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P4Health

NAWA

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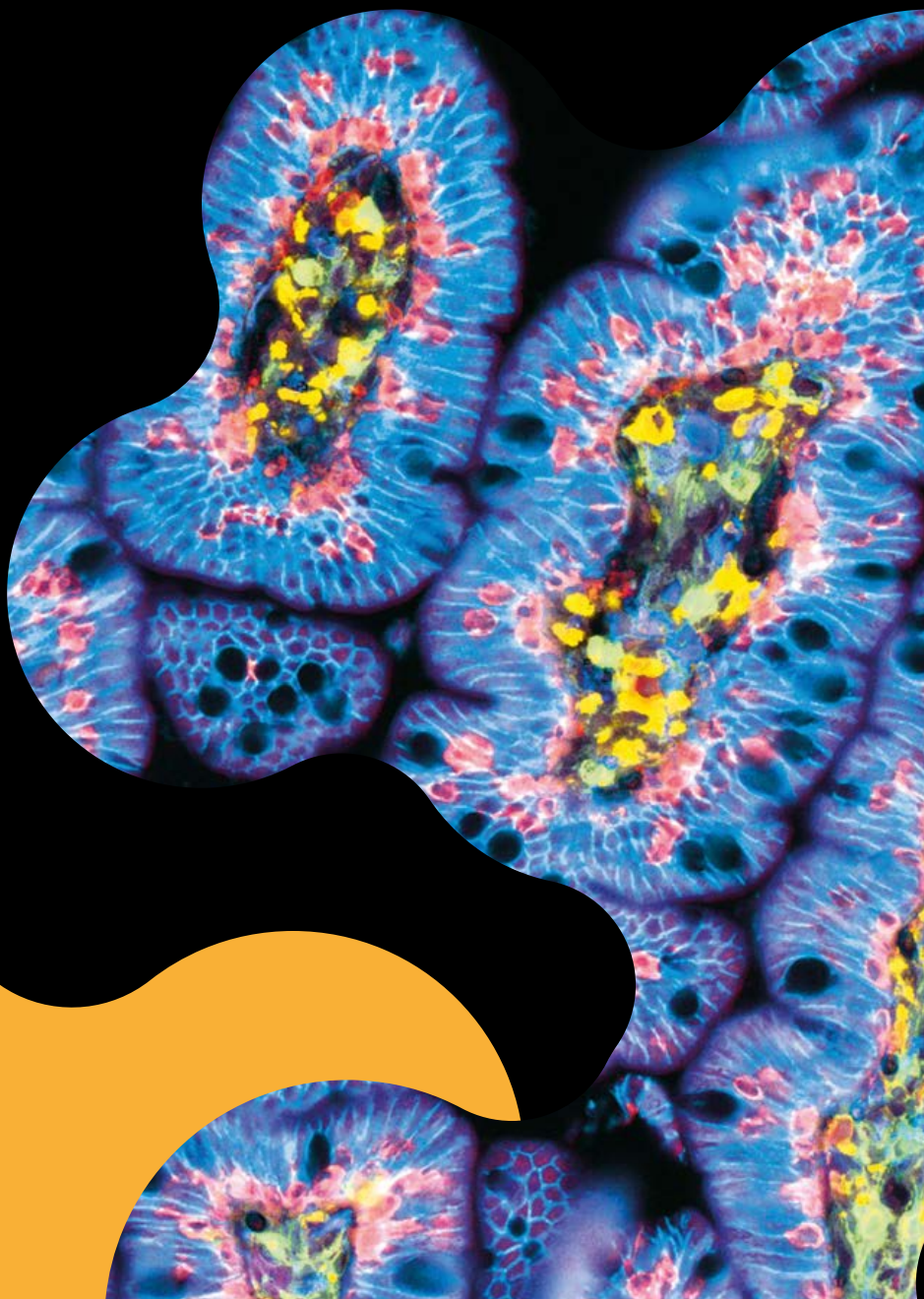
MAY 28-29, 2026
Wrocław, Poland

PORT for Health Oncology 2026



PORT FOR HEALTH ONCOLOGY

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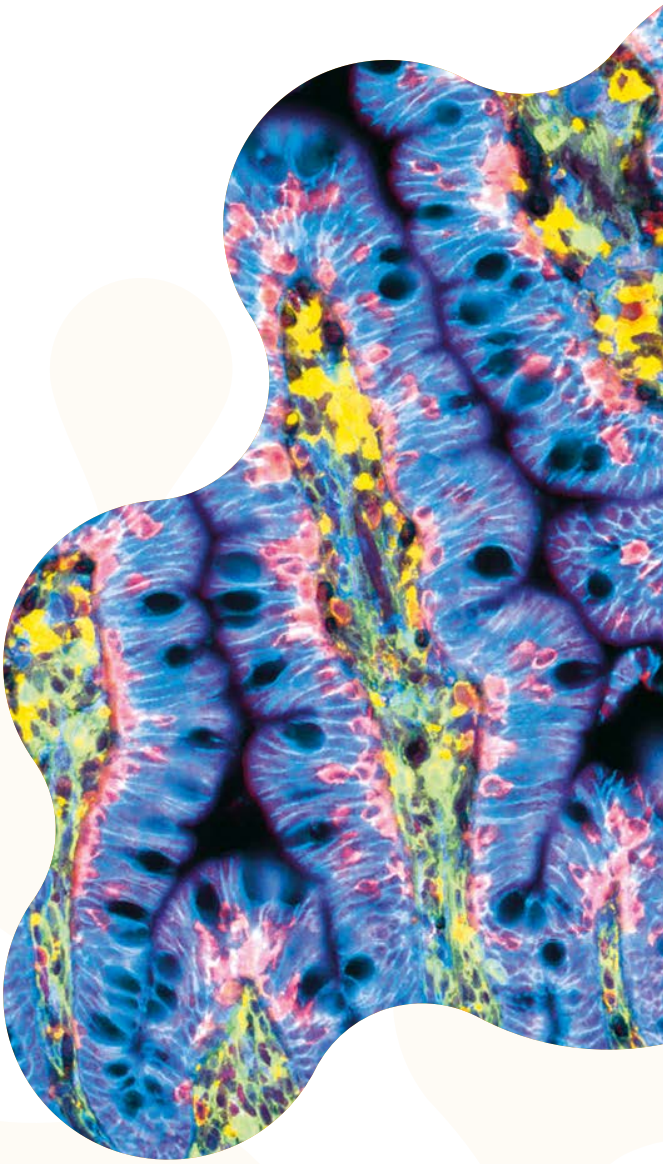
1. PORT FOR HEALTH CONFERENCE



PORT for Health Conference is an annual event organized by Łukasiewicz – PORT since 2021. The conference series brings together scientists, clinicians, industry representatives, and technology experts to **discuss current challenges and advances in biomedical research and healthcare**. Each edition is dedicated to a specific scientific theme with direct implications for human health, following a biennial cycle alternating between neuroscience and oncology. Over the years, PORT for Health has become a permanent element of the Łukasiewicz – PORT scientific and educational agenda, creating a platform for interdisciplinary dialogue, knowledge exchange, and international collaboration.

Today, PORT for Health is evolving into a recognized European forum for intensive cross-sector exchange, connecting **academic research, clinical practice, biotechnology, and industry**. The conference serves not only as a scientific meeting, but also as a **strategic environment for translating research outcomes into practical applications, fostering technology transfer, and expanding networks of industrial and institutional collaborators**. Previous editions attracted outstanding international participants and contributed to the development of international scientific consortia and research partnerships, including collaborations with leading biomedical and pharmaceutical organizations.

PORT for Health: Oncology 2026 is an international scientific conference dedicated to the **latest advances in cancer research and their translation into clinical practice**. The conference explores emerging directions in oncology, including **tumor biology, immunotherapy, neuro-oncology, precision medicine, AI-supported biomedical technologies, and translational research**. Bringing together leading researchers, physicians, innovators, and industry professionals, the event creates a space for scientific discussion, exchange of expertise, and development of new collaborations across disciplines and sectors.

health.port.org.pl

2. ŁUKASIEWICZ – PORT

The conference is organized by **Łukasiewicz – PORT – a modern interdisciplinary research and development center** based in Wrocław, Poland. Łukasiewicz – PORT conducts advanced research in the areas of **health, biotechnology, engineering, and materials science**, with a strong emphasis on **translating scientific discoveries into practical applications**. Its activities combine **fundamental and applied research, collaboration with hospitals and clinicians, development of digital health solutions, bioengineering, immunotherapies, and biobanking infrastructure**.

port.lukasiewicz.gov.pl

3. PORT FOR BUSINESS

An important component of the conference ecosystem is PORT for Business – a dedicated brokerage session organized as a satellite event to PORT for Health. The initiative was created to **strengthen collaboration between academia, healthcare, biotechnology companies, investors, and technology partners**. PORT for Business provides a structured environment for discussing **commercialization pathways, validating emerging technologies with industry stakeholders, identifying implementation challenges, and building long-term strategic partnerships**.

4. P4HEALTH CENTER OF EXCELLENCE FOR PRECISE PHENOTYPING AND BIODATABANKING

PORT for Health: Oncology 2026 is also part of the P4Health initiative – one of the most ambitious precision medicine projects currently developed in Europe. P4Health is establishing a Center of Excellence dedicated to precise phenotyping and biological data banking, designed to **accelerate the development of personalized medicine through advanced technologies, integrative data analysis, and international scientific collaboration**.

The initiative brings together expertise from Poland, the United Kingdom, and France to **develop precise diagnostic and therapeutic approaches tailored to the individual needs of patients**. By combining biomedical research, clinical data, advanced analytics, and translational infrastructure, P4Health aims to **transform personalized medicine** from a predominantly research-driven concept into a practical and scalable component of future healthcare systems.

p4health.eu

5. WROCŁAW

Wrocław is a vibrant metropolis in southwestern Poland. Its rich history – where diverse traditions, cultures, and religions intertwine – blends seamlessly with the dynamic present of a rapidly developing city.

Wrocław (a city with county rights) is the capital of the Lower Silesian Voivodeship. Situated on the Oder River, it is a unique city of 12 islands and 112 bridges. In the heart of the city, strollers are drawn to the Old Town Promenade stretching along the moat and the Botanical Garden located on Ostrów Tumski. The green riverside boulevards can also be admired from the decks of cruise ships and gondolas, which are among Wrocław's many tourist attractions.

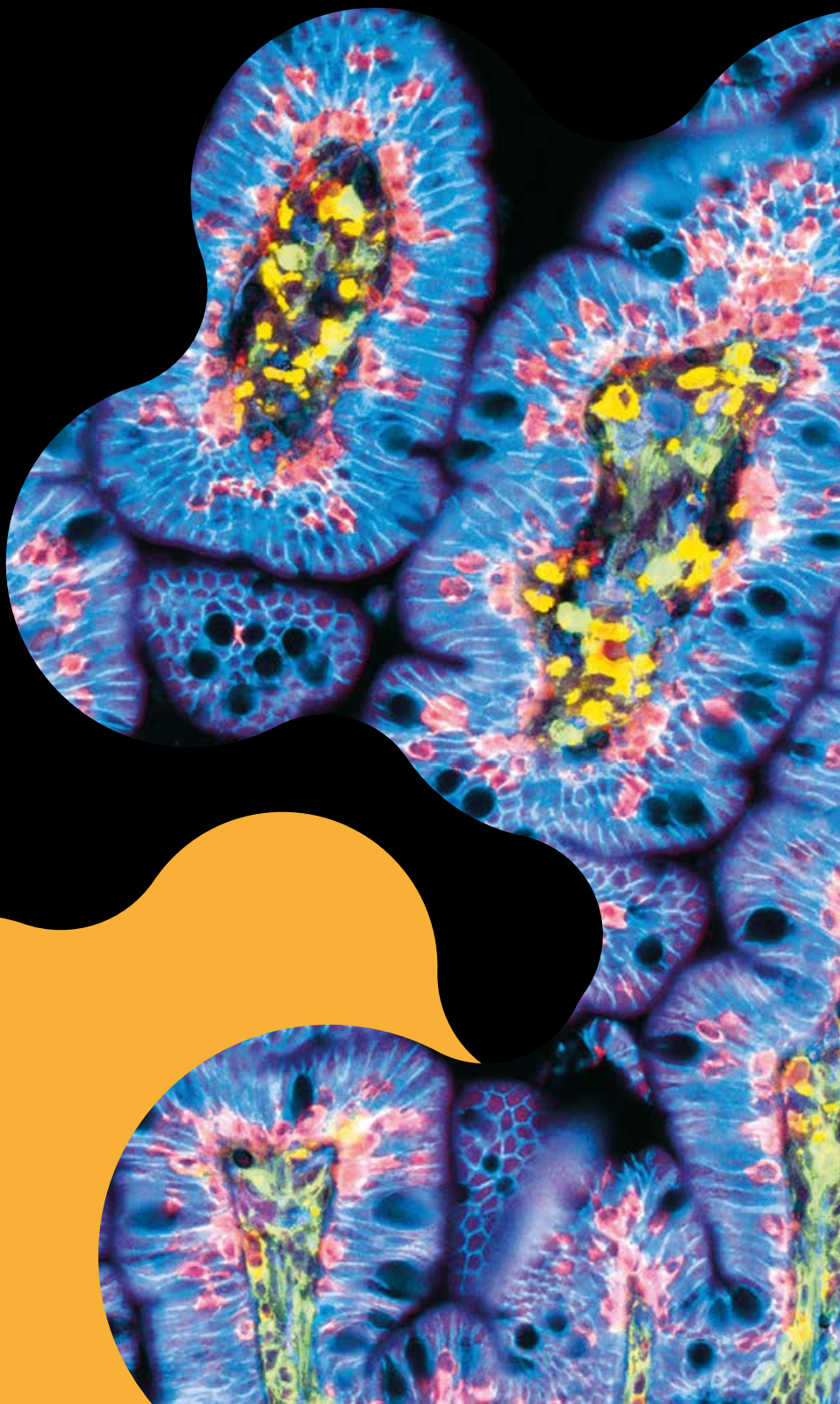
As the capital of the Lower Silesian Voivodeship, Wrocław has become an **important center for biomedical research, clinical collaboration, and interdisciplinary science**. The city hosts a dynamic scientific community spanning **oncology, immunology, neuroscience, molecular biology, biotechnology, and medical engineering**. The city is home to several universities and research institutions involved in brain and health-related studies, including the Łukasiewicz Research Network – PORT Polish Center for Technology Development, Wrocław University of Science and Technology (Politechnika Wrocławska), University of Wrocław, Wrocław Medical University, University of Environmental and Life Sciences (UPr), the University School of Physical Education (AWF), and the Hirszfeld Institute of Immunology and Experimental Therapy. Together, these institutions create **strong opportunities for translational research** and collaboration between academia, clinicians, and industry.

Honorary Patronage:

Strategic Partner:

Sponsors:

PROGRAM



PORT FOR
HEALTH
ONCOLOGY

Thursday 28th May 2026

9.00—9.15 **Official Opening**

CANCER BIOLOGY

CHAIR: **MAREK WAGNER**

9.15—9.45 **Aurélie Poli**

Luxembourg Institute of Health, Luxembourg

AllergoOncology: Lessons Learned from the Allergy-Glioblastoma Connection

9.45—10.15 **Joanna Poźniak**

KU Leuven, Belgium

Cytotoxic NK Cells Impede Response to Checkpoint Immunotherapy in Melanoma with an Immune-Excluded Phenotype

10.15—10.45 **Camille Chatelain**

Lund University, Sweden

Inducing Immunogenic Tertiary Lymphoid Structures Across Cancer Types With Dendritic Cell Reprogramming

10.45—11.15 **Break**

11.15—11.45 **Tim Halim**

Cancer Research UK Cambridge Institute, United Kingdom

The Role of ILC2 in Tissue Homeostasis and Neoplasia

11.45—12.15 **Beatriz German**

IGBMC, France

Analysis of the Chromatin Accessibility Landscape in Patients with Localized Prostate Cancer

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CANCER NEUROSCIENCE

CHAIR: **MATEUSZ KUCHARCZYK**

12.15—12.45 **Andrew Shepherd**

University of Texas MD Anderson Cancer Center, USA

Latent Neuropathy in Colorectal Cancer: Implications for Cancer Survivorship

12.45—13.15 **Christina Møller Andreasen**

University of Southern Denmark, Denmark

Remodelling of the Bone Microenvironment During Cancer Infiltration: Insights from Multiplex Imaging and Spatial Transcriptomics

13.15—14.15 **Lunch Break**

14.15—14.45 **Christoph Klose**

Charité – Universitätsmedizin Berlin, Germany

Enteric Nervous System-Derived VIP Restrains Differentiation of LGR5+ Stem Cells Towards the Secretory Lineage Impeding Type 2 Immune Programs

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CANCER THERAPY

CHAIR: **GRZEGORZ CHODACZEK**

14.45—15.15 **Tobias Bald**

University of Bonn, Germany

Targeting the Dark Matter of Cancer with AI-Designed Mini Binder

15.15—15.45 **Helena Florindo**

University of Lisbon, Portugal

Engineering Nanomedicines for Targeted Neuroimmune Modulation

15.45—16.00 **Monika Ślęzak**

Industry Contact Point (Łukasiewicz – PORT), Poland

Strategic Priorities in Cancer Therapy: Navigating the 2026 Cancer Mission Calls

16.00—16.30 **Break**

16.30—17.30 KEYNOTE: **Frank Winkler**

CHAIR: **Mateusz Kucharczyk**

Universitätsklinik Heidelberg, Germany

Cancer Neuroscience of Brain Tumors

19.00 Conference Networking Event

Official Dinner

Przystań & Marina Restaurant

Księcia Witolda 2, 50–202 Wrocław

INVITED GUESTS ONLY

Friday 29th May 2026

CANCER THERAPY

CHAIR: **PATRYCJA GAZIŃSKA**

9.00—9.30 **Sheeba Irshad**

King's College London, United Kingdom

Spatial Reprogramming of Immune Surveillance in Breast Cancer:

From Immune Control to Immune Failure

9.30—10.00

Stefaan Van Gool

IOZK Immun-Onkologisches Zentrum Köln, Germany

The War Against Glioblastoma Needs More Than Standard of Care

10.00—10.30

Jürgen Kuball

University Medical Center Utrecht, The Netherlands

Uncovering the Spatial Regulation of γδ T Cells: Toward Receptor-Guided Immunotherapy

10.30—11.00

Break

11.00—11.30

Wojciech Szlasa

Lower Silesia Center for Oncology, Pulmonology and Hematology, Poland

CAR-T Cell Therapy in Lymphomas, Acute Lymphoblastic Leukemia, and Multiple Myeloma

11.30—12.00

Sébastien Wälchli (online)

Oslo University Hospital, Norway

Expanding CAR Targets to Non Protein Antigens

12.00—12.30

Gabri van der Pluijm

Leiden University Medical Center, The Netherlands

Potentiating Immunotherapy of Urological Cancers with Oncolytic Viruses

12.30—13.00 **Helen Kakkassery**

King's College London, United Kingdom

Advancing BIA-ALCL Research Through a UK–PORT Alliance—From Biobanking to Immune Discovery

13.00—14.00 **Lunch Break**

PORT FOR BUSINESS
Company Session

CHAIR: **GRZEGORZ CHODACZEK**

14.00—14.10 **Marek Sipowicz**

WPD Pharmaceuticals, Poland

Beyond glioblastoma—WPD Pharmaceuticals

14.10—14.20 **Zbigniew Zasłona**

Molecure, Poland

The Development of USP7 Inhibitor for Cancer Immunotherapy

14.20—14.30 **Milena Mazan**

Ryvu Therapeutics, Poland

Leveraging Cancer Biology for Therapeutic Innovation: Clinical and Discovery Advances at Ryvu

14.30—14.40 **Justyna Adamyczk**

Enamine, Poland

Enabling Early Drug Discovery: Integrated Screening Capabilities and a BRD4/CRBN PROTAC Case Study

14.40—14.50 **Marek Kudła**

Ardigen, Poland

Transforming Multimodal Complexity into Precision Oncology Insights

14.50—15.00 **Artur Wnorowski**

Biotechna, Poland

Synergistic Nanotechnology for Targeted Therapeutics in Oncology

15.00—15.10 **Agata Drewniak-Maksymów**

JJP Biologics, Poland

JJP-1008 as a Novel Checkpoint Inhibitor

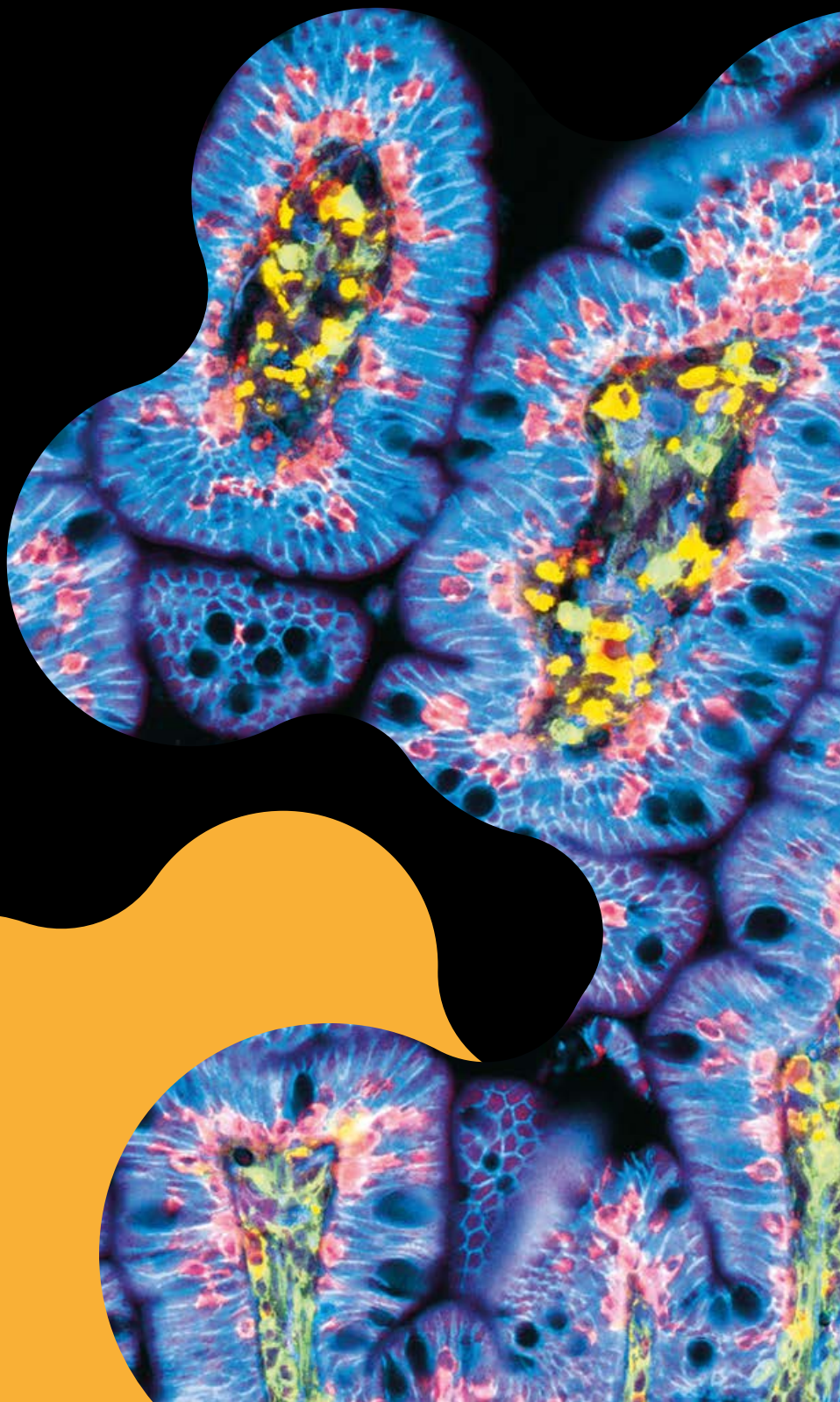
15.10—15.20 **Jakub Knurek**

Mabion, Poland

Development of Biological Drugs for Oncological Indications at Mabion

15.20—15.30 **Official Closure**

SPEAKERS



PORT FOR
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ONCOLOGY

CANCER BIOLOGY

Aurélie Poli

Luxembourg Institute of Health, Luxembourg

BIO:

Highly dedicated and creative scientist, my ambition is to make breakthrough in deciphering how to circumvent glioma mediated local and systemic immunosuppression and to translate our discoveries into more effective immunotherapeutic strategy to support patients' outcomes.

In 2005, after obtaining my Master's Degree in Biotechnology, I started as a research engineer in Dr. J. Zimmer group at the CRP-Santé, Luxembourg, the predecessor to the Luxembourg Institute of Health, Luxembourg (LIH). The research group interest focused on the biology of Natural Killer (NK) cells in both the context of TAP deficiency and their regulation by neurotrophic factors. I developed a strong background in immunology with a specialization in NK cell biology.

TITLE:

AllergoOncology: Lessons Learned from the Allergy-Glioblastoma Connection

ABSTRACT:

This presentation delves into the fascinating intersection of allergy and oncology, with a particular focus on glioblastoma. We will define the field of AllergoOncology, describing the current

interface between allergology and oncology and discussing various pathways involved in the association between allergy and glioblastoma.

We will delve into our recent work demonstrating the protective role of allergic airway inflammation against glioblastoma progression in a murine model, and its implications on systemic and local immunity. Investigating how allergic inflammation delays glioblastoma progression in mice reveals intricate immune mechanisms, including the transcriptional reprogramming of microglia and the potentiation of local and adaptive systemic immunity (Doi: 10.1111/all.15545) .

Additionally, we will discuss our collaborative efforts between the European Academy of Allergy and Clinical Immunology (EAACI) and the European Association of Neuro-Oncology (EANO), aiming to refine the classification of allergic diseases and gliomas for enhanced research in AllergoOncology (Doi: 10.1111/all.15994).

Through these discussions, we aim to provide a comprehensive understanding of the AllergoOncology nexus, emphasizing the importance of preclinical models, biomarker research, and collaborative efforts in advancing our knowledge and improving patient outcomes in this intriguing field.

Joanna Poźniak

KU Leuven, Belgium

BIO:

Joanna holds a Bachelor's degree in Molecular Biology and an international Master's degree in Biotechnology from Adam Mickiewicz University in Poznań, Poland. She completed her PhD at the University of Leeds as part of the Marie Skłodowska-Curie Early Training Network (MELGEN), where her research focused on characterizing immune infiltration across a large cohort of primary melanoma transcriptomes using bioinformatics and statistical approaches.

She is currently a Senior Postdoctoral Researcher in Bioinformatics at the Marine Lab (VIB–KU Leuven, Belgium), supported by Marie Skłodowska-Curie and STK Belgian fellowships. Her work focuses on melanoma heterogeneity and responses to immunotherapy, employing single-cell sequencing and spatial transcriptomics. Her contributions have been recognized with the prestigious Society for Melanoma Research (SMR) Young Investigator Award.

TITLE:

Cytotoxic NK Cells Impede Response to Checkpoint Immunotherapy in Melanoma with an Immune-Excluded Phenotype

ABSTRACT:

Immune checkpoint blockade (ICB) targeting PD-1 has transformed melanoma therapy, yet half of patients do not benefit, often due to unclear resistance mechanisms. Immune

exclusion-the inability of lymphocytes to enter the tumor nest-is a key barrier, and studies using only baseline or late post-treatment samples may miss early immune dynamics.

We hypothesized that treatment-induced changes occur after a single ICB cycle. In an interventional study, we profiled melanoma with single-cell RNA-seq, analyzing 46 tumor samples from 23 patients, including 20 matched pre – and early on-treatment biopsies. We identified 20 immune cell types and linked their abundance to response. Consistent with prior work, responders were enriched for CD8+ T cell subsets, particularly CD8+ CXCL13+ T cells, at both timepoints. Surprisingly, cytotoxic natural killer (NK) cells were significantly enriched in non-responders early on treatment, validated in two independent melanoma cohorts and one breast cancer scRNA-seq cohort.

Spatial transcriptomic and proteomic analyses from the same lesions showed that in non-responders, NK and CD8+ T cells co-accumulated at the tumor rim but failed to penetrate the core, consistent with immune exclusion; responding tumors showed co-localization within the parenchyma. In an immune-excluded mouse melanoma model, pharmacologic NK cell depletion enhanced CD8+ T cell infiltration into the core and enabled tumor clearance with anti-PD-1. Mechanistically, NK cells were recruited to excluded regions via CX3CR1 and constrained CD8+ T cell infiltration and function. These findings identify an unexpected NK cell–mediated resistance axis and suggest targeting NK recruitment or activity to overcome immune exclusion and improve ICB responses.

Camille Chatelain

Lund University, Sweden

BIO:

Camille Chatelain is a Marie Skłodowska-Curie postdoctoral researcher in the Pereira lab in Lund Stem Cell Center (LSCC, Sweden). Her research centers on remodeling the tumor microenvironment with immunotherapies.

During her PhD in the Research Center for Cancerology and Integrated Immunology of Nantes-Angers (CRCI2NA, France), she investigated the use of oncolytic viruses to modulate tumor-associated macrophages toward anti-tumor phenotypes. She then secured a Marie Skłodowska-Curie Action grant to join Filipe Pereira's team in Sweden.

TITLE:

Inducing Immunogenic Tertiary Lymphoid Structures Across Cancer Types With Dendritic Cell Reprogramming

ABSTRACT:

Tertiary lymphoid structures (TLS) in tumors are associated with improved responses to immunotherapy, yet their controlled induction remains elusive. We have developed an approach reprogramming tumor cells in vivo into type-1 conventional dendritic cell (cDC1)–like cells to rewire the tumor microenvironment and initiate de novo formation of immunogenic TLS (imgTLS) in melanoma, colon, bladder, breast and pancreatic cancer across different genetic backgrounds.

cDC1-like cells activated lymphotoxin, TNF, and interferon programs, engaged early with T cells and required CD40 signaling to induce mature imgTLS containing BCL6^{+} germinal centers, independently of endogenous cDC1. Spatial transcriptomics revealed DC-LAMP+ CCR7^{+} migratory cDC1 programs, nucleating immune niches enriched in CD4^{+}, CD8^{+} T cells, and B cells. ImgTLS sustained B cell-supported T cell responses and promoted systemic immunity by clonal expansion in abscopal tumors.

Our study place cDC1 at the apex of TLS induction and establish cellular reprogramming as a generalizable mechanism to engineer lymphoid niches within tumors and overcome resistance to immunotherapy.

Tim Halim

Cancer Research UK Cambridge Institute, United Kingdom

BIO:

Dr. Halim is a Group Leader at the Cancer Research UK Cambridge Institute, University of Cambridge. He obtained his PhD in 2013 from the University of British Columbia (Canada), after which he moved to the laboratory of Dr. Andrew McKenzie at the MRC Laboratory of Molecular Biology (UK) on a CIHR Banting Fellowship. He joined the CRUK-CI faculty in 2017 with additional support from a Royal Society/ Wellcome Trust Sir Henry Dale Fellowship.

In early 2024 Dr. Tim Halim was awarded £3.3 million through the Wellcome Trust Discovery scheme to explore how immune cells called 'type-2 lymphocytes' influence the behaviour of fibroblasts, particularly during health and disease states. The Wellcome Discovery Award provides funding for established researchers to pursue bold and creative research ideas and deliver significant shifts in understanding related to human life, health and wellbeing.

The Halim Group will investigate the project titled "Regulatory roles of type-2 lymphocytes on local fibroblast biology in health and disease". The Halim laboratory focusses on innate lymphoid cell (ILC) biology, and how innate immunity interacts locally with immune and non-immune cells in the pancreas. Their goal is to elucidate how the local environment influences both disease and normal tissue behaviour, with an aim to exploit these organ-specific features for more targeted drug interventions.

TITLE:

The Role of ILC2 in Tissue Homeostasis and Neoplasia

ABSTRACT:

ILC2 are tissue-resident immune cells that play an important role in regulating type-2 inflammation. Beyond driving inflammatory responses, ILC2 are also emerging as regulators of tissue and immune homeostasis. We have shown that ILC2 directly interact with regulatory T cells to constrain the magnitude of the adaptive type-2 immune response. Moreover, we found ILC2 as important tissue-resident immune cells in the healthy pancreas, where they can interact with specific fibroblast subsets; by exerting both positive and negative selective pressures, ILC2 can orchestrate fibroblast homeostasis in healthy, inflamed and cancer conditions.

Beatriz German

IGBMC, France

BIO:

Beatriz Germán Falcón holds a Bachelor's degree in Pharmacy from Universidad San Jorge and an international Master's degree in Biomedical Research from University of Navarra, Spain. She completed her international PhD at the University of Strasbourg as part of the LabEX-INRT Program at Institut de Génétique et de Biologie Moléculaire et Cellulaire (IGBMC), where her research focused on the epithelial regulation of immune responses in inflammatory disorders and melanoma. She subsequently pursued postdoctoral training at IGBMC and later in the laboratory of Dr. Ellis at Cedars-Sinai Medical Center and the Center for Prostate Disease Research USA, where, supported by a Department of Defense Young Investigator Award and a Department of Defense Idea Award as co-Principal Investigator, she investigated the epigenetic and microenvironmental mechanisms driving prostate cancer plasticity, immune evasion, and therapeutic resistance.

She is currently a Senior Postdoctoral Researcher at Laverny's Lab (IGBMC) supported by an FRM Retour en France fellowship, where her research focuses on the epigenetic control of neutrophil-driven immune suppression in cancer.

TITLE:

Analysis of the Chromatin Accessibility Landscape in Patients with Localized Prostate Cancer

ABSTRACT:

Significant group-based variation in prostate cancer (PCa) outcomes remains a major global health challenge. African American (AA) men have the highest incidence and approximately double the mortality of Caucasian American (CA) men. Although disparities in healthcare access contribute, worse outcomes persist even with comparable care, suggesting biological differences in tumor behavior. While emerging studies have begun to define epigenomic alterations in PCa, the contribution of large-scale chromatin remodeling to population-level outcome differences remains unclear.

To address this, we profiled localized PCa from AA and CA men using single-cell spatial ATAC-seq and H3K27ac spatial CUT&Tag on prostatectomy specimens from the Center for Prostate Disease Research/Walter Reed National Military Medical Center. Machine learning–based annotation (Cellcano) enabled identification of lineage-specific chromatin states. Furthermore, AA demonstrated significant lower accessible chromatin associated with CCCTCbinding factor (CTCF) which is a critical zinc finger protein crucial for regulating gene expression, epigenetic marks, and maintaining 3-dimensional genome structure.

We have validated this finding by CTCF immunohistochemistry in an independent TMA that includes 99 patients with distribution of CA and AA prostatectomy samples. Moreover, integration of H3K27ac profiling revealed several differentially regulated enhancer regions near specific genes across samples. Collectively, our current findings provide critical insights into the divergent chromatin accessibility profiles between AA and CA men with localized PCa, which likely underlie distinct transcriptional responses that can determine therapeutic resistance and tumor progression. These data shed light on the complex epigenomic mechanisms driving the group-based variations in PCa outcomes and may inform the development better treatment strategies.

CANCER NEUROSCIENCE

Andrew Shepherd

University of Texas MD Anderson Cancer Center, USA

BIO:

Dr. Andrew Shepherd conducts preclinical research investigating the mechanisms that link inflammation to the pain induced by various forms of injury, including joint damage and chemotherapy-induced neuropathy, with a particular emphasis on signaling related to the renin-angiotensin system. The ultimate goal of the Shepherd Lab in the Department of Symptom Research is to identify interventions that can mitigate or prevent the development of pain associated with these chronic disease states.

Dr. Shepherd earned his bachelor's degree in molecular cell biology in 2003 and a doctoral degree in neuroimmunomodulation in 2006, both from the University of Manchester in Great Britain. His training and expertise focus on rodent models of pain, behavioral readouts of pain sensitivity, and assays of primary rodent and human sensory-neuron function.

Dr. Shepherd co-chairs MD Anderson's Pain Research Consortium, a unique platform that brings together clinical and basic science pain experts for interdisciplinary discussion, innovative problem-solving, and shaping the future of pain management.

TITLE:

Latent Neuropathy in Colorectal Cancer: Implications for Cancer Survivorship

ABSTRACT:

Colorectal cancer survivors are at increased risk of developing neurological issues, particularly peripheral neuropathy and chronic pain. Although pre-existing neuropathy is a risk factor for chronic pain, tumor-induced neuropathy has not been firmly established in pre-clinical models. Consistent with clinical observations, we show that mice with colorectal cancer develop peripheral neuropathy, which was associated with subtle locomotor deficits, without overt hypersensitivity.

We detected widespread differences in pro-inflammatory cytokines and lipid metabolites in peripheral nerves from tumor-bearing mice. Macrophage accumulation, myelin decompaction and ryanodine receptor oxidation were associated with dysfunctional calcium homeostasis and reduced spike amplitude in sensory neurons. Similar alterations in plasma inflammatory mediators and lipid metabolites were associated with neuropathy and macrophage accumulation in peripheral nerves of rhesus macaques with colorectal cancer.

Consistently, mice with colorectal cancer exhibit greater pain sensitivity following treatment with oxaliplatin. These findings suggest colorectal cancer is causally linked to a subacute form of chronic inflammatory demyelinating polyneuropathy across species, which may represent an under-reported, yet important risk factor for neurological dysfunction in colorectal cancer survivors.

Christina Møller Andreasen

University of Southern Denmark, Denmark

BIO:

Dr. Moeller Andreasen is an Associate Professor, PhD, at the Research Unit of Pathology, Department of Clinical Research, University of Southern Denmark, where she leads the Bone & Cancer Research team investigating bone metastases in breast and prostate cancer.

Her research focuses on the cellular and molecular dynamics of the bone microenvironment during metastatic progression, with particular emphasis on how tumor–stroma interactions shape metastatic colonization, dormancy, and therapeutic resistance. She is a current member of the Academy of the European Calcified Tissue Society, actively contributing to bone research and mentoring within the European scientific community.

TITLE:

Remodelling of the Bone Microenvironment During Cancer Infiltration: Insights from Multiplex Imaging and Spatial Transcriptomics

ABSTRACT:

Bone is a dynamic tissue shaped by continuous interactions among osteoclasts, osteoblasts, stromal cells, immune populations, and the surrounding extracellular matrix. When cancer cells infiltrate bone, this finely balanced microenvironment undergoes extensive structural and molecular remodelling that supports metastatic growth.

This presentation uses principles of normal bone remodelling as a foundation for understanding how tumor cells reshape the bone niche during metastasis. Drawing on patient-derived samples primarily from breast and prostate cancer, the talk showcases multiplex imaging and spatial transcriptomics data that map interactions between cancer cells, fibroblasts, vasculature, nerves, and other key components of the metastatic microenvironment.

The session will also include practical guidance on pre-analytical tissue handling to ensure high-quality samples for spatial transcriptomics. Together, these insights highlight how spatially resolved technologies can deepen our understanding of bone metastasis biology in cancer.

Christoph Klose

Charité – Universitätsmedizin Berlin, Germany

BIO:

Christoph Klose earned his PhD in Molecular Medicine from Albert-Ludwigs-University in Freiburg in 2007. Following his doctoral studies, he conducted post-doctoral research at Weill Cornell Medicine in New York City before establishing an independent Emmy-Noether research group at the Department of Microbiology, Infectious Diseases, and Immunology of Charité—Universitätsmedizin Berlin.

Dr. Klose's research has significantly contributed to our understanding of immune cell lineage commitment and development, with a particular focus on innate lymphoid cells (ILCs).

In recognition of his contributions, he was awarded the Robert Koch post-doctoral Immunology award in 2015 and has been listed as a Highly Cited Researcher by Clarivate Analytics since 2020. The main focus areas of his research group are (1) The interaction between the immune system and the nervous system, particularly at barrier surfaces, and (2) the role of ILC2s in type 2 immunity.

TITLE:

Enteric Nervous System-Derived VIP Restrains Differentiation of LGR5+ Stem Cells Towards the Secretory Lineage Impeding Type 2 Immune Programs

ABSTRACT:

Intestinal epithelial homeostasis is sustained by the continuous differentiation of stem cells that are located at the bottom of the intestinal crypts. Epithelial renewal is a highly dynamic process that receives signaling input from various cellular systems to secure barrier function and nutrient uptake.

Here, we addressed the role of the enteric nervous system (ENS) in this process. We identify a pivotal function of the ENS in controlling epithelial proliferation, differentiation, and mucosal homeostasis. Neuronal Vasoactive Intestinal Peptide (VIP), acting via its receptor VIPR1 expressed by epithelial stem cells, restrains proliferation and differentiation towards the secretory lineage. Deficiency of VIP or VIPR1 led to an increase in secretory epithelial cells, including tuft cells, increased IL-25 expression, and activation of ILC2.

Functionally, VIP deficiency improved worm expulsion and exacerbated allergic lung inflammation. Our data expose a previously unappreciated role for the ENS in dictating epithelial cell fate decisions, thereby establishing a neuro-epithelial unit as a critical checkpoint for ILC2 and type 2 immunity, complementing the well-known regulators of barrier integrity and mucosal homeostasis, namely the commensal microbiota, the epithelial barrier, and the immune system.

Tobias Bald

University of Bonn, Germany

BIO:

Dr. Tobias Bald, serves as Professor for Tumor Immunobiology at the Institute for Experimental Oncology at the University Hospital Bonn, Germany. He obtained his PhD with highest honors from the Faculty of Medicine at the University of Bonn in 2015, focusing on the role of inflammatory responses in melanoma progression and the significance of the type I IFN system in cancer immunotherapy efficacy. Following the receipt of an EMBO Long-term Fellowship, Dr. Bald joined Dr. Mark Smyth's lab at the Queensland Institute for Medical Research, Brisbane, Australia, in 2016.

In 2018, he was granted an early career fellowship, enabling him to establish a junior group to investigate the impact of the Nectin-Network in the tumor microenvironment. In 2020, Dr. Bald became a member of the DFG cluster of excellence ImmunoSensation and founded the Laboratory for Tumor Immunobiology (www.baldlab.com) at the University Hospital Bonn. His research focuses on understanding the regulation of tumor-infiltrating immune cells to develop innovative cancer therapies.

TITLE:

Targeting the Dark Matter of Cancer with AI-Designed Mini Binder

ABSTRACT:

Directing chimeric antigen receptor (CAR) T cell immunotherapies against intracellular or secreted tumour specific neoantigens remains a significant challenge, limiting the potential cancer target repertoire clinically. To address this, we have leveraged AI-based protein design to generate peptide-MHC class I-specific minibinders that mimic T-cell receptor (TCR) recognition, integrated into a CAR platform. Using immunopeptidomics, we have a identified a HLA-A*02 restricted peptide derived from IGFBPL-1, frequently expressed by small-cell lung cancer cells.

To experimentally identify functional TCR-mimetic minibinders, we implemented a high-throughput library screening strategy. Selected TCR-mimetic minibinders were incorporated as binding modules into second generation CAR T cell constructs. Peptide-specific activation of T cells expressing IGFBPL1 minibinder-CAR was detected upon co-culture with IGFBPL1 peptide-pulsed HLA class I-matched target cells, demonstrating functional tumor recognition.

We subsequently demonstrated target specific activation of our IGBFPL1 minibinder-CAR against endogenous expression of IGFBPL1 peptide on multiple HLA-A*02 SCLC cell lines. Ongoing experiments are further characterizing CAR functionality and cytotoxicity, with optimisation continuing in both the refinement of the artificial IGFBPL1 specific minibinders and the optimal CAR receptor construct pairing.

Overall, this AI-based platform represents a significant advance in targeting previously inaccessible intracellular oncoproteins and neoantigens with CAR T cells, as well as potentially other modalities like T cell engagers in the future.

Helena Florindo

University of Lisbon, Portugal

BIO:

Helena Florindo graduated in Pharmaceutical Sciences in 2003 (University of Lisbon) and obtained her PhD degree in Pharmaceutical Technology in 2008 (University of Lisbon), in collaboration with the University of London.

Currently, she is a Full Professor in the Department of Pharmacy, Pharmacology, and Health Technologies at the Faculty of Pharmacy, University of Lisbon. Since 2015, she has been the head of the BioNanoSciences – Drug Delivery & Immunoengineering Research Group, at the Research Institute for Medicines (iMed. ULisboa), University of Lisbon.

Helena is also a member of the Portuguese Medicines Agency Evaluation Board (INFARMED) and an expert to the European Medicines Agency (EMA), thus supporting the evaluation of marketing authorization procedures for new drugs and biologics. This knowledge in regulatory sciences also guides the research within her research group, which has been motivated by the immune-oncology field toward the rational development of functionalized nanobiomaterials as novel immunotherapies for cancer treatment.

TITLE:

Engineering Nanomedicines for Targeted Neuroimmune Modulation

ABSTRACT:

Targeted neuro-immune modulation offers a promising strategy to counteract the profound immunosuppression characteristic of solid tumors. Although immune checkpoint inhibitors (ICI) have transformed cancer therapy, their efficacy remains limited in tumors with highly suppressive microenvironments and is frequently accompanied by immune-related toxicities. Breast cancer (BC), pancreatic ductal adenocarcinoma (PDAC), and melanoma exemplify tumors in which restricted immune-cell infiltration and potent immunosuppressive cues hinder therapeutic success. We present multifunctional nanomedicines designed to reprogram the tumor microenvironment (TME) and potentiate ICI efficacy. These nanoparticles (NP) were rationally designed to target dendritic cells (DC) and key immunosuppressive pathways by incorporating tumor-associated antigens, TLR ligands (CpG and Poly(I:C)), and regulators of dominant suppressive mediators within the TME. Surface engineering strategies enhanced DC activation and improved NP trafficking and accumulation in tumors. Cy5.5-labeled NP demonstrated efficient in vivo internalization by DC and induced strong upregulation of co-stimulatory molecules (CD80, CD86, CD40) compared with non-carbohydrate carriers.

NP immune-modulating and anti-tumor impact were validated ex vivo using patient-derived organoids and in vivo across melanoma, PDAC, and BC mouse models. Nanomedicines elicited potent antigen-specific immune responses, reshaped the immunological profile of the TME, and significantly inhibited tumor progression. Synergistic therapeutic benefits were observed when NP were combined with ICI, particularly αOX40. In 4T1 and E0771 tumor-bearing mice, this combination resulted in marked tumor regression and prolonged survival.

In summary, the engineered nanomedicine system effectively reprograms immune pathways within the TME and sensitizes solid tumors to immune checkpoint modulation, offering a powerful strategy to enhance cancer immunotherapy.

Monika Ślęzak

Industry Contact Point (Łukasiewicz – PORT), Poland

BIO:

Monika joined Łukasiewicz – PORT in March 2022 as Coordinator of the Industry Contact Point for Medical Technologies and Health. She has over 10 years of research experience in molecular and cellular biology within the drug and vaccine development field, with a particular focus on preclinical studies, including in vitro and animal models such as mice and zebrafish. She earned her PhD in 2009 from the Jagiellonian University in Kraków. Her doctoral dissertation focused on the genotoxic effects of acute carbon monoxide poisoning in humans. Since 2008, she has been involved in managing research projects funded by the European Union, including Horizon 2020, Horizon Europe, IMI, and IHI programs, serving as both project manager and research project leader. Since 2023, she has acted as an evaluator for competitions organized by the European Commission.

TITLE:

Strategic Priorities in Cancer Therapy: Navigating the 2026 Cancer Mission Calls

ABSTRACT:

The European Cancer Mission constitutes one of the most ambitious components of the EU research and innovation framework, aiming to accelerate translational oncology, improve

patient outcomes, and strengthen cross-border collaboration in cancer prevention, diagnosis, and therapy. The forthcoming 2026 Horizon Europe Cancer Mission calls introduce a new generation of funding priorities focused on precision medicine, advanced therapeutics, AI-supported oncology, clinical validation infrastructures, and implementation-oriented healthcare innovation.

This presentation provides a strategic overview of the 2026 Cancer Mission landscape with particular emphasis on opportunities related to cancer therapy development and deployment. The session will analyse expected thematic directions and policy drivers, as well as discuss practical aspects of competitive proposal preparation, including partnership strategies, stake-holder engagement and technology readiness considerations.

The session aims to support participants in effectively navigating the evolving Horizon Europe funding environment and identifying high-potential opportunities within the 2026 Cancer Mission portfolio.

KEYNOTE

Frank Winkler

Universitätsklinik Heidelberg, Germany

BIO:

Frank Winkler grew up in Hamburg, where he attended the Wilhelm-Gymnasium. After graduating from high school, he studied human medicine at the University of Hamburg with stays in Freiburg, Cape Town and London at the National Hospital for Neurology and Neurosurgery.

In 1999, he began his training at the Neurological Clinic, Großhadern Hospital of the Ludwig Maximilian University of Munich. From 2002 to 2004, he completed a post-doctoral programme at Harvard University. During this time, he conducted research on the influence of the vascular system on brain tumours.

In 2012, he was appointed professor of Experimental Neuro-Oncology at the Department of Neurology in Heidelberg, where he has been senior physician since 2014. His Experimental Neuro-Oncology research group is based at the German Cancer Research Centre (DKFZ) in Heidelberg. His wife Eva Winkler is a specialist in haematology/oncology at Heidelberg University Hospital and a member of the German Ethics Council.

The laboratory led by Frank Winkler has used neuroscience methods to develop a new understanding of malignant adult brain tumours, glioblastomas and brain metastases. Key discoveries from this work have helped to establish the new field of cancer neuroscience

research. These include malignant multicellular tumour networks that are highly functional and resilient and driven by developmental neurobiological factors, including pacemaker-like tumour cells in network nodes and excitatory synapses between brain neurons and various incurable brain tumour entities that drive brain tumour growth, invasion, and metastasis.

Frank Winkler has initiated clinical trials investigating how brain tumours in humans can be better controlled by disrupting neuro-cancer networks.

More information can be found on Wikipedia.

TITLE:

Cancer Neuroscience of Brain Tumors

ABSTRACT

Sheeba Irshad

King's College London, United Kingdom

BIO:

Professor Sheeba Irshad is a Clinician–Scientist in Cancer Immunology at King's College London and a Consultant Breast Cancer Medical Oncologist at Guy's and St Thomas' NHS Foundation Trust. She directs the Breast Cancer Now King's Research Unit, integrating immunology, oncology, and clinical-trial design to understand how immune dysfunction drives cancer progression and treatment failure.

Her research combines high-dimensional immune profiling, spatial and single-cell analysis to identify biomarkers of response and resistance and to guide immunotherapy development. Professor Irshad's team also investigates how ancestry, systemic stressors, and chronic inflammation influence immune outcomes and cancer disparities, and she co-leads Team SAMBAI, a Cancer Grand Challenge initiative exploring the biological basis of global cancer inequities.

She led the national SOAP study, which informed UK COVID-19 vaccine policy for cancer patients, and is Chief Investigator of the Outlier study examining exceptional survivors of metastatic cancer.

TITLE:

Spatial Reprogramming of Immune Surveillance in Breast Cancer: From Immune Control to Immune Failure

ABSTRACT:

Understanding how anti-tumour immunity is organised and disrupted within the tumour microenvironment remains a central challenge in breast cancer. In this study, we integrate spatially resolved imaging approaches across matched tumour and lymph node samples to define how immune surveillance is structured and how it fails during disease progression.

We identify distinct spatial immune niches associated with effective tumour control, characterised by coordinated localisation of B cells. In contrast, immune failure is marked by disrupted cellular architecture, altered immune cell trafficking, and the emergence of spatially restricted, dysfunctional immune states. Notably, we observe a reprogramming of B cell populations and their interactions with T cells, suggesting a critical role for humoral–cellular crosstalk in sustaining anti-tumour immunity.

By mapping these transitions across tumour and draining lymph node compartments, we propose a model in which immune surveillance is governed not only by cell composition but by spatial organisation and intercellular connectivity. These findings provide a framework for understanding resistance to treatments and highlight spatially defined immune interactions as potential therapeutic targets.

Stefaan Van Gool

IOZK Immun-Onkologisches Zentrum

Köln, Germany

BIO:

Stefaan Van Gool, MD, PhD is trained in pediatric neuro-oncology. He developed dendritic cell vaccines for glioblastoma, and treated the first patient in Europe in 2001. After an academic career, he became the medical director of the Praxis für Immunonkologie & Translationale Medizin.

TITLE:

The War Against Glioblastoma Needs More Than Standard of Care

ABSTRACT:

Improving overall survival for patients with Glioblastoma remains a challenge. We installed individualized multimodal immunotherapy as part of a multiphase combined treatment strategy (PMID 38548421). For the current real world data analysis, 104 patients were selected out of the database with the criteria: treatment after 27/05/2015, adults between 18 and 70y, GB diagnosis documented IDH1wt status, known status of OS, absence of second malignancy and known MGMT promoter methylation status (meth versus unmeth).

Sex distribution was 18 female / 25 male in meth patients versus 23 /38 in unmeth patients. Median age at intake was 54y versus 50y. The extent of resection was 12 R0, 28 < R0 and 3 not

available versus 25, 28 and 8. Median KPI at intake was 70 and 70. Patients received in median 37 versus 31 sessions of mEHT, 37 versus 32 injections of NDV and 2 versus 1 DC vaccines. Median OS and percentage 2y OS were 30 months and 69.8% in meth patients versus 20 months and 37.4% in unmeth patients.

There were no major adverse reactions (AR), but the burden of AR is increasing when using checkpoint inhibitors. The beneficial effect of this treatment strategy on OS and quality of life in a larger group of real world patients confirms earlier data. It remains unclear how to draw evidence out of individualized medicine, although it is felt necessarily for patients with Glioblastoma.

Jürgen Kuball

University Medical Center Utrecht,

The Netherlands

BIO:

Prof. Dr. Jürgen Kuball is Chair of the Department of Hematology, and Director of the Bone Marrow Transplantation Program at the UMC Utrecht Cancer Centre. His research activities focus on tumor immunology and the ATMP development of a next generation of engineered immune cells, with an emphasis on the therapeutic potential of individual receptors derived from γδ T cells. His clinical activities focus around acute leukemia and allogeneic stem cell transplantation. He is an active member of European Society for Blood and Marrow Transplantation (EBMT), and has chaired the Legal Regulatory Affairs Committee of the EBMT since 2019.

Prof. Kuball received his medical degree in Hematology at the University of Mainz, Germany, and completed his post-doctoral fellowship at the Fred Hutchinson Cancer Center in Seattle, WA, USA. He joined the Department of Hematology at the UMC Utrecht in 2007 as a hematologist and immunologist. In 2010, he became a VIDI-laureate, and the chair of the Applied & Tumor-Immunology section within the Laboratory of Translation Immunology. He has chaired the Department of Hematology (adults) since 2013.

TITLE:

Uncovering the Spatial Regulation of γδ T Cells: Toward Receptor-Guided Immunotherapy

ABSTRACT:

Gamma delta (γδ) T cells are a unique type of immune cell that can help the body detect and destroy cancer. However, their exact role in solid tumors has remained unclear, partly because traditional laboratory models do not fully reflect how these cells behave in the human body. In this talk, I will present a comprehensive strategy to study γδ T cells directly in patients with colorectal cancer, using matched samples from blood, healthy colon tissue, primary tumors, and liver metastases as well as novel engineering techniques by using advantage of γδ T cell receptors and optimal co-stimulation.

Wojciech Szlasa

Lower Silesia Center for Oncology, Pulmonology and Hematology, Poland

BIO:

Physician at the Hematology Department of DCOPIH, assistant at the Department of Molecular and Cellular Biology at the Wroclaw Medical University (UMW) in Wrocław. Conducts research on CAR-T cell therapy, bispecific antibodies, and the combination of radiotherapy with immunotherapy in hematological malignancies.

He gained experience in cell therapy during an internship in Würzburg—one of Europe's leading CAR-T centers—as well as through collaboration with the Université de Lorraine (Nancy), focusing on molecular modeling and gene electrotransfer across cancer cell membranes. He integrates experimental work with computational modeling and the translation of results into clinical hemato-oncology.

TITLE:

CAR-T Cell Therapy in Lymphomas, Acute Lymphoblastic Leukemia, and Multiple Myeloma

ABSTRACT:

CAR-T cell therapy represents one of the most important examples of adoptive immunotherapy successfully translated into modern oncology practice. The treatment is based on the collec-

tion of a patient's own T lymphocytes, their genetic modification to express a chimeric antigen receptor, and subsequent reinfusion after lymphodepleting chemotherapy. This approach enables T cells to recognize selected tumor-associated antigens and initiate a cytotoxic immune response independently of conventional HLA-mediated antigen presentation.

To date, the most established clinical role of CAR-T therapy has been in B-cell malignancies and multiple myeloma. In diffuse large B-cell lymphoma, CAR-T cells have become a standard therapeutic option for selected patients with relapsed or refractory disease, including those treated in the second-line setting after early relapse or primary refractoriness to immunochemotherapy. In mantle cell lymphoma, CAR-T therapy is used in heavily pretreated patients, particularly after failure of Bruton tyrosine kinase inhibitors. In B-cell acute lymphoblastic leukemia, CAR-T cells have enabled deep and durable remissions in patients with relapsed or refractory disease, including pediatric and young adult populations. In multiple myeloma, BCMA-directed CAR-T constructs have shown high response rates in patients previously exposed to multiple lines of therapy.

Despite its transformative efficacy, CAR-T cell therapy is associated with a distinct toxicity profile, including cytokine release syndrome, immune effector cell-associated neurotoxicity syndrome, prolonged cytopenias, infections, and hypogammaglobulinemia. Therefore, careful patient selection, timely referral, specialized center experience, and multidisciplinary collaboration are essential for safe and effective treatment delivery.

The development of CAR-T therapy illustrates how rapidly the boundaries between basic science, cellular engineering, translational research, and clinical oncology are evolving. It also highlights the need for integrated cooperation between clinicians, molecular diagnostics experts, immunologists, cell therapy laboratories, and healthcare systems to make advanced cellular therapies broadly accessible to patients.

Sébastien Wälchli

Oslo University Hospital, Norway

BIO:

Dr. Sébastien Wälchli is Group Leader at Oslo University Hospital, where he established a CAR discovery platform and has contributed to the development of over 20 CAR molecules, including CD37CAR, OSCAR, and K101CAR.

His work combines molecular engineering and T-cell biology to advance personalized cellular therapies, within national and EU-funded immunotherapy programs. He has co-founded Zelluna Immunotherapy, authored more than 100 publications, and holds 15 patents.

TITLE:

Expanding CAR Targets to Non Protein Antigens

ABSTRACT:

Accurate identification of tumor-specific markers is essential for advancing CAR-based therapies, especially in solid tumors where protein antigens rarely offer sufficient specificity. Post-translational modifications, particularly glycosylation, provide an alternative class of targets with improved selectivity. Among these, the sialyl-Tn (STn) antigen is widely expressed across epithelial cancers but largely absent in healthy tissues.

We present AM-series of monoclonal antibodies with unprecedented specificity for the STn and no detectable reactivity toward normal tissues. Incorporation of the AM52.1 scFv into a second-generation CAR design yielded AM52.1CAR T cells that selectively killed STn-expressing cancer cell lines and patient-derived organoids while sparing STn-negative controls. In multiple preclinical models—including gastric, tubo-ovarian, and colorectal mucinous peritoneal metastases—AM52.1CAR T cells demonstrated robust antitumor activity. These results highlight the promise of targeting non-protein antigens to expand CAR T-cell therapy into complex solid tumors.

Gabri van der Pluijm

Leiden University Medical Center,

The Netherlands

BIO:

Dr. Gabri van der Pluijm is Associate Professor at the Department of Urology, Leiden University Medical Center (Leiden, the Netherlands) at which he is heading the Urology Research Laboratory. His research is translational and ranges from 2D and 3D tumour-immune cell co-culture models in vitro to preclinical and 'near-patient' in vivo disease models for the study of the pathogenesis of tumour progression and experimental treatment of urological cancers (prostate, bladder and renal cancer).

His group is currently focusing on potentiating current immunotherapeutic approaches by oncolytic viruses and/or drug repurposing (cancer neuro-immunology), aiming at improving the treatment outcomes of patients with urological malignancies. Dr. van der Pluijm is (and has been) involved as coordinator and principal investigator in multiple national and international research consortia and research, including EU-sponsored European Training Networks. He is tutor of Medical and Biomedical Science students.

TITLE:

Potentiating Immunotherapy of Urological Cancers with Oncolytic Viruses

ABSTRACT:

Despite the promising effects of immunotherapy in other solid cancers, prostate cancer have remained largely unresponsive. In bladder cancer, immunotherapy has emerged as a promising therapeutic strategy but not all patients show clinically-desirable responses to immune-checkpoint inhibitors.

We hypothesize that oncolytic viruses (OVs) may unleash the full potential of cancer immunotherapy. OVs represent a promising therapeutic avenue, as OV-treatment combines tumour cell lysis with activation of the immune system and mounting of effective anti-tumour responses. For this, we evaluated and compared the oncolytic and immunostimulatory properties of multiple OVs , i.e. wildtype reovirus (R124) , a spontaneous mutant reovirus (jin-3) and a new oncolytic derivative of the Gorilla-derived Human AdenoVirus-B (HAdV-lumc007) 'GoraVir' in preclinical, 'near-patient' and syngeneic models of prostate and bladder cancer.

Both reoviruses variants and GoraVir effectively infected and lysed bladder and prostate cancer cells in 3D-cultures, ex-vivo cultured human tumour tissue slices and patient-derived xenograft (PDX) models. Notably, jin-3 particularly, induces a dose-dependent expression of immunogenic cell death markers, interferon-stimulated genes and inflammatory cytokines.

Additionally, co-culturing reovirus – and to a lesser extent GoraVir – infected bladder and prostate tumouroids with peripheral blood monocytes resulted in a significant and dose-dependent cancer cell lysis and elevated production of CXCL10 and IFNgamma. Administration of jin-3 to immunocompetent mice with a subcutaneously growing murine prostate cancer (TRAMP-C2) increased the infiltration of CD4+ and CD8+ effector cells in the tumour micro-environment resulting in tumor cell killing and cancer regression. Taken together, OVs elicit robust immuno-stimulatory responses highlighting their potential as anti-tumour agents.

Helen Kakkassery

King's College London, United Kingdom

BIO:

Helen Kakkassery is a Cancer Biology PhD Graduate, holds an MRes in Translational Cancer Medicine (Distinction), and earned a First-Class BSc (Hons) in Biomedical Science with a specialization in Immunology**.**

TITLE:

Advancing BIA-ALCL Research Through a UK–PORT Alliance—From Biobanking to Immune Discovery

ABSTRACT:

Breast implant-associated anaplastic large cell lymphoma (BIA-ALCL) is a rare T-cell lymphoma arising in the context of chronic inflammation, yet its systemic immune landscape remains poorly defined. This study aimed to characterise immune alterations in BIA-ALCL patients (n=22) compared with control cohorts, including implant-positive (BIA-ALCL–negative; n=15) and implant-negative (n=22) individuals. High-dimensional immune profiling and Olink-based proteomic analysis were performed to quantify circulating immune cell subsets and serum biomarkers across disease and health. Comparative analyses were conducted between BIA-ALCL, healthy donors, and control groups. In addition, in vitro assays were performed to assess the effects of smooth and textured implant shells on the immune repertoire of peripheral blood from implant-negative healthy donors.

High-dimensional flow cytometry reveals a divergent immune signature in BIA-ALCL differentiating from the control cohorts where a proinflammatory shift was observed. BIA-ALCL is characterised by a pro-inflammatory, memory-skewed immune phenotype, with reduced Th2 and increased Th1/Th17.1 polarisation in CD4 T cells, alongside loss of naïve and expansion of memory subsets across T and NK cells. Immune activation is dysregulated across CD4, CD8, γδ T cells and NK cells, with reduced CD25/CD69, variable CD30 expression, and context-dependent exhaustion, particularly enriched in Vδ1 γδ T cells.

Additionally, BIA-ALCL is characterised by impaired antigen presentation and innate remodelling, with altered B-cell maturation, reduced pDCs, and dysfunctional monocyte compartments. Collectively, these findings define an immunologically dysregulated systemic environment in BIA-ALCL, providing insight into the disease biology.

PORT FOR BUSINESS
Company Session

Marek Sipowicz

WPD Pharmaceuticals, POLAND

BIO:

Physician with over 20 years of experience in leading clinical trials across oncology (hematologic malignancies and solid tumors), neuropsychiatry, diabetes, and cardiovascular medicine.
A board-certified gynecologist trained in Poland, with a postdoctoral fellowship completed at the National Cancer Institute in Bethesda, USA.

A highly regarded expert in the pharmaceutical industry, with a track record of initiating and managing global Phase I–IV clinical trials, including large-scale mortality studies. Spent over two decades in leadership roles at Servier, an international pharmaceutical company, serving as Director of Clinical Operations in Oncology in France and Director of Clinical Research in Australia.

TITLE:

Beyond glioblastoma — WPD Pharmaceuticals

ABSTRACT:

WPD Pharmaceuticals is a clinical-stage biotechnology company focused on developing novel therapies for oncology with special emphasis on brain cancer. WPD-401 is a novel and potent

drug candidate coming from the collaboration between WPD Pharmaceuticals and Wake Forest University. It is a multivalent targeted cytotoxic drug conjugate, which is engineered to target four receptors concomitantly: interleukin 13 receptor alpha 2 (IL-13RA2) and 3 ephrin receptors (EphA2, EphA3 and EphB2), that are overexpressed in patients with glioblastoma (GBM), the most prevalent primary brain tumor of dismal prognosis.

The cytotoxic is a drug conjugate composed of targeting moieties and a microtubular inhibitor DM1. Four targeted receptors are expressed in nearly 100% of glioblastomas, thus, WPD-401 can target glioma cells in almost all the patient population while sparing normal brain. The potency of WPD-401 in treating GBM has been already validated in preclinical models including studies in dogs. A phase I clinical trial in dogs with spontaneous gliomas using a cocktail of ligand-bacterial cytotoxin conjugates that target IL-13RA2 and EphA2 receptors has shown great promise. Additionally, all 4 receptors targeted by WPD-401 are also expressed in various other solid tumors, including melanoma, pancreas, TNBC, colon cancer, kidney, lung or ovary.

In conclusion, WPD-401 is an innovative, highly effective drug candidate that has the potential to revolutionize treatment for high unmet medical needs in glioblastoma, TNBC and other indications.

Zbigniew Zasłona

Molecure, Poland

BIO:

Dr. Zbigniew Zasłona serves as Scientific Director at Molecure, where he leads the Biology department. He earned his PhD in 2010 at the University of Giessen and the Max Planck Institute for Heart and Lung Research in Germany, studying mechanisms that modulate innate immunity in the context of pneumonia.

After completing his doctorate, he continued his research as a postdoctoral fellow at the University of Michigan (USA), focusing on pharmacological studies of GPCR receptors, particularly lipid mediators of inflammation.

Since 2015, he has worked as a research scientist at Trinity College Dublin (Ireland) in the Department of Biochemistry and Immunology, as well as at the newly established biotech company Sitryx, where he was responsible for anti-inflammatory drug discovery programs.

Dr. Zasłona is an internationally recognized expert in inflammatory processes and lung diseases, and has been frequently invited to deliver lectures at international scientific conferences.

TITLE:

The Development of USP7 Inhibitor for Cancer Immunotherapy

ABSTRACT:

Ubiquitin-specific protease 7 (USP7) regulates the stability and fate of many proteins, thereby influencing cellular processes. Elevated level of USP7 in cancer contributes to tumor progression by modulation of tumor microenvironment. USP7 has a variety of substrates, including MDM2 (Murine double minute 2) and the tumor suppressor p53—well known targets in cancer drug development. We have explored USP7-MDM2-p53 pathway in the context of immunomodulation.

After administering OAT-4828, we observed a significant reduction in tumor volume in syngeneic mouse models of colon, melanoma cancer, and leukemia. This reduction was associated with a substantial increase in T cell activation, as evidenced by higher levels of CD69 and CD44, as well as increased production of Granzyme B and IFN-γ. Antitumor activity of OAT-4828 was improved when combined with anti-PD-1 antibodies, while the T cell depletion completely abrogated the therapy outcome.

OAT-4828 caused increased production of IL-2 and upregulation of CD25 and CD69 in human T cells, which was associated with MDM2 downregulation and p53 stabilization. Similarly, inhibition of USP7 lead to downregulating MDM2 in macrophages, inhibiting their M2-like functions. These results indicate that the main mechanism of action of OAT-4828 is based on the activation and improved cytotoxic functions of T cells and promoting anti-cancer activity relevant to the tumor microenvironment.

USP7-MDM2-p53 is implicated in function of immune cells and tumor microenvironment. Altogether we confirm USP7 as an attractive target for cancer immunotherapy and present the rationale for the development of USP7 inhibitors for clinical use.

Milena Mazan

Ryvu Therapeutics, Poland

BIO:

Dr. Milena Mazan is a Head of Translational Medicine at Ryvu Therapeutics, with a PhD in Cancer Research and over 10 years of experience in oncology, translational research, biomarker strategy, and clinical development. Her work focuses on bridging exploratory research and clinical application through biomarker-driven studies supporting the development of novel cancer therapies. At Ryvu, Dr Mazan is responsible for the development and execution of the translational research plan for most advanced compounds, coordinating preclinical research activities, external scientific collaborations, and biomarker analyses supporting clinical development. Her current work contributes to the advancement of treatment strategies in acute myeloid leukemia and solid tumors.

Dr Mazan completed her MSc studies at Jagiellonian University and the University of Orléans, and obtained her PhD at the Manchester Cancer Research Centre, where she studied transcriptional mechanisms involved in hematopoiesis and malignant transformation. She later worked at the Wellcome Trust Sanger Institute in Cambridge, focusing on leukemia biology and therapeutic target identification.

Since joining Ryvu in 2016, she has held senior scientific and leadership roles and contributed to oncology programs including romaciclib and dapolsertib.

Learn more about Ryvu.

TITLE:

Leveraging Cancer Biology for Therapeutic Innovation: Clinical and Discovery Advances at Ryvu

ABSTRACT:

Ryvu Therapeutics is a clinical-stage biopharmaceutical company advancing a broad and differentiated oncology portfolio designed to address high-unmet medical needs by targeting key mechanisms in cancer biology, from dysregulated kinases to synthetic lethality and precision medicine platforms. Our pipeline comprises both innovative clinical candidates and discovery programs that exemplify novel approaches to treating challenging malignancies.

At the forefront of our clinical efforts is romaciclib (RVU120), a first-in-class, orally bioavailable selective CDK8/CDK19 inhibitor that modulates transcriptional programs driving cancer cell survival. Romaciclib is being evaluated across multiple Phase II studies, with focus on relapsed or refractory acute myeloid leukemia (AML) and myelofibrosis, where encouraging signals of activity and a good safety profile have been observed. Other clinical studies are investigating romaciclib in lower-risk myelodysplastic syndromes and pediatric medulloblastoma. In parallel, dapolsertib (MEN1703/SEL24), a dual PIM/FLT3 kinase inhibitor licensed to the Menarini Group, is progressing in Phase II development in diffuse large B-cell lymphoma (DLBCL).

Beyond these clinical assets, Ryvu's ONCO Prime discovery platform supports the discovery of novel precision targets in CRC, TNBC and lung cancers. Separately, Ryvu is advancing ADC programs that incorporate innovative payloads, thereby broadening the range of potential future therapies.

Together, these programs reflect Ryvu's commitment to advancing precision oncology by integrating deep biological insights with translational science, fostering collaborations that accelerate the delivery of impactful therapies for patients.

Justyna Adamyczk

Enamine, Poland

BIO:

Justyna Adamczyk is a Lead Scientist and Project Manager at Enamine, a global provider of small molecules and integrated drug discovery services. With over seven years of industry experience, she leads and supports early-stage drug discovery programs, with a focus on the design and execution of high-throughput and targeted screening strategies.

Her expertise spans protein–ligand interaction studies, assay development, and targeted protein degradation, integrating biophysical and cell-based approaches. She brings extensive experience in high-throughput screening (HTS), including the establishment of screening platforms and the delivery of complex screening campaigns across diverse target classes.

TITLE:

Enabling Early Drug Discovery: Integrated Screening Capabilities and a BRD4/CRBN PROTAC Case Study

ABSTRACT:

Enamine is a global provider of integrated drug discovery solutions supporting early-stage research through access to compound libraries and experimental screening capabilities. Its

platform enables efficient progression of discovery projects by integrating assay development, high-throughput screening workflows, and biophysical and cell-based characterization methods.

Targeted protein degradation has emerged as a powerful approach in cancer drug discovery, enabling modulation of previously undruggable targets through induced protein degradation mechanisms.

Using a BRD4/CRBN PROTAC case study, we will present a stepwise screening approach for identifying initial degrader candidates. The workflow includes primary screening, followed by biophysical characterization and cell-based assessment of target engagement and degradation effects. Particular emphasis will be placed on assay selection strategy, data triaging, and progression criteria applied during hit identification.

The example illustrates how complementary experimental readouts are integrated to support evaluation of early PROTAC candidates and guide progression within early-stage discovery programs.

Marek Kudła

Ardigen, Poland

BIO:

Dr. Marek Kudła, serves as the Director of Bioinformatics at Ardigen, where he provides strategic leadership at the intersection of biological research and computational data analysis.

His journey began at the University of Warsaw, leading him to a PhD at Cold Spring Harbor Laboratory and research at UC Berkeley, where he refined his expertise in genomics and transcriptomics.

With a career defined by bridging the gap between complex biological data and drug discovery, Marek now oversees high-performing, interdisciplinary teams of bioinformaticians, software engineers, and data scientists, focusing on the collaborative path from experimental insight to therapeutic impact.

TITLE:

Transforming Multimodal Complexity into Precision Oncology Insights

ABSTRACT:

The clinical success of oncology therapies rests on ability to look beyond single biomarkers to decipher the complex interplay between patient genetics, the tumor microenvironment, and systemic immune responses. As the volume of multi-omic and clinical data grows, the challenge lies in converting this vast complexity into actionable clinical insights. Ardigen bridges this gap by

integrating advanced machine learning approaches with deep domain expertise in immunology and data science. Our approach transforms disparate, high-dimensional datasets into reliable, interpretable biomarkers and patient stratification models.

Multimodal Biomarker Discovery

In oncology, relying on single-metric biomarkers often misses the nuanced reality of patient response. By integrating heterogeneous datasets into cohesive prediction models we can shift the focus from isolated variables to complex, multimodal signatures to consistently outperform standard clinical benchmarks, enabling more accurate patient stratification and superior survival prediction.

Mechanism-Driven Insights for Drug Resistance

We utilize explainable AI to uncover the hidden drivers of treatment resistance. By deconvoluting the complex immune landscape and mapping subtle molecular patterns, we help our partners identify novel therapeutic combinations designed to restore anti-tumor immunity and bypass resistance pathways.

Scalable AI Infrastructure for R&D Acceleration

Through robust, generalized machine learning frameworks, we extract biological insights from massive, heterogeneous datasets enabling rapid biomarker discovery across a wide range of indications, significantly de-risking the transition from early-stage research to clinic-ready diagnostic development. By turning complex, noisy biological data into interpretable, scientifically grounded insights, Ardigen empowers our partners to better navigate the complexities of modern oncology.

Artur Wnorowski

Biotechna, Poland

BIO:

Artur Wnorowski, PhD, DSc (Pharm) is a professor at the Medical University of Lublin and Scientific Director at BS Biotechna. He specializes in pharmaceutical sciences and biotechnology, with academic and research experience spanning drug development and translational biomedical research.

He earned his PhD in pharmaceutical sciences in 2015 and habilitation in 2023 at the Medical University of Lublin. He previously graduated in biotechnology from the Warsaw University of Life Sciences.

TITLE:

Synergistic Nanotechnology for Targeted Therapeutics in Oncology

ABSTRACT:

Biotechna SA is an innovative biotechnology company dedicated to addressing unmet medical needs in oncology through the development of targeted therapies for solid tumors. Our proprietary approach leverages nanotechnology platforms capable of delivering highly specific, synergistic combinations of active pharmaceutical ingredients, including small molecules and small interfering RNA (siRNA), directly to the tumor microenvironment. By optimizing the physi-

cochemical properties of these delivery systems, we aim to improve cellular uptake, overcome adaptive chemoresistance, and reduce off-target toxicities compared to traditional therapies.

Our investigational program for Triple-Negative Breast Cancer (TNBC) utilizes a novel nanocarrier conjugate delivering a synergistic combination of two active substances. In xenograft models, it demonstrates a four-fold increase in the Maximum Tolerated Dose (MTD) compared to the free drug combination without induction of intrinsic immune activation. Furthermore, this targeted delivery translated to significant primary tumor shrinkage and anti-metastatic activity, achieved using a reduced, lower dose-density schedule (every-other-week versus weekly).

Agata Drewniak-Maksymów

JJP Biologics, Poland

BIO:

Agata Drewniak-Maksymów is a Translational Science Lead at JJP Biologics, specializing in oncology, immunology, and precision medicine. With 10 years of experience in the biopharmaceutical industry, she has held leadership roles at Sanofi and Kiadis Pharma, where she led biomarker and translational strategies across immuno-oncology and cell therapy programs.

Her work focuses on integrating biomarker-driven approaches to support proof of mechanism, patient selection, and clinical development. Holding a PhD in Immunology from University of Amsterdam , she is also a lecturer and scientific consultant, bridging complex science with clinical application.

TITLE:

JJP-1008 as a Novel Checkpoint Inhibitor

ABSTRACT:

JJP Biologics is a Polish biotechnology company developing novel biologics for the treatment of a range of disease indications. We have multiple (pre)clinical programs that we plan to develop until clinical proof of concept. JJP-1008 is a pioneering humanized and stabilized IgG4-κ monoclonal antibody targeting CD270.

High HVEM expression on tumors correlates with poor prognosis and an immunosuppressive tumor microenvironment, making it an ideal target for immuno-oncology interventions. Tumors, including melanoma, breast, lung and colon cancers, exploit CD270 signaling

pathways to escape immune attack, leading to disease progression and resistance to existing immunotherapies.

Through our deep mechanistic insights, we engineered JJP-1008 to offer a much-needed novel treatment option for patients who do not respond to current PD-1/PD-L1 therapies, as identified through tumor expression profiling of CD270.

JJP-1008 blocks the immunosuppressive interactions of CD270 with inhibitory receptors CD160 and BTLA, while preserving and even enhancing immune activation through CD258 (LIGHT). This balanced modulation reactivates the immune system's ability to recognize and kill cancer cells, overcoming tumor immune evasion.

Preclinical studies have demonstrated anti-tumor activity of JJP-1008 in both in vitro and in vivo models, including melanoma, supporting its potential as a transformative checkpoint inhibitor and potential broad application in solid tumors. The program is currently advancing through preclinical development, with ongoing CMC activities supporting future clinical translation.

Jakub Knurek

Mabion, Poland

BIO:

Jakub Knurek is a marketing specialist at Mabion Biologics with expertise in biotech and pharma marketing. His work focuses on translating biologics development, drug manufacturing, and analytical services into clear, value-driven communication that supports business development, brand differentiation, and market visibility. With a scientific foundation in biologics and experience related to analytical method transfer during Mabion's collaboration with Novavax, as well as exposure to monoclonal antibodies such as rituximab and cetuximab, Jakub bridges scientific understanding with strategic communication. In his presentation, "Development of Biological Drugs for Oncological Indications at Mabion," he will share insights into Mabion's role in supporting the development and manufacturing of biologic drug candidates for oncology.

TITLE:

Development of Biological Drugs for Oncological Indications at Mabion

ABSTRACT:

The high attrition rates observed in early-stage oncology biologics development underscore persistent gaps in translational and process sciences that compromise progression from discovery to clinical evaluation. Combining data from published literature, clinical trial registries, regulatory case examples, and meta-analyses of development outcomes, we systemat-

ically identified and categorized predominant failure modes affecting therapeutic biologics, including monoclonal antibodies and engineered protein modalities.

Failure pathways were stratified into:

- biological-target mischaracterization,
- product developability and manufacturing liabilities,
- clinical translation challenges.

A notable instance illustrating target selection failure is matuzumab, a humanized anti-EGFR antibody whose development was halted after disappointing efficacy in phase II colorectal cancer trials despite promising preclinical rationale, reflecting deficiencies in translating mechanistic understanding into clinical benefit. Similarly, depatuxizumab mafodotin, an EGFR-targeted antibody-drug conjugate, ceased enrolment due to lack of anticipated clinical activity in glioblastoma, exemplifying translational attrition rooted in target modality and model predictivity. In the domain of immunogenic and pharmacokinetic liabilities, failure to adequately assess or mitigate immunogenic responses and developability issues remains a recurrent impediment, as evidenced by the foundational risk analyses in biologics development and immunogenicity literature.

Quantitative synthesis further indicates that early developability screening, biophysical profiling, and integration of predictive biological markers are critical to de-risk candidates before costly clinical investments. These findings suggest that enhanced integration of mechanistic pharmacology, CMC science, and predictive biomarker frameworks could reduce failure rates, expedite translational decision-making, and align early-stage oncology biologics with precision medicine objectives.