Global Engage

SINGLE CELL CONGRESS: ASIA

11-12 JULY 2024, NTU@ONE-NORTH,SINGAPORE / HYBRID

Global Engage is pleased to announce their Single Cell Congress Asia, which will take place in person on the 11th and 12th of July 2024 at NTU@one-north, Singapore. The conference is co-hosted with the Genome Institute of Singapore (GIS). With 200 attendees and over 20 talks and a panel discussion on a unified track spanning two conference days, the event will highlight the latest advancements in the field, discover novel research findings, and delve into how the integration of single-cell technologies is facilitating the exploration of innovative therapeutic strategies. The conference will feature a dynamic exhibition room showcasing technology providers and other solutions, over 7 hours of networking, with the opportunity to connect with prominent leaders in the space.

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TIM STUART

Principal Scientist, Laboratory of Regulatory Genomics, GIS, A*STAR, Singapore

JINMIAO CHEN

Principal Investigator, GIS, A*STAR, Singapore

KEYNOTE SPEAKERS

JOSEPH POWELL

Director, Cellular Genomics Futures Institute, Garvan Institute, Australia

HONGKUI ZENG

Executive Vice President & Director, Allen Institute for Brain Science, USA

XUEGONG ZHANG

Professor, Pattern Recognition and Bioinformatics, Tsinghua University, China

CONFIRMED SPEAKERS & CHAIRS

BOXIANG LIU, Assistant Professor, National University of Singapore, Singapore

CHUNG CHAU HON, Team Leader, Division of Genomic Technology, RIKEN, Japan

CHUCK HERRING, Postdoctoral Researcher, Harry Perkins Institute of Medical Research, Australia

JONG-EUN PARK, Principal Investigator & Assistant Professor, KAIST, Korea

JOYCE KANG, MD/PhD Candidate, Harvard University, USA

JAY W SHIN *(Chair)*, Senior Principal Scientist, GIS, A*STAR; Adjunct Associate Professor, NUS, Singapore

JIAN HE, Associate Professor, Shanghai Jiao Tong University School of Medicine, China

GUANGDUN PENG, Principal Investigator and Deputy Director, Center for Cell Lineage and Development, Guangzhou Institutes of Biomedicine and Health, Chinese Academy of Sciences, China

AIBIN HE, Professor & Principal Investigator, Peking University, China

ZHI-JIE CAO, Post-doctoral Fellow, Peking University, China

TIM STUART *(Chair)*, Principal Scientist, Laboratory of Regulatory Genomics, GIS, A*STAR, Singapore

Visit our **website** for further information regarding this conference!

CONGRESS SCHEDULE

DAY 1 - 11TH JULY 2024, THURSDAY

0800 **Registration & Morning Coffee**

0845 **Global Engage Welcome Address & Chairperson's Opening Remarks**

Session Chair: Jay W. Shin, Senior Principal Scientist, GIS, A*STAR; Adjunct Associate Professor, NUS Singapore

0855 **KEYNOTE PRESENTATION**

CELL TYPE DIVERSITY AND ORGANIZATION IN THE MAMMALIAN BRAIN

To understand the function of the brain and how its dysfunction leads to brain diseases, it is essential to uncover the cell type composition of the brain, how the cell types are connected with each other and what their roles are in circuit function. At the Allen Institute, we have built multiple technology platforms, including single-cell transcriptomics, spatial transcriptomics, single and multi-patching electrophysiology, 3D reconstruction of neuronal morphology, and brain-wide connectivity mapping, to characterize the molecular, anatomical, physiological, and connectional properties of brain cell types in a systematic manner, towards the creation of multi-modal cell atlases for the mouse and human brains¬. We have now generated a comprehensive and high-resolution transcriptomic and spatial cell type atlas for the whole adult mouse brain, revealing extraordinary cellular diversity and underlying rules of brain organization. It establishes a foundational resource for deep and integrative investigations of cellular and circuit function, development, and evolution of the mammalian brain.

HONGKUI ZENG
Executive Vice President & Director, Allen Institute for Brain Science, USA

SINGLE-CELL MULTI OMICS SESSIONS

0940 **A SINGLE-CELL ATLAS OF TRANSCRIBED CIS-REGULATORY ELEMENTS IN THE HUMAN GENOME**

Transcribed cis-regulatory elements (tCREs), such as promoters and enhancers, are fundamental to modulate gene expression and define cell identity. The detailed mapping of tCREs at single-cell resolution is essential for understanding the regulatory mechanisms that govern cellular functions. Prior tCRE catalogs, limited by bulk analysis, have often overlooked cellular heterogeneity. We have constructed a tCRE atlas using single-cell 5'-RNA-seq, capturing over 340,000 single-cells from 23 human tissues and annotating more than 175,000 tCREs, substantially enhancing the scope and granularity of existing cis-regulatory element annotations in the human genome. This atlas unveils patterns of gene regulation, revealing connections between broadly expressed promoters and cell type-specific distal tCREs. Assessing trait heritability at single-cell resolution with a novel tCRE module-based approach, we uncovered the nuanced trait-gene regulatory relationships across a continuum of cell populations, offering insights beyond traditional gene-level and bulk-sample analyses. Our study bridges the gap between gene regulation and trait heritability, underscoring the potential of single-cell analysis to elucidate the genetic foundations of complex traits. These insights set the stage for future research to investigate the impact of genetic variations on diseases at the individual level, advancing the understanding of cellular and molecular basis of trait heritability.

CHUNG CHAU HON
Team Leader, Division of Genomic Technology, RIKEN, Japan

SEEKGENE

1005 **A SPONTANEOUS INFECTION MODEL OF CHRONIC GRANULOMATOUS DISEASE (CGD) DECODED BY SINGLE-CELL TRANSCRIPTIONAL RNA-SEQUENCING WITH SPATIAL INFORMATION (SEEKSPACETM)**

Chronic granulomatous disease (CGD) is a rare and hereditary immunodeficiency disorder attributed to a malfunction in the NADPH oxidase 2 (NOX2) of phagocytes. Owing to the deficiency of NOX2-derived reactive oxygen species, individuals with CGD exhibit increased susceptibility to pathogens, including bacteria and fungi. This defect also contributes to an excessive inflammatory response, leading to tissue damage. In this study, we established a CGD infection model in the Ncf2-/- genetic background through controlled environmental exposure. Within Specific Pathogen-Free (SPF) facilities, Ncf2-/- mice showed no signs of infection. However, under Clean grade conditions, these mice spontaneously developed fibro-encapsulated necrotizing lung granulomas. Neutrophils and monocyte-derived macrophages were significantly increased in the lung tissues of CGD mice, developing a NOS2high neutrophil subset with inflammatory transcriptional characteristics and a unique monocyte-derived macrophage subset with both M1 and M2 polarization.

Moreover, the distinct macrophage subset was predominantly localized to the peripheral zone of the granuloma in CGD mice, actively remodeling the extracellular matrix by expressing Fn1 and Mmp12. Treatment of CGD mice with MIF inhibitor 4IPP can effectively block MIF activity, inhibit NLRP3 expression and IL-1βproduction, thereby reducing neutrophil infiltration in lung tissue and correcting myeloid skewed hematopoiesis. Besides , by knocking out the myeloid-specific pro-survival gene Morrbid, the apoptosis of myeloid cells in CGD is enhanced, leading to an effective reduction in the progression of systemic inflammation. In summary, through controlling the environmental factors, we not only constructed a model of CGD infection, but also revealed the key drivers of CGD hyperinflammation.

ZHIGANG CAI
Professor, Tianjin Medical University, National Laboratory of Experimental Hematology, Tianjin General Hospital, China

1035 **MORNING REFRESHMENTS I 1-2-1 PARTNERING MEETINGS I POSTER PRESENTATIONS**

SINGLE-CELL MULTI OMICS SESSIONS

Session Chair: Jay W. Shin, Senior Principal Scientist, GIS, A*STAR; Adjunct Associate Professor, NUS, Singapore

1135 **USING SPATIAL OMICS TECHNOLOGY TO UNDERSTAND EMBRYO DEVELOPMENT**
Spatial transcriptomics technology has emerged as a powerful tool to study cell-cell interactions and tissue architecture by preserving the positional information of cells. This technology offers a unique perspective for dynamic observation of biological processes from both in situ and three-dimensional angles. We employed spatial transcriptomics to investigate the molecular mechanisms underlying tissue development, regeneration, and disease progression. We developed innovative spatial transcriptomics techniques and data integration algorithms to construct spatial reference coordinate systems, enabling the reconstruction of spatial location at single-cell resolution. We demonstrated the potential of spatial transcriptomics in guiding the development and differentiation of pluripotent stem cells both in vivo and in vitro.

GUANGDUN PENG
Principal Investigator and Deputy Director, Center for Cell Lineage and Development, Guangzhou Institutes of Biomedicine and Health, Chinese Academy of Sciences, China

1200 **EFFECTS OF SCALING FACTORS ON SEQUENCING DEPTH BIAS IN SCRNA-SEQ**
Sequencing depth variations in single-cell RNA sequencing (scRNA-seq) data pose a recognized challenge in downstream analyses. Traditionally, normalization based on unique molecular identifier (UMI) counts per cell, followed by scaling to a consistent depth using factors like '10k' or counts per million (CPM), is employed. Our study emphasizes the pivotal role of the scaling factor in mitigating sequencing depth discrepancies, revealing increased resolution but heightened depth bias with higher scaling factors, particularly between 10k and CPM. Notably, at CPM and beyond, scRNA-seq data tends to become binary. We demonstrate that simpler normalization methods, coupled with an appropriate scaling factor, outperform the advanced methods, effectively reducing sequencing depth bias impact. Our findings underscore the efficacy of downsampling and proportion fitting in controlling sequencing depth differences. These techniques serve as efficient batch integrators on uniformly sequenced data, outperforming established methods in preserving biological signals, particularly in disease and development contexts.

CHUCK HERRING
Postdoctoral Researcher, Harry Perkins Institute of Medical Research, Australia

1225 **EARLY CAREER RESEARCHER PRESENTATION**

CONTACT KHAMINI@GLOBAL-ENGAGE.COM TO SUBMIT ABSTRACT

1240 **SOLUTION PROVIDER PRESENTATION**

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1310 **LUNCH I 1-2-1 PARTNERING MEETINGS I POSTER PRESENTATIONS**

EMERGING SINGLE-CELL ANALYSIS TOOLS AND TECHNOLOGIES SESSIONS

Session Chair:

DAY 1 - 11TH JULY 2024, THURSDAY

1415 **KEYNOTE PRESENTATION**

AI FOUNDATION MODELS TO LEARN THE MOLECULAR LANGUAGE OF CELLS

AI foundation models such as Large Language Models (LLMs) have achieved unprecedented success in a broad range of tasks in natural language processing, computer vision and AIGC. These successes have shown the great potential of large deep-learning generative models in grasping and discovering comprehensive relationships among elements of complex systems from massive data. Cells are complex molecular systems composed of complicated sets of genomic, epigenomic, transcriptomic and proteomic elements. Their highly orchestrated activities make the cells functioning in accordance with their types, states and their temporal, spatial and functional relations/interactions with other cells. High-throughput single-cell technologies such as single-cell RNA-seq and single-cell ATAC-seq are producing more and more data for cells of different types and states. Our experiments showed that it is possible to design AI foundation models to reveal the complicated relationships of gene expressions and other elements in different types of cells. Such foundation models can be applied to a large variety of downstream tasks such as cell type annotation, gene regulation analysis, drug response prediction, perturbation, etc. and can outperform methods that were developed specifically for those tasks. The underlying relationships learned inside the foundation models can also provide hints for new discoveries that would be otherwise hard to be probed. I'll talk about our latest progress in single-cell foundation models scFoundation and scMulan and their applications and discuss the possible roadmap of building AI models to understand and represent the complex molecular language of cells.

XUEGONG ZHANG
Professor, Pattern Recognition and Bioinformatics; ISCB Fellow, Tsinghua University, China

1500 **EARLY CAREER RESEARCHER PRESENTATION**

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1515 **SOLUTION PROVIDER PRESENTATION**
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1545 **AFTERNOON REFRESHMENTS I 1-2-1 PARTNERING MEETINGS I POSTER PRESENTATIONS**

1645 **FINI-SEQ: QUASI-SPATIAL SINGLE-CELL TRANSCRIPTOME BASED ON PHYSICAL PROPERTIES DEFINES EARLY AGING ASSOCIATED NICHE IN LIVER TISSUE**

Aging is characterized as a condition of gradually losing the ability to maintain homeostasis, resulting in disruptions to physiological well-being and increased susceptibility to chronic diseases and mortality. Previous research has utilized single-cell RNA-sequencing (scRNA-seq) technology to study age-related changes at a cellular level, and identify gene expression alterations associated with aging. However, due to the limitations of scRNA-seq in capturing subtle variations within tissues, the precise age-dependent changes have not been clearly defined. This study introduces a novel method called fibrotic niche enrichment sequencing (FiNi-seq), which enables the profiling of cell types and molecular signatures that were previously overlooked by scRNA-seq. Using our newly developed FiNi-seq method, we conducted a study on the livers of young and old mice. Our findings unveiled heightened fibrotic characteristics associated with the aging process. We were able to observe microscopic fibrotic niches within the tissue at single-cell level, and find novel cell types that inhabit age-related fibrotic processes. Integrating transcriptomic analysis with spatial transcriptomics data, we identified that these niches are spatially clustered and are prevalent in the portal zone of the liver. Through tissue staining, we were able to validate the existence and localization of cell types that drive age-related fibrotic changes.

JONG-EUN PARK
Principal Investigator and Assistant Professor, KAIST, Japan

1710 **TOWARDS SYSTEMATIC CAUSAL INFERENCE OF GENE EXPRESSION REGULATION**

Systematically elucidating the structure of gene regulation is the foundation for identifying key functional regulators and designing therapeutic interventions thereof. However, accurate mapping of the regulatory network is often hampered by the heterogeneity of genomics evidence and spurious data correlations. To address these challenges, we proposed a graph-linking strategy that utilizes prior regulatory knowledge to bridge the gap between heterogenous genomics modalities, enabling accurate harmonization of single-cell multi-omics data and integrative regulatory inference. Building on mechanistic regulatory evidence gleaned from such integration, we further incorporate perturbation effects from single-cell CRISPR screens and devise an end-to-end differentiable causality-aware regulatory model that is capable of distinguishing genuine regulatory interactions from spurious correlations. Our model exhibits superior performance both in terms of recovering known causal structures and predicting counterfactual effect of unseen perturbations, illustrating the potential of rationally designed computational models for enhancing causal understanding of intricate regulatory systems and informed intervention design.

ZHI-HIE CAO
Postdoctoral Fellow, Biomedical Pioneering Innovation Center, Peking University, China

1735 **DISCOVERING DYNAMIC HLA EXPRESSION QUANTITATIVE TRAIT LOCI AT SINGLE CELL RESOLUTION**

The human leukocyte antigen (HLA) genes mediate the adaptive immune response and play a critical role in autoimmune and infectious diseases, transplantation, and cancer. While coding variation in HLA genes has been extensively documented, regulatory genetic variation modulating HLA expression levels has not been comprehensively investigated. Here, we mapped expression quantitative trait loci (eQTLs) for classical HLA genes (HLA-A, B, C, DPA1/B1, DQA1/B1, DRB1) across 1,073 individuals and 1,131,414 single cells from three tissues. To correct biases in sequencing read alignment due to polymorphic HLA genes, we developed scHLApers, a pipeline to accurately quantify single-cell HLA expression using personalized reference genomes. We identified cell-type-specific cis-eQTLs for every classical HLA gene. Modeling eQTLs at single-cell resolution revealed that many eQTL effects are dynamic across cell states even within a cell type. HLA-DQ genes exhibit particularly cell-state-dependent effects within myeloid, B, and T cells. For example, a T cell HLA-DQA1 eQTL (rs3104371) is strongest in cytotoxic cells. Dynamic HLA regulation may underlie important interindividual variability in immune responses.

JOYCE KANG
MD/PhD Candidate, Harvard University, USA

1800 **END OF DAY 1 | NETWORKING DRINKS RECEPTION**

CONGRESS SCHEDULE

DAY 2 - 12TH JULY 2024, FRIDAY

0855 **Chairperson's Opening Remarks**

Session Chair: Tim Stuart, Principal Scientist, Laboratory of Genome Function, GIS, A*STAR, SINGAPORE

0900 **KEYNOTE PRESENTATION**
INTERSECTING POPULATION GENETICS, STEM CELL BIOLOGY, AND CELLULAR GENOMICS TO STUDY COMPLEX HUMAN DISEASE
Genetic variants can contribute to disease in many ways. In complex diseases, hundreds to thousands of variants independently contribute to disease risk, and an accumulation of risk alleles – often combined with specific environmental exposures –is required to develop the disease phenotype. The overwhelming evidence showing enrichment of disease-associated variants in regulatory regions suggests that regulation of gene expression is likely a dominant mediator for disease risk. Expression quantitative trait loci (eQTL) analysis links disease risk-SNPs to downstream expression effects. An essential next step is defining the cellular contexts in which disease risk-SNPs affect gene expression levels. This will help better understand the molecular and cellular mechanisms by which disease risk is conferred and inform therapeutic strategies. This talk will cover a body of work on how single-cell sequencing technology can be scaled to enable the type of population genetics studies required to address these biological questions. I will present recent research on how we have resolved how genetic variation acts at the level of individual cells in immune cell and stem cell systems and outline the next steps in translating these findings into clinical impact.

JOSEPH POWELL
Director, Cellular Genomics Futures Institute, UNSW / Garvan Institute, Australia

0945 **SINGLE-CELL AND CELL-FREE MULTIOMICS FOR TRACING CELL FATE'S HISTORY**

AIBIN HE
Professor and Principal Investigator, Peking University, China

1010 **SOLUTION PROVIDER PRESENTATION**

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1040 **MORNING REFRESHMENTS I 1-2-1 PARTNERING MEETINGS I POSTER PRESENTATIONS**

1140 **45-MINUTE PANEL DISCUSSION**
EXPLORING FRONTIERS: SINGLE CELL TECHNOLOGIES IN DISEASE RESEARCH
This session aims to discuss the following key areas:
- Latest breakthroughs and innovations in single-cell technologies
- Challenges and opportunities in adopting cutting-edge single-cell analysis tools
- Ethical considerations associated with single-cell analysis, especially in human studies
- Case studies and future directions

1225 **EARLY CAREER RESEARCHER PRESENTATION**

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1310 **LUNCH I 1-2-1 PARTNERING MEETINGS I POSTER PRESENTATIONS**

DAY 2 - 12TH JULY 2024, FRIDAY

SINGLE CELLS IN TRANSLATIONAL RESEARCH SESSIONS

Session Chair: Tim Stuart, Principal Scientist, Laboratory of Genome Function, GIS, A*STAR

1415 **A FACILE STRATEGY OF SINGLE-CELL EXOME SEQUENCING**

Genomics sequencing has become a common approach to study the molecular mechanism and genetic characteristics of diseases. With the continuous progress of technology, single-cell genomics sequencing has become the premium approach to improve the resolution of research. However, single-cell whole genome sequencing generally have the disadvantages of large redundancy of data, low sequencing depth and coverage, difficult data interpretation, and high error rate at present stage, moreover, it is difficult to discriminate true biological variants from technical artifacts, which is not conducive for deep mining of low-abundance variants, and the high cost limits its application in large-scale, high-throughput single-cell studies. Furthermore, though composing a very small fraction (1.1%) of the genome, mutations in the exome are thought to harbor 85% of mutations that have a large effect on disease. Thus, vigorous development of single-cell exome sequencing with higher resolution, higher detection rate, and cost-effectively will substantially improve research efficacy. This strategy demonstrates an ingenious procedure of isolating single cells from tumor tissue and performing single-cell whole exome sequencing. The integration of the single-cell handling and single-cell exome sequencing procedure enabled the improvement of coverage and low-abundance variants detection, also cost-effective, time-saving, and easy to handle. Before that, the team hold several related patents, which mainly focus on single-cell (nuclei) sequencing and single-cell multi-omics analysis. In this study, He et al. demonstrated a strategy for deciphering the tumor microenvironment of hepatoblastoma using single-cell exome sequencing. An innovative single-cell whole-exome sequencing protocol was developed by integrating single-cell handling with whole-exome sequencing. This work detailed described a series of steps involved in single-cell sorting, whole-genome amplification, amplification uniformity assessment, exome capture, whole-exome library construction, sequencing, and analysis. The uniformity assessment of amplification with genomic multi-loci qPCR could exclude samples with a potential risk of amplification bias before exome capture to guarantee amplification homogeneity and genome coverage. Compared with single-cell whole-genome sequencing, this protocol largely ameliorates the detection rate of low-frequency mutations, enhances the resolution of studies and contributes to genetic heterogeneity study in tumors. In summary, this paper systematically detailed account of the procedure of single-cell whole exome sequencing using hepatoblastoma samples, and evaluated the effectiveness of this solution through library quality control and sequencing analysis, established the stability and importance of the protocol in detecting low-frequency mutations and revealing the genetic heterogeneity of human diseases. It provides effective reference and guidance for the evolution and ecology of tumor, clinical diagnosis and etc. At the same time, the precautions and trouble-shooting to common troubles during the experimental process and detailed reagents and consumables lists provides in this work enhances the reproducibility, transparency, availability, and repeatability of this protocol.

JIAN HE
Associate Professor, Shanghai Jiao Tong University School of Medicine, China

1440 **TOWARDS A CELL-TYPE-SPECIFIC UNDERSTANDING OF COMPLEX DISEASES**

High-throughput genotyping and sequencing have led to the discovery of thousands of disease-associated variants. Because most of these variants lie in non-coding regions, their functional mechanisms remain unclear. To identify genetic effects underlying complex diseases, it has become increasingly important to investigate the proper cell types and contexts. We demonstrate the power of cell-type-specific assays for three complex diseases. Coronary artery disease (CAD) is the leading cause of death globally. Approximately 40 - 60% of CAD severity can be attributed to genetic factors. GWAS meta-analyses have uncovered more than 100 significant loci, but most are difficult to interpret because they reside in non-coding regions. We found that coronary artery smooth muscle-specific genetic regulatory mechanisms are highly enriched in CAD GWAS signals. By jointly analyzing eQTL and GWAS datasets, we identified five risk genes. TCF21 and SMAD3 were subsequently validated by single-cell analysis in atherosclerotic mouse models. Age-related macular degeneration is one of the leading causes of blindness in elderlies. It has been estimated that genetic factors explain 45% - 70% of the variation in the severity of age-related macular degeneration. Retinal pigment epithelium (RPE) is vital in ocular development but is underrepresented in genetic regulation studies. By jointly analyzing RPE eQTL and AMD GWAS, we identified several risk genes including RDH5. In particular, we found that the eQTL regulatory SNP also regulates splicing. Experimental validation confirms that the minor allele leads to aberrant splicing and subsequently RNA non-sense-mediated decay. This result revealed the genetic mechanism of RDH5 regulation and confirmed RDH5 as a risk gene for age-related macular degeneration, making it a potential target for drug development.

BOXIANG LIU
Assistant Professor, National University of Singapore, Singapore

DAY 2 - 12TH JULY 2024, FRIDAY

1505 **HOST SINGLE-CELL IMMUNE PROFILE IN RESPONSE TO DENGUE VIRUS ACROSS TIME AND SEVERITY**

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. Here, we single-cell immune profile peripheral blood mononuclear cells from DENV- infected donors across time and severity outcomes. We identified critical transcriptional switched day-by-day at febrile phase right before fever subsided and a significant change in immune repertoire upon convalescence. When compared between severity outcomes, drastic differences in intrinsic cellular response were found in viral target cells. Further, distinct NK phenotypes as well as T cells and plasmablasts were observed in different severity outcomes. Overall, our study provides an in-depth global immune response to DENV infection, revealing potentially distinct response for protection and pathogenicity, which could impact in vaccine development and clinical management improvement.

PONPAN MATANGKASOMBUT
Principal Investigator, Team Leader and Associate Professor, Mahidol University, Thailand

1530 **CLOSING REMARKS | END OF CONFERENCE**

POSTER PRESENTATION

Poster presentation sessions will take place in breaks and alongside the other breakout sessions of the conference. Your presentation will be displayed in a dedicated area, with the other accepted posters from industry and academic presenters. We also issue a poster eBook to all attendees with your full abstract in and can share your poster as a PDF after the meeting if you desire (optional). Whether looking for funding, employment opportunities or simply wanting to share your work with a like-minded and focused group, these are an excellent way to join the heart of this congress. In order to present a poster at the forum you need to be registered as a delegate. Please note that there is limited space available and poster space is assigned on a first come first served basis (subject to checks and successful registration)

Submission instructions
We will require the form to be submitted by **15th June 2024**. This is the formal deadline however space is another limiting factor so early application is recommended. Therefore please contact us with any questions you have as soon as possible.