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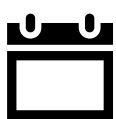
Towards a Universal Map of Human Cell- State Dynamics

Prof. Jong-Eun Park

Associate Professor, KAIST GSMSE

Chief Investigator, IBS Korea Virus Research Institute

Host: Jay Shin



Thursday 13 August 2026
10.00am – 11.00am



GIS L2 – Seminar Room
60 Biopolis Street, Genome, Singapore 138672

About The Speaker

Dr. Jong-Eun Park is a Professor at KAIST whose research bridges RNA biology, single-cell genomics, and computational modeling of complex human immune systems. He began his scientific career with biochemical and sequencing-based studies on RNA processing, elucidating molecular mechanisms of miRNA and non-coding RNA maturation. During his postdoctoral fellowship at the Wellcome Sanger Institute, he joined the Human Cell Atlas consortium and led major efforts to profile developing human immune organs at single-cell resolution, including the Human Thymus Atlas. His work reconstructed human T-cell developmental trajectories and repertoire formation, providing foundational resources for understanding human immune ontogeny. He also contributed to the development of BBKNN, a widely adopted algorithm for scalable integration of large single-cell datasets.

Since establishing his independent laboratory at KAIST, his research has centered on integrative single-cell and spatial genomics to study immune-tumor and tissue microenvironment interactions across cancer, inflammatory, and infectious diseases. His group develops large-scale data integration frameworks and multimodal atlases to model cell states, cell-cell interactions, and tissue organization in human health and disease. Recently, he has expanded this effort by joining the Korea Virus Research Institute in the Institute for Basic Science (IBS) as a Chief Investigator, where his team is advancing computational and experimental modeling of cell-cell interactions and tissue systems using single-cell and spatial omics.

About The Seminar

The growing interest in virtual cell models highlights the need for comprehensive, accurately annotated maps of human cell states across tissues, individuals, and disease contexts. Such models require not only broad cellular coverage, but also precise characterization of cell identity, state variation, and context-dependent regulatory programs.

We present a cross-tissue single-cell and spatial cell atlas, PANGEA, designed to systematically characterize human cell states across organs and disease contexts. Comprehensive metadata curation and hierarchical classification were performed to harmonize terminology across studies. Integration of the remapped transcriptomes markedly reduced batch effects and enabled robust identification of cell types, as well as organ-, disease-, and sex-associated gene signatures.

Building on the PANGEA atlas, we also developed Sceletto, a set of atlas-informed algorithms for marker discovery and batch correction. By leveraging the breadth and harmonized structure of PANGEA, Sceletto enables robust identification of cell-type and cell-state markers across heterogeneous datasets, while improving cross-study comparability through scalable batch correction.

Finally, we performed large-scale Perturb-seq experiments in fibroblast systems to causally dissect the regulatory logic underlying cell-state transitions. By overexpressing 200 transcription factor open reading frames in dermal fibroblasts and mesenchymal stem cells, we systematically mapped gene regulatory networks that govern fibroblast activation and lineage plasticity. These perturbation-based causal inferences complement the cross-tissue atlas by distinguishing universal regulatory programs from context-specific mechanisms that shape human cell-state dynamics.

Together, this work establishes an integrated framework that links population-scale cell atlas construction, atlas-informed computational methods, and experimental perturbation. By combining comprehensive cell-state mapping with causal regulatory inference, our work provides a foundation for virtual cell models and advances a predictive understanding of human cell identity, plasticity, and disease-associated state transitions.