Program

International Academy for Advanced Oncology

IAAO 2026

Pioneering the Next Frontier in Cancer Research

2026 July 31 (Fri) 13:00~19:00
August 1 (Sat) 9:00~15:00
@ Toranomon Hills Forum

C-FINDs
CHUGAI FOUNDATION FOR INNOVATIVE DRUG DISCOVERY SCIENCE

Pioneering the Next Frontier in Cancer Research

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Chair: **Kiyohiko Hatake, MD, PhD**, Professor, Akasaka Sanno Medical Center, Japan

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14:30 **Hiroyuki Mano, MD, PhD**, President, National Cancer Center, Japan

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IAAO 2027

Date : Friday July 23 – Saturday July 24, 2027
Venue : Toranomon Hills Forum, Tokyo

Opening Remarks

Motoo Ueno

President, Chugai Foundation for Innovative Drug Discovery Science (C-FINDs)

As president of the foundation, I am very pleased to host the International Academy for Advanced Oncology, IAAO. I would like to express my sincere gratitude to all of the distinguished guests, experts, and investigators attending this forum from overseas and Japan.

The IAAO is a major C-FINDs event that reflects three guiding principles: Top-level science", "Development of young researchers", and "Global perspective". We are pleased to welcome you to this forum, IAAO 2026, organized by C-Finds.

This year's theme, "Pioneering the Next Frontier in Cancer Research," reflects our strong belief in the importance of integrating basic research with translational science. From uncovering the molecular mechanisms of tumorigenesis to designing and evaluating novel therapies, we sincerely hope that this conference will foster cross-disciplinary exchange and contribute meaningfully to future breakthroughs in cancer research.

We are also deeply honored to welcome such distinguished speakers from around the world. The recent progress in oncology has been truly remarkable. However, the path toward conquering cancer depends not on a single discovery, but on the collective and synergistic accumulation of diverse research efforts. Each participant in this conference holds a vital piece of this shared knowledge. We would therefore like to express our heartfelt gratitude to all of you for joining us today and for generously sharing your expertise.

I wish to express our profound gratitude to the members of the advisory board for their expert contributions in shaping the program: Dr. Chabner, Dr. Tabernero, Dr. Mano, Dr. Hatake, Dr. Ishioka, Dr. Kitagawa, Dr. Nishikawa, Dr. Ohtani, and Dr. Toi. Their unwavering dedication and expert input were indispensable. Thanks to their efforts, we are able to present a comprehensive agenda that captures both depth and breadth across the field.

In closing, I would like to thank all the participants again. C-FINDs' sincere wish is that the IAAO forum becomes an important venue for the exchange of information that advances the fight against cancer and, concurrently, empowers patients to deal with their treatment proactively and with hope. I do hope that this two-day event will be a highly informative and fruitful time for everyone.

Session 1

Frontline of Cancer Immunotherapy: Cell Therapy and Vaccines

1-1. TILs for solid tumors – The Next Frontier

Speaker: Elena Garralda, MD, PhD
(Director, UITM; Codirector, VHIO Clinical Research, Spain)

1-2. Immune-Modulatory Vaccines: Reprogramming the Immunosuppressive Tumor Microenvironment

Speaker: Mads Hald Andersen, PhD, DSc, DMSc
(Professor and Director, CCIT-DK, Herlev Hospital, Denmark)

1-3. Redefining Intra-Tumor Heterogeneity through the Lens of the Immunopeptidome

Speaker: Yardena Samuels, PhD
(Professor, Weizmann Institute, Israel)

TILs for Solid Tumors – The Next Frontier

Speaker

Elena Garralda, MD, PhD

Director, Research Unit for Molecular Cancer Therapy (UITM) – "*CaixaResearch"*
Codirector, Clinical Research Program
Vall d'Hebron Institute of Oncology (VHIO),
Barcelona, Spain

Chair

Josep Tabernero, MD, PhD

Director, Vall d'Hebron Institute of Oncology (VHIO), Spain
Head of the Medical Oncology Department at the Vall d'Hebron University Hospital, Spain

Elena Garralda, MD, PhD

Research Summary

Dr. Elena Garralda is a distinguished translational oncologist specializing in early-phase drug development and precision medicine. Her research focuses on pioneering next-generation cancer therapies, including novel bispecific antibodies, T-cell engagers, and adoptive cell therapies such as tumor-infiltrating lymphocytes (TILs). Utilizing advanced genomic platforms, she identifies critical biomarkers to predict patient response to immunotherapy. By bridging molecular discovery and clinical application, her work facilitates personalized, biomarker-driven treatment strategies. Ultimately, her research strives to optimize therapeutic efficacy and improve survival outcomes for patients with refractory and advanced-stage malignancies.

Professional Experience/Awards

Dr. Elena Garralda is the Director of the Research Unit for Molecular Cancer Therapy (UITM) – "*CaixaResearch"* and the Codirector of the Clinical Research Program at the Vall d'Hebron Institute of Oncology (VHIO) in Barcelona, Spain. In this capacity, she oversees one of Europe's premier centers for Phase I and complex early-phase clinical trials, focusing on targeted therapies and innovative immunotherapies.

Beyond her institutional leadership, Dr. Garralda holds several influential positions within the international oncology community. She chairs the European Organization for Research and Treatment of Cancer (EORTC) RECIST Task Force, where she spearheads the development of global standards for assessing tumor response. Reflecting her standing in the field, she is the Chair for the 38th EORTC–NCI–AACR (ENA 2026) Symposium, a flagship forum for molecular targets and cancer therapeutics.
Dr Garralda is PI and Project Coordinator of several EU-funded projects (e.g., CCE-DART, PCM4EU, CanSERV, PragmaTIL) focused on precision oncology and biomarker-driven clinical trials delivering personalized cancer care across Europe and, in 2025, the PragmaTIL Trial, was awarded with the Excellence category of the PRECISEU Best Practice Recognition.
A committed educator, she is faculty coordinator for European Society for Medical Oncology (ESMO) programs, specializing in clinical trial methodology and early drug development, committee member of the *"AACR Trailblazer Research Grants"* and the *"Clinical and Translational Cancer Research Grants",* member of the Scientific Program Committee for ASCO 2027 and faculty of the *"EORTC-ESMO-AACR Methods in Clinical Cancer Research (MCCR)"* annual workshop since 2021. She is also a regular Chair and Speaker at ASCO, AACR or ENA congresses. Dr. Garralda has authored over 150 peer-reviewed publications in high-impact journals, such as The New England Journal of Medicine, Nature Medicine, and the Journal of Clinical Oncology. She was Section Editor for EJC until this year and is currently Associate Editor for ESMO Open.
Her career is distinguished by extensive scientific citations, numerous invited international lectures, and a pivotal role in transitioning novel oncology agents from the laboratory to the clinic.

Education

PhD in Oncology (Cum Laude) - Universidad Autónoma de Barcelona, Spain
Master's Degree in Molecular Oncology - Spanish National Cancer Research Centre (CNIO), Madrid
Medical Oncology – Hospital Universitario 12 Octubre de Madrid, Spain
Medical Degree (MD) - Universidad Autónoma de Madrid, Spain

Abstract of the lecture

Adoptive cell therapy with ex vivo expanded tumor-infiltrating lymphocytes has demonstrated the potential to induce durable tumor regression in patients with metastatic melanoma. Increasing evidence indicates that TIL products frequently contain T cells recognizing tumor-specific neoantigens, and that neoantigen-reactive TILs are important mediators of antitumor activity in both immune checkpoint blockade and TIL-based cellular therapy. Because neoantigens are selectively expressed by tumor cells and are not subject to central thymic tolerance, they represent highly attractive targets for personalized cancer immunotherapy.
Despite the established activity of TIL therapy in melanoma, extending this benefit to epithelial and immune checkpoint blockade-resistant solid tumors remains a major challenge. In this context, our work on NEXTGEN-TIL supports the feasibility of generating TIL products enriched for neo-antigen recognition. In preclinical studies, TILs were successfully expanded from core tumor biopsies across multiple solid tumor types, with tumor- and neoantigen-reactive T cells detected in patient-derived cultures. These findings provided the rationale for a phase I clinical trial evaluating a neoantigen-selected TIL product in patients with advanced epithelial tumors and immune checkpoint blockade-resistant solid cancers.
This presentation will discuss strategies to improve and broaden the clinical impact of TIL therapy, including neoantigen-based TIL selection, manufacturing optimization from tumor biopsies, and improved treatment feasibility. It will also highlight European initiatives such

as PragmaTIL, which explores pragmatic academically driven TIL therapy approaches, including the administration of TILs together with IL-2 analogs, with the aim of enhancing efficacy, reducing toxicity, and expanding access to personalized T-cell therapies for patients with solid tumors

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Immune-Modulatory Vaccines: Reprogramming the Immunosuppressive Tumor Microenvironment

Speaker

Mads Hald Andersen, PhD, DSc, DMSc

Professor and Director, National Center for Cancer Immune Therapy (CCIT-DK)
Copenhagen University Hospital Herlev, Denmark

Chair

Josep Tabernero, MD, PhD

Director, Vall d'Hebron Institute of Oncology (VHIO), Spain
Head of the Medical Oncology Department at the Vall d'Hebron University Hospital, Spain

Mads Hald Andersen, PhD, DSc, DMSc

Research Summary

Dr. Mads Hald Andersen is a world-renowned expert in cancer immunology and immunotherapy, celebrated for his pioneering work in immunomodulatory vaccination. With over 25 years of experience, he has redefined therapeutic cancer vaccines by moving beyond conventional tumor-antigen targeting to focus on "prohibiting the inhibitors." His research centers on identifying T-cell responses against immune-suppressive proteins, such as IDO, PD-L1, and arginase. By targeting these molecules, his innovative strategies reshape the tumor microenvironment to foster robust anti-tumor immunity. This approach has successfully translated into numerous clinical trials, evaluating novel vaccines in combination with standard-of-care therapies for multiple solid tumors.

Professional Experience/Awards

Dr. Mads Hald Andersen is Clinical Professor at the Department of Immunology and Microbiology, University of Copenhagen, and Director of the National Center for Cancer Immune Therapy (CCIT-DK) at Herlev and Gentofte Hospital, Denmark. He also serves as Coordinating Professor at the Technical University Hospital of Greater Copenhagen. In these roles, he leads a translational environment spanning antigen discovery, preclinical cancer immunology, immunomonitoring, and early clinical development, with a focus on T-

cell-mediated immune regulation and therapeutic targeting of suppressive mechanisms in the tumour microenvironment.
During his career, Dr. Andersen has published more than 250 international peer-reviewed journal articles. He is also an inventor with 25 patents or patent applications, supporting translation of immune-modulatory therapeutic concepts. In parallel, he has contributed to biotechnology translation, including co-founding SurVac and IO Biotech.
Beyond his institutional responsibilities, Dr. Andersen has played a central role in building translational cancer immunotherapy infrastructure in Denmark, including co-founding the Tumor Immunology Group at the Danish Cancer Society in 2001 and the Center for Cancer Immune Therapy at Herlev Hospital in 2006, which later evolved into CCIT-DK. He currently serves as Deputy Chairman of the Danish Cancer Society's Scientific Committee and acts as adviser and reviewer for several international research foundations, alongside extensive national and international collaborations across academia, hospitals, and biotechnology partners

Education

Doctor of Medical Science	University of Copenhagen, Denmark
Doctor of Science	Technical University of Denmark, Denmark
PhD in Medicine/Oncology	Technical University of Denmark, Denmark
Master of Science	Technical University of Denmark, Denmark

Abstract of the lecture

Immune regulation in cancer comprises both inhibitory and counter-regulatory forces. Pro-inflammatory autoreactive T-cells that recognize immunoregulatory pathways represent an effector component that can be therapeutically expanded. The lecture provides a brief historical overview of the discovery of these T-cells (anti-regulatory T-cells; anti-Tregs) that recognize regulatory immune-cells and central immunoregulatory pathways, and how these observations motivated immune-modulatory vaccines (IMVs) as a therapeutically approach. IMVs differ from conventional cancer-vaccine strategies directed against tumor-associated or tumor-specific antigens on malignant cells by targeting tumor-microenvironment antigens (TMAs), defined as antigens expressed by immune-suppressive regulatory cells within the tumor microenvironment. TMAs are expressed across malignant, myeloid, regulatory, endothelial, and stromal compartments and include clinically advanced targets such as indoleamine-2,3-dioxygenase (IDO) and PD-L1, as well as next-generation targets such as arginase-1 (ARG1) and TGF-β.
Current clinical experience with IDO and PD-L1-directed IMVs is reviewed, including early-phase studies reporting robust immunogenicity, favorable tolerability, and signals of activity, particularly in combination with immune-checkpoint blockade. Recent phase-III data in first-line advanced melanoma combining an IDO/PD-L1 vaccine with anti-PD-1 therapy are discussed with emphasis on hypothesis-generation for patient selection, including observations of strong signals in PD-1-naïve disease and in PD-L1-negative tumors.
Mechanistic insights are summarized to explain how IMVs may reprogram the immunosuppressive tumor microenvironment through coordinated $CD8^+$ T-cell-mediated cytotoxicity against TMA-expressing suppressive cells and $CD4^+$ T-cell cytokine-driven modulation of myeloid and stromal states. These effects may support improved antigen presentation, increased immune infiltration, and amplification of endogenous tumor-specific immunity.
Finally, next-generation IMVs targeting ARG1 and TGF-β are presented as strategies to address immune-exclusion, metabolic suppression, and desmoplastic stroma, key barriers in cold tumors. The lecture concludes by positioning IMVs as rational combination partners that can boost and broaden adaptive immunity, thereby complementing immune-checkpoint blockade, cellular therapies, and conventional tumor-antigen vaccines.

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3. IOB-013/KN-D18 investigators, Hassel JC, et al. IO102-IO103 immune-modulatory cancer vaccine and pembrolizumab in melanoma. *Ann Oncol* 2026; 37: 999-1010.

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Redefining Intra-Tumor Heterogeneity Through the Lens of the Immunopeptidome

Speaker

Yardena Samuels, PhD

Professor, Department of Molecular Cell Biology
The Knell Family Professorial Chair
Weizmann Institute of Science, Rehovot, Israel

Chair

Josep Tabernero, MD, PhD

Director, Vall d'Hebron Institute of Oncology (VHIO), Spain
Head of the Medical Oncology Department at the Vall d'Hebron University Hospital, Spain

Yardena Samuels, PhD

Research Summary

Professor Yardena Samuels is a distinguished cancer geneticist and pioneer in melanoma genomics and tumor immunology who, as a postdoctoral fellow in Bert Vogelstein's laboratory, was the first to uncover the high frequency of PIK3CA mutations in human cancers, a landmark discovery that fundamentally shaped targeted therapy development. Her laboratory at the NIH and later the Weizmann Institute subsequently identified numerous driver mutations in cutaneous melanoma, demonstrated that metalloproteinases can act as tumor suppressors, identified ERBB4 as the most highly mutated tyrosine kinase in melanoma, published the first melanoma whole exomes, and contributed to the TCGA comprehensive genomic classification of cutaneous melanoma. More recently, she has pioneered the field of tumor immunopeptidomics, uncovering immunogenic peptides derived from hotspot mutations, microbiome-derived antigens presented on tumor MHC molecules, and aberrant peptides generated by translational errors under metabolic stress, while establishing that silencing translation fidelity can dramatically increase tumor immunogenicity. By combining cutting-edge genomics, functional immunology, and innovative mouse models, her laboratory continues to advance our understanding of how tumor heterogeneity and mutational processes shape the anti-tumor immune response, with the ultimate goal of improving the efficacy of next-generation immunotherapies.

Professional Experience/Awards

Dr. Yardena Samuels is a Professor in the Department of Molecular Cell Biology at the Weizmann Institute of Science, where she holds the Knell Family Professorial Chair. A preeminent leader in the global oncology community, she served as the President of the European Association for Cancer Research (EACR) from 2024 until June 2026, a role that underscored her influence in shaping international research priorities.
Her professional journey includes a transformative postdoctoral fellowship at Johns Hopkins University under the mentorship of Professor Bert Vogelstein. It was during this tenure that she made the seminal discovery of PIK3CA mutations, which remains a cornerstone of precision oncology. At the Weizmann Institute, she has established a world-class laboratory dedicated to uncovering the molecular mechanisms of melanoma and the complex interactions within the tumor microenvironment.
Dr. Samuels' scientific excellence has been recognized with numerous prestigious accolades, including the Pezcoller Foundation–EACR Cancer Researcher Award and the Youdim Family Prize for Excellence in Cancer Research. Her prolific research output includes extensive publications in top-tier journals such as Nature, Science, and Cell. As a highly sought-after expert, she frequently delivers keynote addresses at major international symposia, including AACR and Keystone Symposia. Beyond her research, Dr. Samuels is a dedicated mentor, committed to training the next generation of scientists. Her integrative approach, bridging genomics, immunology, and clinical oncology, continues to drive innovation in identifying therapeutic targets and understanding the factors that dictate patient response to immunotherapy.

Education

Postdoctoral Fellowship	Johns Hopkins University, USA (Under Prof. Bert Vogelstein)
PhD in Cancer Genetics	Ludwig Institute for Cancer Research, University College London, UK
Bachelor of Science (BSc)	Cambridge University, Cambridge, UK

Abstract of the lecture

Intra-tumor heterogeneity is a fundamental feature of cancer that influences disease progression, immune evasion, and therapeutic response. This talk will explore the complex interplay between genomic, epigenetic, and antigenic diversity within tumors and how these layers of heterogeneity shape anti-tumor immunity. Particular emphasis will be placed on the tumor immunopeptidome, which provides the immune system with a dynamic and highly informative snapshot of the physiological and pathological state of cancer cells.

I will discuss how both canonical and non-canonical neopeptide repertoires contribute to intra-tumor heterogeneity and influence immune recognition. Beyond mutation-derived neoantigens, emerging sources of tumor-specific peptides, including those derived from intracellular microbial antigens, aberrant protein synthesis, and unconventional translation events, substantially expand the landscape of potential immune targets.
Importantly, the immunopeptidome is highly dynamic and responds to a broad range of intrinsic and extrinsic cues. I will present examples demonstrating how inflammation, nutrient deprivation, translation aberrations and even circadian rhythms reshape antigen presentation, thereby modulating the signals conveyed to the immune system about the disease state of tumor cells.

Together, these studies highlight the immunopeptidome as both a sensor and a mediator of tumor cell state. Understanding the mechanisms that govern its composition may enable us to redefine intra-tumor heterogeneity, transforming it from a major obstacle to effective cancer therapy into a source of novel therapeutic opportunities.

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Session 2

Frontline of Cancer Immunotherapy: T Cell Regulation

2-1. Reprogramming the Tumor Microenvironment with Lipid Targeting

Speaker: Ping-Chih Ho, PhD
(Full Member, Ludwig Institute for Cancer Research; Professor, University of Lausanne, Switzerland)

2-2. Tumor-Derived Mitochondrial Transfer Shapes the Tumor Microenvironment and Therapeutic Outcomes

Speaker: Yosuke Togashi, MD, PhD
(Professor and Chair, Okayama University, Japan)

2-3. Rethinking Immune Responses in Breast Cancer

Speaker: Sherene Loi, MD, PhD
(Professor and Medical Oncologist, Peter MacCallum Cancer Centre, Australia)

Reprogramming the TME with Lipid Targeting

Speaker

Ping-Chih Ho, PhD

Full Member, Ludwig Institute for Cancer Research (Lausanne Branch)
Professor, Department of Oncology, University of Lausanne (UNIL), Switzerland

Chair

Hiroyoshi Nishikawa, MD, PhD

Professor and Chairperson, Division of Cancer Immune Multicellular System Regulation, Center for Cancer Immunotherapy and Immunobiology (CCII), Kyoto University Graduate School of Medicine
Chief, Division of Cancer Immunology, Research Institute / Exploratory Oncology Research and Clinical Trial Center (EPOC), National Cancer Center Japan

Ping Chih Ho, PhD

Research Summary

Dr. Ping-Chih Ho is a pioneering investigator in cancer immunometabolism, specializing in how metabolic processes regulate immune cell fate within the tumor microenvironment. His research elucidates the complex metabolic crosstalk between cancer and infiltrating immune cells, demonstrating how nutrient competition and metabolic byproducts—such as lactate and lipids—drive T-cell exhaustion. By identifying these "metabolic checkpoints," Dr. Ho develops novel strategies to reprogram tumor-infiltrating lymphocytes (TILs) and CAR-T cells. His work aims to enhance the metabolic fitness and persistence of immune cells under hostile conditions, ultimately advancing innovative metabolic approaches to improve the efficacy of cancer immunotherapy.

Professional Experience/Awards

Dr. Ping-Chih Ho is a Full Member of the Ludwig Institute for Cancer Research (Lausanne Branch) and a Professor in the Department of Oncology at the University of Lausanne (UNIL), Switzerland. He is an internationally recognized authority on the energetic constraints governing anti-tumor immunity, having emerged as a central figure in European translational cancer research.
Before his tenure in Switzerland, Dr. Ho made formative scientific contributions at Yale University, where he established the foundation for his work on immune cell metabolic reprogramming. His research program is distinguished by its ability to translate fundamental mechanistic insights into therapeutic strategies, particularly in enhancing the

anti-tumor efficacy of T-cell-based therapies.
Dr. Ho's scientific excellence has been recognized through numerous prestigious honors, including the European Research Council (ERC) Starting Grant, Swiss Science National Foundation Consolidator Grant, Cancer Research Institute Llyod J. Old STAR Investigator Award, and the EMBO Young Investigator Award. He is the first Europe-based researcher receiving AAI BD Biosciences Investigator Award in the past 35 years and elected as AAAS fellow in 2025. His prolific academic record includes over 100 influential peer-reviewed publications in premier journals such as *Nature*, *Cell*, and *Nature Immunology*.
As a leader in his field, Dr. Ho is frequently invited to speak and organize sessions at major international conferences, including AACR, SITC, and EACR. He also serves on various scientific advisory boards, contributing his expertise to the broader oncology and immunology communities. Through his commitment to translational science, Dr. Ho is redefining the landscape of immunotherapy by integrating metabolic fitness as a key pillar of effective cancer treatment, driving the development of next-generation therapies for patients with refractory malignancies.

Education

Postdoctoral Fellowship Yale University School of Medicine, USA
PhD in Pharmacology / Biomedical Sciences University of Minnesota, USA (2011)
Bachelor of Science (BSc) Taipei Medical University, Taiwan

Abstract of the lecture

Tumor cells develop various strategies to evade immune surveillance, one of which is the modulation of the metabolic state of the tumor microenvironment (TME). In response to metabolic stress in the TME, several tumor-infiltrating immune subsets upregulate CD36 to take up lipids. This leads to impaired anti-tumor immunity, as intratumoral regulatory T cells (Tregs) exhibit increased survival and suppressive activity, while $CD8^+$ T cells become more susceptible to ferroptosis and exhaustion. Here, we develop a humanized anti-CD36 IgG4 antibody, PLT012, against the lipid-binding domain of CD36 with excellent safety and favorable pharmacokinetic features in mice and cynomolgus monkey. PLT012 alone or in combination with PD-L1 blockade or standard-of-care immunotherapy results in robust anti-tumor immunity in both immunotherapy-sensitive and -resistant hepatocellular carcinomas (HCCs). Notably, PLT012 also reprograms immune landscape of human HCC ex vivo. Our findings provide proof-of-concept evidence that PLT012 effectively reprograms anti-tumor immunity in HCC, positioning it as a first-in-class immunotherapy targeting CD36.

References

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2. Xu S, Chaudhary O, ... **Ho PC**, Witztum JL, Emu B, Cui G, Kaech SM. Uptake of oxidized lipids by the scavenger receptor CD36 promotes lipid peroxidation and dysfunction in CD8+ T cells in tumors. *Immunity* 2021; 54:1561-1577.e7.

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Tumor-Derived Mitochondrial Transfer Shapes the Tumor Microenvironment and Therapeutic Outcomes

Speaker

Yosuke Togashi, MD, PhD

Professor and Chair, Department of Tumor Microenvironment
Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan

Chair

Hiroyoshi Nishikawa, MD, PhD

Professor and Chairperson, Division of Cancer Immune Multicellular System Regulation, Center for Cancer Immunotherapy and Immunobiology (CCII), Kyoto University Graduate School of Medicine
Chief, Division of Cancer Immunology, Research Institute / Exploratory Oncology Research and Clinical Trial Center (EPOC), National Cancer Center Japan

Yosuke Togashi, MD, PhD

Research Summary

Dr. Yosuke Togashi is a distinguished physician-scientist specializing in tumor immunology and translational oncology. His research investigates the complex interplay between oncogenic signaling pathways and the immune microenvironment. He is internationally recognized for his pioneering work on regulatory T cells (Tregs), particularly in elucidating how cancer cell-intrinsic genomic alterations and metabolic reprogramming drive immunosuppression and resistance to PD-1/PD-L1 blockade. Utilizing state-of-the-art technologies such as single-cell RNA sequencing and spatial transcriptomics, Dr. Togashi's program bridges molecular discovery with the development of personalized immunotherapies. His work aims to translate these deep immunological insights into effective strategies for treating refractory and resistant malignancies.

Professional Experience/Awards

Dr. Yosuke Togashi is Professor and Chair of the Department of Tumor Microenvironment at Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Japan. In this leadership role, he spearheads advanced research focused on the mechanisms of immune evasion in cancer. Before joining Okayama University, Dr. Togashi held a pivotal position at the National Cancer Center East, where he led high-impact

studies integrating cancer genomics with comprehensive immunological profiling, establishing himself as a leader in translational oncology.
As a clinical oncologist by training, Dr. Togashi possesses a unique perspective that allows him to bridge the gap between laboratory benchwork and patient care. His research program has made significant contributions to understanding why certain patients fail to respond to standard checkpoint inhibitors. His prolific academic output includes numerous publications in top-tier journals such as Nature, Nature Immunology, Immunity, and Clinical Cancer Research.
Dr. Togashi's scientific excellence has been recognized through several prestigious honors, most notably the Japan Cancer Association (JCA) Young Investigator Award, which highlights his impact on the next generation of cancer research. He is an active and influential member of the international scientific community, contributing to the American Association for Cancer Research (AACR), the Society for Immunotherapy of Cancer (SITC), and the European Society for Medical Oncology (ESMO). As a frequent invited speaker and program committee member for these organizations, Dr. Togashi continues to shape global discourse on immune evasion and the development of next-generation personalized cancer immunotherapies.

Education

PhD in Medical Science	Kindai University Graduate School of Medicine, Japan (2015)
Medical Degree (MD)	Kyoto University Faculty of Medicine, Japan (2006)

Abstract of the lecture

Immune checkpoint inhibitors (ICIs) undergo a major challenge from T-cell exhaustion within the tumor microenvironment (TME). Given that metabolic and mitochondrial dysfunction drive this exhausted phenotype, we investigated the underlying role of mitochondrial genetics in this process. Mitochondrial DNA (mtDNA) sequencing of tumor-infiltrating lymphocytes (TILs) revealed somatic mutations in approximately 40% of cases, many of which were identical to those in paired tumor cells, suggesting inter-cellular organelle transfer. *In vitro* assays confirmed that tumor-derived mitochondria are transferred to TILs via extracellular vesicles and tunneling nanotubes, establishing a high degree of mutant heteroplasmy or almost homoplasmy that directly impairs T-cell function. In preclinical models, this mutant mitochondrial transfer compromised anti-PD-1 efficacy and tumor rejection. Concurrently, clinically, the presence of tumor mtDNA mutations correlated with shorter survival in ICI-treated patients. Interestingly, this organelle transfer extended to myeloid populations; while normal mitochondrial transfer promoted antitumor macrophage maturation, mutant mitochondria selectively induced hypoxia/VEGFA-enriched, glycolytic programs, expanding pro-angiogenic macrophage subsets. Functionally, this mutant transfer accelerated neovascularization, thereby sensitizing tumors to anti-angiogenic therapy. Our findings reveal that tumor-to-immune mitochondrial transfer dictates immune cell fate, linking inter-cellular organelle genetics to distinct therapeutic vulnerabilities.

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Rethinking Immune Responses in Breast Cancer

Speaker

Sherene Loi, MD, PhD, FRACP

Professor and Medical Oncologist
Lab Head, Translational Breast Cancer Genomics and Therapeutics Laboratory
Head, Breast Oncology Trials Unit
Peter MacCallum Cancer Centre, Melbourne, Australia

Chair

Hiroyoshi Nishikawa, MD, PhD

Professor and Chairperson, Division of Cancer Immune Multicellular System Regulation, Center for Cancer Immunotherapy and Immunobiology (CCII), Kyoto University Graduate School of Medicine
Chief, Division of Cancer Immunology, Research Institute / Exploratory Oncology Research and Clinical Trial Center (EPOC), National Cancer Center Japan

Sherene Loi, MD, PhD, FRACP, FAHMS, FAA

Research Summary

Professor Sherene Loi is a medical oncologist and translational breast cancer researcher whose work focuses on the interaction between tumour biology, host immunity, and therapeutic response. She is internationally recognised for her contributions to breast cancer immunology, including the clinical development and implementation of tumour-infiltrating lymphocytes as prognostic and predictive biomarkers, and for her leadership in early- and late-stage clinical trials that have helped integrate PD-1 checkpoint blockade into standard breast cancer treatment.
Her research program integrates clinical trials with high-dimensional genomic, transcriptomic, single-cell, spatial, and immune profiling approaches to understand why some breast cancers are naturally immune-infiltrated and responsive to immunotherapy, while others remain immune-excluded or resistant. A central aim of her work is to translate these biological insights into biomarker-driven therapeutic strategies for patients with triple-negative, HER2-positive, and high-risk estrogen receptor-positive breast cancers.
Professor Loi recent research investigates how reproductive life events shape breast cancer risk, tumour immune microenvironments, and clinical outcomes. This work provides a framework for understanding how physiological tissue remodeling can alter anti-tumour immune surveillance and may reveal new opportunities for prevention, risk stratification, and immunotherapy development.

Professional Experience/Awards

Dr. Sherene Loi is a Medical Oncologist, Professor at the University of Melbourne and Head of the Translational Breast Cancer Genomics and Therapeutics Laboratory at the Peter MacCallum Cancer Centre in Melbourne, Australia.
Dr. Loi is a central figure in international collaborative research. She is the Scientific Chair of the International Breast Cancer Study Group (IBCSG),one of the largest global breast cancer trial cooperative groups, and Chair of the Kathleen Cunningham Foundation Consortium for Familial Breast Cancers (kConFab). Her honors include election to the Australian Academy of Science (2026), election to the Australian Academy of Health and Medical Sciences (2018), the Prime Minister's Prize for Life Scientist of the Year (2021), the AAHMS Jian Zhou Medal (2021), the AACR Outstanding Investigator for Breast Cancer Research Award (2020) and the ESMO Breast Cancer Award (2020). She is the inaugural National Breast Cancer Foundation of Australia Endowed Chair (2017). She has been listed as a top 1% highly cited researcher by Clarivate Web of Science since 2018.

Education

PhD in Medical Science University of Melbourne / Peter MacCallum Cancer Centre, Australia
Fellowship of the Royal Australasian College of Physicians (FRACP) Medical Oncology Specialist Board Certification
Medical Degree (MD) University of Monash, Australia

Abstract of the lecture

Tumour-infiltrating lymphocytes are among the most reproducible immune biomarkers in breast cancer. In triple-negative breast cancer, higher levels of stromal TILs are strongly associated with improved prognosis and greater sensitivity to immune checkpoint blockade. However, why some breast cancers are highly immune-infiltrated while others remain immune-poor is still incompletely understood. Most current models focus on tumour-intrinsic features, including genomic instability, antigenicity, interferon signalling, and immune evasion. Less is known about how host physiology and normal breast tissue states shape the immune contexture from which breast cancers arise.
This lecture will examine reproductive history as one potential determinant of breast immune fitness.

Our recent work investigates how parity, lactation, and post-lactational mammary gland remodeling influence anti-tumour immune surveillance. The clinical implications are particularly relevant to triple-negative breast cancer, where immune infiltration has major prognostic and therapeutic significance. By integrating clinical cohort analyses, single-cell profiling, spatial immune analysis, and experimental models, this work provides a framework for understanding how physiological reproductive events may influence tumour initiation, TIL formation, immune escape, and response to immunotherapy. More broadly, the presentation will highlight how studying the origins of immune infiltration in breast cancer may identify new approaches for risk stratification, prevention, immune biomarker development, and rational immunotherapy strategies.

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Session 3

New Frontier in Cancer Biology

3-1. Dark Transcriptome-derived Neoantigens

Speaker: Akihide Yoshimi, MD, PhD
(Chief, National Cancer Center Research Institute, Japan)

3-2. The Viral Basis of Cancer

Speaker: David T Ting, MD
(Scientific Director, Mass General Brigham Cancer Institute, USA)

IAAO2026

Dark Transcriptome-derived Neoantigens

Speaker

Akihide Yoshimi, MD, PhD

Chief, Division of Cancer RNA Research
National Cancer Center Research Institute (NCCRI),
Tokyo, Japan

Chair

Chikashi Ishioka, MD, PhD

Professor Emeritus, Tohoku University, Japan
Director, JR Sendai Hospital, Japan

Akihide Yoshimi, MD, PhD

Research Summary

Dr. Akihide Yoshimi is a physician-scientist and internationally recognized leader in RNA biology and hematologic malignancies. His research focuses on understanding and therapeutically targeting dysregulated RNA processing in cancer. During his seminal postdoctoral training at Memorial Sloan Kettering Cancer Center, he made foundational contributions to elucidating the roles of spliceosomal mutations, including SF3B1, SRSF2, and U2AF1, in hematologic malignancies. Currently, his research program integrates advanced functional genomics with long-read and single-cell technologies to develop RNA-targeted therapeutic strategies. Dr. Yoshimi also aims to develop cancer mRNA vaccines based on dark transcriptome-derived neoantigens across multiple cancer types.

Professional Experience/Awards

Dr. Akihide Yoshimi is the Chief of the Division of Cancer RNA Research at the National Cancer Center Research Institute (NCCRI) in Tokyo, Japan. He leads a multidisciplinary research program focused on understanding and therapeutically targeting dysregulated RNA processing in cancer. Trained as both a physician and a basic scientist, Dr. Yoshimi bridges fundamental RNA biology, cancer genomics, and clinic.
During his postdoctoral training at Memorial Sloan Kettering Cancer Center in New York, he made important contributions to elucidating how spliceosomal gene mutations, including alterations in *SF3B1*, *SRSF2*, and *U2AF1*, drive aberrant mRNA isoform expression and

leukemogenesis. Since returning to Japan, his laboratory has integrated functional genomics, long-read sequencing, and single-cell technologies to identify RNA-based therapeutic vulnerabilities in hematologic malignancies and solid tumors.
Dr. Yoshimi has received competitive recognition and research support, including the ASH Scholar Award, LLS CDP Achievement Award, and JCA-Mauvernay Award. He has authored more than 100 peer-reviewed publications in leading journals such as *Nature*, *Cell*, *Nature Medicine*, and *Cancer Discovery*. His current work also explores cancer mRNA vaccines targeting dark transcriptome-derived neoantigens across multiple cancer types.

Education

PhD in Medical Science	The University of Tokyo, Japan (2011)
Medical Degree (MD)	The University of Tokyo, Japan (2003)

Abstract of the lecture

Cancer immunotherapy relies on the identification of tumor-specific antigens that are both immunogenic and broadly applicable across patients. However, personalized mutation-derived neoantigen approaches often require individualized design and complex manufacturing processes, whereas shared cancer antigens frequently show detectable expression in normal tissues and may have limited immunogenicity due to immune tolerance. To address this unmet need, we have explored the “dark transcriptome,” particularly transposable element-derived transcripts with non-canonical splicing, as a previously underappreciated source of cancer neoantigens.
By developing a computational discovery platform supported by long-read RNA sequencing data from 107 primary patient samples across multiple cancer types, we identified approximately 430,000 unannotated transcripts, including more than 90,000 cancer-specific dark transcripts. These transcripts were frequently derived from non-canonical genomic regions and included candidates with highly restricted expression in cancer cells and little or no detectable expression in normal tissues. Notably, tumors expressing selected dark transcripts displayed features suggestive of enhanced tumor immunogenicity, including broader transposable element activation and increased intratumoral CD8-positive T-cell infiltration.
To evaluate their biological and immunological relevance, we integrated in vitro peptide stimulation assays, ELISpot assays, antigen-prediction approaches, and immunopeptidomics analyses. These studies revealed that a subset of candidate dark transcript-derived peptides are translated, processed within cancer cells, presented by MHC molecules, and capable of inducing T-cell responses. Together, these findings support the concept that the dark transcriptome represents a rich and largely unexplored reservoir of shared cancer neoantigens. These efforts may open a new avenue for broadly applicable, RNA-informed cancer immunotherapy targeting antigens beyond the canonical cancer genome.

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The Viral Basis of Cancer

Speaker

David T. Ting, MD

Scientific Director, Mass General Brigham Cancer Institute, USA
Associate Professor of Medicine, Harvard Medical School, USA

Chair

Chikashi Ishioka, MD, PhD

Professor Emeritus, Tohoku University, Japan
Director, JR Sendai Hospital, Japan

David T. Ting, MD

Research Summary

Dr. David T. Ting is a prominent physician-scientist and an internationally recognized leader in cancer genomics and translational oncology. He is widely known for his pioneering investigations into the “dark matter” of the cancer genome, focusing on the aberrant expression of non-coding RNAs and repeat element sequences. Using state-of-the-art technologies such as single-cell RNA sequencing and spatial transcriptomics, his laboratory elucidates how these repeat elements drive malignant transformation and modulate tumor-immune interactions. Furthermore, Dr. Ting is at the forefront of developing innovative RNA-based liquid biopsy approaches, aiming to achieve early cancer detection and therapeutic intervention through minimally invasive, blood-based molecular assays.

Professional Experience/Awards

Dr. David T. Ting serves as the Scientific Director and the Zebunisha Juma Endowed Chair of the Mass General Brigham Cancer Institute. He is an Associate Professor of Medicine at Harvard Medical School. In these capacities, he leads high-impact initiatives that bridge sophisticated molecular biology with clinical innovation, ensuring the rapid translation of scientific discoveries into bedside patient care. Dr. Ting’s research has redefined our understanding of genomic regulation, particularly through his discovery that satellite RNAs act as potent inducers of inflammation and immune suppression. This work has opened

new therapeutic avenues and shifted the paradigm of how "dark genome" is viewed in oncology. As a highly productive investigator, he has authored over 120 peer-reviewed publications in premier journals such as Science, Nature, and Cell.
His scientific excellence is recognized through his active participation in the global oncology community, where he is a frequent invited speaker at major conferences including AACR, AACR-Japanese Cancer Association joint meeting, and GI ASCO. Dr. Ting is also a leader in integrating artificial intelligence and high-dimensional data into precision oncology, helping to shape the future of personalized medicine. His work has led to the formation of 3 biotech companies. Beyond his research, he is dedicated to mentoring the next generation of physician-scientists. His ongoing commitment to translational research continues to drive the development of more precise, effective, and minimally invasive diagnostic and therapeutic strategies for cancer patients worldwide.

Education

Science Baccalaureate (SB) in Chemical Engineering and Biology	MIT, USA
Medical Degree (MD)	Harvard Medical School, USA
Internal Medicine Residency	Massachusetts General Hospital, USA
Medical Oncology Fellowship	Dana-Farber/Mass General Hospital, USA

Abstract of the lecture

The aberrant expression of repeat elements from the “dark genome” is a common feature found in cancer. The majority of these are non-coding RNAs that have shown the ability to stimulate a viral-like innate immune response. In addition, we have found repeat RNAs are able to “infect” the microenvironment via extracellular vesicles causing broad effects on the tumor microenvironment and cellular plasticity. This inflammatory process appears to be important for cancer cell plasticity that leads to immune evasion and metastatic dissemination. However, this viral stress also presents a liability for cancer cell survival. Cancer cells utilize the long interspersed nuclear element 1 (LINE-1) retrotransposon, one of the few protein coding repeat elements in the human genome, as an adaptive response to manage viral like repeats. LINE-1 is comprised of ORF1, a ribonucleotide protein, and ORF2, a reverse transcriptase and endonuclease. We have shown that suppression of ORF1 and ORF2 has distinct effects on cancer cell behavior. Nucleoside reverse transcriptase inhibitors commonly used for viral infections are able to suppress ORF2 with effects demonstrated in a clinical trial of metastatic colorectal cancer patients. Suppression of ORF1 with shRNAs have shown that ORF1 is necessary to attenuate the anti-viral response stimulated by repeat RNAs leading to significant effects on cancer cell viability and additional disruption of the tumor microenvironment in animal models. Collectively, cancers activate the dark genome to generate a viral response that drives cancer progression, but opens a vulnerability that can be therapeutically exploited.

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Session 4

Mechanism of Cancer Initiation

4-1. Somatic Mutations in Ageing, Precancer and Autoimmune Disease

Speaker: Inigo Martincorena, PhD
(Group Leader, Wellcome Sanger Institute, UK)

4-2. Spatial Profiling of Human Tumors in Three Dimensions

Speaker: Peter Sorger, PhD
(Otto Krayer Professor of Systems Pharmacology, Harvard Medical School, USA)

Somatic Mutations in Ageing, Precancer and Autoimmune Disease

Speaker

Iñigo Martincorena, PhD

Group Leader, Somatic Evolution Group
Wellcome Sanger Institute, Cambridge, United Kingdom

Chair

Naoko Ohtani, MD, PhD

Professor of Department of Pathophysiology, Osaka Metropolitan University, Graduate School of Medicine, Japan

Iñigo Martincorena, PhD

Research Summary

Dr. Iñigo Martincorena is a visionary genomic scientist specializing in somatic evolution. His pioneering research utilizes ultra-high-precision DNA sequencing and innovative computational approaches to map somatic mutations in healthy human tissues. His work has fundamentally reshaped our understanding of oncogenesis by revealing that normal organs are mosaics of mutant clones, often harboring cancer-driver mutations decades before clinical manifestation. Additionally, he has demonstrated that mutation rates across mammalian species inversely correlate with lifespan and that somatic mutations contribute to inflammation and autoimmunity, providing a molecular framework for aging. By dissecting the boundary between normal physiology and malignant transformation, Dr. Martincorena provides critical insights into the earliest stages of cancer and tissue homeostasis.

Professional Experience/Awards

Dr. Iñigo Martincorena is the Group Leader of the Somatic Evolution Group at the Wellcome Sanger Institute in Cambridge, United Kingdom. In this capacity, he leads a world-class research program dedicated to understanding how mutation, selection, and clonal expansion operate across the human lifespan. His studies have revealed the "hidden landscape" of somatic evolution, challenging long-held assumptions about the genomic

integrity of non-cancerous tissues and significantly impacting the fields of aging and oncology.
Dr. Martincorena's academic career began with formative research at the University of Cambridge and the European Bioinformatics Institute (EMBL-EBI), where he focused on the evolution of mutation rates. Since establishing his independent laboratory at the Sanger Institute, he has emerged as a central figure in international genomics, known for integrating large-scale data with elegant evolutionary theory.
His scientific excellence has been recognized through numerous prestigious honors, including election to EMBO and the Josef Steiner Award for Cancer Research. Dr. Martincorena has authored seminal publications in premier journals such as Nature, Science, and Cell, which have been widely cited as foundational to the modern study of somatic evolution and epithelial mosaicism. As a highly sought-after expert, he frequently delivers keynote addresses at major international symposia, including AACR and EACR. Through his continued commitment to advancing genomic science, he is laying the essential groundwork for new strategies in cancer prevention, early detection, and aging.

Education

PhD in Evolutionary Genomics	University of Cambridge / European Bioinformatics Institute (EMBL-EBI), UK (2012)
Bachelor of Science (BSc) in Biology	University of Navarra, Spain

Abstract of the lecture

Somatic mutations underpin cancer development and have been speculated to contribute to ageing and a range of diseases. The ability to sequence cancer genomes has transformed our understanding of the genetics and evolution of a wide range of cancers. However, owing to technical limitations, much less is known about how cells in our tissues accumulate mutations during ageing. For over a decade, our work has focused on studying somatic mutation and selection in normal tissues, revealing that clones with driver mutations colonise our tissues as we age. These discoveries have implications for our understanding of precancer evolution and the pathogenesis of other chronic diseases. In this talk, I will briefly summarise this work and describe our recent work unveiling an unknown landscape of somatic evolution in autoimmunity.

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Spatial Profiling of Human Tumors in Three Dimensions

Speaker

Peter Sorger, PhD

Otto Krayer Professor of Systems Pharmacology, Harvard Medical School
Director, Harvard Program in Therapeutic Science (HiTS)
Director, Laboratory of Systems Pharmacology (LSP), United States

Chair

Naoko Ohtani, MD, PhD

Professor of Department of Pathophysiology, Osaka Metropolitan University, Graduate School of Medicine, Japan

Peter Sorger, PhD

Research Summary

Dr. Peter Sorger is a preeminent leader in systems biology and systems pharmacology, specializing in quantitative approaches to drug discovery and precision oncology. His research focuses on the quantitative analysis of cell signaling networks and drug mechanisms of action within human tissues and animal models. His laboratory is at the forefront of recent developments in highly multiplexed 2D and 3D tissue imaging as a means of understanding the fundamental biology of tumor-immune interaction, particularly in early stage cancers. In addition, by integrating cancer biology, engineering, and advanced computational modeling, Dr. Sorger also aims to develop diagnostic tests that can better predict which patients will respond to targeted therapies given individually and in combination. His work provides critical insights into the future of precision medicine and clinical trial design.

Professional Experience/Awards

Dr. Peter Sorger is the Otto Krayer Professor of Systems Pharmacology at Harvard Medical School. He is the founding Director of the Harvard Program in Therapeutic Science (HiTS) and its primary experimental component, the Laboratory of Systems Pharmacology (LSP). These interdisciplinary initiatives are dedicated to transforming drug development through the integration of mathematics, experimental biology, and clinical research. Under his

leadership, HiTS programs have become international hubs for innovative research that bridges basic science and clinical application.
Prior to his appointment at Harvard, Dr. Sorger served as a distinguished faculty member at the Massachusetts Institute of Technology (MIT), where he made foundational contributions to the field of systems biology. He is a central figure in large-scale international collaborative efforts, including the NCI-funded Human Tumor Atlas Network (HTAN), where he spearheads the establishment of global standards for spatial biology and high-dimensional data integration.

His scientific excellence is reflected in over 350 influential peer-reviewed publications, many in premier journals such as *Nature*, *Cell*, and *Science*. He has trained over 40 students and fellows who now direct their own laboratories in the US, Asia, and Europe. As a highly sought-after mentor and thought leader, he frequently serves as a keynote speaker at major international conferences, advocating for rigorous, systems-level approaches to oncology. His commitment to open science and the development of public-domain software and data tools continues to shape the future of cancer research, which an increasing focus on realizing the promise of precision approaches to cancer care for patients worldwide.

Education

PhD in Biology Trinity College, University of Cambridge, UK (1993)
Bachelor of Arts (AB) in Biochemistry
Harvard University, Cambridge MA USA
Postdoctoral Fellowship
University of California, San Francisco (UCSF), USA (Under Prof. Harold Varmus)

Abstract of the lecture

Solid tumors evolve in competition with the immune system and must also attract resources such as nutrients and oxygen needed to proliferate and invade normal tissue. This results in complex spatial distribution of tumor, immune, and stromal cells within the tumor microenvironment (TME).
Highly multiplexed tissue imaging (spatial proteomics), in combination with other single cell methods, promises to reveal the composition and regulation of TME features in molecular detail but the great majority of data collected to date, including all classical histopathology, is based on hematoxylin and eosin (H&E) or immunohistochemistry (IHC) of thin 2D tissue slices. We have recently shown that no cells in such 5 micron tissue section are intact and that this substantially impacts cell type identification and interaction analysis.
I will describe the development of a suite of computational and experimental methods for performing 3D image-based profiling of human tumor sections at different resolutions and spatial scales ranging from ~50 micron thick (with confocal imaging) to 1 mm thick (with cyclic light sheet fluorescence microscopy; LSFM). These types of 3D cyclic immunofluorescence (CyCIF) make it possible to map cell-cell interactions in complex tissue assemblies based on the proximity of cell membranes rather than simply the location of nuclei, as commonly done in spatial tissue analysis. I will describe how 50-plus channel super resolution CyCIF on thick sections makes it possible to precisely characterize processes such as T cell-tumor engagement. At longer length scales, cyclic LSFM enables large multi-cellular assemblies such as secondary and tertiary lymphoid structures to be studied in detail. I will close with a description of how different types of 3D profiling can be combined on human clinical specimens, from virtual H&E imaging to cyclic LSFM to high-plex high-resolution confocal microscopy. Finally, I will discuss how 3D imaging of human tissues can inform more routine 2D approaches.

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Session 5

Mechanism of Cancer Evolution

5-1. Molecular Dissection of Breast Cancer Development and Therapy Response in Murine Models

Speaker: Jos Jonkers, PhD
(Head, Molecular Pathology; Professor, Molecular Experimental Oncology (NKI), Netherlands)

5-2. Inflammageing as a Driver of Cancer: from Tumour Promotion to Disease Prevention

Speaker: Charles Swanton, MBPhD, FRS
(Clinical Director, Francis Crick Institute; Chair, UCL; Director & CI, TRACERx, UK)

Molecular Dissection of Breast Cancer Development and Therapy Response in Murine Models

Speaker

Jos Jonkers, PhD

Head, Division of Molecular Pathology
Professor of Molecular Experimental Oncology
Netherlands Cancer Institute (NKI), Amsterdam, The Netherlands

Chair

Masakazu Toi, MD, PhD

Director, Tokyo Metropolitan Cancer and Infectious Diseases Center Komagome Hospital, Japan

Jos Jonkers, PhD

Research Summary

Dr. Jos Jonkers is a world-renowned geneticist and an internationally recognized leader in the molecular pathology of breast cancer. He is pioneering the development of sophisticated genetically engineered mouse models (GEMMs) that faithfully recapitulate the genomic complexity and clinical progression of human breast cancer. His research focuses on elucidating genetic drivers of cancer initiation and therapeutic resistance, specifically regarding alterations in BRCA1, BRCA2, and TP53. By integrating GEMMs with functional genomics and advanced organoid systems, his laboratory has generated critical insights into hereditary breast cancer and invasive lobular carcinoma. His discoveries regarding PARP inhibitor resistance have directly informed the development of novel combination treatment strategies.

Professional Experience/Awards

Dr. Jos Jonkers serves as the Head of the Division of Molecular Pathology at the Netherlands Cancer Institute (NKI) in Amsterdam and is Professor of Molecular Experimental Oncogenetics and Cancer Therapeutics at Leiden University. In these capacities, he leads one of Europe's premier research programs dedicated to the molecular

dissection of breast cancer, bridging the gap between basic genetic research and preclinical drug development.
Throughout his distinguished career, Dr. Jonkers has been a central figure in the European cancer research community. He has held numerous leadership and advisory positions. His work is characterized by the use of advanced preclinical models to accelerate the clinical implementation of personalized therapies.
His scientific excellence is reflected in more than 300 peer-reviewed publications in premier journals such as Nature, Nature Genetics, Cancer Cell and Cancer Discovery. Dr. Jonkers' contributions have been recognized with several of the most prestigious honors in science, including an ERC Synergy Grant, a Cancer Research UK Cancer Grand Challenge Grant, and the Stand Up To Cancer Laura Ziskin Prize in Translational Research, which underscores his role as a leader in innovative oncology research. As a highly sought-after expert, he is a frequent keynote speaker and scientific organizer for major international symposia, including AACR, EACR and ESMO. Beyond his research, he is a dedicated mentor, committed to training the next generation of cancer biologists. His ongoing efforts continue to define the molecular standards for understanding breast cancer heterogeneity and improving therapeutic outcomes for patients worldwide.

Education

PhD in Molecular Biology	Netherlands Cancer Institute (NKI) / University of Amsterdam, Netherlands (1999)
Master of Science (MSc) in Molecular Sciences	Wageningen University, Netherlands (1989)

Abstract of the lecture

Genetically engineered and patient-derived xenograft (PDX) models of human cancer are powerful preclinical in vivo platforms that not only permit us to gain a detailed insight into the specific genetic changes that drive tumor initiation and progression but also provide powerful tools to study the mechanisms underlying drug response and acquired resistance. Once these processes are understood in sufficient detail it may be possible to design combination therapies that not only cause complete remissions but also eliminate remnant cells that elicit recurrent disease.
We have developed genetically engineered mouse models and PDX models for ductal carcinoma in situ (DCIS) and invasive breast cancer, including BRCA1/2–associated hereditary breast cancer and E-cadherin–mutated invasive lobular carcinoma. In addition, we have developed somatic genome editing approachges for rapid modelling of breast cancer in mice and rats using intraductal injection of lentiviruses. We have successfully used these models and technologies to (i) identify factors that contribute to invasive progression of DCIS, (ii) identify and validate cancer driver genes in BRCA1–mutated triple-negative breast cancer and E-cadherin–mutated ILC, (iii) identify and validate mechanisms of acquired resistance to targeted therapeutics such as PARP inhibitors, and (iv) identify strategies to overcome PARP inhibitor resistance.

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Inflammageing as a Driver of Cancer: from Tumour Promotion to Disease Prevention

Speaker

Charles Swanton, MBPhD, FRCP, FMedSci, FAACR, FRS

Clinical Director and Principal Group Leader, The Francis Crick Institute Chair in Personalised Cancer Medicine, UCL Cancer Institute
Director and Chief Investigator, CRUK UCL Lung Cancer Centre of Excellence / TRACERx

Chair

Masakazu Toi, MD, PhD

Director, Tokyo Metropolitan Cancer and Infectious Diseases Center Komagome Hospital, Japan

Charles Swanton, MBPhD, FRCP, FMedSci, FAACR, FRS

Research Summary

Dr. Charles Swanton is a world-leading clinician-scientist and a preeminent authority on cancer evolution and precision oncology. He is best known for founding and leading the TRACERx program, a landmark longitudinal study that demonstrated how intratumor heterogeneity drives therapeutic resistance and metastatic progression. His research provides a transformative framework for understanding cancer as a dynamically evolving ecosystem. Recently, his groundbreaking work on environmental carcinogenesis revealed how air pollution promotes malignancy by triggering inflammatory pathways in cells with pre-existing mutations. By integrating evolutionary biology with clinical practice, Dr. Swanton seeks to develop evolution-informed therapeutic strategies to counteract the adaptive capacity of advanced cancers.

Professional Experience/Awards

Dr. Charles Swanton is a senior Principal Investigator of the Cancer Evolution and Genome Instability Laboratory, and Deputy clinical director at the Francis Crick Institute. He combines his research with clinical duties at UCLH as a Consultant thoracic oncologist, focused on how tumours evolve over space and time. His research branched evolutionary histories of solid tumours, processes that drive cancer cell-to-cell variation in the form of

new cancer mutations or chromosomal instabilities, and the impact of such cancer diversity on effective immune surveillance and clinical outcome. Charles is chief investigator of TRACERx, a lung cancer evolutionary study, the national PEACE autopsy program, and the TRACERx EVO study.

Charles was made Fellow of the Royal College of Physicians in April 2011, appointed Fellow of the Academy of Medical Sciences in 2015, awarded the Royal Society Napier Professorship in Cancer in 2016 and elected Fellow of the Royal Society in 2018, Fellow of the Academy of the American Association for Cancer Research in 2020.

Charles has been awarded several prizes including the San Salvatore prize for Cancer Research (2017) Massachusetts General Hospital, Jonathan Kraft Prize for Excellence in Cancer Research (May 2018), the ESMO Award for Translational Cancer Research (2019), the Weizmann Institute Sergio Lombroso Award in Cancer Research (2021), the MSKCC Paul Marks Prize for Cancer Research (2021), the Jeantet-Collen Prize for Translational Medicine (2024), the Gustave Roussy Prize (2025), and the INSERM International prize. He was elected as a member of the U.S. National Academy of Medicine, recognising his contributions to understanding tumour evolution and advancing translational cancer research in October 2025. In 2026 he was the recipient of the Jan Waldenstrom Prize, the Sjoberg Prize, the Burkitt Medal, the EACR Mike Price Gold medal Award and elected Fellow of the Science Museum.

Education

MBPhD
Imperial Cancer Research Fund Laboratories (1999) and Cancer Research UK clinician scientist/medical oncology training (2008)

Fellow of the Royal College of Physicians (FRCP)
Specializing in Medical Oncology

Abstract of the lecture

Lung cancer remains the leading cause of cancer death worldwide, yet our ability to identify at-risk individuals before malignancy emerges and to intercept disease before it becomes established remains limited. Evidence is emerging that macrophage and myeloid programs sit at the heart of lung cancer biology across its entire natural history, from the earliest pollutant-driven inflammatory insult through to malignant progression and recurrence and that this inflammatory biology now leaves detectable plasma proteomic signals in the blood that can be harnessed for risk prediction and molecular cancer prevention.
At initiation, particulate matter exposure provokes alveolar macrophages to release IL-1β, which primes mutant epithelial cells to expand. In lineage-restricted EGFR-mutant mouse models, diverse epithelial lineages, basal, club, neuroendocrine and AT2 , converge on a keratin 8^{+}/claudin 4^{+} alveolar transitional state (KAC) within the alveolar niche, with KAC expansion driven by IL-1β and restrained by its blockade. This tumour-promoting inflammatory state is reflected in a 14-protein plasma signature, derived from an unbiased machine learning approach on UK Biobank proteomics data from 50,000 individuals and validated across eight independent cohorts including a predominantly never-smoker Taiwanese population. The signature is induced by pollution, oncogenic EGFR and IL-1β, predicts incident lung cancer years before diagnosis, and when applied retrospectively to the CANTOS trial identifies those who derive lung cancer preventive benefit from anti-IL-1β therapy, bringing the number-needed-to-treat to prevent one lung cancer into a clinically actionable range.
The same myeloid axis governs the later trajectory of disease. Age-related clonal haematopoiesis, particularly involving TET2-mutant clones, seeds tumours with mutant

monocytes that accumulate as CD11b^{+} macrophages, remodel the microenvironment, accelerate tumour organoid growth, and independently predict recurrence and death across TRACERx NSCLC patients and over 49,000 individuals in the MSK-IMPACT pan-cancer cohort. This phenomenon, termed tumour-infiltrating clonal haematopoiesis (TI-CH) connects the ageing haematopoietic system directly to cancer evolution.
A coherent picture is emerging: pollutant- and age-driven myeloid inflammation creates and sustains a tumour-promoting niche from initiation through to progression. The circulating inflammatory signals this biology generates now allow the enrichment of prevention trials for those most likely to benefit, providing a tractable framework for precision lung cancer prevention.

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Session 6

Breakthrough Technology: Innovation with AI

6-1. Emerging Innovations at the Interface of AI and Translational Cancer Research

Speaker: Eliezer Van Allen, MD
(AI Chair, Dana-Farber Cancer Institute; Professor, Harvard Medical School, USA)

6-2. Generative AI for Decoding Tumor Evolution and Immunotherapy Response

Speaker: Elham Azizi, PhD
(Associate Professor, Columbia University, USA)

6-3. AI-driven Approach for Drug Discovery and Medical Science

Speaker: Yoshihiro Yamanishi, PhD
(Professor, Nagoya University; Chief, Aichi Cancer Center Research Institute, Japan)

Emerging Innovations at the Interface of AI and Translational Cancer Research

Speaker

Eliezer M. Van Allen, MD

Chandra Nohria Family Chair for AI in Cancer Research at Dana-Farber
Chief, Division of Population Sciences, Dana-Farber Cancer Institute
Professor of Medicine, Harvard Medical School
Institute Member, Broad Institute of MIT and Harvard

Chair

Kiyohiko Hatake, MD, PhD

Professor, Akasaka Sanno Medical Center, Japan

Eliezer M. Van Allen, MD

Research Summary

Dr. Eliezer Van Allen is a world-renowned medical oncologist and computational biologist specializing in clinical computational oncology. His research focuses on developing and applying computational strategies to translate high-dimensional cancer data into clinical insights. His laboratory develops advanced machine learning and artificial intelligence approaches to integrate genomic, imaging, and clinical data, aiming to uncover novel resistance mechanisms and predict therapeutic responses. Ultimately, his work seeks to standardize the responsible deployment of AI and other computational approaches to enhance precision cancer medicine and improve patient outcomes.

Professional Experience/Awards

Dr. Eliezer Van Allen is the Chief of the Division of Population Sciences and holds the Chandra Nohria Family Chair for AI in Cancer Research at the Dana-Farber Cancer Institute. He also serves as a Professor of Medicine at Harvard Medical School and is an Institute Member of the Broad Institute of MIT and Harvard. In these roles, he leads interdisciplinary efforts to integrate data science with oncology, positioning him as a global leader in the adoption of artificial intelligence in medicine.

As a dedicated physician-scientist, Dr. Van Allen bridges the gap between sophisticated computational modeling and bedside patient care. His prolific scientific output includes over 200 peer-reviewed publications in premier journals such as Nature, Science, and The New England Journal of Medicine. His research is characterized by a strong commitment to open science and large-scale data sharing, which has fundamentally influenced how the oncology community handles complex genomic datasets.
Dr. Van Allen's scientific excellence has been honored with numerous prestigious accolades, including the AACR Waun Ki Hong Award and the Foundation for the National Institute of Health Trailblazer Prize. He is a highly sought-after advisor and keynote speaker for major international organizations, including ASCO, AACR, and the Parker Institute for Cancer Immunotherapy. Through his leadership in clinical computational oncology, Dr. Van Allen continues to redefine the standards of precision medicine, advocating for the ethical and effective use of high-dimensional data to deliver personalized care to cancer patients worldwide.

Education

Bachelor of Science (BSc) in Symbolic Systems	Stanford University, USA (2003)
Medical Degree (MD)	David Geffen School of Medicine at UCLA, USA (2007)
Internal Medicine Residency	University of California, San Francisco (UCSF), USA
Medical Oncology Fellowship	Dana-Farber/Partners Cancer Care, USA

Abstract of the lecture

The rapid growth of artificial intelligence (AI) based innovations across numerous domains has created major opportunities for enhancing cancer research and improving clinical are. These include the rise of agentic AI for pursuing all aspects of the biological discovery process in cancer, including typical bioinformatics tasks all the way through to complex scientific reasoning and hypothesis generation. In addition, similar AI strategies when applied in large clinical settings may créate new opportunities for enhancing precisión medicine strategies across all aspects of cancer care. In this presentation, we will explore emerging concepts at the interface of AI and translational cancer innovations. We Will also examine how AI tools may impact clinical care in ways that could dramatically change practice, and inform how scientists and clinicians can advocate for safe and ethical AI that may improve cancer patient care.

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Generative AI for Decoding Tumor Evolution and Immunotherapy Response

Speaker

Elham Azizi, PhD

Herbert & Florence Irving Associate Professor of Cancer Data Research
Associate Professor of Biomedical Engineering
Columbia University, United States

Chair

Kiyohiko Hatake, MD, PhD

Professor, Akasaka Sanno Medical Center, Japan

Elham Azizi, PhD

Research Summary

Dr. Elham Azizi is a pioneering computational biologist and biomedical engineer leading the intersection of artificial intelligence, single-cell biology, and cancer genomics. Her research develops advanced machine-learning methodologies, including deep generative models and causal inference frameworks, to analyze high-dimensional single-cell and spatial genomics data. By reconstructing the dynamic cellular "circuitry" of the tumor microenvironment, she elucidates how cellular plasticity and communication drive tumor progression and immune evasion. Conceptualizing tumors as dynamic ecosystems, her work provides critical insights into biomarker discovery and personalized immunotherapy across diverse disease contexts, such as leukemia and breast cancer, ultimately aiming to transform patient care through data science.

Professional Experience/Awards

Dr. Elham Azizi is the Herbert & Florence Irving Associate Professor of Cancer Data Research and an Associate Professor of Biomedical Engineering at Columbia University. She holds multi-departmental affiliations with Computer Science, the Data Science Institute,

and the Herbert Irving Comprehensive Cancer Center. In these roles, she leads a highly interdisciplinary research program dedicated to decoding the complexity of the tumor microenvironment through the lens of artificial intelligence.
Her scientific excellence has been recognized with some of the most prestigious honors for early-career investigators. Notably, she has been recognized in the 2026 Class of 40 Under 40 in Cancer and was awarded the 2025 Vilcek Prize for Creative Promise in Biomedical Science and the 2024 Takeda–New York Academy of Sciences Innovators in Science Award. Her contributions have also been recognized through selection as an Allen Distinguished Investigator, the National Science Foundation (NSF) CAREER Award, and the Chan Zuckerberg Initiative Science Diversity Leadership Award, highlighting her commitment to both scientific innovation and inclusivity.
As an emerging leader in the global cancer research community, Dr. Azizi frequently delivers keynote addresses at major international conferences, including AACR and SITC. Her unique background in electrical engineering and bioinformatics allows her to bridge the gap between rigorous computational theory and complex biological systems. Her work continues to redefine the standards of clinical data integration, advocating for the use of machine learning to uncover the resistant and plastic states of cancer cells. Through her continued advocacy for translational data science, she is shaping the future of precision oncology and inspiring the next generation of interdisciplinary scientists.

Education

Postdoctoral Fellowship	Memorial Sloan Kettering Cancer Center / Columbia University, USA (Under Prof. Dana Pe'er)
PhD in Bioinformatics	Boston University, USA (2014)
MS in Electrical Engineering	Boston University, USA (2010)
BS in Electrical Engineering	Sharif University of Technology, Iran (2008)

Abstract of the lecture

Cancer immunotherapies have transformed the treatment landscape across multiple malignancies, yet durable responses remain limited to a subset of patients. A major challenge is understanding how tumor evolution, cellular plasticity, and interactions within the tumor microenvironment shape therapeutic response and resistance. Recent advances in single-cell and spatial multi-omic technologies now enable comprehensive characterization of these processes at unprecedented resolution, generating datasets whose complexity requires new artificial intelligence approaches for biological discovery.

In this talk, I will discuss how generative AI models can be used to decode the cellular and molecular mechanisms governing anti-tumor immunity. Using examples from melanoma, leukemia, and breast cancer, I will present novel computational frameworks that integrate single-cell transcriptomic, spatial, and histopathologic data to characterize immune-tumor ecosystems, identify determinants of immunotherapy response, and uncover mechanisms of immune escape. These approaches include attention-based models that infer spatially organized cell-cell communication networks, probabilistic frameworks that disentangle intrinsic and microenvironmental drivers of cellular behavior, and generative models that reconstruct continuous cell-state trajectories directly from patient specimens.

I will highlight how these methods have enabled the discovery of T cell populations associated with successful donor lymphocyte infusion in relapsed leukemia, revealed the spatiotemporal clonal dynamics and phenotypic plasticity of T cells during human graft-versus-host disease, identified tumor- and microenvironment-derived programs associated with resistance to immune checkpoint blockade in melanoma, and characterized spatial immune and metabolic niches that contribute to treatment failure in solid tumors.

Building on these advances, we are developing foundation-scale models trained across large single-cell atlases that learn generalizable representations of cellular identity and dynamics, enabling prediction of future cell states and responses to therapeutic perturbation guiding precision and personalized therapies.

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AI-driven Approach for Drug Discovery and Medical Science

Speaker

Yoshihiro Yamanishi, PhD

Professor, Graduate School of Informatics, Nagoya University

Chief at Division of Interdisciplinary Research and Development (R&D), Aichi Cancer Center Research Institute

Chair

Kiyohiko Hatake, MD, PhD

Professor, Akasaka Sanno Medical Center, Japan

Yoshihiro Yamanishi, PhD

Research Summary

Dr. Yoshihiro Yamanishi is a leading authority in bioinformatics and chemoinformatics, specializing in machine learning for genomic and data-driven drug discovery. His research focuses on in silico modeling of molecular interaction networks, including protein-protein and compound-protein interactions. His laboratory leverages heterogeneous omics datasets—spanning the genome, transcriptome, and metabolome—to identify novel therapeutic targets and search for drug candidate compounds. Recently, he has pioneered single-cell drug efficacy analysis and generative AI models for de novo molecular design. By bridging informatics and the life sciences, Dr. Yamanishi's work aims to transform the pharmaceutical landscape through the responsible integration of artificial intelligence and medical science.

Professional Experience/Awards

Dr. Yoshihiro Yamanishi is a Professor at the Graduate School of Informatics at Nagoya University. A central figure in the global computational biology community, he is a member of the Board of Directors at the International Society for Computational Biology (ISCB). He served the President of the Japanese Society for Bioinformatics (JSBi) in 2023-2024. In

these leadership roles, he spearheads initiatives to integrate artificial intelligence into life sciences and healthcare innovation on both a national and international scale.
Throughout his distinguished career, Dr. Yamanishi has held significant academic positions at Curie Institute, Kyushu University and Kyushu Institute of Technology, where he established his reputation as an expert in AI-driven drug discovery. His prolific scientific output includes over 150 peer-reviewed publications in premier journals such as Nature Computational Science, Nature Communications, Bioinformatics, and Nucleic Acids Research. His research is characterized by a unique ability to develop sophisticated algorithms that address complex biological questions, from metabolic pathway reconstruction to the design of new molecular entities.
Dr. Yamanishi's scientific excellence has been recognized with multiple prestigious honors for his contributions to drug discovery and preventive healthcare. As a highly sought-after expert, he frequently deliver presentations at major international symposia, including ISMB and ECCB. Beyond his research, he is a dedicated mentor and an advocate for the digital transformation of the biomedical research landscape. His ongoing commitment to advancing informatics continues to shape the future of healthcare, ensuring that data-driven breakthroughs are translated into effective therapeutic strategies for patients worldwide.

Education

PhD in Science	Kyoto University, Japan (2005)
Master of Science (MSc) in Environmental Science and Engineering	Okayama University, Japan (2001)
Bachelor of Science (BSc)	Okayama University, Japan (1999)
Postdoctoral Fellowship	École Nationale Supérieure des Mines, France (2005–2006)

Abstract of the lecture

In recent years, drug discovery has become increasingly difficult. Computational approaches are expected to promote the efficiency of drug development processes. Recent developments in biotechnology have contributed to the increase in the amounts of high-throughput data in the genome, transcriptome, proteome, interactome, phenome and diseasome. These biomedical big data can be useful resources for drug development processes. Machine learning methods are expected to play key roles in the big data analysis. In this study, we developed novel machine learning methods to predict therapeutic targets of diseases, to search for drug candidate molecules, and to design new chemical structures of drug candidate molecules, by integrating various biomedical data on compounds (e.g., chemical structures, clinical phenotypes, gene expression patterns, target molecules) and diseases (e.g., disease-causing genes, disordered pathways, environmental factors, and clinical information). A unique feature of our data-driven approach is that it clarifies all the potential target proteins (including off-targets) of compounds, estimates the mechanisms of action at the pathway levels, and generates molecular structures of drug candidate compounds autonomically. In my talk at the conference, we will show some of the applications to therapeutic target identification, compound screening, polypharmacology therapy, and drug molecular structure design for a variety of diseases including cancers.

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Access Map

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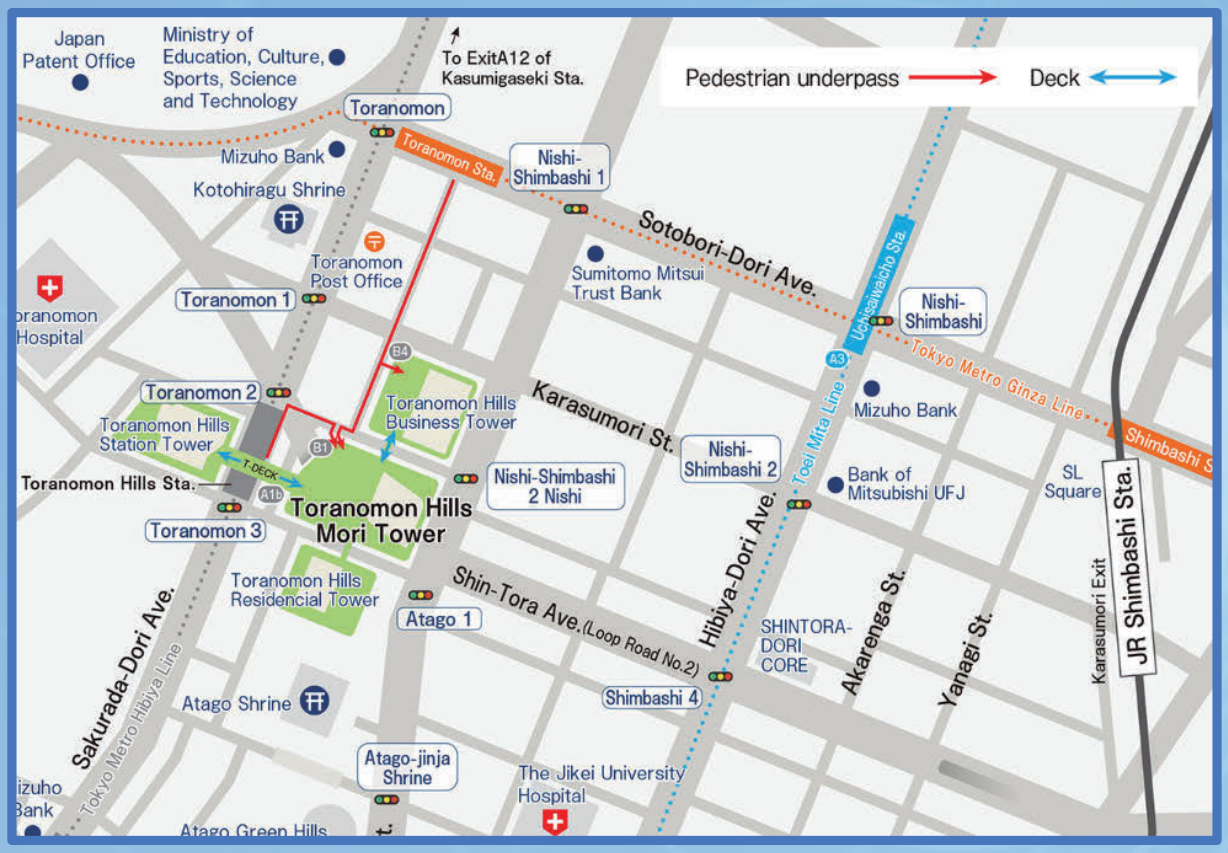

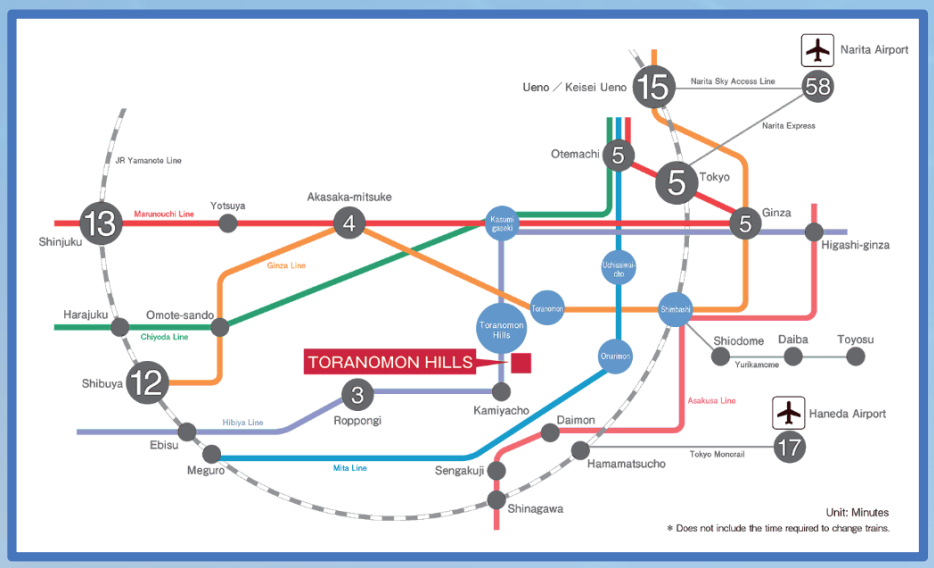

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