



# Asia Pacific Spatial and Single-Cell Biology Innovation Conference

June 20-21, 2025 | Thailand Bangkok

## ABSTRACT BOOK

### Transforming Biology and Medicine Through Spatial and Single-Cell Innovation

Explore the future of science and medicine at the Asia Pacific Spatial and Single-Cell Biology Innovation Conference in Bangkok. Delve into breakthroughs in tissue architecture, tumor heterogeneity, immunotherapy prediction, multi-omics, and AI-driven analysis. Join experts and researchers as they showcase the transformative impact of spatial and single-cell biology on biomedical research and clinical applications—all in the vibrant heart of Asia.

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# Welcome! Sawasdee! สวัสดี!

## **Asia Pacific Spatial and Single-Cell Biology Innovation (AP-SCIBIO 2025)**

June 20-21,2025  
SD Avenue Hotel, Bangkok, Thailand

The Organizing Committee extends a sincere and warm welcome to all participants of the Asia-Pacific International Conference on Science and Biological Engineering 2025 (AP-SCIBIO 2025), held in the vibrant city of Bangkok, Thailand. We are truly honored to host this distinguished gathering of researchers, academics, and industry professionals from across the Asia-Pacific region and beyond.

This conference serves as a crucial platform to explore the dynamic and rapidly evolving fields of science and biological engineering. We have curated a program that we believe showcases cutting-edge research, fosters insightful discussions, and encourages collaborations that will drive innovation and address some of the most pressing challenges of our time.

We are delighted to feature a diverse range of presentations from leading experts, covering a spectrum of topics within science and biological engineering. We trust that this conference will provide a stimulating environment for the exchange of knowledge, the sharing of novel findings, and the forging of lasting connections. It is our hope that AP-SCIBIO 2025 will inspire new perspectives and catalyze future advancements in these vital disciplines.

We wish you a productive, engaging, and memorable conference experience here in the captivating city of Bangkok.

Sincerely,

**AP-SciBio2025 Organizing Committee**

## Our Team



**Chanitra Thuwajit**  
Principal Investigator



**Somponnat Sampattavanich**  
Principal Investigator



**Punn Augsornworawat**  
Principal Investigator



**Natini Jinawath**  
Principal Investigator



**Varodom Charoensawan**  
Principal Investigator



**Peti Thuwajit**  
Principal Investigator



**Ponpan Matangkasombut-  
Choopong**  
Principal Investigator



**Rungdawan Wongsamart**  
Post-Doctorate Fellow



**Kessaya Waidee**  
Research Liaison

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# Symposium Agenda

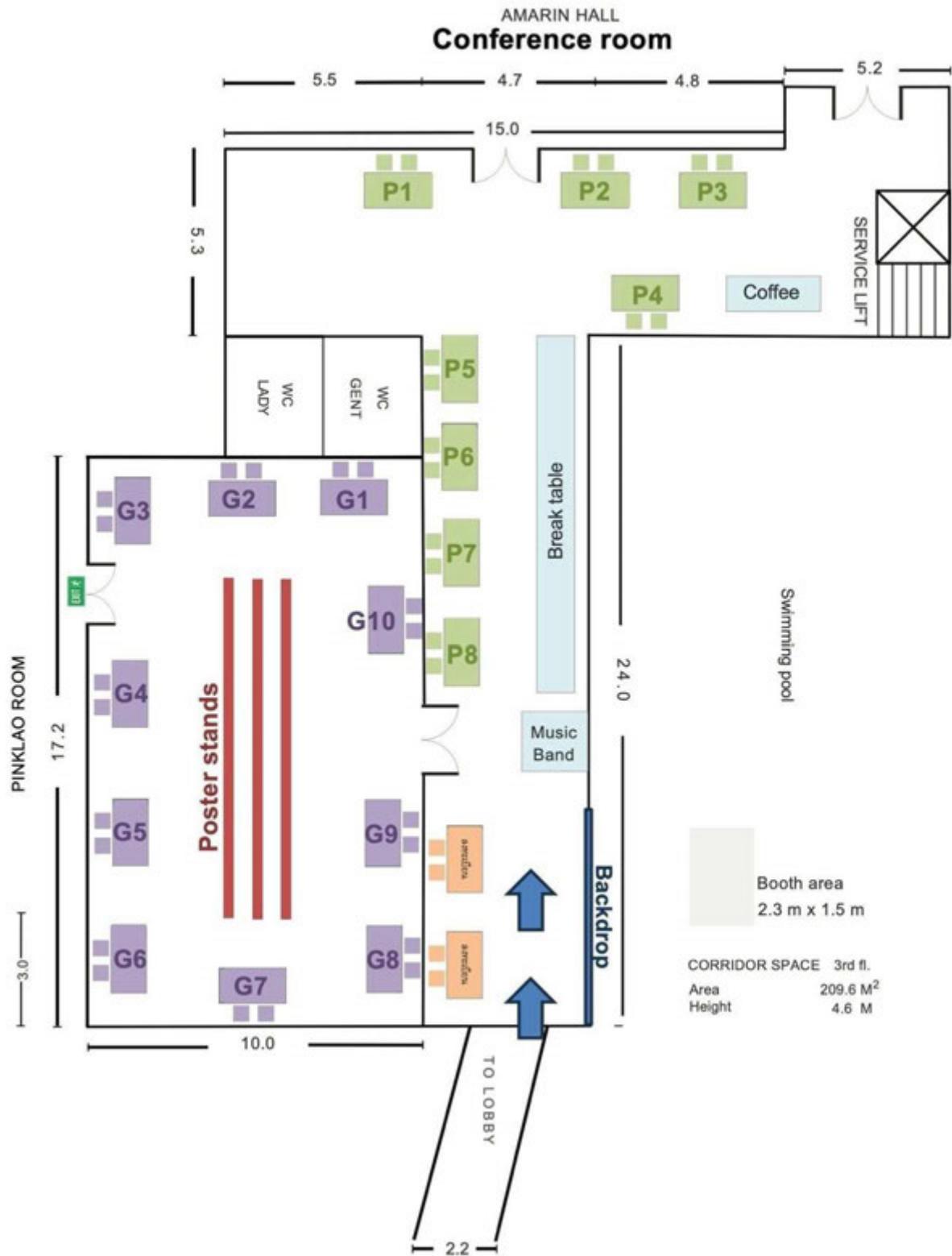
## Day 1 – June 20, 2025

TIME	EVENT
07.45-08.15	<b>Registration</b>
08.15-08.30	<b>Opening Ceremony</b>
08.30-09.20	<b>Plenary Talk 1:</b> Suzuki Yutaka, University of Tokyo, Japan
	<b>Session 1 – Decoding Tissue Architecture</b>
09.20-09.40	Shyam Prabhakar, Genome Institute of Singapore
09.40-10.00	Ankur Sharma, Garvan Institute
10.00-10.20	Varodom Charoensawan, Mahidol University
<b>10.20-11.00</b>	<b>Morning Break</b>
11.00-11.20	Worachart Lert-itthiporn, Khon Kaen University
11.20-11.40	Industrial Speaker: Paul Rasmussen, Genomax & Vizgen
11.40-11.50	Nikhil Rao, Syncell Inc. (Selected Short Talk)
<b>11.50-13.00</b>	<b>Lunch: Benton Berigan, Bruker Spatial Biology</b>
	<b>Session 2 – Harnessing Spatial and Single-Cell Insights to Understand Tumor Heterogeneity</b>
13.00-13.20	Arutha Kulasinghe, Wesley Research Institute
13.20-13.40	Ramanuj DasGupta, CRUK-Scotland & GIS
13.40-14.00	Ruby Yun-Ju Huang, National Taiwan University
14.00-14.20	Natini Jinawath, Mahidol University
14.20-14.40	Somponnat Sampattavanich, Mahidol University
14.40-15.00	Industrial Speaker: Advont Chua, Miltenyi Biotec + Hollywood International
15.00-15.10	Evgeny Denisov, Tomsk National Research Medical Center
<b>15.10-15.40</b>	<b>Afternoon Break</b>
	<b>Session 3 – Spatial and Single-Cell Approaches to Studying Diseases and Developing Immunotherapies</b>
15.40-16.00	Woong Yang Park, Seoul National University
16.00-16.20	Chanitra Thuwajit, Mahidol University
16.20-16.40	Methichit Wattanapanich, Mahidol University
16.40-17.00	Waradon Sungnak, Mahidol University
17.00-17.20	Industrial Speaker: Benson Lim, 10x Genomics
17.20-17.30	Pariyada Tanjak, Mahidol University (Selected Short Talk)
<b>17.30-19.00</b>	<b>Poster Session &amp; Networking</b>
19.00-22.00	<b>Gala Dinner</b>

## Day 2 – June 21, 2025

TIME	EVENT
07.45-08.15	<b>Registration</b>
08.10-08.20	<b>Morning Remarks</b>
	<b>Session 4 – Pioneering Advances in Spatial and Single-cell Multi-omics</b>
08.20-08.40	Sizun Jiang, Harvard Medical School
08.40-09.00	Tim Stuart, Genome Institute of Singapore
09.00-09.20	Ponpan M. Choopong , Mahidol University
09.20-09.40	Xi Chen, SUSTech
09.40-10.00	Industrial speaker: James Mansfield, Standard BioTools
10.00-10.10	Johnny Yu, Tahoe Therapeutics
<b>10.10-10.40</b>	<b>Morning Break</b>
	<b>Session 5 – Expanding Spatial and Single-cell Multi-omics to Broader Disease Applications</b>
10.40-11.00	Jong-Eun Park, KAIST
11.00-11.20	Punn Augsornworawat, Mahidol University
11.20-11.40	Pimpayao Sodsai, Chulalongkorn University
11.40-12.00	Saranyoo Ponnikorn, Thammasat University
12.00-12.20	Industrial Speaker: John Wei-Yuan Yu, MGI-Tech Singapore
12.20-12.30	Wenyang Dong, Changping Laboratory
<b>12.30-13.40</b>	<b>Lunch: Edwin Hauw, Element Bioscience</b>
13.40-14.30	<b>Plenary Talk 2:</b> Faisal Mahmood, Brigham and Women's Hospital, USA
	<b>Session 6: AI-Driven Computational Methods for Spatial and Single-Cell Omics</b>
14.30-14.50	Joe Poh Sheng Yeong, Singapore General Hospital
14.50-15.10	Dong Sung Lee, Seoul National University
15.10-15.30	Theerawit Wilaiprasitporn, VISTEC
<b>15.30-16.00</b>	<b>Afternoon Break</b>
16.00-16.20	Quan Nguyen, University of Queensland
16.20-16.40	Industrial Speaker: William Tan, Cytiva + Bang Trading
16.40-16.50	Desiree Abdurrachim, MSD
16.50-17.40	<b>Plenary Talk 3:</b> Kris Chatamra, Queen Sirikit Centre for Breast Cancer
<b>17.40-18.00</b>	<b>Poster Prize Announcement</b>

# Conference Layout



# **Speaker List**

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## **Plenary Speakers**

1. Suzuki Yutaka, University of Tokyo
2. Faisal Mahmood, Harvard University
3. Kris Chatamra, Queen Sirikit Centre for Breast Cancer

## **Invited Academic Speakers**

1. Shyam Prabhakar, Genome Institute of Singapore
2. Ankur Sharma, Garvan Institute
3. Varodom Charoensawan, Mahidol University
4. Worachart Lert-itthiporn, Khon Kaen University
5. Arutha Kulasinghe, Wesley Research Institute
6. Ramanuj DasGupta, CRUK-Scotland & Genome Institute of Singapore
7. Ruby Yun-Ju Huang, National Taiwan University
8. Natini Jinawath, Mahidol University
9. Somponnat Sampattavanich, Mahidol University
10. Woong Yang Park, Seoul National University
11. Chanitra Thuwajit, Mahidol University
12. Methichit Wattana8panich, Mahidol University
13. Waradon Sungnak, Mahidol University
14. Sizun Jiang, Harvard Medical School
15. Tim Stuart, Genome Institute of Singapore
16. Ponpan M. Choopong, Mahidol University
17. Xi Chen, Southern University of Science and Technology (SUSTech)
18. Jong-Eun Park, KAIST
19. Punn Augsornworawat, Mahidol University
20. Pimpayao Sodsai, Chulalongkorn University
21. Saranyoo Ponnikorn, Thammasat University
22. Joe Poh Sheng Yeong, Singapore General Hospital
23. Dong Sung Lee, Seoul National University
24. Theerawit Wilaiprasitporn, VISTEC
25. Quan Nguyen, University of Queensland

## **Industrial Speakers**

1. Paul Rasmussen, Genomax & Vizgen
2. Advont Chua, Miltenyi Biotec + Hollywood International
3. Benson Lim, 10x Genomics + Bio-Active Co., Ltd.
4. James Mansfield, Standard BioTools Inc. + Biomed Diagnostics
5. John Wei-Yuan Yu, MGI-Tech Singapore
6. William Tan, Cytiva + Bang Trading
7. Benton Berigan, Bruker & Prima Scientific
8. Edwin Hauw, Element Biosciences + Prima Scientific

## **Selected Short Talk Speakers**

1. Nikhil Rao, Syncell Inc.
2. Evgeny Denisov, Tomsk National Research Medical Center
3. Pariyada Tanjak, Mahidol University
4. Johnny Yu, Tahoe Therapeutics
5. Wenyang Dong, Changping Laboratory
6. Desiree Abdurrachim, MSD

## **Gala Dinner Speakers**

1. Manop Pithupakorn, Mahidol University
2. Kid Parchariyanon, SeaX Ventures
3. Arutha Kulasinghe, Wesley Research Institute
4. Ruby Yun-Ju Huang, National Taiwan University
5. Joe Poh Sheng Yeong, Singapore General Hospital

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# **Plenary Talks**

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## Plenary Talk 1

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### Title: Spatial analysis to reveal gene expression diversities

**Yutaka Suzuki**

Life Science Data Research Center, Graduate School of Frontier Sciences, The University of Tokyo

Short bio: Professor at the University of Tokyo's Department of Computational Biology and Medical Sciences, Yutaka Suzuki specializes in transcriptomics, gene expression, and promoter analysis. His research integrates multi-omics approaches to study diseases such as cancer and neuropsychiatric disorders.



**Abstract:** In this presentation, I'd like to discuss the latest spatial analytical platforms for understanding micro-heterogeneity of cancers. Particularly, a focus will be put on the interaction between tumor cells and their surrounding immune cells. Such a microenvironment is believed to be a decisive factor for tumor development and anti-cancer-drug responses. Recently, we have characterized spatial RNA profiles obtained from 30 lung adenocarcinoma patients at various stages, including non-invasive and later invasive stages. These samples were subjected to the spatial transcriptome sequencing analysis in conjunction with higher resolution analysis using *in situ* RNA profiling. The detailed inspection on each case and the following computational modeling based on the observed diverse profiles revealed that the drastic changes in phenotypic appearances of tumor cells are frequently triggered by their interaction with immune cells. The phenomena coincide with the induction of a series of cellular expression programs, which enable transforming tumor cells and their breaking-through against the immune cell barrier, collectively allowing their further progression. The study provides us with actual features how lung tumor develops through interaction within their microenvironments. Moreover, even newer platforms have been introduced to enable protein analysis on the same platform. I'd be happy if my presentation will be a good starting point to further discuss on the future perspectives on the spatial multi-omics analysis.

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## Plenary Talk 2

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### Faisal Mahmood

Brigham and Women's Hospital, USA

Short bio: Dr. Faisal Mahmood is an Associate Professor of Pathology at Harvard Medical School and Brigham and Women's Hospital. He leads the Mahmood Lab, focusing on developing machine learning and medical image analysis methods for objective diagnosis, prognosis, and biomarker discovery in computational pathology.



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## **Plenary Talk 3**

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### **Title: The Breast: carcinoma to single cells**

**Kris Chatamra**

Queen Sirikit Centre for Breast cancer, Thailand

Short bio: Dr. Kris Chatamra is associated with the Queen Sirikit Centre for Breast Cancer in Thailand and has delivered plenary talks on breast cancer, focusing on the progression from carcinoma to single cells.



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# **Session 1**

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## Title: Spatial and single cell omics reveals the diversity of cells and humans

**Shyam Prabhakar**

Genome Institute of Singapore, Singapore

Short bio: Senior Group Leader in Computational and Systems Biology at A\*STAR's Genome Institute of Singapore, Shyam Prabhakar holds a PhD in Applied Physics from Stanford University. His lab focuses on gene-regulatory mechanisms in diseases using single-cell and spatial omics technologies.



**Abstract:** To characterize the spatial architecture and cellular interactions of tumours in colorectal cancer (CRC), we used Xenium spatial RNA profiling on over 9 million cells from 63 samples and 34 patients. Our results reveal how the organized architecture of the normal colon becomes disrupted in cancer. We identify molecular markers related to biological signatures such as stem-like properties and response to hypoxia that exhibit spatial patterning within tumor glands. We also identify tumor-enriched spatial neighborhoods, including a tumor budding neighborhood enriched at the tumor-normal interface of invasive samples.

Switching to human diversity, we generated the Asian Immune Diversity Atlas (AIDA), a multi-national single-cell RNA-sequencing (scRNA-seq) healthy reference atlas of human immune cells. AIDA comprises 1,265,624 circulating immune cells from 619 donors, spanning 7 population groups across 5 Asian countries, and 6 controls. We found that sub-continental diversity, age, and sex pervasively impacted cellular and molecular properties of immune cells, with implications for disease risk and mechanisms. AIDA enables analyses of multi-ancestry disease datasets and facilitates the development of precision medicine efforts in Asia and beyond.

## Title: Understanding embryonic origins of tumours

Ankur Sharma

Laboratory Head Oncofetal Ecosystem • Translational Genomics, Garvan Institute of Medical Research, Australia

Short bio: Dr. Ankur Sharma leads a laboratory at the Garvan Institute of Medical Research and is recognized for discovering the Oncofetal ecosystem in liver cancer. He specializes in single-cell genomics and spatial transcriptomics.



**Abstract:** Cancer cells exhibit behaviour similar to fetal tissues, like quick growth, cellular reprogramming, and immune system suppression. We discovered the 'fetal-like' reprogramming of microenvironment in liver tumors, which we termed as 'Oncofetal ecosystem'. This unique microenvironment suppresses immune response. We also found cells that fetal-like reprogramming impacts the effectiveness of cancer treatments. This fetal-like reprogramming also appears during inflammation and in other solid tumors. In this talk I will discuss implication of oncofetal ecosystem and spatial transcriptomics in precision oncology and predicting immunotherapy response.

## **Title: Exploring Cellular Complexity and Immune Profiles in a Thai population**

**Varodom Charoensawan**

Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Short bio: Associate Professor at Mahidol University's Faculty of Science, Varodom Charoensawan focuses on systems biology, bioinformatics, and gene regulation. He earned his PhD from the University of Cambridge and leads the Integrative Computational BioScience Centre.



**Abstract:** Human variation starts at the molecular level, from the genetic information imprinted in the DNA to the ultimate phenotypic outcomes, including cellular variations, physiological processes, physical development and susceptibility to illness. While the vast majority of human diversity studies focus on genomic variations, their outcomes are largely influenced by the regulation of gene expression. In this talk, I will describe our latest progress, in collaborations with several research groups in Thailand and worldwide, on establishing a single-cell omics facility. This effort has contributed to the country's core capacity and competency in using single-cell resolution technique to map out the cellular diversities and dynamics of human immune cells in response to external stimuli such as viral infections and cancer models. I will also give an overview of our latest contribution to the international consortium Human Cell Atlas (HCA) via the Asian Immune Diversity Atlas (AIDA) project. This project uncovers the landscape of the complex immune variations of diverse Asian populations, which have been influenced by different environments and endemic infectious diseases over several centuries.

# Title: Integrated Tissue-and-Blood Single-Cell Transcriptomic Profiling Reveals the Cellular and Immune Landscape of Meningioma

**Worachart Lert-itthiporn**

Department of Biochemistry, Faculty of Medicine, Khon Kaen University

Short bio: Lecturer in the Department of Biochemistry at Khon Kaen University, Worachart Lert-itthiporn's research encompasses bioinformatics, molecular biology, and oncology, with a focus on cholangiocarcinoma.



**Abstract:** Meningioma, the most common benign tumor of the central nervous system, can cause significant symptoms depending on location and grade. While single-cell RNA-sequencing (scRNA-seq) has mapped the immune niche of normal meninges, its extension to both tumour tissue and circulation is limited. This study aimed to characterize the cellular and immunological architecture of meningioma locally (tumour + adjacent dura) and systemically (peripheral blood). We analyzed two scRNA-seq datasets. (i) A public cohort comprising 5 meningiomas and 7 patient dura samples was processed with Seurat, DoubletFinder, marker-guided annotation, inferCNV, and donor-level hierarchical clustering. (ii) Pre-operative peripheral-blood mononuclear cells (PBMCs) from 5 meningioma patients and 5 controls were profiled with 10x Genomics v3.1 and demultiplexed with Demuxlet/Cell Ranger. The outcome demonstrated that endothelial, mesenchymal, and immunological cells are the main cell types, with comparable relative abundances. The mesenchymal clusters were predisposed towards neoplastic characteristics with high copy number variation, high expression of tumor markers, and distinct person clustering pattern. Systemically, NK, CD4+ T, and B cells were higher in meningioma patients than in controls, while CD8+ T, monocyte, and dendritic-cell (DC) frequencies were lower. Pathway enrichment revealed up-regulation of CD8-activation and vacuolar-acidification signatures in CD8+ T cells and DCs, respectively. Collectively, these findings demonstrate that meningioma causes mesenchymal genetic instability and noticeable systemic immunological changes while maintaining a generally stable local immune environment. A framework for analyzing tumor-stroma crosstalk and creating immune-based biomarkers for meningioma treatment is provided by the integrated tissue-and-blood single-cell atlas.

**Keywords:** Meningioma, Single-cell RNA sequencing (scRNA-seq), Immune microenvironment, Peripheral blood mononuclear cells (PBMCs)

# Title: From Surface to Subcellular Depths: High-Resolution Spatial Biology with MERFISH 2.0

**Paul Rasmussen**

VP of Sales (APAC), Vizgen Inc.

**Short Bio:** Paul Rasmussen has over 25 years of experience in the biotech industry, spanning bench science, field applications, technical sales, and leadership. He has deep hands-on and commercial knowledge of the qPCR, microarray, NGS, and spatial biology universe, acquired through two decades at Applied Biosystems, NanoString Technologies, and now Vizgen. Currently based in Singapore, Paul is now responsible for overseeing Vizgen's commercial buildout in the Asia Pacific region.



**Abstract:** High-plex spatial transcriptomics at single-cell resolution has transformed biological research, offering unprecedented insights into complex systems. However, archived tissues preserved using formalin fixation and paraffin embedding (FFPE) pose a challenge due to degraded or crosslinked RNA, limiting transcriptomic analysis.

Multiplexed Error-Robust Fluorescence In Situ Hybridization (MERFISH) technology addresses this challenge by enabling direct RNA profiling *in situ* with exceptional sensitivity and spatial resolution. MERFISH achieves high accuracy and plexity by tiling probes along the length of transcripts—a method particularly difficult to apply in fragmented RNA samples like FFPE. To overcome this limitation, MERFISH 2.0 chemistry and an optimized sample preparation workflow were developed to improve transcript detection efficiency in low-quality tissues, supporting the measurement of up to 1,000 genes.

Implemented on the MERSCOPE Ultra™ Platform, MERFISH 2.0 supports tissue sections up to 3 cm<sup>2</sup> and expands the utility of spatial transcriptomics to degraded samples. With broad applications across biology, MERFISH 2.0 unlocks new possibilities for leveraging archival tissues in spatial genomics research.

## **Title: Study of whole cell proteome- the proteomic difference of astrocyte between mouse brain cortical and hippocampal regions (Selected Short Talk)**

**Nikhil Rao**

Syncell Inc.

Short bio: Chief Commercial Officer at Syncell Inc., Nikhil Rao holds a PhD in Bioengineering from the University of California, San Diego. He has held leadership roles at 10x Genomics and Becton, Dickinson & Company, contributing to advancements in spatial biology.



**Abstract:** Astrocytes are a subtype of glial cells in the Central Nervous System. They play roles in neurogenesis, synaptogenesis, blood-brain barrier permeability, and maintaining extracellular homeostasis. Interestingly, different populations of astrocytes localized in specific regions of the brain area have unique morphological and functional characteristics. The aim of this study is to characterize proteomic differences of astrocyte across various regions of the mouse brain. Here, we used a microscopy-based proteomics platform Microscoop® which perform ultra-content microscope-guided photo-biotinylation to render study of astrocyte proteomes in the cortical and hippocampal regions.

Astrocytes in mouse brain cryo-sections are immunostained against the glial fibrillary acidic protein (GFAP) and recognized by Microscoop® as the target at which the two-photon laser precisely illuminates. The light effectively activates proprietary photosensitive biotin-based probes free floating around the sample and triggers protein biotinylation. Under the Microscoop®, this cycle runs automatically across thousands of fields of view so that sufficient proteins are biotinylated for the subsequent pull down and LC-MS/MS-based proteome identification. This unique workflow, termed optoproteomics, allows subcellular proteomic discovery in high specificity, sensitivity and resolution.

In this study, proteomes of cortical or hippocampal astrocytes were established. Furthermore, well-known astrocytes markers including GFAP, LAMC3, PARK7 etc. were found. Upon comparing differences in the two proteomes, several candidates that have not been reported in existing literature and were more enriched in either cortex or hippocampus were selected for immunofluorescent staining. The cortex enriched candidates PLEKHB1 and SYBP3 colocalized with the astrocyte marker GFAP and are found to be more abundant in cortex astrocytes than that of hippocampus. In contrast, the hippocampus enriched candidate MINK1 were positively validated in hippocampus astrocyte but not in cortex astrocytes. Future study of the functional relevance of these protein candidates could help unravel the roles of regional specific astrocytes and generates multiple testable hypotheses. It also provides a method of defining cell types, subtypes, and states using whole proteome analysis vs. transcriptomic. The Microscoop® platform enables unbiased study of spatial proteomes, facilitating the discovery of novel astrocyte markers significant to specific brain region.

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# Luncheon 1

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## Title: Streamlining assay development for spatial proteomics

**Benton Berigan**

Bruker

Short bio: Benton Berigan, PhD is an interdisciplinary scientist leading the product development of the CellScape instrument. He received his PhD in Biological Sciences and completed a postdoctoral fellowship in Chemical and Biomedical Engineering at the University of Missouri, USA. During his research career, he developed a fluorescence-based approach to screen temperature-sensitive proteins for thermogenetic neurostimulation. Additionally, he pioneered a technique to study bladder function in mice using a custom miniaturized fluorescence microscope (miniscope).



**Abstract:** Developing custom assays for spatial analysis can often be a time-consuming and complex task. The CellScape™ Precise Spatial Proteomics instrument, together with EpicIF™ technology, offers an efficient and reliable solution, streamlining the process from project initiation to high-quality data generation.

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## **Session 2**

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# **Title: Spatial medicine in lung cancer - insights gained from ultradeep profiling of the tumour microenvironment**

**Arutha Kulasinghe**

Wesley Research Institute, Australia

Short bio: Associate Professor at the University of Queensland, Arutha Kulasinghe leads the Clinical-oMx Lab and serves as the Scientific Director of the Queensland Spatial Biology Centre. He has pioneered spatial transcriptomics and proteomics in the Asia-Pacific region.



**Abstract:** Immune checkpoint inhibitors (ICI) have improved clinical outcomes for patients with advanced non-small cell lung carcinoma (NSCLC), however a subset of patients remain treatment resistant. Spatial biology offers key intelligence into cellular coordinates *in situ*, with accurate interpretation requiring both biological and computational consideration. We analysed >600 NSCLC biopsies taken from patients prior to ICI treatment and applied a deep-learning model to classify cells into distinct phenotypes, map spatial regions for tissue compartments and metabolic neighbourhoods, and perform statistical comparisons for the measured features in these regions for clinical benefit at 6 months following ICI treatment. Geometric profiling of spatial interactions at a range of scales was accomplished through feature engineering, followed by statistically robust stability selection of features. Modelling of the patient cohort relapse events by survival analysis demonstrated that these features largely recapitulate a role for metabolic and dynamics of the tumour microenvironment and predict patient response to ICI.

# Title: Spatially Resolved Single Cell Analysis of Bevacizumab-induced Enhancement of anti-PD1 Response in Nasopharyngeal Cancer

Ramanuj Dasgupta

Cancer Research UK, United Kingdom

Short bio: Professor of Cancer Systems Biology at the University of Glasgow and Senior Group Leader at A\*STAR's Genome Institute of Singapore, Ramanuj Dasgupta specializes in precision oncology and cancer evolution. He obtained his PhD in Developmental and Stem Cell Biology from the University of Chicago.



**Abstract:** Clinical outcome data from our recent Phase II randomized study aimed at interrogating the benefit of combining anti-VEGF therapy (Bevacizumab) with immune checkpoint blockade (ICB, Pembrolizumab) in advanced nasopharyngeal cancer (NPC) showed a ~4x increase in overall survival rate (ORR) in the combinatorial arm compared to anti-PD1 monotherapy (Lancet Oncology 2025). In this study, we employed single cell RNA-seq and spatial transcriptomic analyses on longitudinal, serial biopsies from patients with matched clinical outcome data, to explore the underlying molecular mechanisms and prognostic biomarkers associated with how anti-angiogenic therapy may regulate anti-PD1 response. Notably, our single cell-spatiotemporal analyses revealed that one week of Bevacizumab monotherapy alone can remarkably alter and prime the tumour ecosystem into an immune-active state. Specifically, we elucidate the anti-VEGFA-induced alterations in cell types, cell states, and spatial neighbourhoods that promote vascular normalization, and a proinflammatory switch in myeloid cells as well as cancer-associated fibroblasts (CAFs), which facilitate reversal of immune tolerance, and restoration ICB response. Additionally, gene regulatory network analysis and interrogation of spatially-aware cell-cell interactions associated with emergence cell-states, and dynamic alterations in response-associated spatial niches, revealed potential pathways and cross-regulatory interactions that may control therapy response. Altogether, our study reveals a critical role for stromal remodelling in promoting immunotherapy response, while opening new avenues of investigation into alternative targets for combinatorial interventions to enhance immunotherapy response.

# Title: Epithelial-mesenchymal gradient and plasticity in ovarian clear cell carcinoma

Ruby Yun-Ju Huang

College of Medicine, International College, National Taiwan University,  
Taiwan

Short bio: Professor Ruby Yun-Ju Huang is a renowned clinician-scientist specializing in ovarian cancer and epithelial-mesenchymal transition (EMT) research. She is best known for defining the early events of EMT and proposing a paradigm shift from a binary to a continuous spectrum model.



**Abstract:** Ovarian clear cell carcinoma (OCCC) is a rare cancer type with significant relevance to East Asian women with critical unmet needs for novel therapeutic options. It is a histological subtype with distinct pathological features, molecular profiles, and biological functions. At the bulk transcriptomic level, there are distinct epithelial (EpiCC) and mesenchymal (MesCC) subtypes. EpiCC (epithelial-like), which is associated with early-stage disease, has a higher rate of mutations in the SWI/SNF complex, and exhibits higher expression of genes related to metabolic and metallothionein-binding pathways; and MesCC (mesenchymal-like), which is associated with late-stage disease and enrichment of genes involved in immune-related pathways and stromal signals. One crucial question is whether there is indeed molecular and functional transition between EpiCC and MesCC subtypes. We used OCCC cell lines to create an isogenic partial EMT (pEMT) model to explore the functional changes along the epithelial-mesenchymal gradient. OCCC cells undergoing pEMT had metabolic shifts and lost the expression of *LCN2*, an iron metabolism-related gene, possibly via the concomitant down-regulation of *SOX9*. *LCN2* expression correlated with a spatially resolved OXPHOS-enriched tumor signature, low EMT, and better outcomes in OCCC. Single-cell spatial transcriptomics profiling using CosMx further identified 9 geospatially distinct cancer cell populations including the *LCN2*-high cancer subclone with a high epithelial score. *SOX9* induction could partially restore the epithelial-ness in the *LCN2*-low cells suggesting that plasticity in OCCC could be achieved via transcriptional reprogramming. Our findings provide further insights in the epithelial-mesenchymal plasticity of OCCC.

## **Title: Spatial transcriptomics analysis of malignant transformation in HNSCC**

**Natini Jinawath**

Faculty of Medicine Ramathibodi Hospital Mahidol University

Short bio: Associate Professor and Head of the Translational Medicine Program at the Faculty of Medicine Ramathibodi Hospital, Mahidol University. Dr. Jinawath's research focuses on the molecular etiology of genetic diseases, including cancers and neurodevelopmental disorders, utilizing high-throughput omics technologies.



**Abstract:** Field cancerization refers to the presence of genetically altered cells extending beyond visible tumor margins, contributing to the development of second primary tumors and local recurrence, and resulting in poorer survival outcomes in patients with head and neck squamous cell carcinoma (HNSCC). To investigate the molecular signatures underlying this process, we performed spatial transcriptomic profiling on 20 HNSCC tissues from 10 HNSCC patients, each with matched primary tumor (PT) and second primary tumor (SPT) samples. Using a custom 321-gene panel, we identified spatially distinct markers associated with malignant transformation and field cancerization. Spatial mapping enabled precise localization of gene expression patterns across malignant transformation states of squamous epithelial cells and provided a method to identify molecularly positive surgical margins. These findings highlight the utility of spatial transcriptomics in characterizing epithelial gene expression patterns and suggest that incorporating spatial and cell-type-specific information may improve our understanding and prediction of HNSCC progression and prognosis.

# Title: Advancing Precision Medicine through Spatial Biology: Insights from Rare Cancer Applications

**Somponnat Sampattavanich**

Faculty of Medicine Siriraj Hospital, Mahidol University

Short bio: Assistant Professor in the Department of Pharmacology at the Faculty of Medicine Siriraj Hospital, Mahidol University. A King Scholar and MIT alumnus, Dr. Sampattavanich co-founded the Siriraj Laboratory for Systems Pharmacology. His research encompasses precision medicine, biosensor technology, and diagnostics.



**Abstract:** Understanding tumor heterogeneity is central to advancing precision oncology. Spatial biology, particularly when integrated with single-cell analysis, offers powerful tools to dissect the cellular composition and architecture of tumors with unprecedented resolution. In this presentation, I will share how we apply a range of spatial profiling technologies—including high-plex immunofluorescence and spatial transcriptomics—to investigate tumor heterogeneity in rare cancers such as tumor-induced osteomalacia (TIO), pancreatic cancer, and cholangiocarcinoma.

In TIO, we identified novel diagnostic biomarkers by spatially resolving protein expression patterns within tumor tissue, aiding clinical diagnosis in a disease often marked by elusive pathology. In pancreatic cancer, we analyzed the immune microenvironment across molecular subtypes—classical, basal, hybrid, and null—revealing distinct immunophenotypic features and stromal organization that may influence response to therapy. For cholangiocarcinoma, we are developing prognostic biomarkers based on spatially defined interactions between tumor and immune cells, offering new strategies for risk stratification and therapeutic targeting.

These examples underscore how spatial and single-cell technologies can illuminate the complexity of the tumor microenvironment and support the development of data-driven, personalized treatment approaches. Our findings demonstrate the critical role of spatially resolved biology in understanding tumor heterogeneity and translating molecular insights into clinical impact.

## **Title: Spatial protein and RNA analysis on the same tissue section with the MACSima Imaging platform.**

**Advont Chua Jia Wang**

Regional Product Marketing Manager, Miltenyi Biotec



Short bio: Advont graduated from the Nanyang Technological University Lee Kong Chian School of Medicine under the Interdisciplinary Graduate School's Future Medicine PhD Programme in 2018. He joined Miltenyi Biotec in 2019 as a Local Technical Support and Application Specialist specialising in lightsheet fluorescence 3D imaging of large organisms with the Ultramicroscope platform, and ultra-high plex imaging with the MACSima™ platform. After leading the Local Technical Support and Application team for 1.5 years, he moved to his existing role in 2023 as a Regional Product Marketing Manager. During this time, he and his collaborators established the Centre of Excellence for Spatial Biology and Immune Imaging at Singapore Immunology Network, A\*STAR, Singapore to promote innovation in biological research by providing access to cutting edge imaging techniques to local and regional research communities.

**Abstract:** The recent increase in image-based, spatially-resolved technologies enables researchers to profile the tumor microenvironment (TME) by capturing gene expression profiles within tissue sections. However, a significant limitation of these technologies is the lack of ability to resolve protein and RNA information in the same section, as well as conveniently analyse multimodal data sets. Here, we share a spatial RNA detection method, RNAsky, using Miltenyi Biotec's MACSima™ Platform as an automated, multiomic approach. Our method integrates spatial proteomics and transcriptomics data to provide in-depth profiling with single-cell resolution on the same tissue section. Using this approach, we combined a custom 48 plex RNA panel with a panel of 45 antibodies to conduct a multiomic profiling of colorectal cancer. Through our dedicated MACS® iQ View analysis software, spatially separated subpopulations of cancer associated fibroblasts (CAF) were revealed. Functional characterisation of cellular neighborhoods and the cell-to-cell interactions occurring within the TME revealed that one of the CAF populations inhibiting T cells. These findings will deepen our understanding of tumor progression in colorectal cancer and potentially other solid tumors.

# Title: Molecular makeup of early-onset oral cancer (Selected Short Talk)

Evgeny Denisov

Cancer Research Institute, Tomsk National Research Medical Center

Short bio: Dr. Evgeny Denisov is a cancer biologist at the Cancer Research Institute, Tomsk National Research Medical Center, Russia. He leads the Laboratory of Cancer Progression Biology and specializes in tumor metastasis, recurrence, and early-onset cancers. His research applies single-cell and spatial transcriptomics to study cancer evolution. He has published over 300 scientific works and holds 10 patents.



**Abstract:** Early-onset oral cancer (EOOC) demonstrates an increase in incidence, aggressiveness, and poor response to therapy. Classic etiological factors such as tobacco, alcohol, and human papillomavirus are not related to EOOC. Mechanisms of the development and progression of this cancer remain unclear. Here, we performed genome, exome, 16S rRNA metagenome, single-cell and spatial transcriptome sequencing of EOOC (<45 years old; n=35) as compared to oral cancer in older adults ( $\geq 45$  years; n=25). EOOC patients frequently harbored germline variants in the AHNAK2, KLK10, POPDC2, TTLL4 and other genes. EOOC demonstrated high frequency of somatic mutations in the TP53 and LOC112267881 genes, copy number aberration in the DEFB125 gene, and alterations in the genes involved in the MAPK and Rap1 signaling pathways. EPHB6 and CDKN2A mutations were related to EOOC recurrence. Tumor cells showed an increase in gene expression associated with phosphorylation, glycolysis, and non-canonical MAPK and Hippo signaling pathways. The EOOC microenvironment was found to be immunosuppressive due to increased number of M2 and TREM2+C1QB+ macrophages and myeloid-derived suppressor cells (MDSCs) and decrease in cytotoxic T and plasma cells. Nevertheless, the invasive edge demonstrated an immune response involving cytotoxic T and NK cells through GZMB-PGRMC1 and GZMB-IGF2R interactions. T cell-mediated immunity was also observed in adjacent normal tissue via upregulation of HLA-DR and T cell marker genes. In addition, EOOC invasive edge was enriched by vascular mimicry supported by M2 macrophages. EOOC microbiome was characterized by increase in Acidovorax caeni, Streptococcus infantis and Treponema amylovorum and decrease in Bacteroides acidifaciens, Bibersteinia trehalosi, Kocuria rhizophila and Pseudomonas veronii. Thus, EOOC demonstrates a distinct molecular makeup including specific cell populations, genes, mutations, signaling pathways, and bacterial species that can be potential diagnostic, prognostic, and therapeutic markers. This work was performed by The Consortium “Etiology and Pathogenesis of Oral Cancer in Young Adults” (EPOY: <https://epoy.org/>) and supported by Russian Science Foundation (# 22-15-00308).

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# **Session 3**

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# Title: Spatially resolved IFN- $\gamma$ signaling controls immunotherapy response

Woong-Yang Park

Geninus Inc. and Translational Genomics Center, Samsung Medical Center, Sungkyukwan University School of Medicine, Seoul Korea

Short bio: Professor at the Samsung Genome Institute, Samsung Medical Center, and Sungkyunkwan University, Seoul, South Korea. Dr. Park obtained his MD and Ph.D. from Seoul National University and completed postdoctoral training at Rockefeller University. His research focuses on cancer genomics, tumor evolution, and the tumor microenvironment.



**Abstract:** Immune checkpoint inhibitors (ICIs) targeting the PD-1 pathway have significantly advanced cancer treatment; however, their efficacy in colorectal cancer (CRC) remains limited primarily to tumors exhibiting deficient DNA mismatch repair (dMMR) or high microsatellite instability (MSI). The tumor microenvironment (TME) is increasingly recognized as a crucial determinant of therapeutic outcomes, encompassing intricate interactions among cancer cells, immune infiltrates, stromal elements, and vasculature arranged in specific spatial contexts. The spatial arrangement of immune cells and signaling pathways within tumors significantly influences immunotherapy effectiveness, contributing to variable responses among tumors with similar genomic profiles. Among critical immune pathways, interferon-gamma (IFN- $\gamma$ ) signaling emerges as a pivotal mediator of anti-tumor immunity, coordinating immune activation and tumor cell recognition. However, the precise spatial organization of IFN- $\gamma$  signaling within the CRC TME and its association with immune cell localization remain inadequately understood. We utilized high-resolution spatial transcriptomics at single-cell resolution to thoroughly characterize the immune landscape of colorectal tumors and identify determinants of response to anti-PD-1 therapy. By integrating spatial data with detailed transcriptomic profiles from tumor samples, we discovered that therapeutic efficacy hinges upon the spatial heterogeneity of IFN- $\gamma$  signaling. We identified distinct microenvironmental niches characterized by specific cellular compositions and spatial interactions significantly correlated with clinical outcomes independent of MSI status. We elucidate the profound impact of immune spatial architecture on immunotherapy response in colorectal cancer. The spatial organization of immune interactions emerges as a fundamental factor influencing clinical outcomes. This study not only identifies novel biomarkers for enhanced patient stratification but also reveals potential therapeutic targets for combinational approaches, aiming to optimize immunotherapy efficacy in both MSI and MSS colorectal cancers.

# Title: Spatial Dissection of Tumor–Microenvironment Interactions Reveals Drivers of Aggression, Immune Evasion, and Therapy Resistance

Chanitra Thuwajit

Department of Immunology, Faculty of Medicine Siriraj Hospital, Mahidol University



Short bio: Associate Professor and Head of the Department of Immunology at the Faculty of Medicine Siriraj Hospital, Mahidol University. Dr. Thuwajit's research centers on the tumor microenvironment and cancer immunotherapy, particularly the role of cancer-associated fibroblasts in various cancers.

**Abstract:** Spatially resolved transcriptomic, proteomic, and single-cell technologies offer unprecedented resolution to interrogate the tumor-stroma-immune interface across diverse cancer contexts. In this multi-cancer investigation, we employed spatial bulk transcriptomics, multiplex spatial proteomics, and spatial single-cell transcriptomics to elucidate the mechanisms by which tumor budding (TB), a morphological marker of invasion and metastasis, drives tumor progression and immune modulation in triple-negative breast cancer (TNBC) and colorectal cancer (CRC). In TNBC, spatial transcriptomic profiling of cancer-associated fibroblast (CAF)- and cancer cell-enriched compartments identified upregulation of pro-invasive and immunomodulatory genes, including *LIF*, *ZNF235*, *FAM83A*, *PRICKLE2*, *ATP7A*, and *EDIL3*, while CODEX-based spatial proteomics revealed marked depletion of cytotoxic immune infiltrates in high TB tumors. In CRC, GeoMx spatial transcriptomics demonstrated TB-associated elevation of *CTNNB1* and *CCND1* at the invasive front, correlating with poor prognosis; functional modeling in 3D spheroids confirmed suppression of β-catenin-mediated TB by curcumin. In non-small cell lung cancer (NSCLC), CosMx spatial single-cell transcriptomics delineated distinct CAF subpopulations, with enrichment of LGALS1 (Galectin-1)-expressing CAFs in immune checkpoint inhibitor (ICI)-non-responder, implicating stromal-derived immunosuppression in therapy resistance. Further spatial single-cell transcriptomic analyses of NSCLC samples stratified by ICI response status were planned to validate these findings. Additionally, integrated profiling of cancer and immune cell transcriptomes were performed to identify predictive biomarkers of ICI responsiveness. In conclusion, by leveraging spatially resolved technologies, we identified candidate molecular drivers and cell populations associated with aggressive phenotypes and therapy resistance. Our findings pave the way for precision theranostic strategies that target spatially defined tumor-stroma-immune interactions to improve patient outcomes.

# **Title: Single cell transcriptomic profiling reveals iPSC-derived NK cells as Functional Surrogates for Primary NK Cells in Cancer Immunotherapy**

**Methichit Wattanapanitch**

Siriraj Center for Regenerative Medicine, Research Department,  
Faculty of Medicine Siriraj Hospital, Mahidol University

Short bio: Associate Professor at the Siriraj Center for Regenerative Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University. Dr. Wattanapanitch specializes in developing cell-based therapies for regenerative medicine and studying disease pathogenesis using patient-specific induced pluripotent stem cells.



**Abstract:** Induced pluripotent stem cells (iPSCs) provide an unlimited source of cells capable of differentiating into all cell type in the body. At the SiCRM, we focus on generating iPSC-derived immune cells, including T cells, NK cells, and macrophages, for disease modeling and therapeutic applications. In this study, we established a feeder-free monolayer differentiation protocol to generate hematopoietic stem/progenitor cells (HSPCs) that mimics embryonic hematopoietic development. These HSPCs were further differentiated into functional NK cells (iNK cells). Single-cell RNA sequencing confirmed that the transcriptomic profile of our iNK cells closely resembles that of peripheral blood (PB)-NK cells. Importantly, our iNK cells demonstrated superior cytotoxicity against cholangiocarcinoma and breast cancer cell lines compared to the NK-92 cell line, both in monolayer and tumor spheroid cultures. Additionally, iPSCs can be genetically engineered with chimeric antigen receptors (CARs) to enhance immune cell targeting and antitumor function. Our platform offers great promise as a next-generation immunotherapy, paving the way for innovative cancer treatments.

## Title: Decoding Immune Responses to Viral Infections

**Waradon Sungnak**

Faculty of Science, Mahidol University

Short bio: Lecturer in the Department of Microbiology at the Faculty of Science, Mahidol University. Dr. Sungnak earned his Ph.D. in Immunology from Harvard University and completed a postdoctoral fellowship at the Wellcome Sanger Institute. His research involves single-cell omics analysis of immune responses and contributions to the human cell atlas.



**Abstract:** Single-cell omics technologies are redefining our ability to dissect immune responses to viral infections at high resolution, enabling integrated analyses of cellular phenotypes, transcriptional programs, and adaptive immune repertoires. These approaches facilitate the characterization of context-specific immune landscapes and the reconstruction of clonal architecture, gene usage patterns, and transcriptional signatures of adaptive immune cells through single-cell V(D)J sequencing. Applied to viral infections such as SARS-CoV-2 and dengue virus (DENV), we have revealed distinct immune cell states and repertoire features associated with infection status and clinical outcome. Cross-sectional and longitudinal profiling uncovers distinctions in effector and regulatory subsets, offering insights into potentially protective versus pathogenic immune responses. Together, targeted immune profiling and repertoire analysis help delineate potential mechanisms of immune activation, regulation, and dysregulation in the context of acute viral infections.

## **Title: Access the full richness of biological complexity with single cell and spatial multiomics**

**Benson Lim**

10x Genomics, Singapore



Short bio: Benson Lim is a Senior Science and Technology Advisor at 10x Genomics in Singapore, where he covers the SEA region, Singapore, and India. He has a background in molecular genetics and holds a Ph.D. from the University of Melbourne, Australia. Prior to joining 10x, Benson has held various roles in the biotech industry, including FAS Manager at MGI-Tech and Senior FAS at Bio-Rad Laboratories. Before transitioning to the industry, Benson dedicated over 10 years to the academic sector, where he published several papers and delivered research presentations at international conferences. Additionally, Benson actively contributes to the scientific community by serving as a reviewer for international journals.

**Abstract:** Developing treatments for complex diseases requires building a complete understanding of both disease and treatment-response mechanisms. As we navigate a century where transformative advances in biology will reshape the way we deliver human health, translational and clinical researchers need approaches that provide actionable insights that can, ultimately, be leveraged to improve how diseases are diagnosed and treated.

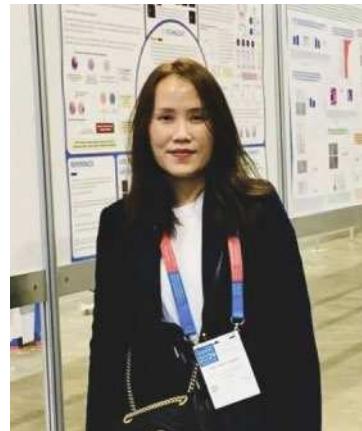
Join us to learn how single cell, spatial, and *in situ* innovations from 10x Genomics can help you push the boundaries of your translational and clinical research. Discover novel therapeutic targets, explore how therapeutics modulate disease-associated cell populations and states, gain insights into mechanisms governing therapeutic toxicity, and understand resistance mechanisms governed by transcriptomic and epigenetic remodeling. Enabling deeper insight into cancer, immunology, neuroscience, and immuno-oncology, 10x Genomics gives researchers the ability to see biology in new ways.

# **Title: Spatial single-cell image and transcriptomic profile resolve the heterogeneity of myeloid in the consensus molecular subtypes of metastatic colorectal cancer for precision immunotherapy (Selected Short Talk)**

**Pariyada Tanjak**

Siriraj Cancer Center, Faculty of Medicine Siriraj Hospital, Mahidol University

Short bio: Researcher at the Siriraj Cancer Center, Faculty of Medicine Siriraj Hospital, Mahidol University. Dr. Tanjak's work focuses on cancer biology, particularly colorectal cancer, and she has presented research on KRAS mutations and the tumor immune microenvironment.



**Abstract:** Molecular classification of colorectal cancer using bulk transcriptomics has identified subtypes with distinctive genotypic and phenotypic traits, commonly known as the consensus molecular subtypes (CMS). While CMS classification has clinical relevance in non-metastatic colorectal cancer, its applicability to metastatic colorectal cancer remains poorly understood. Here, we classified CMS on primary colorectal cancer tissues from 224 patients including 172 patients with non-metastatic colorectal cancer and 52 patients with metastatic colorectal cancer, using bulk transcriptomic sequencing. We further applied spatial transcriptomic profiling to decipher the effect of tumor microenvironment (TME) on CMS prediction in metastatic colorectal cancer. In addition to mapping the intratumoral heterogeneity of CMS and their associated microenvironment, we used spatial single-cell imaging to identify differences in the myeloid compartment, particularly macrophages, across CMS in metastatic colorectal cancer. We spatially localize the myeloid subsets based on CMS and their tissue localization between primary colorectal cancer and metastatic tissues. Spatial transcriptomic profiling revealed cancer cells were predominantly classified as CMS2, whereas CMS1, CMS3, and CMS4 were primarily localized within the TME. This finding suggested that the TME played a crucial role in influencing CMS classification. Spatial single-cell imaging identified macrophages and mast cell were increased in metastatic sites. We detected that SPP1+ macrophages with anti-inflammatory gene group (APOE, CD163) were highest in CMS4. Spatial single-cell image and transcriptomic profile revealed the molecular and immunological landscape of CMS-classified colorectal cancer. Our comprehensive analysis of the cellular landscape within the context of CMS colorectal cancer provides insights into its molecular heterogeneity, thereby contributing to the advancement of personalized therapeutic approaches.

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## **Session 4**

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## Title: Lessons from Spatial Omics: Do we need Everything Everywhere All at Once?

**Sizun Jiang**

Harvard Medical School, Harvard University, Massachusetts,  
United States of America

Short bio: Dr. Sizun Jiang is an Assistant Professor at Harvard Medical School and a Principal Investigator at Beth Israel Deaconess Medical Center. His research focuses on host-pathogen chromatin interactions, spatial omics technologies, and the development of tools like viralMIBI and CODEX for advanced imaging and sequencing.



**Abstract:** In this presentation, I will navigate the rapidly evolving landscape of spatial-omics technologies, challenging the prevailing assumption that more data inherently leads to better science. Drawing from our lab's work at Harvard Medical School, I'll demonstrate how the dazzling proliferation of multi-modal data acquisition methods often overshadows what matters most: thoughtful experimental design driven by specific biological questions. Through case studies in various host-disease interaction contexts, I will showcase how spatial-omics experiments can be elegantly iterative rather than exhaustively comprehensive. This approach allows researchers to interrogate the same precious tissue samples with sequentially deeper questions, strategically selecting modalities based on initial findings. I'll present our framework where each analytical layer builds upon previous discoveries, creating an efficient path to reveal fundamental biological insights about spatial organization in disease. The talk will offer practical strategies for researchers to escape the 'collect everything' trap and instead design hypothesis-driven spatial experiments that maximize biological insight while minimizing technical complexity and cost. I'll conclude with an outlook on how this philosophy can accelerate discovery across diverse disease systems while making spatial technologies more accessible to the broader scientific community.

## Title: Scaling single-cell epigenomic analysis with regulatory element modules

Tim Stuart

Genome Institute of Singapore (GIS), Singapore

Short bio: Dr. Tim Stuart is a Principal Investigator at the Genome Institute of Singapore. His work focuses on single-cell epigenomics, exploring how different cell states are encoded in the genome. He has contributed to the development of tools such as Seurat and Signac for analyzing single-cell transcriptomic and chromatin state data.



**Abstract:** Single-cell epigenomic assays offer new ways to understand how the activity of DNA regulatory elements may shape cellular states and fates. Recent advances in experimental methods now allow profiling of millions of cells, enabling a much more reliable quantification of these epigenomic states, particularly for rare cells. However, the analysis of such data is still impeded by two computational challenges. First, there is a lack of reusable features for epigenomic analysis, and each dataset requires de novo peak calling. This makes comparison of published datasets difficult, as they cannot be directly compared. Second, current analysis methods cannot scale to process large single-cell epigenomic datasets, requiring excessively long runtimes and large memory resources. To address these challenges developed a novel approach for single-cell epigenomic analysis based on feature aggregation. We leveraged epigenomic data from thousands of published datasets to identify groups of co-accessible regulatory elements, which we term Regulatory Element Modules (REMO). We further developed an open-source software toolkit, implemented in Rust, that provides fast and memory-efficient quantification of single-cell epigenomic data. REMO enables accelerated analysis of single-cell epigenomic data with lower memory requirements, and provides reusable features applicable to a broad range of tissue types, offering a robust framework for consistent and scalable analysis of single-cell epigenomic data.

## **Title: Heterogeneity of CD8+ T cells Response in Natural Dengue Virus Infection**

**Ponpan M. Choopong**

Faculty of Science, Mahidol University, Bangkok, Thailand



Short bio: Associate Professor Ponpan Matangkasombut Choopong is a faculty member at the Department of Microbiology, Faculty of Science, Mahidol University. Her research interests include immune responses in dengue viral infections, single-cell RNA sequencing in human immunology, and the Human Cell Atlas: Asian Immune Diversity Atlas.

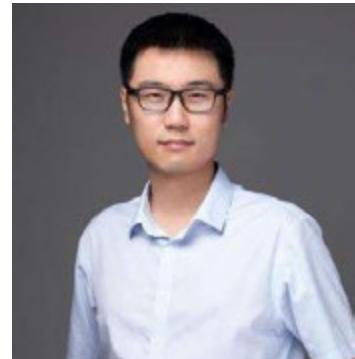
**Abstract:** A mosquito-borne dengue virus (DENV) infection remains a public health threat especially in tropical countries. In-depth understanding of systemic immune response to DENV that could provide either protection or adverse outcomes is needed. CD8+ T cells play major role in protection and immunopathogenesis but how they play the diverse role in different context is not clearly understood. Here, we single-cell immune profile peripheral blood mononuclear cells and DENV-specific CD8 T cells from DENV- infected donors across time and severity outcomes. We identified heterogeneity of CD8 and DENV-specific CD8+ T cells. Differential subcluster abundance and distinct TCR usage across disease severity outcome and disease course were observed. Overall, our study provides an in-depth understanding of CD8+ responses to DENV infection, revealing potentially distinct response for protection and pathogenicity, which could impact in vaccine and targeted treatment design.

## **Title: Dual-omics profiling of chromatin accessibility and 5' gene expression by SCHAFR-seq**

**Xi Chen**

SUSTech, China

Short bio: Dr. Xi Chen is an Associate Professor of Biostatistics at the University of Miami's Miller School of Medicine. He leads the Translational Statistical Bioinformatics Lab, focusing on integrating data science and statistics with genomic research to advance understanding of cancer and other complex conditions.



**Abstract:** Very few single-cell RNA-seq methods can capture 5' end of the mRNA. Previous studies have shown that 5' RNA-seq has the unique advantage of locating transcriptional start sites and capturing RNAs transcribed from active cis-regulatory elements. Here, we developed single-cell chromatin accessibility and five-prime RNA-seq (SCHAFR-seq) for the joint profiling of chromatin accessibility and 5' gene expression in the same cell. We demonstrated that SCHAFR-seq is sensitive, flexible and works across multiple platforms. The data provide valuable information about gene regulatory mechanisms that are often missed by standard 3'-based multiomic methods.

## Title: Scaling Up Discovery: Highplex Proteomic Imaging at High Throughput

**James Mansfield**

Standard BioTools

Short Bio: James R. Mansfield is a scientist with over 30 years of experience, the last 20 of which have been in multiplex pathology and the *in situ* phenotyping of immune cells. He is currently the VP of Imaging at Standard BioTools, where he is working on promoting and developing the Hyperion imaging system. Previously he was Senior Vice President at Visiopharm, where he was instrumental in developing their Phenoplex multiplex analysis software. Before that, he was the product manager for the Vectra multiplex imaging system and inForm analysis software at PerkinElmer. He is an associate editor of the American Journal of Nuclear Medicine and Molecular Imaging, holds 7 patents, has over 100 publications, and has served as an invited speaker, session chair, and organizer at a variety of international conferences.



**Abstract:** Spatial biology is rapidly emerging as a cornerstone of biomedical research, offering critical insights into tissue architecture, cell-to-cell interactions, and the microenvironment's role in health and disease. Within this field, spatial proteomic imaging is particularly valuable for mapping functional protein expression *in situ*, providing context-rich data that genomics and transcriptomics alone cannot capture. As the field matures, there is a growing need to move beyond small, exploratory datasets toward larger-scale, high-throughput studies that can power translational and clinical research.

Advancements in imaging mass cytometry (IMC) are meeting this need by enabling highplex proteomic imaging at unprecedented throughput. With new acquisition modes—tissue mode for rapid survey imaging, preview mode for region-of-interest targeting, and cell mode for detailed single-cell resolution—IMC now supports the acquisition of quantitative, multiplexed datasets from over 100 slides per week. Its ease of assay development (often completed within half a day) and high linear dynamic range further position IMC as an ideal platform for scaling spatial proteomic discovery from bench to bedside.

## **Title: Tahoe-100M: A Giga-Scale Single-Cell Perturbation Atlas for Context-Dependent Gene Function and Cellular Modeling (Selected Short Talk)**

**Johnny Yu**

Tahoe Therapeutics

Short bio: Dr. Johnny Yu is the Chief Scientific Officer and Co-founder of Vevo Therapeutics, a biotechnology company specializing in single-cell drug discovery. He previously conducted research at UCSF, focusing on RNA-binding proteins and single-cell RNA sequencing, contributing to significant publications in cancer genomics.



**Abstract:** Building predictive models of the cell requires systematically mapping how perturbations reshape each cell's state, function, and behavior. Here, we present Tahoe-100M, a giga-scale single-cell atlas of 100 million transcriptomic profiles measuring how each of 1,100 small-molecule perturbations impact cells across 50 cancer cell lines. Our high-throughput Mosaic platform, composed of a highly diverse and optimally balanced “cell village”, reduces batch effects and enables parallel profiling of thousands of conditions at single-cell resolution at an unprecedented scale. As the largest single-cell dataset to date, Tahoe-100M enables artificial-intelligence (AI)-driven models to learn context-dependent functions, capturing fundamental principles of gene regulation and network dynamics. Although we leverage cancer models and pharmacological compounds to create this resource, Tahoe-100M is fundamentally designed as a broadly applicable perturbation atlas and supports deeper insights into cell biology across multiple tissues and contexts. By publicly releasing this atlas, we aim to accelerate the creation and development of robust AI frameworks for systems biology, ultimately improving our ability to predict and manipulate cellular behaviors across a wide range of applications.

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# **Session 5**

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## Title: Cross-tissue single-cell and spatial cell atlas to understand human diseases

Jong-Eun Park

Korea Advanced Institute of Science & Technology (KAIST),  
Daejeon, Republic of Korea

Short bio: Dr. Jong-Eun Park is an Assistant Professor at the Graduate School of Medical Science and Engineering, Korea Advanced Institute of Science and Technology (KAIST). Her research encompasses single-cell transcriptomics and its applications in understanding neurological disorders.



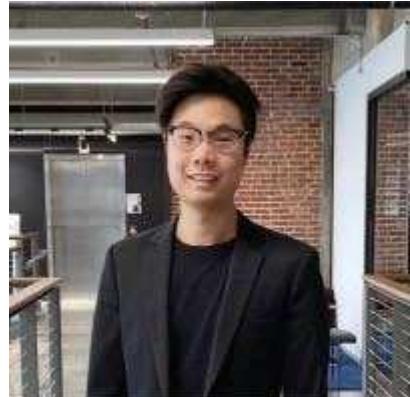
**Abstract:** The recent advent of single-cell RNA sequencing technology has enabled the detailed characterization of human cells in various organs from diverse disease states. As single-cell data continues to pour out, it has become crucial to integrate them effectively into a comprehensive atlas. However, the discrepancy in metadata terminology and bioinformatic analysis pipeline across publicly deposited datasets often hinders the integration at the cell count matrix level. In this study, using an automatic public data search process, we unbiasedly collected over 20 million single-cell transcriptomic profiles from more than 500 independent studies, which contain more than 2,000 single-cell transcriptome datasets from diverse human organs and disease states. We invented a single-cell data remapping pipeline for the efficient re-analysis of the whole dataset from the raw sequence files at its highest genome coverage while excluding the biases from computational data processing steps. Metadata information has been curated and classified to provide harmonized terminology for the entire dataset. The integration of remapped single-cell transcriptome dataset minimizes the batch effect, allowing for the robust identification of cell types and the organ-specific, disease-specific, and sex-specific gene signatures for each cell type. As our remapping pipeline utilizes a genomic binning approach, the splicing patterns and intergenic transcripts were also retrieved, maximizing the interpretability of the single-cell transcriptome. Using this reference atlas of human cell types, we provide a universal reference for the deconvolution and interpretation of multi-organ spatial transcriptomics data collection. Finally, we have applied large language model to replicate the manual curation process, which could reach up to ~90% accuracy. In conclusion, we represent a fully curated, annotated, and harmonized cell network that could provide a fundamental axis for future data integration.

# Title: Maturation of $\beta$ Cells in Human Pancreatic Islets

Punn Augsornworawat

Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Short bio: Dr. Punn Augsornworawat is a faculty member at the Department of Immunology, Faculty of Medicine Siriraj Hospital, Mahidol University. His research focuses on regenerative medicine, single-cell sequencing, and stem cell-derived islets for diabetes therapy.



**Abstract:** Type 1 diabetes (T1D) is caused by autoimmune-mediated destruction of pancreatic islets, resulting in insulin deficiency. Although islet transplantation can restore glycemic control, donor islets are scarce and unsuitable for widespread clinical use. Stem cell-derived islets (SC-islets) offer a scalable alternative, as human pluripotent stem cells can be differentiated into insulin-producing cells. However, these SC-islets remain functionally immature compared to native islets. Single-cell transcriptomic analyses reveal significant differences between SC-islets and native human islets, including divergent gene expression profiles, epigenetic landscapes, and microenvironmental contexts. Native islets also contain rare, specialized cell types that may contribute to proper islet function and structural organization. These insights underscore the need to better understand the cellular complexity of native islets to guide the development of more mature and functional SC-islets for regenerative therapy.

# Title: Single-cell RNA sequencing reveals the Immunological profile of neonatal umbilical cord blood monocytes in newborn infants of chronic HBV-infected mothers

Pimpayao Sodsai

Department of Microbiology, Chulalongkorn University, Bangkok, Thailand



Short bio: Dr. Pimpayao Sodsai is affiliated with Mahidol University and has contributed to research on the complete genome sequences of *Mycobacterium farcinogenes* strains isolated from clinical specimens in Thailand.

**Abstract:** Hepatitis B virus (HBV) infection is a major global health concern, causing liver inflammation and cancer in chronically infected patients. Newborns who acquire HBV from infected mothers are more susceptible to chronic development than adults. Trained immunity triggered by HBV exposure in utero has been reported, characterized by innate immune cell maturation and increased ability of cord blood (CB) immune cells to respond to unrelated pathogens in vitro. However, the mechanisms controlling this phenomenon remain unclear. To assess the impact of HBV exposure on newborns, we conducted immunological and epigenetic analyses of CB monocytes from neonates born to HBV-infected mothers.

**Methods:** Umbilical CB samples were collected from neonates born to both HBV chronically infected and healthy mothers during delivery. CD14<sup>+</sup> monocytes were isolated and stimulated with a TLR8 agonist. Cytokine profiles in the cultured supernatant were measured using Luminex assay. Expression levels of histone modification enzyme genes in HBV-exposed CB monocytes were evaluated via Taqman real-time PCR technique. Additionally, we investigated H3K4me3 levels of selected genes using chromatin immunoprecipitation quantitative PCR. Transcriptomic profiles of CB monocyte populations were assessed using single-cell RNA sequencing.

**Results:** Our findings revealed significantly upregulated production of IL-12p40 but not other cytokines including IL-6, IL-1 $\beta$ , TNF- $\alpha$ , IFN- $\alpha$ 2, IFN- $\gamma$ , and IL-10 in HBV-exposed CB monocytes compared to healthy controls upon stimulation. To elucidate the role of epigenetics in regulating HBV-trained immunity in monocytes, we examined mRNA expression of histone modification enzymes, which showed no significant difference between HBV-exposed CB monocytes and healthy controls. Similarly, H3K4me3 levels of selected genes (IL-12p40, IL-6, IL-10, TNF- $\alpha$ , and IL-1 $\beta$ ) at the promoter regions of stimulated CB monocytes did not differ between HBV-exposed CB monocytes and healthy controls. Further characterization of transcriptomic profiles at single-cell resolution identified 10 monocyte subclusters resembling both monocyte and macrophage subpopulations. Notably, the most abundant cell type in unstimulated HBV-exposed CB samples was M2 macrophages (40.8%), compared to controls (19.8%). Upon stimulation, the frequency of activated M2 macrophages increased in HBV-exposed CB, while activated M1 macrophage frequency increased in healthy controls.

# **Title: Spatial Decoding of Hair Loss: Mapping the Immune–Hair Follicle Interface in Androgenetic alopecia**

**Saranyoo Ponnikorn**

Chulabhorn International College of Medicine, Thammasat University

Short bio: Dr. Saranyoo Ponnikorn is an Assistant Professor at Chulabhorn International College of Medicine, Thammasat University. He completed his Ph.D. in Biochemistry at Mahidol University and specializes in molecular biology, cell signaling, stem cell translational research, proteomics, and bioinformatics.



**Abstract:** Androgenetic alopecia (AGA) is characterized by progressive hair follicle (HF) miniaturization, depletion of progenitor cells, and localized immune dysregulation. To elucidate the spatial mechanisms underlying these processes, we employed spatial transcriptomics on scalp biopsies from patients with early androgenetic alopecia (AGA) and controls. Our analysis revealed distinct immunological landscapes in peri-infundibular regions, characterized by the enrichment of CD4<sup>+</sup> T cells and a Th2-skewed response. Concurrently, progenitor cell zones exhibited upregulation of EMT- and ECM-related genes such as TGFB2, FN1, and TWIST1, alongside elevated protein expression, indicating a fibrotic shift in the HF niche. These spatially correlated immune and molecular changes suggest that chronic microinflammation contributes to the exhaustion of stem cells and tissue remodeling. Our findings underscore the utility of spatial transcriptomics in decoding immune–epithelial crosstalk in AGA and point toward novel therapeutic targets for regenerative intervention.

# Title: Deciphering Transcript Heterogeneity in Early Zebrafish Development with Single-Cell Long-Read Sequencing

John Wei-Yuan Yu

MGI

Short Bio: Dr. John WEI-YUAN YU works as a Senior Business Development at MGI-TECH APAC, since 2022. He is the Founder of IUVENTUS Pte Ltd. Previously, he was employed as Chief Scientific Officer at Lifestrands Genomics till April 2022. Dr. Yu completed his PhD in Biology and Biochemistry from the University of Bath. He has a course Masters in Business Administration and Management, General from The London School of Economics and Political Science (LSE). He completed his Bachelor's degree in Public Health from Taipei Medical University. He is keen in developing the market for the single cell and spatial genomics technology and is also working in clinical WGS projects.



**Abstract:** Recent advances in long-read sequencing have unlocked new potential for resolving transcript isoform diversity at single-cell resolution. In this talk, we present the first application of MGI's single-cell RNA-seq combined with CycloneSeq—a nanopore-based long-read technology—on over 35,000 zebrafish embryonic cells. This integrated workflow enables accurate, full-length isoform reconstruction across diverse developmental cell types. We benchmark its performance against established platforms highlighting CycloneSeq's unique strengths: high isoform recovery (>90%), detection of >8,000 novel splicing events, and scalable throughput at reduced cost. Our findings demonstrate that MGI DNBeLab TaiM4 single cell RNAseq library + CycloneSeq is a competitive and accessible platform for large-scale, single-cell transcriptomics and splicing analysis. This study sets a new standard for decoding transcriptional complexity during development and showcases the potential of cost-effective long-read strategies in single-cell omics.

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# Luncheon 2

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## Title: High Dimensional Biology: Single Cell & Spatial Multiomics Powered by AVITI24

**Edwin Hauw**

Element Biosciences

Short bio: Edwin is a highly accomplished biotechnology and life sciences executive with over 20 years of marketing and product management experience. Before joining Element, he served as Vice President of Marketing at 10X Genomics, where he steered product management efforts and marketing. Over his six-year tenure, he played a pivotal role in propelling the business growth to exceed \$500 million. Prior to this, Edwin was Senior Director of Product Management at Pacific Biosciences, guiding commercial and product development. His experience also includes senior roles at Applied Biosystems and Affymetrix, Inc. Edwin began his career as a software engineer. He holds an MBA with a focus on Finance and Marketing from the University of Southern California, Marshall



**Abstract:** Today, NGS technologies are increasingly used as a read out for multi-omics studies. However, researcher often rely on multiple assays and instruments upfront to generate segmented multiomic data. Each assay can be time-consuming, resource-intensive, expensive, and susceptible to batch effects. Moreover, sample preparation requirements may be mutually incompatible, posing additional challenges. These factors necessitate difficult choices in experimental design to maximize throughput and the value of datasets.

We present AVITI24, the first fully integrated platform capable of providing reliable information on RNA and protein expression, localization, genomic sequence, and morphology at the subcellular level from a single sample. The Teton assays for AVITI24 enable, for the first time, simultaneous multiomic profiling of up to 1–2 million cells per run. Through Avidity Sequencing™ Chemistry, a novel probe scheme, subcellular resolution imaging, and integrated AI-based primary analysis, we have created a powerful assay engine for single-cell and spatial applications.

The AVITI24 system can process up to 24 samples simultaneously today, delivering high-plex mRNA, protein, targeted sequencing, cell-paint phenotyping, and morphology data in less than 24 hours with a 30-minute hands-on workflow. We will showcase applications demonstrating the value of observing the time course of cell dynamics across a myriad of use cases. Finally, we will describe an exciting roadmap ahead.

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# **Session 6**

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# Title: Spatial-powered AI Pathology H&E 2.0, Spatial Proteogenomics & Spatial Medicine: A new hype of new hope?

**Joe Yeong**

Institute of Molecular and Cell Biology (IMCB), A\*STAR, Singapore

Short bio: Dr. Joe Yeong is a Group Leader at the Institute of Molecular and Cell Biology (IMCB), A\*STAR, Singapore, and serves in the Department of Anatomical Pathology at Singapore General Hospital. His research focuses on immunopathology, immune microenvironment across various cancers, immunoprofiling for immunotherapy, and the development of multiplex IHC and spatial transcriptomics techniques.



**Abstract:** The growing cancer patient demographic far exceeds the limited availability of clinical trials, with delays in trial matching and patient recruitment contributing to a >90% trial failure rate and an annual loss of >USD 9 billion. Immune profiling, including immunohistochemistry (IHC) and multiplex IHC, is essential for efficient trial recruitment and biomarker discovery to identify patient subsets who may benefit from trials. However, its widespread adoption is hindered by high costs, technical complexity, limited tissue availability, and labour-intensive processes. Given our track record in spatial biology and developing AI tools to advance drug development in clinical trial (on-going Phase 1b), it demonstrates our ability to integrate cutting-edge technology and positions us uniquely to innovate effective solutions. The current trend of combining immunotherapy with antibody-drug conjugates increases the need for biomarker testing, both individually and in combination, to guide therapeutic decision. With challenges such as limited tissue availability, high cost, and manpower constraints, artificial intelligence (AI) presents a promising solution. In this talk, I will mainly present how to accelerate the process of patient triage during clinical trial recruitment by developing a functional prototype that leverages on our AI-guided concept known as H&E 2.0, for the screening of multiple actionable and clinically relevant predictive biomarkers for clinical trials. On the other hand, in the horizon of spatial medicine, using a recently published study (Nature 2025), I will also demonstrate how to leverages spatial proteogenomics to provide critical insights into the TME and guide clinical decision-making. Spatial proteogenomics integrates spatial proteomics and proteogenomics using LC-MS/MS technology to map protein expression within tissue regions while linking it to genomic and transcriptomic variations. This approach goes beyond traditional proteomics, uncovering not only canonical proteins but also mutant and “dark” proteins—small, noncanonical proteins critical to tumor progression and immune modulation. By providing spatial and molecular insights into tumor heterogeneity and immune interactions, spatial proteogenomics offers unparalleled potential for advancing cancer biology and precision oncology.

## **Title: Epigenomic and chromosomal architectural reconfiguration in developing human brain**

**Dong Sung Lee**

Department of Medicine Seoul National University College of Medicine, South Korea

Short bio: Dr. Dongsung Lee is affiliated with Seoul National University and has research interests in single-cell multi-omics, genomics, epigenomics, next-generation sequencing, and DNA studies.



**Abstract:** The human frontal cortex and hippocampus play critical roles in learning and cognition. We investigated the epigenomic and 3D chromatin conformational reorganization during the development of the frontal cortex and hippocampus, using more than 53,000 joint single-nucleus profiles of chromatin conformation and DNA methylation (sn-m3C-seq). The remodeling of DNA methylation predominantly occurs during late-gestational to early-infant development and is temporally separated from chromatin conformation dynamics. Neurons have a unique Domain-Dominant chromatin conformation that is different from the Compartment-Dominant conformation of glial cells and non-brain tissues. We reconstructed the regulatory programs of cell-type differentiation and found putatively causal common variants for schizophrenia strongly overlap with chromatin loop-connected, cell-type-specific regulatory regions. Our data demonstrate that single-cell 3D-regulome is an effective approach for dissecting neuropsychiatric risk loci.

# **Title: Hybrid Calibration for Robust Tumor Proportion Score Prediction in Non-Small Cell Lung Cancer Using Sparse Annotations and Unsupervised Adaptation**

**Theerawit Wilaiprasitporn**

School of Information Science and Technology, Vidyasirimedhi  
Institute of Science and  
Technology, Rayong, Thailand

Short bio: Dr. Theerawit Wilaiprasitporn is a faculty member at the Vidyasirimedhi Institute of Science and Technology (VISTEC), specializing in neural engineering technology for closed-loop EEG-based brain-computer interfaces.



**Abstract:** Accurate estimation of the Tumor Proportion Score (TPS) is critical for guiding immunotherapy in non-small cell lung cancer (NSCLC). Deep learning models applied to whole-slide images (WSIs) have emerged as promising tools for automated TPS assessment. However, these models frequently exhibit poor generalization across laboratories due to domain shifts arising from minor variations in staining protocols and scanner settings. This challenge is further exacerbated by the practical scarcity of annotated data, hindering reliable model adaptation to new clinical sites with limited labeled examples.

To address this, we introduce a hybrid calibration pipeline that leverages sparse annotations effectively. Starting from a ResNet-50 backbone pretrained on data from the Memorial Sloan-Kettering (MSK) cohort, we first apply lightweight fine-tuning using the minimal available local annotations. Subsequently, we implement Dynamic Unsupervised Adaptation (DUA) on the remaining unlabeled WSIs to align batch-normalization statistics prior to inference. This two-stage calibration strategy effectively harmonizes model performance across sites with minimal manual annotation effort. Our results highlight that integrating sparse fine-tuning with unsupervised adaptation significantly mitigates inter-cohort discrepancies, enabling robust TPS estimation in clinical environments constrained by limited annotated data. This hybrid pipeline provides a practical, cost-effective method to facilitate the deployment of deep learning-based TPS scoring in resource-limited hospitals.

# Title: Robust and interpretable prediction of gene markers and cell types from spatial transcriptomics data

Quan Nguyen

QIMR Berghofer Medical Research Institute

Short bio: Dr. Quan Nguyen is conducting research and development on blockchains and distributed ledger technologies. His expertise includes graph drawing, visual analytics, blockchain, and parallel and distributed computing.



**Abstract:** Spatial transcriptomic (ST) imaging and sequencing data enables us to link tissue morphological features with thousands of unseen gene expression values, opening a horizon for understanding tissue biology and breakthroughs in digital pathology. Deep learning models are emerging to predict gene expression or classify cell types using images as the only input. Such models hold a huge potential for clinical applications, but require improvements in interpretability and robustness. We developed STimage as a comprehensive suite of models for both regression (predicting gene expression) and classification (mapping tissue regions and cell types) tasks. STimage is the first to thoroughly address robustness (uncertainty) and interpretability. While existing models focus on predicting highly variable genes, STimage predicts functional genes and identify highly predictable genes. Using diverse datasets from three cancers and one chronic disease, we assessed the model performance for in-distribution and out-of-sample distribution. STimage is robust to technical variations across platforms, data types, sample preservation, disease types. Further, we implemented an ensemble approach, incorporating pre-trained foundation models, to improve performance and reliability, especially for the case of a small training dataset. With single cell resolution Xenium data, STimage could classify cell types for millions of individual cells. Applying STimage for proteomics data like CODEX, we found that STimage can predict gene expression consistent with the protein expression pattern. Finally, we showed that using STimage predicted values based on just imaging input, we could stratify patient survival groups. Overall, STimage advances spatial transcriptomics by improving the prediction of gene expression from traditional histopathological images, making it more accessible for tissue biology research and digital pathology applications.

## **Title: Standardized primary tissue dissociation approach to a better chance of success.**

**William Tan**

**Cytiva**

Short bio: William Tan is a sales specialist for the Genomics & Diagnostics portfolio at Cytiva. His work involves collaborating and supporting researchers as well as manufacturers in identifying the right materials for their research and development of diagnostics assays. With experience in tissue engineering and tissue diagnostics enables laboratories working with tissues to develop a method to reducing tissue loss and wastage.



**Abstract:** Tissue dissociation is a fundamental step in any research projects that involve cells derived from primary tissues from humans or animal models. Current methods have been a key problem to insufficient and “low-quality” cells for subsequent research experiments, which often results in poor and inconsistent results. Especially in single cell sequencing research, having sufficient viable cells is important to ensure the sequencing experiments can be performed. By arresting the issues at its root case, with a fast and gentle tissue dissociation, high-quality viable single cells can be achieved.

# **Title: Zonal hepatocyte reorganization post-bariatric surgery: Insights from integrated spatial transcriptomics and AI digital pathology (Selected Short talk)**

**Desiree Abdurrachim**

MSD

Short bio: Dr. Desiree Abdurrachim is a Principal Scientist at MSD, specializing in biomedical imaging, AI digital pathology, and spatial biology. She has presented plenary talks on topics such as carcinoma to single cells.



**Abstract:** The liver possesses distinct zoned micro-architecture that exhibits variations in oxygen gradient, metabolic profiles, and morphogenetic fields, resulting in significant zone-specific differences in gene expression, epigenetic features, and regenerative capacity. Under conditions of stress and injury, the zonal organization of the liver undergoes substantial alterations, making it crucial to investigate zonal and spatial biology. This work aims to elucidate the interplay between liver zonation and the underlying mechanisms of Metabolic dysfunction-Associated Steatohepatitis (MASH) regression using spatial transcriptomics and AI digital pathology.

**Methods:** We performed spatial transcriptomics on paired-liver biopsy samples from a MASH patient pre-bariatric surgery (fibrosis stage 3) and 3.6 years post-surgery (fibrosis stage 1) using CosMx SMI Human Universal Cell Characterization Panel (1000-plex). H&E staining was performed following spatial transcriptomics to integrate zonal analysis using in-house AI digital pathology.

**Results:** We characterized 23,783 and 13,536 cells with mean total transcripts per cell of 152 and 252 for pre- and post-surgery sample, respectively. Data analysis revealed 3 main hepatocyte populations from each timepoint. In the pre-surgery sample, all hepatocyte populations had a higher presence in portal tract (PT) than in central vein (CV), which indicates disruption in zone-specific hepatocyte organization. However, in the post-surgery sample, one hepatocyte population had a higher presence in CV, one in PT, and one uniformly distributed, suggesting reorganization of hepatocyte populations. This reorganization is also suggested by the neighborhood enrichment score in the post-surgery sample where a pattern of decreased co-occurrence of cholangiocytes with hepatocyte population enriched in CV region was observed.

**Conclusion:** Integration of spatial transcriptomics and AI digital pathology honed our understanding of spatial biology in MASH. Our pilot data revealed the disruption in the liver zonation in MASH, which could be recovered post-bariatric surgery intervention. Further studies should involve larger sample size and/or in preclinical setting to deepen our understanding and bridge translational gap.

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## **Poster Abstracts**

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**P01: Spatial single-cell image and transcriptomic profile resolve the heterogeneity of myeloid in the consensus molecular subtypes of metastatic colorectal cancer for precision immunotherapy**

Authors: Pariyada Tanjak, Amphun Chaiboonchoe, Thanawat Suwatthanaruk, Kullanist Thanormjit, Onchira Acharayothin, Jutapak Jenkitkonchai, Nutchavadee Vorasan, Pasith Prayoonrat, Tharathorn Suwatthanarak, Watsaphon Tangkullayanone, Atthaphorn Trakarnsanga, Asada Methasate, Siriluck Prapasrivorakul, Ananya Pongpaibul, Krittiya Korphaisarn, Woramin Riansuwan, Bhoom Suktitipat, Somponnat Sampattavanich, Varodom Charoensawan, Manop Pithupkakorn, Vitoon Chinswangwatanakul

**Presenter: Pariyada Tanjak**

Affiliation: Siriraj Cancer Center, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

**Abstract:**

Molecular classification of colorectal cancer using bulk transcriptomics has identified subtypes with distinctive genotypic and phenotypic traits, commonly known as the consensus molecular subtypes (CMS). While CMS classification has clinical relevance in non-metastatic colorectal cancer, its applicability to metastatic colorectal cancer remains poorly understood. Here, we classified CMS on primary colorectal cancer tissues from 224 patients including 172 patients with non-metastatic colorectal cancer and 52 patients with metastatic colorectal cancer, using bulk transcriptomic sequencing. We further applied spatial transcriptomic profiling to decipher the effect of tumor microenvironment (TME) on CMS prediction in metastatic colorectal cancer. In addition to mapping the intratumoral heterogeneity of CMS and their associated microenvironment, we used spatial single-cell imaging to identify differences in the myeloid compartment, particularly macrophages, across CMS in metastatic colorectal cancer. We spatially localize the myeloid subsets based on CMS and their tissue localization between primary colorectal cancer and metastatic tissues. Spatial transcriptomic profiling revealed cancer cells were predominantly classified as CMS2, whereas CMS1, CMS3, and CMS4 were primarily localized within the TME. This finding suggested that the TME played a crucial role in influencing CMS classification. Spatial single-cell imaging identified macrophages and mast cell were increased in metastatic sites. We detected that SPP1+ macrophages with anti-inflammatory gene group (APOE, CD163) were highest in CMS4. Spatial single-cell image and transcriptomic profile revealed the molecular and immunological landscape of CMS-classified colorectal cancer. Our comprehensive analysis of the cellular landscape within the context of CMS colorectal cancer provides insights into its molecular heterogeneity, thereby contributing to the advancement of personalized therapeutic approaches.

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**P02: Islet-Resident Macrophages Show Preferential Interaction with Immature Pancreatic Beta Cells.**

Authors: Paramin Siriphoeumphoonsuk, Nathadol Jomsiriwattana, Punn Augsornworawat

**Presenter: Paramin Siriphoeumphoonsuk**

Affiliation: Department of Immunology, Faculty of Medicine, Siriraj Hospital, Mahidol University

**Abstract:**

Macrophages are known immune cells associated with immune activation, leading to inflammation of pancreatic islet  $\beta$ -cells. In this study, we analyzed the gene expression landscape of pancreatic islets using a single-cell RNA sequencing dataset from both adult and fetal islet samples and found that adult islets expressed both mature and immature forms of  $\beta$ -cells, which islet-resident macrophages were prone to sending immune activation signals to immature  $\beta$ -cells over its mature form.

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**P03: Spatial Transcriptomic Profiling Reveals  $\beta$ -Catenin-Driven Tumor Budding as a Prognostic Marker and Therapeutic Target in Colorectal Cancer**

Authors: Suyanee Thongchot, Phimmada Hatthakarnkul, Punn Augsornworawat, Somponnat Sampattavanich, Peti Thuwajit, Ananya Pongpaibul, Attaporn Trakarnsanga, Napat Angkathunyakul, Joanne Edwards, Chanitra Thuwajit

**Presenter: Suyanee Thongchot**

Affiliation: Department of Immunology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

**Abstract:**

Tumor budding (TB) is a potent prognostic marker associated with poor survival outcomes due to its high metastatic potential in colorectal cancer (CRC). This study the immunohistochemistry (IHC) of pan-cytokeratin (PanCK) in 388 CRC cases revealed high TB was significantly associated with patient poor survival. Furthermore, low-grade and high-grade TB CRC cases were investigated the gene expression profiles of cancer cells and immune cells at the tumor core, invasive front and stromal areas with GeoMx Digital Spatial Profiler. Notably, CTNNB1/ $\beta$ -catenin expression was significantly elevated at the invasive margin in high-grade TB cases compared to low-grade TB cases. In PanCK-positive, high TB invasive regions had higher expression of CCND1/cyclin D1 compared to high TB tumor core. The IHC, multiplex IHC/IF, and Gene Expression Profiling Interactive Analysis databases, confirmed high TB and high CTNNB1/ $\beta$ -catenin were strongly associated with poorer prognosis. The 3D-CRC cells-derived spheroids showed that  $\beta$ -catenin was impaired under curcumin treatment. Our data support the TB grade score improves the prognostic evaluation of CRC patients. Moreover, a therapeutic strategy targeting curcumin to suppress  $\beta$ -catenin-regulated TB formation may offer a promising approach to improving survival outcomes in CRC patients.

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**P04: Simultaneous mapping of 3D genome structure and chromatin modification in single cells with DNA deaminase**

Authors: Wenyang Dong, Jiankun Zhang, Leyi Dai, Jinxin Phaedo Chen, Runsheng He, Honggui Wu, Fanchong Jian, Yijun Liu, Yu Tian, Shengyuan Luo, Xiaoliang Sunney Xie

**Presenter: Wenyang Dong**

Affiliation: Changping Laboratory

**Abstract:**

The interplay between three-dimensional chromatin structure and epigenetic modifications is crucial for gene regulation and cell functions. A variety of methods such as CUT&RUN and CUT&Tag have been developed for probing histone modifications. However, enzymatic cleavage of DNA in these methods hinders their multi-omics integration with other DNA profiling techniques. Here, by extending the use of double-stranded (dsDNA) deaminase in gene editing, we introduce a new method for mapping histone modification by ANtibody-associated DeamInasE (ANDIE). At bulk level, the histone modifications identified by ANDIE showed high degree of consistency with ChIP-seq and CUT&Tag. Unlike cleavage-based methods, ANDIE leaves genomic DNA after deamination unfragmented and amplifiable and suitable for single-cell measurements. In doing so, we achieved simultaneous mapping of histone modifications and the 3D genome structure in a single cell by combining ANDIE with Hi-C. This overcomes the cellular diversity and heterogeneity within tissues or populations. Notably, single-cell ANDIE with Hi-C significantly improved the detectability of targeted histone modifications at the single-cell level, with an order of magnitude increase in the number of unique reads detected when compared with other techniques. As such, we measured the spatial distribution of clustered histone modifications in the reconstructed single-cell 3D genome structures. We also observed a correlation between histone

modification and A/B compartment, uncovered the link between the alterations in histone modifications and changes in chromatin structures. In summary, this integrated approach provides high-resolution maps of chromatin organization and chromatin modifications for elucidating epigenetic interaction during gene regulation in a single-cell basis.

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**P05: Cancer-Associated Fibroblast Gene Signature in Triple-Negative Breast Cancer with High Tumor Budding Reveals LIF as a Novel Therapeutic Marker**

Authors: Pimchanok Phankeaw, Suparada Khanaruksombat, Warapan Numprasit, and Punn Augsornworawat, Malee Warnnissorn, Peti Thuwajit, and Chanitra Thuwajit

**Presenter: Pimchanok Phankeaw**

Affiliation: Faculty of Medicine, Siriraj hospital, Mahidol University

**Abstract:**

Breast cancer has the highest incidence of female cancer worldwide. The survival rate of the patients depends on several factors including the degree of tumor budding (TB) found in the tumor microenvironment (TME). Cancer-associated fibroblasts (CAFs) are the most abundant component in TME. However, the exact crosstalk between TB and CAFs on TNBC progression is still unclear. This study aims to explore the transcriptomic profiling of CAFs in high TB-TNBC compared to a low TB group and indicates novel therapeutic markers using bioinformatics analysis. Whole Transcriptomic analysis of CAF regions in 13 cases of TNBC formalin-fixed paraffin-embedded tissues was performed by Templated Oligo-Sequencing (TempO-Seq). The data demonstrated the upregulated genes in high TB TNBC cases with aggressive CAF phenotype were ZNF235, ANKRD30B, SLC26A2, SPDYC, CDKN1C, GPRC5B, LIF, and FAM83A and the TB-related pathways were Phosphate ion homeostasis, Monocyte chemotaxis, MAPK cascade, and ERK1/ERK2 cascade. Additionally, LIF (leukocyte inhibitor factor) was found in high TB upregulated genes with IGF2, CX3CR1, and FGF4. Recombinant LIF protein could induce TNBC cell migration significantly compared to the untreated cells. These findings highlight both the gene signature of high TB CAFs-containing TNBC cases and LIF as a novel target to attenuate TB-driven TNBC progression.

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**P06: Single-Cell Transcriptomic Profiling of Peripheral Blood Immune Cells in Systemic Lupus Erythematosus**

Authors: Pisacha Somsak, Nathadol Jomsiriwattana, Pimchanok Phankeaw, Punn Augsornworawat

**Presenter: Pisacha Somsak**

Affiliation: Department of immunology, Faculty of medicine Siriraj hospital, Mahidol university

**Abstract:**

Systemic lupus erythematosus (SLE) is a multifaceted autoimmune disease marked by dysregulated immune responses and diverse clinical manifestations. To better understand the immune landscape in SLE, this study leverages publicly available single-cell RNA sequencing datasets to characterize peripheral blood mononuclear cells (PBMCs) from affected individuals. Using established analytical workflows, we identified and annotated major immune cell populations and examined their transcriptional profiles at single-cell resolution. This characterization highlights the cellular heterogeneity present in SLE and provides a valuable reference for future studies aiming to dissect immune dysfunction in autoimmune diseases.

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**P07: Study of whole cell proteome- the proteomic difference of astrocyte between mouse brain cortical and hippocampal regions**

Authors: Chien-Chang Huang, Chiung-Yun Chang, Weng Man Chong, Chun-Kai Huang, Hsiao-Jen Chang, Jung-Chi Liao

**Presenter: Nikhil Rao**

Affiliation: Syncell Inc.

**Abstract:**

Astrocytes are a subtype of glial cells in the Central Nervous System. They play roles in neurogenesis, synaptogenesis, blood-brain barrier permeability, and maintaining extracellular homeostasis. Interestingly, different populations of astrocytes localized in specific regions of the brain area have unique morphological and functional characteristics. The aim of this study is to characterize proteomic differences of astrocyte across various regions of the mouse brain. Here, we used a microscopy-based proteomics platform Microscoop® which perform ultra-content microscope-guided photo-biotinylation to render study of astrocyte proteomes in the cortical and hippocampal regions.

Astrocytes in mouse brain cryo-sections are immunostained against the glial fibrillary acidic protein (GFAP) and recognized by Microscoop® as the target at which the two-photon laser precisely illuminates. The light effectively activates proprietary photosensitive biotin-based probes free floating around the sample and triggers protein biotinylation. Under the Microscoop®, this cycle runs automatically across thousands of fields of view so that sufficient proteins are biotinylated for the subsequent pull down and LC-MS/MS-based proteome identification. This unique workflow, termed optoproteomics, allows subcellular proteomic discovery in high specificity, sensitivity and resolution.

In this study, proteomes of cortical or hippocampal astrocytes were established. Furthermore, well-known astrocytes markers including GFAP, LAMC3, PARK7 etc. were found. Upon comparing differences in the two proteomes, several candidates that have not been reported in existing literature and were more enriched in either cortex or hippocampus were selected for immunofluorescent staining. The cortex enriched candidates PLEKHB1 and SYBP3 colocalized with the astrocyte marker GFAP and are found to be more abundant in cortex astrocytes than that of hippocampus. In contrast, the hippocampus enriched candidate MINK1 were positively validated in hippocampus astrocyte but not in cortex astrocytes. Future study of the functional relevance of these protein candidates could help unravel the roles of regional specific astrocytes and generates multiple testable hypotheses. It also provides a method of defining cell types, subtypes, and states using whole proteome analysis vs. transcriptomic. The Microscoop® platform enables unbiased study of spatial proteomes, facilitating the discovery of novel astrocyte markers significant to specific brain region.

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**P08: Association of Plasma Matrix Metalloproteinase-9 and Osteopontin Levels with Apolipoprotein E Genotypes of Patients with Mild Cognitive Impairment and Alzheimer's Disease**

Authors: Aleczandria R. Esguerra, RMT, MSMT : Ann Florence V. Belvis, PhD

**Presenter: Aleczandria Esguerra**

Affiliation: St. Luke's Medical Center

**Abstract:**

This study investigates the association between Apolipoprotein E (ApoE) genotypes and biomarkers of neurodegeneration, Matrix Metalloproteinase-9 (MMP-9) and Osteopontin (OPN) in individuals with Mild Cognitive Impairment (MCI) and Alzheimer's disease (AD). The ε2/ε3 genotype correlates with the lowest MMP-9 and OPN levels, suggesting a protective effect against neuroinflammation and neurodegeneration. In contrast, the ε3/ε4 genotype exhibits the highest concentrations of these markers, reflecting elevated AD risk, impaired amyloid-β clearance, and heightened inflammatory responses. The

$\epsilon 3/\epsilon 3$  genotype displays intermediate biomarker levels, lacking the protective benefits of  $\epsilon 2$  but avoiding the risks linked to  $\epsilon 4$ . Sex-specific differences are evident, with females showing higher MMP-9 concentrations than males. These findings underscore the influence of ApoE genotype and sex on AD pathology and propose these as potential diagnostic biomarkers and therapeutic targets. Further research is needed to elucidate their mechanistic roles in modulating neurodegeneration and inflammation in AD progression.

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**P09: Single-cell transcriptomics analysis reveals immunosuppressive mechanisms of PGE2-EP4 signaling in human inflammatory bowel disease**

Authors: Siwakorn Punyawatthanakool, Shuh Narumiya

**Presenter: Siwakorn Punyawatthanakool**

Affiliation: Department of Pharmacology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Thailand

**Abstract:**

Inflammatory bowel disease (IBD) is a chronic inflammatory disorder driven by a complex interplay among immune cells, stromal cells, and the intestinal epithelium. Prostaglandin E<sub>2</sub> (PGE<sub>2</sub>), an immunosuppressive lipid mediator, has been shown to exert protective effects in IBD, as evidenced by the exacerbation of gastrointestinal inflammation following either cyclooxygenase (COX) inhibition or genetic ablation of PGE<sub>2</sub> receptors. However, the precise mechanisms by which PGE<sub>2</sub> alleviates IBD symptoms remain poorly understood. To address this, we analyzed a published single-cell RNA sequencing dataset comprising 979,990 cells from three healthy controls and thirty-eight IBD patients enrolled in an anti-TNF $\alpha$  clinical trial (TAURUS-IBD). Our analysis revealed that IBD is characterized by dual CD4 $^+$  T cell subset dysregulation, with concurrent activation of both Th17 and interferon- $\gamma$  pathways, which correlated with increased epithelial cell apoptosis. We found that PGE<sub>2</sub> was produced by myeloid and epithelial cells, while its receptor—particularly PTGER4 (EP4)—was expressed across T cells, myeloid cells, stromal cells, and epithelial cells. Notably, high expression of PTGS2, the gene encoding COX-2, was associated with reduced interferon- $\gamma$  signaling and decreased Th17 cell infiltration at inflammatory sites. Furthermore, CD4 $^+$  and CD8 $^+$  T cells from patients with elevated PTGS2 expression exhibited downregulation of mitochondrial respiration and ribosomal protein synthesis, indicative of suppressed cellular activation. Collectively, these findings demonstrate that PGE<sub>2</sub> exerts broad immunosuppressive effects in IBD and suggest that pharmacological activation of PGE<sub>2</sub> signaling—such as through EP4 agonists—may represent a promising therapeutic strategy for IBD management.

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**P10: Hippo pathway in breast cancer cells induces NCAM1+ $\alpha$ SMA+ fibroblasts to modulate immunosuppressive tumor microenvironment**

Authors: Chanida Thinyakul<sup>1,2</sup>, Yasuhisa Sakamoto<sup>1,12</sup>, Mayuko Shimoda<sup>1,12</sup>, Yanliang Liu<sup>1</sup>, Suyanee Thongchot<sup>2,3</sup>, Omnia Reda<sup>4</sup>, Akihiro Nita<sup>1</sup>, Romgase Sakamula<sup>5</sup>, Somponnat Sampattavanich<sup>5</sup>, Ayato Maeda<sup>1</sup>, Paweenapon Chunthaboon<sup>1</sup>, David Nduru<sup>1</sup>, Mayumi Niimura<sup>1</sup>, Yohei Kanamori<sup>1</sup>, Peti Thuwajit<sup>2</sup>, Keiichi Nakayama<sup>6,7</sup>, Kun-Liang Guan<sup>8</sup>, Yorifumi Satou<sup>4</sup>, Chanitra Thuwajit<sup>2</sup>, and Toshiro Moroishi<sup>1,9,10,11</sup>

**Presenter: Chanida Thinyakul**

Affiliation: Faculty of Medicine Siriraj Hospital, Mahidol University

**Abstract:**

Cancer cells adeptly manipulate the tumor microenvironment (TME) to evade host antitumor immunity. However, the role of cancer cell-intrinsic signaling in shaping the immunosuppressive TME remains

unclear. Here, we found that the Hippo pathway in cancer cells orchestrates the TME by influencing the composition of cancer-associated fibroblasts (CAFs). In a 4T1 mouse breast cancer model, Hippo pathway kinases, large tumor suppressor 1 and 2 (LATS1/2), promoted the formation of neural cell adhesion molecule 1 (NCAM1)+ alpha-smooth muscle actin ( $\alpha$ SMA)+ CAFs expressing the transforming growth factor- $\beta$  (TGF-  $\beta$ ), identified by single cell RNA sequencing. These CAFs were associated with T cell inactivation and dysfunction. Depletion of LATS1/2 in cancer cells resulted in a less immunosuppressive TME, indicated by the reduced proportions of NCAM1+ $\alpha$ SMA+ CAFs and dysfunctional T cells. Notably, similar Hippo pathway-induced NCAM1+ $\alpha$ SMA+ CAFs were observed in human breast cancer, highlighting the potential of TME-manipulating strategies to reduce immunosuppression in cancer immunotherapy.

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### **P11: Spatial Immune Profiling Reveals Localized Immune Suppression in High Tumor Budding Triple-Negative Breast Cancer**

Authors: Suparada Khanaraksombat, Hay Mar Oo, Simran Venkatraman, Pranisa Jamjuntra, Suyanee Thongchot, Somponnat Sampattavanich, Doonyapat Sa-Nguanraksa, Malee Warnnissorn, Peti Thuwajit, and Chanitra Thuwajit

#### **Presenter: Suparada Khanaraksombat**

Affiliation: 1Department of Immunology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700 Thailand, 2Siriraj Center of Research Excellence for Cancer Immunotherapy, Research Department, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700 Thailand

#### **Abstract:**

Triple-negative breast cancer (TNBC) is the most aggressive breast cancer subtype, characterized by high rates of distant metastasis and poor clinical outcomes. Building on our previous identification of an immune-oncogenic gene signature derived from spatial transcriptomic analyses of immune cell-enriched regions in high tumor budding (TB) TNBC, this study investigates the immune cell landscape underlying this aggressive phenotype. We employed multiplex immunohistochemistry (mIHC) using the CODEX Phenocycler Fusion platform to characterize the tumor immune microenvironment in TNBC tissues with high (n=2) and low (n=2) immune-oncogenic signatures. Our analysis revealed a significantly diminished immune infiltrate in high immune-oncogenic TNBC, particularly a marked reduction in CD3 $\varepsilon$ + T cells and activated cytotoxic T cells (GZB+ CD8+). Furthermore, effector immune cells including CD4+ T cells, regulatory T cells, B cells, and M1 macrophages were spatially distant from activated cytotoxic T cells, whereas immunosuppressive M2 macrophages were found in closer proximity, suggesting a localized immune suppression mechanism. Extending this observation to a larger cohort, high TB TNBC cases (n=31) exhibited significantly fewer GZB+CD3 $\varepsilon$ +CD8+ T cells compared to low TB cases (n=34, p=0.012), supporting the association between tumor budding and impaired immune activation. These findings underscore the prognostic and therapeutic relevance of spatial immune architecture in TNBC and highlight potential approaches for the development of precision immunotherapies targeting immune-excluded and immunosuppressed tumor niches.

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### **P12: Spatial resolved tumour microenvironment features associated with outcome to therapy of head and neck cancers**

Authors: Naomi Berrell, James Monkman, Meg Donovan, Tony Blick, Ken O'Byrne, Rahul Ladwa, Chin Wee Tan, Arutha Kulasinghe

#### **Presenter: Naomi Berrell**

Affiliation: Wesley Research Institute

#### **Abstract:**

Despite advancements in various cancer treatments, including chemoradiotherapy, surgery and immunotherapies, treatment for head and neck cancers (HNC) remains a challenge. For patients with HNC, relapse or recurrent disease will occur in half of all patients, and these patients have a median overall survival of 8.9 (HPV negative tumours) and 18 months (HPV positive tumours). Immunotherapies are showing long term and durable responses in a subset of patients, but we lack robust, predictive biomarkers for response in HNC. Understanding the tumour microenvironment (TME) may reveal key biological differences between patient responses and could help us predict therapy response. To explore the TME we applied geospatial whole-transcriptome profiling (NanoString GeoMx Digital Spatial Profiler) and spatial proteomics profiling (Akoya PhenoCycler-Fusion) on a tumour-microarray. The cohort included 25 tumour cores of advanced head and neck tumours, 12 of which were collected from 2 immunotherapy responsive patients (5 cores) and 4 immunotherapy-resistant patients (8 cores). Through mapping the whole transcriptome of tumour and stromal regions, we identified dysregulated metabolic pathways within the tumour, including hypoxia and glycolysis, to be associated with patients who experienced a poor response to immunotherapy. Additionally, when we assessed the relationship of cellular interactions and locations within the TME using single-cell spatial proteomics, the infiltration of CD8 T cells into the tumour mass was higher in patients who had improved response to immunotherapy. Furthermore, we found that the presence of cellular neighbourhoods that were rich in natural killer cells, dendritic cells and macrophages were more common in responsive patients, whereas tumour-dense neighbourhoods were more frequent in non-responsive patients. By harnessing the power of high-plex spatial transcriptomics and proteomics we identify novel alterations within the TME that could be contributing to immunotherapy resistance and response in HNC.

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**P13: Spatial Transcriptomic Profiling Reveals Ancestral Mesenchymal Subclone Driving Metastatic Progression in Advanced Ovarian Clear Cell Carcinoma Associated With Clinical Outcome**

Authors: Thang Le Truong, Duncan Yi-Te Wang, Ko-Chen Chen, Wei-Chou Lin, Tuan Zea Tan, Lin-Hung Wei, Ruby Yun-Ju Huang

**Presenter: Thang Truong Le**

Affiliation: National Taiwan University

**Abstract:**

**Background:** Advanced ovarian clear cell carcinoma (OCCC) is characterized by extensive metastasis and complex cellular heterogeneity. The spatial organization and clonal evolution of cancer subpopulations driving metastasis remain poorly understood.

**Methods:** In the discovery phase, the CosMx SMI 1K panel was used to profile the cellular landscape from three FFPE tumor samples of ovarian, peritoneal, and colonic metastatic sites in stage IV OCCC. Validation was performed using the GeoMx DSP CTA panel on 364 PanCK-positive ROIs from nine patients, followed by integration analysis with single-nucleus RNA sequencing (snRNA-seq) data from ten published OCCC patients and whole-transcriptomic profiles from an independent cohort of 102 clinical samples.

**Results:** A total of 25 single-cell types were identified, including 9 cancer cells (labelled a-i). Among them, the cancer “f” was consistently present across all metastatic sites and was inferred by trajectory analysis as the ancestral clone from which other subclones emerged. Cancer f cells exhibited a higher mesenchymal score than epithelial score ( $P < 0.001$ ) and showed niche-forming tendencies. CellChat analysis indicated autocrine signaling activities in cancer f cells, with SPP1 and LIFR pathways enriched in the peritoneal and colonic sites, respectively. Deconvolution analyses onto GeoMx DSP PanCK ROIs and integration analyses onto snRNA-seq cohorts revealed positive correlations between cancer f enrichment and mesenchymal transition score ( $P < 0.001$ ). Notably, higher enrichment of cancer f was associated with worse overall survival (OS) and progression-free survival (PFS) in OCCC patient cohorts.

Conclusions: Spatial transcriptomics revealed an ancestral cancer subclone with mesenchymal features and autocrine signalling that drives metastatic progression and correlates with poor survival outcomes in OCCC.

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#### **P14: Tahoe-100M: A Giga-Scale Single-Cell Perturbation Atlas for Context-Dependent Gene Function and Cellular Modeling**

Authors: Jesse Zhang, Airol A Ubas, Richard de Borja, Valentine Svensson, Nicole Thomas, Neha Thakar, Ian Lai, Aidan Winters, Umair Khan, Matthew G. Jones, Vuong Tran, Joseph Pangallo, Efthymia Papalexi, Ajay Sapre, Hoai Nguyen, Oliver Sanderson, Maria Nigos, Olivia Kaplan, Sarah Schroeder, Bryan Hariadi, Simone Marrujo, Crina Curca Alec Salvino, Guillermo Gallareta Olivares, Ryan Koehler, Gary Geiss, Alexander Rosenberg, Charles Roco, Daniele Merico, Nima Alidoust, Hani Goodarzi, Johnny Yu

**Presenter: Johnny Yu**

Affiliation: Tahoe Therapeutics

Abstract:

Building predictive models of the cell requires systematically mapping how perturbations reshape each cell's state, function, and behavior. Here, we present Tahoe-100M, a giga-scale single-cell atlas of 100 million transcriptomic profiles measuring how each of 1,100 small-molecule perturbations impact cells across 50 cancer cell lines. Our high-throughput Mosaic platform, composed of a highly diverse and optimally balanced “cell village”, reduces batch effects and enables parallel profiling of thousands of conditions at single-cell resolution at an unprecedented scale.

As the largest single-cell dataset to date, Tahoe-100M enables artificial-intelligence (AI)-driven models to learn context-dependent functions, capturing fundamental principles of gene regulation and network dynamics. Although we leverage cancer models and pharmacological compounds to create this resource, Tahoe-100M is fundamentally designed as a broadly applicable perturbation atlas and supports deeper insights into cell biology across multiple tissues and contexts. By publicly releasing this atlas, we aim to accelerate the creation and development of robust AI frameworks for systems biology, ultimately improving our ability to predict and manipulate cellular behaviors across a wide range of applications.

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#### **P15: Multiomic characterization of the immune landscape in human pancreatic islets**

Authors: Jomsiriwattana Nathadol, Augsornworawat Punn

**Presenter: Nathadol Jomsiriwattana**

Affiliation: Department of Immunology, Faculty of Medicine Siriraj Hospital

Abstract:

**Introduction and Rationale:** Type I Diabetes is associated with the autoimmune-led destruction of the pancreatic insulin-producing beta cells, most likely caused by the dysregulation of antigen presenting cells. Islet-resident macrophages, one type of antigen presenting cell, have been shown to be crucial for islet development and for maintaining immune balance, which may potentially link to autoimmune diabetes. However, much of our current understanding is derived from mouse studies, which have not comprehensively covered all sequencing profiles relevant to human contexts. Although tissue-resident immune cell populations have been extensively characterized in various organ sites, investigation into the immune cell composition within human pancreatic islets remains limited. In this study, we use single-cell sequencing analysis to thoroughly characterize human islet immune cells, enhancing our understanding of their roles in both developmental and diabetes contexts.

**Methods:** In this study, we employed single-cell multiomic sequencing to analyze the transcriptome (scRNA-seq) and chromatin accessibility (scATAC-seq) profiles of five healthy human adult pancreatic

islets. We utilize the Seurat, and Signac package to perform the analysis. Briefly, datasets were compiled and processed by filtering out low quality cells and normalized to allow for gene expression interpretation. Cellular populations were identified using key gene markers. Minor population such as immune cells were identified using high expression levels of PTPRC (CD45), CD74, and LYZ. Immune cells were subset, re-clustered, and characterized based on lineage-specific markers and regulatory accessibility landscapes. Cross-tissue comparison was performed against resident macrophages from lymph nodes.

**Results:** Our analysis revealed a diverse immune cell repertoire within the human islet microenvironment, including distinct populations of monocytes/macrophages, plasma cells, and potentially other leukocyte subtypes. Notably, both plasma cells and islet-resident macrophages exhibited transcriptional and epigenetic signatures indicative of tissue residency and immune activation. Furthermore, single-cell multiomic comparisons with macrophages from various organs demonstrated that islet-resident macrophages share greater lineage similarity with other tissue-resident macrophages than with monocyte-derived macrophages.

**Conclusions:** In conclusion, our findings support the hypothesis that pancreatic tissue-resident macrophages may act as key mediators of local immune activation, potentially influencing susceptibility to autoimmune diabetes. These findings contribute to our understanding of immune regulation within pancreatic islets and may have implications for the development of therapeutic strategies for autoimmune diabetes.

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#### **P16: Integrative Identification of Tumor Features in Meningioma Using Marker-Based Annotation, inferCNV, and Hierarchical Clustering**

Authors: Sereerat T , Ukhampun P , Lert-itthiporn W

**Presenter: Thanapong Sereerat**

Affiliation: Department of Biochemistry, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand

**Abstract:**

Meningioma, a tumor formed in meninges, is one of the most prevalent primary brain tumors. Even if it is commonly benign, the location and high-grade meningioma can cause serious symptoms and aggressive characteristics. Recent studies have characterized the cell-types composition and function of meningioma cells apart from normal adjacent tissues. However, the strategies to uncover tumor features have not adequately studied especially in benign tumors. This study aimed to propose the strategies to reveal tumor features based on single-cell RNA sequencing (scRNA-seq). The publicly scRNA-seq dataset were downloaded via Zenodo (DOI: 10.5281/zenodo.6473604), including 5 meningioma and 7 normal adjacent dura. Data preprocessing involved standard quality control and doublet detection, resulting in 49,857 cells for downstream analysis. The strategies to investigate tumor features are canonical markers, inferring copy number variations (CNVs) from scRNA expression via inferCNV, before validation by person's hierarchical clustering. The expression of CLU, PTN, LEPR, and SSTR2 genes were specifically high in mesenchymal cluster, compared to other cell types. The inferCNV results showed that the common CNVs profiles of meningioma, including loss in chromosomes 1, 4, 6, and 22, and gain in chromosomes 17 and 20, were highly specific to mesenchymal cluster. The hierarchical heatmap confirmed that distinct expression profiles of mesenchymal cells between meningioma and normal adjacent tissue. In summary, the combination of tumor markers, CNVs, and hierarchical heatmap can be used to reveal the tumor characteristics in scRNA expression, shedding the light on the tumor-stromal cells interaction studies.

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#### **P17: Spatial Single-Cell Analysis Identifies Galectin-1 Fibroblasts-Associated with Immune Checkpoint Inhibitor Resistance in Non-small Cell Lung Cancer**

Authors: Chantra Kamnerdnond, Pimchanok Phankeaw, Kanjana Laosuntisuk, Jutapak Jenkitkonchai, Kittiporn Punuch, Varodom Chareonsawan, Peti Thuwajit, and Chanitra Thuwajit

**Presenter: Chantra Kamnerdnond**

Affiliation: Faculty of Medicine Siriraj hospital, Mahidol University

**Abstract:**

Non-small cell lung cancer (NSCLC) is a highly aggressive malignancy with significant global morbidity and mortality. Despite advances in treatment, including immune checkpoint inhibitors (ICIs), approximately 25% of patients develop resistance, which is influenced by both tumor-intrinsic factors and the immunosuppressive tumor microenvironment (TME). Cancer-associated fibroblasts (CAFs), as key components of the TME, play a crucial role in promoting tumor progression and ICI resistance. This study investigated spatial single-cell transcriptomic profiles in NSCLC tissues from two ICI responders (R) and one non-responder (NR), with a focus on the heterogeneity of CAF subtypes between responders and non-responders. Spatial transcriptomics was conducted on three formalin-fixed, paraffin-embedded (FFPE) NSCLC samples using the NanoString CosMx Spatial Molecular Imager with a 1,000-plex RNA panel. Bioinformatic analyses included cell type annotation, including CAF identification, and quantification of cells expressing genes of interest based on cell type and ICI response status. Fifteen distinct cell types were identified, including three tumor subtypes, ten immune/stromal populations, and two non-immune stromal cell types. A total of approximately 9,500 CAFs were identified based on elevated expression of COL1A1, COL1A2, COL3A1, FN1, and TAGLN, and were further clustered into seven subpopulations. Notably, Cluster 4 CAFs were enriched in the NR sample and depleted in R samples, and demonstrated high expression of LGALS1, encoding the immunoregulatory protein galectin-1 (Gal-1). CAFs exhibited the highest LGALS1 expression (56.8%) compared to tumor cells (52.3%) and other cell populations. LGALS1-positive CAFs were more prevalent in the NR sample (58.7%) than in responders (55.9%). These findings suggest that CAFs, particularly those in Cluster 4, are a source of Gal-1 and may contribute to immune evasion and ICI resistance in NSCLC. Additional experiments are required before conclusion but these data support the potential of the specific CAFs as therapeutic targets to improve immunotherapy outcomes.

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**P18: Spatial Immune Signatures of Super Responders to Immune Checkpoint Inhibitors in Non-Small Cell Lung Cancer**

Authors: Romgase Sakamula, Thanaphon Likhityungyuen, Tauangtham Anekpuritanang, Krittiya Korphaisarn, Somponnat Sampattavanich

**Presenter: Romgase Sakamula**

Affiliation: Siriraj Center of Research Excellence in Cancer Precision Medicine and Systems Pharmacology, Faculty of Medicine Siriraj Hospital, Mahidol University

**Abstract:**

Immune checkpoint inhibitors (ICIs) have transformed the treatment landscape of non-small cell lung cancer (NSCLC), yet patient responses remain heterogeneous. PD-L1 expression, while widely used as a predictive biomarker, does not consistently correlate with clinical outcomes, highlighting the need for a deeper understanding of tumor microenvironment (TME) features that influence ICI efficacy. This study analyzed pre-treatment NSCLC tissue from ICI-treated patients stratified by response and PD-L1 expression. Using tissue-based cyclic immunofluorescence (t-CyCIF), we performed high-dimensional profiling of immune cell populations and their spatial organization within the TME. Although total immune cell density did not significantly differ between responders and non-responders, responders exhibited a higher density and tumor-normalized ratio of tertiary lymphoid structures (TLS), indicating enhanced local immune priming. Cellular neighborhood (CN) analysis revealed that responders were

enriched for pan-immune CNs, with close spatial association between dendritic cell- and CD3<sup>+</sup> T cell-enriched regions. In contrast, non-responders displayed increased co-localization of cytotoxic T cell- and Treg-enriched regions, suggesting immunosuppressive spatial interactions. These findings underscore the importance of spatial immune architecture, beyond cell type abundance, in modulating ICI response. Incorporating spatial context into biomarker strategies may improve the prediction of therapeutic outcomes in NSCLC.

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### **P19: Spatial Profiling Reveals ENPP1 as a Diagnostic Marker and Highlights CD68<sup>+</sup> Cell Heterogeneity in Tumor-Induced Osteomalacia**

Authors: Karn Mahaparn, Romgase Sakamula, Thanaphon Likhityungyuen, Somponnat Sampattavanich, Chutintorn Sriphrapradang, Artit Jinawath, Thawee Songpatanasilp, Sittichoke Watcharamasbonkkot, Sutipat Pairojboriboon, Kantang Satayasoontorn, Sorranart Maungsomboon, Rita Ruangroj, Taungtham Anekpuritanang, Chindanai Hongsaprabhas, Voranuch Thanakit, Pojchong Chotiyarnwong, Ekasame Vanitcharoenkul, Krittawat Suwanpukdee, Pannin Thanapipatsiri, Rapin Phimolsarnti, Apichat Asavamongkolkul and Chandhanarat Chandhanayingyong

#### **Presenter: Romgase Sakamula**

Affiliation: Siriraj Center of Research Excellence in Cancer Precision Medicine and Systems Pharmacology, Faculty of Medicine Siriraj Hospital, Mahidol University

#### **Abstract:**

Tumor-induced osteomalacia (TIO) is a rare paraneoplastic syndrome characterized by FGF23-overproducing mesenchymal tumors, leading to hypophosphatemia and impaired bone mineralization. Although genes such as ENPP1, FGFR1, and DMP1 are implicated in phosphate regulation, the spatial and cellular mechanisms underlying FGF23 dysregulation remain poorly defined. We applied tissue-based cyclic immunofluorescence (t-CyCIF) to formalin-fixed, paraffin-embedded (FFPE) samples from seven TIO patients from five medical centers in Thailand to map the spatial distribution of ENPP1, FGFR1, FGF23, DMP1, and CD68. Tumor and non-tumor regions were annotated by pathologists, and single-cell marker expression was quantified. Unexpectedly, ENPP1 expression was consistently and significantly enriched in tumor regions at both the tissue and single-cell levels, outperforming other candidate markers in distinguishing tumor from non-tumor areas. This supports its potential utility as a diagnostic biomarker for localizing phosphaturic mesenchymal tumors. Single-cell clustering further revealed distinct CD68<sup>+</sup> subpopulations, including multinucleated osteoclast-like cells that co-expressed elevated FGFR1 and FGF23, suggesting a role in amplifying FGF23 signaling through immune-stromal crosstalk. Our findings position ENPP1 as a promising spatial biomarker for TIO diagnosis and highlight the functional heterogeneity of CD68<sup>+</sup> cells within the tumor microenvironment. Ongoing spatial transcriptomic analysis will further define these subpopulations and their potential as therapeutic targets.

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### **P20: Denosumab Reprograms the Tumor Immune Microenvironment via SPP1 Suppression and Cytotoxic T-Cell Activation**

Authors: Zezhuo Su (Research Assistant Professor), Kelvin Sin Chi Cheung (Clinical Assistant Professor), and Jason Pui Yin Cheung (Clinical Professor)

#### **Presenter: Zezhuo Su**

Affiliation: Department of Orthopaedics and Traumatology, School of Clinical Medicine, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong SAR, China.

#### **Abstract:**

Denosumab, a RANK ligand inhibitor, is widely used to inhibit osteoclastogenesis in osteoporosis, bone metastasis, and giant cell tumour of bone (GCTB). Beyond its skeletal effects, RANK signalling

modulates immune pathways, including NF- $\kappa$ B activation and expression of SPP1 (osteopontin), a gene implicated in CXCL9:SPP1 macrophage polarization and tumour progression. Here, we investigate denosumab's role in antitumor immunity using single-cell transcriptomics and pan-cancer analyses. Single-cell RNA sequencing was performed on nine human GCTB samples (six untreated, three denosumab-treated without prior therapies) to map tumour microenvironment changes. Paired pre- and post-denosumab samples from nine additional GCTB patients were analysed to validate immune dynamics. A pan-cancer cohort spanning 34 malignancies was interrogated to assess broader clinical relevance.

In GCTB, denosumab reduced SPP1 expression across myeloid subsets and increased cytotoxic CD8+ T-cell infiltration with enhanced activity. Validation in paired samples confirmed SPP1 downregulation and cytotoxic T-cell expansion post-treatment. Pan-cancer analysis revealed an inverse correlation between SPP1 and CD8A expression, with a elevated CD8A:SPP1 ratio predictive of improved overall survival in 14 cancer types, outperforming CD8A or SPP1 alone as a prognostic marker. Our study identifies denosumab as a dual-action therapy that suppresses SPP1-mediated immunosuppression and promotes cytotoxic T-cell responses. These findings provide clinical evidence for repurposing denosumab to enhance antitumor immunity, particularly in SPP1-high malignancies, and highlight the CD8A:SPP1 ratio as a novel prognostic biomarker. This work bridges osteoimmunology and cancer immunotherapy, offering translational insights for innovative therapeutic strategies.

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**P21: Non-invasive tape-stripping technique for spatial melanoma diagnosis**

Authors: Pannawich Thirabowonkitphithan, Kevin Yang, Skaidre Jankovskaja, Gustav Christensen, Kari Nielsen, and Tautgirdas Ruzgas

**Presenter: Pannawich Thirabowonkitphithan**

Affiliation: 1. Department of Biomedical Science, Faculty of Health and Society, Malmö University, Malmö, Sweden 2. Biofilms – Research Centre for Biointerfaces, Malmö University, Malmö, Sweden

**Abstract:**

Melanoma, the deadliest common skin malignancy, proceeds through many stages with different cellular and molecular features. Detection and treatment in an early stage are curable and critical to offer better patient outcomes. To achieve early melanoma detection, a non-invasive diagnostic approach involving tape-stripping to harvest epidermal cells from suspicious lesions is investigated. We assessed a tape-stripping approach for spatial melanoma biomarker detection, integrating molecular testing for gene expression analysis with structural analysis using scanning electron microscopy (SEM) for stratum corneum roughness measurement. RNAscope, a spatially resolved technology, effectively discovered housekeeping genes in tape-stripped skin samples, demonstrating the method's efficiency for RNAs detection in the skin samples. The efforts are now directed to detecting melanoma-associated biomarkers. SEM investigation showed unique surface roughness patterns across melanoma stages. The findings revealed that stage pT3a melanoma showed a rougher surface than non-lesional skin and the other stages except pT2a stage. Notably, stage pT2a melanoma showed more roughness than non-lesional skin, while no significant differences were observed among Tis and pT1a stages. These findings suggest that the tape-stripping technique, when combined with a spatial transcriptomic tool and surface analysis, could offer a non-invasive method for spatial melanoma diagnosis and staging. The combination of genetic and structural information might have the potential to improve early detection and distinction of melanoma stages. Currently we are working to provide proof-of-concept of the clinical applicability of this non-invasive approach.

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**P22: Spatial Immune Landscape and Prognostic Significance of Molecular Subtypes in Thai Pancreatic Ductal Adenocarcinoma**

Authors: Hay Mar Oo, Napat Angkathunyakul, Phensri Niamyim, Ananya Pongpaibul, Krittiya Korphaisarn, Tauangtham Anekpuritanang, Somponnat Sampattavanich

**Presenter: Hay Mar Oo**

Affiliation: Department of Pharmacology, Faculty of Medicine Siriraj Hospital

**Abstract:**

Pancreatic ductal adenocarcinoma (PDAC) is an aggressive cancer characterized by rapid progression and resistance to conventional therapies, resulting in limited effective therapeutic options. In order to better understanding of the immunological heterogeneity of PDAC, we profiled the tumor microenvironment in formalin-fixed paraffin-embedded (FFPE) samples from 27 Thai patients with locally advanced or metastatic PDAC using tissue-based cyclic immunofluorescence (t-CyCIF). Single-cell quantification and spatial segmentation were performed by Qupath software. We classified tumors into four molecular subtypes: Classical, Hybrid, Basal, and Null, based on the expression of GATA6 and CK5. Compared to patients with Basal or Null subtypes, patients with Classical and Hybrid subtypes had significantly greater overall survival rates ( $p < 0.001$ ). These two favorable subtypes displayed the higher infiltration of B cells, CD4<sup>+</sup> and CD8<sup>+</sup> T cells, and M1 macrophages, which were characteristics of immune-inflamed ("hot") microenvironments. On the contrary, the Null and Basal subtypes showed immunological-cold characteristics with less invasion of immune cells. Pan-immune hotspots (CN1) were shown to be considerably enriched in the Classical subtype ( $p < 0.05$ ) and to be associated with better clinical outcomes ( $p < 0.05$ ) according to spatial neighborhood analysis. Additionally, CD8 T cell proximal densities were higher in classical tumors (within 25–200  $\mu\text{m}$  of tumor cells), indicating stronger local cytotoxic immune responses. However, the Basal subtype displayed closer proximity of immunosuppressive CD163<sup>+</sup>PD-L1<sup>+</sup> cells to CD8<sup>+</sup> T cells and higher immunosuppressive scores. Taken together, our results highlight the prognostic value of combining spatial immunophenotyping and molecular classification in Thai PDAC patients.

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**P23: Targeting Reversible Dormancy to Prevent Recurrence in PARP Inhibitor–Treated Ovarian Cancer**

Authors: Suttipun Suriya, Supawan jamnongsong, Somponnat Sampattavanich

**Presenter: Suttipun Suriya**

Affiliation: Department of Pharmacology, Faculty of Medicine Siriraj Hospital, Mahidol University

**Abstract:**

Cancer dormancy and recurrence are key challenges to durable responses in ovarian cancer treated with PARP inhibitors. To elucidate reversible phenotypic states and uncover therapeutic vulnerabilities, we integrated single-cell phenotypic analysis, bulk transcriptomics, and combinatorial drug screening across BRCA-mutant and BRCA-wild-type ovarian cancer models. PARP inhibitor treatment induced a dormant, non-proliferative state marked by polyploid giant cells and senescence-like features. Drug withdrawal led to cell cycle re-entry in a subset of cells, confirming dormancy reversibility. BRCA-wild-type models showed more rapid regrowth than BRCA-mutant counterparts, suggesting genotype-specific differences in escape dynamics. Bulk RNA sequencing across three temporal states, naive, dormancy (day 7 of treatment), and regrowth (day 10 post-withdrawal), revealed stepwise activation of gene programs involved in metabolic reactivation, stress response, and cell cycle progression. Drug screening with Olaparib combinations uncovered genotype-dependent vulnerabilities: anti-inflammatory agents showed broad efficacy, while metabolism inhibitors, DNA damage response, and cellular stress were more effective in BRCA-wild-type lines. These findings demonstrate that PARP inhibitor-induced dormancy is reversible and targetable, and BRCA status informs differential vulnerabilities. Our

integrative framework supports the development of state- and genotype-specific combination therapies to prevent recurrence and enhance the durability of PARP inhibitor responses in ovarian cancer.

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#### **P24: Immunophenotypic Diversity of Tumor-Associated Macrophages in Undifferentiated Pleomorphic Sarcoma and Myxofibrosarcoma**

Authors: Diana, Taungham Anekpuritanang, Chandhanarat Chandhanayyingyong, Somponnat Sampattavanich

##### **Presenter: Ms Diana**

Affiliation: Department of Pharmacology, Faculty of Medicine, Siriraj Hospital, Mahidol University

##### **Abstract:**

Undifferentiated pleomorphic sarcoma (UPS) and myxofibrosarcoma (MFS) are among the most common adult soft tissue sarcomas, yet treatment outcomes remain poor, particularly in advanced stages. Tumor-associated macrophages (TAMs), the predominant immune cells in the tumor microenvironment (TME), are known to contribute to tumor progression, metastasis, immunosuppression, and resistance to therapy. We hypothesized that distinct TAM subsets and their spatial interactions with immune and tumor cells across primary, recurrent, and metastatic lesions could serve as prognostic markers in UPS and MFS. To investigate this, we employed tissue-based cyclic immunofluorescence (t-CyCIF) to perform multiplexed immunostaining on formalin-fixed, paraffin-embedded (FFPE) tissue sections from UPS and MFS patients. The antibody panel included markers for myeloid and lymphoid cells, immune checkpoints, vasculature, proliferation, and stromal components. Preliminary analyses revealed distinct immune landscapes between UPS and MFS. We observed a higher prevalence of macrophages compared to lymphoid lineage cells in both tumor types. Further analysis categorized macrophages into three distinct tumor-associated macrophage (TAM) phenotypes: CD68+CD163+ M2, CD68+CD206+ M2, and CD68+CD163+CD206+ M2 macrophages. Notably, the distribution and frequency of these phenotypes varied between paired primary and metastatic lesions of UPS and MFS, suggesting dynamic changes in the tumor microenvironment during disease progression. Spatial neighborhood analysis suggested enhanced macrophage activity in metastatic UPS, potentially contributing to immune suppression or tumor support. Survival analysis confirmed that UPS was associated with poorer prognosis compared to MFS, consistent with prior clinical data. These findings highlight the immunophenotypic heterogeneity of TAMs in soft tissue sarcomas and their potential prognostic significance. Distinct TAM profiles and spatial immune contexts in UPS and MFS may inform future immunomodulatory treatment strategies.

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#### **P25: Gene Expression Profiling of Tumor Budding in Triple-Negative Breast Cancer Using Spatial Transcriptomics**

Authors: Pennapa Plypongisa, Rungdawan Wongsamart, Panisa Janyasupab, Pranisa Jamjantra, Doonyapat Sa-Nguanraksa, Malee Warnnissorn, Peti Thuwajit, Apichat Suratanee, Kitiporn Plaimas, and Chanitra Thuwajit

##### **Presenter: Pennapa Plypongisa**

Affiliation: Faculty of Medicine Siriraj Hospital

##### **Abstract:**

Tumor budding (TB) is a histopathological feature associated with tumor aggressiveness and adverse clinical outcomes across various cancers. In triple-negative breast cancer (TNBC), the underlying molecular mechanisms and gene expression changes linked to TB remain poorly defined. This study employed TempO-Seq® spatial bulk transcriptomics to analyze gene expression profiles of cancer cells in 13 TNBC tissue samples stratified by TB levels. Differential expression and mutual information analyses identified seven candidate genes associated with high TB: PRICKLE2, NGFR, LRIT3, ATP7A,

FAM110C, and EDIL3 (up-regulated), and LRRCC1 (down-regulated). To validate these findings, a quantitative real-time RT-PCR was performed on three additional TNBC samples. Results confirmed significant up-regulation levels of PRICKLE2, ATP7A, and EDIL3 in high TB cases compared to those in normal mammary cell, MCF-10A, while LRRCC1 was consistently down-regulated but not reach statistical significance. Further validation with larger sample cohorts is necessary to confirm these associations. Nonetheless, the identified gene expression patterns may offer valuable insights into the biological mechanisms underlying TB and highlight candidate biomarkers for prognosis and therapeutic stratification in this aggressive breast cancer subtype.

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**P26: Multi-modal Spatial Omics Integration for Ovarian Clear Cell Carcinoma Recurrence Prediction Using SpatialGlue Framework**

Authors: Mqondisi Fortune Mavuso

**Presenter: Mqondisi Fortune Mavuso**

Affiliation: National Taiwan University, International College

**Abstract:**

Background: Ovarian clear cell carcinoma (OCCC) exhibits pronounced spatial heterogeneity and immune infiltration patterns that correlate with recurrence. Our NanoString DSP analysis identified five immune-mimicry clusters linked to outcomes, but integrating proteomic and transcriptomic spatial data remains a challenge.

Methods: In this pilot study (n=4), we will apply SpatialGlue—a dual-attention graph neural network—to fuse GeoMx DSP proteomic data with transcriptomic cancer-testis antigen (CTA) expression profiles. SpatialGlue's intra-modality attention will model spatial protein-expression relationships, while its cross-modality layer will align protein and RNA features. We will fine-tune a supervised classification head on recurrence labels and use unsupervised embeddings to discover novel spatial neighborhoods. Expected Results: We hypothesize that multi-modal integration will (1) uncover coordinated protein–RNA spatial programs tied to recurrence, (2) map cross-talk between immune hot/cold regions and CTA expression, and (3) outperform single-modality classifiers by at least 10% AUROC. Attention maps will reveal interpretable spatial biomarkers.

Significance: This first application of advanced spatial AI to OCCC could yield actionable, spatially informed biomarkers and inform precision therapy. The framework and code will be shared to enable application to other histologically complex cancers.

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**P27: NGS-Guided Elucidation of Drug D's Cytotoxic Mechanism in Colorectal Cancer Cells via Mitochondrial-ER Stress Axis**

Authors: Gergorius Gena Maran, Huong Thi Nguyen, Tran Thu Ha, Chi-Ying F. Huang

**Presenter: Gergorius Gena Maran**

Affiliation: Center for Child Health, Universitas Gadjah Mada / Master Program in Biopharmaceutical Sciences, Institute of Biopharmaceutical, NYCU Taiwan

**Abstract:**

Colorectal cancer (CRC) remains among the most common malignancies worldwide and constitutes a major contributor to cancer-associated mortality. The efficacy of existing chemotherapeutic regimens is often hindered by adverse side effects and the emergence of resistance, thereby underscoring the necessity for alternative therapeutic approaches. In this study, we utilized next-generation sequencing (NGS)-based transcriptomic profiling to investigate the therapeutic repurposing potential of Drug D, a clinically utilized steroid hormone precursor, for the treatment of CRC. Transcriptomic analysis of CRC cells exposed to Drug D revealed a significant downregulation of genes

associated with NADPH metabolism, mitochondrial function, and oxidative stress regulation, thereby informing downstream mechanistic investigations. The cytotoxic effects of Drug D were substantiated through sulforhodamine B (SRB) and clonogenic survival assays. Moreover, flow cytometric analysis demonstrated that Drug D increased both total and mitochondrial reactive oxygen species (ROS) levels dose-dependently.

Further validation via immunoblotting indicated that Drug D activates the integrated stress response (ISR) through upregulation of ATF4 and CHOP, while concurrently suppressing the Nrf2/KEAP1 antioxidant defense axis. In addition, Drug D attenuated mitochondrial biogenesis, as evidenced by reduced PGC1 $\alpha$  expression, and impaired mitochondrial dynamics by downregulating S-OPA1, Mfn2, and Drp1. Notably, mitophagy was not induced; instead, the accumulation of mitochondrial stress correlated with the activation of intrinsic apoptotic pathways, as indicated by elevated caspase-3 and caspase-9 activities.

These findings demonstrate that NGS-driven transcriptomic analysis can effectively elucidate Drug D's mechanism of action in CRC, particularly its impact on mitochondrial and ER stress pathways, thereby supporting its potential as a candidate for precision therapeutic development.

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## **P28: Zonal hepatocyte reorganization post-bariatric surgery: Insights from integrated spatial transcriptomics and AI digital pathology**

Authors: Clare Yong, Christopher Hendra, Ashmita Saigal, Serene Lek, Gideon Ho, Thomas Forest, Richard Baumgartner, Chih-Liang Chin, Saswata Talukdar, Raluca Pais, Vlad Ratziu, Asad Abu Bakar Ali, Desiree Abdurrachim

**Presenter: Desiree Abdurrachim**

Affiliation: MSD

### **Abstract:**

**Background:** The liver possesses distinct zoned micro-architecture that exhibits variations in oxygen gradient, metabolic profiles, and morphogenetic fields, resulting in significant zone-specific differences in gene expression, epigenetic features, and regenerative capacity. Under conditions of stress and injury, the zonal organization of the liver undergoes substantial alterations, making it crucial to investigate zonal and spatial biology. This work aims to elucidate the interplay between liver zonation and the underlying mechanisms of Metabolic dysfunction-Associated Steatohepatitis (MASH) regression using spatial transcriptomics and AI digital pathology.

**Methods:** We performed spatial transcriptomics on paired-liver biopsy samples from a MASH patient pre-bariatric surgery (fibrosis stage 3) and 3.6 years post-surgery (fibrosis stage 1) using CosMx SMI Human Universal Cell Characterization Panel (1000-plex). H&E staining was performed following spatial transcriptomics to integrate zonal analysis using in-house AI digital pathology.

**Results:** We characterized 23,783 and 13,536 cells with mean total transcripts per cell of 152 and 252 for pre- and post-surgery sample, respectively. Data analysis revealed 3 main hepatocyte populations from each timepoint. In the pre-surgery sample, all hepatocyte populations had a higher presence in portal tract (PT) than in central vein (CV), which indicates disruption in zone-specific hepatocyte organization. However, in the post-surgery sample, one hepatocyte population had a higher presence in CV, one in PT, and one uniformly distributed, suggesting reorganization of hepatocyte populations. This reorganization is also suggested by the neighborhood enrichment score in the post-surgery sample where a pattern of decreased co-occurrence of cholangiocytes with hepatocyte population enriched in CV region was observed.

**Conclusion:** Integration of spatial transcriptomics and AI digital pathology honed our understanding of

spatial biology in MASH. Our pilot data revealed the disruption in the liver zonation in MASH, which could be recovered post-bariatric surgery intervention. Further studies should involve larger sample size and/or in preclinical setting to deepen our understanding and bridge translational gap.

---

### P29: Molecular makeup of early-onset oral cancer

Authors: Evgeny Denisov, Elena Kolegova, Marina Patysheva, Elizaveta Prostakishina, Denis Kulbakin, Andrey Polyakov, Liliya Yakovleva, Mikhail Kropotov, Igor Reshetov, Evgeny Choinzonov

**Presenter: Evgeny Denisov**

Affiliation: Cancer Research Institute, Tomsk National Research Medical Center

Abstract:

Early-onset oral cancer (EOOC) demonstrates an increase in incidence, aggressiveness, and poor response to therapy. Classic etiological factors such as tobacco, alcohol, and human papillomavirus are not related to EOOC. Mechanisms of the development and progression of this cancer remain unclear. Here, we performed genome, exome, 16S rRNA metagenome, single-cell and spatial transcriptome sequencing of EOOC (< 45 years old; n=35) as compared to oral cancer in older adults ( $\geq 45$  years; n=25). EOOC patients frequently harbored germline variants in the AHNAK2, KLK10, POPDC2, TTLL4 and other genes. EOOC demonstrated high frequency of somatic mutations in the TP53 and LOC112267881 genes, copy number aberration in the DEFB125 gene, and alterations in the genes involved in the MAPK and Rap1 signaling pathways. EPHB6 and CDKN2A mutations were related to EOOC recurrence. Tumor cells showed an increase in gene expression associated with phosphorylation, glycolysis, and non-canonical MAPK and Hippo signaling pathways. The EOOC microenvironment was found to be immunosuppressive due to increased number of M2 and TREM2+C1QB+ macrophages and myeloid-derived suppressor cells (MDSCs) and decrease in cytotoxic T and plasma cells. Nevertheless, the invasive edge demonstrated an immune response involving cytotoxic T and NK cells through GZMB-PGRMC1 and GZMB-IGF2R interactions. T cell-mediated immunity was also observed in adjacent normal tissue via upregulation of HLA-DR and T cell marker genes. In addition, EOOC invasive edge was enriched by vascular mimicry supported by M2 macrophages. EOOC microbiome was characterized by increase in Acidovorax caeni, Streptococcus infantis and Treponema amylovorum and decrease in Bacteroides acidifaciens, Bibersteinia trehalosi, Kocuria rhizophila and Pseudomonas veronii. Thus, EOOC demonstrates a distinct molecular makeup including specific cell populations, genes, mutations, signaling pathways, and bacterial species that can be potential diagnostic, prognostic, and therapeutic markers. This work was performed by The Consortium “Etiology and Pathogenesis of Oral Cancer in Young Adults” (EPOY: <https://epoy.org/>) and supported by Russian Science Foundation (# 22-15-00308).

---

### P30: Single-cell immune profiling of breast cancer patients under chemotherapy

Authors: Tatiana Gerashchenko, Anastasia Fedorenko, Marina Patysheva, Anton Fedorov, Anastasia Filatova, Pavel Iamshchikov, Evgeny Denisov.

**Presenter: Tatiana Gerashchenko**

Affiliation: Research Institute of Molecular and Cellular Medicine, Peoples' Friendship University of Russia (RUDN University)

Abstract:

The functional status of the immune system in triple-negative breast cancer (TNBC) plays a critical role in determining the efficacy of neoadjuvant chemotherapy (NACT). Only half of TNBC patients achieve a pathological complete response (pCR), while the remaining ones have a very poor prognosis. Early

prediction of tumor response to NACT, based on the unique functional characteristics of the immune cells, is essential for optimizing treatment strategies. Here we aimed to assess the functional state of immune blood cells in TNBC patients based on their response to NACT. Mononuclear cells were purified from the peripheral venous blood of 10 TNBC patients before NACT, after the 3rd and 21st days of NACT using a ficoll density gradient. Single cells were sequenced using BD Rhapsody Express (Becton Dickinson, USA) and Chromium Fixed RNA (10x Genomics, USA) technologies on NextSeq 2000 (Illumina, USA) and Genolab M (GeneMind Biosciences, China) platforms. The data were analyzed using Seurat and SingleR. Analysis of single-cell sequencing data revealed distinct populations of mononuclear leukocytes, including B- (CD79+), NK- (KIR2DL+), DC- (CD135+), T- (CD4+), T- (CD8+), T-regulatory (CD25+, FOXP3+) cells and monocytes (CD14+CD16- and CD14+CD16+). Patients who achieved pCR during NACT showed an increase in adaptive immune response effectors (CD4+, CD8+ T lymphocytes) and a decrease in the number of innate immunity cells (NK cells, monocytes). T cells exhibited overexpression of genes such as GNLY, NFKBI, JUN, FOS, and FosB involved in cytotoxic activation, proliferation, and differentiation, while monocytes showed overexpression of MNDA, ABCA1 related to myeloid differentiation, and NK cells - cells displayed overexpression of NFKBIA and NFKBIZ stimulating T cell differentiation. Conversely, patients with disease progression demonstrated a decrease in adaptive immune response effectors and an increase in innate cells. NK cells exhibited overexpression of CD69, GNLZ, GZMA, GZMB, and GZMH genes involved in nonspecific cytotoxicity, while monocytes showed overexpression of BCL6, CCR1 genes involved in the regulation of the immune response. In summary, poor response to NACT in TNBC patients was associated with altered activation of adaptive immunity cells. This work is supported by Russian Science Foundation (grant #22-75-10128).

---

### **P31: Transcriptional similarities between embryonic and tumor cells reveal clues to cellular plasticity**

Authors: Maxim Menyailo, Anna Khozyainova, Daria Zhigalina, Anastasia Korobeynikova, Maria Tretyakova, Anton Fedorov, Evgeny Topolnitsky, Evgeny Rodionov, Sergey Miller, Evgeny Denisov

**Presenter: Maxim Menyailo**

Affiliation: TNRMC

#### Abstract:

**Introduction:** Cancer remains one of the leading causes of mortality worldwide, primarily due to therapy resistance, metastasis, and recurrence. These phenomena are associated with tumor cell plasticity, which enables them to alter their phenotype. The process of oncogenesis is similar to embryogenesis, as both utilize common signaling and molecular pathways in their development. However, while embryogenesis progresses toward differentiation, oncogenesis often exhibits a reverse trend. Comparing the transcriptional profiles of early embryonic cells and tumor cells can help uncover the mechanisms of cellular plasticity and refine markers for progenitor and stem cells.

**Material and method:** The study was conducted using a model of embryoid bodies (day 18 of spontaneous differentiation) and samples of lung adenocarcinoma tumors obtained from four patients. Embryoid bodies were cultured from induced pluripotent stem cells (iPSCs), forming rudiments of all three germ layers. Single-cell RNA libraries were prepared from cell suspensions using the 10X Genomics platform. Data analysis was performed using the Cell Ranger pipeline and additional tools for cell typing.

**Result and discussion:** The analysis revealed the presence of cells from all three germ layers and pluripotent stem cells in the embryoid bodies. In the tumor samples, major populations of immune, stromal, and epithelial cells were identified, along with tumor cells. Data integration resulted in the identification of 26 transcriptionally distinct clusters. Clusters specific to embryoid bodies consisted primarily of neuronal progenitors, while tumor-specific clusters exhibited epithelial marker expression but lacked the expression of genes associated with the epithelial-mesenchymal transition and stemness.

Analysis of tumor cells transcriptionally similar to embryoid body cells revealed stem-like tumor cells resembling primitive ectoderm and endoderm. Some clusters demonstrated transitional states between epithelial and mesenchymal phenotypes, indicating dedifferentiation of tumor cells.

Conclusion: Comparison of the transcriptional profiles of embryoid bodies and tumor cells allowed the identification of various tumor cell states: epithelial, epithelial-stem, dedifferentiated, and transitional toward a mesenchymal state. Further research should focus on deeper profiling of both tumor and embryonic cells, as well as modeling dynamic processes. This will shed light on the mechanisms of cellular plasticity and pave the way for the development of novel cancer therapies.

---

### P32: Single-cell transcriptomic analysis of rare cancers: JMML and soft tissue sarcomas

Authors: Ikonnikov A.V., Volchkov E.V., Kopantseva E.E., Fetisov T.I., Menyailo M.E., Gurzhikhanova M.Kh., Poryadina E.A., Kirsanov K.I., Yakubovskaya M.G., Denisov E.V., Maschan M.A.

#### Presenter: Alexander Ikonnikov

Affiliation: Research Institute of Molecular and Cellular Medicine, Peoples' Friendship University of Russia (RUDN University), 115093 Moscow, Russia

#### Abstract:

Objectives: Single-cell RNA sequencing (scRNA-seq) provides detailed insights into the cellular composition of tumors, making it especially valuable for studying rare cancers. Juvenile myelomonocytic leukemia (JMML) and undifferentiated pleomorphic sarcoma (UPS) are aggressive malignancies with complex cellular heterogeneity and limited treatment options. This study applies scRNA-seq to characterize cell populations in JMML and UPS patient samples and to explore potential molecular features relevant to disease progression and therapy.

Materials and Methods: BM cells from 8 JMML patients with PTPN11, NF1, NRAS and KRAS gene mutations were used. The control group included public scRNA-seq data of bone marrow (BM) samples from 7 healthy children. Additionally, 1 tissue sample from patients with undifferentiated pleomorphic sarcoma were analyzed. Single-cell libraries were prepared using the 10x Genomics Chromium Controller (10x Genomics, USA) and the Single Cell 3' Reagent Kit. Sequencing was performed using the Genolab M (GeneMind, China). The bioinformatics analysis involved cell and gene filtering, data normalization, integration, and cell type annotation was performed using Scanpy v1.9.5 and Seurat v. 5.0.3. Batch effect correction was performed with the Harmony package. Metabolic pathway analysis was conducted using GSEAp and DecoupleR. Aneuploidy analysis for tumor cell annotation was performed with the SCEVAN package. Gene regulatory network analysis was conducted using pySCENIC package.

Results: JMML samples exhibited increased MAPK pathway activity in hematopoietic progenitors and enhanced myelomonocytic proliferation. The JMML sample with aggressive clinical progression and a PTPN11 mutation showed a significant number of hematopoietic stem cells (HSCs), early progenitors, and erythroid lineage cells. Additionally, granulocyte/monocyte progenitors from JMML samples were characterized by upregulated GTPase and histone deacetylation pathways, while mRNA splicing pathways and oxidative phosphorylation processes were downregulated in JMML group compared to healthy BM (FDR < 0.05). Batch-corrected principal component correlation (PCA) analysis of transcriptional profiles in cells revealed negative correlation between JMML and healthy controls. HSCs in JMML samples are characterized by significantly higher activity of regulons associated with the regulation of hematopoiesis (GATA2, PBX1). Hyperactivation of MEIS1 and the ERG oncogene may indicate dysregulated hematopoiesis.

Four subpopulations of tumor cells and seven subpopulations of tumor microenvironment have been identified in UPS sample. The expression of chemoresistance genes has been detected, including ULK1,

LUM, GPNMB, KLF4 (doxorubicin and ifosfamide), CAVIN1 (doxorubicin), and AHNAK2 (gemcitabine) in tumor cells and ETS1 (gemcitabine) in TME.

**Conclusions:** This study identified molecular processes that distinguish JMML bone marrow progenitor cells from healthy bone marrow - hyperactivation of MAPK pathways and hematopoiesis-specific regulons. Putative chemoresistance genes identified in tumor cells from UPS sample may provide insights valuable for the development of targeted therapeutic strategies.

---

### **P33: Single-cell analysis of immune microenvironment and tumor-immune interactions in soft tissue sarcoma**

Authors: Kopantseva E.E., Ikonnikov A.V., Fetisov T.I., Toropov, A.L., Menyailo M.E., Tarakykova, Bokhyan, B.Y., Kozlov, N.A, A.A.Kirisanov K.I., Yakubovskaya M.G., Denisov E.V.

**Presenter: Elena Kopantseva**

Affiliation: Research Institute of Molecular and Cellular Medicine, Peoples' Friendship University of Russia (RUDN University)

**Abstract:**

Soft tissue sarcomas (STS) are rare, complex, aggressive tumors of mesenchymal origin that are currently classified into more than 70 subtypes. Due to their rarity, they are frequently misdiagnosed. And even after a correct diagnosis, their treatment is hampered by the lack of knowledge of their molecular mechanisms and the subsequent lack of therapeutic targets. Immunotherapy has been shown to hold promise for treatment of some types of sarcomas, which suggests that studying the tumor-immune interactions in STS is of special importance.

The aim of this project was to study the immune microenvironment and the interactions between tumor and the immune populations in different types of STS at the single cell level. The tumor samples from 11 patients with synovial sarcoma (SyS), 7 patients with undifferentiated pleomorphic sarcoma (UPS), 5 patients with myxofibrosarcoma (MFS), and 1 patient with pleomorphic rhabdomyosarcoma (pRMS) were prepared for single cell analysis. The tumor samples were fixed and dissociated according to the Tissue Fixation & Dissociation for Chromium Fixed RNA Profiling protocol (10x Genomics). Single cell transcriptome libraries were constructed according to the Chromium Fixed RNA protocol (10x Genomics) and sequenced on the Genolab M platform (GeneMind). The sequence files were analyzed using Cell Ranger 7.1.0, DoubletCollection, Seurat package, SCEVAN, and CellChat packages.

The UPS and MFS display a diverse immune cell niche, with CD4+ and CD8+ T cell populations, NK cells, dendritic cells and macrophages. The CD4+ T cells in these two types of STS do not possess a high expression of FOXP3, which suggests low T reg infiltration. MFS, in comparison with UPS, has a more pronounced cluster of plasmacytoid dendritic cells ( IL3RA, IRF7, IRF8, LILRA4, CLEC4C). The T cell populations in pRMS largely correspond to those of UPS. However, in pRMS there is a distinct group of Treg cells, multiple markers of exhaustion are present in cytotoxic T cells (LAG3, HAVCR2, EOMES, CD74), and the macrophages display myeloid checkpoint markers (SIGLEC1, SIRPA, CSFR1, HAVCR2, CD74). The analysis of ligand-receptor pairs indicates the presence of MIF-CD74 interactions between all pRMS tumor clusters and the immune cells, as well as APP, PTN, and CXCL12 signaling predominantly between non-myogenic tumor clusters and immune cells. The immune microenvironment in SyS is markedly poor, which has been noted in previous reports. However, 2 out of 11 synovial sarcoma samples possess cytotoxic T cells with increased activation and exhaustion signatures, as well as macrophages with anti-tumor properties. This corresponds with the presence of a unique poorly differentiated HOX11+ tumor cluster, which is functionally enriched in genes related to embryonic development (HOX11, HOX10, MAPK) and glycolytic metabolism (LXH9). In conclusion, we have conducted single-cell analysis of the tumor-immune landscape in several major types of STS. The presence of the pronounced plasmacytoid dendritic cell cluster in MFS may serve as

a diagnostic tool to help distinguish MFS from UPS. The discovered tumor-immune connections (MIF signalling, APP signalling) in pRMS can be targeted to help the immune cells recognize and attack cancer cells. The HOX11+ tumor population in SyS may have value as a diagnostic indicator to predict the progression of this malignancy.

---

#### **P34: Mapping Cellular Diversity Across 18 Human Organs Using High-Resolution Spatial Transcriptomics**

Authors: Cheng Yee Chung, Gregor Kent, David Klinzing, Ufuk Degirmenci

**Presenter: Ufuk Degirmenci**

Affiliation: Next Level Genomics

Abstract: Spatial transcriptomics with the CosMx 6k panel enables deep biological insights from a single slide, offering both exceptional efficiency and high resolution. By integrating supervised and unsupervised cell typing approaches, spatial profiling reveals distinct molecular signatures and cellular dynamics across 18 different organs, capturing diverse tissue contexts with remarkable clarity. Key examples include estrogen receptor signaling in breast tissue, collagen formation in skin, extracellular matrix remodeling in tongue, and adaptive immune responses in tonsil, underscoring the powerful insights derived from individual slides. Importantly, this data is generated from local populations in Singapore, enhancing its relevance for regional studies and translational research. While individual slides provide substantial discoveries, analyzing multiple slides significantly amplifies biological understanding, supporting comparative analyses across disease states, patient cohorts, and experimental conditions. These findings demonstrate the transformative potential of spatial transcriptomics for advancing molecular pathology and precision medicine. Next Level Genomics is committed to empowering researchers and clinicians with cutting-edge spatial biology solutions and access to locally generated datasets, driving groundbreaking discoveries and clinical innovation.

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#### **P35: Spatial Whole Transcriptome Profiling Uncovers Tumor Microenvironment Signatures and Novel Biomarkers for Chemotherapy Response in Thai Cholangiocarcinoma**

Authors: Supawan Jamnongsong, Patipark Kueanjinda, Hay Mar Oo, Suchada Srifa, Ananya Pongpaibul, Siwanon Jirawatnotai, Krittiya Korphaisarn, Somponnat Sampattavanich

**Presenter: Supawan Jamnongsong**

Affiliation: Siriraj Center of Research Excellence for Systems Pharmacology, Department of Pharmacology, Faculty of Medicine Siriraj Hospital, Mahidol University

Abstract: Cholangiocarcinoma (CCA) is a heterogeneous malignancy of the biliary tract, often diagnosed at advanced stages when surgical resection is not feasible. Despite gemcitabine combined with platinum-based chemotherapy being the standard first-line treatment, therapeutic resistance is common and predictive biomarkers are lacking. To address this, we employed spatial whole transcriptome profiling to investigate tumor microenvironmental (TME) features associated with chemotherapy response in Thai CCA patients. Spatial transcriptomics revealed that tumors from chemosensitive patients were enriched in immune cell infiltration, notably CD4<sup>+</sup> and CD8<sup>+</sup> T cells, and showed suppression of ERK/MAPK signaling, E2F targets, and G2M checkpoint pathways. In contrast, chemoresistant tumors displayed an immunosuppressive TME, characterized by regulatory T cells, exhausted T cells, and elevated signatures of hypoxia and epithelial–mesenchymal transition (EMT). These findings were independently validated using multiplexed tissue imaging, confirming distinct spatial immune landscapes. To translate these insights into clinical practice, we developed a gene expression-based biomarker panel, CCA20, capable of stratifying patients by chemotherapy response and prognosis. Notably, CCA20 maintained predictive performance across samples with varying tumor purity and ethnic backgrounds. In summary, our study

highlights the utility of spatial transcriptomic profiling in uncovering TME-linked mechanisms of chemotherapy resistance in CCA and introduces CCA20 as a promising biomarker for guiding personalized treatment strategies.

---

### **P36: Association of Eosinophil and Neutrophil Indices with Response to First-Line Pembrolizumab-Gemcitabine Plus Platinum in R/M HNSCC**

Authors: Sau Yee Kok, Nabilah Tuan Zaid, Audrey Weng Yan Lee, Jia Wern Pan, Joanna Mei Ch'wan Lim, Thomas George Kallarakkal, Chin Heng Fong, Ibtisam Muhamad Nor, Nahjatul Kursyiah, Yoke Fui Wong, Wan Zamaniah Wan Ishak, Sok Ching Cheong, Kue Peng Lim

**Presenter: Sau Yee Kok**

Affiliation: Cancer Research Malaysia

Abstract: Despite improved outcomes with pembrolizumab combined with chemotherapy in recurrent/metastatic head and neck squamous cell carcinoma (R/M HNSCC), predictive biomarkers for treatment response remain limited. In this interim analysis of single-arm phase II trial (NCT05286619), we evaluated clinical and immune-related predictors of response in 19 R/M HNSCC patients (data cutoff: March 15, 2025) receiving first-line pembrolizumab plus gemcitabine and platinum (GP). This prospective cohort stratified patients into responders (n=10; complete or partial response) and non-responders (n=9; stable or progressive disease) based on RECIST evaluation at cycle 8 (6 months). Associations with baseline PD-L1 combined positive score (CPS), tumour-infiltrating lymphocyte (TIL), stromal TIL scores, and haematological indices from peripheral blood were examined. Univariate analysis revealed that the eosinophil-to-lymphocyte ratio (ELR) was significantly higher ( $p < 0.05$ ) in responders [0.17 (0.02-0.20)] compared to non-responders [0.09 (0.03-0.16)]. In contrast, the neutrophil-to-lymphocyte ratio (NLR) demonstrated dynamic predictive value. Responders, particularly those achieving complete response, displaying higher baseline NLR followed by a progressive decline during treatment, whereas non-responders showed no significant NLR change. No significant associations were observed between PD-L1 CPS, TILs or stromal TILs and treatment response. Elevated baseline ELR in responders suggests that eosinophils may contribute to anti-tumour immunity through immune cell recruitment or modulation of the tumour microenvironment. A declining NLR in responders may reflect suppression of neutrophil-driven inflammation, which may be critical for effective chemoimmunotherapy. Our findings support further investigation into baseline tumour immune subtypes encompassing immune cell subsets, checkpoint molecule expression, and tumour-intrinsic features to better characterize the interaction between the immune landscape and tumour biology in relation to treatment response. This approach may help identify immune profiles associated with favourable responses to chemoimmunotherapy in R/M HNSCC.

---

### **P37: Advances in integration of spatial transcriptomics and cell morphology to study cancer-associated fibroblasts heterogeneity**

Authors: Hai C.T. Nguyen, Joe Poh Sheng Yeong, David Joon Ho

**Presenter: Hai Nguyen**

Affiliation: National Cancer Center, Republic of Korea

Abstract: Cancer-associated fibroblasts (CAFs) are a heterogeneous and spatially organized component of tumor microenvironment that play critical roles in tumor progression, immune modulation, and therapy resistance. Recent technological advances in spatial transcriptomics (ST) have transformed our ability to profile gene expression *in situ*. At the same time, computational pathology enables the extraction of morphological and contextual information from histological stains. Integrative frameworks that combine these modalities now allow illuminating the complex landscape of CAF biology with high spatial

resolution. This poster explores cutting-edge approaches that unify transcriptomic and morphological data to dissect CAF subtypes and their spatial dynamics.

We highlight METI (Morphology-Enhanced Spatial Transcriptome Analysis Integrator), a deep learning framework integrating hematoxylin and eosin (H&E)-stained histology with ST to identify tissue architecture and infer cell states. STAIG (Spatial Transcriptomics Analysis via Image-Aided Graph Contrastive Learning) leverages machine learning to predict spatial gene expression from histological features, bridging gaps in transcriptomic resolution. Additionally, GIST (Gene expression and histology Integration for SpaTial cellular profiling) refines spatial domain detection by harmonizing tissue morphology with gene expression. Lastly, SpotLight (Spatial Profiling of Tumors by Leveraging Imaging and Transcriptomics) enables deconvolution of bulk ST data to localize specific CAF subpopulations using single-cell references. These tools have uncovered functionally distinct CAF subsets by bridging spatial omics and histology. These innovations demonstrate how multimodal spatial analysis can decode complexity of stroma tissue and enrich therapeutic strategies targeting CAF-driven immunosuppression. However, current approaches combining ST with morphological data encounter issues such as resolution disparities, absence of validated ground truth, and insufficient CAF-specific modeling. These limitations hinder precise characterization of CAF heterogeneity. Future efforts should focus on creating interpretable, CAF-targeted tools, and developing multi-modal atlases across tumor types to more effectively capture the complexity and dynamics of CAFs within the tumor microenvironment.

---

### **P38: Systematic Benchmarking of Normalization and Batch Effect Correction Methods in Spatial Proteomics**

Authors: Sydney Rechie S. Necesario, Ko-Chen Chen, Maria José Carbajal Hernández, Thang Truong Le, Alice Hsiang Kuo Yang, Wei-Chou Lin, Lin-Hung Wei, Ruby Yun-Ju Huang

**Presenter: Sydney Rechie Necesario**

Affiliation: Department of Biomedical Engineering, National Taiwan University

**Abstract:** Spatial proteomics enables multiplexed quantification of proteins within intact tissue sections, offering a powerful framework to resolve cellular organization, tumor microenvironment (TME) dynamics, and both intra- and intertumoral heterogeneity *in situ*. Notably, the GeoMx Digital Spatial Profiling (DSP) platform facilitates comprehensive spatial protein expression across histologically defined compartments, yet technical variability, particularly slide-specific batch effects, remains a major source of confounding variation. Although normalization and batch effect correction are essential for ensuring data integrity, a systematic evaluation of preprocessing strategies tailored to spatial proteomics has been lacking. Here, we benchmarked 16 normalization methods and five batch correction strategies using high-plex spatial proteomic profiling with the 580-plex Immuno-Oncology Proteome Atlas (IPA) panel on the GeoMx DSP platform across 324 regions of interest (ROIs) from six patients, including ovarian clear cell carcinoma, endometriosis, and recurrent disease, with both MET fusion-positive and –negative profiles. Data were collected across two experimental batches, introducing measurable slide-specific variation. Using a multi-metric evaluation framework, we assessed each method's ability to correct unwanted variation while preserving biologically meaningful spatial structure. Among all approaches, the combination of trimmed mean of M-values (TMM) normalization with ComBat-SVA batch correction consistently demonstrated superior performance, maintaining distinctions in immune composition, tumor progression, and MET fusion-associated features. This study establishes a robust preprocessing framework for spatial proteomics and provides a reference standard to guide normalization and batch correction choices in future spatial omics analyses.

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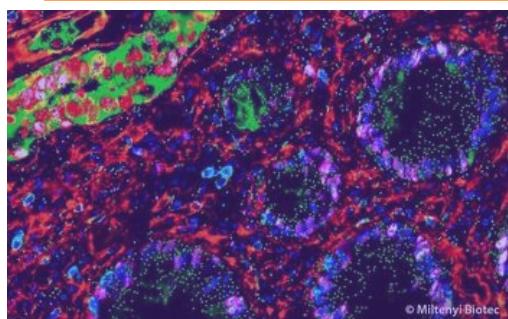
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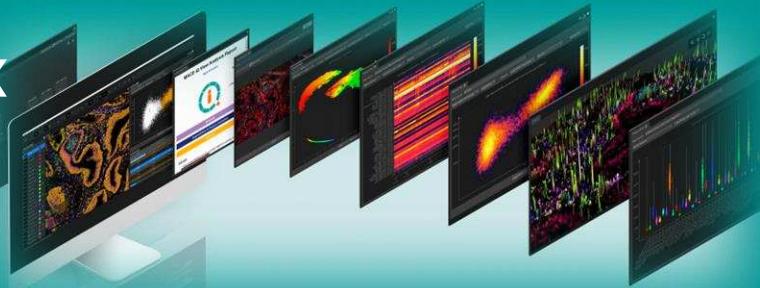
Application note  
**From tissue sample to publication-ready results in record time**

► Discover MACSima Platform's streamlined same-section multiomics workflow for spatial biology

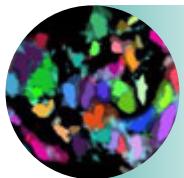


# Simplify complex analysis

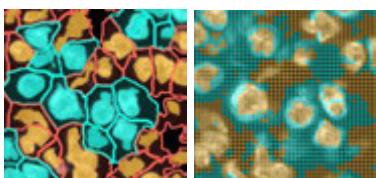
with MACS® iQ View  
Image Analysis Software



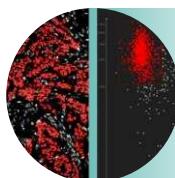
## Key features



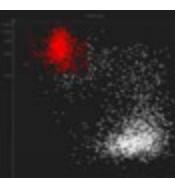
Easy & Fast Segmentation



**Segmentation method:**  
Nucleus – adv. morphology for tissue (left panel)  
Cytoplasm – Constrained donut (left panel)  
SuperPixel, 5 pixel (right panel)



Gating



Feature plot (Scatter plot)  
Gated region in red

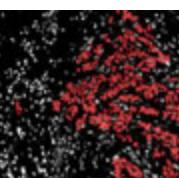
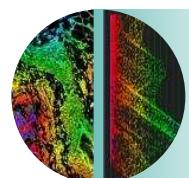
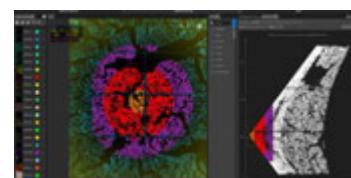


Image view  
Gated cells in red



Distance maps

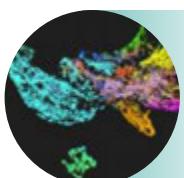


Distance mapping facilitates the definition of cell groups based on their position relative to your cells of interest.

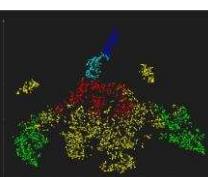
Pre-defined segmentation options based on proven and fast algorithms let you start your analysis immediately. Alternatively, you can flexibly use your own trusted segmentation mask.

Phenotype your cells based on biomarker expression, location, or cell shape properties through your favourite gating strategies.

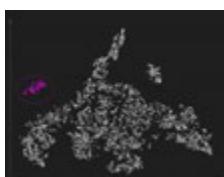
Define distance zones across tissue and analyze the immunophenotypes of your cells across their spatial arrangement using heat map of additional markers



Clustering & dimension reduction



t-SNE displayed on scatter plot with K-means clusters highlighted



t-SNE displayed on scatter plot with manual gate (purple) highlighted

Perform meaningful and unbiased data analysis of a large number of features via t-SNE, UMAP, and k-means clustering, and more.

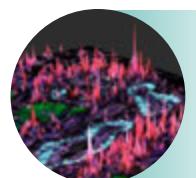


Workflow editor

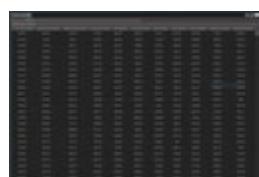


The workflow editor allows you to organize your analysis steps visually in an intuitive manner.

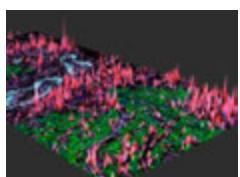
Track all the selections, gating, and combinations you applied during the analysis and can easily follow your whole train of thought. Easily apply stored analysis workflows to other datasets.



Data visualization & export



All data are accessible in several table formats allowing downstream statistical calculation.



Heightmap: 3D representation of an expression pattern.

Images and videos are also easy to export which allows you to create composite images and highlight areas of interest for data presentation.

### List of selected publications



Discover how the MACSima Platform is enabling fundamental, translational, and clinically relevant discoveries.

### Application note

**Analyzing complex spatial data with MACS® iQ View**





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Capture rare populations



Deeper profiling



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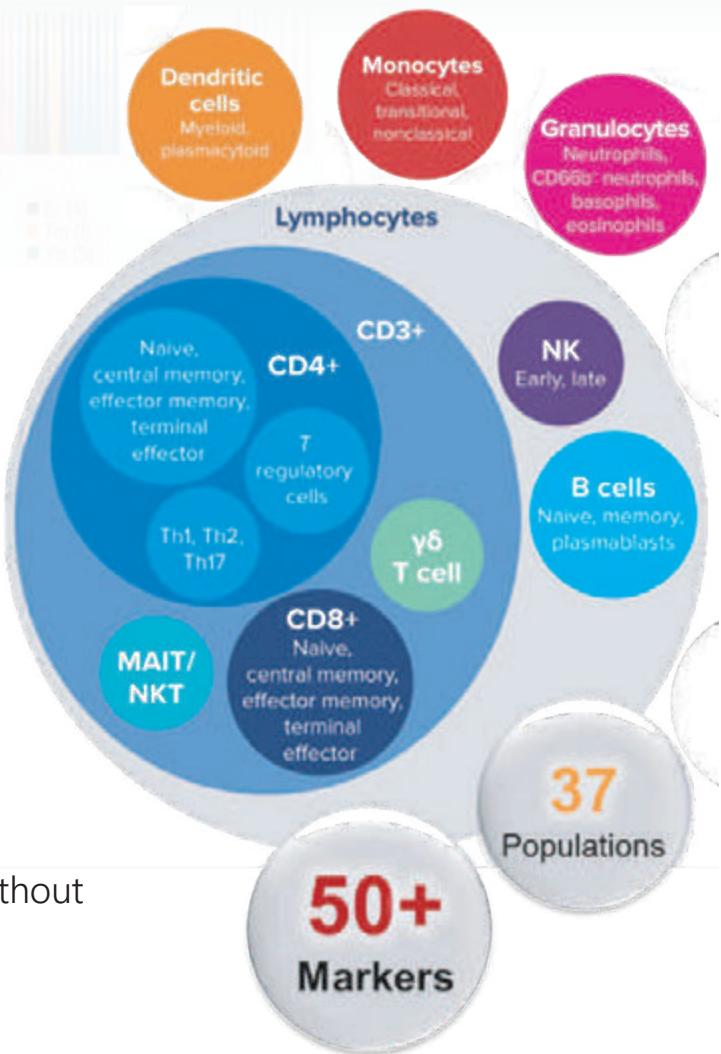


Sample multiplexing



Fixed-and-shipped

Sample for signal preservation without bleaching problem



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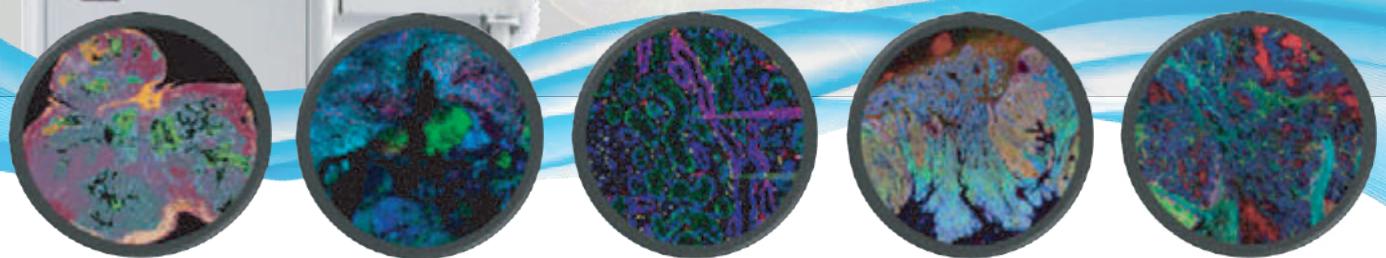


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40-plus markers imaged simultaneously



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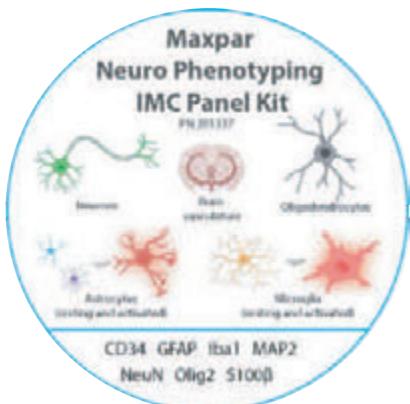
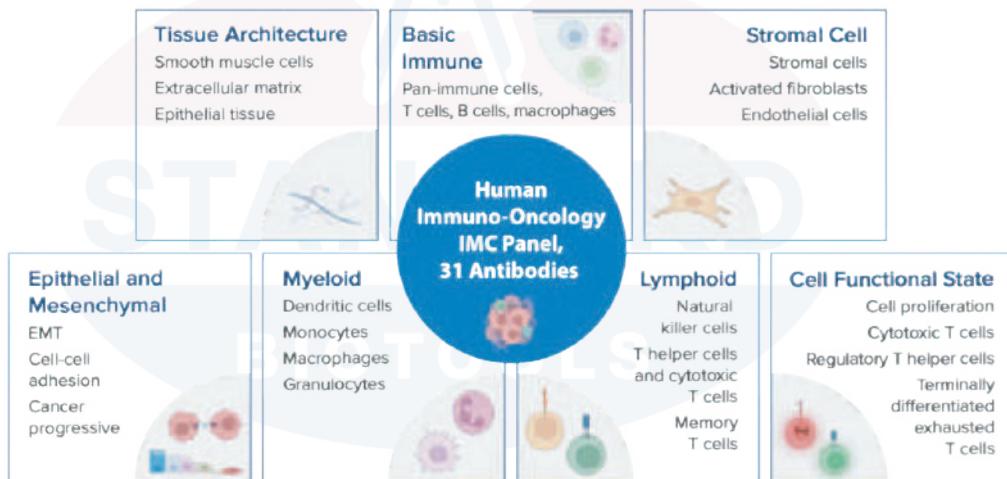


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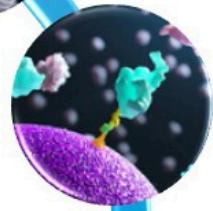
### Scale Bioscience

QuantumScale Single Cell RNA



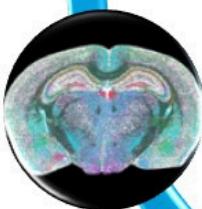
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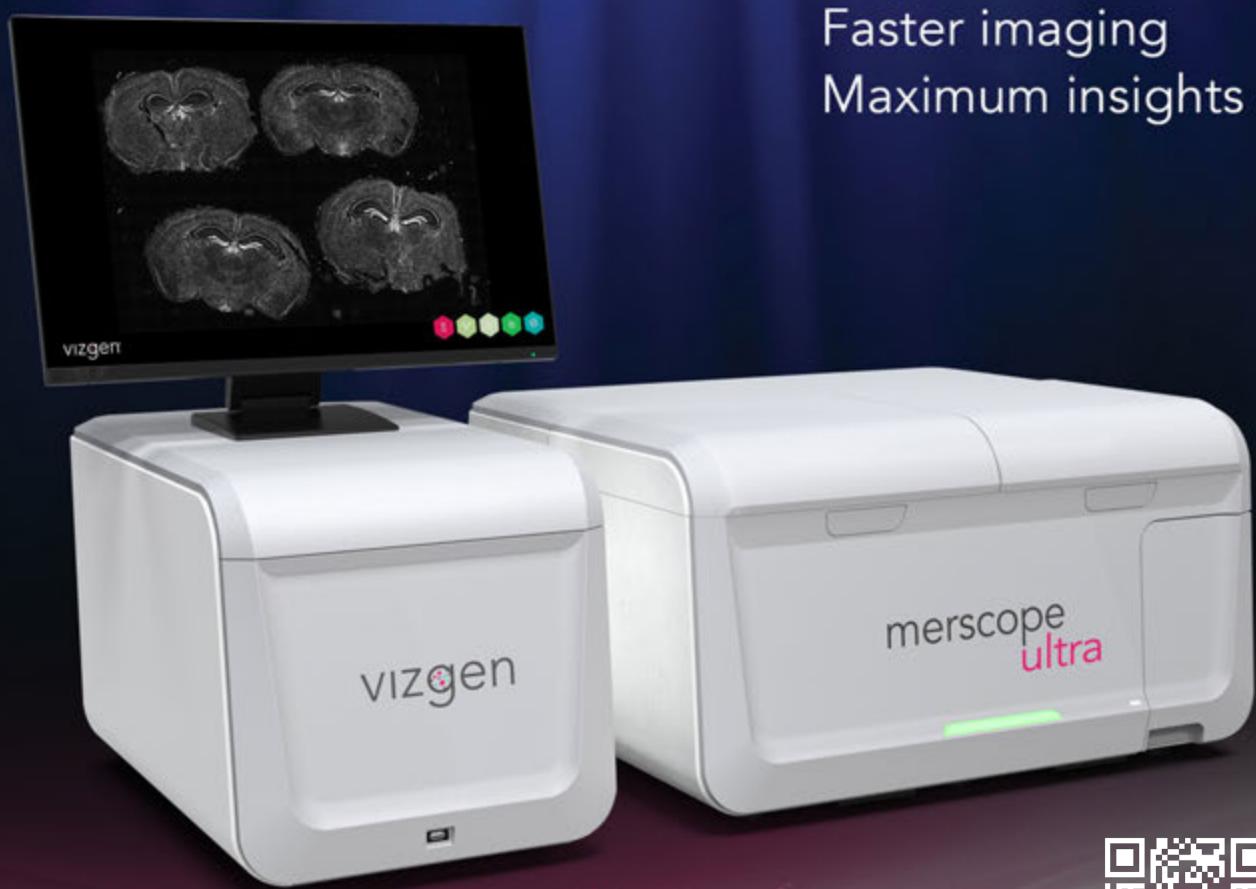
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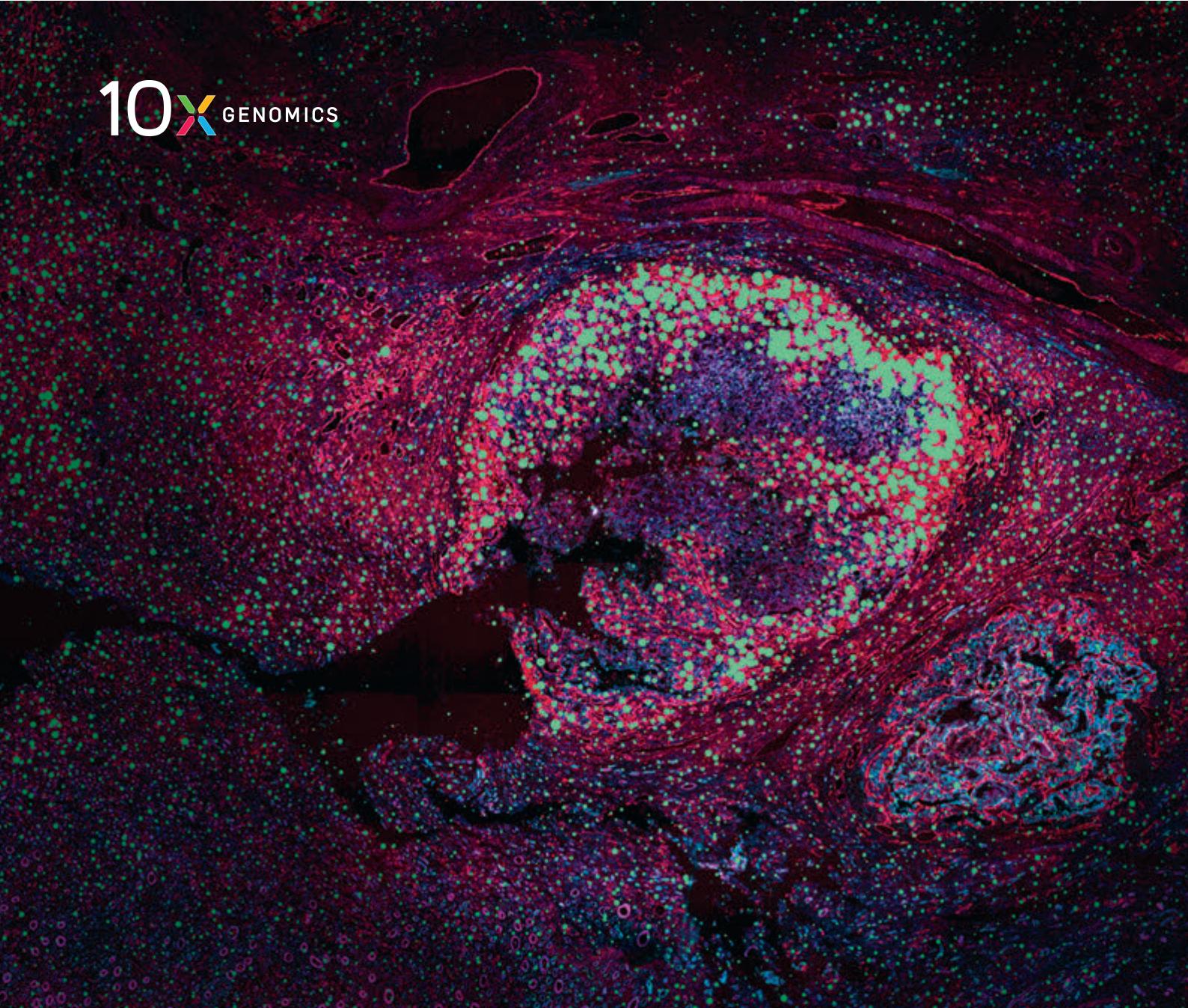
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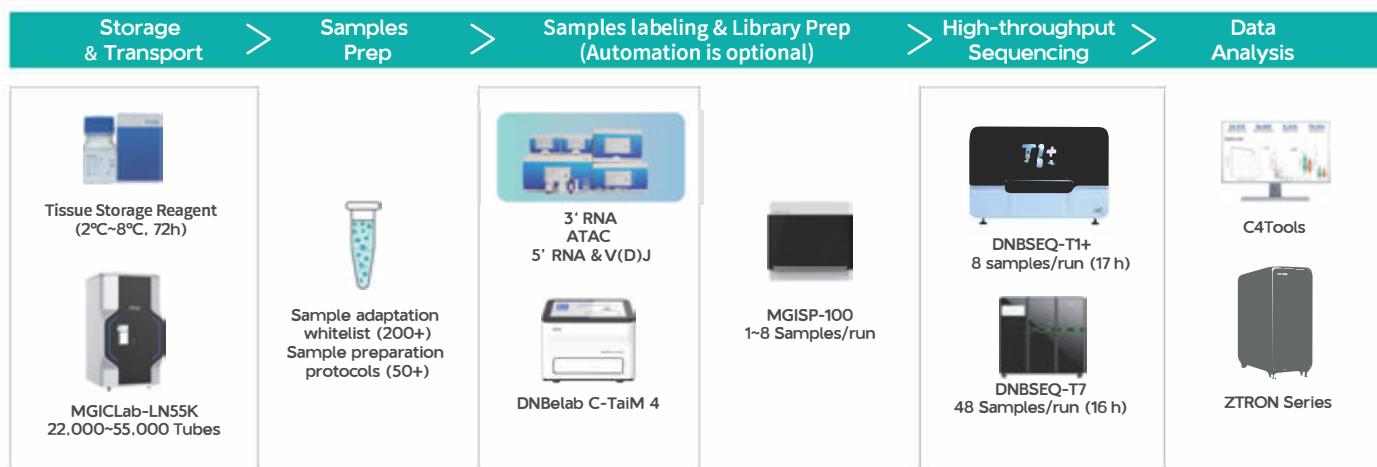
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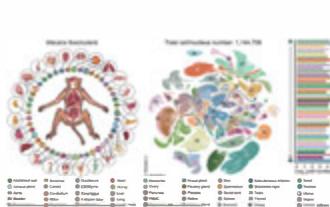
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## ■ One-stop Platform for Single-cell Omics Research



## ■ Application Cases

Case 1 Single-cell atlas of Macaca fascicularis



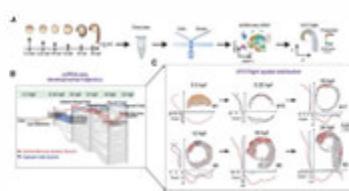
Han, L. et al, Nature 2022

Case 2 Human totipotent stem cell



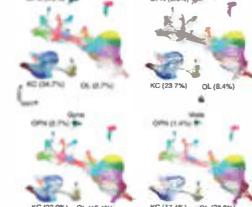
Mazid, MA. et al, Nature 2022

Case 3 Zebrafish development and differentiation



Liu, C. et al, Developmental Cell 2022

Case 4 Ant brain nuclei



Li, Q. et al. Nature Ecology & Evolution 2022

### Samples

45 organs or tissues of  
Macaca fascicularis

### Samples

Human embryonic-like cells  
at the 8-cell stage (8CLC)

### Samples

Zebrafish embryo

### Samples

Ant brain

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x  
3cm

2cm  
x  
2cm

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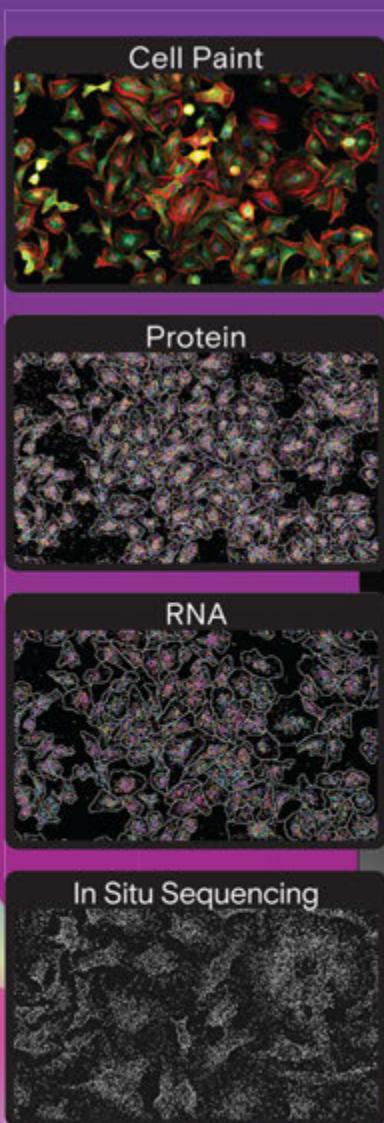
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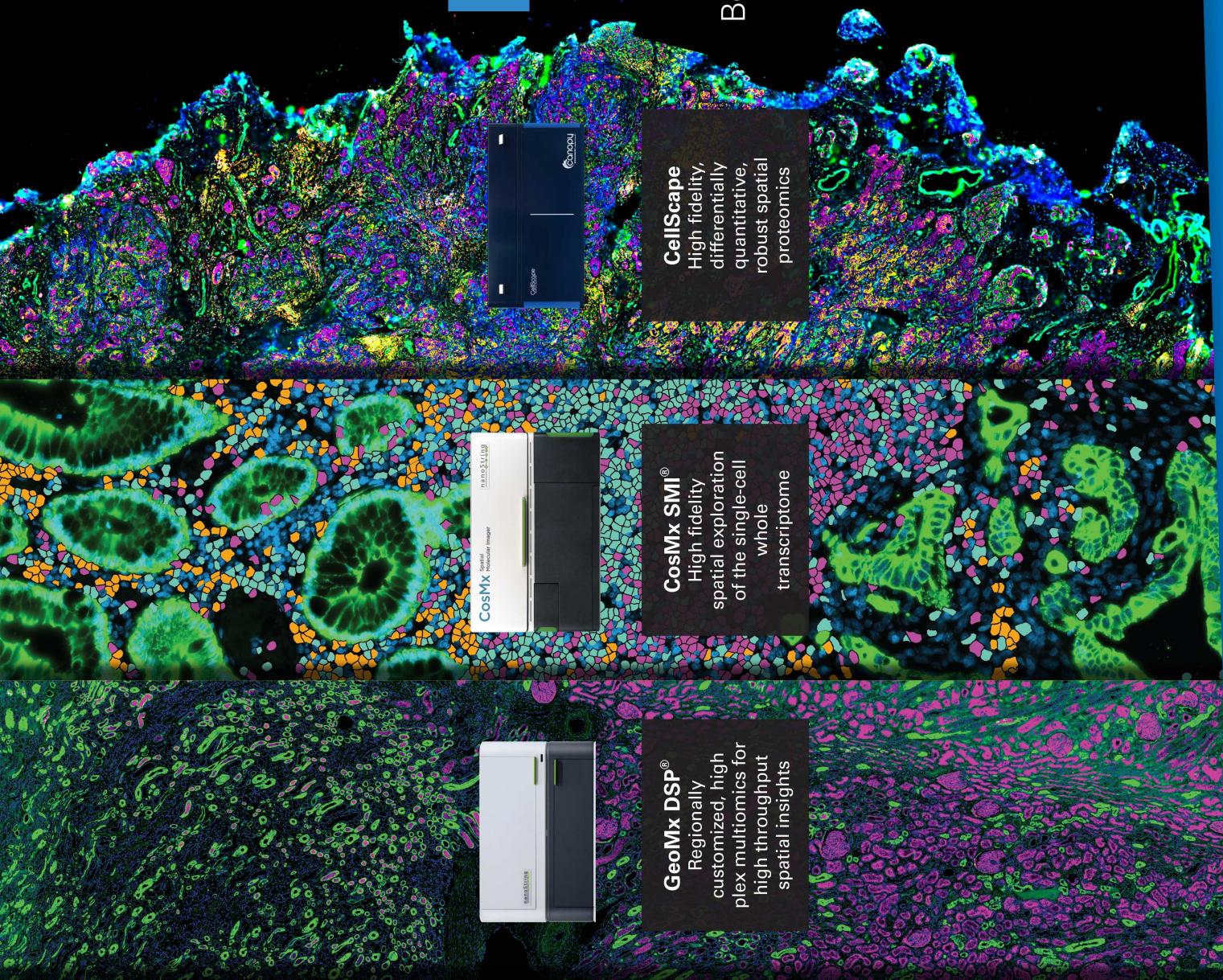


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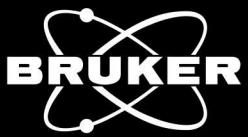


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## 60-marker human IO antibody panel

Immune Cell Typing		Lymphocyte Profiling		Tissue Architecture		Tumor-Specific Markers	
CD68	HLA-A	CD107a	Granzyme B	CD31	Beta-actin	Keratin 14	Iba1
CD4	HLA-DR	CD57	TOX	E-Cadherin	Podoplanin	Bcl-2	EpCAM
CD44	CD14	FOXP3	TCF-1	SMA	Collagen IV	PanCK	GPI100
CD45RO	CD20	CD21	CD79a	Vimentin	Caveolin	Keratin 8/18	TP63
CD45	CD3e	CD38	CD39	CD34	b-Catenin1	ER	Keratin 5
CD11c	CD56						
CD8							
Immune Activation		Proliferation		Myeloid Profiling			
IFNG	PD-L1	Histone H3 Phospho (Ser28)		iNOS	CD11b		
IDO1	VISTA	PCNA		CD66	CD206		
PD-1	LAG3	Ki67		MPO	CD209		
ICOS				CD163			

## 24-marker mouse FFPE IO antibody panel

T Cell Markers		Structural, Proliferation and Tumor Markers		Macrophage Markers	
CD3e	T cell	Vimentin	Mesenchymal cells, epithelial-mesenchymal transition (EMT)	CD68	Macrophage
CD4	Helper T cell	CollA1	Extracellular matrix	CD206	M2 macrophage
CD8	Cytotoxic T cell	Caveolin	Integral membrane protein	F4/80	Macrophage
FOXP3	Regulatory T cell	PanCK	Epithelial cells, tumor marker	Immune Cell Markers	
CD44	Activated T cell	Ki67	Proliferation marker	CD11c	Panleukocytic
CD45R/B220	Naive T cell	Other Cell Type Markers		Ly6g	Peripheral neutrophils, monocytes and granulocytes
B Cell Markers		CD31	Endothelial cells	FCRy	Multiple immune cell types
CD20	B cell	CD36	Adipocyte progenitors	CD45	Nucleated hematopoietic cells
		Ter119	Erythroid cells	Iba1	Microglia
				SI00A9	Myeloid differentiation

42-marker Human Neuro Panel is coming soon!

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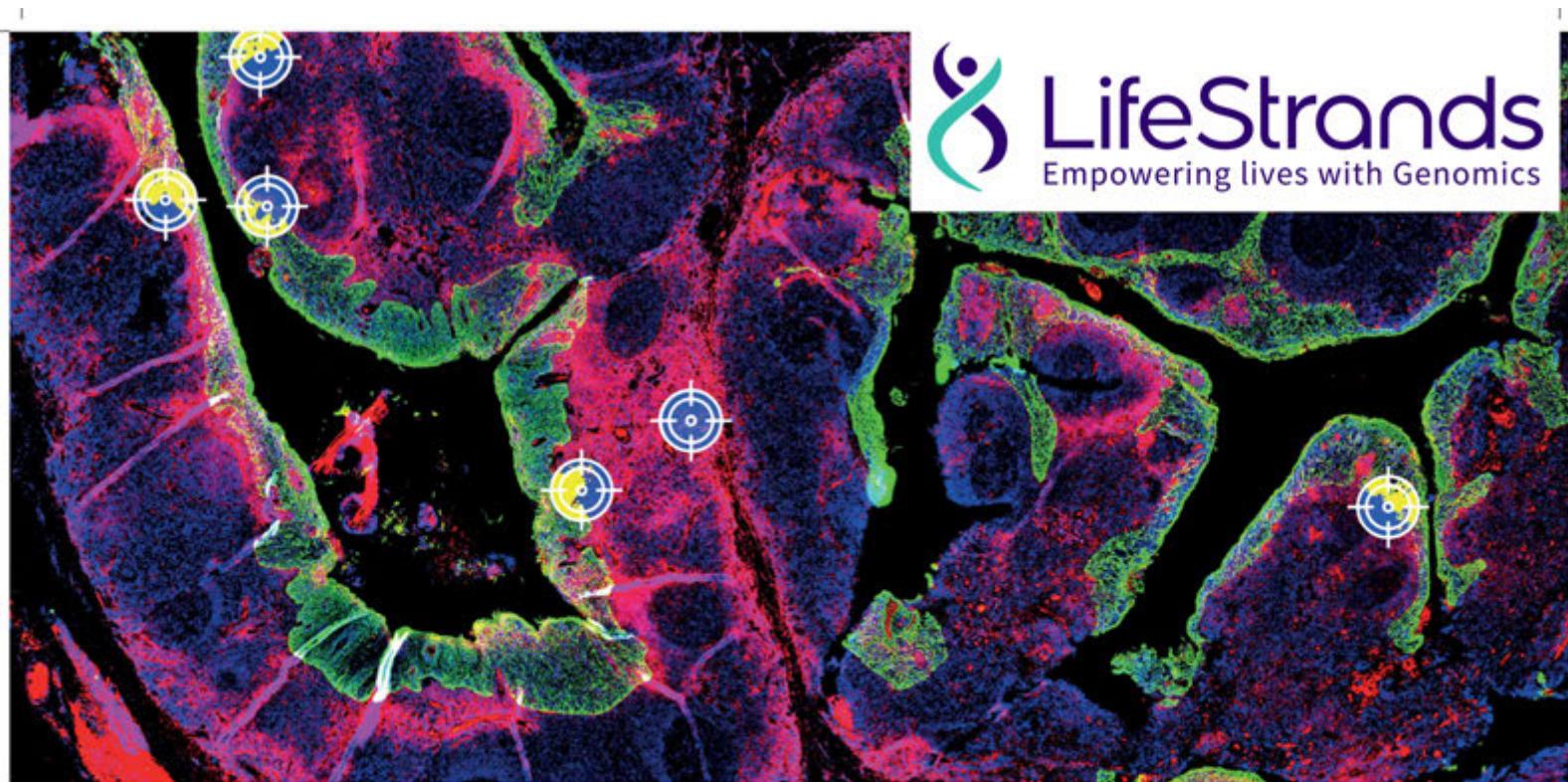
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From tissue to insight — LifeStrands Genomics supports every step of your spatial journey, with trusted expertise across clinical, academic, and pharma projects.

### Clinical Aligned Workflow

Conducted in a CAP-accredited, ISO 15189-certified laboratory

### End to End Solutions

From tissue sectioning to sequencing to spatial bioinformatics

### Trusted by Spatial Experts

Pathologists, Oncologists, Scientists, and Academics across Asia

### Certified Service Providers

CosMx, GeoMx, Visium, MerScope Ultra, RNAScope

### Experience with over 50+ Spatial Projects

Delivered across various tissue types and sample formats

### Tailored Solutions for Any Samples

FFPE, TMA, Organoids, Xenograft, Human, Mouse, Monkey, Plants

### Need Spatial Advice? Ask Us Anything.

Not sure if GeoMx, CosMx, Visium, or Vizgen is right for your project?

Talk to us. As the only certified, platform-agnostic spatial genomics service provider in Asia, we'll guide you to the best fit for your biological question, budget, and sample type.

**TALK TO US  
ABOUT YOUR  
RESEARCH**

[spatial@lifestrandsgx.com](mailto:spatial@lifestrandsgx.com)

[spatial.lifestrandsgx.com](http://spatial.lifestrandsgx.com)





# Solutions for Spatial Biology and Single Cell Analysis

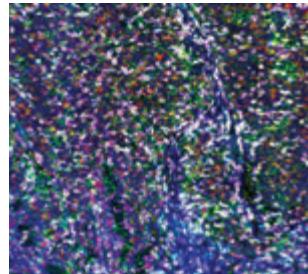
## SignalStar™ Multiplex Immunohistochemistry

SignalStar Multiplex Immunohistochemistry (mIHC) is a methodology for studying FFPE tissue samples. It uses antibodies, oligonucleotides, and fluorophores to identify which cells are present, where they are located, and what functions they perform.

With SignalStar mIHC you can amplify multiple biomarkers simultaneously in FFPE tissue with high sensitivity and specificity, and generate results on up to 8 biomarkers in just two days.

### Advantages of SignalStar mIHC:

- Flexible and easy design and redesign of panels
- Results in 2 days
- Amplify your signal –detect biomarkers with low expression levels
- Use your existing fluorescent imaging instrumentation
- Reproducible every time—our validation process begins with the IHC-P validation, followed by antibody validation for SignalStar Multiplex IHC.



SignalStar™ multiplex immunohistochemical analysis of paraffin-embedded human gastric adenocarcinoma

Scan the QR code  
for an in-depth  
overview of this  
novel technology



## InTraSeq™ Single Cell Analysis

Detect RNA alongside intracellular and cell surface proteins in one experiment at the single-cell level.

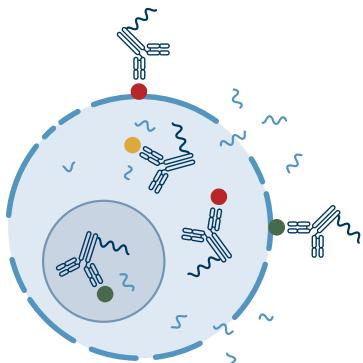
InTraSeq (Intracellular Protein and Transcriptomic Sequencing) facilitates deeper understanding of cell biology by:

- Uncovering biological insights not revealed with traditional single-cell RNA sequencing (scRNA-seq) methods
- Highlighting the dynamics between transcription and translation in single-cell datasets
- Dissecting cell signaling mechanisms by quantifying post-translational modifications at the single-cell level

InTraSeq 3' technology is developed and validated by CST, using the 10x Genomics Chromium Single Cell 3' Reagent Kits with Feature Barcoding technology.

### Advantages of InTraSeq:

- A straightforward, four-step protocol  
1 hour of benchwork with multiple stopping points before starting 10x Genomics single cell experiment
- Investigate intracellular protein signaling while guaranteeing robust RNA signal
- Identify hard-to-detect cells with unbiased depth-of-coverage
- Explore multiple molecular mechanisms in one experiment



Scan the QR code  
for an in-depth  
overview of this  
NEW technique



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IVIS® Spectrum Series



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Radiometric detection



TRI-CARB



Micro Beta



Quantulus™ GCT



Sample Oxidizer



Cell Harvesters

Cell counting and image cytometry



Celigo Image Cytometer



Cellaca MX High-throughput Cell Counter



Cellaca PLX Image Cytometry System

Microplate readers



VICTOR® Nivo



VICTOR® Kira



EnVision® Nexus

Microplates



Drug discovery reagents



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NovogeneAIT Genomics is a leading multi-omics services and solutions provider headquartered in Singapore. It is formed as a strategic joint venture between Novogene and AITbiotech, in 2016. Operating from our state-of-the-art sequencing center in Singapore, we are dedicated to advancing genomic research by offering unmatched data quality that empowers researchers across diverse sectors, including healthcare and agriculture, to realize their research goals. visit [novogene.com/amea-en](http://novogene.com/amea-en).



## FLEXIBLE SINGLE CELL SOLUTIONS



### TISSUE PRESERVATION & PROCESSING

- 72h tissue preservation
- Automated tissue dissociation/homogenization and nuclei isolation



### FLEXIBLE SINGLE CELL ANALYSIS

- Standard: Automate cell partitioning & barcoding
- Full automation: Sequencing-ready libraries
- Manual: Instrument-free



### MULTI-OMICS KITS

- (Full-length) transcriptome
- Full-length immune repertoire
- Targeted variant detection
- Time-resolved transcriptome
- Glycosylation
- Combined genome & transcriptome

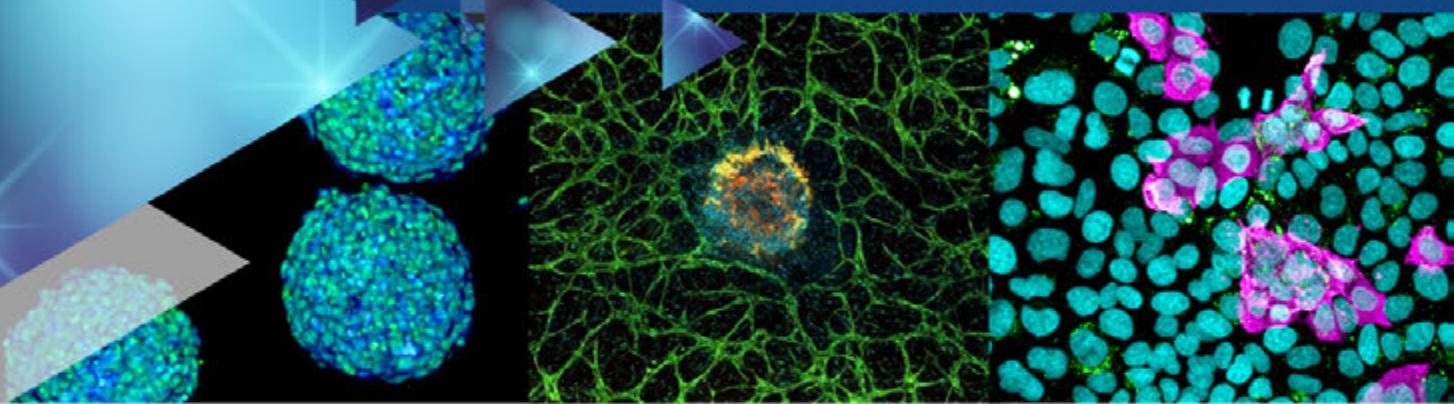


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## Living Cell Analysis Solutions

### Single-Cell Analysis Solutions

- Single Cell Targeting for Analysis
- Automatic sampling of whole cells or subcellular regions
- Maintains cell location and morphology information for repeatable extractions.
- Nano - point delivery with minimal cell damage



Model : SS2000



Model : CQ3000

### High-Content Analysis Solutions

- Single Cell Targeting for Analysis
- Automatic sampling of whole cells or subcellular regions
- Maintains cell location and morphology information for repeatable extractions.
- Nano - point delivery with minimal cell damage



Model : CV8000

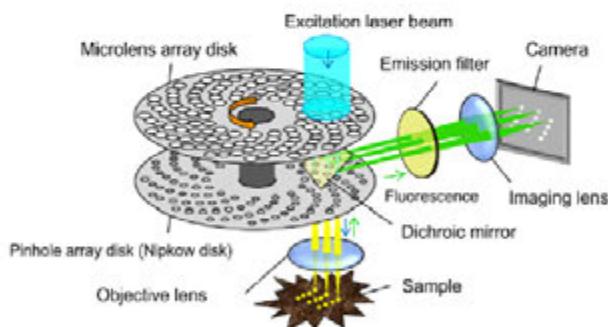
### Microlens-Enhanced Dual Spinning Disk Confocal Solutions

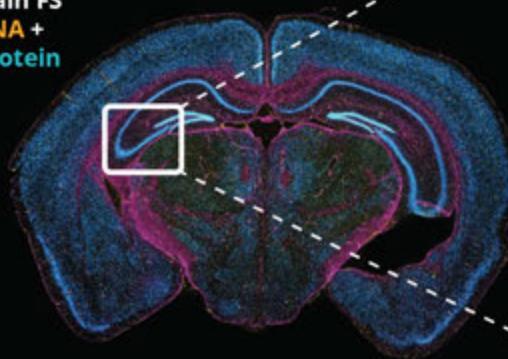
- Low phototoxicity
- Low photobleaching
- Fast, gentle, and clear imaging
- High-quality data for 3D cell analysis

*Fast & Clear Imaging*

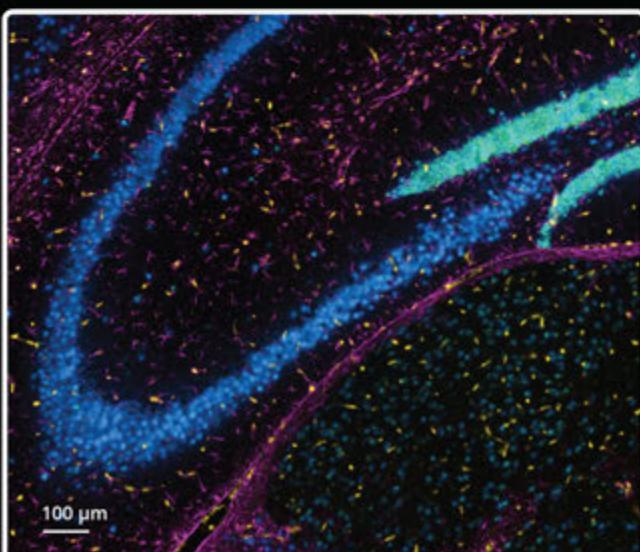
*Low Photo-Toxicity/Low Photo Bleaching*

*Live Cell Imaging of 3D cultures*




**R&D SYSTEMS**
**TOCRIS**
**NOVUS  
BIOLOGICALS**
  
**Lunaphore**
  
**ACD**
  
**Lunaphore**  
a biotechne brand
  
**ACD**  
a biotechne brand
**Mouse brain FS  
12-plex RNA +  
12-plex protein  
panel**


1 mm

*Prox1 Cldn5 + GFAP NeuN*

100 μm

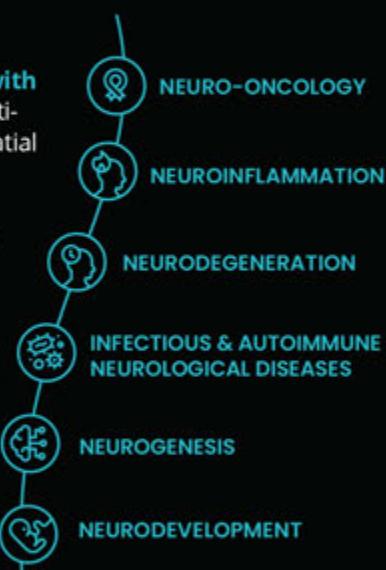
<b>RNA</b>	Slc17a7	Rbfox3	Mbp	Tubb3	Slc17a6	Prox1	Pdgfrb	Cldn5	Gfap	Sox10	Tmem119	Gad1
<b>Protein</b>	NeuN	GFAP	F4/80	OLIG2	IBA1	PDGFRB	MBP	CD14	CD31	aSMA	CD11b	CD45

## Spatial multiomics profiling of fixed frozen tissue sections on COMET™

Generate a molecular map of the mouse brain with spatial multiomics. Understanding cellular interactions in the complex brain tissue requires precise spatial analysis. COMET™ automates spatial multiomics profiling, integrating RNAscope™ HiPlex Pro and sequential immunofluorescence (seqIF™) to enable simultaneous, protease-free detection of RNA and protein in FFPE and fixed frozen tissue sections.

Spatial multiomics of fixed frozen brain tissue is now available on COMET™. For the first time, our spatial multiomics protocol has been applied to map the complexity of the mouse brain. We enable the detection of key neuronal RNA and protein markers.

Expanding possibilities in neuroscience and beyond. COMET™ enables the co-detection of RNA and protein on fixed frozen mouse brain tissue sections, opening possibilities for discovery in neuro-oncology, neuroinflammation, neurogenesis and beyond.


  
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protocol


# MICROSCOOP / MINT

**Microscoop Mint uses microscopy-guided photo-biotinylation to enable superior resolution, specificity, sensitivity, and efficiency.**

A uniquely advanced solution to prep for mass spec analysis, the platform's capabilities and streamlined workflows offers advantages over traditional methods like laser capture microdissection (LCM), antibody-based imaging workflows, and proximity labeling.



## Highlights



### Unbiased Discovery

Extract all proteomic information without antibodies or targeted panels or panels across an entire sample.



### Broad Sample Compatibility

Use with FFPE/fresh frozen tissue samples or fixed cells.



### Superior Sensitivity

Discover low copy number proteins from increased dynamic range due to subcellular protein isolation.



### Reveal

Novel proteomes of organelles, cells and organisms.



### Identify Targets Faster

Discover novel biomarkers or therapeutic targets of disease-associated locations.



### High Resolution

~350 nm precision at sub-cellular scale.

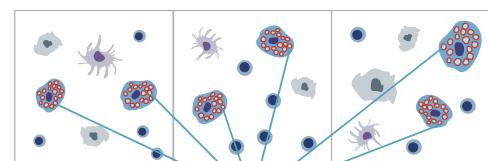


### Unmatched specificity

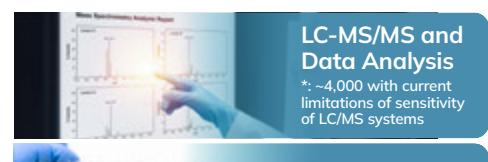
Two photon guided photo-biotinylation identifies only proteins in regions of interest.

## Spatial Protein Purification

### Regions of Interest (ROI)



### Collect All ROIs Only



### LC-MS/MS and Data Analysis

\*: ~4,000 with current limitations of sensitivity of LC/MS systems



### Proteomic Readout

i.e. ELISA, Western Blotting, FIA



### Protein Sequencing

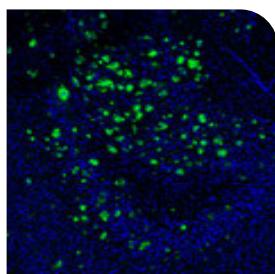
## Product Specifications

Syncell's hardware-firmware-software integrated mechatronic system enables accurate and fast control of scanners, lasers, microscope, camera, epi-illumination light source and peripheral devices.

- Microscoop system (optical engine & electrical controller)
- Inverted epifluorescence microscope
- Epifluorescence illumination light source
- Two-photon laser for Microscoop photolabeling
- Software package
- Filter sets for microscope
- Camera

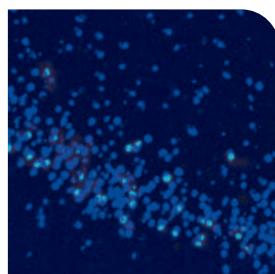
## Broad Discovery Applications

### Neurodegenerative Disease



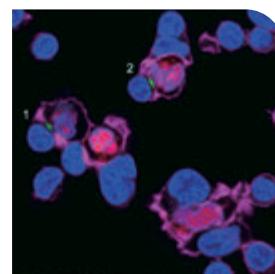
Amyloid β

### Target Discovery for Drug or Cell Therapy



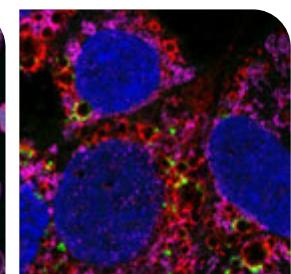
ALS Aggregates in Human Brain Section

### Immuno-oncology & Cancer Biology



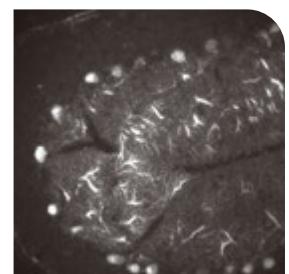
Immune Synapses

### Basic Cell Biology



Mitochondria-lipid Droplet Interface

### Tissue Atlassing



Purkinje Cells