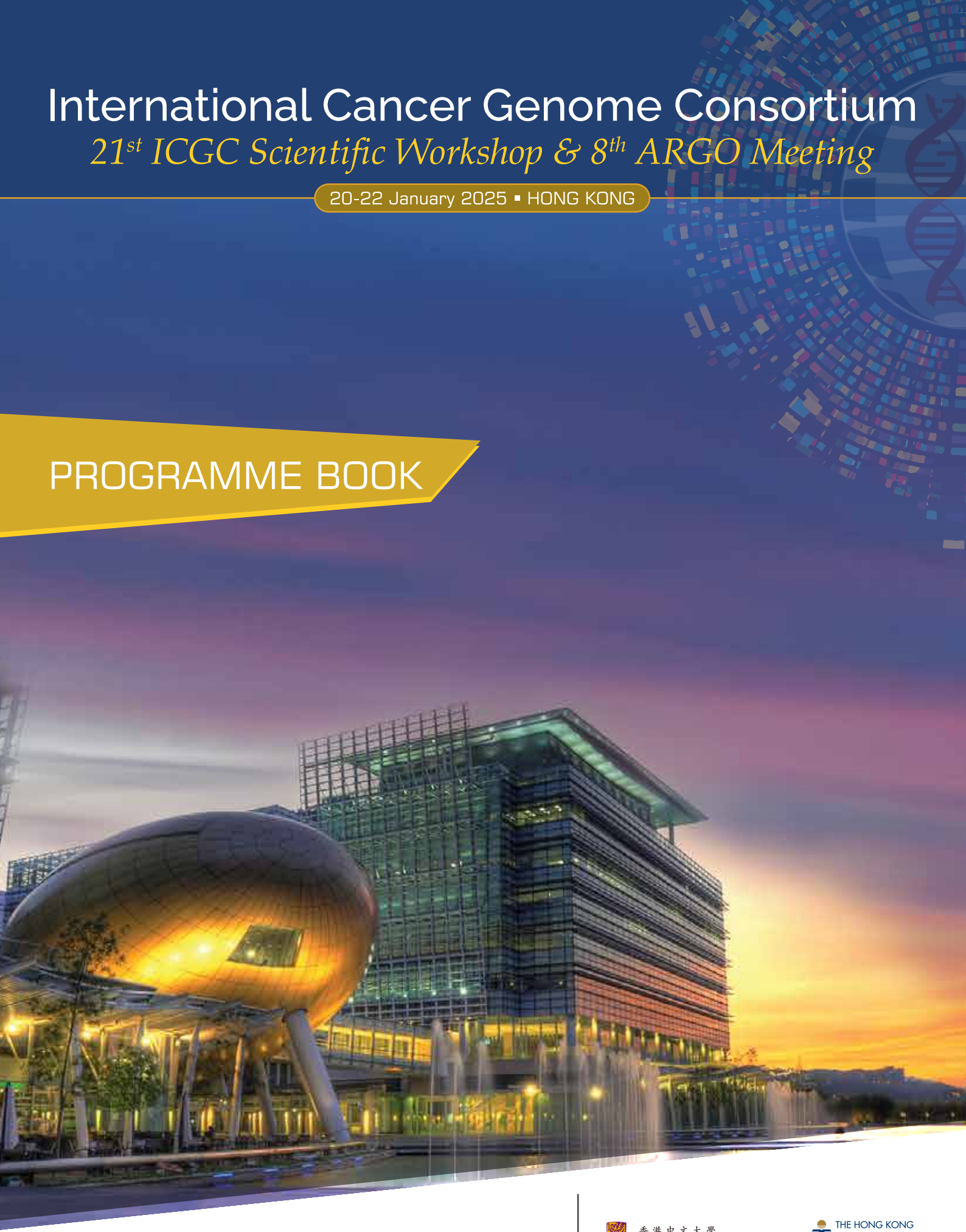
International Cancer Genome Consortium

21st ICGC Scientific Workshop & 8th ARGO Meeting

20-22 January 2025 ▪ HONG KONG

PROGRAMME BOOK



ICGC International Cancer Genome Consortium

ARGO Accelerating Research in Genomic Oncology

香港中文大學 The Chinese University of Hong Kong

THE HONG KONG UNIVERSITY OF SCIENCE AND TECHNOLOGY

HKSTP

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WELCOME MESSAGE

Dear Colleagues,

We are delighted to announce the taking place on 20-22 January 2025 of
The 21st Scientific Workshop of the International Cancer Genome Consortium (ICGC), and
The 8th Meeting of Accelerating Research in Genomic Oncology (ARGO)
at the Hong Kong Science Park, a welcoming innovation park in the heart of the New Territories.

Although genomic work has always been the core business of our research universities and the two medical schools for decades, the Hong Kong Genome Institute was only established in Science Park recently in 2021. It is therefore timely and relevant for us to have the coming ICGC Events in the ionic "MICE" (Meeting, Incentives, Conferences & Exhibitions) venues of the Hong Kong Science Park.

It is a "by invitation" internal meeting of ICGC, but we welcome our local workers and students of the cancer genome to attend the educational plenaries, keynote speeches, insights and updates. While members of the ICGC are engaged seriously with their internal committee meetings, workers of the field are encouraged to enjoy the meticulously prepared posters.

The environment of the Science Park Conference Centre encourages in-depth discussion and the development of close collaboration. We welcome and value your physical presence, looking forward to seeing all of you there.

Andrew V. BIANKIN
International Cancer Genome Consortium (ICGC)

Wai S. POON
Division of Neurosurgery
Department of Surgery
The Chinese University of Hong Kong

Hannah H. XUE
Division of Life Science
The Hong Kong University of Science and Technology

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ORGANISATION

Organiser

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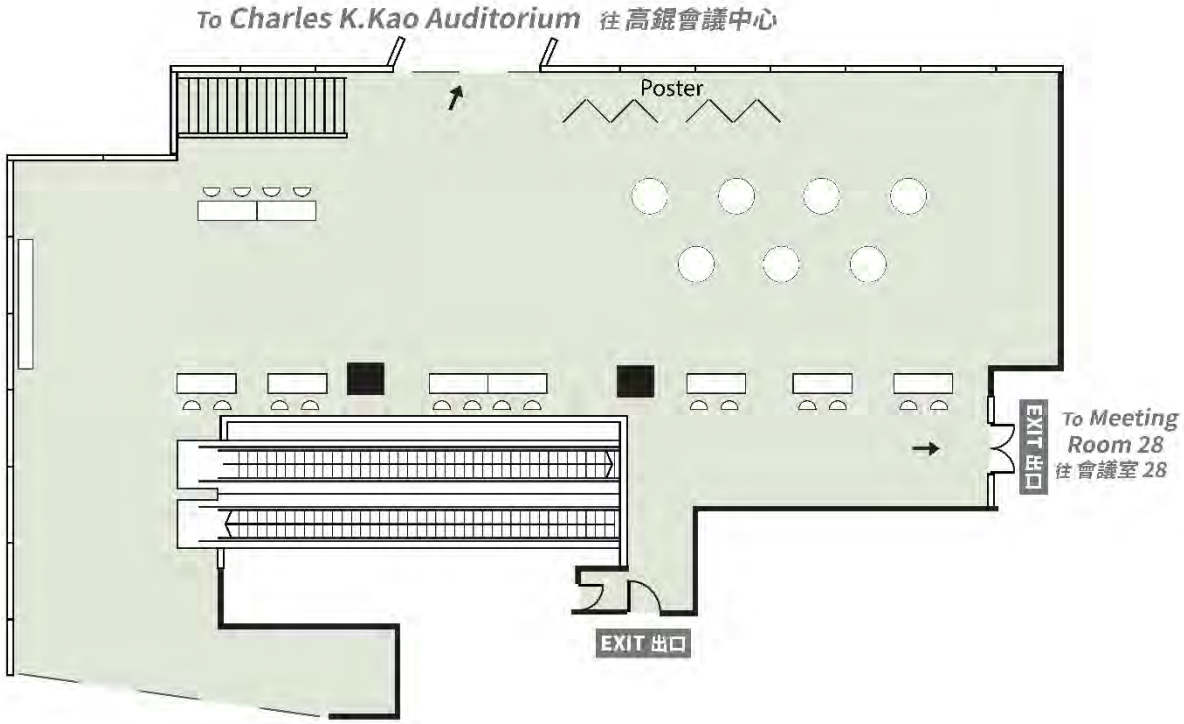
VENUE FLOOR PLAN



SCIENTIFIC PROGRAMME

All communicated times in the Scientific Programme are Hong Kong Time. Hong Kong is 8 hours ahead of Greenwich Mean Time (i.e. GMT+8 hours).

	20 Jan 2025 (Mon) Day 1	21 Jan 2025 (Tue) Day 2	22 Jan 222025 (Wed) Day 3
0830-0900	Registration & Putting up Posters	Registration & Putting up Posters	Registration & Putting up Posters
0900-0930	Welcome Address	Keynote Presentations from Patients and Their Families	**Real World Precision Oncology**
0930-1000	**Keynote Lecture I**		
1000-1030	*Coffee Break* *Poster & Exhibition*	Genome Project II	
1030-1100	ICGC-ARGO Vision & Update		*Coffee Break* *Poster & Exhibition*
1100-1130		*Coffee Break* *Poster & Exhibition*	Group Discussion - ICGC Current & Future Strategy
1130-1200		Understanding Today's Cancer Genomics	
1200-1230	*Lunch* *Poster & Exhibition*		Summary & Closing
1230-1300		*Lunch* *Poster & Exhibition* *(Executive Committee & Programme Leads Meeting 13:00-14:00)*	
1300-1330	ICGC-ARGO Programme Updates		
1330-1400			
1400-1430			
1430-1500	*Coffee Break* *Poster & Exhibition*	ICGC AGRO Projects	
1500-1530	Genome Projects I		
1530-1600		*Coffee Break* *Poster & Exhibition*	
1600-1630	**Keynote Lecture II**	**Panel Discussion – AI for Precision Oncology**	
1630-1700			
1700-1730	*Welcome Reception*	Tour of HKSP	
1730-1800			
1800-1830		*Scientific Workshop Dinner*	
1830-2100			

SCIENTIFIC PROGRAMME

Day 1: 20 January 2025 (Monday)

08:30 – 09:00	Registration and Poster Set-up	Pre-function Hall of Charles K. Kao Auditorium

Opening		**Charles K. Kao Auditorium**
MC : Wai Sang POON		
09:00 – 09:30	**Welcome by the Organiser** *Andrew BIANKIN* *Executive Director, International Cancer Genome Consortium (ICGC)*	
	Welcome Speech *Chung-mau LO, BBS, JP* *Secretary for Health, Government of the Hong Kong Special Administrative Region* *Chair Professor of Hepatobiliary and Pancreatic Surgery, The University of Hong Kong*	
	Welcome Speech *Dennis Y.M. LO, JP* *Vice-Chancellor and President, Li Ka Shing Professor of Medicine & Professor of Chemical Pathology, CUHK*	
	Welcome Speech *Gracie NG* *Chief of Staff and Acting Chief Corporate Development Officer, Hong Kong Science & Technology Parks Corporation*	
	Group Photograph	
Keynote Lecture I		**Charles K. Kao Auditorium**
Chairpersons : Danny T.M. CHAN		
09:30 – 10:05	**Circulating DNA in Cancer Diagnosis** *Dennis Y.M. LO, JP* *Vice-Chancellor and President, Li Ka Shing Professor of Medicine & Professor of Chemical Pathology, CUHK*	
10:05 - 10:40	*Break, Exhibition & Poster Viewing*	*Pre-function Hall*
ICGC-ARGO Vision and Update		**Charles K. Kao Auditorium**
10:40 - 12:00	*Andrew BIANKIN, Executive Director of ICGC* *Amber JOHNS, Project Developer of ICGC* *Lincoln Stein, Management Committee Member of ICGC* *Melanie COURTOT, Member of ICGC*	
12:00 - 13:00	*Lunch, Exhibition & Poster Viewing*	*Pre-function Hall*

SCIENTIFIC PROGRAMME

Day 1: 20 January 2025 (Monday)

ICGC-ARGO Programme Updates	**Charles K. Kao Auditorium**
Chairperson : Takayuki YOSHINO	
13:00 – 14:45	**ICGC ARGO Clinical Data Dictionary** *Linda XIANG* *Ontario Institute for Cancer Research, Toronto, Canada*
	Deciphering Clinical Signatures of Colorectal Cancer through Multi-Omics Big Data *Kui WU* *BGI Genomics, Shenzhen, China*
	The Pan Prostate Cancer Group – An Overview *Colin COOPER* *University of East Anglia, Norwich, UK*
	The Mutographs Project: Achievements and Expectations on Enhancing Our Understanding of Causes on Cancer *Sandra PERDAMO* *International Agency for Research on Cancer, Lyon, France*
	National Genomics Data Center of China *Jianmin WU[1] & Yiming BAO[2]* *[1]Center for Cancer Bioinformatics. Peking University Cancer Hospital & Institute, Beijing, China* *[2]National Genomics Data Center, Beijing Institute of Genomics, Chinese Academy of Science, Beijing, China*
14:45 - 15:15	*Break, Exhibition & Poster Viewing* — *Pre-function Hall*
Genome Projects I	**Charles K. Kao Auditorium**
Chairperson : Jianmin WU	
15:15 - 16:15	**TGFB1 Induced TET3 Dependent Regulation of OTX2 Super Enhancer Hypomethylation Promotes Group 3 medulloblastoma Progression** *Ziwei WANG* *Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong*
	A Series Cases of Lhermitte-Duclos Disease with Surgical Interference *Ziyang CHEN* *Guangdong Sanjiu Brain hospital, Guangzhou, China*
	Comprehensive Genomic and Transcriptomic Characterization of Glioblastoma Reveals the Impact of Extrachromosomal DNA on Tumor Heterogeneity and Therapeutic Vulnerabilities *Aya EL HELALI* *Department of Clinical Oncology, School of Clinical Medicine, The University of Hong Kong, Hong Kong*

SCIENTIFIC PROGRAMME

Day 1: 20 January 2025 (Monday)

Keynote Lecture	**Charles K. Kao Auditorium**
Chairperson : Wai S. POON	
16:15– 16:45	**Population Cancer Genomics Through the Lens of The Hong Kong Genome Project** *Dr Brian H.Y. CHUNG & Dr Su Vui LO* *Hong Kong Genome Institute, Hong Kong*
16:45 - 18:15	*Welcome Reception*

SCIENTIFIC PROGRAMME

Day 2: 21 January 2025 (Tuesday)

08:30 – 09:00	Registration and Poster Set-up	Pre-function Hall of Charles K. Kao Auditorium

Keynote Presentations from Patients and Their Families		**Charles K. Kao Auditorium**
Chairperson : Amber JOHNS		
09:00 – 09:45	**Establishing a Brain Tumour Centre for Otto** *Jennifer CHEN*	
	To Accompany Woody in His Last Journey *Snow LI*	
	Dual Citizenship, in The Kingdom of The Well and The Sick *Charissa Ann CHAU & Antoine d'HAUSSY*	
Genome Projects II		**Charles K. Kao Auditorium**
Chairperson : Peter Y.M. WOO		
09:45 – 11:00	**Tyrosine Kinase Inhibitor Could Reconstruct Immune Microenvironment in Lung Cancer Brain Metastasis *via* CTLA4** *Wei HUA* *Huashan Hospital, Fudan University, Shanghai, China*	
	Stromal Architecture and Fibroblast Subpopulations with Opposing Effects on Outcomes in Hepatocellular Carcinoma *Xiaofang CHEN* *College of Life Sciences, University of Chinese Academy of Sciences, Beijing, China*	
	Local Cranial Radiation Combined with Third-generation TKIs Improve Leptomeningeal Metastasis Disease-free Survival and Reduce Leptomeningeal Metastasis Rate in Patients with EGFR-mutated NSCLC and Brain Metastasis *Qian WANG* *Guangdong Sanjiu Brain Hospital, Guangzhou, China*	
	Forward and Backward Evolution of Cancer Cells *Hannah H. XUE[1] & Wai S. POON[2,3]* *[1]Division of Life Science, Hong Kong University of Science and Technology, [2]Department of Surgery, The Chinese University of Hong Kong, [3] Neuromedicine Center, The University of Hong Kong- Shenzhen Hospital*	
	Spatially Visualised Proteomics Platforms and Standardisation *Peng XIA* *Hong Kong Bayomics Biotechnology Co.*	
11:00 - 11:30	Break, Exhibition & Poster Viewing	Pre-function Hall

SCIENTIFIC PROGRAMME

Day 2: 21 January 2025 (Tuesday)

Understanding Today's Cancer Genomics		**Charles K. Kao Auditorium**
Chairperson : Hannah H. XUE		
11:30 – 12:30	**Investigation of Glioma Evolution for Precision Neuro-oncology** *Jiguang WANG* *Division of Life Science, Department of Chemical and Biological Engineering, Hong Kong University of Science and Technology, Hong Kong*	
	Spatiotemporal Genomics of Human Medulloblastoma at Single-nucleus Resolution *Ziwei WANG* *Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong*	
	Survival Impact and Cost-Effectiveness of Precision Oncology: Health System Evidence from British Columbia, Canada *Dean A. REGIER* *Regulatory Science Lab, BC Cancer Research Institute & School of Population and Public Health, Faculty of Medicine, University of British Columbia, Vancouver, Canada*	
12:30 - 14:30	*Lunch & Poster Viewing*	*Pre-function Hall*
	Executive Committee and Programme Leads Meeting (13:00-14:00)	*Meeting Room 24*
ICGC ARGO Projects		**Charles K. Kao Auditorium**
Chairperson : Colin COOPER		
14:30 – 15:30	**Landscape of ADC Target Gene Expression and Their Association with Efficacy of ADC in Advanced Solid Tumors: SCRUM-Japan MONSTAR-SCREEN-2** *Takao FUJISAWA* *Department for The Promotion of Drug and Diagnostic Development, National Cancer Center Hospital East, Kashiwa, Japan*	
	Unlocking Germline and Somatic Variations in Lung Cancer Brain Metastasis *Hannah H. XUE* *Division of Life Science, Hong Kong University of Science and Technology, Hong Kong*	
	Organoid Models of Gastric Cancer (Invited Lecture) *Suet Yi LEUNG* *Department of Pathology, School of Clinical Medicine, The University of Hong Kong, Hong Kong*	
15:30 - 16:00	Break, Exhibition & Poster Viewing	Pre-function Hall
Panel Discussion		**Charles K. Kao Auditorium**
Moderator: Andrew BIANKIN		
16:00 – 17:00	**Artificial Intelligence for Precision Oncology: What Data Do We Really Need**	
17:00 - 18:00	**Tour of Hong Kong Science Park**	
18:00 - 21:00	**Scientific Workshop Dinner**	

SCIENTIFIC PROGRAMME

Day 3: 22 January 2025 (Wednesday)

08:30 – 09:00	Registration	Pre-function Hall

Real World Precision Oncology	**Charles K. Kao Auditorium**
Chairperson : Melanie COURTOT	
09:00 – 10:30	**Ensuring Equity in Genomics: WHO Guidance on Human Genome Data Collection, Access, Use, and Sharing and its Implications for the ICGC-ARGO Project** *Kazuto KATO* *Graduate School of Medicine, Osaka University, Suita, Japan*
	Real World Experience of Utilizing Formalin Fixed Paraffin Embedded Tissue (FFPE) in Whole Genome and Transcriptome Sequencing for Personalized Oncogenomics *Melissa K. MCCONECHY* *Canada's Michael Smith Genome Sciences Centre, Vancouver, Canada*
	Real-World Data for Precision Oncology: Validation of Large Language Models for Automatic Data Extraction from Electronic Health Records *Deirdre WEYMANN[1,2]* *[1]Regulatory Science Lab, BC Cancer Research Institute, Vancouver, Canada* *[2]Faculty of Health Sciences, Simon Fraser University, Burnaby, Canada*
	Personalized OncoGenomics (POG): Advancing Precision Medicine in British Columbia *Howard J. LIM* *Division of Medical Oncology, BC Cancer – Vancouver, Vancouver, Canada*
10:30 - 11:00	Break, Exhibition & Poster Viewing — Pre-function Hall
Group Discussion	**Charles K. Kao Auditorium**
Moderators : Andrew BIANKIN, Wai S POON, Hannah H. XUE, Takayuki YOSHINO	
11:00 – 12:00	**ICGC-ARGO Current and Future Strategy** To involve all ICGC members for in-depth dialogue and structured discussion
	Summary and Closing *Andrew BIANKIN, ICGC*

ABSTRACTS

ARGO Clinical Data Dictionary

Hardeep Nahal-Bose[1], Peter Lichter[2], Ursula Weber[2], Linda Xiang[1], Edmund Su[1], Melanie Courtot[1], ICGC ARGO Tissue & Clinical Annotation Working Group
[1]Ontario Institute for Cancer Research
[2]German Cancer Research Center

Background and Objective

The International Cancer Genome Consortium Accelerating Research in Genomic Oncology (ICGC-ARGO) is a global initiative to sequence the germline and tumor genomes of 100,000 cancer patients across 13 countries and 22 tumor types. By integrating genomic data with comprehensive clinical information including treatment outcomes, lifestyle factors, environmental exposures, and family history, ICGC-ARGO addresses a diverse spectrum of cancers. This integration aims to accelerate the translation of genomic insights into clinical applications, driving advancements in diagnosis, treatment, early detection, and prevention. However, one of the project's significant challenges is harmonizing extensive clinical datasets from various tumor types and across international programs.

Method and Results

To address this, the ICGC-ARGO Clinical Data Dictionary was collaboratively developed by the Ontario Institute for Cancer Research (OICR) Data Coordination Centre and the ICGC-ARGO Tissue & Clinical Annotation Working Group. Accessible at https://docs.icgc-argo.org/dictionary, the dictionary specifies a minimal set of clinical fields required for submission, ensuring consistent and high-quality data collection. It employs an event-based data model to capture relationships between clinical events and enables longitudinal data collection. Grounded in common data elements (CDEs) and international standardized terminology, the model comprises 15 schemas, including 6 core schemas: Sample Registration, Donor, Specimen, Primary Diagnosis, Treatment, and Follow Up. There are also 5 schemas detailing specific treatments such as Chemotherapy, Immunotherapy, Surgery, Radiation, and Hormone Therapy. Additionally, 4 optional schemas capture extended clinical variables, including exposure, family history, biomarkers, and comorbidities. The data model consists of 53 ID fields, 79 core fields, and 112 extended fields, with each clinical field is defined by a data tier and an attribute classification, reflecting the importance of the field in terms of clinical data completeness. Validation rules are enforced to ensure data integrity and correctness, while interoperable with other data standards, such as mCODE/FHIR (Minimal Common Oncology Data Elements) to enhance compatibility.

Conclusion

As a comprehensive clinical data model, the ICGC ARGO Data Dictionary enables the collection of high-quality clinical data linked to genomic data, facilitating robust analyses and addressing key clinical questions in cancer research. Its further adoption by international initiatives, such as the European-Canadian Cancer Network (EuCanCan) and the Marathon of Hope Cancer Centres Network (MOHCCN), highlights its broad applicability, reusability, and impact on advancing precision oncology research.

ABSTRACTS

Deciphering Clinical Signatures of Colorectal Cancer through Multi-Omics Big Data

Cong Lin[1,2,3,#], Huanzi Zhong[1,4,#], Fuqiang Li[1,3,4,#], Zhun Shi[1,4,#], Meizhen Wu[1], Huahui Ren[1,4], Tian Luo[1], Fangming Yang[1,4], Ting Zhu[1,2], Luís Nunes[5], Shida Zhu[1,4], Huanming Yang[2], Bengt Glimelius[5], Tobias Sjöblom[5], **Kui Wu**[1,2,3,4,*]

[1]Institute of Intelligent Medical Research (IIMR), BGI Genomics, Shenzhen 518083, China
[2]HIM-BGI Omics Center, Zhejiang Cancer Hospital, Hangzhou Institute of Medicine (HIM), Chinese Academy of Sciences, Hangzhou 310000, China
[3]Guangdong Provincial Key Laboratory of Human Disease Genomics, Shenzhen Key Laboratory of Genomics, BGI Research, Shenzhen 518083, China
[4]BGI Genomics, Harbin 150023, Heilongjiang, China
[5]Department of Immunology, Genetics and Pathology, Science for Life Laboratory, Uppsala University, Uppsala, Sweden

Background: Colorectal cancer (CRC) is the third most prevalent cancer and the second leading cause of cancer-related mortality worldwide. Precision medicine of CRC requires understanding of comprehensive molecular characteristics including intratissue microbiota.

Objective: To advance the understanding of CRC pathogenesis, identify driver events and find prognostic features, as well as deepening the understanding of intratissue microbiota in CRC and its complex interplay with tumor genomic alterations and prognosis.

Methods: We obtained high-quality whole-genome sequences (average 53X) from patient-matched tumour and unaffected control samples along with tumour transcriptome sequence (average 30M paired reads) from 1,063 CRC. Of the 1,063 CRCs, 943 were primary tumour surgical specimens and 120 were primary tumour biopsies. Mutational signatures and driver genes were identified through whole genome analysis. Evolutional timing of mutated genes was analyzed to determine the sequence of genomic events during CRC carcinogenesis. We then performed integrated analyses of gene mutations and gene expressions to further understand the effect of alterations on molecular function. Associating with high quality clinical information, we then attempted to determine prognostic mutation and gene expression signatures in CRC. From high-depth WGS data, high-quality non-human reads were extracted and mapped against the Unified Human Gastrointestinal Genome (UHGG) catalog, along with comprehensive clinical follow-up, we performed a thorough profiling of the presence and relevance of tissue-resident microbiota in primary CRC.

Results: On the basis of the total mutation count, CRC tumours were divided into hypermutated (HM) group with >23.16 mutations per megabase and non-hypermutated (nHM) group. We identified 96 mutated driver genes, 9 were not previously implicated in colorectal cancer and 24 had not been linked to any cancer. Two distinct patterns of pathway co-mutations were observed, timing analyses identified nine early and three late driver gene mutations, and several signatures of colorectal-cancer-specific mutational processes were identified. Mutations in WNT, EGFR and TGFβ pathway genes, the mitochondrial CYB gene and 3 regulatory elements along with 21 copy-number variations and the COSMIC SBS44 signature correlated with survival.

ABSTRACTS

Gene expression classification yielded five prognostic subtypes with distinct molecular features, in part explained by underlying genomic alterations. Microsatellite-instable tumours were divided into two classes with different levels of hypoxia and infiltration of immune and stromal cells. Distinct microbiome enrichment patterns between right- and left-sided colon were observed in both tumor and normal adjacent tissues (NATs). A set of stable microbial features was identified in hypermutated tumors, with multiple taxa demonstrating correlations with mutations in host driver genes and DNA damage repair genes. *Fusobacterium* species were prognostic in patients with consensus molecular subtype (CMS) 4 tumors, while pks- island or elevated *Enterobacteriaceae* levels correlated with unfavorable prognosis specifically in patients with CMS2 tumors. In NATs, high *Akkermansia* levels were associated with poorer survival. Finally, risk scores based on tumor or NAT resident taxa could predict CRC prognosis independent of established clinical factors. The prognostic taxa were closely associated with pathways involved in CRC oncogenesis, including hypoxia, immunological and metabolism status.

Conclusions: This study constitutes the largest integrated genome and transcriptome analysis of colorectal cancer, and interlinks mutations, gene expression, intratissue microbiota and patient outcomes. The identification of prognostic mutations and expression subtypes as well as intratissue microbiota can guide future efforts to individualize colorectal cancer therapy.

ABSTRACTS

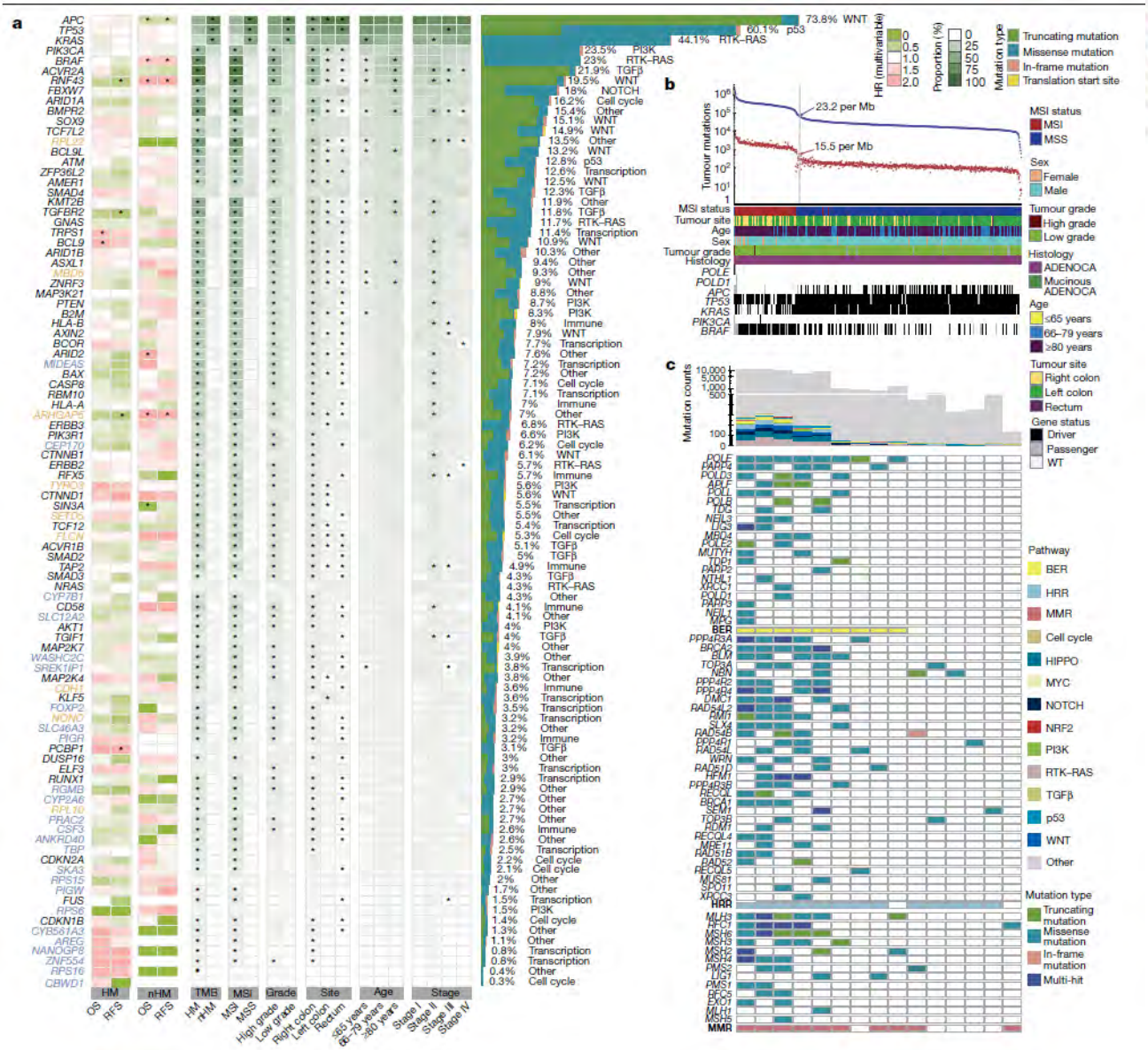


Figure 1: Mutational landscape of 1,063 CRC genomes

ABSTRACTS

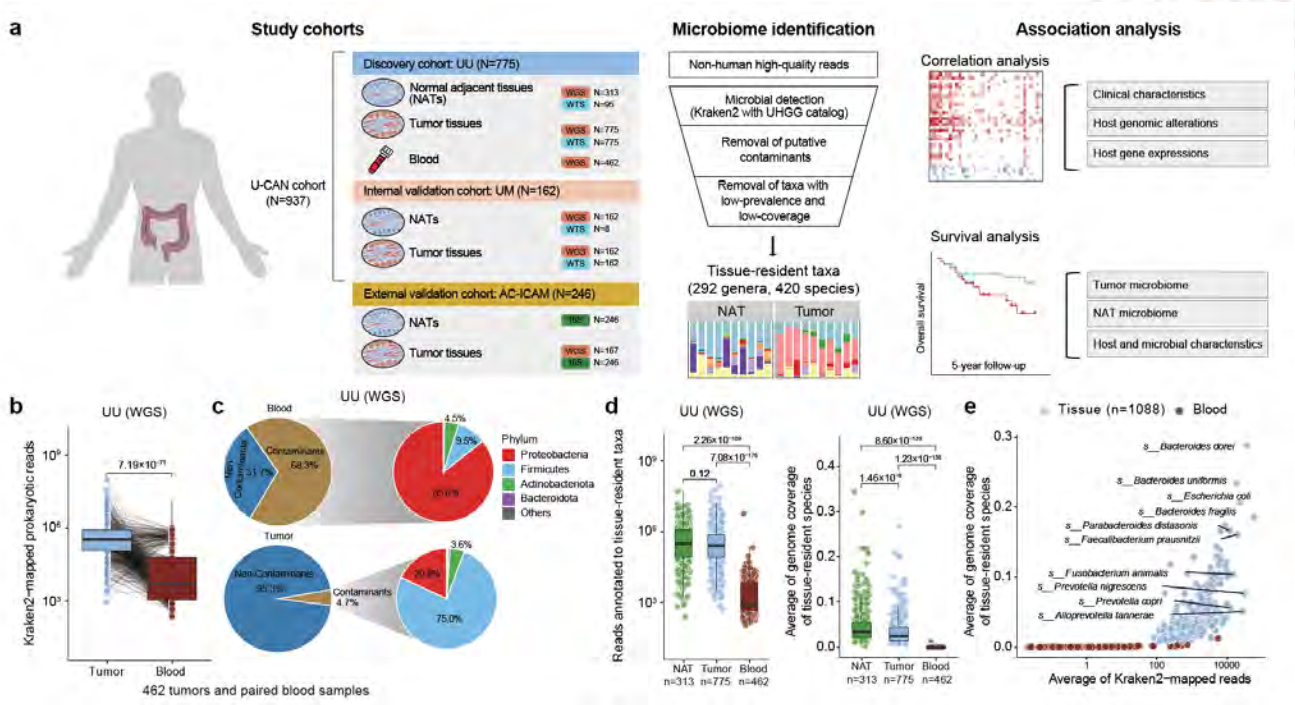


Figure 2: Identification and validation of tissue-resident microbiota in CRC patients

ABSTRACTS

The Pan Prostate Cancer Group – An Overview.

Colin Cooper[1], David Wedge[2] & Ros Eeles[3] on behalf of the Pan Prostate Cancer Group (PPCG) Steering Committee.
[1]University of East Anglia, UK, [2]Manchester Cancer Research Centre, University of Manchester, UK, [3]Institute of Cancer Research, London, UK

The PPCG (www.panprostate.org) involves researchers from the UK, Finland, Germany, Denmark, France, Australia, USA, Canada, and South Africa. Although predominantly a core community of around 40 bioinformatics experts, the PPCG also includes molecular biologists, mathematicians, genetic anthropologists, histopathologists, trials experts, oncologists, and surgeons. Its structure comprises technical (PP-COMP), pathology (PP-PATH) and clinical data/ethics (PP-CLIN) working groups, all reporting to the PPCG Steering Committee. Harmonised analyses of 'omics data (including WGS, RNA-Seq and methylome data) from 2021 clinically well-annotated unique donors (including from early onset, and metastatic disease) have been completed, with processing through 45+ pipelines.

Integrated data from the PP-COMP, PP-CLIN and PP-PATH working groups then became available for analysis. To contribute to the analysis of data, participants and collaborators complete a Concept Form, outlining a research project that they intend to carry out using PPCG data. Over 70 interlinked proposals have been received and approved to date. To allow coordination of the research process within the consortium these proposals have each been assigned to one of 12 Analysis Groups. Leads of each Analysis group are tasked with (i) synthesising and crafting the projects for allocated Concept Forms; (ii) linking to other themes; (iii) developing PPCG projects that will lead to clinical benefit and publications and (iv) setting up links as appropriate to clinical trials.

Utilising WGS data, linked to pathology and clinical data, the analysis working groups within PPCG are making novel discoveries. Prior to this data release, we reported the association of rare germline variants with rapid biochemical recurrence (Burns *et al*. 2022). Concurrent with the release of PPCG data (planning for late 2025), we intend to craft a collection of papers describing the mutational processes affecting prostate cancer, the process of evolution of this disease, delineate the role of hypoxia in driving aggressive prostate cancer, characterise the landscape of genetic variation in mitochondria and in nuclear genomes; provide evidence for novel bacterial species in patients with prostate cancer; and applying deep learning to digital pathology.

Members of the PPCG Steering Committee

Vanessa Hayes, University of Sydney, Australia; Andreas Gruber, University of Konstanz, Germany; Rosalind Eeles, Chris Foster and Zsofia Kote-Jarai, Institute of Cancer Research, London, UK; Housheng He, University of Toronto, Canada; Christopher Hovens, The University of Melbourne, Australia; David Wedge and Rob Bristow, Manchester Cancer Research Centre, University of Manchester, UK. Daniel Brewer and Colin Cooper, University of East Anglia, UK, Vincent Gnanapragasam, University of Cambridge, UK, George Steven Bova, Tampere University, Finland; Adam Butler; Wellcome Sanger Institute, UK; Benedikt

ABSTRACTS

TGFB1 induced TET3 dependent regulation of *OTX2* super enhancer hypomethylation promotes Group 3 medulloblastoma progression

Ziwei Wang[1], Xuan Chen[2], Dongming Han[2], Jiankang Li[2], Tao Jiang[3], Hailong Liu[3]
[1]Faculty of Medicine, Chinese University of Hong Kong
[2]BGI Research, Shenzhen, China
[3]Department of Neuro-pathology, Sanbo Brain Hospital, Capital Medical University, Beijing, China

Background: DNA methylation patterns have been extensively utilized for medulloblastoma (MB) classification. However, the functional implications of enhancer methylation in MB remain poorly understood.

Methods: We conducted bisulfite-based genome-wide sequencing on 189 human MB cases. Upon molecular classification, 80 cases was compared against 8 normal cerebellums, revealing differentially methylated regions (DMRs) . Integration with gene expression data facilitated the identification of DMR target genes, subsequently validated in the 109 cases. Further, leveraging sn-ATACseq, we evaluated the association between DMRs in chromatin accessibility regions and tumorigenesis. Finally, we investigatedd the impact of upstream epigenetic events involving aberrant methylation on tumor development.

Results: We observed a negative correlation between the methylation levels of DMR overlapping super enhancers (seDMRs) and the expression of targeted genes, particularly in Group_3 MB (G3-MB), the most aggressive subgroup. Genes targeted by hypomethylated seDMRs were significantly enriched in early-stage cerebellum development hallmarks, specifically the rhombic lip subventricular zone (RL^{SVZ}), but not in the late stage. Hypomethylated seDMRs of *OTX2* were associated with elevated chromatin accessibility and oncogenic activation of progenitor-like MB tumor cells, leading to worse prognosis. Integrative epigenetic analysis revealed highly active OTX2 binding sites within its own hypomethylated seDMRs, indicating feedback auto-regulation. Deletion of motif at this enhancer region reduced *OTX2* expression and attenuated tumor progression. Further, we found that ten-eleven translocation 3 (TET3), a DNA methylation eraser, was recruited by OTX2 to catalyze active DNA demethylation at binding motifs, thereby organizing chromatin and stimulating gene expression. *TET3* was upregulated by TGF-β1/Smad2 signaling. Based on these findings, nanoparticle-coated small interfering RNAs (siRNA) targeting *TET3* were designed, effectively suppressing xenograft progression.

Conclusion: Our study highlights the increased transcriptional activity of *OTX2* through cell-type-specific super enhancer demethylation, which is dependent on TGF-β1/Smad-induced TET3. This identifies a potential therapeutic strategy for G3-MB.

Key words: Medulloblastoma, Epigenomics, Tumorigenesis

ABSTRACTS

A Series Cases of Lhermitte-Duclos Disease with Surgical Interference

Ziyang Chen[1, 2, 3]†, Hui Ouyang[1, 2, 3]*, Yanfeng Fan [1, 2, 3]*
[1]Department of Neurosurgery, Guangdong Sanjiu Brain Hospital, Guangzhou, China;
[2]Institute of Clinical Teaching Center, Jinan University, Guangzhou, China;
[3]Institute for Brain Research and Rehabilitation, South China Normal University, Guangzhou, China;

OBJECTIVE Lhermitte-Duclos disease (LDD) is an infrequent disorder which overlap features both of neoplasm and hamartoma in cerebellar hemispheres. It has been considered to be relevant to phosphatase and tensin homologue (*PTEN*) hamartoma tumor syndrome, also known as Cowden's syndrome (CS). In order to thorough investigate the correlative factors, and provide potential interference idea, here we present a retrospective analysis and description among series cases.

MEDTHOD Through thorough research in our multidisciplinary team, 3 total cases were identified. We performed a retrospective search to investigate clinical Information such as onset ages, clinical manifestations, surgical treatment, follow-up results.

RESULTS All three female patients, of age ranging from 18 to 53 years. Two cases presented with raised intracranial tension and headache, one patient had cerebellar signs. Duration of symptoms varied from 1 to 36 months. Two patients underwent partial resection, one patient underwent complete removal. Two patients obtained a good prognosis, while one patient had a poor prognosis due to increased intracranial edema and underwent unplanned reoperation.

CONCLUSION The routine genetic testing for PTEN expression is very necessary in diagnosis of LDD. Gross total resection is benefit for the management of LDD when neurological compromise exists due to mass effect.

Keywords: Lhermitte-Duclos disease, Dysplastic cerebellar gangliocytoma, Cowden syndrome, Hamartoma, Surgical interference

ABSTRACTS

Comprehensive Genomic and Transcriptomic Characterization of Glioblastoma Reveals the Impact of Extrachromosomal DNA on Tumor Heterogeneity and Therapeutic Vulnerabilities

Wenshu Tang[1#], Wing Lun Lee[2#], Cario W.S. Lo[1], Annie T.W. Chu[1], Karrie Mei-Yee Kiang[3], Wei Ma[1], Amy H.Y. Tong[1], Dingge Ying[1], Jamie S.L. Kwok[1], David Shih[4], Gilberto K.K. Leung[3,5], Brian H.Y. Chung[1,6,°], Aya El Helali[3,°],
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Introduction:
Glioblastoma (GB) is a highly aggressive and malignant brain tumor that poses significant treatment challenges due to its limited therapeutic options. The integration of whole-genome sequencing (WGS) and single-cell transcriptomic profiling is crucial for the detection of actionable genetic alterations and for comprehensively understanding the cellular functional dynamics.

Methods:
In this study, we conducted both short-read and long-read whole-genome sequencing, coupled with single-nucleus RNA sequencing (snRNA-seq), on a cohort comprising 42 patients diagnosed with GB in China. This multifaceted approach enabled a thorough evaluation of intra- and extrachromosomal genomic alterations and facilitated the classification of transcriptomic subtypes in GB.

Results:
Our investigations unveiled amplified oncogenes, specifically EGFR, MYC, CDK4, and PDGFRA, which were found on extrachromosomal DNA (ecDNA). Notably, EGFR ecDNA exhibited a unique DNA methylation profile associated with gene expression programs that modulate cellular differentiation.

Conclusion:
The findings of our study bear profound implications for the management of GB patients and highlight novel avenues for future research across diverse population strata. Our discoveries provide essential foundational insights that could propel GB research forward and foster the development of personalized therapeutic strategies.

ABSTRACTS

Tyrosine kinase inhibitor could reconstruct immune microenvironment in lung cancer brain metastasis *via* CTLA4

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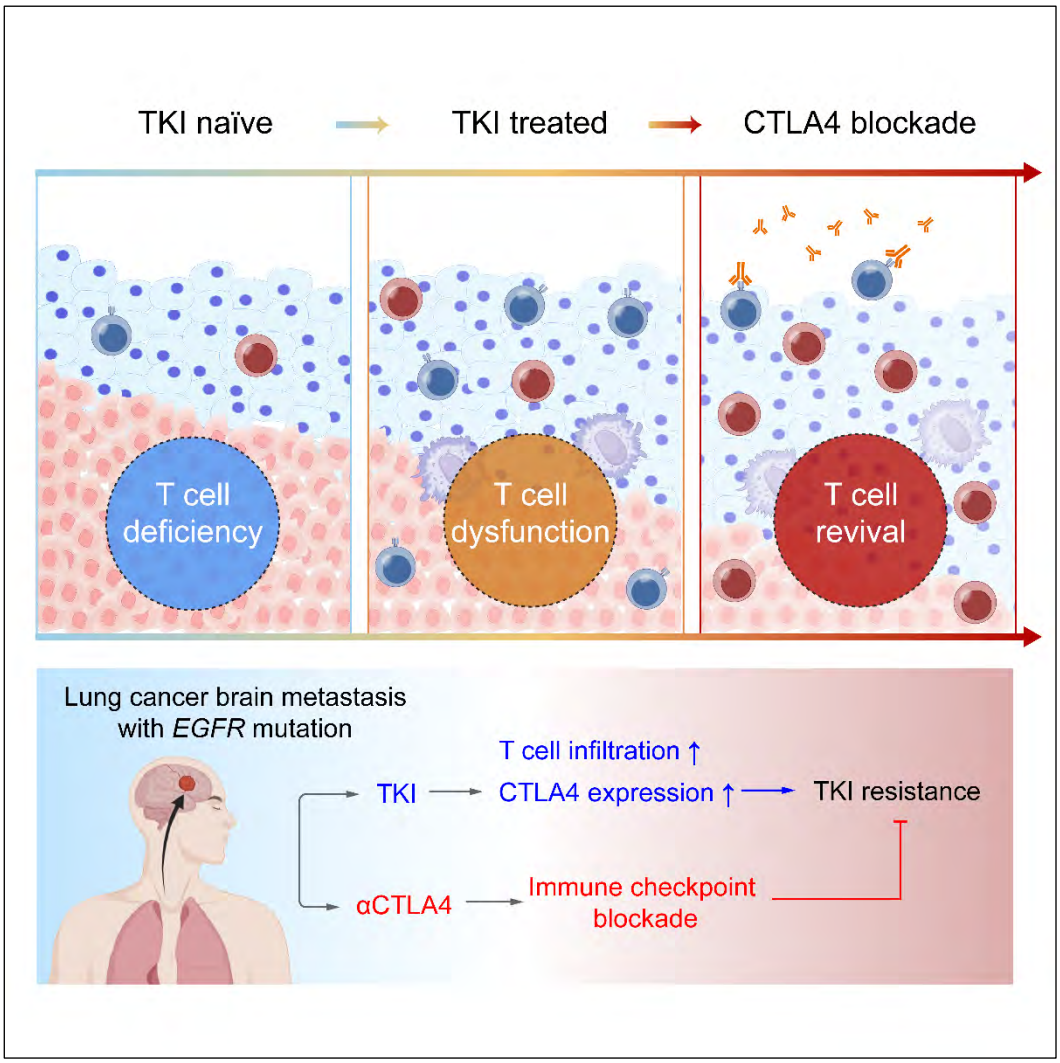

Background:
The brain is one of the most common distant metastatic sites for lung cancer, and patients with lung cancer brain metastasis (LCBM) have a very poor prognosis. Although tyrosine kinase inhibitors (TKIs) can improve the prognosis of LCBM, the development of acquired resistance severely limits their efficacy. The immune microenvironment plays a significant role in TKI acquired resistance.

ABSTRACTS

Objective:
This study aims to reveal the immune microenvironment mechanisms of TKI acquired resistance and provide new targets for overcoming TKI resistance in the clinic.

Methods:
Based on a cohort of over 400 LCBM cases collected from Huashan Hospital, we selected 31 samples for single-cell sequencing to characterize the immune microenvironment profiles of LCBM before and after TKI treatment. Additionally, using a large-scale clinical sample and tumor organoids, we investigated the molecular mechanisms of immune microenvironment reshaping during TKI treatment through mass cytometry, multiplex immunohistochemistry, lineage tracing, CUT&Tag, and other methods.

Results:
Comparing the immune microenvironment of LCBM before and after TKI treatment, we found that while TKI could increase T cell infiltration, it also upregulated the expression of the T cell immune checkpoint CTLA4, inhibiting its effector functions. Mechanistic studies showed that TKI could induce immunogenic cell death in sensitive lung cancer cells, releasing the HMGB1 signal molecule, which further activated the T cell NF-κB signaling pathway, and the transcription factor p65 activated the target gene CTLA4 expression. Single-cell analysis of mice treated with TKI/CTLA-4 inhibitor combination therapy revealed that the combined treatment could relieve the functional suppression of T cells and enhance their tumor-killing capabilities.

Conclusion:
This study, for the first time, depicts the immune microenvironment profiles of LCBM before and after TKI treatment at single-cell resolution, revealing that TKI can induce immunogenic cell death in EGFR-mutated lung cancer cells, release signal molecules, and upregulate the expression of the immune checkpoint CTLA4 in T cells, mediating targeted drug resistance. It provides new insights for overcoming TKI resistance in the clinic.

ABSTRACTS

Stromal Architecture and Fibroblast Subpopulations with Opposing Effects on Outcomes in Hepatocellular Carcinoma

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Background and Objective: Liver cancer is a highly lethal malignancy that causes around 800,000 deaths annually, with hepatocellular carcinoma (HCC) comprising 75~85% of the total cases. Approximately 50-60% of HCC patients are diagnosed as advanced-stage, receiving systemic therapies. However, even the first-line immune checkpoint blockades (ICBs) offer limited efficacy and response duration. Of note, HCC typically arises in a stroma-rich environment, with liver fibrosis existing in 80-90% of the patients secondary to common etiologies, such as chronic viral infection, metabolic syndrome, and alcohol abuse. Intratumor stroma, occupying tumor mass, has been associated with T-cell exclusion and ICB efficacy in HCC. Meanwhile, the fibrotic rings (FR), surrounding tumor mass, also known as tumor encapsulation, is reported to have contradictory prognostic roles in HCC. Elucidating the biological function and regulatory mechanisms of spatially different stroma components may inform novel therapeutic options in HCC.

Methods: We identified two distinct stromal archetypes ("FR^+" with FR surrounding tumor mass and "FR^-" harboring prominent intratumor stroma but without FR) using whole-slide images (WSIs) of hematoxylin and eosin (H&E) stained tissue sections. To investigate both the prognostic and molecular features, we systematically reviewed 1,169 treatment-naïve HCC cases from 2 well-documented cohorts (cohort1, n=1,010; cohort2, n=159) and identified two major stromal archetypes. To study both the acellular ECM components and the cellular components that mainly contribute to the regional stromal features, we conducted spatial proteomics of 127 formalin-fixed paraffin-embedded (FFPE) samples from 30 treatment-naïve HCC patients (FR^-, n=15; FR^+, n=15) using laser capture microdissection (LCM). In addition, we generated Stereo-seq data of 28 HCC samples (patients n=28; FR^+, n=18; FR^-, n=10), paired with snRNA-seq (n=21), scRNA-seq (n=12), and snATAC-seq (n=26).

Results: We revealed that the two stromal archetypes had distinct prognostic, immunologic, and ECM profiles. Firstly, we found that patients with FR^+ tumors had better survival compared to FR^- tumors. Secondly, we revealed that regional stroma had distinct protein profiles by spatial proteomics workflow. Surrounding FR of FR^+ tumors enriched in ECM organization, wound healing, and cell migration pathways.

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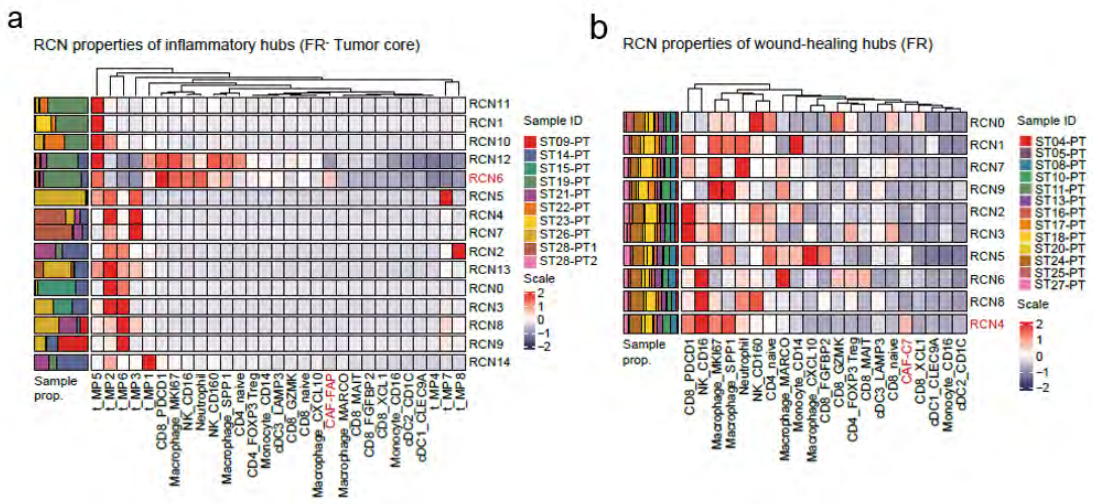
Intratumor stroma of FR- tumors showed strong enrichment of actin cytoskeleton organization, RAC-GTPases, and leukocyte activation. Moreover, we found that two fibroblast subpopulations (CAF-FAP and CAF-C7) were separately enriched at the intratumor stroma and surrounding FR, and identified the RUNX1 and USF2 as potential regulators for CAF-FAP and CAF-C7 differentiation, respectively. We discovered two functional units, one is the intratumor inflammatory hub featured by CAF-FAP plus CD8_PDCD1 proximity and the other is the surrounding FR's wound-healing hub with CAF-C7 plus Macrophage_SPP1 colocalization. Finally, the vitro experiment showed that inhibiting CAF-FAP combined with anti-PD-1 in orthotopic HCC models led to improved tumor regression than either monotherapy.



a, c RCN properties of inflammatory hubs at the tumor core of FR- tumors (**a**) and wound-healing hubs at the FR of FR- tumors (**b**).

Conclusion: Our findings introduced novel spatially confined CAF biology and stroma-targeted strategies for HCC, raising the concept that those CAF subsets fit their regional transcriptional programs and intercellular crosstalk to the tissue context.

ABSTRACTS

Local cranial radiation combined with third-generation TKIs improve leptomeningeal metastasis disease-free survival and reduce leptomeningeal metastasis rate in patients with EGFR-mutated NSCLC and brain metastasis

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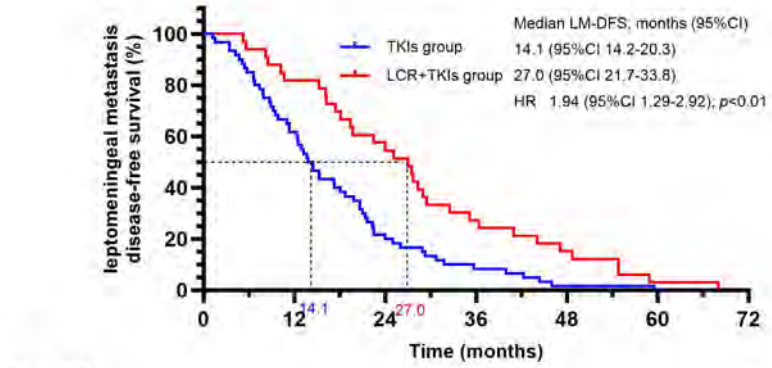
Background: Studies have shown that third-generation tyrosine kinase inhibitors (TKIs) prolong overall survival (OS), and the central nervous system progression-free survival (CNS-PFS) in patients with lung cancer and brain metastases (BM). However, the role of local cranial radiation (LCR) combined with third-generation TKIs in the following the progression to leptomeningeal metastases (LM) in EGFR (epidermal growth factor receptor)-mutated non-small cell lung cancer (NSCLC) with BM remains unclear. This study investigates whether LCR combined with third-generation TKIs can influence the occurrence LM in EGFR-mutated NSCLC with BM.

Objective: To explore the effect of LCR combined with third-generation TKIs on subsequent LM in patients with EGFR-mutated NSCLC with BM.

Methods: Patients diagnosed with EGFR exon 19del or exon 21 L858R mutant NSCLC with LM in Guangdong Sanjiu Brain Hospital were recruited into the study between January 1, 2018 and December 31, 2023. All were diagnosed with BM using an MRI, and received third-generation TKIs before progression to LM. They were separated into two groups according to whether they received LCR during the period from BM to LM on not. The primary endpoints were the LM disease-free survival (LM-DFS) and LM rate. The secondary endpoints were the OS after brain metastasis (BM-OS) and leptomeningeal metastasis (LM-OS).

Results and conclusion: A total of 93 patients were enrolled, 60 in the TKIs group and 33 in the LCR+TKIs group. The median LM-DFS of the TKIs group and the LCR+TKIs group were 14.1 months (95% CI 14.2-20.3 months) and 27.0 months (95% CI 21.7-33.8 months), respectively (P<0.01). 2-year LM rates in TKIs group and LCR+TKIs group were 80.0% and 45.5%, respectively (P<0.01). A multivariate Cox regression analysis showed that no LCR, EGFR exon 21 L858R mutation, and the presence of extracranial metastases were risk factors for LM-DFS. On summary, LCR combined with third-generation TKIs have the ability to improve LM-DFS and decrease LM rate compared with third-generation TKIs alone. Further studies are required to ascertain this effect.

ABSTRACTS



No. at risk							
TKIs group	60	37	12	5	1	0	0
LCR+TKIs group	33	27	18	9	5	1	0

ABSTRACTS

Forward and Backward Evolution of Cancer Cells

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Background and Objectives: Evolution is often depicted as a steady march toward increasingly better beings from simple to complex forms, and traditionally, evolution biology studies forward, but not, backward evolutions. A recent study published on Dec. 13, 2024 at The Conversation, reported that Ferns' 'backward' evolution reveals Life's meandering path, providing evidence that organisms with less specialization in one particular reproductive strategy are more capable to adapt — even if that means reverting to previous evolutionary versions (1).

Methods and Results: This interesting notion coincided with our observations published earlier this year on the backward evolution of cancer cells from mitosis to non-mitosis for division or reproduction, and this backward evolution was reversable from non-mitotic to mitotic division, under favorable cell culture conditions such as decreased anticancer drug concentrations (2). This backward evolution of cancer cells also coincided with the forward and backward mutations in cancer genomes in forms of both single nucleotide variations and copy number variations, reported by us six years ago (3).

Conclusion: The forward and backward evolution of cancer cells at sequence, structure and whole genome levels requires more in depth studies to understand its relationship to cancer metastasis, recurrence, heterogeneity and drug resistance.

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ABSTRACTS

Spatiotemporal Genomics of Human Medulloblastoma at Single-nucleus Resolution

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Medulloblastoma (MB) is the most common malignant brain tumor of childhood. While single-cell transcriptomic approaches have revealed a great deal of the cellular hierarchy and heterogeneity between individual MB cells, how these various cell types are organized in in three-dimensionsional space is currently unknown. We have now comprehensively analyzed single-nucleus RNA transcriptomics, chromatin accessibility, and spatial transcriptomic profiles from 51 human MB spanning the four molecular subgroups, complemented by bulk whole genome/bisulfite, and bulk RNA sequencing data from 332 human MB samples.

We determine the cellular hierarchy of tumors with division into MB cancer stem-like cells (MB-CSCs), cycling cells, and more differentiated-like populations. Gene expression signature for different levels of the hierarchy are strongly correlated to clinical outcomes. To more accurately understand the cellular origin and the developmental trajectories of MB cells, they are compared to cell populations from the normal developing rhombic lip in both *Mus Musculus* and *Homo Sapiens*. Subsequently we illustrate the spatial relationships between different levels of the differentiation hierarchy from patient derived operation materials, which further emphasizes the geographically heterogeneous nature of MB. We identify somatic copy number variants (CNVs), as well as transcriptional signatures that are only present in specific, geographically defined subclones of tumors when studied at single cell resolution. The existence of distinct, geographically adjacent, but isolated subclones of the tumor, harboring different somatic CNVs suggests a model in which MB is not in fact truly monoclonal, but rather a competitive agglomeration of co-existing subclones. Inclusion of spatial genomics in our analysis of MB supports a new model of the disease, in which adjacent but distinct subclones, each with varying somatic genetic drivers co-exist with their own microenvironment, as well as their adjacent clones. The extent to which there is co-operative signaling between distinct clones at their borders is currently unknown. The high frequency of isolated subclones, and the failure of any one subclone to become further malignant and sweep to clonal dominance suggests that co-operation between subclones contributes to tumor growth and persistence. Translational approaches to study MB that fail to account for geographic heterogeneity may not lead to novel effective therapies, whereas understanding the biology between interacting clones could suggest novel approaches of therapy that will be effective across all clones in a given tumor.

ABSTRACTS

Survival Impact and Cost-Effectiveness of Precision Oncology: Health System Evidence from British Columbia, Canada

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Background: Precision oncology uses next generation sequencing (NGS) to identify therapeutic targets independent of cancer type. Globally, health system adoption of precision oncology is uneven due to uncertain real-world clinical and economic impacts. Real world evidence (RWE) of efficacy and health system cost-effectiveness is key to equitable access for precision oncology.

Objective: We determine the cost-effectiveness of a tumour-agnostic NGS panel compared to single-gene testing for advanced cancers.

Setting: Patients with advanced non-small cell lung cancer (NSCLC), melanoma, and colorectal cancer in British Columbia, Canada.

Methods: We used real-world patient-level data to emulate a pragmatic randomized controlled trial. We 1:1 matched NGS patients with controls using genetic algorithm-based matching, a non-parametric semi-supervised machine learning approach that finds optimal matches that maximize balance based on a generalized weighted Mahalanobis distance metric. Following the emulation of random treatment assignment, we estimated the magnitude of survival difference in the Kaplan-Meier curve using the Cox proportional hazard ratio (HR). We calculated mean life years gained (LYG), incremental costs, net monetary benefit (public healthcare payer perspective; 2021 CAD; willingness to pay $50,000 per LYG) using inverse probability of censoring weighted linear regression and nonparametric bootstrapping.

Results: Our eligible population included 3,588 patients, 2,033 who received NGS and 1,555 who received single-gene testing. From that eligible population, we matched 1,142 patients receiving NGS to 1,142 controls. Our machine learning model achieved balance on all included covariates, successfully emulating a randomized trial. We found a statistically significant reduced hazard of death (HR: 0.85, 95% CI: 0.77, 0.94, p-value=0.002]. Incremental LYG were 0.15 (95%CI: 0.06, 0.25), which translated to 2 months of additional survival. Incremental health system costs were $4,473 (95%CI: $-1,255, $10,364), with NGS testing costing $1,200 per patient. The net-benefit was $3,171 (95% CI -$2,090, $9,305) and there was an 87% probability that BC's tumour-agnostic NGS panel was cost-effective

ABSTRACTS

Implications: Tumour-agnostic targeted or virtual panels for advanced cancers is cost-effective for health systems at an NGS price of $1,200 per patient. RWE from real-world data are key to supporting policy for population-wide reimbursement of precision oncology.

Declaration of funding: This research was supported by Genome British Columbia/Genome Canada (G05CHS) and the Terry Fox Research Institute.

ABSTRACTS

Landscape of ADC Target Gene Expression and Their Association with Efficacy of ADC in Advanced Solid Tumors: SCRUM-Japan MONSTAR-SCREEN-2

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Background:
Approved ADCs have been developed for a diverse array of targets. However, a predictive biomarker for ADCs remains elusive, except for HER2. Herein, we evaluated the landscape of ADC target gene expression and their association with efficacy of ADC by mRNA expression level of targeted genes in advanced solid tumors from the SCRUM-Japan MONSTAR-SCREEN-2, a nationwide molecular profiling project (UMIN000043899).

Objective: To evaluate the landscape of ADC target gene expression and relationship between mRNA expression levels of ADC target genes and therapeutic efficacy of ADCs in patients with advanced solid tumors.

Method: Patients with advanced solid tumors were enrolled; whole exome / transcriptome sequencing by Caris Life Sciences (Phoenix, AZ, USA) and immunohistochemistry (IHC) of HER2 were performed.

Results: Among 2,037 patients enrolled as of July 2023, mRNA expression data of baseline tissue samples were available in 1,414 pts across 35 cancer subtypes: most common subtypes were colorectal (n=332), gastric (n=181), pancreatic (n=114) and breast cancers (n=106). mRNA expression of ADC target genes was observed across diverse cancer types. *ERBB2* mRNA and HER2 IHC expression were highly concordant (AUC,

0.92). Since HER2 overexpression in IHC corresponded to the top 9.3 percentile of mRNA level across all solid tumors, we defined the cutoff for high mRNA expression as the top 9.3 percentile. Sixty-two percent of the high mRNA expression of target genes for FDA-approved ADCs, such as HER2, Nectin-4, TROP-2, Tissue factor and Folate receptor, was found in off-label cancer types. In 46 patients treated with ADC monotherapy, the high mRNA expression group tended to have a better objective response rate (ORR) and significantly better progression-free survival (PFS) than the non-high mRNA expression group (ORR: 48.0% vs 28.6%, P = 0.23, PFS: 6.9 vs 3.0 months, hazard ratio [HR]: 0.38, 95% CI: 0.17-0.84, *P* = 0.01). The significant difference in PFS was independent of ADC targets and cancer types.

Conclusion:
High mRNA levels were associated with better therapeutic efficacy of ADCs, highlighting the potential of mRNA level as a predictive biomarker of ADCs.

ABSTRACTS

Unlocking Germline and Somatic Variations in Lung Cancer Brain Metastasis

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Background: Lung cancer is the most common cancer in the world and a major source of brain metastasis, which has an increasingly high incidence and adverse prognosis.

Objectives: The Hong Kong Brain Metastasis (HKBM) project under the umbrella of ICGC-ARGO aims to unlock the germline and somatic variations underlining the risk of primary lunch cancer metastasis to the brain through in depth genomic and functional studies.

Methods: AluScan next generation sequencing on Illumina platform has been applied to exam genome-wide sequence variations in form of single nucleotide variation (SNV) and structure variations in form of copy number variation (CNV), with white blood cell samples and tumor tissue samples for germline and somatic genomes, respectively.

Results: SNV analysis gave rise to destined mutation profiles for primary lunch cancers and brain metastatic cancers. In particular, CG to TG forward mutations were clearly higher in early stages of primary lung cancers compared to later stage and brain metastatic cancers. Genome wide CNV analysis revealed that a wave of copy number deletions occurred at onset of primary lung cancers which continued in both forward and reversed directions visible as CNV-gains and CNV-losses in advance lung cancers and more so in brain metastatic cancers. Moreover, feature enrichment analysis revealed that enhanced CNV-gains enriched with evolutionary young transposable elements were prominent in the germline genome of brain metastasis group of patients.

Conclusion: The present study has pointed out that lung cancer brain metastasis not only showed prominent mutation profiles, and forward-reversed sequence and structural variations in somatic genomes, but also distinct genomic feature enrichment in germline genome. The findings from this study provide insights to the development of diagnostic approaches to staging, prognosis, early detection and prevention of lung cancer brain metastasis.

ABSTRACTS

Ensuring Equity in Genomics: WHO Guidance on Human Genome Data Collection, Access, Use, and Sharing and its Implications for the ICGC-ARGO Project

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In November 2024, the World Health Organization (WHO) published a set of guiding principles aimed at establishing equitable practices in the collection, access, use, and sharing of human genome data. These principles, developed by the WHO Technical Advisory Group on Genomics (TAG-G), represent a significant effort to promote fairness and inclusivity in genomic research and its medical applications globally.

The WHO Guidance underscores the critical importance of informed consent and privacy considerations, advocating for transparency and clear communication regarding the use of genomic data. It emphasizes the need to address inequities by fostering inclusivity and eliminating disparities in access to genomic technologies. In particular, the document highlights the necessity of enabling participation and benefits for underrepresented groups, including populations in low- and middle-income countries. Furthermore, the Guidance calls for enhanced global cooperation and capacity building, urging stakeholders to invest in infrastructure development and to cultivate genomic literacy within individual nations.

The principles outlined in this document are envisioned as a framework for fostering equitable practices in genomic medicine and research worldwide. They hold particular relevance for projects like the International Cancer Genome Consortium Accelerating Research in Genomic Oncology (ICGC-ARGO), where the application of these guidelines could serve to promote inclusivity and fairness. Additionally, the Guidance offers a benchmark for evaluating whether national and international initiatives adhere to its equitable standards, facilitating accountability and progress in the field of genomics.

ABSTRACTS

Real World Experience of Utilizing Formalin Fixed Paraffin Embedded Tissue (FFPE) in Whole Genome and Transcriptome Sequencing for Personalized Oncogenomics

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Background: The Personalized Oncogenomics (POG) Program at British Columbia Cancer, Canada, is a prospective clinical trial (NCT02155621) with enrollment of over 2000 metastatic and advanced pan-cancer patients. Whole genome and transcriptome sequencing and analysis (WGTA) is performed to inform potential therapeutic options for individual patients. The analytical pipeline, including lab and bioinformatics, has been designed and optimized for the analysis of fresh biopsy tissue. However, patient accrual has increased and obtaining fresh tissue from cancer patients can be challenging and sometimes risky.

Objective: We sought to explore the use of FFPE tumour tissue that may be available through clinical standard of care processes, to make WGTA more accessible to a wider patient population. Use of FFPE-derived tumour tissue in WGTA can be challenging as FFPE tissue is optimized for pathology diagnosis, and is associated with nucleic acid artifacts and degradation resulting in suboptimal nucleic acid quality for use in molecular assays. We evaluated the use of FFPE specimens for WGTA, to assess the impact of sequencing artifacts induced by formalin fixation and storage to refine quality metrics and bioinformatic filtering.

Methods: FFPE tissue blocks were reviewed by a pathologist to estimate tumour content, then extracted for total nucleic acid (TNA). Whole genome and RNA-Seq libraries were constructed and sequenced using Illumina paired-end sequencing to over 80X WGS coverage and 200 million RNA-Seq reads. Quality control (QC) metrics were assessed at three different gates: 1) laboratory QC, 2) post sequencing non-alignment-based QC, 3) post analysis alignment-based QC.

Results: The FFPE cohort described encompasses 122 FFPE samples sequenced from 120 patients across 20 different tumour types. Results were successfully reported using both WGS and transcriptome in 83 (68%) cases, with a quality disclaimer reported in 10 cases. There were 31 (25%) where DNA WGS only results were reported, and only 8 (7%) cases that did not produce any results. Based on results of comparative analyses, improvements were made to the TNA extraction protocol, small variant filtering, and QC metrics to identify poor quality WGS and RNA-based libraries. Additional optimization is required for the refinement of artifact filtering for small variant and copy number identification. Sample quality is associated with variation in preservation, storage, laboratory protocols, and bioinformatics analysis tools. RNA QC metrics to assess diversity (duplicates, %TPM = 0), and transcript quality (transcript integrity number) were informative to assess RNA quality for reporting expression outliers.

ABSTRACTS

Conclusions: This real-world experience supports the use of WGTA on FFPE-derived tissue to provide potential therapeutic treatment options for cancer patients within the POG research program. Analysis requires optimization of lab protocols and analysis pipelines to accommodate for noise and artifacts, and overall failure rate, especially for RNA. FFPE-derived libraries can vary in quality. Careful recording and analysis of metadata, which includes processing and storage information, can help identify process optimization and may help to shape best practices. FFPE WGTA will continue to be refined in order to expand testing to a broader patient population.

ABSTRACTS

Real-World Data for Precision Oncology: Validation of Large Language Models for Automatic Data Extraction from Electronic Health Records

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Background: Globally, precision oncology access is highly variable and often limited to clinical trials or basic research settings. Improved patient access requires healthcare systems to rapidly integrate research data with other real-world health and equity information to generate evidence for precision oncology that supports decision-making. This is an application of learning healthcare. Regrettably, real-world patient data to support learning healthcare is frequently siloed within electronic health records (EHRs) and is often infeasible to manually abstract.

Objective: We sought to validate natural language processing (NLP) and large language models (LLMs) for automating real-world data extraction from EHRs to enable learning healthcare.

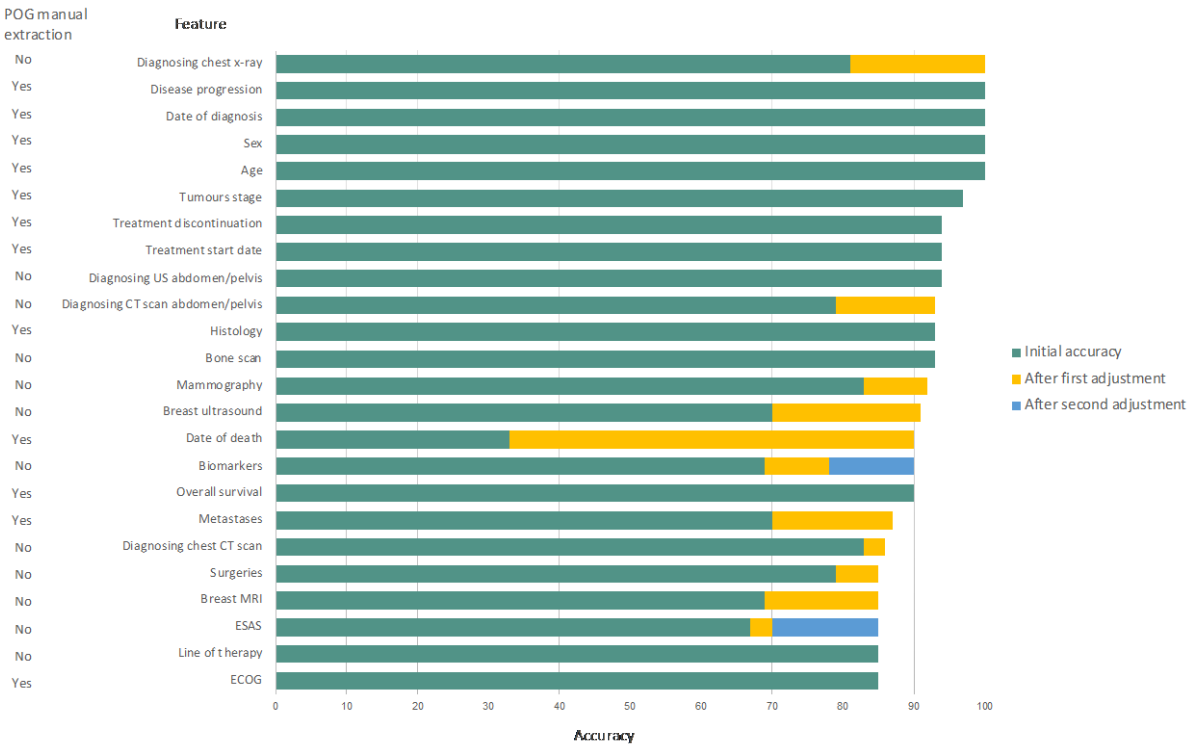
Methods: Our validation study focused on all patients from British Columbia, Canada, diagnosed with metastatic breast or advanced lung cancer, who gave informed consent and enrolled in BC Cancer's Personalized OncoGenomics (POG) program between July 2012 and August 2017, and had high quality comprehensive clinical tumor profiles generated. We obtained Provincial Health Services Authority (PHSA) and BC Cancer EHR documents for care received between 1981 and 2021. We deployed a commercially available LLM engine (DARWEN™) using NLP to extract 24 clinical features of interest for each patient from these documents. Features spanned demographics, disease characteristics, and treatment information, some of which the POG program manually extracts from EHRs (see Figure 1). We compared model outputs to manually curated ground truths for each feature, estimating precision, recall, F1 and overall accuracy. We set a benchmark accuracy threshold at ≥85% and made adjustments to any features whose accuracy remained below this threshold by: modifying the data input into the model or the output of the model; directing the model via parameterization; or fine-tuning the model with additional labelled data.

ABSTRACTS

Results: Of the 722 adult patients who enrolled in POG over the period, 148 patients diagnosed with metastatic breast cancer and 63 with advanced lung cancer met our study criteria. For these patients, we obtained 113,024 EHR documents, consisting of 194 distinct document types. Without additional feature processing or fine-tuning of the LLM engine, 13 of the 24 clinical features achieved benchmark accuracy for data extraction (Figure 1). These features were mainly well-defined demographics and dates identifiable in clinician notes, with the highest accuracies of 100% observed for age, sex, and date of diagnosis. The remaining 11 features required further adjustments to meet the defined benchmark. For example, difficulty contextualizing hedging language encountered within radiology reports initially resulted in lower extraction accuracy (70%) for identifying the earliest date of metastatic disease. Training the model with additional labelled examples enabled adaptation to the nuances observed in this style of language, resulting in better overall accuracy (87%).

Conclusions: High-quality real-world data can be extracted efficiently and at scale from patient EHRs using NLP and LLMs. While limited accuracy may initially arise due to nuances of language and contextual differences across jurisdictions, we demonstrate how institution-specific model training and refinements can address these challenges. Automating extraction of real-world data may facilitate life-cycle evidence generation for precision oncology that guides health system decision-making and supports learning healthcare.

Figure 1: Estimated accuracy of NLP data feature extraction compared to ground truth



Research Support: *This research was supported by the PRecision oncology Evidence Development in Cancer Treatment (PREDiCT) program. PREDiCT is funded by Hoffmann-La Roche Limited and the Canadian Personalized Healthcare Innovation Network.*

ABSTRACTS

Personalized OncoGenomics (POG): Advancing Precision Medicine in British Columbia

Howard J. Lim, Nathalie LeVasseur, Alannah Smrke, Sophie Sun, Daniel J. Renouf, Stephen Yip, Dean A. Regier, Kasmintan Schrader, Seven J.M. Jones, Marco A. Marra, Janessa Laskin

Background: The Personalized OncoGenomics (POG) program at BC Cancer integrates whole genome and transcriptome sequencing and analysis (WGTA) into clinical oncology to provide comprehensive insights into cancer biology, enable therapy alignment, and build foundational resources for precision medicine research. This initiative has positioned BC as a leader in the integration of genomics into routine cancer care and clinical research.

Program Overview: POG enrolls patients with hard to treat cancers for genomic profiling, generating DNA and RNA-based data to inform systemic therapy. Multidisciplinary molecular tumour board discussions guide clinical decision-making by identifying actionable targets and treatment strategies. The program also serves as a hub for collaboration, aligning with the Hereditary Cancer Program, clinical trials, TFRI's Marathon of Hope initiative, and other precision medicine projects to expand its impact on the genomic and clinical landscape.

Future Directions: By fostering partnerships and integrating advanced genomic technologies, POG continues to evolve as a cornerstone of precision medicine in BC. Ongoing efforts to incorporate data from other precision medicine programs, expand research collaborations, and streamline workflows aim to further the program's reach and impact on patient care.

Conclusions: POG exemplifies the transformative potential of genomics in oncology, bridging research and clinical practice to deliver innovative, patient-centered cancer care. Through strategic collaborations and a commitment to advancing genomic medicine, POG is shaping the future of precision oncology in British Columbia and beyond.

CONFERENCE INFORMATION

Date
20 – 22 January 2025 (Monday - Wednesday))

Time Zone
All communicated times in the Scientific Programme are Hong Kong Time. Hong Kong is 8 hours ahead of Greenwich Mean Time (i.e. GMT + 8 hours).

Meeting Venue
Hong Kong Science Park, Shatin, New Territories, Hong Kong.

Main meeting venue: Charles Kao Auditorium, 1/F, Building 10W
Exhibition area: Pre-Function Hall, 1/F, Building 10W
Meeting room for executive meeting: Meeting Room 23-24, 2/F, Building 10W

Official Language
The official language of the Meeting is English.

Registration
Registration counter will be located at Pre-function Hall outside the Charles Kao Auditorium and open on:
20 January 2025 Monday 08:30-16:00
21 January 2025 Tuesday 08:30-17:00
22 January 2025 Wednesday 08:30-12:00

Trade Exhibition
An exhibition will be held during the Meeting on 20 – 22 January 2025.

Certificate of Attendance
Electronic Certificate of Attendance will be issued upon will be issued within two weeks after the Meeting.

Liability and Insurance
The Organiser is not responsible for injury or damage involving persons and property during the Meeting. Participants are advised to make their own arrangements for their medical, travel and personal insurance.
Equipment and all related display materials installed by Exhibitors / Sponsors are not insured by the Organiser, and the Organiser under no circumstances will be liable for any loss, damage or destruction caused to equipment, goods or property belonging to Exhibitors/Sponsors.
Disclaimer
Whilst every attempt is made to ensure that all aspects of the Meeting as mentioned in this website will take place as scheduled, the Organising Committee reserves the right to make changes should the need arise.

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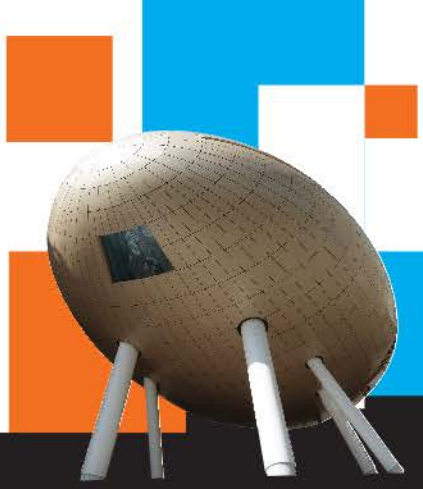

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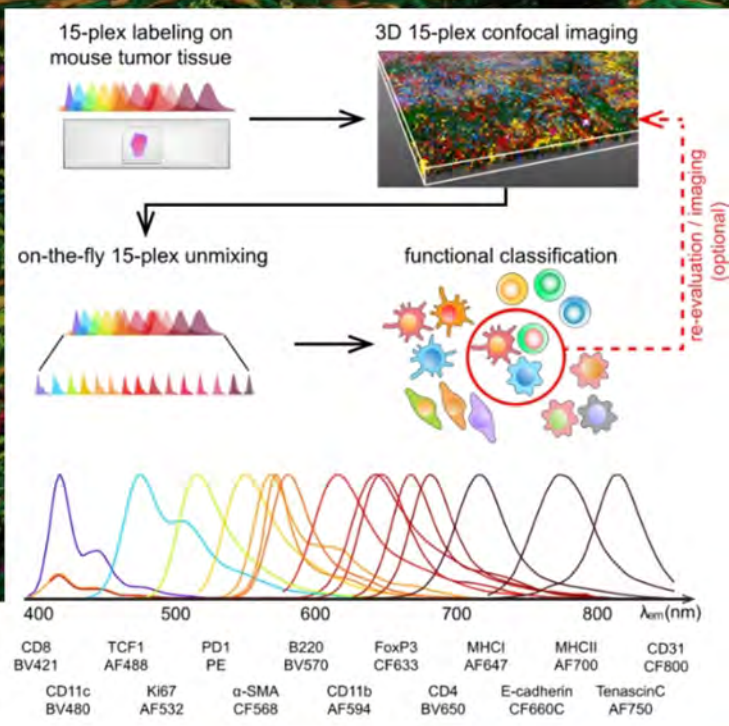

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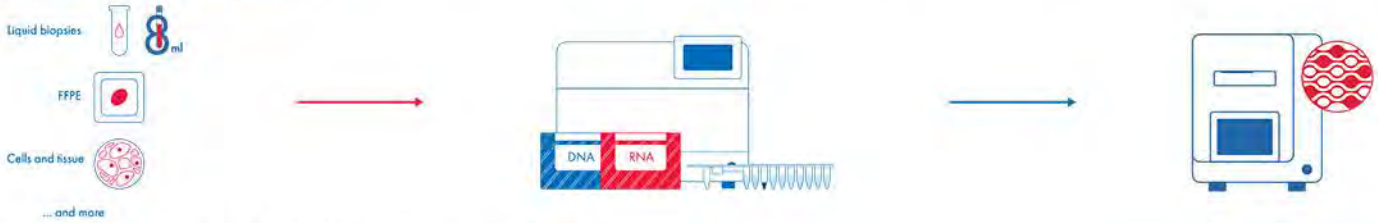

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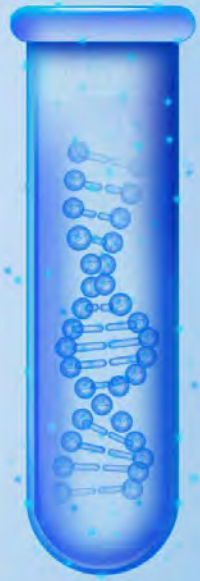

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