FarU Immunology Meeting

Abstract Book

7 JUNE 2025
9:00 - 18:20

Prof. Karol Taylor Lecture Hall
Intercollegiate Faculty of Biotechnology, UG & MUG
Abrahama 58, 80-307 Gdańsk

FarU Immunology Meeting

This event is dedicated to uniting the immunological community in the Tri-City area.

The conference aims to foster collaboration and knowledge exchange by bringing together scientists from the three Fahrenheit Universities who are involved in immunology research. Attendees will have the opportunity to:

- share their research
- take part in discussions
- engage in networking

The event is free for registered users.

Fahrenheit Universities

MEDICAL UNIVERSITY OF GDAŃSK

University of Gdańsk

Gdańsk Branch

FarU

The Fahrenheit Union of Universities in Gdańsk was established upon the joint request of the Rectors of the Medical University of Gdańsk, Gdańsk University of Technology and the University of Gdańsk. The senates of the three leading universities of Pomerania adopted relevant resolutions and endorsed the wording of the Statutes specifying the range of responsibilities, the bodies and the way of managing that new organisation.

The Medical University of Gdańsk, Gdańsk University of Technology and University of Gdańsk have been undertaking joint initiatives in the scientific, educational and organisational areas for many years. Owing to the creation of the Union of Universities, it is possible to enhance current cooperation and create in Pomerania one of the strongest academic centres in Poland.

Fahrenheit Universities

MEDICAL UNIVERSITY OF GDAŃSK

University of Gdańsk

Gdańsk Branch

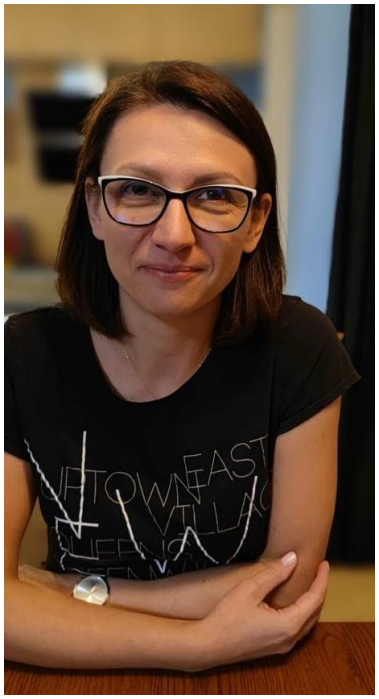
Organizing Committee

Dr hab. n. med. Danuta Gutowska-Owsiak, prof. UG (University of Gdańsk)

The Chairwoman of the Gdańsk Branch of the Polish Society for Fundamental and Clinical Immunology and Co-Chairwoman of the European Epidermal Barrier Research Network. Danuta graduated from the Medical University of Gdańsk and then devoted her career to experimental immunology. She received a PhD degree from the University of Liverpool, which she followed with 8 years of postdoctoral training at the University of Oxford, incl. the work for which she was distinguished with the "Young Investigator Award" by the British Society for Investigative Dermatology.

In 2017, Danuta established the Laboratory of Experimental and Translational Immunology at the Intercollegiate Faculty of Biotechnology UG-MUG in Gdańsk. She has a strong interest in both epidermal barrier formation and the immunity in the skin, as well as extracellular vesicles as mediators of cellular communication. Together with her team, she investigates allergic inflammation, as well as basic immunological mechanisms of T cell and dendritic cell biology, including more translational angle. The work of "DGO Lab" is fully supported by grants from National Science Centre and the Foundation for Polish Science, incl. EU cofunds and the COST action programme.

Dr hab. med. Anna Wardowska (Medical University of Gdańsk)

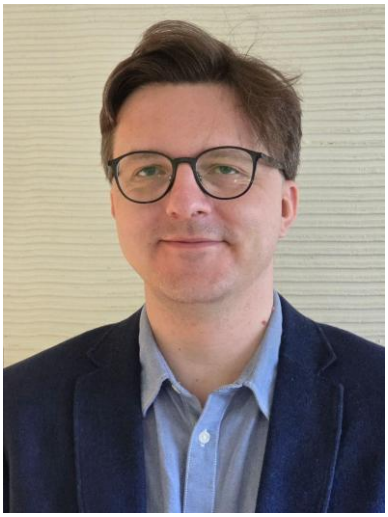


Dr. Hab. Ania Wardowska, a biotechnologist by education and an immunologist by passion, vice-chair of the Gdańsk Branch of the Polish Society for Fundamental and Clinical Immunology. She has been studying the immunopathogenesis of systemic lupus erythematosus (SLE) and the mechanisms of immunomodulation for years. In recent years, she has been looking for answers to the question of whether and how COVID-19 infection affects the course of the underlying disease in SLE patients. In addition, in cooperation with Dr. Hab. Marta Spodzieja from the University of Gdańsk, she studies the effect of peptides on the regulation of immune cell activity, also in patients with lupus. She hopes that the attempt to use the immune checkpoints will lead to the development of effective immunomodulatory therapies.

Dr hab. Katarzyna Lisowska, prof. GUMed (Medical University of Gdańsk)

Dr. Hab. Katarzyna Lisowska works as an associate professor at the Department of Physiopathology, Medical University of Gdańsk. Katarzyna's main interest is the immunology of chronic kidney disease (CKD). She completed her doctoral thesis under the supervision of Prof. Alicja Dębska-Ślizień at the Department and Clinic of Nephrology, Transplantology, and Internal Medicine of the Medical University of Gdańsk. For years, she has been studying the influence of hemodialysis and recombinant human erythropoietin on the functioning of T lymphocytes. She is currently starting research aimed at understanding how chronic stress affects the function of the immune system in patients after kidney transplantation. The results obtained may contribute to the creation of recommendations for the implementation of psychological support programs for transplant patients in the future, to minimize the impact of stress and negative perception of the disease on their health.

Dr hab. Tomasz Ślebioda (Medical University of Gdańsk)



Tomasz Ślebiota graduated in biotechnology from the Intercollegiate Faculty of Biotechnology, University of Gdańsk, and the Medical University of Gdańsk in 2006. He received a PhD in immunology from the University of Southampton (UK) in 2010. Presently, he works at the Department of Histology, Medical University of Gdańsk. His entire research career has been focused on the biology of T cells, with a special emphasis on the TL1A/DR3 axis in the costimulation of T cells. Currently, Tomasz is working on the development of novel modifications of CAR-T cells, investigating the molecular interactions of daratumumab with T-ALL cells, and is also involved in studies on extracellular vesicles in glioblastoma development.

Dr hab. Weronika Hewelt-Belka (Gdańsk University of Technology)

Weronika Hewelt-Belka works as an Associate Professor at the Department of Analytical Chemistry, Faculty of Chemistry, Gdańsk University of Technology, where she obtained her PhD in chemical sciences. Her research focuses mainly on the development and application of new methods for lipidomic analysis using advanced hyphenated techniques. She has experience in high-resolution mass spectrometry and high-performance chromatography techniques for the lipidomic analysis of various types of biological samples, including human milk, bacterial cells, and extracellular vesicles. She currently leads an NCN project aiming at the development of new LC-MS-based tools for the separation and lipidomic characterization of extracellular vesicles. Collaborating with Polish and international teams, she uses analytical chemistry to address key biological questions in immunology, personalized medicine, and nutrition.

Organizing Team

- **Reza Abouali,** MSc, PhD candidate – Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology, University of Gdańsk and Medical University of Gdańsk
- **Mikołaj Klimczuk,** MSc, PhD candidate – Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology University of Gdańsk and Medical University of Gdańsk
- **Aniela Kosobucka,** BSc, MSc student – Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology, University of Gdańsk and Medical University of Gdańsk
- **Martyna Misztal,** MSc, PhD candidate – Department of Physiopathology, Faculty of Medicine, Medical University of Gdańsk
- **Argho Aninda Paul,** MSc, PhD candidate – Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology, University of Gdańsk and Medical University of Gdańsk

Conference Sponsors

We gratefully acknowledge the generous support of our sponsors, whose contributions have been instrumental in making this conference possible. Their commitment to advancing scientific research and innovation is reflected in their active engagement with the academic and scientific community. Below, you will find introductions from each of our sponsors, highlighting their mission, vision, and role in supporting scientific excellence.

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Speakers

Keynote Speaker: Prof. Graham Ogg

Radcliffe Department of Medicine

University of Oxford, Oxford, England

Graham Ogg is a Professor of Dermatology, a Deputy Director of the MRC Translational Immune Discovery Unit (TIDU) at the University of Oxford, and a Leader of the Translational Dermatology Unit at the Weatherall Institute of Molecular Medicine. Graham completed his doctoral training at the University of Oxford and his clinical training in Oxford and London.

He is a Medical Research Council (MRC) Programme Leader, a recent recipient of the Wellcome Discovery Award, a National Institute of Health Research (NIHR) Senior Investigator and the UK's Clinical Lead for Dermatology. His numerous appointments include the Interim Director of the MRC Human Immunology Unit, Chairman of the British Society of Investigative Dermatology, Chairman of the British Association of Dermatologists Research Committee, and a specialty clinical lead for the NIHR Clinical Research Network. He is also an elected Fellow of the Academy of Medical Sciences, and a Co-Founder and Director of Orbit Discovery Ltd, and T-Cypher Bio Ltd.

In his research work, Graham investigates mechanisms underlying the role of T cells in inflammatory skin disease. His main focus is on studying CD1a, an MHC-like molecule, which can present lipid antigens to CD1a-reactive T cells. Such T cells are now known to contribute to different forms of cutaneous inflammation. The pathway opens experimental medicine approaches to help delineate the biology underlying clinical observation, to progress towards translational benefit for patients with skin disease.

Clinical immunology

Prof. dr hab. Ewelina Grywalska

Prof. Ewelina Grywalska is a clinical immunologist and professor at the Medical University of Lublin. She serves as the Regional Consultant in Clinical Immunology and is the President of the Polish Society of Experimental and Clinical Immunology. Her research focuses on immunology, oncology, and autoimmune diseases.

Lecture title: **Humoral immunodeficiency associated with chronic lymphocytic leukemia**

Prof. dr hab. Marek Niedoszytko

Prof. Marek Niedoszytko is an allergist, pulmonologist, and internist who heads the Department of Allergology at the Medical University in Gdansk. His research is focused on asthma, mastocytosis, MCAS, Hymenoptera, drug, food allergy, allergen immunotherapy, and desensitization. The studies on allergen immunotherapy in Hymenoptera and inhalant allergy will be presented during the talk. Close cooperation between clinicians, immunologists, and geneticists allows us to design tailored therapy for individual patients.

Lecture title: **Allergen immunotherapy as the only causative treatment of allergy**

Dr Anna Masiak

She is a physician specializing in internal medicine, rheumatology, and clinical immunology and a university teacher at the Medical University of Gdańsk (MUG). Her primary field of interest is the clinical diversity of systemic vasculitides, with a particular focus on granulomatosis with polyangiitis. During the lecture, she will present the pathogenesis of GPA in relation to the clinical presentation and treatment options.

Lecture title: **Granulomatosis with polyangiitis from pathogenesis to clinical presentation and treatment**

Dr Jacek Rutkowski and Dr Piotr Wysocki

They are medical doctors specializing in radiation oncology. They are also involved in research on immunomodulation in cancer treatment and participate in multiple clinical trials focused on melanoma, lung cancer, and gynecological malignancies.

Lecture title: **Taming Complexity: Immunomodulation in cancer – challenges and opportunities**

Experimental Immunology

Dr hab. Aleksandra Rutkowska

Dr hab. Aleksandra Rutkowska is a neuroscientist and group leader at the Medical University of Gdańsk with a research focus on neuroimmune interactions in the central nervous system. She completed her MSc and PhD in Neuroscience at Trinity College Dublin. Her current work further explores how EBI2/oxysterol signalling shapes immune–glial communication, particularly in the context of blood–brain barrier regulation and remyelination in multiple sclerosis. Ola's research aims to uncover immune-driven mechanisms that support repair processes in the inflamed CNS.

Lecture title: **Bridging CNS remyelination and immune regulation through EBI2/oxysterol signaling**

Dr hab. Andrea Lipińska

Dr hab. Andrea Lipińska is a Research Associate at the Department of Virus Molecular Biology of the Intercollegiate Faculty of Biotechnology and the Head of the BSL3+ Microbiology Laboratory at IFB – a passionate virologist, specialising in virus interactions with the immune system and virus immune evasion. She will give an introduction to virus immune subversion and tell the story of a herpesvirus small protein UL49.5, hijacking the cellular protein degradation pathway to compromise antigen presentation and antivirus immune response.

Lecture title: **A small viral thief and a cellular VIP protein – how viruses hijack cellular pathways for immune subversion**

Prof. dr hab. Natalia Marek-Trzonkowska

Prof. Natalia Marek-Trzonkowska is the director of the International Center for Research on Anticancer Vaccines (ICCVS) at the University of Gdańsk. Her research focuses on the clinical application of immune cells. During her talk, she will discuss the importance of understanding the diversity of populations comprising the cancer microenvironment, as well as the impact of a research model on cancer immunogenicity.

Lecture title: **Limitations of preclinical models and cancer heterogeneity – challenges for cancer immunotherapy**

Dr hab. Patrycja Koszałka

Dr hab. Patrycja Koszałka is an assistant professor at the Laboratory of Cell Biology and Immunology, Institute of Medical Biotechnology and Experimental Oncology, Medical University of Gdańsk. Her primary research focuses on nucleotide metabolism's influence on the tumor microenvironment in cancer development and progression, applying murine models. She will present the challenges associated with analyzing the immunomodulatory role of CD73, an extracellular adenosine-generating enzyme, in breast cancer development and progression.

Lecture title: **CD73 (ecto-5'-nucleotidase) regulatory role in anticancer immune response – using murine models to research the mechanism and clinical response**

Translational Immunology

Dr Wojtek Siwek

Wojtek Siwek is a group leader at the International Centre for Cancer Vaccine Science (ICCVS), University of Gdańsk. His doctoral training was in biochemistry at the International Institute of Molecular and Cell Biology in Warsaw (IIMCB), followed by postdoctoral fellowships at the Gulbenkian Institute (Portugal), the University of Oxford (UK), and Harvard Medical School (USA). He will be presenting on transcriptional memory and our laboratory's aim to translate this research into the clinic.

Lecture title: **Mechanisms of interferon-gamma (IFNγ) transcriptional memory**

Prof. dr hab. Marcin Okrój

Marcin Okrój is employed at the Medical University of Gdańsk as a professor and the head of the research group interested in the complement system, a constituent of innate immunity. The group is focused on developing new diagnostic tools, complement-based approaches to immunotherapy, and interactions of the complement system with tumor cells. The recent invention to be presented during the conference is a universal antibody that seeks and destroys tumor cells resistant to numerous first-line anticancer mAbs.

Lecture title: **Anti-C4d antibody – a universal agent targeting tumor cells resistant to first-line immunotherapy**

Dr hab. Danuta Gutowska-Owsiak

Danuta Gutowska-Owsiak leads the Laboratory of Experimental and Translational Immunology at the Intercollegiate Faculty of Biotechnology UG and MUG. Together with her team, she investigates structural and immunological skin barrier and crosstalk between non-immune and immune cells, including the role of extracellular vesicles. She is interested in both very basic mechanisms and translational aspects of allergy, atopic dermatitis, and beyond. She will present results on the induction of tolerogenic function in dendritic cells.

Lecture title: **Metallothionein upregulation as a potential novel mechanism driving induction of tolerogenic functionality in “flash DC-10” cells induced by a brief exposure to interleukin 10**

Dr hab. Tomasz Ślebioda

Tomasz Ślebioda is an assistant professor at the Department of Histology, Medical University of Gdańsk. His primary research interests are focused on the biology of T cells and their role in antitumor response. He will present the role of CAR-T cells in antitumor therapy, challenges associated with this type of treatment, and the potential novel costimulatory domain that can enhance the efficacy of CAR-T cells.

Lecture title: **Death receptor 3 as a potential costimulatory domain for CAR-T cells**

Joint Venture

Dr hab. Weronika Hewelt-Belka

Weronika Hewelt-Belka works as an Associate Professor at the Department of Analytical Chemistry, Faculty of Chemistry, Gdańsk University of Technology. Her research focuses mainly on the development and application of new methods for lipidomic analysis using advanced hyphenated techniques. During the presentation, she will discuss the application of lipidomics as a tool in immunological research of extracellular vesicles. She will focus on lipid analysis using advanced LC-MS techniques, enabling detailed characterization of the lipid profiles of EVs and their role in immunological processes.

Lecture title: **Lipidomics as a tool in immunological research of extracellular vesicles – analytical perspectives**

Prof. dr hab. Michał Pikuła

Prof. Michał Pikuła is a Full Professor and Group Leader at the Laboratory of Tissue Engineering and Regenerative Medicine, Department of Embryology at the Medical University of Gdansk (MUG), Poland. His scientific interests focus on the biology of skin cells, adipose-derived stem cells (ADSCs), wound healing, and the development of pro-regenerative compounds, including peptides, exosomes, and plasma-derived components. He will give a speech about the safety of biomaterials and peptides in cell biology, immunology, chemistry, and tissue engineering.

Lecture title: **Immunological safety of biomaterials and peptides for regenerative medicine**

Dr hab. Jakub Mieczkowski

Jakub Mieczkowski is a computational biologist specializing in multiomic analysis, with a focus on the epigenetic regulation of gene expression and early detection of cancer. He leads a research group at the Medical University of Gdańsk and collaborates closely with clinicians to develop minimally invasive diagnostic tools. His work combines advanced genomic technologies, including single-cell and spatial transcriptomics, with AI-driven analysis of clinical imaging data. He is also actively involved in international consortia, including advisory roles at the European Space Agency.

Lecture title: **Radiation-induced dysregulation of hematopoietic differentiation: insights from CD34+ cells**

Dr inż. Ilona Kłosowska-Chomiczewska

Dr inż. Ilona Kłosowska-Chomiczewska is a researcher at Gdańsk University of Technology, specializing in colloidal science and digestion models. She works on the development, optimization, and application of in vitro models simulating the human digestive system. She will present digestion models that mimic the human gastrointestinal tract and discuss their potential application in assessing the fate of food and drug components that may trigger inappropriate immune responses, particularly under conditions of impaired intestinal mucus barrier and altered nutrient absorption.

Lecture title: ***In vitro*** **digestion models as tools to unravel immunological responses to food compounds**

Abstracts

1. **Demographic and clinical presentation of neuropsychiatric manifestations of SLE in patients diagnosed with lupus nephropathy** – Adamski et al.

2. **Changes in peripheral T cell subsets in undifferentiated arthritis patients progressing to rheumatoid arthritis** – Bzoma et al.

3. **Molecular and functional effects of EBI2 receptor signaling in glial cells and remyelination in organotypic cerebellar slices** – Fatimah and Rutkowska

4. **Photodynamic disarmament of *S. aureus* toxins in atopic dermatitis** – Jaśkiewicz et al.

5. **Characterization of tumor LDs and sEVs and their role in modulating CD1a-restricted T cell responses** – Klimczuk et al.

6. **Intracellular lipid transfer as a possible factor shaping the repertoire of CD1a antigens in the membranes of small extracellular vesicles** – Kosobucka et al.

7. **Immunomodulatory properties of peptide ligands of BTLA-HVEM complex** – Misztal et al.

8. **Analysis of extracellular vesicle lipid content: comparing direct injection LC-MS to traditional extraction methods for immunological applications** – Młynarczyk et al.

9. **The most efficient IL-10-induced tolerogenic dendritic cells upregulate metallothionein expression** – Panek et al.

10. **Controllable release of immunogen for the induction of specific T cell response** – Paul et al.

11. **CD1 antigens can be found in the lungs and are enriched in small extracellular vesicles isolated from bronchoalveolar lavage fluid** – Abouali et al.

12. **CD73 regulates the lytic to non-lytic switch of pyroptosis in metastatic breast cancer** – Serafin et al.

13. **Interferon-α increases expression of ISGylation-related genes and accumulation of profilaggrin in differentiated keratinocytes** – Siedlar et al.

14. **The key player of the immune system that impacts all – gut microbiome** – Sobiecki et al.

15. **Podocytes synthesize RNA of all IgG receptors, which level varies depending on glycemia** – Typiak et al.

16. **Investigating the neuroimmunomodulatory effects of psychedelics via 5-HT_2A and Sigma-1 receptors** – Yu and Rutkowska

17. **The pro-inflammatory status of monocytes in patients with systemic lupus erythromatosus (SLE)** – Zielińska et al.

1. Demographic and clinical presentation of neuropsychiatric manifestations of SLE in patients diagnosed with lupus nephropathy

Adamski I[1], Komorniczak M[2], Bułło-Piontecka B[2], Dębska-Ślizień A[2], Wardowska A[3]

[1]Student Scientific Circle at the Department of Physiopathology, Medical University of Gdańsk, Poland

[2]Department of Nephrology, Transplantology and Internal Diseases, Medical University of Gdańsk, Poland

[3]Department of Physiopathology, Medical University of Gdańsk, Poland

Neuropsychiatric systemic lupus erythematosus (NPSLE) is one of the most severe forms of SLE due to high risk of death and disability, and indicates the involvement of the nervous system by immunopathological processes. This pathomechanism is diverse in individual manifestations of NPSLE, complicated and not fully understood. However, it is known that many elements of the immune system are involved.

The aim of this study was the demographic analysis and identification of the most common manifestations of NPSLE in accordance with the criteria of the American College of Rheumatology from 1999 in patients diagnosed with SLE and lupus nephritis (LN). Moreover, we tried to discuss the immunopathology of this phenomenon.

The analysis included patients under the care of the Department of Nephrology, Transplantology and Internal Diseases, MUG in Gdańsk since 2010 until May 2025. A retrospective analysis was performed on patients with SLE and LN who were diagnosed with involvement of the central and/or peripheral nervous system due to the underlying disease, confirmed by laboratory tests, imaging tests, and the simultaneous exclusion of other potential causes, which represented 24,62% of all lupus patients under the clinic's care.

32 patients were examined: 26 women (81.25%) and 6 men (18.75%). The main manifestations of NPSLE included headaches (59.375%), cerebrovascular diseases (43.75%), polyneuropathy/mononeuropathies (40.625%), seizure disorder (25%), movement disorders (12.5%), cognitive disorders (9.375%), anxiety disorders (3.125%), aseptic meningitis (3.125%). 50% of women and 83.3% of men experienced severe forms of NPSLE, reckoned in this analysis as cerebrovascular disease and/or epilepsy and/or psychosis.

Patients suffering from SLE and LN most often develop the following manifestations: headaches, cerebrovascular diseases, polyneuropathy/mononeuropathy, and seizure disorder. It suggests a severe course of NPSLE in this group. The major limitation of the study is the small size of the study group, therefore the analysis of a larger group of patients is required to confirm the findings. The present picture suggests the involvement of the nervous system by immunological processes – a comparison of the clinical and laboratory picture of

patients with the available literature suggests that the excess immune complexes, deposited in the body, cause LN, may also lead to damage and unsealing of the blood-brain barrier, thus allowing the spread of pathological immunological processes in the central nervous.

2. Changes in peripheral T cell subsets in undifferentiated arthritis patients progressing to rheumatoid arthritis

Izabella Bzoma[1], Edyta Brzustewicz[1], Agnieszka Daca[1], Maria Szarecka[2], Małgorzata Sochocka-Bykowska[2], Jacek M. Witkowski[1,3], Ewa Bryl[1]

[1]Department of Physiopathology, Faculty of Medicine, Medical University of Gdansk, 80-210 Gdansk, Poland.

[2]Pomeranian Rheumatology Center, 81-759 Sopot, Poland

[3]Department of Embryology, Faculty of Medicine, Medical University of Gdansk, 80-210 Gdansk, Poland.

Early treatment of rheumatoid arthritis (RA) has been shown to prevent joint damage and disability. However, reliable biomarkers that allow early differentiation of RA from undifferentiated arthritis (UA) are still needed. This study aimed to investigate peripheral $CD4^+$ and $CD8^+$ T cell subpopulations in patients progressing from UA to RA (UA→RA) and to identify potential predictive biomarkers of such progression.

Fifty-eight untreated UA patients with at least one peripheral joint showing signs of inflammation lasting 2–4 months were enrolled in the study. Peripheral blood was collected at baseline, and T cell phenotyping was performed using multicolor flow cytometry. Clinical diagnoses were confirmed 6 to 12 months after sample collection, and a retrospective (post hoc) analysis was conducted.

UA patients who later progressed to RA had significantly higher percentages of $CD4^+CD3^+CD95^+$, $CD4^+CD3^+CD95L^+$ cells, and double-positive apoptotic marker-expressing cells ($CD4^+CD3^+CD95^+CD95L^+$), compared to healthy controls. Similarly, $CD8^+$ T cells from UA→RA patients showed increased percentages of $CD8^+CD3^+CD95^+$ and $CD8^+CD3^+CD95L^+$ subsets.

Our findings indicate that changes in specific T cell subpopulations and apoptotic marker expression are detectable in the early stages of undifferentiated arthritis. These alterations may serve as early biomarkers for progression to rheumatoid arthritis.

3. Molecular and functional effects of EBI2 receptor signaling in glial cells and remyelination in organotypic cerebellar slices

Fatimah[1], Aleksandra Rutkowska[1]

[1]Department of Anatomy and Neurobiology, Medical University Gdańsk, Gdańsk, Poland

Epstein-Barr virus-induced gene 2 (EBI2, GPR183) and its oxysterol ligand 7α,25-dihydroxycholesterol (7α,25OHC) play a critical role in immune cell migration and have been implicated in neuroinflammatory and neurodegenerative diseases, including multiple sclerosis (MS). EBI2 is expressed in various immune cells such as B and T cells, dendritic cells, and natural killer cells as well as in astrocytes, oligodendrocytes, and hematopoietic stem cells. In the central nervous system (CNS), EBI2 is upregulated in inflamed glial cells and facilitates astrocyte-macrophage communication. Astrocytes and microglia produce oxysterols in response to inflammatory stimuli, further modulating EBI2 signaling. EBI2 is also transiently upregulated during oligodendrocyte maturation, and 7α,25OHC promotes oligodendrocyte progenitor cells (OPCs) migration, suggesting a role in remyelination. High expression of EBI2 in immune cells and their accumulation in MS lesions supports the idea that the EBI2/7α,25OHC pathway facilitates the migration of inflammatory cells to sites of CNS inflammation in MS. These findings suggest EBI2 signaling is a key regulator of neuroimmune interactions and myelin biology, highlighting its therapeutic potential in demyelinating diseases such as MS.

This study will further investigate the molecular and functional effects of EBI2 signaling in glial cells including astrocytes, microglia, and OPCs isolated from EBI2 knockout (KO) and WT mice. Cells will be treated with cytokines (e.g., TNF, IL-17), lysophosphatidylcholine (LPC) or lipopolysaccharide (LPS) and CF3-7α,25OHC to assess EBI2-dependent pathways. Functional outcomes in glial cells such as differentiation, maturation, and inflammatory responses will be evaluated using ELISA, qPCR, Western blot (WB), immunocytochemistry (ICC) along with (re)myelination in organotypic cerebellar slices. The central hypothesis suggests that EBI2 promotes glial cell function and enhances remyelination in the CNS.

Funding:

This project received funding from the National Science Centre, Poland, grant registration number: 2022/47/D/NZ3/02613 (AR).

4. Photodynamic disarmament of *S. aureus* toxins in atopic dermatitis

Patrycja Ogonowska[1], Adrian Kobiela[2], Danuta Gutowska-Owsiak[2], Dominika Goik[1] , Maciej Jaśkiewicz[1] Joanna Nakonieczna[1*]

[1]Laboratory of Photobiology and Molecular Diagnostics, Intercollegiate Faculty of Biotechnology, University of Gdańsk and Medical University of Gdańsk, Poland

[2]Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology, University of Gdańsk and Medical University of Gdańsk, Poland

*correspondence E-mail: joanna.nakonieczna@biotech.ug.edu.pl

Staphylococcus aureus is a well-known opportunistic pathogen capable of causing severe infections such as pneumonia and osteomyelitis [1]. However, it preferentially targets the skin, facilitated by specific virulence factors that promote adhesion and the cytolysis of neutrophils [2,3]. Among these factors, superantigens (SAgs) play a critical role in the exacerbation of skin lesions in atopic dermatitis (AD) by binding to MHC class II molecules and T-cell receptors, resulting in uncontrolled T-cell activation, proliferation, and cytokine release [4]. The most common and potent SAgs secreted by *S. aureus* include staphylococcal enterotoxins, namely SEA, SEB, SEC, SED, and TSST-1]. A significant number of recurrent skin infections have been attributed to methicillin-resistant *S. aureus* (MRSA), which limits conventional therapeutic options. Antimicrobial photodynamic inactivation (aPDI) has emerged as a promising alternative, utilizing non-toxic photosensitizers in combination with light of specific wavelengths to generate reactive oxygen species (ROS) [5]. ROS can damage essential biological molecules, leading to bacterial cell death without promoting resistance development. Multiple studies have demonstrated the efficacy of aPDI against *S. aureus* infections, including its potential to inactivate bacterial virulence factors [6]. In this study, we evaluated the capacity of aPDI using Rose Bengal (RB) and new methylene blue (NMB) to inactivate *S. aureus* superantigens *in vitro* and reduce their biological activity. Our results indicate that the efficacy of aPDI depends on the specific photosensitizer and light combination. Notably, RB combined with green light significantly inactivated enterotoxins, leading to a marked reduction in T-cell proliferation. In contrast, NMB combined with red light showed limited inactivation of the tested toxins. These findings suggest that optimized aPDI protocols may serve as an effective strategy for reducing *S. aureus* colonization and superantigen-mediated inflammation in atopic dermatitis.

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5. Characterization of tumor LDs and sEVs and their role in modulating CD1a-restricted T cell responses

Mikołaj Klimczuk[1], Aniela Kosobucka[1], Michał Młynarczyk[2], Weronika Hewelt-Belka[2] and Danuta Gutowska-Owsiak[1]

[1]Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology, University of Gdańsk & Medical University of Gdańsk, Poland

[2] Department of Analytical Chemistry, Gdańsk University of Technology, Poland

Lipid droplets (LDs) and small extracellular vesicles (sEVs) are increasingly recognized as important players in the regulation of immune responses and cancer progression. Beyond their traditional roles in lipid storage and cellular communication, LDs and sEVs intersect at the interface of cellular lipid metabolism and immune modulation.

We hypothesise that in tumor cells, reprogramming of the lipid metabolic pathways could alter the lipid composition of sEVs and LDs. Specifically, the abundance of non-permissive (inhibitory) and permissive (stimulatory) lipids may change possibly affecting their ability to modulate lipid-specific T cell responses.

Samples of LDs and sEVs were isolated from bladder (SV-HUC-1, RT4, T24) and breast (HB2, MCF7) normal and cancer cell lines. Morphological parameters of LDs were assessed using the Tomocube HT-2 microscope. Preliminary lipidomic mass spectrometry was performed to assess the lipid composition of isolated sEVs derived from bladder cell lines. The effect of CD1a-restricted T cell responses was performed with IFNγ ELISpot assay with CD1a-transfected K562 cells serving as antigen presenting cells.

In sEVs isolated from the bladder lines, lipidomic mass spectrometry detected differences in the content of nonpermissive lipids in the tumorigenic lines in contrast to non-tumorigenic.

We determined that the addition of lipid antigens to the coculture of CD1a-K562 cells with T cells reduced CD1a-dependent IFNγ responses in comparison to the control unpulsed cells. Additionally, pulsing with lipids derived from LDs resulted in increased IFNγ responses in comparison to the lipids from sEVs of the same line.

The gathered results suggest that lipids contained within secreted sEVs by tumors of different invasiveness differ in the content of nonpermissive and permissive ligands. Additionally, sEV lipids secreted by tumors contribute to differential activation of the CD1a-restricted T cells and may promote tumor evasion.

Understanding the crosstalk between LD and sEV lipid composition provides promising insights for decoding tumor-immune interactions.

6. Intracellular lipid transfer as a possible factor shaping the repertoire of CD1a antigens in the membranes of small extracellular vesicles

Aniela Kosobucka[1], Mikołaj Klimczuk[1], Rafał Sądej[2], Danuta Gutowska-Owsiak[1]

[1]Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology University of Gdańsk and Medical University of Gdańsk

[2]Division of Molecular Enzymology and Oncology, Intercollegiate Faculty of Biotechnology University of Gdańsk and Medical University of Gdańsk

Lipid droplets (LDs) are considered the main intracellular source of lipids. Small extracellular vesicles (sEVs) mediate intercellular communication and may contain antigenic lipids for CD1a-mediated presentation. The lipidome of sEVs can be involved in immune modulation, however the intracellular origin of antigenic lipids remains unclear. This study investigates the potential role of LDs as a lipid source for sEVs and explores the role of lipid transfer proteins (e.g. VPS13A) in this process.

Lipid droplet parameters in different cell lines and the effect of rapamycin-induced LD blockade in HepG2 cells were analysed using holotomographic microscopy. sEVs were isolated from HepG2 cells via differential ultracentrifugation and characterised by Western Blot and NTA. Using publicly available Kaplan-Meier data, mRNA expression levels of lipid transfer proteins were correlated in different cancer types with recurrence-free survival and progression-free survival under immune checkpoint inhibitor therapy. A VPS13A knockout HepG2 cells were obtained using CRISPR technology.

In tumour lines compared to control, the same trend of decreased refractive index values of LDs is observed, and similar effect is present after rapamycin treatment of HepG2 cells. Higher expression of VPS13A, ATG9A and ATG2A is positively correlated with recurrence-free survival of patients with different tumours and their progression-free survival in response to immunotherapy. An opposite trend is observed for VMP1 enzyme, which high expression negatively correlates with both survival and response to immunotherapy of cancer patients.

Differences in refractive index values of LDs may reflect changes in their contents after metabolic rewiring during tumorigenesis. Expression of lipid transfer proteins is correlated with recurrence of different tumours and their response to immunotherapy, possibly indicating the important role of intracellular lipid transfer in modulation of anti-tumour T cell responses. However, lipidomic and functional analyses of sEVs produced by analysed cells need to be conducted to draw more final conclusions.

7. Immunomodulatory properties of peptide ligands of BTLA-HVEM complex

Martyna Misztal[1], Marta Spodzieja[2], Anna Wardowska[1]

[1]Department of Physiopathology, Faculty of Medicine, Medical University of Gdańsk, Poland

[2]Department of Biomedical Chemistry, Faculty of Chemistry, University of Gdańsk, Poland

BTLA (B and T Lymphocyte Attenuator) and HVEM (Herpes Virus Entry Mediator) are a pair of receptors that form an inhibitory immune checkpoint (ICP). The BTLA-HVEM interaction is distinct from other inhibitory ICPs, such as PD-1 or CTLA-4, because BTLA ligation inhibits lymphocyte activation, while the binding of HVEM induces pro-survival signaling. This unique feature makes the BTLA-HVEM checkpoint an attractive therapeutic target in both cancer and autoimmune diseases where the immune system is dysregulated.

Two chemically synthesized peptides, BTLA (35-43) and BTLA (33-64), were designed based on the fragment of the BTLA protein sequence that binds the HVEM protein. Peptides were previously confirmed to block the formation of the BTLA-HVEM complex in vitro. Studies using cell lines have also shown that these peptides can inhibit HVEM-induced pro-survival signaling. To assess the biological activity of these peptides, peripheral blood mononuclear cells (PBMC) were isolated from healthy donors and cultured in vitro in the presence of the peptides. To evaluate whether peptides can reduce the strength of the immune cell activation, cells were previously stimulated with a human anti-CD3 antibody. After 3 and 5 days of culture, PBMCs were collected, and the levels of lymphocyte activation markers were measured using flow cytometry.

A modest decrease in the expression of activation markers CD25 and HLA-DR was observed in both groups, treated with peptide BTLA (35-43) and peptide BTLA (33-64) at both incubation times. Additionally, the differences were present in the percentage of the population expressing each marker, as well as the number of molecules on each cell, measured using the mean fluorescence index (MFI).

These results suggest that the peptides may suppress the immune response, supporting further studies using cells from patients with autoimmune diseases.

8. Analysis of extracellular vesicle lipid content: comparing direct injection LC-MS to traditional extraction methods for immunological applications

Michał Młynarczyk[1], Jorge Matinha-Cardoso[2], Felicja Gajdowska[3], Danuta Gutowska-Owsiak[3], Paulo Oliveira[4], Weronika Hewelt-Belka[1]

[1]Department of Analytical Chemistry, Faculty of Chemistry, Gdańsk University of Technology, Gdańsk, Poland

[2]Interdisciplinary Centre of Marine and Environmental Research (CIIMAR/CIMAR), University of Porto, Matosinhos, Portugal

MCbiology Doctoral Program, ICBAS – Instituto de Ciências Biomédicas Abel Salazar, Universidade do Porto, Porto, Portugal

[3]Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology of University of Gdansk and Medical University of Gdansk, University of Gdansk, Gdansk, Poland

[4]Interdisciplinary Centre of Marine and Environmental Research (CIIMAR/CIMAR), University of Porto, Matosinhos, Portugal, Department of Biology, Faculty of Sciences, University of Porto, Porto, Portugal

Extracellular vesicles (EVs) are nanosized, membrane-bound nanobioparticles secreted by both eukaryotic and prokaryotic cells. They carry diverse bioactive molecules, including proteins, lipids, nucleic acids, and membrane-bound receptors, playing essential roles in cell communication, immune modulation, and disease progression. Their lipid composition is particularly significant due to its influence on vesicle structure, function, and interaction with immune cells.

However, lipidomic characterization of EVs is often limited by small sample volume and the need for complex lipid extraction procedures. In this study, we evaluated a direct injection liquid chromatography-mass spectrometry (DI-LC-MS) approach to lipidomic profiling of EVs, bypassing the need for conventional extraction techniques, like liquid-liquid extraction(LLE), or solid-phase extraction (SPE). Using reverse-phase liquid chromatography coupled with quadrupole time-of-flight mass spectrometry (RP-LC-Q-TOF-MS), we analysed EVs isolated from eukaryotic and bacterial cells sources, including human plasma, fetal bovine serum, cyanobacterial cultures. Our results demonstrate that DI-LC-MS provides lipidome coverage comparable to traditional extraction methods (e.g., liquid-liquid extraction), while significantly reducing chemical background, analysis time, and the risk of analyte loss. This streamlined workflow allows for the analysis of minimal sample volumes (e.g., 0.1 µL), making it especially suitable for immunological applications where sample availability is often limited. The ability to rapidly and reproducibly analyze EV lipidomes without prior extraction enhances the feasibility of applying lipidomics in immunological studies. DI-LC-MS proves to be a

powerful tool for investigating the lipid composition of EVs in the context of immune responses, inflammation, and disease, supporting the development of novel diagnostic and therapeutic strategies.

This research was funded by the National Science Centre, Poland, grant number (2021/43/D/ST4/02872).

9. The most efficient IL-10-induced tolerogenic dendritic cells upregulate metallothionein expression

K. Panek[1], M. Klimczuk[1], R. Abouali[1], F. Santoni de Sio[2], A. Siedlar[1], A. Kobiela[3], S. Gregori[2], D. Gutowska-Owsiak[1]

[1]Experimental and Translational Immunology Group, Intercollegiate Faculty of Biotechnology of University of Gdansk and Medical University of Gdansk, University of Gdansk, Gdansk, Poland

[2]Mechanisms of Peripheral Tolerance Group, Immune Core, San Raffaele Telethon Institute for Gene Therapy, Milan, Italy

[3] Medical Research Council (MRC) Human Immunology Unit, Medical Research Council (MRC) Weatherall Institute of Molecular Medicine, National Institute for Health Research (NIHR) Oxford Biomedical Research Centre, Radcliffe Department of Medicine, University of Oxford, Oxford, United Kingdom

Tolerogenic dendritic cells (tolDC) induce a state of immunological tolerance in T cells. The time of provision of antigen and tolerizing stimulus affects the tolDCs phenotype and impacts the establishment of antigen-specific responses. Here, we aimed to understand the mechanism of DC characteristics acquisition depending on the time of IL-10 stimulation during the antigen exposure.

DCs were differentiated from monocytes by IL-4/GM-CSF and treated with CEFT antigen pool and lipopolysaccharide on day 5. A tolerizing factor (IL-10) was provided on days 3, 4, 5 or 6 for 24h. After 7 days, differently generated DC populations were characterized phenotypically (CD40, CD80, CD86, HLA-DR marker expression), included in a co-culture with autologous T cells in an IFN-γ ELISpot assay, and RNA isolated from DC models was sequenced.

The best tolerogenic phenotype and function of tolDC was obtained when IL-10, LPS and antigens were provided together (on day 5), however, provision of IL-10 24h after the LPS and antigen stimulation (on day 6) failed to induce tolerogenic characteristics. From Differential Gene Expression (DGE) analysis, we found that the most tolerogenic DC subset, D5 tolDC, significantly increased the expression of genes encoding members of the metallothionein family (MT) compared to the control cells, mature DC. Also, *MT1G* and *MT1H* genes were the most significantly downregulated genes in D6 tolDC compared to D5 tolDC. qPCR experiments confirmed DGE results.

MT proteins are responsible for zinc buffering, which is a crucial element for maintaining immunological homeostasis, including antigen-specific tolerance. Our findings could be important for the development of improved therapeutic approaches in the fields of autoimmune diseases, allergies and graft acceptance.

10. Controllable release of immunogen for the induction of specific T cell response

Argho Aninda Paul[1], Ewa Czechowska[1], Yusuke Sakai[2], Jonathan Heddle[2], Piotr Karaś[1] Margarida Rei[3], Danuta Gutowska-Owsiak[1]

[1]Experimental and Translational Immunology Group "DGO Lab", Intercollegiate Faculty of Biotechnology of University of Gdańsk & Medical University of Gdańsk, Gdańsk, Poland.

[2]Malopolska Centre of Biotechnology, Uniwersytet Jagielloński w Krakowie, Krakow, Poland.

[3] MRC Human Immunology Unit, University of Oxford, Oxford, United Kingdom.

Immunogen are often metabolized rapidly or excreted from the body in addition efficacy of the immunogens in its putative forms markedly reduced due to the nonspecific cell and tissue distribution, these limitation can be overcome by using nanocarriers of different type. In our study, we used a nanocarrier designed to release the cargo upon the specific trigger.

To assess the controlled release of the cargo, peptide specific IFN-γ ELISpot assay was performed. In brief, NY-ESO-1 (157-165) peptide, a model immunogen; was loaded on the nanocarrier at different loading ratio (nanocarrier: peptide= 1:1000,1:500,1:250 & 1:100) and peptide loaded nanocarriers were pre-treated with or without peptide releasing cue followed by pulsing of those nanocarrier at 0.1, 0.25, 0.5 & 1nM concentration to T2 (APC) cells for 4 hours. Pulsed T2 cells were co-cultured with NY-ESO-1 specific 1G4 transduced primary $CD8^+$ cells in ELISpot plate for 16 hours and IFN-γ spots were considered as T cell activation response.

Compared to the control nanocarrier (without the releasing trigger), specific release of the peptide yielded approximately 140-200% higher T cell response, in four tested loading ratio and concentration, indicating the controllability of the antigen release. Among these four tested loading ratio and four concentration, 1:500 yielded highest T cell response compared to other three loading ratio in all four tested concentration where the highest T cell response obtained with 0.5nM of nanoparticle.

Since the controllable release of the peptide leads to significant increase in T cell response this nanoparticle can be used for the provision of immunogen to drive specific T cell response.

11. CD1 antigens can be found in the lungs and are enriched in small extracellular vesicles isolated from bronchoalveolar lavage fluid

Reza Abouali[1], Michał Młynarczyk[2], Jagoda Mantej[1], Alicja Janowicz[3], Karolina Kita[4], Weronika Hewelt-Belka[2], Danuta Gutowska-Owsiak[1]

[1]Laboratory of Experimental and Translational Immunology, Intercollegiate Faculty of Biotechnology, University of Gdansk, Gdansk, Poland

[2]Department of Analytical Chemistry, Faculty of Chemistry, Gdańsk University of Technology, Gdansk, Poland

[3]Department of Pulmonology & Allergology, Medical University of Gdańsk, Gdansk, Poland

[4]Department of Allergology, Medical University of Gdańsk, Gdansk, Poland

Small extracellular vesicles (sEVs) mediate immune regulation in the lungs, facilitating antigen presentation and lipid signaling. While immune responses mediated by proteins isolated from bronchoalveolar lavage fluid (BALF) have been extensively studied, the lipid composition of BALF-derived sEVs and their functional role in CD1-restricted immune responses remain largely unexplored. This study aims to determine lipid composition of the BALF and lung sEVs for future investigations of lipid presentation pathways.

BALF samples were collected from 17 patients with inflammatory lung diseases, including interstitial lung disease and asthma. sEVs were isolated via ultracentrifugation and characterized using nanoparticle tracking analysis (NTA) for size and concentration, transmission electron microscopy (TEM) for morphology, and Western blotting for protein markers. Lipidomic analysis was carried out using Liquid Chromatography-Quadrupole Time-of-Flight Mass Spectrometry.

Analysis of BALF samples revealed a consistent population of sEVs. The presence of sEVs was confirmed by NTA, TEM, and Western blot, showing expected particle size, intact vesicular structures, and expression of CD9, CD63, and Flotillin, with no detectable ApoA1, Calnexin, indicating no lipoprotein contamination. Computational lipid analysis identified multiple lipids detected in BALF-derived sEVs. Preliminary lipidomic results confirmed enrichment of relevant lipid classes in BALF-derived sEVs we isolated. These results suggest that BALF-derived sEVs may carry immunologically relevant lipids involved in CD1-mediated antigen presentation, warranting further functional validation.

These findings confirm the successful isolation of pure BALF-derived sEVs, validating their suitability for analysis of the lipid content. Computational and experimental lipid profiling identified CD1-presented lipids within sEVs, suggesting a potential role in lipid-mediated immune modulation. Future work will characterize these lipids and their role in CD1 antigen presentation.

12. CD73 regulates the lytic to non-lytic switch of pyroptosis in metastatic breast cancer

Paweł Kamil Serafin[1,2], Wiktoria Laprus[1], Marika Bolcewicz[3], Patryk Mucha[4], Kamila Bulak[5], Patrycja Koszałka[1]

[1]Laboratory of Cell Biology and Immunology, Institute of Medical Biotechnology and Experimental Oncology, Intercollegiate Faculty of Biotechnology University of Gdańsk and Medical University of Gdańsk, Medical University of Gdańsk, Poland;

[2]Experimental and Translational Immunology Group "DGO Lab", Intercollegiate Faculty of Biotechnology University of Gdańsk and Medical University of Gdańsk, University of Gdańsk, Poland;

[3]Department of Medical Laboratory Diagnostics—Fahrenheit Biobank BBMRI.pl, Faculty of Pharmacy, Medical University of Gdańsk, Poland;

[4]Department of Biochemistry, Faculty of Medicine, Medical University of Gdańsk, Poland;

[5]Department of Pathomorphology and Forensic Veterinary Medicine, University of Life Sciences in Lublin, Poland

CD73 (ecto-5'-nucleotidase), an enzyme generating extracellular adenosine, has a significant role in tumor progression. Its depletion has inhibited breast cancer (BC) growth and metastasis in syngeneic murine models. However, the link between CD73 expression and BC outcome is highly inconclusive for clinical data. The objective was to assess CD73's role in BC progression using a mouse model reflecting its complexity.

A murine model of chemically induced breast cancer was applied to analyze CD73 knock-out (KO)-induced changes in main parameters of cancer progression and gene expression at the transcriptome (RNA-seq) and proteome (IHC) levels. ELISA and dot blot analysis of patients' serum confirmed results from the murine model.

CD73 depletion significantly decreased the overall survival in mice with multiple mammary gland tumors (MGTs) and distant metastases. It did not affect the incidence or multiplicity of metastases; however, it increased the growth rate of all next developing tumors (next MGTs) and decreased their latency. Transcriptomic analysis of the next MGTs indicated a CD73 KO-induced activation of pyroptosis, Gasdermin (GSDM)-dependent pro-inflammatory programmed cell death. Activation of upstream genes regulating pyroptosis was confirmed at the protein level with a change in the cyclic GMP-AMP synthase (cGAS) - stimulator of interferon genes (STING) signaling pathway. However, the IHC analysis for cGAS, p-STING, GSDMD-NT, CASP1, and ALIX indicated a CD73 KO-induced switch from lytic to the non-lytic mode of pyroptosis. This switch was confirmed in patients' serum with dot blot analysis for GSDMD and ELISA quantification for IL-1β level.

CD73 KO can increase the tumor burden at the metastatic stage of breast cancer progression by deregulating pyroptosis. That may have implications for anti-CD73 therapy, both as monotherapy and in combination with immunotherapy for advanced BC.

13. Interferon-α increases expression of ISGylation-related genes and accumulation of profilaggrin in differentiated keratinocytes

Aleksandra Małgorzata Siedlar[1], Argho Aninda Paul[1], Danuta Gutowska-Owsiak[1]

[1]Experimental and Translational Immunology Group, Intercollegiate Faculty of Biotechnology of University of Gdansk and Medical University of Gdansk, University of Gdansk, Gdansk, Poland

Filaggrin is a protein with a key role in the skin. In keratinocytes, filaggrin is stored in granules as a profilaggrin. During differentiation, profilaggrin is released as monomers in a control manner since their excessive accumulation causes premature cell death.

Previously, we showed that profilaggrin undergoes turnover via the ubiquitin-proteasome pathway. Similar to ubiquitination, ISGylation process also results in covalent tagging of target proteins, but it is proposed that it may compete with or modulate ubiquitination, with a differential impact on protein stability. Interferon stimulation increases expression of *ISG-15* and related enzymes, leading to ISG-15 binding to target proteins. This study investigates the role of interferon-α-stimulated ISGylation in profilaggrin turnover.

Differentiated N/TERT-1 keratinocytes were stimulated with interferon-α. The effect on cell viability was assessed, and the induction of ISGylation-related genes was investigated by qPCR. The impact on ISG-15 and profilaggrin protein was evaluated by immunoblotting.

Interferon-α had no significant impact on the N/TERT-1 viability. Increased *ISG-15, UBE1L, UBCH8*, *HER5* and *HER6* expression indicated activation of the ISGylation pathway. ISG-15 protein expression peaked at 8h and profilaggrin accumulated in N/TERT-1 over the inspected period.

Interferon-α upregulated the expression levels of enzymes involved in ISGylation pathway, while ISG-15 was detected at gene and protein levels. This led to profilaggrin accumulation in differentiated keratinocyte. Our findings suggest a potential regulatory role of ISGylation process in the turnover of filaggrin.

14. The key player of the immune system that impacts all – gut microbiome

Jurand Sobiecki[1], Wiktoria Manowska[1], Julia Kwoczek[1], Oliwia Kosecka[1]

[1]University of Gdańsk, Institute of Psychology

The gut microbiome has emerged as a central regulator of immune system functioning, increasingly recognized for its profound impact on both physical and psychological health. Early-life exposures, such as breastfeeding and mode of birth, are critical for microbiota formation and subsequent immune development. Disruptions in this process have been linked to a higher risk of chronic inflammatory conditions and altered stress responses later in life. Furthermore, growing evidence suggests that psychotropic medications, while widely used, may impair immune function by modulating cytokine networks and lymphocyte profiles. Therefore, exploring alternatives, such as synbiotics, is vital. From an evolutionary perspective, immune system efficiency is closely tied to adaptive traits essential for survival, such as assertiveness and stress resilience. The gut-immune-brain axis represents a dynamic interface where microbial ecosystems have co-evolved with host defenses and behavior regulation. Understanding this complex relationship offers promising insights into the biological underpinnings of psychosocial functioning. Supported by initiatives under the Champions of Collaboration and SEA-EU frameworks, we aim to present an interdisciplinary line of research focused on the interplay between gut microbiota, immune regulation, and psychological traits. Integrating findings from immunology, microbiology, and psychology, our work highlights the microbiome as a key mediator in health outcomes and a potential target for novel interventions. This poster will outline the conceptual foundations and future directions for microbiota-informed strategies in enhancing immune competence and mental resilience, emphasizing the critical role of evolutionary processes in shaping these interactions.

15. Podocytes synthesize RNA of all IgG receptors, which level varies depending on glycemia

Marlena Typiak[1], Aleksandra Sobczak[1], Zofia Dobrzyńska[1], Moin A. Saleem[2], Anna Jaźwińska[3]

[1] Department of General and Medical Biochemistry, Faculty of Biology, University of Gdansk, Poland
[2] Bristol Renal, University of Bristol, Bristol, United Kingdom
[3] Regional Center of Blood Donation and Blood Treatment in Gdansk, Poland

Diabetes results from prolonged high blood glucose level, causing tissue inflammation with increased immune complexes' (ICs) formation and glomerular deposition. This results in diabetic kidney disease (DKD) in 30-40% of patients. Since, kidney glomeruli have very few leukocytes within, some glomerular cells (like podocytes) developed immune features, aiming to defend kidneys against metabolic imbalance. Podocytes perform phagocytosis of immunoglobulin G (IgG)-opsonized particles and are professional antigen presenting cells. Thus, we have deduced that they should produce receptors for Fc fragment of IgG (FcγR), which initiate phagocytosis of IgG-based ICs, and that FcγR synthesis could change in hyperglycemia. Therefore, human podocytes were cultured in standard (SG) or high (HG) glucose concentration. Human ovarian cell line was used as a negative control and leukocytes as a positive control of FcγR-coding genes' transcription. Real-time PCR was implemented to assess levels of *FCGR1A*, *FCGR2A*, *FCGR2B*, *FCGR2C*, *FCGR3A*, *FCGR3B*, *FCGRT*, *FCRL4*, *FCRL5* and *TRIM21* expression in relation to *ACTB*. We have shown that podocytes produce RNA for all the known FcγRs. Only *FCRL4* and *FCRL5* were expressed at a low level. *TRIM21* expression was higher in podocytes than leukocytes. Surprisingly, FcγR gene expression was present in ovarian cells, with lower expression of *FCGRT* and *TRIM21* than in podocytes. *FCGR1A* and *FCGR2C* were expressed significantly higher in podocytes grown in HG vs SG, *FCGR2A* showed a similar trend. *FCGR2B, FCGR3A* and *TRIM21* RNA synthesis was lower, whereas *FCGR3B* and *FCGRT* expression was indifferent in HG vs SG. The increased expression of *FCGR1A*, *FCGR2A* and *FCGR2C* in hyperglycemia with decreased production of *FCGR2B* RNA (coding for the only immune response-inhibiting FcγR), could translate into exaggerated IC phagocytosis and antigen presentation at the cell surface, leading to initiation or increase of renal tissue inflammation during DKD.

Funding: the University of Gdansk, 533-D000-GF79-23. Published in: Gene 958 (2025) 149501.

16. Investigating the neuroimmunomodulatory effects of psychedelics via 5-HT$_2$A and Sigma-1 receptors

Ying Yu[1], Aleksandra Rutkowska[1]

[1]Medical University of Gdańsk, Division of Anatomy and Neurobiology, M. Skłodowskiej-Curie 3A, 80-210 Gdańsk, Poland

Psychedelics—such as LSD, DMT, 5-MeO, MDMA, ketamine and psilocybin—are increasingly studied for their psychoactive properties, yet their direct cellular effects on neuroinflammation and neuroprotection remain poorly understood. These compounds are known to activate serotonin 5-HT$_2$A and Sigma-1 receptors, which play key roles in regulating neuronal survival, neuroplasticity and inflammatory signaling pathways. However, their potential involvement in promoting myelination or protecting against inflammation-induced demyelination has not been explored. Similarly, their effects on modulating blood–brain barrier (BBB) integrity and permeability under normal and inflammatory conditions have yet to be investigated.

This project aims to determine whether psychedelics can protect against demyelination and neuron damage by activating anti-inflammatory signaling via 5-HT$_2$A and Sigma-1 receptors. In parallel, we will assess their effects on BBB integrity and permeability under both normal and inflammatory conditions.

Organotypic cerebellar slices were subjected to chemical demyelination using lysophosphatidylcholine (LPC). Slices were co-treated with DMT, either alone or in combination with selective antagonists: ketanserin (5-HT$_2$A receptor antagonist) and BD-1063 (Sigma-1 receptor antagonist). The release of pro-inflammatory cytokines were measured using ELISAs. Gene expression changes were assessed by RT-qPCR, including markers for oligodendrocyte precursor cells (OPCs), oligodendrocytes and myelin, neurons, pro-inflammatory transcription factors, BBB components and the expression of 5-HT$_2$A and Sigma-1 receptors. Human tri-cell BBB model was treated with psychodelics with or without a cocktail of pro-inflammatory cytokines IL17/TNFα and the antagonists (ketanserin or BD). Gene expression changes were assessed by RT-qPCR, including pro-inflammatory transcription factors, BBB components and the expression of 5-HT$_2$A and Sigma-1 receptors.

Treatment with psychedelics inhibited pro-inflammatory signaling in our *ex viv*o and *in vitro* models. The anti-inflammatory effects were inhibited with receptor antagonists indicating 5-HT$_2$A and Sigma-1 receptors mediated effects.

This project and future findings may offer a cellular basis for exploring psychedelics as potential therapies in neuroimmune and neurodegenerative disorders. They may have important implications for neuroinflammatory conditions such as multiple sclerosis and dementias as well as for neurovascular disorders like stroke.

17. The pro-inflammatory status of monocytes in patients with systemic lupus erythematosus (SLE)

Katarzyna Zielińska[1], Michał Komorniczak[2], Barbara Bułło-Piontecka[2], Alicja Dębska-Ślizień[2], Katarzyna A. Lisowska[1], Anna Wardowska[1]

[1]Department of Pathophysiology, Faculty of Medicine, Medical University of Gdańsk, Gdańsk, Poland

[2]Department of Nephrology, Transplantology and Internal Medicine, Faculty of Medicine, Medical University of Gdańsk, Gdańsk, Poland

In SLE the excessive accumulation of apoptotic material activates innate and adaptive immunity, leading to chronic inflammation. Monocytes - the critical cells in orchestrating innate immune response, can be divided into three subpopulations: classical and intermediate (pro-inflammatory) and non-classical (anti-inflammatory). DAMPs recognized via TLRs activate monocytes, which respond in overproduction of pro-inflammatory cytokines. Therefore, assessed in this study parameters – percentages of monocyte subsets, expression of TLRs and cytokines could be used as markers of disease activity/proinflammatory status of monocytes.

Peripheral blood from 10 SLE patients from Department of Nephrology, Transplantology and Internal Diseases and 10 healthy donors was analyzed by flow cytometry to assess percentages of monocyte subpopulations, expression of TLR2 and TLR4 and by qPCR to measure the expression of genes associated with pro- and anti-inflammatory response (IL-1, IL-6, TNFα, IL-10, TGF-β).

Statistically significant increase of TGF-β expression - was observed in healthy control group compared to SLE patients. Moreover, the median values of TLR2 MFI on non-classical monocytes were detected in SLE patients with active disease compared to patients in remission state. Also the percentages of TLR expressing monocytes differ between patients and healthy individuals. Additionally, patients with active SLE were characterized with higher median expression of IL-6, IL-1, IL-10 compared to inactive disease.

Parameters associated with proinflammatory phenotype were slightly raised in the group of SLE patients, especially in patients with active disease. Only minor differences between SLE group and healthy control group might be related to the limited size of research group, applied immunosuppressive therapies or possible appearance of endotoxin tolerance in SLE patients permanently exposed to DAMPs. The study must be continued on extended group of patients, to draw more specific conclusions.

FarU Immunology Meeting

CONFERENCE PROGRAMME

7th June 2025

9:00	9:10	**Conference Opening –** Danuta Gutowska-Owsiak Welcome words: Katarzyna Zygmunt (FarU) and Wiesław Laskowski (UG)	
CLINICAL IMMUNOLOGY SESSION Chairs: Katarzyna Lisowska, Marek Niedoszytko			
9:10	9:35	Humoral immunodeficiency associated with chronic lymphocytic leukemia *Special guest lecture - PTIDiK*	Ewelina Grywalska (Medical University of Lublin)
9:35	9:55	Allergen Immunotherapy as the only causative treatment of allergy	Marek Niedoszytko (MUG)
9:55	10:20	Granulomatosis with polyangiitis from pathogenesis to clinical presentation and treatment	Anna Masiak (MUG)
10:20	10:40	Timing Complexity: Immunomodulation in cancer: challenges and opportunities	Jacek Rutkowski /Piotr Wysocki (MUG)
10:40	11:00	**Coffee Break**	
EXPERIMENTAL IMMUNOLOGY SESSION Chairs: Danuta Gutowska-Owsiak, Wojciech Siwek			
11:00	11:25	Bridging CNS remyelination and immune regulation through EBI2/oxysterol signalling	Aleksandra Rutkowska (MUG)
11:25	11:50	A small viral thief and a cellular VIP protein – how viruses hijack cellular pathways for immune subversion	Andrea Lipińska (UG)
11:50	12:10	Limitations of preclinical models and cancer heterogeneity – challenges for cancer immunotherapy	Natalia Marek-Trzonkowska, (UG)
12:10	12:30	CD73 (ecto-5'-nucleotidase) regulatory role in anticancer immune response – using murine models to research the mechanism and clinical response	Patrycja Koszałka (MUG)
12:30	12:50	**Sponsor Session - BD**	
12:50	13:50	**Lunch and Poster Session**	

KEYNOTE SPEAKER			
13:50	14:50	Lipid-reactive T cells and the skin: biology and therapeutic relevance	Graham Ogg (University of Oxford)
TRANSLATIONAL IMMUNOLOGY SESSION Chairs: Michał Pikuła, Ewa Bryl			
14:50	15:10	Mechanisms of interferon-gamma (IFN-γ) transcriptional memory	Wojciech Siwek (UG)
15:10	15:30	Anti-C4d antibody – a universal agent targeting tumor cells resistant to first-line immunotherapy	Marcin Okrój (MUG)
15:30	15:55	Metallothionein upregulation as a potential novel mechanism driving the induction of tolerogenic functionality in "flash DC-10" cells induced by a brief exposure to interleukin 10	Danuta Gutowska-Owsiak (UG)
15:55	16:20	Death receptor 3 as a potential costimulatory domain for CAR-T cells	Tomasz Ślebioda (MUG)
16:20	16:40	**Coffee Break**	
JOINT VENTURE SESSION Chairs: Weronika Hewelt-Belka, Tomasz Ślebioda			
16:40	17:00	Lipidomics as a tool in immunological research of extracellular vesicles – analytical perspectives	Weronika Hewelt-Belka (GUT)
17:00	17:25	Immunological safety of biomaterials and peptides for regenerative medicine	Michał Pikuła (MUG)
17:25	17:50	Radiation-induced dysregulation of hematopoietic differentiation: insights from CD34+ cells	Jakub Mieczkowski (MUG)
17:50	18:10	*In vitro* digestion models as tools to unravel immunological responses to food compounds	Ilona Kłosowska-Chomiczewska (GUT)
18:10	18:20	**Awards and Meeting Closure**	

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