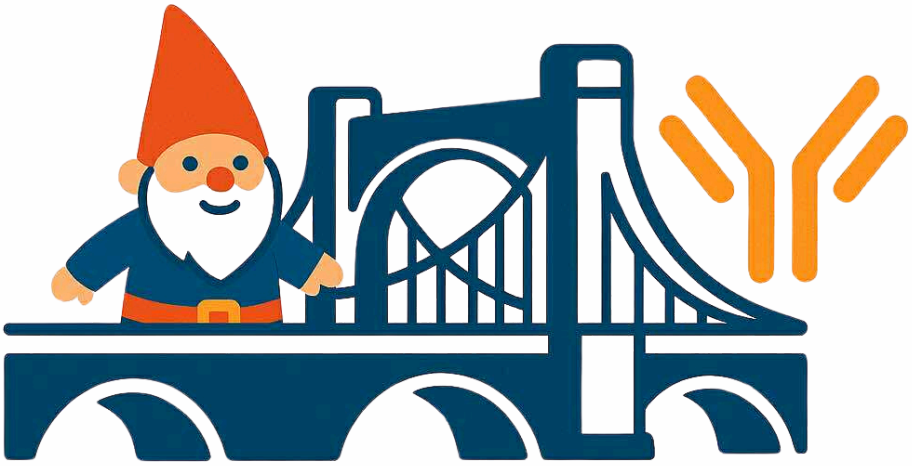
Marcin Kulasek
Minister of Science and Higher Education
Republic of Poland



# IMMUNOBRIDGE

## Polish Young Immunologists 1st Symposium

## Wrocław, Poland 17-20.03.2026

POLISH YOUNG IMMUNOLOGISTS

Instytut Immunologii i Terapii Doświadczalnej im. L. Hirszfelda Polskiej Akademii Nauk

PAN POLSKA AKADEMIA NAUK

POLSKIE TOWARZYSTWO IMMUNOLOGII DOŚWIADCZALNEJ I KLINICZNEJ
Polish Society for Fundamental and Clinical Immunology

WCA Wrocławskie Centrum Akademickie

yefis

**Marcin Kulasek**
Minister of Science and Higher Education
Republic of Poland

**This conference is held under the Honorary Patronage of the Minister of Science and Higher Education.**

## Scientific Committee of ImmunoBridge:

- Assoc. Prof. Paulina Niedźwiedzka-Rystwej, PhD **(general chair)**
- Prof. Katarzyna Bogunia-Kubik
- Prof. Anna Chełmońska-Soyta
- Prof. Urszula Demkow
- Prof. Andrzej Gamian
- Ewa Oleszycka, PhD
- Prof. Andrzej Siwicki
- Assoc. Prof. Bogumiła Szponar, PhD

## Organizing Committee of ImmunoBridge:

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- MSc Julia Depta
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- BSc Wiktoria Grzybowska
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- Ewa Oleszycka, PhD
- BSc Zuzanna Pochwała
- MSc Natalia Rzepka
- MSc Karolina Rusewicz
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- Weronika Jasińska, PhD
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POLISH YOUNG IMMUNOLOGISTS

The **Polish Young Immunologists (PYI)** is a section of the Polish Society of Experimental and Clinical Immunology (PTIDiK). The fundamental objective of the PYI is to integrate students and young scientists, as well as to encourage research in the domain of immunology among young professionals in Poland.

The section was founded in December 2024. The members of the Board for the first term are:
President: Filip Lewandowski (Szczecin)
Vice-president: Ewa Oleszycka (Krakow)
Board secretary: Wiktoria Grzybowska (Wrocław)
Board member: Nicole Kryniecka (Wrocław)

Facebook: **Polish Young Immunologists**
Instagram: **@polish_young_immunologists_pyi**
LinkedIn: **Polish Young Immunologists**

The **Polish Society of Fundamental and Clinical Immunology (PTIDiK)** brings together researchers and clinicians working in the field of immunology, fostering collaboration between experimental and clinical disciplines.

POLSKIE TOWARZYSTWO IMMUNOLOGII DOŚWIADCZALNEJ I KLINICZNEJ
Polish Society for Fundamental and Clinical Immunology

Its main goals include supporting scientific research, promoting education in immunology, organizing conferences, workshops, and seminars, as well as encouraging the development of young scientists. PTIDiK also cooperates with national and international scientific organizations to strengthen the exchange of knowledge and experience. The current President of the Society is Prof. Ewelina Grywalska, with Assoc. Prof. Paulina Niedźwiedzka-Rystwej serving as President-Elect

The **Institute of Immunology and Experimental Therapy** was founded in 1952 by the **Polish Academy of Sciences**. The Institute was established by **Professor Ludwik Hirszfeld**, a renowned Polish immunologist and microbiologist, who served as its inaugural director. He established the Polish school of immunology and was the pioneer of a new scientific discipline – seroanthropology.

He identified the Rh factor and elucidated the phenomenon of serological conflict between mother and fetus, achievements that led to his nomination for the Nobel Prize in Physiology or Medicine in 1950.

After his passing, the leadership of the institute was taken over by Professor **Stefan Ślopek**, a specialist in medical microbiology and immunology. He held this position for 31 years, making him the longest-serving Director of the Hirszfeld Institute. He conducted pioneering research on therapies for infectious diseases of the gastrointestinal and respiratory systems, as well as on the process of phagocytosis. He also explored the therapeutic applications of bacteriophages, including their use in treating sepsis.

Professor **Andrzej Gamian** currently serves as Director, as well as Head of the Department of Medical Biochemistry at Wrocław Medical University and the Department of Infectious Diseases Immunology

# Useful information

## Means of transport

You can check public transport on desired routes in real time as well as purchase tickets in **Jakdojade** app.

**From the train station/hotel to the Institute**:
Trams available on this route: 9 and 15*

# IMMUNOBRIDGE 2026 SCHEDULE

## TUESDAY, MARCH 17

| | |
|---|---|
| 11:00-14:00 | Registration |
| 11:30-11:45 | Official Opening Ceremony |
| 11:45-12:00 | About Polish Young Immunoligsts |
| 12:00-13:00 | Keynote speach 1: **Assoc. Prof. Paulina Niedźwiedzka- Rystwej** |
| 13:00–13:45 | Lunch |
| 13:45–15:30 | Session 1 Cancer Immunology (Chair: **Assoc. Prof. Paulina Niedźwiedzka-Ryswej**) |
| 15:30–16:00 | Coffee break |
| 16:00-18:00 | Workshop 1: The use of real-time PCR in SNP genotyping |
| 19:00-21:00 | Networking |

## WEDNESDAY, MARCH 18

| | |
|---|---|
| 08:30–12:30 | Registration |
| 09:00–10:00 | Keynote Speaker (**PhD Odrei Stephanek**) |
| 10:00–10:30 | Coffee break |
| 10:30–11:30 | Session 2 Innate immunology (Chair: **Prof. Joanna Cichy**) |
| 11:30 - 12:30 | Invited speaker 1: **PhD Agnieszka Razim** |
| 12:30-13:15 | Lunch |
| 13:15–14:00 | Session 3 Neuroimmunology (Chair: **PhD MD Ali Jawaid**) |
| 14:00-15:00 | Invited speaker 2: **PhD MD Ali Jawaid** |
| 15:00–15:30 | Coffee break |
| 15:30-17:30 | Flashtalk session + Workshop „The Art of Public Speaking" (Chair: **PhD MD Ali Jawaid**) |
| 19:00–21:00 | Networking |

## THURSDAY, MARCH 19

| | |
|---|---|
| 08:30–13:00 | Registration opens |
| 09:00–10:00 | Keynote Speaker (**PhD Katarzyna Sitnik**) |
| 10:00–10:30 | Coffee break |
| 10:30–11:30 | Session 5 Immunology of infectious diseases (Chair: **Assoc. Prof. Mariola Paściak**) |
| 11:30-12:30 | Invited speaker 3: **PhD Louis Boon** |
| 12:30-13:15 | Lunch |
| 13:15–13:45 | Session 4 Microbiome and Immunology (Chair: **PhD Aleksandra Kołodziejczyk**) |
| 13:45-14:45 | Invited speaker 4: **PhD Aleksandra Kołodziejczyk** |
| 14:45-15:45 | Invited speaker 5: **Assoc. Prof. Wojciech Juzwa** |
| 15:45–16:15 | Coffee break |
| 16:00 - 17:30 | **Poster session** + Best Poster Awards |
| 19:00–21:00 | Networking |

## FRIDAY, MARCH 20

| | |
|---|---|
| 09:00–10:00 | Keynote Speaker (**PhD Roisin McManus**) |
| 10:00–10:30 | Coffee break |
| 10:30–11:15 | Session 6 Hipersesitivity (Chair: **Assoc. Prof. Andrzej Eljaszewicz**) |
| 11:15-12:15 | Invited speaker 6: **Assoc. Prof. Andrzej Eljaszewicz** |
| 12:15 - 13:00 | Official Closing + Best Presentation Awards |
| 13:00 - 13:45 | Lunch |

**IMMUNOBRIDGE**
**Polish Young Immunologists**
**1st Symposium**
**Wrocław, Poland 17-20.03.2026**

# Keynote speakers

**Assoc. Prof. Paulina Niedźwiedzka-Rystwej, PhD**, professor at the University of Szczecin, leader of the research team of experimental immunology and immunobiology of infectious and cancer diseases at the Institute of Biology of the University of Szczecin, Poland, Director of the Center for Experimental Immunology and Immunobiology in Infectious Diseases and Cancer, University of Szczecin, Szczecin, Poland, Deputy Director of the Institute of Biology at the University of Szczecin. She has been associated with the University of Szczecin since the beginning of her scientific career in 2005.

Author and co-author of over 200 scientific papers on veterinary immunology, virology, and immunobiology of infectious and cancer diseases, with a total IF of over 260. She conducts research on the mechanisms of immune response in infectious and cancerous diseases, focusing on phenomena such as apoptosis, autophagy, and T-cell exhaustion. Her work includes, among others, analysis of the immune response in rabbits infected with various strains of the Lagovirus europaeus virus and research on the activation of apoptosis in infections caused by this pathogen. In addition, she evaluates the antimicrobial and anticancer properties of bee products, such as honey and propolis, studying their therapeutic potential in the context of cancer and infectious diseases.

She is the founder of the IMMUNO Apiary, established in 2022 at MOBI Kulice. She is also the co-author of translations of textbooks on immunology and the president-elect of the Polish Society for Experimental and Fundamental Immunology. She has been a keynote speaker at numerous international immunology conferences, co-founder of the First International School of Immunology in Poland. Recipient of grants from the National Science Centre, and the National Centre for Research and Development in the field of veterinary immunology and infectious diseases in humans. Winner of awards for scientific activity, including the West Pomeranian Nobel Prize, as well as teaching awards, e.g., Faculty Mentor, Medal of the National Education Commission. Certified mentor and tutor.

At the ImmunoBridge 2026 conference, Prof. Paulina Niedźwiedzka-Rystwej will deliver a keynote lecture titled **„Exhausted but Not Defeated: How PD-1 Pathways Influence Anti-Cancer Immunity”** and will serve as an **expert in the session on oncoimmunology.**

# Keynote speakers

**Ondřej Stěpánek, PhD,** is an expert in the field of T cell receptor (TCR) signaling and adaptive immunity. He has more than 15 years of experience in research on T cell biology, spanning molecular mechanisms of receptor signaling, thymic development, and eU̓ector and regulatory T cell responses. Following his doctoral training at Charles University and the Institute of Molecular Genetics (IMG) in Prague, he pursued postdoctoral research at the University Hospital Basel, where he made fundamental contributions to our understanding of TCR tolerance mechanisms.

Since establishing the Laboratory of Adaptive Immunity at IMG in 2016, Dr Stěpánek has developed an at IMG in 2016, Dr. Stěpánek has developed an internationally recognized research program that dissects how T cells integrate signals from their receptors and co-receptors to make fate decisions. His group has uncovered unique roles of co-receptor–bound LCK in helper and cytotoxic T cells, identified a new IL-17 receptor subunit critical for T cell-mediated autoimmune pathology, and revealed novel mechanisms governing self-tolerance and functional T-cell diversity.

To directly connect mechanism to physiology, his team makes extensive use of animal models, including syngeneic tumor models, autoimmune models driven by IL-17 signaling, and thymic development models elucidating lineage choice and co-receptor/LCK control of self-reactivity. These approaches have enabled the lab to bridge molecular immunology with disease-relevant biology.

Dr. Stěpánek is the recipient of an ERC Starting Grant (2019) and an ERC Consolidator Grant (2025), the only ERC grants in the field of immunity and immunotherapy (LS6) ever awarded in Czechia, and an EMBO Installation Grant. He is also a laureate of the J.E. Purkyne Fellowship of the Czech Academy of Sciences and the Eastern Star Award of the European Federation of Immunological Societies. His laboratory regularly publishes in prestigious journals such as Nature Immunology, Nature Communications, EMBO Journal, eLife, and actively collaborates across Europe and beyond.

In addition to leading cutting-edge research, Dr. Stěpánek is deeply committed to mentoring the next generation of immunologists, having supervised more than 15 PhD and MSc students. He teaches immunology courses at Charles University and serves on national and international evaluation panels. He is regularly invited to speak at international conferences and seminars.

At the ImmunoBridge 2026 conference, Dr. Stěpánek will deliver a keynote lecture titled **“Signaling pathways shape the outcomes of T-cell fate choices.”**

# Keynote speakers

**Katarzyna Sitnik, PhD,** studied biotechnology at the Jagiellonian University in Kraków and subsequently earned her PhD in biomedicine at the Faculty of Medicine at Lund University in Sweden. She completed postdoctoral training in immunology at the Technical University of Denmark in Copenhagen and in virology at the Helmholtz Centre for Infection Research in Germany.

Since 2024, Katarzyna has been a tenure-track assistant professor and research group leader in stromal cell biology at the Department of Biomedical Sciences at the University of Veterinary Medicine Vienna in Austria. Her research integrates expertise in fibroblast biology, immunology, and virology to investigate how stromal cells contribute to immunomodulation and host–microbe interactions.

In her keynote talk **„Lymphoid Tissue Fibroblasts in Immunity and Infection”**, Katarzyna will discuss the emerging roles of stromal fibroblasts in shaping immune responses and influencing outcomes of infection, highlighting new perspectives and potential therapeutic opportunities.

**Róisín McManus, PhD,** completed her PhD at Trinity College Dublin under the joint supervision of Prof. Marina Lynch and Prof. Kingston Mills (2015) where she studied the effect of infection on the brain with age and Alzheimer's disease (AD). Róisín then moved to Germany where she first worked with Prof. Dr. Michael Heneka (2015-2021) and then Prof. Dr. Eicke Latz (2021-2024) at the Institute of Innate Immunity, University of Bonn and the German Centre for Neurodegenerative diseases (DZNE), where she uncovered a new role for NLRP3 in the progression of AD and dementia. In 2024, Róisín was awarded a Fellowship from the DZNE to establish her own independent research group, 'Translational Neuroimmunology'. The group make use of preclinical models and clinical samples to uncover how environmental factors like diet contribute to the progression of dementia. Róisín and her team are particularly interested in the role of chronic innate immune activation and metabolic signaling pathways in this process.

At the ImmunoBridge 2026 conference, Dr. Róisín McManus will deliver a keynote lecture titled **„Microglial innate immune signalling as a driver of dementia”**.

# Invited speakers

**Agnieszka Razim, PhD,** is an Assistant Professor at the Hirszfeld Institute of Immunology and Experimental Therapy, Polish Academy of Sciences, and a former Marie Skłodowska-Curie Postdoctoral Fellow at the Medical University of Vienna. She is currently running a SONATA research project. Her research focuses on interactions between the microbiome and mucosal immune systems, particularly in the contexts of allergy and malnutrition; she studies the role of bacterial extracellular vesicles in bacteria–host communication.

She is also a science communicator, a holder of patents, and a recipient of the START2022 scholarship from the Foundation for Polish Science.

During the conference Dr. Agnieszka Razim will deliver a talk titled **„Building your academic CV: defining long-term goals, writing competitive research proposals and more."**

# Invited speakers

**Ali Jawaid, MD, PhD**, is a physician-scientist with training in both clinical and basic neuroscience. He is a Principal Research Investigator at the Research Network Łukasiewicz – PORT Polish Center for Technology Development. He completed his medical studies at the Aga Khan University in Karachi, Pakistan, followed by a fellowship in Neuropsychiatry at Baylor College of Medicine in Houston, USA. He subsequently earned an MD-PhD in Neuroscience in Switzerland (dual PhD degrees from the UZH/ETH International Program in Neuroscience and the UZH MD-PhD Program, 2016), and continued with a postdoctoral fellowship in Neuroepigenetics at ETH Zurich.

Dr. Jawaid has been an independent group leader since late 2020, investigating metabolic and epigenetic factors that shape susceptibility to neuropsychiatric disorders across the lifespan and across generations. He has authored more than 80 publications, including original research articles in top-tier journals such as Nature Neuroscience, Neuron, Nature Communications, EMBO Journal, and Translational Psychiatry, and currently holds an H-index of 33.

He is a member of the FENS-Kavli Network of Excellence, a prestigious platform of 30 outstanding European neuroscientists supported by the Federation of European Neuroscience Societies. Outside of science, he is also a fiction writer, poet, and virtual-reality enthusiast.

During the conference, Dr. Ali Jawaid will **chair the Neuroimmunology Session,** will deliver a lecture titled **„Immunometabolic Pathways of Brain Resilience and Vulnerability Across the Lifespan”** and will lead the workshop **„The Art of Public Speaking”.**

# Invited speakers

**Louis Boon, PhD,** is the Chief Scientific Officer and a Management Board Member of JJP Biologics, an innovative Polish biotechnology company supported by the Starak family, focusing on the development of next-generation therapeutic biologics based on personalized medicine and companion diagnostics. He has founded multiple companies dedicated to the generation and development of therapeutic monoclonal antibodies, primarily in the fields of oncology and inflammation.

Dr. Boon is the author of over 400 publications in international scientific journals in medical biotechnology and an inventor on more than 20 patent applications.

His research focuses on the mechanisms by which autoantigen-specific IgA autoantibodies contribute to the pathogenesis of multiple autoimmune and fibrotic diseases. In particular, he investigates how excessive IgA/autoantigen immune complexes trigger continuous CD89 (FcαRI)-mediated activation of myeloid cells, including neutrophils, leading to severe tissue damage. He has pioneered the use of autoantigen-specific IgA as a biomarker to stratify patients for personalized therapy.

At the ImmunoBridge 2026 conference, Dr. Louis Boon will deliver a lecture titled: **„Anti-CD89 Antibody for Personalized Treatment of IgA-Mediated Inflammatory Disorders: Autoantigen-Specific IgA, a Biomarker with Strong Effector Functions”**.

The lecture will highlight the therapeutic potential of JJP-1212, a humanized anti-CD89 antibody, which interferes with the IgA/CD89 axis to resolve IgA/autoantigen-induced inflammation and prevent tissue damage in a variety of autoimmune disorders, enabling a precision medicine approach for patients with high levels of autoantigen-specific IgA.

# Invited speakers

**Aleksandra Kołodziejczyk, PhD,** is a specialist in single-cell transcriptomics and host–microbiota interactions. During her PhD at the Wellcome Sanger Institute, she developed foundational methods for analysing single-cell RNA-seq data, including approaches to quantify technical variability and identify low-quality cells. She applied these techniques to explore the heterogeneity of T cells and mouse embryonic stem cells, demonstrating that variability within the core pluripotency network is shaped by culture conditions.

In her postdoctoral work, Kołodziejczyk investigated how the microbiota influences immune and metabolic responses across tissues in both animal models and clinical contexts. Using large-scale single-cell profiling, she identified tissue-specific host responses to the microbiota and uncovered shared transcriptional activation programs in liver-resident cells driven by Myc. Her research also revealed microbiota-derived metabolites that regulate body weight after smoking cessation and mechanistic links between hyperglycaemia, dendritic-cell dysfunction, and increased susceptibility to viral infections.

Dr. Kołodziejczyk is also engaged in comparative immunology, studying conserved and divergent antiviral programs in bats, rodents, and primates, with a focus on mucosal immunity and complement system evolution.

During the conference, Dr. Kołodziejczyk will serve as an **expert in the session on microbiome immunology** and will deliver a lecture titled **“Host–Microbiota Interactions in Health and Disease”**

# Invited speakers

**Assoc. Prof. Wojciech Juzwa** has been working since 2010 at the Department of Biotechnology Microbiology of Food at the Poznań University of Life Sciences, dealing with the analysis and sorting of eukaryotic and prokaryotic cells using conventional flow cytometers, cell sorters, and imaging instruments. Wojciech Juzwa specializes in investigation of the structural and functional cellular parameters using routine and self-created analytical protocols, which he develops for various stages of cytometric experiments. Currently, his scientific and translational focus is on:

i) utilizing a combination of cytometry with imaging and cell sorting to improve precision of the definition of the sub-populations for sorting,

ii) troubleshooting in cytometric analysis and cell sorting, and

iii) improving the efficiency and viability of cells in sorting for cell therapy purposes (including Advanced Therapy Medicinal Products (ATMPs)).

The scientific output comprises 79 original research publications, including 70 indexed in the JCR database. The total Impact Factor is 303.183, the MNiSW score amounts to 8,050 points, the number of citations is 1,270, and the h-index is 20. At CYTO-LOGIC, he is the Head of the applications department. As a trainer within the Cyto-Logic Flow Academy, he conducts installation and application training and integrates the implementation of cell isolation systems for the preparation of ATMPs.

During the conference, Dr. Juzwa will deliver a lecture titled **"Who Is Who in the Microbiome? Single-Cell Technologies, Flow Cytometry, and the Architecture of Gut Microbial Communities".**

# Invited speakers

**Assoc. Prof. Andrzej Eljaszewicz** is a medical biotechnologist and immunologist, currently acting as the Director of the Centre for Regenerative Medicine at the Medical University of Białystok, Poland, and serving as the Head of the Tissue and Cell Bank at the University Clinical Hospital in Białystok. He is also the Editor-in-Chief of the Central European Journal of Immunology, the official journal of the Polish Society for Experimental and Clinical Immunology, and an active member of the European Academy of Allergy and Clinical Immunology (EAACI), where he currently serves as Secretary of the Immunology Section.
Dr. Eljaszewicz leads a young and dynamic research team focused on advancing translational approaches in immunology and regenerative medicine. His scientific work centers on the immunopathology of asthma, with particular interest in airway remodeling, extracellular matrix dynamics, and immune regulation. In parallel, he leads translational research on next-generation biological wound dressings, including ECM-based scaffolds, mesenchymal stem cell–derived products, and extracellular vesicles, aimed at improving therapies for chronic and hard-to-heal wounds.

During the conference, Dr. Eljaszewicz will serve as an **expert in the session on hipersesitivity** and will deliver a lecture titled **“Beyond Inflammation: The Role of Allergen Proteases in Airway Remodelling”.**

# Experts

**Prof. Joanna Cichy** is an expert in the immunology of barrier organs with a primary focus on the skin. Her research group explores various aspects of skin defense, including immunology, neurology, antimicrobial products, interactions with microbiota, and adipocytes. Most recently, her work has centered on understanding the interactions between two complementary defense systems in the skin: the immune system with a focus on innate immunity and the sensory neuronal system.

Prof. Cichy earned her PhD in Biochemistry from the Jagiellonian University (UJ) in Kraków, Poland. She has since held positions as a Postdoctoral Fellow at the Wistar Institute USA and as a visiting scholar at Stanford University USA. She is the author of over 90 publications and has received numerous national and international grants, including a Fulbright Fellowship and the Fogarty International Research Collaboration Award. Currently, she serves as a Professor and Head of the Department of Immunology at the Faculty of Biochemistry, Biophysics, and Biotechnology at UJ.

During the conference, Prof. Cichy will serve as an **expert in the session on Innate Immunology.**

# Experts

**Assoc. Prof. Mariola Paściak** is a long standing member of the Laboratory of Medical Microbiology in the Department of Immunology and Infectious Diseases at the Institute of Immunology and Experimental Therapy in Wrocław. She has extensive expertise in elucidating the chemical structures and biological activities of various actinobacterial glycolipids and polysaccharides. As an expert in the taxonomy of Actinobacteria, she, along with her collaborators, established a novel genus called Ruania within the Actinobacteria class and characterised a few new species of Actinobacteria.

Mariola Paściak, a medical microbiologist, possesses unique skills in the deep diagnostics of clinical and environmental bacterial strains. She has significant knowledge and experience with MALDI TOF mass spectrometry, lipidomics, and other analytical methods. Her current research focuses on bacterial extracellular vesicles, including their compositional analysis and biological functions. She is a member of several scientific associations and actively participates in research collaborations, resulting in publications in highly regarded journals. She has supervised bachelor's, master's, and doctoral students and is currently overseeing two PhD students.

Dr. Mariola Paściak will serve as an **expert in the session on the Immunology of Infectious Diseases.**

# Day 1, Tuesday
# 17.03.2026

11:00 - 14:00 Registration
11:00 -16:00 Workshop 1: Stress-Free Cell Sorting: Preserving Fragile and Neuronal Cells with the WOLF Cell Sorter
11:30 - 11:45 Official Opening Ceremony
11:45 - 12:00 About Polish Young Immunologists
12:00 - 13:00 Keynote speech 1:
**Assoc. Prof. Paulina Niedźwiedzka-Rystwej** (University of Szczecin)
*„Exhausted but Not Defeated: How PD-1 Pathways Influence Anti-Cancer Immunity"*
13:00 - 13:45 Lunch
13:45 - 15:30 Session 1: Oncoimmunology
Chair: **Assoc. Prof. Paulina Niedźwiedzka-Rystwej** (University of Szczecin)

- OP1 MSc Filip Lewandowski: *Granzyme B at the crossroads of thyroid autoimmunity and head and neck cancer*
- OP2 MSc Weronika Kruczkowska: *Decoding Glioblastoma's Resistance: How Immune Landscape Shields Tumor Cells from Chemotherapy*
- OP3 MSc Ewa Dejnaka: *Linking mitochondrial metabolism and tumor microenvironment: estrogen-related receptor alpha in canine bladder cancer*
- OP4 Michał Mikitiuk, PhD: *Development of Lysosome Targeting Chimera (LYTAC) for protein degradation in cancer immunotherapy*
- OP5 Jan Barczyński, PhD: *Preclinical development of ROR-1 x PD-L1 and Mesothelin x PD-L1 bispecific antibodies*
- OP6 Michał Skroński, PhD: *MELBA study – analysis of immune checkpoint biomarkers expression in patients with melanoma, lung and gastric cancers*

15:15 - 15:30 Sponsor's speech: Becton Dickinson
15:30 - 16:00 Coffee break
16:00 - 18:00 Workshop 2: The use of real-time PCR in SNP genotyping (capacity: 10)

Networking event:
19:00 - 21:00 **Axe throwing**: 7 siekier **Adress**: Tęczowa 25, 53-601 Wrocław (the organizer provides refreshments on site)

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# Day 2, Wednesday 18.03.2026

8:30 - 12:30 Registration
9:00 - 10:00 Keynote speech 2
**Ondrej Stepanek, PhD**
(Institute of Molecular Genetics of the Czech Academy of Sciences)
*„Signaling pathways shape the outcomes of T-cell fate choices"*
10:00 - 10:30 Coffee break
10:30 - 11:30 Session 2: Innate Immunology
Chair: **Prof. Joanna Cichy** (Jagiellonian University)

- OP7 Ewa Oleszycka, PhD: *Context Matters: The effect of metabolite derivatives on distinct macrophage population*
- OP8 Ilona Aylott, PhD: *Exogenous G-CSF mobilizes an unusual c-kit$^+$ mature neutrophil cluster absent with CoPP-Induced Endogenous G-CSF*
- OP9 MSc Hubert Kasprzak: *Analysis of pro- and anti-inflammatory gene response patterns in patients receiving phage therapy: the impact of biological age*
- *OP10 MSc Bartosz Hanczaruk: The effectiveness of innovative acellular dermal matrices derived from abdominoplasty skin in the management of chronic diabetic wounds.*

11:30 - 11:45 Sponsor's speech: Omixys
11:45 - 12:45 Invited speaker 1
**Agnieszka Razim, PhD** (Institute of Immunology and Experimental Therapy of the Polish Academy of Sciences)
*„Building your academic CV: defining long-term goals, writing competitive research proposals and more"*
12:45 - 13:30 Lunch

IMMUNOBRIDGE

# Day 2, Wednesday 18.03.2026

| | |
|---|---|
| 13:30 - 14:15 Session 3: Neuroimmunology<br>Chair: **Ali Jawaid, MD, PhD** (Lukasiewicz PORT-Polish Center for Technology Development)<br>OP11 Jan Zakrzewski: TDP-43 Aggregation as a Driver of Immune Dysregulation: Emerging Pathways of Microglial Reactivity in Amyotrophic Lateral Sclerosis<br>OP12 MSc Natalia Pocałuń: Keratinocyte-Specific Regnase-1 Deficiency Alters Neuroimmune Crosstalk, Inflammation Kinetics, and Scratching Behavior in an MC903-Induced Model of Atopic Dermatitis<br>OP13 MSc Nicole Kryniecka: From Gums to the Brain: Could Periodontal Disease Contribute to Alzheimer's Disease? | 13.15-15.15 Workshop 3: 3D Cell Imaging Using the Tomocube HT-2 Microscope (group 1, capacity: 5) |

IMMUNOBRIDGE

# Day 2, Wednesday 18.03.2026

14:00 - 15:00 Invited speaker 2
**Ali Jawaid, MD, PhD** (Lukasiewicz PORT- Polish Center for Technology Development)
*„Immunometabolic pathways of brain resilience and vulnerability across the lifespan"*
15:00 - 15:30 Coffee break

| | |
|---|---|
| 15:30 - 17:30 Flashtalk session + Workshop 4<br>The Art of Public Speaking<br>Chair: **Ali Jawaid, MD, PhD** (Lukasiewicz PORT- Polish Center for Technology Development)<br>OF1 MSc Michalina Pęcherz: Physiological oxygen level during endothelial progenitor cell culture increases vessel normalizing potential of extracellular vesicles<br>OF2 MSc Agnieszka Barbach: The significance of heat shock protein 90 in calcitriol and tacalcitol anticancer activity against colorectal cancer cells<br>OF3 BSc Zuzanna Pochwała: Interferons as Mediators of Neuroinflammation and Synaptic Loss in Alzheimer's Disease | 15:15 - 17:15 Workshop 3: 3D Cell Imaging Using the Tomocube HT-2 Microscope (group 2, capacity: 5) |

Networking event:
19:00 - 21:00 **Bowling** Location: Sky Tower, Sky Bowling **Address**: Powstańców Śląskich 95, 53-332 Wrocław (the organizer provides refreshments on site)

IMMUNOBRIDGE

# Day 3, Thursday
# 19.03.2026

8:30 - 13:00 Registration
9:00 - 10:00 Keynote speech 3
**Katarzyna Sitnik, PhD** (University of Veterinary Medicine Vienna, Austria)
*„Lymphoid Tissue Fibroblasts in Immunity and Infection"*
10:00 - 10:30 Coffee break
10:30 - 11:30 Session 4: Immunology of infectious diseases
Chair: **Assoc. Prof. Mariola Paściak** (Institute of Immunology and Experimental Therapy of the Polish Academy of Sciences)

- OP14 MSc Patrycja Burzyńska: *Sialic acid specificity of Laverania Erythrocyte Binding Antigen 140 (EBA-140)*
- OP15 MSc Szymon Mazgaj: *Neutrophil extracellular traps (NETs) in immunity and viral infections*
- OP16 MSc Mariia Tyshchenko: *Keratinocyte-intrinsic Regnase-1 regulates inflammatory skin response to Staphylococcus aureus infection*
- OP17 MSc Laura Ribes-Mertinez: *Preclinical therapeutic potential of phage FT5P against vancomycin-resistant Enterococcus feacium infections*

11:30 - 12:30 Invited speaker 3
**Louis Boon, PhD** (JJP Biologics)
*„Anti-CD89 Antibody for Personalized Treatment of IgA-Mediated Inflammatory Disorders: Autoantigen-Specific IgA, a Biomarker with Strong Effector Functions"*
12:30 - 13:15 Lunch
13:15 - 13:45 Session 5: Microbiome and Immunology
Chair: **Aleksandra Kołodziejczyk, PhD**
(International Institute of Molecular and Cell Biology in Warsaw)

- OP18 MSc Subhasree Venkatasubramaniyan: *Developement of a universal chemically-defined medium for the analysis of „stressed" bacterial EVs in the context of undernutrition*
- OP19 MSc Natalia Rzepka: *Dissecting dendritic cell–microbiota interactions in the intestines: role of bacterial metabolites in shaping adaptive immunity*

13:45 - 14:45 Invited speaker 4
**Aleksandra Kołodziejczyk, PhD**
(International Institute of Molecular and Cell Biology in Warsaw)
*„Host-microbiota interactions in health and disease"*

IMMUNOBRIDGE

# Day 3, Thursday
# 19.03.2026

**14:45 - 15:45** Invited speaker 5
**Assoc. Prof. Wojciech Juzwa** (Poznań University of Life Sciences;Cyto-Logic)
*„Who Is Who in the Microbiome? Single-Cell Technologies, Flow Cytometry, and the Architecture of Gut Microbial Communities"*
**15:45 - 16:15** Coffee break
**16:00 - 17:30** Poster session + Best Poster Awards
Oncoimmunology

- PP1 MSc Anas Waer-Asea: *Modulation of Tumor Immunogenic Phenotype by Electroporation under Normoxia and Hypoxia*
- PP2 MSc Karolina Grycuk: *RVU120 Potentiates NK Cell–Mediated ADCC in Combination with Therapeutic Antibodies*
- PP3 MSc Anna Rudawska: *Bone marrow-derived macrophages loaded with boron carbide nanoparticles as selective boron carriers for crossing the blood-brain barrier toward glioma for boron neutron capture therapy*
- PP4 MSc Anna Andrzejczak: *Distinct Mechanisms, Shared Goal: CAR-T and TCR-T Cell Therapies in Hematologic Malignancies*
- PP5 MSc Krystian Sarat: *Optimizing CAR-T Expansion Conditions for the First Polish Academic Multiple Myeloma Therapy*
- PP6 BSc Szymon Więcek: *Expression and gene polymorphism of miR-21 affect progression-free survival in multiple myeloma patients*
- PP7 Jagoda Mierzejwska, PhD: *Impact of Stattic analogs on STAT3 expression and cytokine secretion in restimulated splenocytes obtained from MC38-bearing mice vaccinated with genetically modified dendritic cells*
- PP8 Agnieszka Szczygieł, PhD: *Effect of Stattic analogs on STAT3 expression and cytokine secretion in genetically modified dendritic cells applied as anticancer vaccines*
- PP9 MSc Katarzyna Węgierek-Ciura: *Co-delivery of IL-12/IL-15/IL-18 engineered dendritic cells in the murine colon cancer model*
- PP10 MSc Julia Depta: *TIGIT in non-small cell lung cancer and its major subtypes, adenocarcinoma and squamous cell carcinoma: evaluation of the TIGIT promoter methylation as well as mRNA and protein expression levels*

IMMUNOBRIDGE

# Day 3, Thursday
# 19.03.2026

Immunology of infectious diseases

- PP11 MSc Weronika Krzyszczuk: *Porphyromonas gingivalis attenuates Herpesviruses replication by gingipains-mediated virion destabilization*
- PP12 BSc Monika Jakoby: *Immunomodulatory and Antiviral Properties of Spirulina platensis: Mechanisms and Therapeutic Potential*
- PP13 MSc Martyna Cieślik: *Phage interactions with gut epithelium confirm the safety of phage therapy after oral administration*

Neuroimmunology

- PP14 BSc Wiktoria Grzybowska: *Beyond Amyloid and Tau: Emerging Biomarkers for Early Diagnosis of Alzheimer's Disease*
- PP15 MSc Dominika Kozakiewicz: *Gut Microbiota Products Modulate Blood–Brain Barrier Integrity: Insights from Bifidobacterium animalis BEVs*

Hipersensitivity

- PP16 MSc Aleksandra Wiekiera: *Immunomodulatory effects of proteolytically inactive gingipains on osteoblast and osteoclast*
- PP17 Patrycja Kwiecińska, PhD: *Investigating Hematopoietic Memory in Long-Term Hematopoietic Stem Cells*
- PP18 BSc Hanna Krzyżanowska: *Polymorphism within gene coding for activating NKG2D receptor and aging – study on a group of healthy Polish representatives*

Microbiome and Immunology

- PP19 MSc Anna Karlik: *Synbiotics Reduce Hypercholesterolemia and Aortic Inflammation in ApoE Knockout Mice via Intestinal Cholesterol Metabolism and T Cell Modulation*

Innate Immunology

- PP20 BSc Magdalena Szaczkowska: *AREL1 E3 Ubiquitin Ligase as a Modulator of Innate Immune Signaling*
- PP21 MSc Karolina Rusewicz: *Understanding the function of DTX3L ligase in the antiviral response induced by pattern recognition receptors (PRRs)*

Networking event:
19:00 - 21:00 **Escape room** Exit 19 **Address**: Ofiar Oświęcimskich 19, 50-069 Wrocław

IMMUNOBRIDGE

# Day 4, Friday
# 20.03.2026

9:00 - 10:00 Keynote speech 4
**Róisín McManus, PhD** (Deutsches Zentrum für Neurodegenerative Erkrankungen)
*„Microglial innate immune signalling as a driver of dementia"*
10:00 - 10:30 Coffee break
10:30 - 11:30 Session 6: Hipersensitivity
Chair: **Assoc. Prof. Andrzej Eljaszewicz** (Medical University of Białystok)

- OP20 MSc Natalia Zagórska: *Genistein modulates autoimmune inflammation in epidermolysis bullosa acquisita*
- OP21 MSc Kornelia Kłosińska: *Comprehensive transcriptomic profiling of circulating platelets in patients with ankylosing spondylitis*
- OP22 MSc Angelika Kruszyńska: *Potential mechanism of lymphoid cell death induced by anti-MHC II antibodies*
- *OP23* Marlena Tynecka, PhD: *Immunosuppressive mesenchymal stem cells regulate arachidonic acid metabolism in experimental T2 low asthma*

11:30 - 12:30 Invited speaker 6
**Assoc. Prof. Andrzej Eljaszewicz** (Medical University of Białystok)
*„Beyond inflammation: the role of allergen proteases in airway remodelling"*
12:30 - 13:00 Official Closing + Best Presentation Awards
13:00 - 13:45 Lunch + Farewell

IMMUNOBRIDGE

# OP1 Oncoimmunology

## Granzyme B at the crossroads of thyroid autoimmunity and head & neck cancer

Filip Lewandowski[1,2,3], Paulina Niedźwiedzka-Rystwej[1,2]
1 Center for Experimental Immunology and Immunobiology of Infectious Diseases and Cancer, University of Szczecin, Poland
2 Second Institute of Biology, University of Szczecin, Poland
3 Doctoral School, University of Szczecin, Poland

Granzyme B (GzmB) is a key effector molecule of cytotoxic lymphocytes, including $CD8^+$ T cells and NK cells, and plays a central role in immune-mediated cytotoxicity [1]. While its function in tumor immunosurveillance is well established, its contribution to autoimmune thyroid diseases (AITD) remains incompletely understood [2]. The present study aimed to evaluate the cytotoxic immune profile, with particular emphasis on Granzyme B expression, in patients with thyroid disorders and to compare these findings with patients diagnosed with head and neck cancers (HNC).

Peripheral blood samples were collected from patients with autoimmune thyroid diseases and from HNC patients. Immunophenotyping of cytotoxic lymphocyte subsets was performed using multiparameter flow cytometry, focusing on $CD3^+CD8^+$ T cells and $CD3^-CD56^+$ NK cells. Granzyme B expression was assessed at both the protein and mRNA levels.

Our results demonstrate distinct patterns of cytotoxic immune activation between autoimmune and neoplastic conditions. Patients with thyroid diseases exhibited altered frequencies of cytotoxic lymphocyte subsets accompanied by modified Granzyme B expression, suggesting ongoing immune activation consistent with autoimmune pathology.

In contrast, HNC patients displayed features indicative of dysregulated or functionally impaired cytotoxic responses, reflected by changes in Granzyme B expression within effector cell populations.

These findings highlight divergent regulation of the cytotoxic axis in autoimmunity and cancer. Granzyme B may serve as a functional biomarker reflecting immune activation status in thyroid disorders and immune dysfunction in malignancy. Understanding these differences may contribute to improved immunological characterization of patients and provide a rationale for targeted immunomodulatory strategies in both autoimmune and oncological settings.

References

Majchrzak A., Lewandowski F., Hrynkiewicz R., Poniewierska-Baran A., Bębnowska D. and Niedźwiedzka-Rystwej P. (2025) Granzyme B and melittin in cancer immunotherapy: molecular mechanisms and therapeutic perspectives in head and neck cancers. Front. Immunol. 16:1628014. doi: 10.3389/fimmu.2025.1628014

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# OP2 Oncoimmunology

## Decoding Glioblastoma's Resistance: How Immune Landscape Shields Tumor Cells from Chemotherapy

Weronika Kruczkowska1, Damian Kołat1, Żaneta Kałuzińska- Kołat1,2, Elżbieta Płuciennik1
1 Departement of Functional Genomics, Medical University of Lodz, Lodz, Poland
2 Departement of Experimental Surgery, Medical University of Lodz, Lodz, Poland

Central nervous system tumors afflict 1.6-1.7% of cancer patients annually, with glioblastoma (GBM) representing the most lethal primary brain malignancy, bearing a devastating 5-year survival rate of merely 12.8% in Europe. Despite aggressive treatment protocols including maximal resection and temozolomide-based radio-chemotherapy, therapeutic resistance remains a formidable obstacle [1,2,3]. This study unveils a critical connection between tumor microenvironment (TME) complexity and chemoresistance in gliomas.

Employing computational deconvolution methods (ESTIMATE and TIMER) on RNA-sequencing data, we conducted comprehensive TME profiling across GBM and low-grade glioma (LGG) cohorts. GBM exhibited dramatically elevated stromal ($p=8.1\times10^{-26}$), immune ($p=2.1\times10^{-31}$), and combined ESTIMATE scores ($p=8.3\times10^{-36}$), indicating substantially enriched microenvironmental infiltration. Detailed immune cell analysis revealed significant GBM enrichment of B lymphocytes ($p=0.00287$), CD8+ T cells ($p=4.1\times10^{-8}$), neutrophils ($p=0.00707$), and macrophages ($p=0.00213$).

Strikingly, chemosensitivity profiling using CancerRxTissue algorithms demonstrated that LGG cells require significantly lower drug concentrations for 50% growth inhibition compared to GBM for both temozolomide ($p=6.35\times10^{-48}$) and carmustine ($p=3.61\times10^{-39}$). This inverse relationship between TME complexity and drug sensitivity suggests that specific immune populations—particularly tumor-associated macrophages—actively orchestrate resistance mechanisms through survival factor secretion, physical barrier formation, or metabolic alterations affecting drug bioavailability.

These findings imply that TME may play a role as an active resistance mediator rather than passive bystander, positioning microenvironment-targeted therapies as promising strategies to overcome chemoresistance. Our bioinformatics approach demonstrates the power of computational deconvolution in identifying actionable therapeutic vulnerabilities, advancing personalized medicine for this devastating disease.

References

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# OP3 Oncoimmunology

## Linking mitochondrial metabolism and tumor microenvironment: estrogen-related receptor alpha in canine bladder cancer

Ewa Dejnaka1, Aleksandra Pawlak1
*1 Wroclaw University of Environmental and Life Sciences, Faculty of Veterinary Medicine, Department of Pharmacology and Toxicology, Wroclaw, Poland*

Increasing evidence indicates that metabolic reprogramming in cancer cells plays a critical role in shaping the tumor microenvironment (TME), which influences antitumor immunity and effectiveness of immunotherapy. In-depth understanding of TME dynamic enables the identification of mechanisms of immune evasion and supports the development of combination strategies that enhance anti-cancer therapies (1). Estrogen-related receptor alpha (ERRα/*ESRRA*) is an orphan nuclear receptor and a master regulator of mitochondrial biogenesis and oxidative metabolism, which is overexpressed in many types of tumors such as breast, prostate, ovarian, colon and oral cancers (2-4). Emerging evidence suggests ERRα modulates TME by promoting immunosuppressive phenotypes, angiogenesis, and metabolic crosstalk with stromal cells (5). However, the contribution of ERRα to TME modulation remains an unexplored area, particularly in comparative oncology models. To address this gap, we focused on canine bladder cancer, which serves as a valuable spontaneous model that mirrors key molecular and microenvironmental features of human muscle-invasive bladder cancer.

This study investigates the *ESRRA* expression in established canine bladder cancer cell lines and patient-derived organoids. We evaluated gene expression level using quantitative PCR (qPCR) using the fluorescent dye SYBR Green.

*ESRRA* expression was detected in all tested cell lines and organoids at the mRNA level.

Given ERRα's known function in regulating mitochondrial metabolism and the TME, confirmation of its expression in canine bladder cancer positions ERRα as a potential new target for anticancer therapy in this type of tumor. Further functional and translational studies are necessary to determine whether targeting ERRα could contribute to metabolic-immune therapeutic strategies in canine and human diseases.

**References**

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2. Ariazi, E., Clark, G., Mertz, J. Estrogen-related receptor alpha and estrogen-related receptor gamma associate with unfavorable and favorable biomarkers, respectively, in human breast cancer. *Cancer Res* **15**, 6510-8 (2002)
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# OP4 Oncoimmunology

## Development of Lysosome Targeting Chimera (LYTAC) for protein degradation in cancer immunotherapy

Michał Mikitiuk1
1JJP Biologics Protein Engineering Lab, Trzy Lipy 3, Gdańsk, Poland

Immunotherapy, using checkpoint inhibitors to block the PD-1- PD-L1 axis, has evolved to the cornerstone of cancer treatment and has dramatically improved the outlook for many cancer patients. Unfortunately, not all patients respond equally to immunotherapy and although the mechanism behind this remains to be elucidated, individual patient differences within the same indication can be significant. Alternative molecules with innovative mechanisms of action are urgently needed for this group of refractory patients. An example of such an approach is using Lysosome Targeting Chimera (LYTAC), which are bispecific, antibody-based molecules that target a selected molecular protein target (intended for degradation) and simultaneously a receptor inducing lysosomal trafficking. In the current program, we developed a PD-L1 targeting LYTAC molecule using an anti-PD-L1 and a high -affinity peptide to bind Insulin-like Growth Factor 2 Receptor. These PD-L1-LYTACs can induce internalization of PD-L1 in both soluble and transmembrane forms in a time- and concentration-dependent manner. Theoretically, these PD-L1-LYTAC molecules should induce a response analogous to the immune checkpoint blockade achieved with anti-PD-L1 antibodies. Surprisingly, the engineered LYTACs inhibited the proliferation of cancer cell-lines better and induced significantly higher levels of tumor cell lysis by human PBMCs in vitro compared to therapeutically approved anti-PD-L1 antibodies. Targeted degradation technologies offer an alternative and promising mechanism to be applied into future therapies, targeting not only oncological indications, but also other disease indications for which the discovery of inhibitors using classical methods of rational drug design is impossible.

# OP5 Oncoimmunology

## Preclinical development of ROR-1 x PD-L1 and Mesothelin x PD-L1 bispecific antibodies

Jan Barczyński1
1 1JJP Biologics Bioanalytics Lab, Trzy Lipy 3, Gdańsk, Poland

The PD-1-PD-L1 axis has been identified as one of the most effective approaches for cancer treatment. Unfortunately, a significant group of patients in various cancers develop resistance. ROR-1 and Mesothelin are proteins that have minimal presence in healthy tissues but are highly overexpressed in several oncological indications, in which these proteins are involved in proliferation and survival. Moreover, their presence on cancer cells has been correlated with poor prognose. PD-L1, in turn, is one of the best-characterized immune checkpoint regulators, identified as a source of immune evasion for many types of cancer. The aim of the current program was to analyze the potential therapeutic value of symmetric bispecific antibody modalities being, ROR-1 x PD-L1 and Mesothelin x PD-L1 with and without effector functions as IgG1 and IgG4, respectively. The obtained bispecific antibodies exhibited the expected physicochemical properties and demonstrated the ability to bind their respective molecular targets and block PD-1/PD-L1 interactions with similar efficacy as anti-PD-L1 antibodies. Functional activity of the bispecific antibodies was assessed in co-cultures of PBMCs and cancer cells (lines Panc1, Panc 05.04, BT20, and RL95-2) and resulted in a significantly increased cancer cell lysis compared to anti-PD-L1 antibodies, with the level of lysis depending on the antibody concentration. Additionally, in co-cultures with Panc1 and RL95-2 cells, a significantly elevated level of cytokines (IL-10, TNFα, IFNγ, Granzyme B) and a reduction in regulatory T-cell subpopulations (CD4+ CD25+ CD127low FoxP3hi and CD4- FoxP3+) were observed. The Mesothelin x PD-L1 antibodies promoted a more effective anti-cancer response than the ROR-1 x PD-L1 antibodies, and IgG1 antibodies exhibited slightly higher activity than IgG4 antibodies, indicating that the IgG1 effector functions had limited contribution. In summary, the use of bispecific antibodies for the simultaneous blockade of ROR-1 and PD-L1 or Mesothelin and PD-L1 is an innovative and promising approach to cancer therapy. However, confirming their effectiveness in more complex models, like tumor spheroids or an animal model, remains to be confirmed.

# OP6 Oncoimmunology

## MELBA study – analysis of immune checkpoint biomarkers expression in patients with melanoma, lung and gastric cancers

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MELBA is an observational, single-center study focused on the analysis of immune checkpoint (IC) biomarkers related to CD270 (HVEM) IC, as well as CD279 (PD-1)/CD274 (PD-L1) in patients with melanoma, lung, and gastric cancer over the course of their immunotherapy with immune-checkpoint inhibitors (ICI). CD270 has 3 known major ligands, of which two - CD160 and CD272 (BTLA) are dominant and immunosuppressive, while CD258 (LIGHT) is immune-activating. High expression of the CD270 on various tumors correlates with poor survival and an immunosuppressive TME, which makes HVEM a potential therapeutic target.

The objectives of the study is to investigate longitudinal dynamics of blood-based soluble IC biomarkers: CD270, CD272, CD160, CD258, CD279 and CD274 in patients treated with standard-of-care (SoC) ICI and to assess the relation between these biomarkers and response to treatment.

The study population includes 150 cancer patients to be enrolled in DCOPiH.

Study analyses will be conducted in cancer tissue samples and patient's blood. Blood samples will be obtained at baseline (before therapy), at 1 and 3 months after the ICI administration, as well as at the time of progression or 6 months after ICI treatment, whichever comes first.

Soluble IC biomarkers levels in serum will be measured with ELISA, while their expression on blood immune cells with flow cytometry and in tumor tissues with immunohistochemistry. Plasma will be used to isolate circulating tumor DNA (ctDNA) for molecular response assessment by next-generation sequencing.

The recruitment will start in Q1 2026, and the results of the study are expected mid-2027.

# OP7 Innate Immunology

## Context Matters: The effect of Metabolite Derivatives on Distinct Macrophage Populations

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4Department of Medical Biotechnology, Faculty of Biochemistry, Biophysics, and Biotechnology, Jagiellonian University, Kraków, Poland

Secretory leukocyte protease inhibitor (SLPI) is a potent inhibitor of serine proteases with additional anti-inflammatory and antimicrobial properties that are crucial for the regulation of inflammation and tissue homeostasis. Although its physiological and pathological roles are increasingly appreciated, the molecular and cellular mechanisms controlling SLPI expression remain incompletely understood. Identifying factors that enhance SLPI production may therefore provide new therapeutic opportunities. Here, we demonstrate that SLPI is produced by activated murine macrophages in an NRF2-dependent manner. In this context, SLPI induction is independent of endogenous itaconate synthesis and instead correlates with intracellular fumarate accumulation. Importantly, our data reveal marked differences between macrophage populations. In activated bone marrow–derived macrophages (BMDMs), SLPI production is strongly enhanced by itaconate and fumarate derivatives that activate NRF2 signaling. In contrast, tissue-resident macrophages display a distinct regulatory pattern. Although LPS stimulation induces SLPI secretion in these cells, treatment with itaconate derivatives suppresses SLPI production in an NRF2-dependent manner.

Collectively, our findings highlight fundamental differences between BMDMs and tissue-resident macrophages in the metabolic regulation of SLPI and demonstrate that immunometabolic interventions can exert divergent effects depending on macrophage origin and cellular context.

# OP8 Innate Immunology

## Exogenous G-CSF mobilizes an unusual c-kit⁺ mature neutrophil cluster absent with CoPP-Induced Endogenous G-CSF

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Emergency granulopoiesis supports host defense during stress states such as chemotherapy-induced neutropenia and inflammation, yet how distinct mobilisation stimuli shape granulocyte output remains incompletely understood. Standard G-CSF–based mobilisation is clinically effective, but the degree of mobilisation varies among patients, motivating interest in alternative approaches that may differentially influence granulocyte maturation and function.

Cobalt protoporphyrin IX (CoPP) is an alternative agent that induces endogenous G-CSF and additional cytokines, and has been reported to mobilize higher numbers of mature granulocytes than recombinant G-CSF (1). We therefore investigated phenotypic and functional differences between granulocyte populations released into peripheral blood following G-CSF or CoPP treatment.

Using unsupervised flow cytometry clustering analysis and immunophenotyping, we identified two distinct granulocyte populations distinguished by c-kit expression: Ly6Gint/hi c-kit$^+$ and Ly6Ghi c-kit$^-$ cells. A c-kit$^+$ granulocyte population was preferentially mobilised following G-CSF treatment but was largely absent after CoPP administration, leading to the hypothesis that c-kit$^+$ granulocytes represented a more immature subset. However, assessment of granulocyte morphology and phagocytic capacity revealed only minor differences in baseline functional and maturational properties between c-kit$^+$ and c-kit$^-$ granulocytes.

Together, these data indicate that c-kit–defined granulocyte populations mobilised during therapeutically driven mobilisation represent phenotypically distinct states with largely overlapping functional capacity, highlighting granulocyte heterogeneity without overt functional divergence.

Acknowledgements
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# OP9 Innate Immunology

## Analysis of pro- and anti-inflammatory gene response patterns in patients receiving phage therapy: the impact of biological age

Hubert Kasprzak1, Maciej Przybylski2, Wojciech Fortuna3, Sławomir Letkiewicz3,4, Paweł Rogóż3, Barbara Bubak1 Andrzej Górski1,3, and Ryszard Międzybrodzki1,3,5
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While bacteriophages are well known for their bactericidal properties, their immunomodulatory effects remain largely unexplored. This study investigates both pro- and anti-inflammatory gene response patterns in patients undergoing phage therapy (PT). Cell populations were isolated from blood samples collected from ten patients before and during PT. The patients received personalized PT due to various chronic bacterial infections resistant to antibiotic treatment. RNA extraction and quantitative PCR (qPCR) were performed to evaluate changes in the expression of 22 selected cytokines, pattern recognition receptors, and signaling molecules in peripheral blood mononuclear cells (PBMCs) and granulocytes. Our findings revealed high inter-individual variability in gene expression change profiles among patients undergoing PT. Although no consistent universal inflammatory pattern emerged, further analyses using hierarchical clustering revealed distinct, compartment-specific regulatory architectures for PBMCs and granulocytes. Furthermore, biological age was identified as a potential determinant of the host response. Specifically, older patients showed higher activation of the innate sensing machinery (e.g., TLR4) in PBMCs, potentially reflecting an age-associated immune "priming". This study demonstrated that chronically administered bacteriophages do not trigger a predictable, systemic immune activation in PT patients. Instead, the immune response is highly individualized and appears to be modulated by specific host-related biological factors, such as age and exhibits distinct compartment-specific dynamics depending on the leukocyte subpopulation. As an exploratory investigation, this work provides essential baseline data and generates hypotheses to guide the rational design of future preclinical and clinical investigations.

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# OP10 Innate Immunology

## The effectiveness of innovative acellular dermal matrices derived from abdominoplasty skin in the management of chronic diabetic wounds

Bartosz Hanczaruk1, Krystian Czołpiński1, Adrian Janucik1, Jordan Holl1, Dawid Groth1, Alicja Walewska1, Marlena Tynecka1, Marcin Moniuszko1,2, Hady Razak Hady3, Andrzej Eljaszewicz1,4
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Chronic wounds represent a significant global health challenge. To address this problem, our research group has developed an innovative human acellular dermal matrix (hADM) derived from abdominoplasty skin from bariatric patients. Here, we aimed to assess the effectiveness of our novel hADM in preclinical models of diabetic wounds.

Skin from post-bariatric patients underwent decellularization using three distinct protocols: hADM1 (1M NaCl and SDS), hADM2 (2M NaCl and SDS), and hADM3 (trypsin and Triton X-100)1. The structural integrity of the extracellular matrix (ECM) and the presence of cellular components were assessed through immunohistochemical and histochemical staining. Additionally, the immunogenic potential of each protocol was examined using a T-cell proliferation assay.

The therapeutic potential of novel hADMs was assessed in both wild-type (WT) mice2 and the leptin receptor knockout diabetic mouse model (db/db), to examine diabetic wound healing. The wound-healing process was monitored daily, and gene expression was analyzed. Among them, the immunogenic potential was lowest in hADM1. The efficacy of all hADM was similar during the inflammatory phase of wound healing. However, in the course of the proliferative phase, hADM1 significantly accelerated wound closure. This was associated with the upregulation of genes involved in collagen synthesis (Col5a3, Col5a2), integrin expression (Itga6, Itgb3), and matrix remodeling enzymes (Mmp9, Mmp1a).

Overall, our findings underscore the potential of hADM1 as a promising treatment for managing chronic diabetic wounds. Nevertheless, further studies are essential to understand the mechanisms underlying its beneficial effects.

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# OP11 Neuroimmunology

## TDP-43 Aggregation as a Driver of Immune Dysregulation: Emerging Pathways of Microglial Reactivity in Amyotrophic Lateral Sclerosis

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Background: TDP-43 is an RNA/DNA-binding protein implicated in the pathogenesis of several neurodegenerative disorders, including amyotrophic lateral sclerosis. Emerging evidence suggests that TDP-43 loss in microglia alters their functional state, contributing to exaggerated synaptic engulfment. This study aims to demonstrate the impact of TDP-43 loss of function on microglial intracellular redox homeostasis, expression of selected immunometabolic genes and the central mediating role of cellular metabolic pathways in exaggerated phagocytosis.

Materials and Methods: TDP-43 was knocked down in murine BV2 and human HMC3 microglial cells using RNA interference. The depletion's effects were studied using reactive oxygen species (ROS) production assays and the quantification of the expression of chosen immunometabolic genes. Live-cell metabolic flux analysis (MFA) was performed after TDP-43 knockdown under glycolytic and mitochondrial stress conditions. Synaptoneurosome uptake was quantified in cells with TDP-43 knockdown before and after metabolic rerouting.

Results: TDP-43 depletion was associated with increased microglial glycolysis in both cell lines, without a concomitant decrease in mitochondrial OXPHOS. This was corroborated by increased glucose uptake, lactate production, and intracellular ATP and ROS levels. Several immunometabolic genes have been shown to demonstrate differential expression. TDP-43 knockdown led to exaggerated engulfment of synaptoneurosomes, which was reversed under metabolic rerouting via supplementation with pyruvate-containing, glucose-free medium.

Conclusions: The study's findings indicate that TDP-43 LOF is mechanistically linked to microglial hyperactivity. Beyond modulating the cellular metabolic profile, it also appears to disrupt redox homeostasis and alter the expression of key genes, thereby further reinforcing the existing literature implicating immunological pathways in ALS pathology.

# OP12 Neuroimmunology

## Keratinocyte-Specific Regnase-1 Deficiency Alters Neuroimmune Crosstalk, Inflammation Kinetics, and Scratching Behavior in an MC903-Induced Model of Atopic Dermatitis

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Atopic dermatitis (AD) is a chronic inflammatory skin disorder characterized by immune dysregulation, epidermal barrier impairment, and severe pruritus driven by neuroimmune interactions. Keratinocytes actively contribute to this process by releasing cytokines and alarmins that modulate both immune cell recruitment and sensory neuron activation. Regnase-1 (MCPIP1) is an RNase that post-transcriptionally regulates inflammatory mediators; however, its role in cutaneous neuroimmune crosstalk remains poorly defined.

We investigated the impact of keratinocyte-specific Regnase-1 deficiency in an MC903-induced AD-like mouse model. Wild-type (WT) and keratinocyte-specific Regnase-1 knockout (Reg1 cKO) mice were treated topically with MC903 for 3, 7, and 14 days. Disease severity was evaluated by ear thickness measurements and clinical scoring. Scratching behavior was quantified to assess pruritus intensity. Histological analysis and flow cytometry were performed to characterize epidermal hyperplasia and immune cell infiltration in skin and peripheral immune organs.

Reg1 cKO mice exhibited temporally altered inflammatory responses compared to WT controls. After 7 days, knockout mice showed significantly increased ear thickness and enhanced immune cell infiltration. Notably, Reg1 cKO mice displayed significant differences in scratching behavior, with altered intensity and dynamics relative to WT animals, indicating dysregulated neuroimmune signaling. By day 14, skin lesions in Reg1 cKO mice extended beyond the treated area despite reduced local immune infiltration compared to WT mice. Additionally, Reg1 cKO mice demonstrated increased leukocyte reservoirs in the bone marrow, suggesting altered systemic immune mobilization.

These findings identify Regnase-1 as a key modulator of both inflammatory and neuroimmune pathways in AD-like skin inflammation.

# OP13 Neuroimmunology

## From Gums to the Brain: Could Periodontal Disease Contribute to Alzheimer's Disease?

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Alzheimer's disease (AD) is the most common cause of dementia and represents a growing global health challenge. According to the Alzheimer's Association over 55 million people worldwide are currently living with dementia, with AD accounting for 60–70% of cases and prevalence is expected to rise dramatically with population aging. The disease is characterized by a long preclinical phase, during which neuropathological changes accumulate years before the onset of cognitive symptoms, highlighting the importance of identifying modifiable risk factors and novel pathogenic mechanisms [1].

For decades the amyloid cascade hypothesis has dominated AD research however, growing evidence suggests that amyloid deposition alone is insufficient to fully explain disease onset and progression. Consequently, alternative and complementary hypotheses have gained increasing attention. Among them, the infectious hypothesis of AD proposes that chronic peripheral infections and sustained systemic inflammation may contribute to neuroinflammation, blood–brain barrier (BBB) dysfunction and neurodegeneration [2].

Periodontal disease (PeD) is a highly prevalent chronic oral inflammatory disease, affecting nearly 50% of adults worldwide and up to 70% of individuals over 65 years of age. It is driven by complex dysbiotic biofilms composed of periopathogens classified by Socransky into distinct microbial complexes. While much of the existing research has focused on Porphyromonas gingivalis and its gingipains, periodontitis involves a broad spectrum of pathogenic species -. Importantly, periopathogens and its metabolites may penetrate peripheral tissues, enter the circulation and potentially cross the BBB, thereby exerting effects on the central nervous system [3,4]. It was presented earlier by our team that poor oral and periodontal health may contribute to cognitive impairment and AD progression. Even mild inflammation in periodontal tissue or gingivitis may already influence peripheral immune cell conditions, which in turn might be related to negative consequences for the brain and mental health [5].

Based on these observations, we are conducting a medical experiment investigating whether PeD treatment restore the balance of the oral microbiome, influences systemic and neuroinflammatory immune markers, as well as cognitive functions in patients at risk of cognitive decline. This research aims to clarify the potential contribution of periodontal inflammation to AD pathogenesis and to explore PeD therapy as a modifiable intervention within a broader preventive strategy.

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# OP14 Immunology of infectious diseases

## Sialic acid specificity of Laverania Erythrocyte Binding Antigen 140 (EBA-140)

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There are an estimated 260 millions malaria cases and up to 600,000 deaths per year caused by parasites of the genus Plasmodium, among which the most malignant species, P. falciparum, belongs to the Laverania subgenus. Parasites of the Laverania subgenus infect both humans and great apes and are largely host-specific. The factors controlling host-specificity are likely multifactorial, but the ability to recognize host receptors has been proposed as one of the potential mechanisms. Understanding these processes is essential for designing vaccines and other tools against malaria.
The Erythrocyte Binding Antigen-140 (EBA-140) is one of the ligands involved in erythrocyte recognition, and all Laverania species possess its orthologue. EBA-140 binds erythrocytes in sialic acid-dependent manner. It has been proposed that differences in human and ape sialic acid (SA) patterns may lead to species specific differences in invasion ligand binding.
We expressed recombinant EBA-140 antigens from P. falciparum (a human parasite) and its closest ancestor, P. praefalciparum (an ape parasite) and assess their ability to bind SAs. Both antigens showed only minor differences in their affinity for human versus ape SAs. We then generated transgenic P. falciparum lines in which part of the native PfEBA-140 was replaced with a sequence from P. praefalciparum. We observed significant growth differences between the transgenic and wild-type lines, but no differences in invasion efficiency were detected among the compared lines.
Our results demonstrate that EBA-140 is not a determinant of host specificity and that its role in Plasmodium invasion can be compensated for by other ligands.

# OP15 Immunology of infectious diseases

## Neutrophil extracellular traps (NETs) in immunity and viral infections

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NETs are one of the key mechanisms of the innate immune response. In response to viruses, neutrophils can release extracellular traps composed of chromatin and antimicrobial proteins. A growing body of research indicates that NETs play a significant role in viral infections. Therefore, the aim was to present the current state of knowledge on the involvement and function of NETs in viral infections in humans and animals, with particular emphasis on the molecular exponents of NETs. NETs, on the one hand, serve a protective function—the resulting networks can capture and immobilize viruses, limiting their spread and the number of infected cells. On the other hand, excessive or abnormally regulated NETs activation is associated with several adverse effects. Importantly, NETs components—including MPO, NE, free DNA, and histones—may serve as valuable biomarkers of disease severity and the degree of neutrophil activation, supporting the diagnosis and assessment of the dynamic course of viral infections.

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# OP16 Immunology of infectious diseases

## Keratinocyte-intrinsic Regnase-1 regulates inflammatory skin response to Staphylococcus aureus infection

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Staphylococcus aureus is a major pathogen implicated in the exacerbation of Atopic Dermatitis (AD), a condition characterized by barrier defects and immune dysregulation. Regnase-1 (Reg1) is an RNA-binding protein essential for immune homeostasis, known to prevent excessive inflammation through degradation of pro-inflammatory cytokine mRNAs. Interestingly, Reg1 expression is aberrant in AD skin lesions, yet the functional consequences of it in keratinocytes during bacterial infection remain poorly understood.

In this study, we investigated the role of Reg1 in skin host defense using a mouse model with keratinocyte-specific deletion of Reg1. To mimic barrier dysfunction, we established a topical S. aureus infection model. Considering the documented role of Reg1 as a negative regulator of inflammation, we hypothesized that its loss would exacerbate inflammatory pathology. Contrarily, Reg1 deficiency conferred a protective phenotype. While wild-type mice developed severe skin lesions characterized by erythema and erosion, Reg1-deficient mice exhibited significantly reduced disease severity.

These findings challenge the conventional view of Reg1 solely as a suppressor of inflammation and suggest that its downregulation in keratinocytes may activate compensatory antimicrobial or tissue-protective pathways. Although the phenotypic outcome is striking, the molecular mechanisms underlying this resistance remain to be elucidated.

Overall, our study reveals an unanticipated role for Reg1 in epithelial-microbial interactions and provides new insights into keratinocyte-mediated defense mechanisms that may guide potential therapeutic strategies for infectious inflammatory skin diseases.

# OP17 Immunology of infectious diseases

## Preclinical therapeutic potential of phage FT5P against vancomycin-resistant Enterococcus faecium infections

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Background. Enterococcus faecium, classified by the World Health Organization as a high-priority ESKAPE pathogen, exhibits intrinsic multidrug resistance, environmental persistence, and strong biofilm formation, facilitating adaptation to hospital settings. Vancomycin-resistant E. faecium (VRE) is an increasing global nosocomial threat, particularly in immunocompromised and critically ill patients. Given limited therapeutic options, alternative strategies such as phage therapy are potentialized. We aimed to characterize phage FT5P and evaluate its preclinical therapeutic potential against VRE.

Methods. Phage FT5P was isolated from hospital wastewater. Morphology was assessed by electron microscopy and genome sequencing. Stability was evaluated across pH 1–10.5, temperatures −80°C to 60°C, and in active and heat-inactivated human serum. Host range was tested against 86 multidrug-resistant clinical isolates. Lytic activity was analyzed through growth curves (MOI 0.1–10). Biofilm prevention and treatment were measured by biomass and viability assays. Synergy with phage FT2P, daptomycin, and ampicillin was assessed using checkerboard assays.

Results. FT5P exhibited myovirus morphology with a 56 kb genome encoding 89 genes and no lysogeny, toxin, virulence, or antimicrobial resistance genes. It remained stable at pH 4.5–10.5 and physiological temperatures, retaining infectivity in inactivated serum but showing complement susceptibility at 24 h. FT5P infected 39% of E. faecium and 6% of E. faecalis strains. Lytic efficacy was strain-dependent, achieving complete inhibition in reference strains. Biofilm prevention reached 70%, while established biofilms showed up to 60% viability reduction. Synergy was observed with FT2P, additive effects with daptomycin, and antagonism with ampicillin.

Conclusions. FT5P demonstrates a safe genomic profile and promising antibiofilm activity against VRE, supporting further exploration in personalized phage therapy approaches.

# OP18 Microbiome and Immunology

## Development of a universal chemically-defined medium for the analysis of „stressed“ bacterial EVs in the context of undernutrition

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Undernutrition represents a significant global health challenge, impacting diverse populations. Its influence on the gut microbiome is complex, causing both quantitative and qualitative shifts that may alter inter-bacterial communication and host interactions, producing bacterial extracellular vesicles (bEVs), which could serve as universal messengers modulating the gut environment and potentially impacting host health.

This project aims to elucidate the role of undernutrition-induced stress on bacterial EV production, focusing on key nutritional deficiencies such as iron, vitamin A, and excess bile salts. To achieve this, we are developing a universal chemically-defined medium (CDM) optimized for the reproducible culture of representative commensal strains under stressed and standard conditions [1]. The CDM enables systematic comparison of bEVs, including proteomic and transcriptomic profiling of bEVs pairs to identify functional cargo involved in microbiome-host interactions.

The insights obtained may pave the way for novel biomarkers or therapeutic strategies targeting microbiome-mediated pathways in undernutrition and related digestive pathologies.

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# OP19 Microbiome and Immunology

## Dissecting dendritic cell–microbiota interactions in the intestines: role of bacterial metabolites in shaping adaptive immunity

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Intestines are a specialized mucosal barrier continuously exposed to microbial and dietary antigens. Immune homeostasis in this environment depends on coordinated interactions between innate and adaptive immune cells. Dendritic cells (DCs) are central to this network, acting as antigen-sampling sentinels that link innate and adaptive immunity. By presenting antigens and providing co-stimulatory signals, DCs guide $CD4^+$ T cell differentiation toward effector subsets or promote tolerance through regulatory T cells.

Although antigen presentation is well characterized, how the gut microbiota influences DC function remains poorly understood. It is unclear whether distinct bacterial species act uniformly across DC subsets or whether specific microbes preferentially modulate particular DC populations to shape downstream T cell responses. Moreover, microbial metabolites may alter DC activation by engaging pattern-recognition receptors or modulating intracellular signaling.

The goal of my research is to define how individual bacterial species and their metabolites influence DC biology and subsequent $CD4^+$ T cell differentiation. To that end, I expose classical DCs isolated from murine spleen to purified microbial metabolites or bacterial culture supernatants and analyze them by bulk RNA sequencing to identify metabolite-induced transcriptional programs. Selected compounds are then tested functionally by pulsing DCs with ovalbumin (OVA) in the presence of metabolites, before co-culture with naïve OT-II $CD4^+$ T cells. I then assess T cell differentiation with flow cytometry by expression of lineage-defining transcription factors and activation markers.

This work will reveal how microbial signals shape DC-T cell interactions, offering insight into microbiota-driven immune regulation and guiding strategies to therapeutically modulate tissue inflammation.

# OP20 Hipersensitivity

## Genistein modulates autoimmune inflammation in epidermolysis bullosa acquisita

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Autoimmune blistering skin diseases are characterized by pathogenic autoantibodies targeting skin components and overactivation of the innate immune system, particularly neutrophil-mediated tissue injury. Among these, epidermolysis bullosa acquisita (EBA) is caused by autoantibodies targeting type VII collagen (COL7), a major component of subepidermal anchoring fibrils tethering epidermis and dermis. Current therapies for autoimmune blistering skin diseases rely on systemic immunosuppression and are associated with severe adverse effects, underlining the need for safer alternatives. Genistein is a plant isoflavone with anti-inflammatory and immunoregulatory properties. In this study, we present preliminary in vitro and in vivo data demonstrating the therapeutic potential of genistein in experimental EBA. To explore potential effector mechanisms, we assessed the impact of genistein on neutrophil activation in vitro. Genistein significantly reduced reactive oxygen species (ROS) release from human neutrophils stimulated with fMLP/LPS in a dose dependent way, demonstrating direct suppression of oxidative effector functions driving blister formation. Building on these findings, we evaluated the therapeutic efficacy of genistein in vivo. Prophylactic oral administration of genistein (40 mg/kg/day) in a passive antibody transfer mouse model of EBA, caused a marked reduction of clinical disease severity compared to vehicle-treated controls. Genistein-treated mice exhibited ~60% decrease in affected body surface area, visibly reduced skin inflammation and blister formation, indicating effective attenuation of autoantibody-mediated tissue damage. These results provide the first experimental evidence that genistein inhibits neutrophil activity in vitro and ameliorates autoantibody-driven skin inflammation in vivo, suggesting that genistein represents a promising alternative for the treatment of autoimmune blistering skin diseases.

# OP21 Hipersensitivity

## Comprehensive transcriptomic profiling of circulating platelets in patients with ankylosing spondylitis

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Background: Ankylosing spondylitis (AS) is characterized by chronic inflammation leading to pathological ossification. Recently, platelets are increasingly recognized as immune modulators containing a dynamic transcriptome that can reflect systemic disease states[1]. This study aimed to characterize the transcriptomic profile of circulating platelets in AS patients to elucidate their specific contribution to disease pathogenesis.

Methods: The study included 35 patients with AS, sampled at baseline and after 3 to 6 months of biological therapy (anti-TNF or anti-IL-17), alongside 21 healthy controls. Total RNA was isolated from platelets and subjected to bulk RNA sequencing. Differential gene expression analysis was performed to identify dysregulated transcripts, followed by Gene Set Enrichment Analysis (GSEA) to evaluate AS-specific biological pathways.

Results: Analysis revealed a distinct transcriptomic profile in AS platelets. We identified significant upregulation of the long non-coding RNA MALAT1, which was recently positively correlated with AS symptom severity[2], and TPI1, encoding a metabolic enzyme connected to other rheumatic disorders[3]. Functional analysis demonstrated enrichment of immune response activation and leukocyte cell-cell adhesion. Crucially, we observed dysregulation of bone remodeling pathways, specifically Wnt signaling and osteoblast differentiation. These alterations highlight a potential regulatory axis involving DKK1 protein, suggesting platelets actively modulate the inflammation-ossification balance, which aligns with recent insights into AS progression[4].

Conclusion: The platelet transcriptome in AS reflects disruptions in both inflammatory and osteogenic pathways. The identification of MALAT1 and Wnt signaling interruptions positions platelets as active participants in AS pathogenesis and a source of potential biomarkers for monitoring therapeutic response to biological agents.

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# OP22 Hipersensitivity

## POTENTIAL MECHANISM OF LYMPHOID CELL DEATH INDUCED BY ANTI-MHC II ANTIBODIES

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The development of monoclonal antibodies (mAbs), targeting cancer-specific antigens and inducing apoptosis of cancer cells is crucial for the advancement of hematological malignancies therapy. The majority of monoclonal antibodies with therapeutic potential need the complement activation or antibody-dependent cellular cytotoxicity for eliciting their anticancer activity [1-2].

In our institute it has developed two monoclonal antibodies, B5 and E11, that recognize antigen DLA-DR (canine MHC II) which exert strong direct and indirect apoptotic effect on canine leukaemia and lymphoma cell lines [3].

The aim of presented research is to investigate the molecular mechanism of apoptosis in cells treated with anti-DLA-DR antibodies. I study the hypothesis that apoptotic effect is not dependent from internalization, but is associated with signal transduction into the cell. Understanding the molecular mechanism of the anti-tumor activity of MHC II mAbs is vital to advance their application both in dogs and humans.

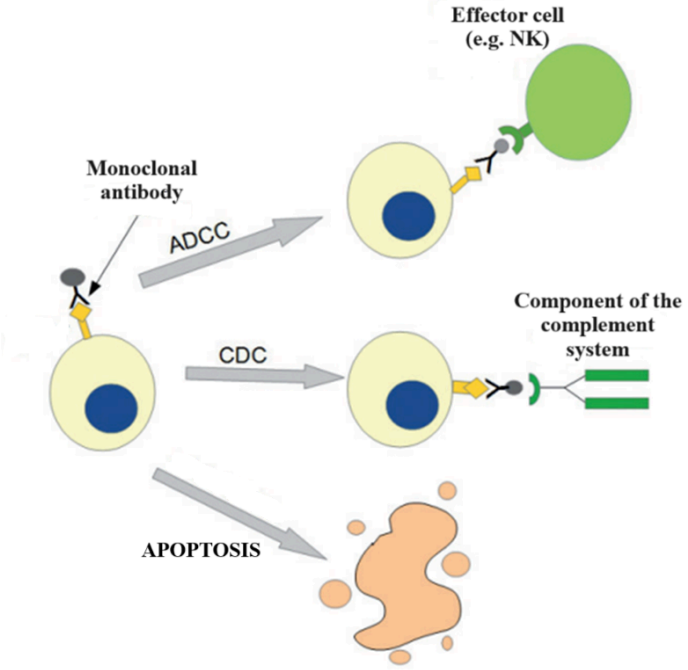


Fig. 1. Mechanisms of monoclonal antibodies cytotoxicity: indirect via antibody-dependent cellular cytotoxicity (ADCC) mediated by natural killer (NK) cells, complement-dependent cytotoxicity (CDC) involving the complement system and direct induction of apoptosis.

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# OP23 Hipersensitivity

## Immunosuppressive mesenchymal stem cells regulate arachidonic acid metabolism in experimental T2 low asthma

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Over the past decades, the immunosuppressive properties of mesenchymal stem cells (MSCs) have been widely confirmed in chronic inflammatory diseases in numerous preclinical studies, followed by clinical trials1,2. The challenges associated with the optimization and regulatory approval of cellular therapies are primarily driven by safety concerns. Nevertheless, mesenchymal stem cells may still be used as a valuable tool to investigate the mechanisms underlying poorly controlled inflammatory diseases, including T2-low asthma. Therefore, here we aimed to mechanism(s) underlying MSC-mediated immunosuppression in low T2 airway inflammation in the experimental asthma model.

C57BL6 mice were challenged with 100 µg of house dust mite (HDM) extract for 5 days in each of two weeks. Moreover, mice received intranasal administration of mesenchymal stem cells on day 6th of the experiment. All animals were sacrificed on day 15th, and bronchoalveolar lavage fluid (BALF) and lung tissue were collected for analysis.

Transcriptomic profiling of lung tissue revealed downregulation of genes associated with lipid metabolism and arachidonic acid signaling. Notably, MSC administration led to reduced expression of genes encoding three consecutive cysteinyl leukotriene-synthesizing enzymes. Consistently, metabolic profiling of BALF demonstrated decreased levels of terminal arachidonic acid metabolites, including PGD2, PGE2, 12-HHTrE, and HETEs family. Immunofluorescence staining (CysLTR1, CysLTR2, LTB4R2) revealed co-localization of leukotriene receptors in airway epithelial cells.

In summary, we revealed a novel mechanism of mesenchymal stem cell–mediated immunosuppression in T2-low airway inflammation in an experimental asthma model. Further studies aimed at identifying therapeutic targets are warranted.

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# PP1 Oncoimmunology

## Modulation of Tumor Immunogenic Phenotype by Electroporation under Normoxia and Hypoxia

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Cancer immunotherapy has transformed oncology, but many tumors evade immune detection due to insufficient immunogenicity. Inducing immunogenic cell death (ICD) has emerged as a strategy to overcome this barrier, ICD triggers exposure of damage-associated molecular patterns (DAMPs) like calreticulin and CXCL10 that alert dendritic cells and initiate adaptive immune responses against tumors [1,3]. Electroporation shows promise for inducing tumor stress and enhancing immunogenicity, yet how specific parameters and microenvironmental factors influence immunogenic remodeling remains poorly characterized [2]. LLC cells were treated with two electroporation protocols—600 V/cm or 1200 V/cm , 8 pulses at 100 or 50 µs duration each, alongside non-electroporated controls . After electroporation cells were maintained under normoxic or hypoxic conditions for 48 hours to track immunogenic changes.
Electroporated cells displayed significant increases in surface CRT and CXCL10. We found the strongest CRT expression in cells cultured in normoxia after treatment with 1200 V/cm pulsed electric field (PEF), whereas CXCL10 upregulation was far more prominent under normoxia than hypoxia. MHC-I expression also increased after electroporation, suggesting better antigen presentation capabilities. Interestingly, normoxia amplified all three immunogenic markers, indicating that oxygen levels play a major role in how electroporation triggers ICD. This study confirms that electroporation-induced stress upregulates ICD markers, particularly calreticulin, alongside elevated CXCL10 chemokine expression and enhanced MHC-I antigen presentation in an oxygen-dependent manner. The amplified responses under normoxia provide insight into how microenvironment shapes ICD. These findings guide strategies for utilizing electroporated tumor antigens in dendritic cell assays and support electroporation-based cancer immunotherapy development.

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# PP2 Oncoimmunology

## RVU120 Potentiates NK Cell–Mediated ADCC in Combination with Therapeutic Antibodies

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Natural killer (NK) cells play a pivotal role in cancer immunosurveillance by eliminating malignant cells through innate cytotoxic mechanisms. A key pathway of NK-cell-mediated tumor killing is antibody-dependent cellular cytotoxicity (ADCC), triggered when NK cells recognize the Fc region of therapeutic antibodies bound to tumor-associated antigens. Monoclonal antibodies such as the anti-CD20 antibody rituximab exploit this mechanism to enhance immune-mediated cancer cell clearance. Since NK-cell activity can be negatively regulated by STAT1 (S727) phosphorylation, CDK8/19 inhibition presents a strategy to potentiate ADCC responses. Accordingly, RVU120, a clinical-stage CDK8/19 inhibitor, represents a promising candidate for improving antibody-based immunotherapies.

Pharmacokinetic and pharmacodynamic (PKPD) studies in immunocompetent mice demonstrated that short-term RVU120 treatment promotes NK-cell maturation and enhances their cytotoxic phenotype, as confirmed by ex vivo co-culture cytotoxicity assays. In contrast, chronic continuous dosing resulted in diminished efficacy and exhibited hallmarks of NK cell exhaustion. In vivo efficacy studies in Raji tumor-bearing mice revealed limited anti-tumor activity of RVU120 monotherapy, whereas rituximab significantly inhibited tumor growth. Notably, combination therapy with RVU120 and rituximab was well tolerated and led to complete tumor regressions, indicating a strong synergistic effect.

These findings highlight the importance of dosing strategy in maximizing RVU120 activity and demonstrate its potential to augment antibody-driven anti-cancer immunity. Ongoing translational studies are expanding the application of RVU120 in combination with additional therapeutic antibodies, including trastuzumab. This combination approach may improve the efficacy of antibody-based therapies and antibody–drug conjugates, ultimately contributing to deeper and more durable clinical responses.

# PP3 Oncoimmunology

## Bone marrow-derived macrophages loaded with boron carbide nanoparticles as selective boron carriers for crossing the blood-brain barrier toward glioma for boron neutron capture therapy

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The use of macrophages as cellular carriers of nanoparticles in anticancer therapy is a promising strategy, called a "Trojan horse". Macrophages have a huge potential to engulf particles, migrate and accumulate at tumor sites, as well as to cross the blood-brain barrier [1]. A particularly promising application of this strategy is the delivery of boron-rich compounds to the tumor microenvironment for boron neutron capture therapy (BNCT). This therapy is a type of targeted radiotherapy that destroys tumors at the cellular level and is primarily intended to treat patients with gliomas [2].

The primary objective of our study was to develop selective cellular carriers of boron carbide (B4C) nanoparticles. First, the uptake and accumulation of B4C nanoparticles in bone marrow-derived macrophages (BMDMs) in three polarization states (M0, M1, and M2) were confirmed using holotomography and ICP-MS. Subsequently, using the Transwell system, we demonstrated that all nanoparticle-loaded BMDM populations exhibited comparable ability to migrate through the brain endothelial cell monolayer, mimicking the blood-brain barrier, towards the glioma microenvironment. In the final stage, we showed that BMDMs loaded with B4C did not affect the viability of glioma spheroids after 6 days of co-culture, compared to control macrophages, which stimulated tumor cell survival. Moreover, the evaluation of the effect of glioma spheroids on macrophage phenotype changes confirmed that M1 macrophages with B4C did not repolarize towards the M2 phenotype during co-culture.

The results provide evidence that macrophages have great potential as boron carriers in BNCT, which may become a new type of radioimmunotherapy.

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# PP4 Oncoimmunology

## Distinct Mechanisms, Shared Goal: CAR-T and TCR-T Cell Therapies in Hematologic Malignancies

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Although CAR-T cell therapy has transformed the treatment of hematologic malignancies, its clinical efficacy is frequently constrained by antigen escape, as observed in multiple myeloma (MM), and by the lack of uniformly expressed, safe surface targets in diseases such as acute myeloid leukemia (AML). These limitations highlight the need for complementary approaches capable of targeting intracellular tumor antigens in an HLA-restricted manner, including T-cell receptor-engineered T cells (TCR-T). Using MM as an experimental model enabling direct side-by-side comparison, we evaluated the phenotype and functional properties of CAR-T and TCR-T cell platforms.

$CD3^+$ T-cells were transduced with lentiviral CAR or TCR constructs. Cytotoxic activity was assessed against luciferase-expressing MM and AML cell lines (HLA-A*02:01) using luciferase-based and LDH assays. T-cell activation, differentiation status, exhaustion markers, and $CD8^+$ T-cell frequency were analyzed by multiparameter flow cytometry.

Compared with CAR-T cells, TCR-T cells exhibited lower expression of activation markers, higher proportion of naïve-like cells, and an increased frequency of $CD8^+$ T-cells. Expression of exhaustion markers was largely comparable between platforms, although CAR-T cells showed higher levels of TIM-3 and LAG-3. Functionally, both CAR-T and TCR-T cells mediated comparable cytotoxicity against MM and AML targets, however, TCR-T cells displayed delayed onset of cytotoxicity while maintaining sustained effector function and sensitivity to low antigen density.

Overall, HLA-A02:01-restricted TCR-T cells efficiently eliminated MM and AML cells in vitro, complementing the rapid but surface antigen-dependent activity of CAR-T cells. These findings support TCR-T cells as a promising alternative or synergistic therapeutic option for HLA-A02:01$^+$ patients with hematologic malignancies.

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# PP5 Oncoimmunology

## Optimizing CAR-T Expansion Conditions for the First Polish Academic Multiple Myeloma Therapy

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Background: Multiple myeloma remains a major clinical challenge despite therapeutic advances. CAR-T therapy has transformed hematologic cancer treatment, but its efficacy depends on optimized manufacturing, including vector design and culture conditions shaping T-cell activation and expansion. Clinical data and scRNA-seq analyses indicate that therapeutic performance is influenced not only by CAR design but also by the culture environment, supporting a systematic evaluation of how culture conditions affect anti-BCMA CAR-T expansion, phenotype, exhaustion, and function.

Methods: We evaluated four GMP-grade media, four activation systems, and multiple cytokine combinations (IL-2, IL-7, IL-15, IL-21) using material from healthy donors. T cells were activated, transduced with an anti-BCMA lentiviral vector (MOI 10), and expanded for ten days. Flow cytometry assessed phenotype, CAR expression, and exhaustion markers. Functional activity was evaluated using luciferase-based killing assays against $BCMA^{+}$ and $BCMA^{-}$ targets. A weighted scoring algorithm integrated phenotypic and checkpoint-related parameters for condition comparison.

Results: Culture conditions strongly influenced CAR-T composition, generating balanced, CD8-enriched, or CD4-predominant products. Naïve-like T cells declined, while terminal effectors remained minimal. IL-21-containing conditions favored central memory phenotypes, with $CD8^{+}CAR^{+}$ T_CM exceeding 60%. PD-1 decreased consistently, while IL-21 reduced TIGIT and PD-1 but increased LAG-3. CAR-T cells achieved >80% tumor lysis at 24 h (E:T 5:1).

Conclusions: Culture optimization significantly impacts CAR-T memory, exhaustion, and function, though donor variability remains a key determinant of product heterogeneity. Flexible and validated manufacturing strategies are essential for reproducible GMP-grade CAR-T production and clinical translation.

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# PP6 Oncoimmunology

## Expression and gene polymorphism of miR-21 affect progression-free survival in multiple myeloma patients

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MicroRNAs (miRNAs) are a class of short, non-coding RNAs that play a crucial role in post-transcriptional gene regulation, influencing various cellular processes. miR-21 is well-characterized oncomir that significantly promotes the growth, survival and therapy resistance of multiple myeloma (MM) cells within the bone marrow microenvironment. Due to its biomarker potential in other cancers, we decided to investigate the prognostic significance of serum miR-21 levels and of MIR21 single nucleotide polymorphism (SNP) in MM patients. miR-21 expression was analysed in serum samples collected from 55 MM patients using TaqMan assays, while MIR21 rs1292037 SNP genotyping was performed on DNA isolated from whole blood of 250 MM patients using LightSNiP assays. PCR was performed on a LightCycler 480 II device.

We observed that miR-21 serum levels were significantly higher in patients with more advanced disease (ISS stages II-III) compared to patients with less advanced disease (ISS stage I) ($p=0.036$). Furthermore, patients with a high level of miR-21 were characterized by shorter progression-free survival than patients with low miR-21 expression ($p=0.048$). This shows the unfavourable role of increased miR-21 serum levels. Genotyping revealed that genotype MIR21 rs1292037 TT was associated with shorter progression-free survival. Our results suggest that miR-21 serum level and rs1292037 genetic polymorphism may be of value as potential prognostic markers for patients with multiple myeloma.

# PP7 Oncoimmunology

## Impact of Stattic analogs on STAT3 expression and cytokine secretion in restimulated splenocytes obtained from MC38 bearing mice vaccinated with genetically modified dendritic cells

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Application of dendritic cell (DC)-based vaccines represents a promising strategy in anticancer immunotherapy by enhancing the reactivity of the immune system. Key cytokines involved in shaping the antitumor response include interleukin (IL)-12 and IL-18, which promote the activation of cytotoxic T lymphocytes and NK cells. However, activation of STAT3 in T cells and NK cells contributes to immunosuppression by reducing cytotoxic activity and IFN-γ production, while promoting tolerance and regulatory phenotypes (Treg/Th17). Therefore, inhibition of STAT3 using small-molecule inhibitors, including Stattic analogs, may restore and strengthen antitumor immunity.

Our study aimed to assess the impact of Stattic analogs on STAT3 expression levels and cytokine production in restimulated splenocytes isolated from MC38 tumor-bearing mice vaccinated with DCs.

To determine the effect of a single administration of DCs transduced to produce IL-12 and/or IL-18 and stimulated with tumor antigens on the activation of a systemic antitumor response, we analyzed the activity and phenotype of spleen cells. Populations of NK cells, CD4+ and CD8+ T lymphocytes were distinguished among the spleen cells, and the percentage of effector and memory lymphocytes was estimated. The level of activation of restimulated spleen cells was determined based on their cytokine production capacity and cytotoxic activity against MC38 tumor cells. To evaluate the STAT3 expression and changes in cytokine production, splenocytes were co-culture in the presence of STAT3 inhibitors.

In conclusion, Stattic analogs modulated STAT3 signaling and altered cytokine profile in restimulated splenocytes obtained from MC38 tumor-bearing mice treated with DCs-based vaccines, potentially enhancing systemic antitumor immune responses.

# PP8 Oncoimmunology

## Effect of Stattic analogs on STAT3 expression and cytokine secretion in genetically modified dendritic cells applied as anticancer vaccines

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Dendritic cells (DCs)-based vaccines are an example of currently conducted immunotherapy using immunostimulants that affect tumor microenvironment (TME) changes. They play a special role in activating antigen-specific CTL lymphocytes in the TME. However, activation of STAT3 in dendritic cells acts as a suppressive regulator, inhibiting their maturation and reducing the expression of MHC II and the costimulatory molecules CD80/CD86. Furthermore, it limits DC migration to lymph nodes by reducing CCR7 expression. Activation of STAT3 also promotes the production of IL-10 and TGF-β, promoting immune tolerance and the development of regulatory T cells. Therefore, inhibition of STAT3 using small-molecule inhibitors, including Stattic analogs, may support their maturation.
This study aimed to assess the effect of Stattic analogs on STAT3 expression levels and cytokine secretion in genetically modified DCs used as anticancer vaccines.
The vaccine cells were characterized in vitro, determining the transduction efficiency and the effect of transduction and stimulation with tumor antigens (TAg) on the differentiation of these cells, compared to control DCs. To initially determine the efficacy of the developed vaccines, the ability of DCs transduced to produce IL-12 and/or IL-18 and stimulated with TAg to activate a specific anti-tumor response was assessed ex vivo. To evaluate STAT3 expression and changes in cytokine production, DCs were co-cultured in the presence of STAT3 inhibitors.
In summary, stimulation of vaccine dendritic cells with STAT3 inhibitors can support their maturation, increase the expression of MHC and costimulatory molecules, and enhance their ability to activate T lymphocytes.

# PP9 Oncoimmunology

## Co-delivery of IL-12/IL-15/IL-18 engineered dendritic cells in the murine colon cancer model

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Dendritic cell (DC)-based vaccines represent a promising strategy for cancer immunotherapy due to their capacity to induce potent antitumor immune responses. Their therapeutic efficacy may be enhanced by cytokines, such as IL-12, IL-15 or IL-18, that can modulate distinct immune cell subsets and act synergistically. In this study, we evaluated the therapeutic efficacy of cytokine-engineered DC vaccines in a murine colon cancer model MC38.

Mice bearing established MC38 tumors were immunized three times (days 17, 24, and 31 post-tumor inoculation) with DC-based cellular vaccines. Dendritic cells were stimulated with tumor-associated antigens and transduced with lentiviral vectors encoding cytokine genes: IL-12, IL-15/IL-15Rα, or IL-18. Combinations of differently modified DCs were also evaluated. Tumor growth was monitored to generate tumor growth curves and assess tumor growth inhibition. Additionally, both local and systemic antitumor immune responses were analyzed.

The highest tumor growth inhibition was observed in groups receiving mixtures of cytokine-modified DCs. These groups also exhibited the most pronounced leukocyte infiltration within the tumor microenvironment. Administration of all DC-based vaccines resulted in a decreased proportion of regulatory T cells (Tregs) and a significant increase in the cytotoxic activity of restimulated splenocytes.

Our findings indicate that combined cytokine-engineered DC vaccines enhance antitumor immune responses and may represent an effective strategy for colorectal cancer immunotherapy.

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# PP10 Oncoimmunology

## TIGIT in non-small cell lung cancer and its major subtypes, adenocarcinoma and squamous cell carcinoma: evaluation of the TIGIT promoter methylation as well as mRNA and protein expression levels

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T cell immunoglobulin and ITIM domain (TIGIT) is a co-inhibitory receptor (ICR) mainly expressed on T and NK cells. TIGIT causes T cells' exhaustion when binding to its ligand CD155 (PVR), which is also a ligand for the stimulatory molecule CD2261. The CD226/CD155 interaction is important in promoting T cell activation. Moreover, CD226 is involved in the biological function of NK cells2. TIGIT has been shown to synergise with PD-1 at the molecular level to inhibit CD226 and is considered a potential therapeutic target in cancer immunotherapy, including non-small-cell lung cancer (NSCLC). Growing evidence indicates that the TIGIT/CD155 axis may constitute a promising treatment target in NSCLC3,4. However, only a few studies analysed TIGIT and CD155 expression in NSCLC and its main histological subtypes, adenocarcinoma (LUAD) and squamous cell carcinoma (LUSC).

In this study, we investigated TIGIT promoter methylation (pyrosequencing), TIGIT expression at the mRNA level (ddPCR), and at the protein level (multiparametric flow cytometry and immunohistochemistry) in surgically resected NSCLC samples, with particular focus on differences between LUAD and LUSC. Additionally, TIGIT expression was investigated on different cell populations in the tumour microenvironment and in peripheral blood. Our results indicate that the NSCLC tumour microenvironment is characterised by increased TIGIT expression across multiple immune cell populations. We also observed a lower level of DNA methylation in the promoter region of the TIGIT gene in tumor tissue compared to non-tumor tissue, which was associated with higher TIGIT mRNA expression.

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# PP11 Immunology of infectious diseases

## Porphyromonas gingivalis attenuates Herpesviruses replication by gingipains-mediated virion destabilization

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Periodontitis (PeD) is a highly prevalent chronic inflammatory disease of the gingiva and clinical evidence indicates that patients with PeD are frequently co-infected with herpesviruses, including HSV-1. A proposed molecular basis for this association is the ability of gingipains, cysteine protease of Porphyromonas gingivalis, a key etiological agent of PeD, to impair host antiviral defenses. However, the direct effects of gingipains on HSV-1 virions remain unknown. Here, we investigated the direct interactions between P. gingivalis gingipains and HSV-1 virions and examined how these interactions influence functional outcomes across the viral replication cycle.

Our data demonstrate that gingipains selectively modify viral structural proteins, resulting in a pronounced reduction in virion stiffness. While these alterations do not impair viral entry into human epithelial cells, they lead to a significant decrease in viral replication at later stages of infection. Mechanistic analysis revealed that gingipains proteolytically cleave viral glycoproteins – gH, gL, gD and gB, required for membrane fusion, potentially compromising efficient delivery of viral DNA to the nucleus. In parallel, mass spectrometry identified gingipain-mediated degradation of viral proteins – ICP0, UL41 and UL46, involved in suppression of host antiviral responses, further contributing to reduced replication efficiency. Notably, a similar phenomenon was observed in neurons, cell types in which HSV-1 establishes latency.

Although P. gingivalis has been shown to facilitate herpesvirus infection, the present data extend our previous observations by demonstrating that direct, extracellular effect on virions may instead limit viral replication, indicating that gingipain-herpesvirus interactions are more complex than initially appreciated. Collectively, these results reveal a previously unrecognized complexity in P. gingivalis-herpesvirus interactions highlighting the need for further investigation.

# PP12 Immunology of infectious diseases

## Immunomodulatory and Antiviral Properties of Spirulina platensis: Mechanisms and Therapeutic Potential

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Spirulina platensis (Arthrospira platensis) is a nutrient-dense cyanobacterium with a long history of human consumption and increasing scientific interest due to its immunomodulatory and antiviral properties. It contains high-quality protein, polysaccharides, vitamins, minerals, and essential fatty acids. Additionally, it is rich in bioactive compounds such as phycocyanobilin and calcium spirulan, that contribute to its biological activity.
Experimental evidence indicates that Spirulina-derived polysaccharides directly activate innate immune responses. This activation involves enhancement of macrophage phagocytic activity, and stimulation of nitric oxide production via inducible nitric oxide synthase. In addition, Spirulina modulates cytokine expression, including Tumor Necrosis Factor alpha (TNF-α) and interleukin 6 (IL-6). Notably, Spirulina exhibits context-dependent immunoregulatory effects, promoting immune activation under physiological conditions while attenuating excessive inflammation in hyperinflammatory states. In parallel, Spirulina demonstrates broad-spectrum antiviral activity against enveloped viruses, including influenza virus, herpes simplex virus, HIV-1, and SARS-CoV-2. These antiviral effects involve both direct inhibition of viral attachment and penetration – particularly mediated by calcium spirulan – and indirect enhancement of host antiviral defenses through consistent upregulation of interferon-gamma (IFN-γ).
The convergent evidence from in vitro, preclinical, and clinical studies supports a dual-action model of Spirulina platensis, combining direct antiviral effects with finely tuned immunomodulation. Its ability to enhance innate immunity while preventing excessive inflammatory responses positions Spirulina as a promising functional and therapeutic adjunct in immune support and viral infection management, warranting further controlled clinical investigation.

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# PP13 Immunology of infectious diseases

## Phage interactions with gut epithelium confirm the safety of phage therapy after oral administration

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Due to the rapidly spreading bacterial resistance to antibiotics, phage therapy is increasingly being considered. In addition to their antibacterial activity, bacteriophages can interact with eukaryotic cells. The oral route is commonly used for drug administration due to its safety and ease of use, and intestinal lymphoid tissue constitutes the largest organ of the immune system. Some bacteriophages can interact with intestinal cells and penetrate them, potentially contributing to phage-dependent systemic immunomodulation.

Five lytic Enterobacter-specific bacteriophages were selected for this study. The aim was to examine their effect on Caco-2 cell viability, their impact on the integrity of the intestinal epithelial cell monolayer, and their ability to translocate through the epithelium (Fig. 1).

Phage lysates were purified using chromatographic methods, and bacterial endotoxin levels were determined. Cell viability following phage exposure was assessed using the MTT assay, while Evans blue staining was used to evaluate monolayer integrity. Epithelial translocation was analyzed using cell culture inserts. Phage titers were determined using routine test dilution and double-layer agar method.

Exposure to phages at titers of 106, 107, or 108 PFU/mL did not affect cell viability. No disruption of epithelial monolayer integrity was observed after phage exposure, even following translocation. The ability to cross an intact, differentiated monolayer in the apical-to-basolateral direction varied depending on the phage.

These results provide further evidence supporting the safety of phage therapy in intestinal epithelial tissue. Additionally, the ability of some phages to translocate through the epithelium suggests potential for targeted delivery to distant inflammatory sites and for exploiting their immunomodulatory properties.

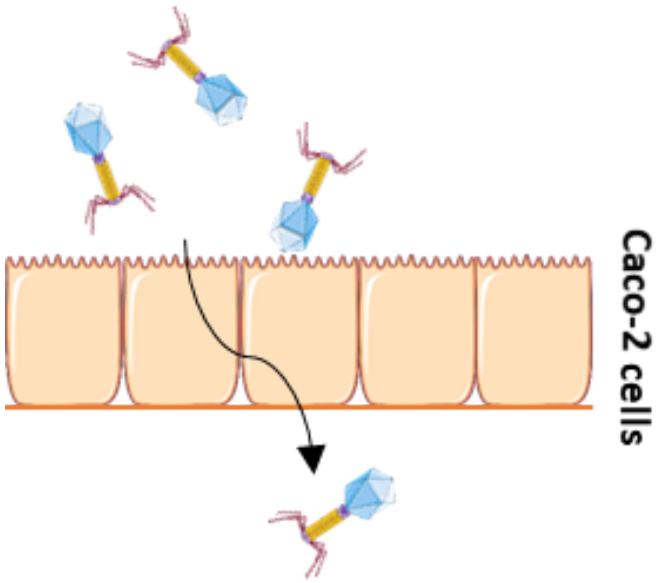


Figure 1. Phage translocation through the epithelium.

# PP14 Neuroimmunology

## Beyond Amyloid and Tau: Emerging Biomarkers for Early Diagnosis of Alzheimer's Disease

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Alzheimer's disease (AD) is the leading cause of dementia and is characterized by progressive cognitive decline preceded by long-lasting, clinically silent neuropathological changes. Early and accurate diagnosis is therefore essential. Biomarkers play a central role in identifying pathological processes before overt clinical symptoms emerge. [1]
Current diagnostic frameworks are based on the AT(N) model, encompassing amyloid deposition (A), tau pathology (T), and neurodegeneration (N). Core cerebrospinal fluid (CSF) and blood biomarkers include decreased Aβ42 or Aβ42/Aβ40 ratio, increased total tau (t-tau), phosphorylated tau (p-tau181, p-tau217), and neurofilament light chain (NfL). However, amyloid and tau abnormalities are not fully disease-specific, as they may occur in other neurodegenerative, vascular, and inflammatory conditions, limiting their etiological exclusivity. [2]
Emerging diagnostic strategies extend beyond single-protein detection toward multi-system, minimally invasive approaches. Neuroinflammatory profiling, microglial activation signatures, immune exhaustion markers, and blood–brain barrier dysfunction indicators provide mechanistic insight into early disease processes. Additional avenues include gut–brain axis–related biomarkers, saliva-based markers, and digital biomarkers supported by artificial intelligence. Integration of classical, inflammatory, vascular, and systemic immune biomarkers with clinical phenotyping may significantly enhance diagnostic accuracy, prognostic assessment, and personalized risk stratification. [3]
Advances in multimodal and minimally invasive biomarker detection are expected to improve early diagnosis and support the development of precision medicine strategies in AD. [4]

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# PP15 Neuroimmunology

## Gut Microbiota Products Modulate Blood–Brain Barrier Integrity: Insights from Bifidobacterium animalis BEVs

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Age-related changes in the gut microbiota contribute to systemic inflammation, which can compromise blood–brain barrier (BBB) integrity and promote neuroinflammation and cognitive decline. While the gut–brain axis is recognized as a crucial regulator of brain health, the impact of commensal bacteria and their secreted products remains unclear.

This study characterizes bacterial extracellular vesicles (BEVs) produced by Bifidobacterium animalis subsp. animalis CCDM 366 (Ba366) and evaluates the protective effects of Ba366 and its BEVs on BBB integrity.

The basic physicochemical properties of BEVs were established: their size and concentration were determined using DLS, with consistent lipid content between batches. Visualization by TEM confirmed their bilayer structure and DLS-measured size. BEVs demonstrated stability for up to 3 months and showed no batch-to-batch differences. Human brain microvascular endothelial cells (HBEC-5i) were used as an in vitro BBB model to examine the effects of Ba366 and BEVs under physiological and inflammatory conditions. Both Ba366 and BEVs were non-cytotoxic to HBEC-5i cells. Ba366 treatment enriched genes related to cell proliferation and neuronal signaling, while BEVs enriched genes involved in ion transport, neurotransmitter signaling, membrane function, and autophagy regulation.

These findings demonstrate that Ba366 and its BEVs modulate BBB integrity and function through complementary molecular pathways. In the context of age-associated gut microbiota dysbiosis and systemic inflammation, Ba366-derived components show promise for preserving or restoring BBB integrity and mitigating neuroinflammatory processes.

Financial support: This work was supported by the Biocodex Microbiota Foundation.

# PP16 Hipersesitivity

## Immunomodulatory effects of proteolytically inactive gingipains on osteoblast and osteoclast

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Periodontitis is an inflammatory condition of the gums leading to the progressive loss of tooth-supporting tissues, including the alveolar bone. The disease is driven by colonization with Porphyromonas gingivalis. The main virulence factors of P. gingivalis are gingipains—arginine- and lysine-specific cysteine proteases. Their enzymatic effects have been extensively described; however, emerging evidence indicates that proteolytically inactive gingipains (piGING) may also exert strong effects on host tissues. piGING display pro-inflammatory activity in epithelial cells and promote the differentiation of Th cells toward the Th17 phenotype. Until now, their impact on the disruption of bone homeostasis has not been investigated. The aim of this study was to examine the direct role of piGING in maintaining the osteoblast/osteoclast balance. We analyzed the biology of osteoblasts (Saos-2) and osteoclast precursors (monocytes) in the presence of piGING. Using TRAP staining, we demonstrated that piGING, particularly RgpB, induces upregulation of osteoclast differentiation. This observation was confirmed by increased expression of genes and proteins involved in bone resorption and remodeling, as measured by qPCR and Western blot (e.g., TRAP, CTSK, DCSTAMP, MMPs). We also observed that piGING attenuated the expression of genes involved in osteoblast differentiation and function, as well as extracellular bone matrix formation (e.g., ALPL, OMD, SPP1, SP7), including the key transcription factor RUNX2. The reduced differentiation potential of osteoblast precursors was confirmed by diminished mineralization (Alizarin Red staining). These findings demonstrate for the first time that piGING promote osteoclastogenesis while impairing osteoblast differentiation, highlighting a previously unrecognized mechanism contributing to periodontal bone loss.

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# PP17 Hipersesitivity

## Investigating Hematopoietic Memory in Long-Term Hematopoietic Stem Cells

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Hematopoietic stem cells (HSCs) ensure lifelong blood cell production through their capacity for self-renewal and multilineage differentiation. Beyond this classical role, emerging concepts suggest that HSCs may retain information about previously encountered stimuli, thereby contributing to a form of long-term hematopoietic memory. The mechanisms by which such memory is established, maintained, and functionally recalled at the stem cell level remain largely unexplored.
The aim of this study is to investigate whether repeated exposure to the same mobilizing stimulus induces persistent, HSC-intrinsic functional adaptations that influence subsequent hematopoietic output. Using a murine model, animals were treated with granulocyte colony-stimulating factor (G-CSF) to mobilize myelopoiesis. One month after this primary stimulation, long-term HSCs (LT-HSCs) were isolated and analyzed either in single-cell colony-forming unit (CFU) assays or following transplantation into lethally irradiated recipients. After hematopoietic reconstitution, recipients were subjected to a secondary mobilization, enabling assessment of HSC responses upon re-exposure to the same stimulus. In parallel, individually sorted LT-HSCs were cultured in vitro in medium supplemented with G-CSF, and the colonies formed from single cells were analyzed using a multimodal workflow combining morphometric imaging (colony size and shape classification), cytological staining, multiparameter flow cytometry, and index sorting. This integrative approach allows high-resolution characterization of colony diversity and lineage output while capturing functional heterogeneity within a phenotypically defined HSC population.
Together, this multilevel strategy provides a robust framework for studying functional adaptations of HSCs following repeated stimulation and establishes a strong foundation for future investigations into the epigenetic regulation of hematopoietic memory.

# PP18 Hipersesitivity

## Polymorphism within gene coding for activating NKG2D receptor and aging – study on a group of healthy Polish representatives

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Aging is associated with progressive alterations in the functioning of the immune system, collectively referred to as immunosenescence, including impaired Natural Killer (NK) cell and cytotoxic T lymphocyte responses. The activating receptor NKG2D, encoded by the KLRK1 gene, plays a key role in NK and CD8+ T cell activation by recognizing stress-induced ligands, and its expression has been shown to change with age. This study aimed to analyze the frequency of selected NKG2D (KLRK1) single nucleotide polymorphisms (SNPs) in young and elderly representatives of the Polish population.

A total of 407 healthy individuals were enrolled in the study, including 139 elderly (>65 years) and 268 young (18–64 years) individuals. The rs1154831 and rs1049174 SNPs in the KLRK1 gene were genotyped using the TaqMan allelic discrimination assay (Applied Biosystems) and carried out on a LightCycler 480 II instrument (Roche Diagnostics).

Significant differences in the distribution of the KLRK1 rs1154831 (but not rs1049174) genotypes were observed between elderly and young Poles. Older Poles were more likely to be GT heterozygotes (59/139 vs 83/268; OR=1.644; p=0.022), while GG homozygosity was less common in this group than in younger individuals (76/139 vs 177/268; OR=0.620; p=0.031). This latter relationship was particularly evident in the female group. Older women were twice as unlikely to carry the GG genotype (43/84 vs 84/122; OR=0.474; p=0.013).

These findings suggest that the rs1154831 polymorphism may be associated with attaining older age, indicating a potential role of NKG2D/KLRK1 genetic variability in immunological aging.

# PP19 Microbiome and Immunology

## Synbiotics Reduce Hypercholesterolemia and Aortic Inflammation in ApoE Knockout Mice via Intestinal Cholesterol Metabolism and T Cell Modulation

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Synbiotics, which combine probiotics (live microorganisms with claimed health benefits) and prebiotics (non-digestible food ingredients), have gained attention for their potential to mitigate high-fat diet (HFD) induced metabolic and cardiovascular dysfunctions. Here, using a novel synbiotic formulation, containing Lactobacillus fermentum, Bifidobacterium longum, Lactococcus lactis and inulin, we assessed its effects on lipid metabolism, intestinal and global T-cell immunity, and short chain fatty acids levels in HFD-fed ApoE -/- mice. Synbiotic treatment significantly reduced LDL cholesterol and hepatic lipid accumulation without affecting body or organ weights. In the intestine, synbiotics prevented HFD-induced changes in cholesterol metabolism-related gene expression, and maintained hepatic cholesterol levels comparable to mice fed with standard chow. Immunologically, synbiotic supplementation specifically reduced Th17 and Th17-like cytotoxic T cell subsets in the mesenteric lymph nodes (msLN), suggesting localized anti-inflammatory effects in the gut-associated lymphoid tissue, without broad changes in total T cell composition in either the msLN or spleen. Importantly, symbiotic treated mice showed no signs of aortic inflammation, unlike vehicle-treated HFD mice. In addition, synbiotic treatment significantly increased fecal butyrate levels. Overall, these findings demonstrate that this novel synbiotic formulation exerts protective metabolic and immunomodulatory effects in HFD-fed ApoE$^{-}$/$^{-}$ mice, likely mediated through modulation of gut lipid metabolism, localized intestinal T-cell responses, and enhanced butyrate production. Together, this highlights the therapeutic potential of synbiotics as a targeted strategy to attenuate diet-induced cardiometabolic and inflammatory dysfunction.

# PP20 Innate Immunology

## AREL1 E3 Ubiquitin Ligase as a Modulator of Innate Immune Signaling

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Apoptosis-resistant E3 ubiquitin protein ligase 1 (AREL1) belongs to the family of E3 ubiquitin ligases responsible for the selective attachment of ubiquitin to target proteins during the ubiquitination process. This is a crucial mechanism that controls protein stability and activity within the cell 1. Despite its potential involvement in immune regulation, the role of AREL1 in signaling pathways initiated by pattern recognition receptors (PRRs) remains poorly characterized. This study is conducted using human THP-1 and Cal-1 cell lines. To generate AREL1 knockout models, a gesicles-mediated CRISPR-Cas9 delivery system was employed in both cell lines, and successful genetic modification was confirmed by Western blot analysis. Following PRR ligand stimulation, the secretion levels of cytokines such as CCL5, TNF-α, and IL-8 were quantified using enzyme-linked immunosorbent assay (ELISA). Our results demonstrate that AREL1 deficiency alters the secretion of key pro-inflammatory cytokines and chemokines, indicating a regulatory role for AREL1 in PRR-mediated innate immune signaling. A more comprehensive understanding of the molecular mechanisms mediated by this ligase may facilitate the identification of novel therapeutic targets for inflammatory and cancer-related diseases1.

# PP21 Innate Immunology

## Understanding the function of DTX3L ligase in the antiviral response induced by pattern recognition receptors (PRRs)

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Every day, the innate immune system defends the host against invading pathogens. The first line of defense relies on pattern recognition receptors (PRRs) that sense pathogen-associated molecular patterns (PAMPs). When viruses invade the cells, receptors such as Toll-like receptors (TLRs), RIG-I-like receptors (RLRs) activate signaling cascades that induce the expression of effector molecules such as interferons and pro-inflammatory cytokines.1 An important process accompanying the activation of signaling pathways is ubiquitination—the covalent attachment of ubiquitin to a protein. While proteins tagged with ubiquitin are often targeted for proteasomal degradation, ubiquitination does not always signal “destruction”. It can regulate protein localization, interactions with other proteins, and enzymatic activity. In this way, ubiquitin encodes complex molecular signals that control the fate and function of proteins.2 DTX3L is a RING domain E3 ligase that, when complexed with PARP9, promotes interferon-stimulated gene expression and antiviral responses. While viruses like SARS-CoV-2 can counteract this system, DTX3L's direct impact on PRR induced signaling during viral infection remains unclear. Therefore, in this study, we investigate DTX3L's molecular role in PRR signaling pathways activated by different viruses.3

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# OF1 Oncoimmunology

## Physiological oxygen level during endothelial progenitor cell culture increases vessel normalizing potential of extracellular vesicles

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Approaches to normalize cancer vasculature became a hope in oncology, as they can enhance anti-cancer therapy effectiveness. However, currently available therapeutics normalize vessels only temporarily, and their use is burdened with limitations [1]. Endothelial progenitor cells (EPCs) may become an alternative, as they possess vessel-restoring potential and can act in a paracrine way [2, 3], which overcomes the limitations of cell-based therapies in cancer. Our research explores EPC-derived extracellular vesicle (EPC-EV) potential to restore cancer vasculature. To fully understand EPC-EV properties, EVs from murine EPC cell lines MagEC 10.5 and MagEC 11.5 produced in different oxygen conditions (normoxia, physioxia, and hypoxia) were studied. EPC-EVs were tested in vitro in functional assays to determine their effects on endothelial cell proliferation, migration, phenotype, and angiogenic properties. Additionally, potential effects on off-target cells like immunocompetent cells and cancer cells were evaluated. Firstly, EVs derived from different oxygen conditions showed distinct properties. Notably, physioxia-derived EVs were able to reduce endothelial cell proliferation in tumor microenvironment-mimicking conditions and altered the morphology of pseudovessels when endothelial cells were co-cultured with cancer spheroids. Finally, although no significant effects on immunocompetent cells were observed, physioxia-derived EPC-EVs managed to partially decrease migration and vimentin expression in melanoma cells. In conclusion, physioxia-derived EPC-EVs seem to harbor cancer vessel normalizing potential, which could be enhanced in the future with the introduction of genetic modifications or EV packaging.

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# OF2 Oncoimmunology

## The significance of heat shock protein 90 in calcitriol and tacalcitol anticancer activity against colorectal cancer cells

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Calcitriol, the biologically active form of vitamin D3, regulates gene transcription through the vitamin D receptor (VDR), which heterodimerizes with the retinoid X receptor. This complex binds vitamin D response elements in target gene promoters, thereby controlling cell growth, differentiation, and survival (1). Tacalcitol, a calcitriol analog, exhibits reduced calcemic activity (2). The role of heat shock protein 90 (Hsp90) in VDR-mediated anticancer activity remains poorly understood. However, chronic exposure of colorectal cancer (CRC) cells to 5-fluorouracil (5-FU) increases Hsp90 expression and activity, contributing to thymidylate synthase expression and drug resistance (3).
This research aims to investigate whether the increased expression and activity of Hsp90 following 5-FU treatment can enhance the sensitivity of CRC cells to the activity of vitamin D3 derivatives (VDDs).
Experiments were performed in the HT-29 and HCT 116 CRC cell lines, their 5-FU-resistant sublines generated by continuous 5-FU exposure, and the 5-FU-sensitive sublines with silenced Hsp90β. VDDs' antiproliferative activity was assessed using the sulforhodamine B assay, and protein expression was analysed by western blot.
5-FU-resistant HT-29 and HCT 116 cells were more sensitive to 5-FU in combination with VDD than 5-FU-sensitive cells. Silencing of Hsp90β significantly reduced the antiproliferative activity of VDDs alone and in combination with 5-FU in HT-29 cells, whereas no such effect was observed in the less VDD-sensitive HCT 116 cells. These results indicate that Hsp90 level may modulate the anticancer activity of vitamin D3 derivatives in the case of the HT-29 CRC cell line.
This research was funded by the National Science Centre, Poland, grant number 2020/39/D/NZ5/02011.

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# OF3 Neuroimmunology

## Interferons as Mediators of Neuroinflammation and Synaptic Los Alzheimer's Disease

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Alzheimer's disease (AD) is characterized by progressive cognitive decline accompanied by β-amyloid and tau pathology as well as chronic neuroinflammation. Increasing evidence suggests that interferons represent an important molecular link between immune activation and neurodegeneration. This review aimed to summarize the role of type I interferons (IFN-I), particularly IFN-β, and type II interferon (IFN-γ) in neuroinflammatory processes associated with AD.

A structured literature search of the PubMed database was conducted up to November 2025 using predefined keywords related to interferons, neurodegeneration, microglia, blood–brain barrier and cGAS–STING signaling. Relevant experimental and clinical studies published within the last decade were included after title/abstract and full-text screening.

Available data indicate that chronic activation of IFN-I signaling, often driven by the cGAS–STING pathway, promotes microglial activation, synaptic loss and cognitive impairment in AD [1]. Elevated IFN-β levels correlate with cognitive decline and brain atrophy in patients, suggesting a role in disease progression [2]. In contrast, controlled exogenous IFN-β administration improves cognition and reduces amyloid burden in experimental models [3]. IFN-γ contributes to neurodegeneration by inducing oxidative stress through repression of the Nrf2 pathway and by destabilizing the blood–brain barrier [4]. Increased interferon-response gene expression in patients with MCI and AD further supports the clinical relevance of interferon signaling as a potential biomarker of disease progression [5].

Interferon signaling represents a dynamic and context-dependent regulator of neuroinflammation in AD. Selective modulation of interferon pathways, including IFNAR and cGAS–STING signaling, may constitute a promising strategy for disease-modifying therapies.

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# Workshops

**„Stress-Free Cell Sorting: Preserving Fragile and Neuronal Cells with the WOLF Cell Sorter"**
**Riccardo Pasculli, Application Specialist, Omixys**

The WOLF Cell Sorter is a microfluidic-based platform designed to enable high-purity cell sorting while minimizing mechanical stress and preserving cell viability. Unlike conventional droplet-based sorters that rely on high pressure and electrostatic deflection, the WOLF system employs low-pressure microfluidic cartridge technology to provide gentle handling of delicate cell types. This approach is particularly advantageous for neuronal cells, which are highly sensitive to shear stress and environmental perturbations. Gentle sorting with the WOLF Cell Sorter supports improved post-sort viability, maintained neurite integrity, and enhanced functional recovery, making it well suited for downstream applications such as primary neuron culture, single-cell analysis, transcriptomics, and regenerative research. By combining precision, sterility, and reduced fluidic stress, the WOLF platform represents a valuable tool for researchers working with fragile neuronal populations.

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# Workshops

## „The use of real-time PCR in SNP genotyping”

Single nucleotide polymorphisms (SNPs) are a type of DNA variation that involve only a single nucleotide position. They are the most common form of genetic variation, with many SNPs responsible for various common traits, but also for increased disease risk. There are various methods of SNP genotyping that can be used depending on the needs of the researcher. This workshop will focus on the use of real-time PCR in studying SNPs. The workshop will be divided into three parts: 1) a “wet lab” part, during which the participants will run the experiment themselves, 2) presentation of the methods and basics behind SNP genotyping and research, 3) analysis of genotyping results generated in the first part of the workshop.
The workshop will take 2 hours and is limited to 10 participants.

## „3D Cell Imaging Using the Tomocube HT-2 Microscope”

This hands-on workshop will be held as part of ImmunoBridge 2026 and is dedicated to advanced methods of three-dimensional cell imaging.
Participants will be introduced to the principles and practical applications of the Tomocube HT-2 microscope, an advanced, label-free 3D imaging system based on quantitative phase imaging (QPI) and holographic tomography. The workshop will demonstrate how this technology enables high-resolution, three-dimensional imaging of live cells without the need for staining, using differences in the refractive index to visualize cellular structures.

In addition to imaging, the workshop will cover quantitative analysis of cellular parameters, a feature that distinguishes Tomocube HT-2 from conventional microscopy systems.

This workshop offers a unique opportunity to gain hands-on experience with the only Tomocube HT-2 system available in Poland, making it a valuable learning experience for researchers interested in advanced cell imaging techniques.
Participants are invited to join this session during ImmunoBridge 2026 to explore state-of-the-art approaches to live-cell 3D imaging.

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# Workshops

## „The Art of Public Speaking"

(Part 1) How to give a confident and compelling scientific presentation
(Part 2) The art of scientific story telling on print and social media

Scientific communication is perhaps the most important aspect of a scientific career, which unfortunately is also the least emphasized in training. Effective presentation skills are part and parcel of a scientist's life; from baby steps (your 'brainbee' presentation) to the ultimate glory (your Nobel prize lecture). The last decade has also brought about a diversification in the scientific presentation formats with the emergence of pitch talks, blitz talks, chalk talks, and of course the COVID-ed complication of giving all these talks online. Finally, #scicomm has witnessed the transformation of a Stanford professor (Andrew Huberman) to a Times-cover celebrity with 7 million followers. Of course, not everyone needs to be the next Huberman, but we all need to nurture our inner Hubermen and Huberwomen because social media is the largest platform for public outreach and is here to stay.

In this 2-hour long interactive workshop (divided into two parts), we will first cover a novel and effective approach to prepare and deliver scientific presentations using PAT (prepare, anticipate, target) and RAT (relax, articulate, translate) components. This approach will be individualized to different formats- large class format lectures, flash/pitch talks, and virtual talks. We will then have a special feature on communicating science through popular science magazines and social media.
The workshop is limited to a maximum of 30 participants.

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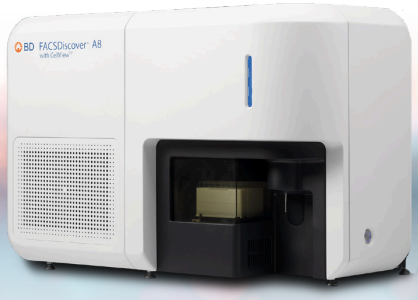


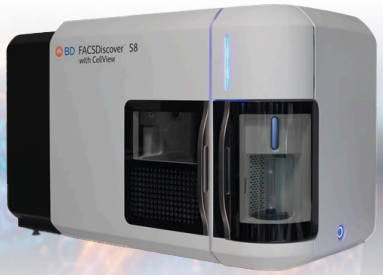


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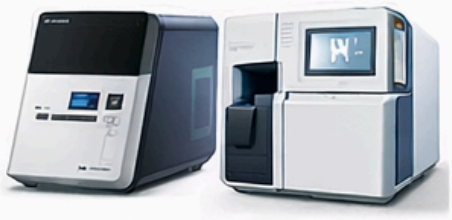

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