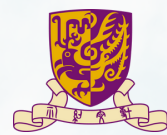


SECOND MEETING OF HONG KONG AND SHANGHAI BRAIN CONSORTIUM -HSBC-2



**FEBRUARY 7, 2026
(SATURDAY)**

**8:45am to 5:00pm
8th Floor, Cordis Hotel, Kowloon**

Session 1

Moderators - Evelyn Lu, Matthew Shing

8:50 am	Opening	Gary Tse
9:00 - 9:30 am	Incorporating CSF-based liquid biopsies in management of CNS tumors	Anthony Liu
9:30 - 10:00 am	Hong Kong Genome Project: Advancing the Genomics Frontier	Brian Chung Wenshu Tang
10:00 - 10:30 am	The Tumor Immune Microenvironment Landscape of Germinoma	John Ding
10:30 - 11:00 am	Immuno-hot and Gemistocytic Subtype of IDH-mutant Astrocytoma Have Worse Prognosis	Wang Jiguang
11:00 - 11:30 am	Coffee and Posters	

Session 2

Moderators - Hovy Wong, Kenneth Wong

11:30 - 12:00 am	Uncovering the Programs that Launch—and Reawaken—Brain Metastasis	Gan Siting
12:00 - 12:30 pm	Pan-Cancer Proteogenomic and Spatial Atlas of 1,000 Metastatic Brain Tumors	Zhang Gao
12:30 - 1:00 pm	Genomic Landscape of Brain Metastases from Renal Cell Carcinoma	David Shih
1:00 - 1:30 pm	Perfusible Vascularized Human Cerebral Brain Organoids	Angela Wu
1:30 - 2:30 pm	Lunch and Posters	

Session 3

Moderators - Mona Hau and Danny Chan

2:30 - 3:00 pm	The Ring Leader, ecDNA as a Master Regulator of GBM Recurrence and Therapy Resistance	Aya El-Helali
3:00 - 3:30 pm	A Population-Based Study of Von Hippel-Lindau Syndrome-associated versus Sporadic Hemangioblastoma: A Territory-wide Review of Patterns-of-Care and Survival Outcomes in Hong Kong	Peter Woo
3:30 - 4:00 pm	CSF-target Methods Assists Precision Diagnosis and Treatment in Diffuse Adult Gliomas	Peter Shi
4:00 - 4:30 pm	Molecular Diagnostics of Brain Tumors : Present and Future	H. K. Ng
4:30 - 5:00 pm	Coffee and Posters	
5:00 pm	End of Conference	

Speakers' profiles have been hyperlinked.

Poster abstracts are available on the website. Click [HERE](#).

PHOTO GALLERY

VIDEO

Organizers:

**Department of Anatomical and Cellular Pathology (ACP), CUHK
Hong Kong Shanghai Brain Consortium (HSBC)**

All poster abstracts are listed here by the alphabetical order of the surnames of the first author.

Mitophagy activation in tumour microenvironment counteracts pro-tumour effects of mitochondrial transfer to glioblastoma cells

Cao Shuhan, Leung K. K. Gilberto, Kiang M. Y. Karrie

Department of Surgery, The University of Hong Kong

Despite advances in cancer treatment, glioblastoma (GBM) remains the most malignant subtype of adult brain tumours, with an overall prognosis of only approximately 18 months. The first-line treatment and current standard of care for GBM, aside from maximum surgical resection, is temozolomide (TMZ). Yet, despite initial promising results in improving survival, drug resistance and tumour relapse remain nearly unavoidable. Mitochondrial transfer between astrocytes and GBM cells have been investigated regarding their effects on GBM tumorigenicity, while mitophagy is crucial in enabling mitochondrial quality control.

To investigate the effects of mitophagy on mitochondrial transfer in GBM cells, we pre-treated Normal Human Astrocyte (NHA) cells containing dsRed-tagged mitochondria with a mitophagy activator for 48 hrs. Subsequently, the treated NHA-dsRed cells were cocultured with eGFP-transfected GBM cells for 16 hrs. FACS was then performed to sort out GBM cells receiving NHA-dsRed mitochondria (double positive signal), and compared against single positive GBM cells for proliferation and migration. The mitophagy-activator pretreated group was compared to an untreated negative control group. Interestingly, when GBM cells received mitochondria from NHA cells pre-treated with a mitophagy activator, cell proliferation and migration were not significantly enhanced compared to those not receiving mitochondria. However, in the negative control group where NHA-dsRed cells were untreated, GBM cells receiving mitochondria showed significantly increased cell proliferation and migration. This suggests that activating mitophagy in tumour microenvironment cells potentially offsets the tumorigenic effects of mitochondrial transfer to GBM cells. In conclusion, combination therapy with a mitophagy-activator may be effective as a novel therapeutic method.

Early Clinical Experience with Belzutifan in Von Hippel-Lindau Disease: A Single-Centre Case Series

Chun Wai Tai¹, Lau Sau Ning Sarah², Li Lai Fung², Lui Wai Man², Leung Ka Kit Gilberto², Cheng King Fai²

1. Faculty of Medicine, University of Hong Kong
2. Department of Neurosurgery, Queen Mary Hospital

Objectives:

To describe the initial clinical application of Belzutifan, a hypoxia-inducible factor 2 alpha (HIF- 2 α) inhibitor, in Von Hippel-Lindau (VHL) disease patients at a tertiary centre, with emphasis on early efficacy, tolerability, and feasibility.

Methods:

A retrospective observational series was conducted on three patients fulfilling VHL disease diagnostic criteria who initiated Belzutifan in the first half of 2025. Clinical records were reviewed for demographics, tumour type, treatment duration, and radiological outcomes. Adverse events were documented using patient reports and laboratory data.

Results:

The cohort (2 males, 1 female) had a mean age at VHL diagnosis of 22 years and a mean age of 49 years at treatment initiation. All patients had CNS haemangioblastoma. The mean treatment duration was 7 months, with a range of 6-9 months. All three patients exhibited radiological evidence of disease regression in their target lesions, constituting a 100% objective response rate. The most significant adverse effect was anaemia which was universal and consistent with Belzutifan's known safety profile. One patient required temporary treatment interruption due to haemoglobin <9g/dL, which subsequently recovered to >9g/dL. No treatment discontinuation occurred. All patients continued regular monitoring with imaging and haematologic assessments.

Conclusion:

Our early institutional experience demonstrates that Belzutifan provides clinically meaningful tumour reduction in VHL patients, consistent with trial data. Side effects, primarily anaemia and fatigue, were present. Given the rarity of VHL and the novelty of Belzutifan, further observation in a larger cohort with longer follow-up is needed to clarify durability of response, optimize side-effect management, and define its role in comprehensive VHL care.

Perfusable Vascularized Human Cerebral Organoid

Feng Xiaohan

Hong Kong University of Science and Technology

Cerebral organoids model human brain development and disease but are limited by the lack of vascularization, causing hypoxia and restricted maturation. We developed a perfusable vascularized human cerebral organoid (pvhCO) by integrating self-assembled intrinsic vasculature with an engineered microfluidic network. Perfusion in pvhCOs reduced hypoxia, enhanced growth, and promoted neuronal and astrocyte maturation beyond non-perfusable (vhCO) and non-vascularized (hCO) controls. Endothelial cells in pvhCO exhibited neural-specific signatures, upregulating genes for barrier function, angiogenesis, and flow response. Functionally, pvhCOs displayed stronger electrical activity and network complexity in electrophysiological tests. This model offers a physiologically relevant human platform for studying neurovascular interactions, disease, and drug testing.

The Covert Cancer Network: Intercellular Mitochondrial Dynamics Models The Tumourigenicity of Glioblastoma

Kwok Chun Wai Aeon¹, Kiang Mei Yee Karrie¹, Leung Gilberto Ka Kit^{1,2}

¹Department of Surgery, The University of Hong Kong

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Mitochondria play an irreplaceably important role in maintaining cellular energy homeostasis and metabolic characteristics. When cells are under stress, mitochondrial functions can deviate from their healthy balance, leading to various pathological conditions. Recent research advances have highlighted the role of mitochondria in glioblastoma (GBM) growth and invasion, ranging from excessive production of reactive oxygen species to dysregulated apoptosis, thereby shaping the protumourigenic landscape within the GBM microenvironment. In addition to the intracellular mitochondrial dysequilibrium in GBM cells, we are interested in how intercellular mitochondrial interactions may rewire GBM functions. Preliminary experiments demonstrate the significance of cell-cell mitochondrial dynamics in GBM. Our research elucidates the novel horizontal cancerous mitochondrial connections and their promising therapeutic potential.

A single-cell resolved atlas of pan-cancer metastatic brain tumors

Yulan Deng^{1,11}, Yu Jiang^{1,2,11}, Hao Duan^{3,11}, Hongbin Lan^{4,11}, Zhenyu Yang^{1,11}, Dainan Zhang^{5,11}, Wanming Hu^{6,11}, Xiuqi Wang⁴, Yuqi Liu⁵, Yuanzhong Yang⁶, Yuan Xie³, Karrie Mei-Yee Kiang^{7,8}, Yu Liu⁴, Xin Zan², Jianguo Xu², Chengjian Zhao⁹, Yue Li¹⁰, Yi Zhang¹⁰, Gilberto Ka-Kit Leung^{7,8,12}, Shiyu Wei^{1,12}, Wang Jia^{5,12}, Yonggao Mou^{3,12}, Gao Zhang^{4,12}, Lunxu Liu^{1,12}

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¹¹These authors contributed equally.

¹²These authors jointly supervised this work.

Abstract

Brain metastases (BrMs) represent a lethal stage of cancer evolution characterized by profound heterogeneity and resistance to therapy. To elucidate the molecular landscape of these tumors, we constructed a comprehensive single-cell and single-nucleus transcriptomic atlas comprising 130 pan-cancer BrM samples across 18 primary tumor types, integrated with 34 gliomas as a comparative brain-native cohort. Our analysis defined 27 robust transcriptional meta-programs (MPs) in malignant cells, spanning cell cycle, stress, hypoxia,

and lineage-related states. Notably, our data suggest a lineage-dependent rewiring of intercellular communication networks among highly interactive MPs in BrMs. Conserved functional states appear to leverage distinct signaling pathways to remodel the neural niche: for instance, breast cancer BrMs predominantly engage CX3C interactions, whereas lung adenocarcinoma BrMs are more closely associated with MIF signaling. Furthermore, comparative analysis with matched primary tumors highlights the evolutionary emergence of divergent metastatic subclones, characterized by high chromosomal instability and the co-enrichment of cell cycle, stress and epithelial-mesenchymal transition (EMT)-related programs. Coupled with a lineage-specific immune landscape, these findings shed light on the diverse adaptive strategies metastatic cells may employ to colonize the brain. This comprehensive transcriptomic atlas serves as a foundational resource for dissecting tumor heterogeneity and exploring potential therapeutic vulnerabilities in BrMs.

Disrupting Glioblastoma Metabolic Resilience via Transplantation of Oxidatively Stressed Adipocyte-Derived Mitochondria

Junbo Liao, Gilberto Ka Kit Leung, Karrie Mei Yee Kiang
Department of Surgery, The University of Hong Kong

Background:

Glioblastoma (GBM) is a highly aggressive brain tumor with a median survival of approximately 15 months. Its resistance to conventional therapies—including surgery, radiation, and chemotherapy—arises largely from its capacity to tolerate oxidative stress and evade ferroptosis, an iron-dependent form of cell death. GBM cells acquire healthy mitochondria from surrounding cells to maintain low-reactive oxygen species (ROS) energy production, thereby suppressing oxidative phosphorylation and limiting oxidative damage.

Objective:

We hypothesized that transplantation of dysfunctional mitochondria derived from oxidatively stressed adipocytes could perturb GBM's metabolic adaptation and trigger ferroptosis.

Methods:

Human preadipocytes and 3T3-L1 cells were differentiated into adipocytes and validated by Oil Red O staining. To induce oxidative stress, adipocytes were treated with 200 μM H_2O_2 for 24 h. Mitochondria were then isolated, quantified (50 μg protein equivalent), and transplanted into U87 and U251 GBM cells, as well as orthotopic mouse models using U87 (BALB/c nude), SB28, and GL261 (C57BL/6) cell lines ($n = 10$ per group). Intratumoral mitochondrial injections (50 μg in 5 μL PBS) were administered on day 7. Ferroptosis was evaluated through ROS levels, lipid peroxidation, mitochondrial potential, GPX4 expression, iron accumulation, and IHC markers (4-HNE, GPX4). Tumor progression was monitored via bioluminescence imaging.

Results:

In vitro mitochondrial transplantation increased ROS 3-fold and lipid peroxidation 2.5-fold ($p < 0.01$), while reducing mitochondrial membrane potential by 60% ($p < 0.05$), lowering GPX4 expression by 70% ($p < 0.01$), and elevating intracellular iron 1.8-fold ($p < 0.05$), consistent with ferroptotic death. In vivo, bioluminescent tumor radiance decreased by 50% in U87, 45% in SB28, and 48% in GL261 models by day 28 ($p < 0.01$). Corresponding IHC confirmed elevated 4-HNE and reduced GPX4 expression.

Conclusion:

Transplantation of oxidatively stressed adipocyte-derived mitochondria effectively disrupts GBM's antioxidant metabolic network and induces ferroptosis both in vitro and in vivo. This strategy harnesses GBM's inherent mitochondrial scavenging behavior, offering a potential therapeutic avenue to overcome its ferroptosis resistance.

Improved Survival of Pediatric High Grade Glioma in the Modern Era

Christy Yuen-kwan Mak¹, Dennis Tak-loi Ku¹

¹Haematology and Oncology Centre, Department of Paediatrics and Adolescent Medicine, Hong Kong Children's Hospital, Hong Kong SAR

Objectives

To evaluate outcomes of pediatric high-grade glioma (HGG) in Hong Kong, comparing treatment eras before and after the establishment of the Hong Kong Children's Hospital in 2019.

Methods

A retrospective review of 68 pediatric HGG patients diagnosed from 1999 – 2025 was performed. Patients were stratified by diagnostic era: pre-2019 (n=51) and \geq 2019 (n=17). Overall survival, molecular testing, use of targeted therapy and extent of surgery were analyzed.

Results

Median overall survival improved in the recent cohort: 1.06 years (range: 0.02-21.44) for patients diagnosed before 2019, compared with 1.82 years (range: 0.01-6.51) in post-2019 cohort, a statistical significant difference (log-rank test $p = 0.02$). Use of molecular diagnostics increased from 18% (9/51) before 2019 to 100% (17/17) thereafter.

Targeted therapy was administered to 4% (2/51) of pre-2019 patients, compared with 65% (11/17) post-2019. Among treated patients, the median survival was longer (1.88 vs 1.06 years) with a significant benefit in survival (log-rank $p = 0.007$). Infant subtypes, including Infant glioblastoma and infant hemispheric glioma (n=7) showed more favourable outcome, with 71% (5/7) achieving long-term survival. Two patients receiving molecular matched targeted therapies had exceptional survival: one with BRAF V600E-mutant gliosarcoma and an acquired PIK3CA mutation survived 5.6 years, and another with PDGFRA amplified glioblastoma survived 3.8 years. Extent of resection did not correlate with improved survival, likely reflecting underlying tumour biology and anatomical constraints..

Conclusion

Survival outcomes for pediatric HGG have improved in the modern era, driven by widespread adoption of molecular diagnostics and increased use of targeted therapies. These findings highlight the growing impact of precision medicine in this population.

The Paradoxical role of SIRT1 in Glioblastoma mediated by the tumor microenvironment.

Anza Mnahal

Department of Surgery, The University of Hong Kong

SIRT1, a NAD⁺-dependent deacetylase, plays a complex role in glioblastoma (GBM), with debates surrounding its function as either a promoter or suppressor of tumorigenesis. This study aims to understand the double roles of SIRT1 in GBM incorporating in vivo and in vitro models. We conducted a series of experimentation to understand the role of SIRT1 starting with comprehensive analysis of SIRT1 expression in patient datasets, followed by SIRT1 knockdown studies in various GBM cell lines (U87, U251, and patient-derived cells) and in orthotopic, subcutaneous and intracranial tumour induction in animal models. Pharmacological interventions using SIRT1 inhibitors were also employed. Our studies uncovered that higher the SIRT 1 expression level, better is the survival in low grade glioblastomas. On the other hand, knockdown of SIRT1 did not affect the in vitro cell proliferation or migration. However, there was significant effect of SIRT1 knockdown on tumor growth. The tumour growth decreased in both intracranial and subcutaneous models. Furthermore, we observed an altered interaction dynamics between tumour cells and the surrounding neurons, caused by SIRT1 knockdown. This indicates that the influence of tumour microenvironment on SIRT1's functional role. This study reveals the dual role of SIRT1 in GBM, highlighting it as a potential therapeutic target. The paradoxical role of SIRT1 inhibition in various models proves the importance of the tumor microenvironment in shaping GBM biology also providing a future target to study.

A proteogenomic atlas of 1,032 brain metastases identifies molecular subtypes, immune landscapes, and therapeutic vulnerabilities

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The molecular heterogeneity of brain metastases hampers therapeutic development for cures. To address this unmet and urgent need, we construct a comprehensive multi-omic, single cell, and spatially resolved atlas of 1,032 pan-cancer brain metastases, identifying four robust molecular subtypes with distinct biological programs and clinical associations. These brain metastases subtypes (BrMS) are defined by unique biological states: neural-like (BrMS1), metabolic (BrMS3), highly proliferative/immune-excluded (BrMS4), and an immune-infiltrated (BrMS2) state featuring a coordinated epithelial-mesenchymal transition program. Patient-derived organoids coupled with targeted drug screening indicate subtype-specific molecular dependencies and putative targets, notably mTOR signaling activation in BrMS3 and CDK4/6 axis activation in BrMS4, while BrMS1 and BrMS2 display distinct radiobiologic and immunologic signatures. This atlas provides a rigorous classification framework of BrMs and offers insights into subtype-specific molecular vulnerabilities.

Nuclear Condensates Drive a Novel Chemoresistance Mechanism in Glioblastoma

Wong Cheuk Lun Ethan*, Kiang Mei Yee Karrie, Leung Ka Kit Gilberto

Department of Surgery, The University of Hong Kong

Background:

Yes-associated protein (YAP) is a transcriptional effector of the Hippo pathway that is commonly overexpressed in cancer. Recent studies indicate that YAP can undergo liquid-liquid phase separation to form nuclear condensates that promote oncogenic transcription, yet the functional role of these condensates on treatment response remains largely unexplored. This study aims to determine whether YAP and its nuclear condensates contribute to temozolomide chemoresistance in glioblastoma (GBM).

Methodology:

Temozolomide-sensitive and temozolomide-resistant U87 and U251 GBM human cell lines were previously established. Temozolomide-resistant cells were maintained in low-dose temozolomide. Here, we investigate whether Hippo signaling contributes to temozolomide chemoresistance. By modulating the Hippo signaling pathway through short-hairpin YAP gene knockdown, the outcome on GBM cancer phenotypes was studied by cell assays (in vitro) and mouse orthotopic xenograft (in vivo). Finally, the therapeutic effect of YAP inhibitor verteporfin is studied.

Results:

Our data showed that YAP is overexpressed in temozolomide-resistant glioblastoma cells. Nuclear translocation of YAP (which becomes its active state) has increased in temozolomide-resistant cells. Temozolomide treatment promotes the formation of YAP nuclear condensates. YAP inhibition demonstrates reduced cell viability and higher susceptibility to temozolomide. Mice with tumor injection of the YAP knockdown cells have significantly reduced tumor size. Similarly, YAP inhibitor resensitizes temozolomide-resistant cells to temozolomide. Mechanistically, YAP knockdown reduces cell stemness, which in turn attenuates temozolomide chemoresistance.

Conclusions:

Our data suggested that YAP nuclear condensates drive temozolomide chemoresistance in glioblastoma. YAP represents a promising therapeutic target, and pharmacological disruption of YAP condensates may be a viable strategy to overcome temozolomide resistance in glioblastoma patients.

Single-Nucleus RNA Sequencing Characterizes Intracranial Germinoma and Its Developmental Origin

Wu Boxiang

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Intracranial germinoma is a central nervous system tumor mostly affecting children and adolescents. Despite survival rates exceeding 90% with chemoradiotherapy, long-term physical and psychological toxicities remain a substantial burden for pediatric and adolescent patients. The cellular origin and pathogenesis of intracranial germinoma are still unclear, hindering the development of effective targeted therapies.

Here, we conducted an analysis using the single-nucleus RNA sequencing (snRNA-seq) dataset of intracranial germinoma, as well as publicly available human embryonic and spermatogenesis atlases. In the low-dimensional embedding space, intracranial germinoma cells clustered adjacent to epiblast cells in the embryonic atlas and primordial germ cells (PGCs) in the spermatogenesis atlas. Our trajectory and pseudotime analyses placed the tumor cells as a branch derived from PGCs within the spermatogenetic lineage, further supporting a PGC origin of intracranial germinoma. Furthermore, intracranial germinoma exhibited high activity of the HIF and JAK–STAT pathways, with prominent activation of HIF-1 α and STAT1. In contrast, MAPK and PI3K pathway alterations reported in a previous genome-wide methylation study were not observed at the transcriptomic level.

Using the snRNA-seq dataset of intracranial germinoma, our study characterizes the transcriptional landscape of this tumor, provides new evidence supporting a primordial germ cell origin, and contributes to understanding the molecular mechanisms of its pathogenesis.

Elucidating the Lactylation-Dependent Regulation of Mitochondrial Transfer from Astrocytes to Glioblastoma Cells

You Zhong Sheng¹, Kiang M Karrie¹, Leung Ka-Kit Gilberto¹

¹Department of Surgery, The University of Hong Kong

Glioblastoma (GBM) remains the most aggressive intracranial tumor, characterized by a dismal prognosis with a five-year survival rate below 10%, largely due to its infiltrative nature, high heterogeneity, and profound therapeutic resistance. A critical mechanism contributing to GBM's resilience is intercellular mitochondrial transfer, a process wherein tumor cells hijack functional mitochondria from surrounding cells within the tumor microenvironment (TME), such as astrocytes. This acquisition enhances their metabolic capacity, proliferative potential, and resistance to chemoradiotherapy. Concurrently, the TME is characterized by high concentrations of lactate resulting from aberrant cancer metabolism (the Warburg effect). Lactate is now recognized not merely as a metabolic byproduct but as a substrate for lactylation, a novel post-translational modification that regulates diverse cellular processes. Recent studies have suggested a potential link between lactylation and organelle transport. However, whether protein lactylation directly modulates intercellular mitochondrial transfer in the context of GBM remains an unexplored and critical question. This study aims to investigate the hypothesis that protein lactylation, driven by the lactate-rich GBM microenvironment, plays a direct regulatory role in the molecular machinery governing mitochondrial transfer between astrocytes and glioma cells. Elucidating this potential mechanism could unveil a novel axis in GBM pathophysiology, offering new therapeutic targets to disrupt tumor progression and ultimately improve patient outcomes.