symptom items. In Phase II, Study 2.1 (n=396) performed item analysis to reduce the preliminary 43-item scale. Study 2.2 (n=418) evaluated the psychometric properties of the final scale, including reliability, construct validity (exploratory factor analysis), criterion validity (Spearman correlations with FACT-G), and known-groups validity (Mann-Whitney U test by ECOG status). Subgroup analyses were conducted by residency status (urban vs. rural) and cancer type.

Results: The final 34-item module comprised 13 core symptoms, 15 immunotherapy-specific items, and 6 interference items. Exploratory factor analysis identified a 6-factor structure (skin, gastrointestinal, cardiac, hepatobiliary, extremity edema/musculoskeletal, and pain/fever dimensions) explaining 77.59% of the total variance. Internal consistency was excellent (Cronbach's α: core subscale 0.917; immunotherapy module 0.878; interference subscale 0.910). Criterion validity was supported by significant correlations with FACT-G physical well-being ($\rho = 0.245–0.674$, $p < 0.01$). Subgroup analyses showed consistent reliability across urban and rural populations and across major cancer types, though rural patients reported significantly higher symptom burden ($p < 0.05$). The newly added item "skin dryness" was endorsed at similar rates across all cancer types (38–41%).

Conclusions: The revised Chinese MDASI-Immunotherapy is a valid, reliable, and comprehensive tool for assessing symptom burden in Chinese cancer patients receiving immunotherapy. Its 6-factor structure aligns with clinically recognized organ-system clusters of immune-related adverse events, supporting its utility for targeted symptom monitoring and management in multi-cancer clinical practice and research.

P19 IMMUNE-VASCULAR DUALITY IN DESCENDING NASOPHARYNGEAL CARCINOMA DELINEATES IMMUNOTHERAPY-RESPONSIVE AND METASTATIC-RISK ECOSYSTEMS

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Background: Clinical benefit from immune checkpoint inhibitors (ICIs) in nasopharyngeal carcinoma (NPC) remains heterogeneous, highlighting the need for biomarkers that reflect the tumor microenvironment. Ascending and descending NPC represent clinically identifiable patterns of disease progression with distinct prognostic and therapeutic characteristics, raising the possibility that they are underpinned by different cellular and spatial ecosystems. However, the microenvironmental basis of these clinical subtypes and its relationship to prognosis and immunotherapy response remain poorly understood. We therefore aimed to identify and validate subtype-associated cellular and spatial biomarkers with potential prognostic and therapeutic relevance in NPC.

Methods: We integrated eight single-cell RNA-sequencing and four spatial transcriptomic datasets, comprising approximately 1.01 million cells from 150 samples and 87,482 spatial spots from 42 tissue sections. Cell-state annotation, bulk and spatial deconvolution, multicellular-hub analysis, ligand-receptor inference, and trajectory analysis were performed. Clinical associations were evaluated using sample-level group comparisons, survival analyses, and Cox proportional hazards models. Key findings were assessed in independent transcriptomic, immunotherapy-treated, and single-cell spatial cohorts.

Results: Descending NPC exhibited two divergent biomarker programs. An immune-active program comprising $CD5^+$ plasmacytoid dendritic cells (pDCs), progenitor exhausted T cells (Tpex), and tertiary lymphoid structure (TLS)-like niches was enriched in descending NPC. $CD5^+$ pDC abundance was associated with increased T-cell infiltration, favorable survival, and response to ICI plus chemotherapy, while its transcriptional signature was enriched in responders across independent melanoma immunotherapy cohorts. Spatial analyses further linked $CD5^+$ pDCs to TLS-like regions and Tpex cells through predicted co-stimulatory interactions, with these relationships reproduced in external pan-cancer spatial datasets. In parallel, a vascular program comprising $CXCL12^+$ fibroblasts and CXCR4-high $ESM1^+$ tip-like endothelial cells was enriched in tumors with advanced nodal involvement and associated with adverse outcomes, VEGF-active angiogenic programs, and spatial CXCL12-CXCR4 signaling.

Conclusions: Descending NPC harbors distinct immune and vascular biomarker programs. The $CD5^+$ pDC-Tpex-TLS axis represents a candidate response-associated biomarker linked to favorable prognosis, whereas the $CXCL12^+$ fibroblast-$ESM1^+$ tip-like endothelial program marks adverse vascular biology. These findings provide a framework for prospective biomarker-guided stratification and evaluation of combined PD-1 and anti-angiogenic therapy.

P20 SPATIAL STRUCTURAL REMODELING DRIVES IMMUNE CHECKPOINT INHIBITOR RESISTANCE IN GASTRIC CANCER

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Background: Immune checkpoint inhibitors (ICIs) have improved outcomes in a subset of gastric cancer (GC) patients, yet primary and acquired resistance remain major challenges across molecular subtypes. Although genomic biomarkers have been extensively investigated, the contribution of tumor spatial organization and its dynamic remodeling during ICI treatment remains poorly understood. We aimed to define spatial structural determinants of ICI resistance beyond tumor genomic features.

Methods: We performed multiplex spatial transcriptomics and proteomics on paired pre- and post-treatment tumor specimens from 50 GC patients, including 20 ICI-sensitive and 30 ICI-resistant cases. Spatial ecological niches, cellular states, immune-tumor interactions, and tissue architectural features were systematically characterized and compared longitudinally.

Results: ICI-responsive tumors displayed relatively ordered and compact growth patterns accompanied by effective immune-cell infiltration and tumor-immune spatial interactions. In contrast, resistant tumors exhibited disorganized and invasive architectures with prominent immune exclusion. Across resistant tumors, we identified a conserved resistance-associated spatial niche characterized by hypoxic-necrotic regions enriched in neutrophils, accompanied by neutrophil extracellular traps (NETs) and inflammatory signaling that promoted stromal remodeling and the formation of physical barriers restricting immune-cell penetration. Longitudinal analysis further showed sustained immune infiltration and preserved tissue organization in responders, whereas non-responders developed reduced immune infiltration and progressive stromal barrier formation following ICI therapy.

Conclusions: Our findings identify spatial architectural disorder as a key determinant of ICI resistance in GC, extending the current focus beyond genomic biomarkers. A hypoxia-necrosis-neutrophil axis and associated stromal remodeling constitute a convergent mechanism of immune exclusion. Quantitative spatial organizational features may provide broadly applicable biomarkers for ICI response across GC subtypes, while targeting these physical and cellular barriers may offer a potential strategy to overcome ICI resistance.

P21 RUBICON RESTRAINS ARGINASE-1 EXPRESSION IN TUMOR-ASSOCIATED MACROPHAGES TO PROMOTE ANTITUMOR IMMUNITY

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Background: Tumor-associated macrophages (TAMs) contribute to immune evasion through metabolic and functional reprogramming, but the mechanisms linking macrophage metabolism to immunosuppression remain incompletely understood. RUBCN (RUN domain Beclin-1 interacting and cysteine-rich containing protein) is a regulator of autophagy and cellular homeostasis; however, its role in TAM-mediated immunosuppression remains poorly understood.

Methods: RUBCN expression and macrophage-associated immune signatures were analyzed using transcriptomic and single-cell RNA sequencing datasets. The effects of RUBCN deficiency on tumor growth, macrophage function, cellular metabolism, and $CD8^+$ T-cell activity were investigated using syngeneic tumor models, bone marrow-derived macrophages (BMDMs), Seahorse metabolic profiling, flow cytometry, RNA sequencing, and macrophage–$CD8^+$ T-cell co-culture assays. The molecular regulation of arginase-1 (Arg1) by RUBCN was examined using co-immunoprecipitation, protein stability assays, and proteasome inhibition.

Results: TAM RUBCN expression was associated with a more favorable immune microenvironment and antitumor immunity. Genetic deletion of Rubcn promoted tumor growth, increased immunosuppressive macrophage accumulation, and reduced $CD8^+$ T-cell infiltration. RUBCN deficiency was accompanied by enhanced oxidative phosphorylation (OXPHOS), increased Arg1 protein abundance, and greater macrophage-mediated suppression of $CD8^+$ T-cell responses. Mechanistically, RUBCN interacted with Arg1 and promoted its proteasome-dependent degradation, thereby limiting Arg1 accumulation in macrophages. Rubcn-deficient BMDMs suppressed $CD8^+$ T-cell proliferation and reduced effector molecule production, including interferon-γ and granzyme B. Restoration of L-arginine availability partially rescued $CD8^+$T-cell proliferation and effector function, supporting a functional role for the RUBCN–Arg1 axis in macrophage-mediated immunosuppression.

Conclusions: Our findings identify RUBCN as a regulator of TAM immunosuppressive function linking macrophage metabolic state to immune regulation. RUBCN promotes Arg1 protein turnover and limits macrophage-mediated L-arginine depletion, thereby preserving $CD8^+$ T-cell effector function. Modulation of the RUBCN–Arg1 axis may represent a potential strategy for reprogramming immunosuppressive TAMs and enhancing antitumor immunity.

P22 IRON SUPPLEMENTATION REVERSES IMMUNOTHERAPY RESISTANCE IN PMMR COLORECTAL CANCER VIA CLEC9A^{+} CDC1 ACTIVATION

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Approximately 85% of colorectal cancer (CRC) patients harbor proficient mismatch repair (pMMR) tumors, rendering them inherently refractory to immunotherapy. These patients also frequently exhibit concurrent iron deficiency or iron deficiency anemia, making the reversal of primary immunotherapy resistance an unmet clinical need. Based on a clinical cohort of 717 CRC patients from Sun Yat-sen University Cancer Center and analysis of the NHANES database, we found that iron deficiency was significantly associated with poor prognosis, while iron supplementation was linked to reduced tumor-related mortality. In mouse models, iron supplementation significantly suppressed tumor growth, enhanced intratumoral CD8^{+} T cell function, and effectively reversed primary resistance to PD-L1 blockade in pMMR colorectal cancer. Single-cell RNA sequencing revealed a marked enrichment of CLEC9A^{+} cDC1 cells within tumors upon iron treatment. Notably, this antitumor effect was virtually abolished in Batf3$^{-/-}$ knockout mice or upon CLEC9A blockade, indicating that iron exerts its antitumor immune effects by enhancing CLEC9A^{+} cDC1 function.Collectively, This study reveals a novel mechanistic role of iron metabolism in overcoming immunotherapy resistance in pMMR CRC through the regulation of CLEC9A^{+} cDC1 function. Our findings offer a new theoretical foundation for the clinical development of iron supplementation as a simple and cost-effective strategy to potentiate immunotherapy.

P23 MULTI-OMICS ANALYSIS OF GPR4-MEDIATED INVASION MECHANISMS AND TARGETED STRATEGY EXPLORATION IN GROWTH HORMONE–SECRETING PITUITARY ADENOMA

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Background: The invasive growth of growth hormone-secreting pituitary adenoma (GHPA) is a primary driver of poor patient outcomes. The high heterogeneity of tumor cells and the tumor microenvironment promotes invasive growth and limits the efficacy of current therapies. Identifying invasion-associated cellular subpopulations and elucidating the molecular mechanisms of invasion are imperative for developing novel targeted strategies.

Objective: To deconstruct the cellular heterogeneity of invasive GHPA, identify invasion-associated subpopulations and their specific markers, elucidate how these markers regulate tumor invasion, and explore novel targeted therapeutic strategies.

Methods: A comprehensive multi-omics approach integrating single-cell and bulk RNA sequencing, spatial transcriptomics, and proteomics, complemented by interpretable machine learning and multivariable regression, was used to identify and validate biomarkers in clinical cohorts of invasive GHPA. Molecular mechanisms were explored through experiments on cell lines and animal models, including GTP pull-down, Western blot, and immunofluorescence, and a tumor cell-macrophage co-culture system to examine immune microenvironment regulation. AI-based structural prediction, high-throughput virtual screening, molecular dynamics simulations, and cell viability assays were integrated for computational drug discovery and target validation.

Results: scRNA-seq analysis revealed for the first time that GHPA tumor cells exhibit a differentiation trajectory from progenitor-like to mature states, with the G-protein-coupled receptor GPR4 identified as a specific marker of the mature, invasion-prone subpopulation. Multi-omics analyses of clinical specimens confirmed that GPR4 expression correlates strongly with clinical invasiveness and serves as an independent risk factor. Mechanistically, GPR4 overexpression promoted GHPA proliferation and migration via activation of CDC42, upregulating downstream effector INF2 and mediating F-actin stress fiber remodeling and epithelial-mesenchymal transition. GPR4 tumor cells recruited tumor-associated macrophages via paracrine CCL7 and induced their osteoclast differentiation through direct cell-to-cell contact. Lead compounds with novel chemical scaffolds targeting GPR4 were identified and confirmed to inhibit GHPA proliferation in a GPR4-dependent manner in vitro.

Conclusions: Invasive GHPAs undergo recapitulation of the pituitary developmental trajectory. GPR4 is a mature tumor cell–specific marker and independent risk factor for clinical invasiveness, enhancing invasion via CDC42-INF2 axis-mediated cytoskeletal remodeling. GPR4 cells secrete CCL7 to promote macrophage-mediated bone invasion. Validated lead compounds targeting GPR4 provide a theoretical foundation and experimental evidence for targeted therapy of invasive GHPA.

Keywords: GPR4; Growth Hormone-Secreting Pituitary Adenoma; Tumor Invasiveness; CDC42-INF2 axis; Tumor Immune Microenvironment; CCL7; CADD

P24 E3 UBIQUITIN LIGASE RNF150 REPROGRAMS GLYCOLYTIC METABOLISM TO SUPPRESS ESOPHAGEAL AND NASOPHARYNGEAL CARCINOMAS VIA PROMOTING UBIQUITINATION OF PFKP AND PFKM

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Background: Metabolic reprogramming supports tumor progression, but the mechanisms linking epigenetic silencing of tumor suppressors to altered metabolism remain unclear. Moreover, the function and underlying mechanisms of RING finger protein 150 (RNF150) as a tumor-suppressive E3 ligase that regulates metabolic reprogramming in esophageal squamous cell carcinoma (ESCC) and nasopharyngeal carcinoma (NPC) remain unclear.

Methods: RNF150 methylation and expression were assessed in tumor cell lines, primary tissues, and public datasets. RNF150 function was examined using CRISPR/Cas9 knockout, ectopic expression, cell-based assays, and xenograft models. Protein interactions, ubiquitination, and stability were analyzed by proteomics, co-immunoprecipitation, cycloheximide chase, and proteasome inhibition. Glycolysis and global metabolic changes were evaluated by glucose uptake, lactate and ATP assays, and untargeted metabolomics.

Results: Methylomic analysis identified RNF150 promoter methylation in ESCC and HNSC tumors compared with adjacent normal tissues, which was associated with reduced expression and poorer survival in ESCC. RNF150 loss increased tumor cell proliferation, migration, and invasion and significantly enhanced tumorigenicity in vivo, whereas RNF150 restoration suppressed tumor growth in vivo. RNF150 localized predominantly to the endoplasmic reticulum/cytoplasm and suppressed glucose uptake, lactate production, and extracellular ATP, thereby attenuating the Warburg effect. Mechanistically, RNF150 bound the PFK1 isoforms PFKP and PFKM, but not PFKL, and promoted their RING-domain-dependent polyubiquitination and proteasomal degradation. RNF150 deficiency stabilized PFKP and PFKM, enhanced glycolytic flux and induced broad metabolic alterations involving methionine and S-adenosylmethionine, purine and oxidative phosphorylation pathways. Re-expression of PFKP or PFKM rescued the anti-proliferative and anti-migratory effects of RNF150 in tumor cells. Conclusions: RNF150 is an epigenetically silenced E3 ubiquitin ligase that functions as a tumor suppressor by targeting PFKP and PFKM for ubiquitin-mediated degradation, thereby restraining oncogenic glycolytic reprogramming. RNF150 promoter methylation may serve as a candidate biomarker for ESCC and NPC, and the RNF150–PFKP/PFKM axis represents a potential therapeutic vulnerability.

Keywords: RNF150, CpG methylation, metabolism, glycolysis, ubiquitination, PFKP, PFKM

P25 TRANSLATING EPIGENETIC SMOKING BIOMARKERS INTO NURSE-LED PRACTICE

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Background: DNA methylation biomarkers of smoking exposure — notably AHRR hypomethylation and epigenetic clocks such as GrimAge — predict smoking-related morbidity and mortality and, unlike static genetic-risk markers, respond dynamically to cessation. This evidence base remains confined largely to genetics/epidemiology literature, with no synthesis assessing its readiness for nurse-led cessation practice.

Objective: To map the predictive validity, causal basis, and individual-level reliability of DNA methylation smoking biomarkers, contextualize this evidence against established biomarker-feedback trials, and identify gaps relevant to nurse-delivered cessation counseling.

Methods: Following JBI methodology and PRISMA-ScR standards, this scoping review evaluates records from PubMed, CINAHL, Scopus, and Web of Science (2000–2025). Two reviewers are screening records and extracting data via a piloted charting form.

Results: Preliminary findings indicate: (1) strong population-level predictive associations between AHRR hypomethylation and COPD/lung cancer risk (HR 4.6–4.9; pooled AUC 0.79–0.81); (2) mixed evidence for a causal role, with inconsistent findings across Mendelian randomization, twin, and population-based designs; (3) limited individual-level reliability (ICC 0.4–0.7 across most epigenetic clocks) despite high technical reproducibility; and (4) confirmed cessation-linked methylation recovery (mean 5.9% increase over 6 months among abstainers). However, this recovery is slower and more heterogeneous than other cessation biomarkers, with up to 18% of confirmed quitters showing no detectable change. Furthermore, while a 2019 Cochrane review found that neither genetic risk feedback nor pooled harm/imaging feedback significantly improved cessation, subsequent individual spirometry-based "lung age" trials (ESPIROTAB, RESET) reported significant benefits (OR 1.42–2.8). To date, no nurse-delivered epigenetic feedback trials have been conducted.

Conclusions: Methylation biomarkers are currently useful as population-level tools rather than individual diagnostic tests. Because they change in response to quitting, they align better with newer harm-visualization approaches than with static genetic feedback, offering a mechanistically justified direction for nurse-led precision cessation counseling.

P26 MULTI-OMIC BIOMARKERS OF SMOKING EXPOSURE IN LUNG CANCER: AN INTERPRETABLE MACHINE LEARNING APPROACH

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School of Nursing, St. Teresa's Hospital, Hong Kong SAR, China

Introduction: Tobacco smoke exposure induces measurable, persistent alterations in tumor DNA methylation, gene expression, and mutational burden, yet tumor mutational burden alone poorly discriminates smoking-exposure status. Few studies have integrated tumor-level methylation, transcriptomic, and mutational data within an interpretable machine learning framework to identify which biomarkers best track smoking exposure in lung cancer independent of histologic subtype. This project addresses that gap.

Methodology: Using The Cancer Genome Atlas, we will assemble a cohort of approximately 500 lung adenocarcinoma and squamous cell carcinoma patients with complete DNA methylation, RNA-sequencing, mutation, and smoking-history data. Differentially methylated positions and differentially expressed genes associated with smoking status will be identified using limma and DESeq2, then combined with TMB into a unified feature set. A gradient-boosted tree classifier will be trained to predict smoking status using patient-level cross-validation to prevent data leakage. SHAP (SHapley Additive exPlanations) values will be computed to rank feature contributions and ensure clinical interpretability.

Findings: Analysis is anticipated to show integrated multi-omic features outperform TMB alone in distinguishing smoking-exposure status. Furthermore, SHAP rankings are expected to highlight known smoking-associated markers, such as AHRR methylation, alongside transcriptomic signatures—providing an interpretable, transparent basis for classification rather than a "black-box" prediction.

Conclusion and recommendations: The proposed model is expected to deliver strong discriminative performance, establishing a framework for using tumor-derived multi-omic signatures as objective, tissue-based corroboration of self-reported smoking history. This will support nurses and interdisciplinary oncology teams in risk stratification and survivorship counseling. Future work should validate this signature in prospectively collected, non-TCGA cohorts. Ultimately, multi-omic corroboration should complement, rather than replace, patient-reported exposure history.

P27 GENOMIC-INFORMED SMOKING CESSATION COUNSELING TO ADVANCE PRECISION CANCER PREVENTION

Ka Kei Alison CHU
School of Nursing, St. Teresa's Hospital, Hong Kong SAR, China

Background: Tobacco use remains the leading preventable cause of cancer. Emerging evidence demonstrates that genomic markers—particularly CHRNA5 and related nicotinic receptor variants, as well as polygenic risk scores (PRS)—can inform smoking behavior, cessation outcomes, and lung cancer risk. Translating these insights into clinical practice requires a rigorous evaluation of their analytic validity, clinical utility, and implementation pathways within learning health systems.

Methods: This integrative literature review synthesizes empirical studies, randomized controlled trials, and translational frameworks published between 2006 and 2026. Sources include genomic association studies, pharmacogenomic trials, behavioral counseling interventions, and public health genomics analyses. Evidence is evaluated across the domains of risk stratification, counseling design, pharmacogenomic treatment pathways, and ethical, legal, and social implications (ELSI).

Results: Genomic predictors such as CHRNA5 and CYP2A6 variants consistently associate with smoking heaviness, relapse risk, and treatment response, while PRS enhance predictive accuracy for cessation success and lung cancer risk beyond standard clinical factors. Although patient receptivity to genetic risk feedback is high, its direct impact on quit rates remains variable. Standalone genomic disclosure shows limited efficacy; however, integrated counseling models that combine risk communication, pharmacotherapy, and behavioral support show great promise. Current evidence supports the readiness of these tools for risk stratification and targeted cessation intensification, whereas pharmacotherapy personalization requires further validation. Key ethical concerns include privacy, consent, disparities in genomic literacy, and the risk of genetic determinism.

Conclusions: Genomic-informed counseling offers a promising pathway for precision cancer prevention by tailoring cessation support and screening strategies to individual genetic risk profiles. Integrating PRS and pharmacogenomic insights into multi-component interventions can optimize quit attempts, treatment adherence, and screening uptake. Realizing this potential will require harmonized trial designs, robust evaluations of clinical utility, and equitable implementation across diverse populations.

P28 CO-ALTERATION ARCHITECTURE AND MOLECULAR FEATURES OF ERBB2-MUTANT NON-SMALL CELL LUNG CANCER

Liuzhen Meng, Xiaoxue Chai, Yongheng Chen, Yuan Li, Tony S K Mok, Herbert Loong, Lili Li

Department of Clinical Oncology, State Key Laboratory of Translational Oncology, Sir YK Pao Center for Cancer, The Chinese University of Hong Kong, Shatin, Hong Kong SAR, China

Background: ERBB2 (HER2) mutations occur in approximately 2%-4% of non-small cell lung cancer (NSCLC) and define a distinct subset with limited targeted therapy options. ERBB2-mutant NSCLC mutational landscapes are known, but clonal architecture, VAF dynamics, and multi-omics correlates of co-altered drivers remain unclear.

Methods: We integrated multi-omics data from 16 published datasets encompassing 9,989 NSCLC samples. ERBB2 mutations were annotated for oncogenicity, co-alteration patterns, VAF trajectories, mutational signatures, and survival associations.

Results: ERBB2 mutations were identified in 351 patients (4.1%). Co-altered tumors exhibited partner-specific patterns enriched for KEAP1, STK11, and TP53 alterations relative to ERBB2-only tumors. In tumors co-altered with EGFR, KRAS, ALK, MET, or BRAF, ERBB2 VAF was significantly lower than in ERBB2-only cases for four of five partners, with reciprocal partner VAF suppression. Within co-altered tumors, ERBB2 and partner VAFs were indistinguishable (all $p>0.23$), consistent with parallel rather than hierarchical clonal evolution. ERBB2 activating mutations were enriched in the high-VAF quartile, whereas other ERBB2 alterations were enriched in the low quartile (both $p=0.041$). Among 330 samples with copy-number data, focal ERBB2 amplification (6.4%) was associated with higher VAF ($p=0.0461$), kinase-domain/inframe insertions mutation ($p=0.03$), and fewer ECD/missense alterations (both $p\leq0.02$). ERBB2 expression was elevated in mutants and further increased in activating-mutant cases ($p=0.0045$). Overall survival did not differ by ERBB2 status, co-alteration, or mutation subtype, although high expression and activating mutations trended toward favorable hazard ratios, suggesting that activating mutations, high VAF, amplification, and elevated expression may define a therapeutically sensitive subset.

Conclusions: ERBB2 status alone lacks a distinct survival signal, likely reflecting contemporary therapies, but activating mutations, high VAF, and elevated expression suggest therapeutic sensitivity. Reciprocal VAF suppression supports parallel clonal evolution across multiple co-altered driver genes. Patient selection beyond mutation presence, incorporating VAF, copy-number, and expression, is needed to refine ERBB2-directed strategies

About CTONG and CTONG Young

The Chinese Thoracic Oncology Group (CTONG) was founded in 2007, led by Professor Yi-Long Wu, an oncology expert of Guangdong Provincial People's Hospital. It aims to design and develop multi-center clinical trials in the field of chest tumor, especially for lung cancer, provide a high level of evidence for clinical practice of thoracic tumor, promote standardization, modernization and internationalization of clinical and research work in thoracic tumor area, and enhancing the diagnosis, treatment standards, and international status of thoracic oncology in the country.

Professor Yi-Long Wu serves as Chair of the expert committee; Professors Li Zhang, Cai-Cun Zhou, and Shun Lu serve as Vice-Chairs, and Professor Qing Zhou acts as Secretary.

CTONG has launched a series of domestic multicenter clinical trials. Its research findings have been frequently presented at major international academic conferences including ASCO, ESMO and WCLC, and published in prestigious journals such as The New England Journal of Medicine, Journal of Clinical Oncology and The Lancet Oncology.

CTONG YOUNG is a young talent development platform for lung cancer professionals established by Professor Yi-Long Wu, Chair of the Chinese Thoracic Oncology Group (CTONG), and his team. It was officially founded on October 27, 2023.

As Professor Yi-Long Wu noted, young scholars within the field have needs for self-development, collaborative research among peers, and multicenter cooperation. However, young physicians are often limited in influence and access to resources. CTONG YOUNG was therefore founded to foster exchanges and collaboration among members and strengthen organizational cohesion. Primarily targeting young physicians from diverse medical centers, CTONG YOUNG serves as a platform for skill advancement, academic exposure, information sharing and networking. It encourages young physicians to grow through practice and generate new momentum to drive the development of the next generation of lung cancer specialists.

It is hoped that the official establishment of CTONG YOUNG will nurture a brand-new talent pipeline in lung cancer, and we anticipate accelerated progress in China's talent training model for lung cancer care and research.

IMMUNO – ONCOLOGY HONG KONG 2026 | LUNCH SYMPOSIUM

Shaping the Future of Long-Term Management in ALK+ aNSCLC:

Turning Durable Evidence Into Everyday Success

7 November 2026 (Saturday) | 13:20 – 14:20 | Grand Ballroom 1 – 3



Chairperson and Speaker

PROF TONY MOK

Chairman, Department of Clinical Oncology, Li Shu Fan Medical Foundation
Professor of Clinical Oncology, The Chinese University of Hong Kong



Speaker

PROF VICTOR LEE

Clinical Professor, Department of Clinical Oncology, The University of Hong Kong



Speaker

DR KEN WONG

Specialist in Clinical Oncology, Honorary Clinical Assistant Professor, The Chinese University of Hong Kong, The University of Hong Kong

13:20 – 13:23	**Welcome and Chair's Introduction**	**Prof Tony Mok**
13:23 – 13:38	**Session 1:** ALK+ aNSCLC in 2026: How Should Long-Term Data Shape First-Line Decisions?	**Prof Tony Mok**
13:38 – 13:53	**Session 2:** Making It Work in Real Life: A Practical Guide to Managing ALK+ aNSCLC in Hong Kong	**Dr Ken Wong**
13:53 – 14:08	**Session 3:** After First Line: Resistance, Re-Biopsy and the Next Wave of ALK Treatments	**Prof Victor Lee**
14:08 – 14:20	**Panel Discussion and Q&A:** Your Toughest ALK Cases: Experts' Answer	**All Participants**

Abbreviations: ALK, anaplastic lymphoma kinase; aNSCLC, advanced non-small cell lung cancer.

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EVOLVING TREATMENT LANDSCAPE IN LUNG CANCER:

BREAKTHROUGH WITH ADCs

7 NOV 2026 | SATURDAY

13:20–14:20
HUNG HOM 1, KERRY HOTEL, HONG KONG

AGENDA

Time	Topic	Faculty
13:20–13:25	Welcome and introduction	Dr Herbert Loong
13:25–13:45	Transforming Precision Medicine with ADCs in NSCLC	Dr Julia Rotow
13:45–14:00	Advancing Targeted Therapeutics in ES-SCLC: The ADC Landscape	Dr Stephanie Saw
14:00–14:20	Panel discussion	Dr Herbert Loong, Dr Julia Rotow, Dr Stephanie Saw

ADC, antibody–drug conjugate; ES-SCLC, extensive-stage small-cell lung cancer; NSCLC, non–small-cell lung cancer.

MEET OUR FACULTY



CHAIRPERSON

Dr Herbert Loong

Clinical Associate Professor, Department of Clinical Oncology

Medical Director, Phase 1 Clinical Trials Centre, The Chinese University of Hong Kong



SPEAKER

Dr Julia Rotow

Clinical Director, Lowe Center for Thoracic Oncology, Dana-Farber Cancer Institute

Assistant Professor, Harvard Medical School, USA



SPEAKER

Dr Stephanie Saw

Consultant (Medical Oncology), National Cancer Centre Singapore

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Optimising the *EGFR*-Mutant NSCLC Treatment Journey:

EMERGING EVIDENCE, EVOLVING FIRST-LINE STRATEGIES, AND PRACTICAL INSIGHTS FROM JAPAN



7 November 2026 (Saturday)

15:45 – 16:45

Grand Ballroom 1-3,
Kerry Hotel, Hung Hom



Prof Hidetoshi Hayashi, *MD, PhD*

Professor, Department of Medical Oncology,
Kindai University,
Faculty of Medicine, Japan

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Advancing Outcomes in Resectable GC/GEJC:

Translating Scientific Breakthroughs in Immunotherapy Into Clinical Practice

Date	Time	Venue
November 8th (Sunday)	12:15 – 13:15	Kerry Hotel (Ballroom 1 – 3)

FACULTY

MODERATOR	SPEAKER	SPEAKER
Prof. Winnie Yeo	**Prof. Ian Chau**	**Prof. Shannon Chan**
Department of Clinical Oncology The Chinese University of Hong Kong	Consultant Medical Oncologist Gastrointestinal and Lymphoma Units Royal Marsden Hospital, UK	Department of Surgery The Chinese University of Hong Kong

AGENDA

TIME	AGENDA	SPEAKER
12:15 – 12:20	Welcome Speech	Prof. Winnie Yeo
12:20 – 12:40	Lecture 1: The Evolving Treatment Landscape and Practical Applications of Immunotherapy in the Perioperative Setting	Prof. Ian Chau
12:40 – 12:55	Lecture 2: Surgical Perspectives on Integrating Immunotherapy in the Perioperative Setting	Prof. Shannon Chan
12:55 – 13:15	Panel Discussion: Case-based Discussion and Q&A Prof. Ian Chau – Case Presentation Prof. Shannon Chan – Case Presentation Panel Q&A	All Faculty

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IMMUNO – ONCOLOGY HONG KONG 2026

The Shifting Paradigm of Immunotherapy Combination Therapy in Early-Stage RCC and MIBC

Sunday, 8 November 2026
12:15-13:15

Ballroom 4, Level 2, Kerry Hotel,
38 Hung Luen Road, Hung Hom Bay,
Kowloon, Hong Kong



Chairperson

Prof. Bryan Li

Clinical Assistant Professor,
Center of Cancer Medicine,
The University of Hong Kong



Speaker

Prof. Terence Friedlander

Robert and Virginia O'Reilly Family Professor of Medicine,
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IMMUNO-ONCOLOGY HONG KONG 2026

The Future Treatment Landscape of **Immunotherapy for Metastatic NSCLC:** New technologies, Bispecific Antibodies, and combinations with ADCs

12:15 - 13:15

8th Nov 2026, Sunday

Hung Hom Room 1, Kerry Hotel, Hong Kong



Chairman:

Prof. Tony MOK

Associate Dean
(Translation and Entrepreneurship)
Chairman, Department of Clinical Oncology
Li Shu Fan Professor of Clinical Oncology
The Chinese University of Hong Kong



Speaker:

Prof. Solange PETERS, MD-PhD

Chair Medical Oncology
Director Oncology Department – CHUV
Lausanne University Hospital



Agenda (Lunch Symposia)

Time	Topic	Chairman/Speaker
12:15-12:20	Opening	Prof. Tony Mok
12:20-13:05	The Future Treatment Landscape of Immunotherapy for Metastatic NSCLC: New technologies, Bispecific Antibodies, and combinations with ADCs	Prof. Solange Peters
13:05-13:15	Discussion	All

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‡Within 6 weeks from the last neoadjuvant treatment dose.[2,3]
§Within 90 days after surgery.[3]

AJCC=American Joint Committee on Cancer; I-O=immuno-oncology; NSCLC=non-small cell lung cancer.

chemo=chemotherapy; I-O=immuno-oncology; NCCN=National Comprehensive Cancer Network; NSCLC=non-small cell lung cancer; pCR=pathological complete response.

References: 1. OPDIVO® Hong Kong Prescribing Information. **2.** Forde PM, Spicer J, Lu S, et al. Neoadjuvant nivolumab plus chemotherapy in resectable lung cancer. *N Engl J Med*. 2022;386(21):1973-1985. **3.** Cascone T, Awad MM, Spicer JD, et al. Perioperative nivolumab in resectable lung cancer. *N Engl J Med*. 2024;390(19):1756-1769. **4.** Forde PM, Peters S, Donington J, et al. Perioperative vs neoadjuvant nivolumab for resectable NSCLC: patient-level data analysis of CheckMate 77T vs CheckMate 816. Oral presentation at WCLC 2024. Abstract PL02.08. **5.** Provencio M, Awad MM, Spicer JD, et al. Clinical outcomes with perioperative nivolumab by nodal status among patients with stage III resectable NSCLC: results from the phase 3 CheckMate 77T study. Oral presentation at ASCO 2024. Abstract LBA8007. **6.** Spicer J, Wang C, Tanaka F, et al. Surgical outcomes from the phase 3 CheckMate 816 trial: nivolumab + platinum-doublet chemotherapy vs chemotherapy alone as neoadjuvant treatment for patients with resectable non-small cell lung cancer. Oral presentation at ASCO 2021. Abstract 8503. **7.** Donington JK, Kuzdzal J, Tanaka F, et al. Surgical outcomes with perioperative nivolumab in patients with resectable NSCLC from the phase 3 CheckMate 77T study. Oral presentation at AATS-ITSOS 2024. **8.** Awad MM, Cascone T, Spicer JD, et al. Clinical outcomes with perioperative nivolumab in patients with resectable NSCLC from the phase 3 CheckMate 77T study. Oral presentation at ELCC 2024. Abstract LBA2. **9.** Spicer JD, Girard N, Provencio Pulla M, et al. Neoadjuvant nivolumab plus chemotherapy vs chemotherapy in patients with resectable NSCLC: 4-year update from CheckMate 816. Oral presentation at ASCO 2024. Abstract LBA8010. **10.** Provencio Pulla M, Awad MM, Cascone T, et al. Perioperative nivolumab vs placebo in patients with resectable NSCLC: clinical update from the phase 3 CheckMate 77T study. Oral presentation at ESMO 2024. Abstract LBA50. **11.** NCCN Guidelines – Non-Small Cell Lung Cancer. v7.2026

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FOLFIRI: Leucovorin calcium, fluorouracil and irinotecan hydrochloride regimen; FOLFOX4: Leucovorin calcium, fluorouracil, and oxaliplatin regimen; mCRC: Metastatic colorectal cancer; mFOLFOX6: Modified leucovorin calcium, fluorouracil, and oxaliplatin regimen; RAS: rat sarcoma virus.

References: 1. Vectibix® Hong Kong Prescribing Information, December 2021. **2.** *MVASI®* Hong Kong Prescribing Information, September 2023. **3.** Douillard JY, et al. Impact of early tumour shrinkage and resection on outcome in patients with wild-type RAS metastatic colorectal cancer. *Eur J Cancer.* 2015;51:1231-1242. **4.** Rivera F, et al. Final analysis of the randomised PEAK trial: overall survival and tumour responses during first-line treatment with mFOLFOX6 plus either panitumumab or bevacizumab in patients with metastatic colorectal carcinoma. *Int J Colorectal Dis.* 2017;32:1791190. **5.** Köhne CH, et al. Impact of primary tumour location and early tumour shrinkage on outcomes in patients with RAS wild-type metastatic colorectal cancer following first-line FOLFIRI plus panitumumab. *Drugs in R&D.* 2019;19:267-275. **6.** Thatcher N, et al. Efficacy and Safety of the Biosimilar ABP 215 Compared with Bevacizumab in Patients with Advanced Nonsquamous Non-small Cell Lung Cancer (MAPLE): A Randomized, Double-blind, Phase III *Study. Clin. Cancer Res.* 2019;25(7):2088-2095. **7.** Data on file, Amgen; [ABP 215 CSR; 2015].

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MVASI, in combination with carboplatin and gemcitabine is indicated for treatment of adult patients with first recurrent platinum-sensitive epithelial ovarian, fallopian tube or primary peritoneal cancers who have not received prior therapy with bevacizumab or other VEGF inhibitors or VEGF receptor-targeted agents. **DOSAGE AND ADMINISTRATION:** Dosage: either 5 mg/kg or 10 mg/kg once every 2 weeks or 7.5 mg/kg or 15 mg/kg once every 3 weeks, **mBC:** 10 mg/kg once every 2 weeks or 15 mg/kg once every 3 weeks. **NSCLC:** *First-line treatment in combination with platinum-based chemotherapy:* 7.5 mg/kg or 15 mg/kg once every 3 weeks, up to 6 cycles of treatment followed by MVASI as a single agent until disease progression. **mRCC:** 10 mg/kg once every 2 weeks. Treatment should be continued until progression of the underlying disease or until unacceptable toxicity. **Epithelial ovarian/Fallopian tube/Primary peritoneal cancer:** *front-line:* 15mg/kg once every 3 weeks in addition to carboplatin and paclitaxel for up to 6 cycles, then as monotherapy; *platinum-sensitive recurrent disease:* 15mg/kg once every 3 weeks in combination with carboplatin and gemcitabine for 6 cycles and up to 10 cycles, then as monotherapy. Method of administration: *initial dose:* IV infusion over 90 mins. If well tolerated, *second dose:* IV infusion over 60 mins. If well tolerated, *subsequent doses:* IV infusion over 30 mins. Do not administer as IV push or bolus or mix with glucose solutions. Dose reduction for adverse reactions is not recommended. If indicated, discontinue or temporarily suspend therapy. No recommendations for use in children (<18 years old). No dose adjustment in the elderly. **CONTRAINDICATIONS:** Hypersensitivity to the active substance or to any of the excipients. Hypersensitivity to Chinese Hamster Ovary cell products or other recombinant human or humanised antibodies. Pregnancy. **WARNINGS AND PRECAUTIONS:** Gastrointestinal (GI) perforations and fistulae: increased risk for the development of GI and gall bladder perforation; intra-abdominal inflammatory process may be a risk factor for gastrointestinal perforations in patients with metastatic carcinoma of the colon or rectum. discontinue therapy permanently in patients who develop GI perforation. Non-GI: Permanently discontinue therapy in patients with tracheoesophageal fistula or any grade 4 fistula. In cases of internal fistula not arising in the GI tract, discontinuation of therapy should be considered. Wound healing: do not initiate for at least 28 days following major surgery or until the surgical wound is fully healed: withhold for elective surgery. Necrotising fasciitis: therapy should be discontinued in patients who develop necrotising fasciitis, and initiate appropriate treatment. Hypertension: Pre-existing hypertension should be adequately controlled prior to treatment. Monitor blood pressure is generally recommended during therapy. The use of diuretics is not advised in patients who receive a cisplatin-based chemotherapy regimen. Permanently discontinue if medically significant hypertension cannot be adequately controlled with antihypertensive therapy, or hypertensive crisis or encephalopathy is developed. Posterior Reversible Encephalopathy Syndrome (PRES): signs and symptoms presented such as seizures, headache, altered mental status, visual disturbance, or cortical blindness, with or without associated hypertension. Diagnosis requires brain imaging for confirmation; recommend treatment of specific symptoms and discontinue therapy. Proteinuria: Patients with a history of hypertension may be at increased risk; monitoring of proteinuria by dipstick urinalysis is recommended prior to starting and during therapy. Permanently discontinue therapy in patients who develop nephrotic syndrome. Arterial thromboembolism: Patients receiving bevacizumab plus chemotherapy, with a history of arterial thromboembolism, diabetes or age greater than 65 years have an increased risk of developing arterial thromboembolic reactions during therapy. Permanently discontinued in patients who develop arterial thromboembolic reactions. Venous thromboembolism: Patients may be at risk of developing venous thromboembolic reactions, including pulmonary embolism under bevacizumab treatment. Discontinue therapy if life-threatening (grade 4) thromboembolic reactions develop, closely monitor inpatients with thromboembolic reactions ≤ grade 3. Haemorrhage: Patients treated with bevacizumab have an increased risk of haemorrhage, especially tumour-associated haemorrhage. Discontinue permanently if grade 3 or 4 bleeding develop. Risk of CNS haemorrhage in patients with untreated CNS metastases has not been prospectively evaluated. Monitor for signs and symptoms of CNS bleeding and discontinue therapy in cases of intracranial bleeding. Caution in patients with congenital bleeding diathesis, acquired coagulopathy or during anticoagulant therapy. Pulmonary haemorrhage/haemoptysis: Patients with recent pulmonary haemorrhage/haemoptysis (> 2.5 mL of red blood) should not be treated with bevacizumab. Congestive heart failure (CHF): Caution in patients with clinically significant cardiovascular disease such as pre-existing coronary artery disease, or CHF; most of the patients who experienced CHF had metastatic breast cancer and had received previous treatment with anthracyclines, prior radiotherapy to the left chest wall or other risk factors for CHF. Neutropenia and infections: Increased rates of severe neutropenia, febrile neutropenia, or infection with or without severe neutropenia (including some fatalities) have been observed in patients treated with some myelotoxic chemotherapy regimens plus bevacizumab. Hypersensitivity reactions (incl. anaphylactic data): Close observation during and following the administration of bevacizumab . If a reaction occurs, the infusion should be discontinued and appropriate medical therapies should be administered. Osteonecrosis of the jaw (ONJ): Cases of ONJ have been reported in cancer patients treated with bevacizumab, the majority of whom had received prior or concomitant treatment with IV bisphosphonates; patients who have previously received or are receiving i.v. bisphosphonates should avoid invasive dental procedures. Aneurysms and artery dissections: The use of VEGF pathway inhibitors in patients with or without hypertension may promote the formation of aneurysms and/or artery dissections. risk should be carefully considered prior to treatment in patients with risk factors such as hypertension or history of aneurysm. Eye disorders: serious ocular adverse reactions have been reported following unapproved intravitreal use of bevacizumab compounded from vials approved for intravenous administration in cancer patients. Systemic effects following intravitreal use: reduction of VEGF concentration has been demonstrated, adverse reactions including non-ocular haemorrhages and arterial thromboembolic reactions have been reported. Ovarian Failure / Fertility: Fertility preservation strategies should be discussed with women of child-bearing potential prior to treatment. **INTERACTIONS:** *Combination of bevacizumab and sunitinib malate:* microangiopathic haemolytic anaemia (MAHA) was reported in patients treated with bevacizumab and sunitinib malate combination; hypertension, elevated creatinine and neurological symptoms were also observed. Findings were reversible upon discontinuation of bevacizumab and sunitinib malate. *Combination with platinum- or taxane-based therapies:* Increased rates of severe neutropenia, febrile neutropenia, or infection with or without severe neutropenia have been observed in patients treated with platinum- or taxane-based therapies in the treatment of NSCLC and mBC. *Combination with EGFR monoclonal antibodies:* No interaction studies have been performed: decreased PFS and/or OS, and increased toxicity was observed. **FERTILITY, PREGNANCY AND LACTATION:** Women of childbearing potential have to use effective contraception during and up to 6 months after treatment. Pregnancy: no clinical trial data on the use of bevacizumab in pregnant women; suspected cause serious birth defects. Breast-feeding: Women must discontinue breast-feeding during therapy and for at least 6 months following the last dose. Fertility: bevacizumab may have temporary adverse effect on female fertility and cause ovarian failure. After discontinuation of bevacizumab treatment, ovarian function recovered in the majority of patients. Long term effects of the treatment with bevacizumab on fertility are unknown. **UNDESIRABLE EFFECTS:** Please refer to full prescribing information for full list of undesirable effects. *Very common adverse reactions:* febrile neutropenia, leucopenia, neutropenia, thrombocytopenia, anorexia, hypomagnesaemia, hyponatraemia, peripheral sensory neuropathy, dysarthria, headache, dysgeusia, eye disorder, lacrimation increased, hypertension, thromboembolism (venous), dyspnoea, rhinitis, epistaxis, cough, rectal haemorrhage, stomatitis, constipation, diarrhoea, nausea, vomiting, abdominal pain, wound healing complications, exfoliative dermatitis, dry skin, skin discolouration, arthralgia, myalgia, proteinuria, ovarian failure, asthenia, fatigue, pyrexia, pain, mucosal inflammation and weight decreased.

Abbreviated Prescribing Information Version: HKMVAPI03

Version date: Sep 2023

Please read the full prescribing information prior to administration and full prescribing information is available upon request.

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Vectibix® (panitumumab) Abbreviated Prescribing Information

Vectibix® (panitumumab) Concentrate for Solution for Infusion 20 mg/ml

INDICATIONS Vectibix is indicated for the treatment of adult patients with wild-type *RAS* metastatic colorectal cancer (mCRC) as first-line in combination with FOLFOX or FOLFIRI, as second-line in combination with FOLFIRI for patients who have received first-line fluoropyrimidine-based chemotherapy (excluding irinotecan) and as monotherapy after failure of fluoropyrimidine-, oxaliplatin-, and irinotecan-containing chemotherapy regimens. **DOSAGE AND ADMINISTRATION** The recommended dose of Vectibix is 6 mg/kg of bodyweight given once every two weeks. Modification of the dose of Vectibix may be necessary in cases of severe (≥ grade 3) dermatological reactions. There is no clinical data to support dose adjustments in the elderly. Vectibix must be administered as an intravenous (IV) infusion via an infusion pump. Prior to infusion, Vectibix should be diluted in sodium chloride 9 mg/ml (0.9%) solution for injection to a final concentration not to exceed 10 mg/ml. Vectibix must be administered using a low protein binding 0.2 or 0.22 micrometre in-line filter, through a peripheral line or indwelling catheter. The recommended infusion time is approximately 60 minutes. If the first infusion is tolerated, then subsequent infusions may be administered over 30 to 60 minutes. Doses higher than 1,000 mg should be infused over approximately 90 minutes. A reduction in the rate of infusion of Vectibix may be necessary in cases of infusion-related reactions. Vectibix must not be administered as an intravenous push or bolus. **CONTRAINDICATIONS** Vectibix is contraindicated in patients with a history of severe or life-threatening hypersensitivity to the active substance or to any of the excipients, and in patients with interstitial pneumonitis or pulmonary fibrosis. The combination of Vectibix with oxaliplatin-containing chemotherapy is contraindicated for patients with mutant *RAS* mCRC or for whom *RAS* mCRC status is unknown. **SPECIAL WARNINGS AND PRECAUTIONS FOR USE** *Dermatologic reactions and soft tissue toxicity:* Dermatologic related reactions, a pharmacologic effect observed with epidermal growth factor receptor (EGFR) inhibitors, are experienced with nearly all patients (approximately 94%) treated with Vectibix. Severe (NCI-CTC grade 3) skin reactions were reported in 23% and life-threatening (NCI-CTC grade 4) skin reactions in < 1% of patients who received Vectibix monotherapy and in combination with chemotherapy. *Pulmonary complications:* Cases of interstitial lung disease (ILD), both fatal and non-fatal, have been reported, mainly from the Japanese population. *Electrolyte disturbances:* Progressively decreasing serum magnesium levels leading to severe (grade 4) hypomagnesaemia have been observed in some patients. Other electrolyte disturbances, including hypokalaemia, have also been observed. *Infusion-related reactions:* Across monotherapy and combination mCRC clinical studies, infusion-related reactions (occurring within 24 hours of an infusion) were reported in Vectibix treated patients including severe infusion-related reactions (NCI-CTC grade 3 and grade 4). Hypersensitivity reactions occurring more than 24 hours after infusion have been reported including a fatal case of angioedema that occurred more than 24 hours after the infusion. *Acute renal failure:* Acute renal failure has been observed in patients who develop severe diarrhoea and dehydration. *Ocular toxicities:* Serious cases of keratitis and ulcerative keratitis, which may lead to corneal perforation, have been reported. Vectibix should be used with caution in patients with a history of keratitis, ulcerative keratitis or severe dry eye. Contact lens use is also a risk factor for keratitis and ulceration. *Patients with ECOG 2 performance status treated with Vectibix in combination with chemotherapy:* Assessment of benefit-risk is recommended prior to initiation of Vectibix in combination with chemotherapy for treatment of mCRC. A positive benefit-risk balance has not been documented in patients with ECOG 2 performance status. **INTERACTIONS** The infusion line should be flushed with physiologic saline solution before and after Vectibix administration to avoid mixing with other medicinal products or intravenous solutions. Vectibix should not be administered in combination with IFL chemotherapy or with bevacizumab-containing chemotherapy. **PREGNANCY, LACTATION AND FERTILITY** *Pregnancy:* There are no adequate data from the use of Vectibix in pregnant women. *Breast-feeding:* It is unknown whether panitumumab is excreted in human breast milk. It is recommended that women do not breast feed during treatment with Vectibix and for 2 months after the last dose. *Fertility:* Panitumumab may impact the ability of a woman to become pregnant. **UNDESIRABLE EFFECTS** Very commonly reported adverse reactions occurring in ≥ 20% of patients were gastrointestinal disorders (diarrhoea (46%), nausea (39%), vomiting (26%), constipation (23%) and abdominal pain (23%)); general disorders [fatigue (35%), pyrexia (21%)]; metabolism and nutrition disorders [decreased appetite (30%)]; infections and infestations [paronychia (20%)]; and skin and subcutaneous disorders [rash (47%), dermatitis acneiform (39%), pruritus (36%), erythema (33%) and dry skin (21%)]. **OVERDOSE** Doses up to 9 mg/kg have been tested in clinical trials. There have been reports of overdose at doses up to approximately twice the recommended therapeutic dose (12 mg/kg). Adverse events observed included skin toxicity, diarrhoea, dehydration and fatigue and were consistent with the safety profile at the recommended dose.

Abbreviated Prescribing Information Version: HKVECPI02

Version date: December 2021

Please read the full prescribing information prior to administration and full prescribing information is available on request.

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For medical inquiries or to report adverse events/product complaint, please contact +852 800 961 142 or email medinfo.JAPAC@amgen.com

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HKG-215-1225-80004 (Jan 2026)

AMGEN

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Tel: +852 2843 1178 Fax: +852 2808 2820

TEVIMBRA®
(tislelizumab)
100 mg concentrate for solution for infusion

A TREATMENT OPTION RECOMMENDED BY **ESMO+NCCN GUIDELINES**[6,9,10]

FOR YOUR PATIENTS WITH **UPPER GI CANCER**

LOOK FURTHER WITH TEVIMBRA

A unique PD-1 inhibitor optimised for lasting inhibition, through potent PD-1 binding and robust T-cell activation[1–5]*



TEVIMBRA is approved in 3 upper GI cancer indications: 1L OSCC, OSCC after prior platinum-based chemotherapy and 1L G/GEJ adenocarcinoma[7]

More than 2 million patients have been treated globally to date across all indications[8]

A well-characterised safety profile established across all approved indications in a broad population of nearly 3,000 patients[7]

INDICATIONS

Gastric / gastroesophageal junction (G/GEJ) adenocarcinoma[7]

TEVIMBRA, in combination with platinum and fluoropyrimidine-based chemotherapy, is indicated for the first-line treatment of adult patients with HER-2-negative locally advanced unresectable or metastatic G/GEJ adenocarcinoma whose tumours express PD-L1 with a tumour area positivity (TAP) score ≥5%.

Oesophageal squamous cell carcinoma (OSCC)[7]

TEVIMBRA, in combination with platinum-based chemotherapy, is indicated for the first-line treatment of adult patients with unresectable, locally advanced or metastatic OSCC whose tumours express PD-L1 with a TAP score ≥5%.

TEVIMBRA as monotherapy is indicated for the treatment of adult patients with unresectable, locally advanced or metastatic OSCC after prior platinum-based chemotherapy.

Abbreviated Prescribing Information:

Abbreviated Prescribing Information: Presentation: Tevimbra (Tislelizumab) Injection 100mg/10ml. Indication: Tevimbra is indicated for the treatment of patients with (1) in combination with Paclitaxel plus Carboplatin or Paclitaxel for injection (Albumin Bound) plus Carboplatin as first-line treatment in patients with unresectable, locally advanced or metastatic squamous NSCLC. (2)Tevimbra in combination with pemetrexed and platinum-containing chemotherapy is indicated for thefirst-line treatment of adult patients with non-squamous NSCLC whose tumours have PD-L1 expression on ≥50% of tumour cells with no EGFR or ALK positive mutations and who have locally advanced NSCLC and are not candidates for surgical resection or platinum-base chemoradiation, or metastatic NSCLC;(3) As monotherapy is indicated for the treatment of adult patients with locally advanced or metastatic non squamous non-small-cell lung cancer (NSCLC), with EGFR genomic tumor aberrations negative and ALK genomic tumor negative, that has progressed after or did not tolerate prior platinum-based chemotherapy and of adult patients with locally advanced or metastatic squamous non-small-cell lung cancer (NSCLC), with EGFR and ALK negative or unknown, that has progressed after or did not tolerate prior platinum-based chemotherapy. (4) in combination with platinum-based chemotherapy, is indicated for the first-line treatment of adult patients with unresectable, locally advanced or metastatic OSCC whose tumours express PD-L1 with a TAP score ≥ 5% ; (5)the treatment of locally advanced or metastatic esophageal squamous cell carcinoma who have disease progression following or are intolerant to first-line standard chemotherapy;(6) In combination with with platinum and fluoropyrimidine-based chemotherapy, is indicated for the first-line treatment of adult patients with HER-2-negative locally advanced unresectable or metastatic gastric or gastroesophageal junction (G/GEJ) adenocarcinoma whose tumours express PD-L1 with a tumour area positivity (TAP) score ≥ 5%. Dosage & Administration: Tevimbra is administered by intravenous infusion. The recommended dose of Tevimbra is 200 mg every 3 weeks. The treatment should last until disease progression or intolerable toxicity. Contraindications: Hypersensitivity to the active substance or to any of the excipients. Special Precautions: Immune-related adverse reactions, Immune-related pneumonitis, Immune-related hepatitis, immune-related skin reactions, immune-related colitis, Immune-related endocrinopathies, Thyroid disorders, Adrenal insufficiency, Hypophysitis, Type 1 diabetes mellitus, immune-related nephritis with renal dysfunction, Other immune-related adverse reactions, Solid organ transplant rejection, Infusion-related reactions, Patients excluded from clinical studies and Patients on controlled sodium diets. Adverse Reactions: The most commonly occurring adverse reactions (≥ 10%) were anaemia, hypothyroidism, cough, rash, pruritus, fatigue, decreased appetite, aspartate aminotransferase increased, alanine aminotransferase increased, blood bilirubin increased. Refer to the full prescribing information for other undesirable effects

*Based on preclinical data.[1-5] †TEVIMBRA is recommended in both 1L OSCC and OSCC after prior platinum-based chemotherapy treatment settings by the ESMO 2025 guidelines.[6]

Abbreviations: 1L, first-line; ESMO, European Society for Medical Oncology; G/GEJ, gastric or gastroesophageal junction; GI, gastrointestinal; OSCC, oesophageal squamous cell carcinoma; PD-1, programmed cell death protein 1; PD-L1, programmed death-ligand 1; TAP, tumour area positivity.
References: 1. Hong Y, et al. *FEBS Open Bio.* 2021;11(3):782–792. 2. Feng Y, et al. AACR annual meeting. March 29–April 3, 2019. Abstract 4048; 3. Zhang T, et al. *Cancer Control.* 2022;29:10732748221111296; 4. Desai J, et al. *J Immunother Cancer.* 2020;8(1):e000453; 5. Zhang T, et al. *Cancer Immunol Immunother.* 2018;67:1079–1090; 6. Obermannova R, et al. *ESMO Open.* 2025;10(2):104134; 7. TEVIMBRA. Hong Kong Abbreviated Prescribing Information, BeOne (Hong Kong); 2025; 8. Data on file : BeOne annual report 2026; 9. NCCN Guidelines Esophageal and Esophagogastric Junction Cancers Version 2.2026 10.NCCN Guidelines Gastric Cancer Version 2.2026
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BeOne

HERNEXEOS®
zongertinib tablets | 60 mg

HERNEXEOS®, A once-daily oral TKI targeting HER2 (ERBB2)-mutant mNSCLC

after prior systemic therapy[1]

NOW APPROVED

ORR[2]*
71%
(95% CI: 60, 80;
n=53/75; P<0.001)

mPFS[2]*
12.4 months
(95% CI: 8.2-NE)



HERNEXEOS® is indicated for the treatment of adult patients with unresectable or metastatic non-squamous non-small cell lung cancer (NSCLC) whose tumors have HER2 *(ERBB2)* tyrosine kinase domain activating mutations, as detected by a validated test, and who have received prior systemic therapy.[1]

This indication is registered under "1+" mechanism.[3]

ORR and PFS by RECIST v1.1 as assessed by BICR.[2]
*HERNEXEOS® was evaluated in BEAMION® LUNG-1, a single-arm, open-label, multi-center trial. Major efficacy outcome measures include ORR and PFS.[2]
BICR=blinded independent central review; CI=confidence interval; ERBB2=erb-b2 receptor tyrosine kinase 2; HER2=human epidermal growth factor receptor 2; mPFS=median progression-free survival; mNSCLC=metastatic non-small cell lung cancer; NE=not evaluated; ORR=objective response rate; PFS=progression-free survival; RECIST=Response Evaluation Criteria In Solid Tumors; TKI=tyrosine kinase inhibitor.
References: 1. HERNEXEOS® Hong Kong Prescribing Information. **2.** Heymach JV, et al. Zongertinib in Previously Treated HER2-Mutant Non-Small-Cell Lung Cancer. N Engl J Med. 2025;392(23):2321-2333. **3.** Pharmacy & Poisons Board of Hong Kong. List of Pharmaceutical Products Registered Under "1+" Mechanism. Available at: https://www.drugoffice.gov.hk/eps/do/en/consumer/news_informations/relicList2.html?indextype=1PLUS. Accessed on 7 May 2026.
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Please scan the QR code to access the abbreviated prescribing information of HERNEXEOS®

Boehringer Ingelheim

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Jemperli (dostarlimab) Injection 500 mg

Let their light *Shine Longer*

All-comers indication now approved



16 months mOS improvement[1*]

Broad disease characteristics included in the trial[2†]

~1-year mPFS[3‡]

Manageable safety profile[1§]

INDICATION

- **JEMPERLI is indicated in combination with carboplatin and paclitaxel for the treatment of adult patients with primary advanced or recurrent endometrial cancer (EC) and who are candidates for systemic therapy.[3]**

*HR compared with CP alone, 0.69; 95% CI, 0.54–0.89; p=0.0002.[1] †Carcinosarcoma and serous adenocarcinoma were included in the trial. P53 subgroup analysis is also available.[2] ‡ HR compared with CP alone, 0.64; 95% CI, 0.51–0.80; p<0.0001.[3] §No new safety signals were observed with additional follow up.[1]
Abbreviations: **CP,** carboplatin + paclitaxel; **EC,** endometrial cancer; **HR,** hazard ratio; **mOS,** median overall survival; **mPFS,** median progression-free survival.
References: 1. Powell MA, et al. Overall survival in patients with primary advanced or recurrent endometrial cancer treated dostarlimab plus chemotherapy in Part 1 of the ENGOT–EN6-NSGO/GOC-3031/RUBY trial. Presented at the Society of Gynecologic Society (SGO) 2024 Annual Meeting on Women's Cancer; 16–18 March 2024; San Diego, US. **2.** Mirza MR, et al. Dostarlimab + Chemotherapy for the Treatment of Primary Advanced or Recurrent Endometrial Cancer: Analysis of Progression-Free Survival and Overall Survival Outcomes by Molecular Classification in the ENGOT-EN6-NSGO/GOG-3031/RUBY Trial. Presented at: ESMO Congress 2023; 20–24 October 2023; Madrid, Spain. **3.** JEMPERLI. Hong Kong Prescribing Information. Version: HK67364_Jemperli concentrate solution for infusion (clean)_20250529. GSK.

Safety information for Jemperli (Dostarlimab)
Immune-related adverse reactions, which may be severe or fatal, can occur. While immune-related adverse reactions usually occur during treatment with PD-1/PD-L1 blocking antibodies, symptoms can also manifest after discontinuation of treatment. Early identification and management of immune-related adverse reactions are essential to ensure use of PD-1/PD-L1 blocking antibodies. Monitor for symptoms and signs of immune-related adverse reactions. Evaluate clinical chemistries, including liver tests and thyroid function tests, at baseline and periodically during treatment.

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Safety Statement:
For adverse event reporting, please call GlaxoSmithKline Limited at (852) 3189 8989 (Hong Kong),
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PM-HK-DST-LBND-250002 (06/2027)
Date of preparation: 07/2025

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Fruquintinib Capsules

Designed for Better Targeting

in patients with Metastatic Colorectal Cancer



ELUNATE® has shown a consistent safety and efficacy profile in mCRC clinical studies - FRESCO, FRESCO-2, and maintain efficacy in patients with prior targeted treatment exposure. [1,2]

International Clinical Practice Guidelines recommend ELUNATE® (Fruquintinib) as a regimen for mCRC patients previously treated with chemotherapy.^[3-6]

ONE Capsule, ONCE Daily*
*3 weeks on & 1 week off

^ in patients who previously treated with oxaliplatin and irinotecan

References: **1.** Li *et al.* Effect of fruquintinib vs placebo on overall survival in patients with previously treated metastatic colorectal cancer: the FRESCO randomized clinical trial. JAMA. 2018;319 (24):2486-96. **2.** Dasari *et al.* Fruquintinib versus placebo in patients with refractory metastatic colorectal cancer (FRESCO-2): an international, multicentre, randomised, double-blind, phase 3 study. Lancet. 2022. **3.** NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Colon Cancer V.2.2026. Accessed 13 July 2026. **4.** NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Rectal Cancer V.2.2026. Accessed 13 July 2026. **5.** Chinese Society of Clinical Oncology (CSCO) Guideline for Colorectal Cancer 2025. Accessed 13 July 2026. **6.** Yoshino *et al.* Pan-Asian adapted ESMO Clinical Practice Guidelines for the diagnosis, treatment and follow-up of patients with metastatic colorectal cancer. ESMO Open. 2023 Vol 8 issue 3.

ELUNATE® Abbreviated Product Information

This advertisement is intended for health care professionals only. Please refer to the approved package insert for detailed prescribing information.

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5 years
of real-world experience

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CONFIDENCE FOLLOWS EXPERIENCE

Reflect on how the trusted SoC for 1L uHCC, backed by 5 years of real-world experience, improves survival outcomes for a wide range of patients[1-11]

TECENTRIQ® + AVASTIN®

Indication:

TECENTRIQ, in combination with bevacizumab, is indicated for the treatment of patients with unresectable or metastatic hepatocellular carcinoma (HCC) who have not received prior systemic therapy

References:

1. Gordan JD, et al. (2024) J Clin Oncol, 42:1830–1850.
2. Singal AG, et al. (2023) Hepatology, 78(6):1922–1965.
3. Vogel A, et al. (2025) Ann Oncol. In press.
4. Reig M, et al. (2022) J Hepatol, 76:681–693.
5. ILCA (2020) Systemic Therapy Guidance.
6. Sangro B, et al. (2025) J Hepatol, 82:315–374.
7. Finn R, et al. (2020) N Engl J Med, 382:1894–1905.
8. Cheng A-L, et al. (2022) J Hepatol, 76:862–873.
9. Fulgenzi C, et al. (2022) Eur J Cancer, 175:204–213.
10. Manfredi, G. et al. (2025) JHEP Reports, https://doi.org/10.1016/ j.jhepr.2025.101431
11. D'Alessio A, et al. (2022) Hepatology, 76:1000–1012.

SoC = Standard of Care; 1L = first line; uHCC = unresectable hepatocellular carcinoma

M-HK-00002494 approved in April 2026 is valid for 2 years or until change is required in accordance with regulatory requirements, whichever comes first.

Roche Hong Kong Limited
Unit 1-2, 24/F, Central Plaza, 18 Harbour Rd, Wan Chai, Hong Kong
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TECENTRIQ® IV Prescribing Information

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ACT Onco® Pro
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Panel:
440 clinically relevant cancer gene variants

Suitable Patients:
Patients with solid tumors
-Identify biomarkers for targeted therapies
-Explore other treatment options
when disease resistance, recurrence, or metastasis happens

Specimen types:
FFPE/Cell Block

Turnaround time:
8 Working Days

Comprehensive Genetic & Proteomics Analysis
SNV, Insertion/Deletion, CNA, Fusions, TMB, MSI, Protein biomarkers

Matched Treatment Recommendations:
Targeted therapy · Immunotherapy · Chemotherapy · Hormonal Therapy · Antibody-drug Conjugates (ADC)

OncoOmicsDx
Tumor Proteomics Testing

Panel:
30/72 protein biomarkers for chemotherapy, targeted therapy, immunotherapy, antibody-drug conjugates (ADC)

Technology:
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Specimen types:
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Comprehensive Package
Take action to identify both genetic and protein profile for the right therapies

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NEW BREAKTHROUGH OVERALL SURVIVAL DATA
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SHE'S ON TRACK
FOR THE EXECUTIVE BOARD
NOT RECURRENCE

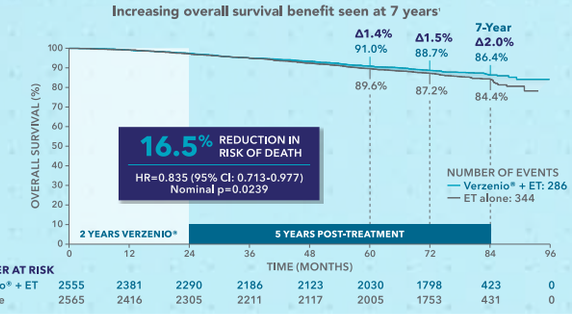
In node-positive, high-risk HR+/HER2- EBC,* **Verzenio® is proven to increase survival** and **deliver long-lasting protection** from recurrence with 2 years of treatment, so you can help give **her a future of opportunity.**†,1,2



In monarchE cohort 1

Verzenio® is the first CDK4/6 inhibitor with a proven overall survival benefit in the curative setting and 2 years of treatment*,†,1,2

Increasing overall survival benefit seen at 7 years†



NUMBER AT RISK

Verzenio® + ET	2555	2381	2290	2186	2123	2030	1798	423	0
ET alone	2565	2416	2305	2211	2117	2005	1753	431	0

Kaplan-Meier curve depicting OS rates at 7 years in Cohort 1 for the monarchE phase 3 global trial.

In monarchE cohort 1

Verzenio® delivers 7 years of sustained IDFS & DRFS benefits and 2 years of treatment*,2

- At the 7-year IDFS analysis, **77.0% of patients remained recurrence-free with Verzenio® + ET** vs 70.1% with ET alone (HR=0.726 [95% CI: 0.648-0.815]; nominal p<0.0001).2
- At the 7-year DRFS analysis, **79.5% of patients remained recurrence-free to incurable metastatic disease with Verzenio® + ET** vs 74.0% with ET alone (HR=0.736 [95% CI: 0.651-0.832]; nominal p<0.0001).2

*Verzenio® in combination with ET (AI or tamoxifen) is indicated for the adjuvant treatment of adult patients with HR+/HER2-, node-positive EBC at a high risk of recurrence. High-risk EBC in Cohort 1 was defined as patients who had ≥4 positive nodes or 1–3 positive nodes and tumors that were ≥5 cm, histological Grade 3—or both.1,2
†A statistically significant OS benefit was seen in the ITT population. Following regulatory consultation at the primary IDFS analysis, the OS analysis plan was amended to increase final events from 390 to 650 to ensure ≥5 years' follow-up. Verzenio® is approved for Cohort 1 (91% of ITT); OS analysis in this subpopulation was not powered or alpha-controlled.1,2

Indication:
Early Breast Cancer
Verzenio® in combination with endocrine therapy is indicated for the adjuvant treatment of adult patients with hormone receptor (HR)-positive, human epidermal growth factor receptor 2 (HER2)-negative, node-positive early breast cancer at high risk of recurrence. In pre- or perimenopausal women, aromatase inhibitor endocrine therapy should be combined with a luteinising hormone-releasing hormone (LHRH) agonist.1

Abbreviations:
AI: aromatase inhibitor; CDK4/6: cyclin-dependent kinase 4 and 6; CI: confidence interval; EBC: early breast cancer; ET: endocrine therapy; HER2-: human epidermal growth factor receptor 2-negative; HR: hazard ratio; HR+: hormone receptor-positive; IDFS: invasive disease-free survival; ITT: intent-to-treat; OS: overall survival.

References:

1. Johnston S, Martin M, O'Shaughnessy J, et al. Overall Survival with Abemaciclib in Early Breast Cancer. Ann Oncol. 2025. doi:10.1016/j.annonc.2025.10.005.
2. Verzenio® Hong Kong Prescribing Information.

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One Proven Solution in Multiple Conditions

Sintilimab injection Tyvyt has been approved in China for seven indications

Lymphoma[1,8]

NSCLC Non-squamous[2,9]

EGFR+ NSCLC[3,9]

NSCLC Squamous[4,9]

Liver Cancer[5,10]

Esophageal Cancer[6,11]

Gastric Cancer[7,12]

INOCRAS

Whole Genome Power. Whole Patient Impact.

Tumor profiling and tumor fingerprinting → Time point monitoring tests



Test for BRAF V600. TARGET it across NSCLC, melanoma, anaplastic thyroid cancer and paediatric glioma.[1,2]

BRAF V600 mutation status must be confirmed using a validated test prior to treatment. Anaplastic thyroid cancer and paediatric glioma indications are specific to BRAF V600E.[1,2]

Tafinlar® (dabrafenib) 50 mg, 75 mg capsules + Mekinist® (trametinib) 0.5 mg, 2 mg tablets

Abbreviations: NSCLC, non-small cell lung cancer. **References: 1.** Tafinlar® HK Prescribing Information, rev. 7 Nov 2025. **2.** Mekinist® HK Prescribing Information, rev. 7 Nov 2025.

MEKINIST® Important note: Before prescribing, consult full prescribing information of MEKINIST® (trametinib). When used in combination with TAFINLAR® (dabrafenib), consult full prescribing information of both products. **Presentation:** ♦ Film-coated tablets containing trametinib dimethyl sulfoxide equivalent to 0.5 mg and 2 mg of trametinib. **Indications:** ♦ Unresectable or metastatic melanoma. Mekinist in combination with dabrafenib is indicated for the treatment of adult patients with BRAF V600 mutation positive unresectable Stage III or metastatic (Stage IV) melanoma. Mekinist as a monotherapy is indicated for the treatment of adult patients with BRAF V600 mutation positive unresectable Stage III or metastatic (Stage IV) melanoma who have not demonstrated clinical activity in patients who have progressed on BRAF inhibitor. ♦ Adjuvant treatment of melanoma. Mekinist in combination with dabrafenib is indicated for the adjuvant treatment of adult patients with melanoma with a BRAF V600 mutation and involvement of the lymph node(s), following complete resection. ♦ Non-small cell lung cancer (NSCLC). Mekinist in combination with dabrafenib is indicated for the treatment of adult patients with advanced non-small cell lung cancer (NSCLC) with a BRAF V600 mutation. ♦ Anaplastic thyroid cancer (ATC). Mekinist in combination with dabrafenib is indicated for the treatment of adult patients with locally advanced or metastatic anaplastic thyroid cancer (ATC) with a BRAF V600E mutation and with no satisfactory locoregional treatment options. ♦ Low-grade glioma. Mekinist in combination with dabrafenib is indicated for the treatment of paediatric patients 1 year of age and older who weigh at least 26 kg with low-grade glioma (LGG) with a BRAF V600E mutation who require systemic therapy. ♦ High-grade glioma. Mekinist in combination with dabrafenib is indicated for the treatment of paediatric patients 1 year of age and older who weigh at least 26 kg with high-grade glioma (HGG) with a BRAF V600E mutation who have progressed following prior treatment and have no satisfactory alternative treatment options. **Dosage and administration:** ♦ **Mekinist® (trametinib) tablets:** Adults: The recommended dose of MEKINIST, used as monotherapy or in combination with dabrafenib, is 2 mg given orally once daily independent of body weight. Paediatrics: The recommended dosage for Mekinist tablets in paediatric patients who weigh at least 26 kg, is based on body weight. A recommended dose for patients who weigh less than 26 kg has not been established. Administration: MEKINIST should be taken without food, at least 1 hour before or 2 hours after a meal. ♦ When MEKINIST and dabrafenib are taken in combination, the dose of MEKINIST is missed, only take the dose if it is more than 12 hours until the next scheduled dose. ♦ **Dose modifications:** The management of adverse events/adverse drug reactions may require treatment interruption, dose reduction, or treatment discontinuation. **Special populations:** ♦ **Children** (< 1 year of age): Safety and efficacy not established. ♦ **Elderly (≥ 65 years):** No dose adjustment required. Moderate or severe: Should be used with caution. ♦ **Contraindication:** Hypersensitivity to the active substance trametinib, dimethyl sulfoxide or any of the excipients. **Warnings and precautions:** ♦ **Left ventricular ejection fraction (LVEF) reductions/Left ventricular dysfunction:** Cases of LVEF decrease reported. Should be used with caution when conditions could impair left ventricular function. Patients should be evaluated for LVEF prior to initiation of treatment with continued evaluation during treatment. Dose modification guidelines should be considered. ♦ **Hemorrhage:** Haemorrhagic events, including major and fatal haemorrhagic events occurred in patients taking Mekinist as monotherapy and in combination with Tafinlar. ♦ **Visual impairment:** Visual disturbances, including chorioretinopathy or retinal pigment epithelial detachment (RPED) and retinal vein occlusion (RVO) occurred. Not recommended for patients with history of RVO. Ophthalmological evaluation should be performed at baseline and during treatment. If new ocular abnormalities are observed, treatment should be interrupted and if confirmed, treatment discontinued. Permanent discontinuation of treatment if RVO occurs. ♦ **Rash:** Observed in Mekinist monotherapy and in combination with Tafinlar. ♦ **Severe cutaneous adverse reactions (SCARs):** SCARs, including Stevens-Johnson syndrome, and drug reaction with eosinophilia and systemic symptoms (DRESS), which can be life-threatening or fatal, have been reported with Mekinist in combination with Tafinlar. Before initiating treatment, patients should be advised of the signs and symptoms and monitored closely for skin reactions. If signs and symptoms suggestive of SCARs appear, Mekinist and Tafinlar should be withdrawn. ♦ **Venous thromboembolism (VTE):** VTE, including deep vein thrombosis (DVT) and pulmonary embolism (PE) can occur when used as monotherapy or in combination with Tafinlar. Patients should seek immediate medical care if they develop symptoms of VTE. ♦ **Pyrexia:** Pyrexia including severe rigors, dehydration, renal impairment, renal failure and renal insufficiency reported. Incidence and severity increased when Mekinist used in combination with Tafinlar. Monitoring serum creatinine and other evidence of renal function impairment during and following severe pyrexia events. Serious non-infectious febrile events observed. For management of pyrexia, therapy with MEKINIST (MEKINIST when used in combination) should be interrupted if the patient's temperature is ≥ 39.0 °C or at the first symptom of pyrexia syndrome. In case of recurrence, therapy can also be interrupted at the first symptom of pyrexia. ♦ **Colitis and Gastrointestinal perforation:** Colitis and gastrointestinal perforation, including fatal outcome, reported. Treatment with Mekinist as monotherapy or in combination with Tafinlar should be used with caution in patients with risk factors for gastrointestinal perforation, including a history of diverticulitis, metastasis to the gastrointestinal tract and concomitant use of medications that are associated with gastrointestinal perforation. If patients develop signs/symptoms of a gastrointestinal perforation, they should immediately seek medical care. ♦ **Hemophagocytic lymphohistiocytosis (HLH):** In postmarketing experience, HLH has been observed with Mekinist in combination with Tafinlar. If HLH is suspected, treatment should be discontinued and appropriate management of HLH should be initiated. ♦ **Tumor lysis syndrome (TLS):** Cases of TLS, including fatal cases, have been reported in patients treated with Mekinist in combination with Tafinlar. Risk factors for TLS include rapidly growing tumors, a high tumor burden, renal dysfunction, and dehydration. Patients with risk factors for TLS should be closely monitored, prophylaxis should be considered (e.g., intravenous hydration and treatment of high uric acid levels) prior to initiating treatment and treated as clinically indicated. **Pregnancy, lactation, females and males of reproductive potential** ♦ **Pregnancy:** Mekinist can be harmful to the fetus. Pregnant women should be advised of the potential risk to the fetus. ♦ **Lactation:** Nursing women should be advised of the potential risk to the baby. ♦ **Females and males of reproductive potential:** Sexually active women should be advised to use effective contraception while on Mekinist and for at least 16 weeks after stopping it. Efficacy of oral or any other systemic hormonal contraceptives may be decreased; an effective alternative method of contraception should be used. Male patients (including those that have had a vasectomy) should be advised to use condoms while on Mekinist and for at least 16 weeks after stopping it. **Infertility:** May impair fertility in humans. **Adverse events with Mekinist monotherapy in melanoma Very common (≥10%):** hypersensitivity, fatigue, peripheral oedema, cough, dyspnoea, diarrhoea, nausea, vomiting, constipation, abdominal pain, dry mouth, rash, dermatitis acneiform, dry skin, pruritus, alopecia, hemorrhage, pyrexia, chills, rash pustular, anaemia, dehydration, vision blurred, periorbital oedema, visual impairment, left ventricular dysfunction, ejection fraction decreased, bradycardia, lymphoedema, epistaxis, pneumonitis, stomatitis, skin chapped, erythema, palmar-plantar erythrodysesthesia syndrome, skin fissures, blood creatine phosphokinase increased, face oedema, mucosal inflammation, asthenia, aspartate aminotransferase increased, alanine aminotransferase increased, blood alkaline phosphatase increased. **Common (≥0.1 to <1%):** cholangitis, retinal vein occlusion, papilloedema, retinal detachment, cardiac failure, interstitial lung disease, gastrointestinal perforation, colitis, rhabdomyolysis. **Adverse events in combination with Tafinlar in metastatic melanoma Very common (≥10%):** urinary tract infection, nasopharyngitis, neutropenia, decreased appetite, headache, dizziness, hypertension, haemorrhage, cough, abdominal pain, constipation, diarrhoea, nausea, vomiting, dry skin, pruritus, rash, dermatitis acneiform, arthralgia, myalgia, pain in extremity, fatigue, oedema peripheral, pyrexia, chills, asthenia, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** cellulitis, folliculitis, paronychia, rash pustular, cutaneous squamous cell carcinoma (SCC) including SCC of the skin, SCC in situ (Bowen's disease) and keratoacanthoma, papilloma including skin papilloma, sebaborrheic keratosis, acrochordon (skin tags), anaemia, thrombocytopenia, leukopenia, dehydration, hypophosphataemia, hyponatraemia, hypokalaemia, hypomagnesaemia, hyperglycaemia, hypercalcaemia, hypoalbuminaemia, hypertriglyceridaemia, insomnia, peripheral sensory neuropathy, dysgeusia, dizziness, syncope, dysarthria, somnolence, headache, dysgeusia, dry mouth, stomatitis, erythema, astrio keratosis, night sweats, hyperkeratosis, alopecia, palmar-plantar erythrodysesthesia syndrome, skin lesion, hyperhidrosis, skin fissures, paronychia, photosensitivity, myalgia, spasms, blood creatine phosphokinase increased, renal failure, mucosal inflammation, influenza-like illness, face oedema, blood alkaline phosphatase increased, gamma-glutamyltransferase increased. **Uncommon (≥0.1 to <1%):** new primary melanoma, hypersensitivity, choroiretinopathy, uveitis, retinal detachment, periorbital oedema, left ventricular dysfunction, cardiac failure, pneumonitis, interstitial lung disease, pancreatitis, gastrointestinal perforation, colitis, rhabdomyolysis, nephritis, renal failure acute. **Adverse drug reactions in combination with Tafinlar in Stage III melanoma following complete resection Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypertension, cough, nausea, diarrhoea, vomiting, abdominal pain, constipation, rash, dry skin, dermatitis acneiform, erythema, pruritus, arthralgia, myalgia, pain in extremity, fatigue, chills, oedema peripheral, influenza-like illness, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** weight decreased, hypophosphataemia, hyperglycaemia, hypercalcaemia, hypoalbuminaemia, hypertriglyceridaemia, peripheral sensory neuropathy, dysgeusia, dizziness, syncope, dysarthria, somnolence, headache, dysgeusia, dry mouth, stomatitis, erythema, astrio keratosis, night sweats, hyperkeratosis, alopecia, palmar-plantar erythrodysesthesia syndrome, skin lesion, hyperhidrosis, skin fissures, paronychia, photosensitivity, myalgia, spasms, blood creatine phosphokinase increased, renal failure, mucosal inflammation, influenza-like illness, face oedema, blood alkaline phosphatase increased, gamma-glutamyltransferase increased. **Uncommon (≥0.1 to <1%):** rhabdomyolysis, renal failure. **Adverse drug reactions in combination with Tafinlar in advanced non-small cell lung cancer Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypotension, nausea, vomiting, diarrhoea, decreased appetite, constipation, erythema, dry skin, rash, pruritus, hyperkeratosis and hyperkeratosis, actinic and seborrheic keratosis and keratosis pilaris, keratoacanthoma, papilloma including skin papilloma, arthralgia, myalgia, pyrexia, asthenia including fatigue and malaise, oedema (generalised and peripheral), chills, blood and urine phosphorus decreased, blood and urine phosphate increased, blood creatine phosphokinase increased, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** cutaneous squamous cell carcinoma, leukopenia, dehydration, detachment of retinal/retinal pigment epithelium, ejection fraction decreased, hypertension, pulmonary embolism, pancreatitis acute, renal failure, tubulointerstitial nephritis. For a complete list, consult full prescribing information. **Adverse drug reactions in combination with Mekinist in locally advanced or metastatic anaplastic thyroid cancer (ATC) Very common (≥10%):** neutropenia, anaemia, leukopenia, hyperglycaemia, decreased appetite, headache, dizziness, haemorrhage, cough, nausea, vomiting, diarrhoea, constipation, dry mouth, rash, myalgia, arthralgia, fatigue, pyrexia, chills, oedema, alanine aminotransferase increased, aspartate aminotransferase increased, blood alkaline phosphatase increased. **Common (≥1 to <10%):** hypophosphataemia, hyponatraemia, detachment of retinal/pigment epithelium, hypertension, rhabdomyolysis, ejection fraction decreased. **Adverse drug reactions in combination with Mekinist in pediatric patients (1 to <18 years of age) Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypotension, nausea, vomiting, diarrhoea, decreased appetite, constipation, erythema, dry skin, rash, pruritus, hyperkeratosis and hyperkeratosis, actinic and seborrheic keratosis and keratosis pilaris, muscle spasms, arthralgia, myalgia, pyrexia, asthenia including fatigue and malaise, oedema (generalised and peripheral), chills, blood and urine phosphorus decreased, blood and urine phosphate increased, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** hypophosphataemia, hyponatraemia, detachment of retinal/retinal pigment epithelium, ejection fraction decreased, hypertension, pulmonary embolism, pancreatitis acute, renal failure, tubulointerstitial nephritis. **Adverse drug reactions in combination with Mekinist in locally advanced or metastatic anaplastic thyroid cancer (ATC) Very common (≥10%):** neutropenia, anaemia, leukopenia, hyperglycaemia, decreased appetite, headache, dizziness, haemorrhage, hypertension, cough, nausea, vomiting, diarrhoea, constipation, dry mouth, rash, myalgia, arthralgia, fatigue, pyrexia, chills, oedema, alanine aminotransferase increased, aspartate aminotransferase increased, blood alkaline phosphatase increased. **Common (≥1 to <10%):** hypophosphataemia, hyponatraemia, detachment of retinal/pigment epithelium, hypertension, rhabdomyolysis, ejection fraction decreased. **Adverse drug reactions in combination with Mekinist in pediatric patients (1 to <18 years of age) Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypotension, nausea, vomiting, diarrhoea, decreased appetite, constipation, erythema, dry skin, rash, pruritus, hyperkeratosis and hyperkeratosis, actinic and seborrheic keratosis and keratosis pilaris, muscle spasms, arthralgia, myalgia, pyrexia, asthenia including fatigue and malaise, oedema (generalised and peripheral), chills, blood and urine phosphorus decreased, blood and urine phosphate increased, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** hypophosphataemia, hyponatraemia, detachment of retinal/retinal pigment epithelium, hypertension, pulmonary embolism, pancreatitis acute, renal failure, tubulointerstitial nephritis. **Adverse drug reactions in combination with Tafinlar monotherapy from post-marketing experience and pooled clinical trials Common (≥1 to <10%):** peripheral neuropathy. **Adverse drug reactions with Tafinlar in combination with Mekinist from post-marketing experience and pooled clinical trials Common (≥1 to <10%):** peripheral neuropathy. **Adverse drug reactions with Tafinlar monotherapy from post-marketing experience and pooled clinical trials Common (≥1 to <10%):** VTE, peripheral neuropathy, ulceration of the bowel. **Uncommon (≥0.1 to <1%):** sarcoidosis, burkitt lymphoma, bowel perforation, diverticulitis, GI perforation. **Adverse drug reactions with Tafinlar in combination with Mekinist from post-marketing experience and pooled clinical trials Uncommon (≥0.1 to <1%):** sarcoidosis, burkitt lymphoma, bowel perforation, diverticulitis, GI perforation. **Interactions:** ♦ Strong inhibitors or inducers of CYP3A4: Dose adjustment may be necessary, respectively. Tafinlar concentration: ♦ Alternative agents should be considered during administration of Tafinlar. ♦ Tafinlar inhibits OATP1B1 and OATP1B3. Therefore caution is recommended at co-administration of TAFINLAR and OATB1B1 or OATP1B3 substrates such as statins. ♦ Tafinlar is a substrate of CYP2C8 and CYP3A4. ♦ Caution should be exercised and additional monitoring of digoxin is recommended when digoxin is used concomitantly with TAFINLAR and at discontinuation of TAFINLAR. For complete interactions, consult full prescribing information. **Packs:** 28's pack. Not all pack sizes may be marketed. **Legal classification:** P1S1S1 **CDS Version:** 3.11 **Reference:** AUS 16 Oct 2025 **Revision date:** 7 Nov 2025

TAFINLAR® Important note: Before prescribing, consult full prescribing information of TAFINLAR® (dabrafenib). When used in combination with MEKINIST® (trametinib), consult full prescribing information of both products. **Presentation:** ♦ Hard capsules. Each capsule contains dabrafenib mesylate equivalent to 50 mg or 75 mg of dabrafenib. **Indications:** ♦ Unresectable or metastatic melanoma. TAFINLAR in combination with trametinib is indicated for the treatment of adult patients with BRAF V600 mutation positive unresectable (Stage III) melanoma. TAFINLAR as monotherapy is indicated for the adjuvant treatment of adult patients with BRAF V600 mutation positive unresectable (Stage III) melanoma, with involvement of the lymph node(s), following complete resection. ♦ Non-small cell lung cancer (NSCLC). TAFINLAR in combination with trametinib is indicated for the treatment of adult patients with advanced non-small cell lung cancer (NSCLC) with a BRAF V600 mutation and with no satisfactory locoregional treatment options. ♦ Anaplastic thyroid cancer (ATC). TAFINLAR in combination with trametinib is indicated for the treatment of adult patients with locally advanced or metastatic anaplastic thyroid cancer (ATC) with a BRAF V600E mutation and with no satisfactory locoregional treatment options. ♦ Low-grade glioma. TAFINLAR in combination with trametinib is indicated for the treatment of paediatric patients 1 year of age and older who weigh at least 26 kg with low-grade glioma (LGG) with a BRAF V600E mutation who require systemic therapy. ♦ High-grade glioma. TAFINLAR in combination with trametinib is indicated for the treatment of paediatric patients 1 year of age and older who weigh at least 26 kg with high-grade glioma (HGG) with a BRAF V600E mutation who have progressed following prior treatment and have no satisfactory alternative treatment options. **Dosage and administration:** ♦ **Tafinlar® hard capsules:** Adults: The recommended dose of TAFINLAR in adult patients with a BRAF V600 mutation who have progressed following prior therapy is TAFINLAR 150 mg (two 75 mg capsules) taken orally corresponding to a total daily dose of 300 mg, independent of body weight. *Paediatrics:* The recommended dose and dose level reductions of TAFINLAR capsules in paediatric patients who weigh at least 26 kg, is based on body weight. A recommended dose of TAFINLAR capsules for patients who weigh less than 26 kg has not been established. *Administration:* TAFINLAR should be taken either at least one hour before, or at least two hours after a meal due to the effect of food on TAFINLAR capsules. ♦ Tafinlar should not be used in patients with a history of hypersensitivity to dabrafenib or any of the excipients. **Warnings and precautions:** ♦ Tafinlar should not be used in patients with BRAF wild-type melanoma. ♦ When TAFINLAR and MEKINIST are taken in combination, the dose of MEKINIST at the same time each day with either the morning dose or the evening dose of TAFINLAR. ♦ **Missed dose:** If a dose is missed, it should not be taken if it is less than 6 hours until the next dose. ♦ **Dose modifications:** Management of adverse reactions may require treatment interruption, dose reduction or treatment discontinuation. **Special populations:** ♦ **Children (≥1 year of age):** Safety and efficacy not established. ♦ **Elderly (≥65 years):** No dose adjustment required. Moderate or severe: Should be used with caution. **Contraindications:** The active substance dabrafenib or any of the excipients. Tafinlar should not be used in patients with BRAF wild-type melanoma. **Warnings and precautions:** ♦ **Hepatic impairment:** Mild or moderate: No dose adjustment required. Severe: Should be used with caution. ♦ **Renal impairment:** Mild or moderate: No dose adjustment required. Severe: Should be used with caution. ♦ **Hepatic impairment:** Mild to moderate: No dose adjustment required. Severe: Should be used with caution. ♦ **Left ventricular ejection fraction (LVEF) reductions/Left ventricular dysfunction:** Cases of LVEF decrease reported. Should be used with caution when conditions could impair left ventricular function. Patients should be evaluated for LVEF prior to initiation of treatment with continued evaluation during treatment. Dose modification guidelines should be considered. ♦ **Hemorrhage:** Haemorrhagic events, including major and fatal haemorrhagic events occurred in patients taking Tafinlar as monotherapy and in combination with Mekinist. Risk factors for TLS include rapidly growing tumors, a high tumor burden, renal dysfunction, and dehydration. Patients with risk factors for TLS should be closely monitored, prophylaxis should be considered (e.g., intravenous hydration and treatment of high uric acid levels) prior to initiating treatment and treated as clinically indicated. **Pregnancy, lactation, females and males of reproductive potential** ♦ **Pregnancy:** Tafinlar can be harmful to the fetus. Pregnant women should be advised of the potential risk to the fetus. ♦ **Lactation:** Nursing women should be advised of the potential risk to the baby. ♦ **Females and males of reproductive potential:** Sexually active women should be advised to use effective contraception while on Tafinlar and for at least 2 weeks after stopping it. If taken in combination with Mekinist, effective contraception should be used. Male patients (including those that have had a vasectomy) should be advised to use condoms while on Tafinlar and for at least 2 weeks after stopping it. If taken in combination with Mekinist, condoms should be used while on treatment and for at least 16 weeks after stopping it. ♦ **Infertility:** Potential risk for impaired spermatogenesis, which may be irreversible. **Adverse events with Tafinlar monotherapy in metastatic melanoma Very common (≥10%):** papilloma including skin papilloma, hyperkeratosis and hyperkeratosis, keratoacanthoma, hyperglycaemia, headache, cough, nausea, vomiting, diarrhoea, constipation, skin effects (actinic keratosis, skin lesion, dry skin, erythema, pruritus), photosensitivity, influenza-like illness, LVEF decrease. **Uncommon (≥0.1 to <1%):** new primary melanoma, hypersensitivity, uveitis, panoretinitis, pannicula itis, retinal vein occlusion, retinal detachment, periorbital oedema, left ventricular dysfunction, cardiac failure, pneumonitis, interstitial lung disease, gastrointestinal perforation, colitis, pancreatitis, rhabdomyolysis, renal failure acute. **Adverse drug reactions in combination with Mekinist in Stage III melanoma following complete resection Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypertension, cough, nausea, diarrhoea, vomiting, abdominal pain, constipation, rash, dry skin, dermatitis acneiform, erythema, pruritus, arthralgia, myalgia, pain in extremity, muscle spasms, pyrexia, fatigue, chills, oedema peripheral, influenza-like illness, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** weight decreased, hypophosphataemia, hyperglycaemia, hypercalcaemia, hypoalbuminaemia, hypertriglyceridaemia, peripheral sensory neuropathy, dysgeusia, dizziness, syncope, dysarthria, somnolence, headache, dysgeusia, dry mouth, stomatitis, erythema, actinic keratosis, night sweats, hyperkeratosis, alopecia, palmar-plantar erythrodysesthesia syndrome, skin lesion, hyperhidrosis, skin fissures, paronychia, photosensitivity, myalgia, spasms, blood creatine phosphokinase increased, renal failure, mucosal inflammation, influenza-like illness, face oedema, blood alkaline phosphatase increased, gamma-glutamyltransferase increased. **Uncommon (≥0.1 to <1%):** rhabdomyolysis, renal failure. **Adverse drug reactions in combination with Tafinlar in pediatric patients (1 to <18 years of age) Very common (≥10%):** pyrexia, vomiting, diarrhoea, abdominal pain, rash, pruritus, arthralgia, myalgia, fatigue, oedema peripheral, influenza-like illness, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** papilloma including skin papilloma, keratoacanthoma, hyperglycaemia, headache, cough, nausea, vomiting, abdominal pain, constipation, skin effects (actinic keratosis, skin lesion, dry skin, erythema, pruritus), photosensitivity, influenza-like illness, LVEF decrease. **Uncommon (≥0.1 to <1%):** new primary melanoma, hypersensitivity, uveitis, panoretinitis, pannicula itis, retinal vein occlusion, retinal detachment, periorbital oedema, left ventricular dysfunction, cardiac failure, pneumonitis, interstitial lung disease, gastrointestinal perforation, colitis, pancreatitis, rhabdomyolysis, renal failure acute. **Adverse drug reactions in combination with Tafinlar in pediatric patients (1 to <18 years of age) Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypertension, nausea, vomiting, diarrhoea, decreased appetite, constipation, erythema, dry skin, rash, pruritus, hyperkeratosis and hyperkeratosis, actinic and seborrheic keratosis and keratosis pilaris, muscle spasms, arthralgia, myalgia, pyrexia, asthenia including fatigue and malaise, oedema (generalised and peripheral), chills, blood and urine phosphorus decreased, blood and urine phosphate increased, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** hypophosphataemia, hyponatraemia, detachment of retinal/retinal pigment epithelium, hypertension, rhabdomyolysis, ejection fraction decreased. **Adverse drug reactions in combination with Mekinist in pediatric patients (1 to <18 years of age) Very common (≥10%):** neutropenia, hyponatraemia, headache, dizziness, haemorrhage, hypotension, nausea, vomiting, diarrhoea, decreased appetite, constipation, erythema, dry skin, rash, pruritus, hyperkeratosis and hyperkeratosis, actinic and seborrheic keratosis and keratosis pilaris, muscle spasms, arthralgia, myalgia, pyrexia, asthenia including fatigue and malaise, oedema (generalised and peripheral), chills, blood and urine phosphorus decreased, blood and urine phosphate increased, alanine aminotransferase increased, aspartate aminotransferase increased. **Common (≥1 to <10%):** hypophosphataemia, hyponatraemia, detachment of retinal/retinal pigment epithelium, hypertension, pulmonary embolism, pancreatitis acute, renal failure, tubulointerstitial nephritis. **Adverse drug reactions in combination with Tafinlar monotherapy from post-marketing experience and pooled clinical trials Common (≥1 to <10%):** peripheral neuropathy. **Adverse drug reactions with Tafinlar in combination with Mekinist from post-marketing experience and pooled clinical trials Common (≥1 to <10%):** VTE, peripheral neuropathy, ulceration of the bowel. **Uncommon (≥0.1 to <1%):** sarcoidosis, burkitt lymphoma, bowel perforation, diverticulitis, GI perforation. **Adverse drug reactions with Tafinlar monotherapy from post-marketing experience and pooled clinical trials Uncommon (≥0.1 to <1%):** sarcoidosis, burkitt lymphoma, bowel perforation, diverticulitis, GI perforation. **Interactions:** ♦ Strong inhibitors or inducers of CYP2C8 or CYP3A4: Dose adjustment may be necessary, respectively. Tafinlar concentration: ♦ Alternative agents should be considered during administration of Tafinlar. ♦ Tafinlar inhibits OATP1B1 and OATP1B3. Therefore caution is recommended at co-administration of TAFINLAR and OATB1B1 or OATP1B3 substrates such as statins. ♦ Tafinlar is a substrate of CYP2C8 and CYP3A4. ♦ Caution should be exercised and additional monitoring of digoxin is recommended when digoxin is used concomitantly with TAFINLAR and at discontinuation of TAFINLAR. For complete interactions, consult full prescribing information. **Packs:** 28's pack. Not all pack sizes may be marketed. **Legal classification:** P1S1S1 **CDS Version:** 3.11 **Reference:** AUS 16 Oct 2025 **Revision date:** 7 Nov 2025

Please review the abbreviated Prescribing Information on this page for full approved indication wording before prescribing. For healthcare professionals only. Adverse events should be reported to Novartis Pharmaceuticals (HK) Limited

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Abbreviations

CT, Chemotherapy; TP, Paclitaxel + Cisplatin; NPC, Nasopharyngeal Carcinoma; ESCC, Esophageal Squamous Cell Carcinoma; OS, Overall Survival; NCCN, National Comprehensive Cancer Network; EMA, European Medicines Agency; PD-1, Programmed Cell Death Protein 1; 1L, First-Line.
*CT – gemcitabine and cisplatin

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A next-generation TKI for *ROS1+* NSCLC, approved for *NTRK* gene fusion+ solid tumors[1]

Recommended treatment option in ***NCCN***[2]***, ESMO***[3]***, ASCO Living Guidelines***[4] for **TKI-naive** and **TKI-pretreated** *ROS1+* NSCLC patients

35.7 months mPFS in **TKI-naive** *ROS1+* NSCLC patients[5]

9.0 months mPFS in **TKI-pretreated** *ROS1+* NSCLC patients[5]

Well-tolerated safety profile

Only 7% patients discontinued **AUGTYRO** due to adverse reactions[5]

References: 1. AUGTYRO® (Reprotrectinib) Capsules Hong Kong Prescribing Information. September 2025. **2.** NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Non-Small Cell Lung Cancer V.7.2026. **3.** Hendriks, L.E.L. et al. Updated treatment recommendations for systemic treatment: from the ESMO oncogene-addicted metastatic NSCLC living guideline. Annals of Oncology. 2025 June 24. **4.** Therapy for Stage IV Non-Small Cell Lung Cancer With Driver Alterations: ASCO Living Guideline, Version 2026.3.0. **5.**Dhillon A, Camidge DR, et al. TRIDENT-1 Investigators. Repotrectinib in ROS1 Fusion-Positive Non-Small-Cell Lung Cancer. N Engl J Med. 2024 Jan 11;390(2):118-131.

AUGTYRO® Capsules 40mg - Abbreviated Prescribing Information

Indications: Augtyro as monotherapy is indicated for the treatment of adult patients with ROS1-positive advanced non-small cell lung cancer (NSCLC). Augtyro is also indicated as monotherapy for the treatment of adult and paediatric patients 12 years of age and older with advanced solid tumours expressing an *NTRK* gene fusion who have received a prior NTRK inhibitor, or have not received a prior NTRK inhibitor and treatment options not targeting NTRK provide limited clinical benefit or have been exhausted. **Dosage and administration:** Patient selection should be confirmed by appropriate validated *ROS1* or *NTRK* testing. The recommended dosage is 160 mg taken orally once daily for 14 days, then increased to 160 mg twice daily until disease progression or unacceptable toxicity. Please refer to the full package insert for dose modifications for adverse reactions. Take Augtyro at approximately the same time each day with or without food. Swallow capsules whole: do not open, crush, chew or dissolve. If a dose is missed or vomiting occurs after a dose, resume the next scheduled dose without taking an extra dose. Avoid grapefruit, grapefruit juice and Seville oranges. **Warnings and precautions:** *Central Nervous System Adverse Reactions:* Augtyro can cause CNS adverse reactions including dizziness, ataxia and cognitive disorders. Advise patients not to drive or operate machinery if affected. Withhold and resume at same or reduced dose upon improvement, or permanently discontinue based on severity. *Interstitial Lung Disease (ILD)/Pneumonitis:* Augtyro can cause ILD/pneumonitis. Monitor for new or worsening pulmonary symptoms. Immediately withhold if suspected and permanently discontinue if confirmed. *Skeletal Fractures:* Skeletal fractures have occurred in adults and paediatric patients. Promptly evaluate patients with symptoms suggestive of fracture. *Hepatotoxicity:* Augtyro can cause hepatotoxicity. Monitor liver function tests including ALT, AST and bilirubin as clinically indicated. *Hepatic Impairment:* Augtyro should not be used in patients with moderate or severe hepatic impairment. *Contraception in female and male:* Augtyro may cause fetal harm. Women of childbearing potential should use highly effective contraception during treatment and for 2 months after the last dose. Augtyro may reduce the effectiveness of hormonal contraceptives. Male patients with female partners of childbearing potential should use condoms during treatment and for 4 months after the last dose. **Drug interactions:** Avoid concomitant use with strong or moderate CYP3A/P-gp inhibitors. Concomitant use may increase repotrectinib exposure and adverse reactions. Avoid grapefruit and grapefruit products. Avoid concomitant use with strong or moderate CYP3A/P-gp inducers as these may reduce efficacy. Co administration of Augtyro with strong or moderate CYP3A4 or P-gp inducers decreases repotrectinib plasma concentrations and should thus be avoided. Repotrectinib is a moderate CYP3A4 inducer, which can decrease progestin or oestrogen exposure to an extent that could reduce the effectiveness of systemically acting hormonal contraceptives including oral contraceptives. **Undesirable effects:** The most common adverse reactions in adults were dizziness, dysgeusia, constipation, paraesthesia, anaemia and dyspnoea. The most common serious adverse reactions were pneumonia, dyspnoea, pleural effusion, pyrexia, muscular weakness, anaemia and pneumonitis. Grade ≥3 adverse reactions occurred in 43% of patients. The most frequent Grade ≥3 laboratory abnormalities included anaemia, dyspnoea, pneumonia, increased creatine phosphokinase, weight increased, increased AST, pleural effusion and decreased neutrophil count.
Please refer to the full prescribing information before prescribing.
Ref: HKPI version September 2025

ZMHKHP20260901002 Expire Date 2027/09/01

For academic reference only by medical and healthcare professionals.

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AUGTYRO® (repotrectinib)

Notes

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KEYTRUDA® (pembrolizumab) injection 100 mg

KEYNOTE-A39

KEYTRUDA® is the FIRST AND ONLY immunotherapy approved in combination with an ADC for the treatment of LA/mUC in Hong Kong[1,2]



KEYTRUDA® (pembrolizumab), in combination with enfortumab vedotin (EV), is indicated for the first-line treatment of unresectable or metastatic urothelial carcinoma in adults.

KEYTRUDA® + EV delivered nearly 2X benefit across 3 endpoints vs. chemotherapy in patients with LA/mUC[3]

At a median follow-up of 17.2 months:[3]

SUPERIOR EFFICACY

OS
Nearly 2X mOS
KEYTRUDA® + EV 31.5 mo vs. Chemotherapy 16.1 mo
(HR=0.47; 95% CI: 0.38-0.58; *p*<0.001)

PFS
Nearly 2X mPFS
KEYTRUDA® + EV 12.5 mo vs. Chemotherapy 6.3 mo
(HR=0.45; 95% CI: 0.38-0.54; *p*<0.001)

CR
Nearly 2X CR
KEYTRUDA® + EV 29.1% n=127/437 vs. Chemotherapy 12.5% n=55/441

The confirmed overall response was higher in the KEYTRUDA® + EV group than in the chemotherapy group (67.7% [95% CI: 63.1-72.1] vs. 44.4% [95% CI: 39.7-49.2]; *p*<0.001)

TRAE ≥Grade 3 occurred in 55.9% of the patients in the KEYTRUDA® + EV group and in 69.5% of those in the chemotherapy group[3]

- The most common TRAE ≥Grade 3 in KEYTRUDA® + EV group were maculopapular rash (7.7%), hyperglycemia (5.0%), and neutropenia (4.8%). The most common TRAE ≥Grade 3 in chemotherapy group were anemia (31.4%), neutropenia (30.0%), and thrombocytopenia (19.4%)[3]

Abbreviations: ADC: Antibody-drug conjugate; AEs: Adverse events; CR: Complete response; EV: Enfortumab vedotin; IV: Intravenous; LA/mUC: Locally advanced or metastatic urothelial carcinoma; mo: Months; mOS: Median overall survival; mPFS: Median progression-free survival; OS: Overall survival; PFS: Progression-free survival; Q3W: Every 3 weeks; TRAE: Treatment-related adverse event.

References: 1. Astellas Pharma Inc. PADCEV (enfortumab vedotin-ejfv) with KEYTRUDA (pembrolizumab) approved by FDA as the first and only ADC plus PD-1 to treat advanced bladder cancer (Press release). 2023. Available from: https://www.prnewswire.com/news-releases/padcev-enfortumab-vedotin-ejfv-with-keytruda-pembrolizumab-approved-by-fda-as-the-first-and-only-adc-plus-pd-1-to-treat-advanced-bladder-cancer-302017114.html. Accessed Dec. 9, 2025. **2.** KEYTRUDA® (pembrolizumab) Solution for Injection 100mg/4mL. Hong Kong Package Insert. Merck Sharp & Dohme. **3.** Powles T, et al. Enfortumab vedotin and pembrolizumab in untreated advanced urothelial cancer. *New Engl J Med.* 2024;390(10):875-88.

Selected Safety Information for KEYTRUDA (pembrolizumab): Contraindications: None. **Precautions:** •Immune-mediated pneumonitis •Immune-mediated colitis •Immune-mediated hepatitis and hepatotoxicity •Immune-mediated endocrinopathies •Immune-mediated nephritis and renal dysfunction •Immune-mediated Dermatologic Adverse Reactions •Other immune-mediated adverse reactions •Infusion-related reactions (including hypersensitivity and anaphylaxis) •Complications of allogeneic HSCT in patients after or prior to treatment with KEYTRUDA treatment •Increased mortality in patients with multiple myeloma when KEYTRUDA is added to a thalidomide analogue and dexamethasone •Embryo-fetal toxicity. **Adverse Events:** Most common adverse reactions (reported in ≥20% of patients) were: •KEYTRUDA as a single agent: fatigue, musculoskeletal pain, rash, diarrhea, pyrexia, cough, decreased appetite, pruritus, dyspnea, constipation, pain, abdominal pain, nausea and hypothyroidism; •KEYTRUDA in combination with chemotherapy or chemoradiotherapy: fatigue/asthenia, nausea, constipation, diarrhea, decreased appetite, rash, vomiting, cough, dyspnea, pyrexia, alopecia, peripheral neuropathy, mucosal inflammation, stomatitis, headache, weight loss, abdominal pain, arthralgia, myalgia, insomnia, palmar-plantar erythrodysesthesia, urinary tract infection, and hypothyroidism; •KEYTRUDA in combination with chemotherapy and bevacizumab: peripheral neuropathy, alopecia, anemia, fatigue/asthenia, nausea, neutropenia, diarrhea, hypertension, thrombocytopenia, constipation, arthralgia, vomiting, urinary tract infection, rash, leukopenia, hypothyroidism, and decreased appetite; •KEYTRUDA in combination with axitinib: diarrhea, fatigue/asthenia, hypertension, hepatotoxicity, hypothyroidism, decreased appetite, palmar-plantar erythrodysesthesia, nausea, stomatitis/mucosal inflammation, dysphonia, rash, cough, and constipation; •KEYTRUDA in combination with lenvatinib: hypothyroidism, hypertension, fatigue, diarrhea, musculoskeletal disorder, nausea, decreased appetite, vomiting, stomatitis, weight loss, abdominal pain, urinary tract infection, proteinuria, constipation, headache, hemorrhagic events, palmar-plantar erythrodysesthesia, dysphonia, rash, hepatotoxicity, and acute kidney injury; •KEYTRUDA in combination with enfortumab vedotin: rash, peripheral neuropathy, fatigue, pruritus, diarrhea, alopecia, weight loss, decreased appetite, dry eye, nausea, constipation, dysgeusia, and urinary tract infection. **For detailed precautions and adverse events, please consult the full prescribing information.** HK-KEY-01117 Nov/2025

MSD

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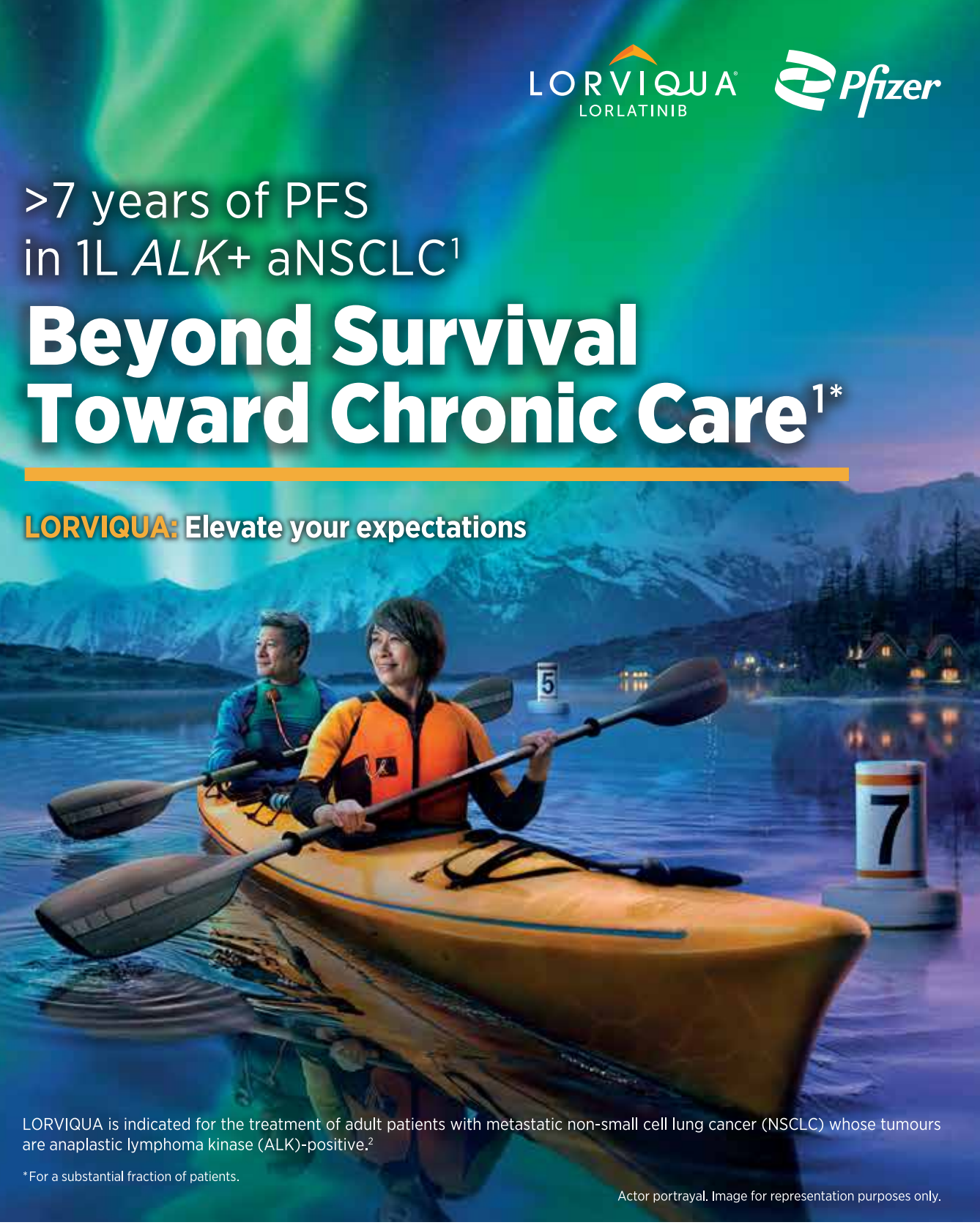
LORVIQUA®
LORLATINIB

Pfizer

>7 years of PFS in 1L *ALK*+ aNSCLC[1]

Beyond Survival Toward Chronic Care[1]*

LORVIQUA: Elevate your expectations



LORVIQUA is indicated for the treatment of adult patients with metastatic non-small cell lung cancer (NSCLC) whose tumours are anaplastic lymphoma kinase (ALK)-positive.[2]

*For a substantial fraction of patients.

Actor portrayal. Image for representation purposes only.

References: 1. Shaw AT, et al. *Ann Oncol*. 2026; doi: https://doi.org/10.1016/j.annonc.2026.05.692. **2.** LORVIQUA® Prescribing Information. Pfizer Corporation Hong Kong Limited. Version February 2022.

Pfizer

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The QR code/URL links to the latest Prescribing Information approved by the Department of Health in Hong Kong and may not be effective and the same as presented in the actual product package.

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