XXX Gliwice Scientific Meetings



Gliwice, November 19-20, 2026

http://gsn.io.gliwice.pl/

Organizers:

Association for the Support of Cancer Research

Maria Skłodowska-Curie National Research Institute of Oncology

Silesian University of Technology

Co-organizer:

Polish Academy of Sciences

Patronage:

Director of the National Research Institute of Oncology

Rector of the Silesian University of Technology

Director of the National Research Institute of Oncology – Gliwice Branch

Mayor of the City of Gliwice

Narodowy Instytut Onkologii
im. Marii Skłodowskiej-Curie
Państwowy Instytut Badawczy

Gliwickie Spotkania Naukowe 2026 - zadanie dofinansowane przez Budżet Badań Naukowych w Narodowym Instytucie Onkologii im. Marii Skłodowskiej-Curie - PIB.
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THIRTY YEARS OF THE GLIWICE SCIENTIFIC MEETINGS

Dear Friends and Colleagues,

This year, we celebrate the 30th anniversary of the Gliwice Scientific Meetings. However, the history of this conference series goes back further. Its origins can be traced to many scientific meetings held irregularly during the 1960s, 1970s, and 1980s and organized by Prof. Mieczysław Chorąży and his team from the Department of Tumor Biology at the Institute of Oncology in Gliwice.

In 1997, Prof. Joanna Rzeszowska-Wolny and her team from the newly established Department of Experimental and Clinical Radiobiology at the Institute of Oncology decided to continue this tradition by establishing a regular forum for scientific exchange. From the very beginning, the organizational and financial foundation of these meetings was provided by the Association for the Support of Cancer Research, an organization established by researchers from the Institute of Oncology together with colleagues from the Silesian University of Technology.

Today, the Gliwice Scientific Meetings are jointly organized by the Association for the Support of Cancer Research, the Maria Skłodowska-Curie National Research Institute of Oncology and the Silesian University of Technology - the two major scientific institutions that have shaped the research landscape of Gliwice.

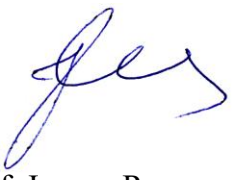
Over the years, the Gliwice Scientific Meetings have covered a broad spectrum of topics, encompassing different aspects of cancer research, molecular and cellular biology, medicine, biotechnology, and bioinformatics. During the past three decades, more than 550 distinguished lecturers from around the world have contributed to the scientific excellence of the conference. The meetings have brought together approximately 3,000 participants from more than 30 countries, who have presented nearly 2,100 posters.

We believe that, over these thirty years, the Gliwice Scientific Meetings have established a strong scientific reputation and have become a recognizable and valued event extending well beyond the Polish scientific community.

On the occasion of our 30th anniversary, we would like to express our warmest gratitude to all the participants, speakers, co-organizers, and sponsors whose commitment and support have made this journey possible.

And yet, this is not the end of the story. The story continues. We look forward to welcoming you to the future editions of the Gliwice Scientific Meetings and to writing the next chapters of this remarkable scientific journey together.

Yours sincerely,



Prof. Joanna Rzeszowska



Prof. Piotr Widłak

FROM ORIGINS TO THE PRESENT:
THE HISTORY OF THE GLIWICE SCIENTIFIC MEETINGS

The Gliwice Scientific Meetings originated as a series of scientific meetings organized irregularly by the Research Division of the Institute of Oncology in the 1960s, 1970s, 1980s, and 1990s. Thus, the history of the Scientific Meetings in Gliwice spans almost six decades, beginning in 1966.

Between 1966 and 1978, twelve Scientific Meetings, sometimes referred to as the "Autumn Scientific Meetings," were organized by Prof. Mieczysław Chorąży and his team from the Department of Tumor Biology. During this early period, various committees of the Polish Academy of Sciences, in which Prof. Chorąży was actively involved, participated in the organization of these scientific conferences in Gliwice. The Meetings were sometimes held in conjunction with other important events. For example, the (7th) Scientific Meeting, held in November 1972, was organized as part of the celebrations marking the 25th anniversary of the Institute of Oncology in Gliwice.



Scientific Meetings (in Gliwice) [1967]

It is worth emphasizing that, despite the constraints imposed by the "Iron Curtain" and the relative isolation of Polish researchers from the international scientific community, the scientific programme of these early Meetings consistently addressed some of the most topical issues in cancer research worldwide. A particularly striking example is provided by the invitation to the (11th) Scientific Meeting, held on 15 December 1977, which announced: "*We would like to devote the meeting to the issue of genome research using 'genetic engineering' methods. In particular, we would like to discuss the current state of research on isolating and cloning specific DNA sequences of eukaryotic cells, summarizing information about restriction enzymes and plasmids used in prokaryotic and eukaryotic DNA cloning experiments."* This topic illustrates how closely the scientific community in Gliwice followed the rapidly developing fields of molecular biology and genetic engineering, even when access to international scientific literature, reagents, technologies, and direct scientific exchange was severely limited.

After a ten-year gap, largely attributable to the political circumstances in Poland, including the period of martial law, an attempt was made to revive the tradition of scientific meetings in Gliwice. On 4–5 May 1989, a conference was organized by Dr Joanna Rzeszowska and Docent Zdzisław Krawczyk. Its main themes were "DNA - Research Methods and Organization in the Cell Nucleus" and "Gene Expression in Specific Cell States." In the following years, the Department of Tumor/Molecular Biology continued to organize scientific conferences, although the term "Scientific Meetings" was no longer used. One of the most notable events of this period was the workshop "Assessment of Cancer Hazard in Silesia," held on 11-13 June 1992. The workshop was sponsored by the National Cancer Institute (NCI), Bethesda, USA, and the Polish Ministry of Environmental Protection. It aimed to bring together and summarize the results of research conducted at the Environmental Mutagenesis Laboratory by the team led by Prof. Mieczysław Chorąży, including Dr. Ewa Grzybowska, Dr. Grażyna Motykiewicz, and Dr. Jan Szeliga. The research was carried out in close collaboration with research teams from the United States and Sweden, led by Prof. Frederica Perera (Columbia University, NY) and Prof. Kari Hemminki (Karolinska Institutet, Stockholm). In her conference summary report prepared for the NCI, Dr Ansley Weston concluded: "*In conclusion, the dramatic pollution of air, water and soil as a result of industrial processes in Poland in Upper Silesia has been identified and well documented. This pollution seems to pose a serious threat to human health and a cancer risk in Silesia.*" The findings attracted considerable international attention and were widely disseminated in the scientific literature, including a landmark paper published in Nature (Perera et al., Molecular and genetic damage in humans caused by environmental pollution in Poland; Nature, 1992, 360, 256–258). Another memorable scientific event took place on 13-14 October 1995, organized by the Department of Molecular Biology to celebrate the 70th birthday of Prof. Mieczysław Chorąży. Entitled "Progress in Tumor Biology," the conference brought together numerous distinguished scientists, as well as Prof. Chorąży's friends and long-standing collaborators. Among the distinguished guests were Prof. Hilary Koprowski, renowned for developing the world's first effective live polio vaccine, and Prof. Robert Gallo, one of the co-discoverers of HIV.



Workshop "Assessment of Cancer Hazard in Silesia"



Conference "Progress in Tumor Biology" [1995]

The final and ultimately successful attempt to revive the tradition of the Scientific Meetings in Gliwice came in 1996, initiated by Docent Joanna Rzeszowska, then Head of the Department of Experimental and Clinical Radiobiology at the Institute of Oncology, and her team. Following a small scientific meeting held in Srebrna Góra, they recognized the need to establish a more permanent organizational framework for continuing this initiative. This led to the establishment of the Association for the Support of Cancer Research, which provided a stable organizational platform for the future meetings. One of the Association's principal statutory objectives was the organization of an annual scientific event known as the "**Gliwice Scientific Meetings**." Over the years, the Association has brought together current and former researchers from the Institute of Oncology and the Silesian University of Technology (SUT), creating a community that has supported and sustained the Meetings across generations of scientists. The Association has played a crucial role in ensuring the continuity of this initiative. In 2026, the Gliwice Scientific Meetings will celebrate their 30th edition in this new organizational format, marking three decades of an enduring scientific tradition that has its roots in the earlier meetings held in Gliwice.



Scientific Meetings (in Srebrna Góra) [1996]

The first meeting to officially bear the name "**Gliwickie Spotkania Naukowe**" was held on November 21-22, 1997 (since then, the penultimate week of November has traditionally been reserved for the Meetings). Its scientific programme devoted to DNA-protein interactions included 20 lectures and 5 posters. The only international guest was Dr. Valentin Schick from Moscow, who, in his lecture "Analysis of DNA and Protein Microchips", presented what was then a pioneering microarray-based technology. The second Meetings, held in 1998, were organized in conjunction with the celebrations marking the 50th anniversary of the Institute of Oncology in Gliwice. The scientific programme comprised four sessions: "Mathematical Models in Biology" "Chromatin Structure and Gene Expression", "Genetic Polymorphism and Cancer," and "DNA Damage, Cell Repair and Death." In total, 18 lectures were delivered, including four by international speakers: Prof. Wolfgang Henning, Prof. Wolf H. Strätling, Prof. Jan Filipski, and Prof. Régen Drouin. From the 5th Edition (year 2001) onward, the conference became fully international, with English adopted as its official language. This marked an important new chapter in the history of the Gliwice Scientific Meetings, establishing the international character that has remained one of their defining features ever since.



IX Gliwice Scientific Meetings [2005]

In the subsequent years, close collaboration with the team of Prof. Andrzej Świerniak at the Institute of Automatic Control (SUT), where Prof. Joanna Rzeszowska moved in 2010, further strengthened the role of the Silesian University of Technology, a vibrant academic institution based in Gliwice, as a co-organizer of the Gliwice Scientific Meetings. Starting with the 10th edition in 2006, the Meetings have been held primarily using the conference facilities of SUT. Today, the main venue is the modern Conference and Education Center of the SUT. Since 2005, the Gliwice Scientific Meetings have been officially co-organized by three partners: the Association for the Support of Cancer Research, the Maria Skłodowska-Curie Institute of Oncology in Gliwice (Basic Research Division), and the Silesian University of Technology (Institute of Automatic Control and then Biotechnology Centre). This long-standing partnership has provided a strong institutional foundation for the continued development and international growth of the Meetings.

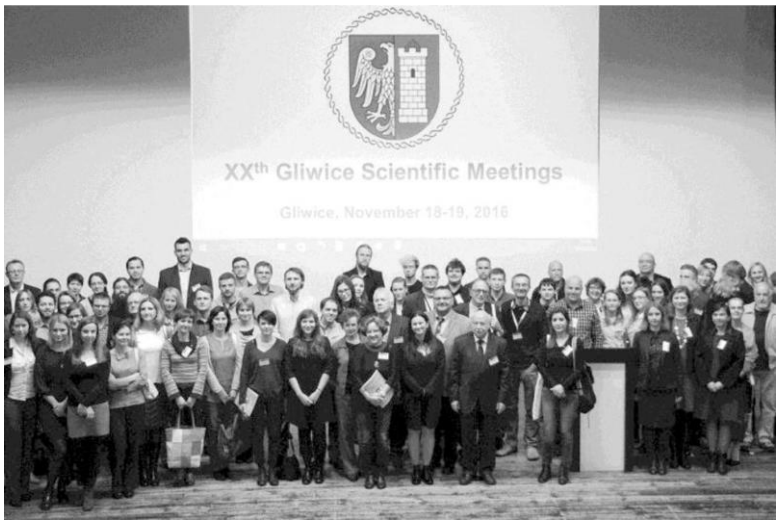
In the following years, several editions of the Meetings were organized to commemorate important milestones or in conjunction with other major scientific events. The 9th Edition (2005) provided an opportunity to celebrate the 80th birthday of Prof. Mieczysław Chorąży. The 13th Edition (2009) was organized jointly with the 2nd Polish-German Cancer Workshop, with the German Cancer Research Center (DKFZ) as a partner. A satellite event accompanying the 15th edition (2011) was the Polish-German-American Workshop on

Progress in Neuro-Oncology, co-sponsored by the DKFZ and MD Anderson Cancer Center.



X Gliwice Scientific Meetings [2006]

During the COVID-19 pandemic, the Meetings adapted to the unprecedented circumstances by moving online for their 24th Edition in 2020, which was held entirely virtually, and adopting a hybrid format for the Jubilee 25th Edition in 2021. Sadly, the latter event was also the first to take place without the presence of Prof. Mieczysław Chorąży, our long-standing Mentor and eminent Scientist, who passed away on 21 February 2021, at the age of 95. In his memory, the Conference Organizers decided that the opening lecture of the Gliwice Scientific Meetings would henceforth be designated the "Professor Mieczysław Chorąży Memory Lecture." The inaugural lecture, delivered in 2021, was given by Prof. Kari Hemminki. In the same year, the Association for the Support of Cancer Research established and funded the Professor Mieczysław Chorąży Scholarship for young scientists. The Jubilee 25th Edition was also accompanied by a satellite workshop entitled "Proteomics of Extracellular Vesicles," organized jointly by the Polish and Finnish Proteomics Societies. The 27th edition, held in 2023, was dedicated to the 70th anniversary of the discovery of the DNA double helix. The Professor Mieczysław Chorąży Memory Lecture 2023 was delivered by Dr. Victor Ambros, who was subsequently awarded the Nobel Prize in Physiology or Medicine in 2024 for his discovery of microRNA and its role in post-transcriptional gene regulation.



XX Gliwice Scientific Meetings [2016]



Major organizers of GSM: Prof. Joanna Rzeszowska and Prof. Piotr Widłak

Over the 30-year history of the Gliwice Scientific Meetings, the scientific sessions have covered a remarkably broad spectrum of topics, consistently addressing some of the most important and enduring questions in biomedical research. These have included chromatin structure and the regulation of gene expression, DNA damage and repair, cellular responses to ionizing radiation and other stress factors, as well as neurobiology and neuro-oncology. The Meetings have also provided a forum for advances in genomics, proteomics and metabolomics. From the very beginning, mathematical modelling of biological processes, computational biology and biostatistics were also among the important scientific areas covered by the Meetings. Their inclusion from the earliest editions reflected the forward-looking character of the conference and its openness to emerging interdisciplinary approaches at the interface of biology, medicine, mathematics and computational science. In parallel, the scientific programme has encompassed a wide range of topics related to environmental and medical biotechnology, the discovery and characterization of novel active compounds, and the development of innovative biomaterials. Throughout the years, the Meetings have thus reflected the dynamic evolution of biomedical science while maintaining a strong focus on fundamental and translational questions of lasting importance. During the past three decades, more than 550 distinguished lecturers from around the world have contributed to the scientific excellence of the conference. The meetings have brought together approximately 3,000 participants from more than 30 countries, who have presented nearly 2,100 posters. Since their inception in 1997, the Gliwice Scientific Meetings have established a strong scientific reputation and have become a well-established and highly regarded scientific event, attracting interest and participation well beyond the Polish scientific community.

GLIWICE SCIENTIFIC MEETINGS – KEY MILESTONES

1966 – The first "Scientific Meetings" were held at the Institute of Oncology in Gliwice, initiated by Prof. Mieczysław Chorąży and organized by the staff of the Department of Tumor Biology.

1966–1978 – "Autumn Scientific Meetings" were organized regularly, with a total of 12 meetings held during this period, except in 1970 and 1975.

1989 – Scientific Meetings were re-established after a decade-long break, on the initiative of Dr. Joanna Rzeszowska and Doc. Zdzisław Krawczyk.

1992 – The workshop "Assessment of Cancer Hazard in Silesia" was organized by the Department of Molecular Biology with the co-sponsorship of the National Cancer Institute (NCI), NIH, USA.

1995 – The scientific conference "Progress in Tumor Biology" was held in conjunction with the celebration of the 70th birthday of Prof. Mieczysław Chorąży.

1996 – The Association for the Support of Cancer Research (ASCR) was established.

1997 – The 1st Gliwice Scientific Meetings were organized by Prof. Joanna Rzeszowska and her team from the Department of Experimental and Clinical Radiobiology, with the support of the Association for the Support of Cancer Research, marking the beginning of the current series of Gliwice Scientific Meetings.

1998 – The 2nd Gliwice Scientific Meetings were organized together with celebrations marking the 50th anniversary of the Institute of Oncology in Gliwice.

2005 – The 9th Gliwice Scientific Meetings were held, with the celebration of the 80th birthday of Prof. Mieczysław Chorąży as a special highlight of the event.

2011 – The 15th Gliwice Scientific Meetings were held, with the Center for Translational Research and Molecular Biology of Cancer, headed by Prof. Piotr Widłak, taking on the leading role in organizing the Meetings on behalf of the Institute of Oncology.

2021 – The 25th Jubilee Gliwice Scientific Meetings were held. The Professor Mieczysław Chorąży Memory Lecture was introduced and delivered for the first time, and the Professor Mieczysław Chorąży Scholarship for Young Scientists was awarded for the first time.

2026 – The 30th Jubilee Gliwice Scientific Meetings are held to celebrate the history and scientific achievements of the former Basic Research Division of the Institute of Oncology in Gliwice, marking the end of an important chapter in its scientific history.

XXX Gliwice Scientific Meetings

Scientific Program

Thursday, 19th November 2026

08:00 – 09:00	Poster Session of the Student Scientific Society of the Silesian University of Technology
09:00 – 09:15	Opening of the Conference
09:15 – 10:00	***Prof. Mieczysław Chorąży Memory Lecture 2026***: **Jan Barciszewski** *(Institute of Bioorganic Chemistry, Poznań):* From Gliwice to the World: the great world of DNA. Mieczysław Chorąży and the breakthroughs of molecular biology.
10:00 – 10:45	**Joanna Rzeszowska** *(Silesian University of Technology, Gliwice)*: The origins of scientific discoveries: biology in Gliwice.
10:45 – 11:15	Coffee Break
11:15 – 13:00	Session "***Heat Shock and cellular response to stress***" (*Chairpersons*: Wiesława Widłak & Dorota Ścieglińska) **Dorota Ścieglińska** (*IGBZ, Jastrzebiec*): Tribute to Professor Zdzisław Krawczyk and Heat Shock Proteins. **Anniina Vihervaara** (*Royal Institute of Technology, Solna*): Kinetics and Mechanisms of Transcriptional Reprogramming during Stress and Differentiation. **Maxim Shevtsov** (*Technical University of Munich*): The Epichaperone Matrix: A Multiscale Network Organizing Tumor Cell Stress Responses. **Patryk Janus** (*MSCI, Gliwice*): Beyond the heat shock response: non-canonical roles of HSF1 in cell survival and hormonal signaling.
13:00 – 14:00	Lunch
14:00 – 16:00	**Poster Session** (Part I – odd numbers and Part II – even numbers) and Coffee
16:00 – 18:00	Session "***Genetic predispositions to cancer and targeted treatment***" (*Chairpersons*: Marek Rusin & Dorota Butkiewicz) **Marek Rusin** (*MSCI, Gliwice*): Genetic predispositions to cancer and targeted treatment – historical intro.

Asta Försti (*DKFZ, Heidelberg*): Germline genetics of colorectal cancer

Wojciech Fendler (*Medical University of Łódź*): Expression patterns of circulating miRNAs associated with DNA repair defects.

Jan Lubiński (*Pomeranian Medical University, Szczecin*): Selenium and arsenic in relation to cancer risk among BRCA1 pathogenic variant carriers

Magdalena Olbryt (*MSCI, Gliwice*): Targeted genomic profiling of melanoma using liquid biopsy.

19:00 – Social event (*Kopalnia Guido, Hala Pomp, poziom 320*)

Concert of the ***Autentikos*** ensemble

Friday, November 20th, 2026

08:00 – 09:00 Poster Session of the Student Scientific Society of the Silesian University of Technology

09:00 – 10:40 Session "***Radiobiology and DNA damage/repair***"

(*Chairpersons*: Dorota Gabryś & Piotr Widłak)

Piotr Widłak (*MUG, Gdańsk*) – historical intro

Christophe Badie *(UKHSA, Oxfordshire)*: From DNA damage to leukaemia development.

Serge Candeias (*CEA, Grenoble*): Influence of radiation parameters on radiation-induced inflammation in different primary human cell lineages.

Dorota Gabryś (*MSCI, Gliwice*): Radiotherapy-induced modulation of the urinary microbiome in patients with prostate cancer.

10:40 – 11:00 Coffee Break

11:00 – 12:45 Session "***Clinical proteomics***"

(*Chairpersons*: Monika Pietrowska & Piotr Widłak)

Piotr Widłak (*MUG, Gdańsk*): Clinical Proteomics – Gliwice Perspective

Maciej Łałowski (*Univ. of Helsinki*): Analyzing the dynamics of microglia during development in health and disease: a functional omics approach.

Laura Cole (*Sheffield Hallam University*): Multi-Modal Mass Spectrometry Imaging to Investigate Drug-Induced Response.

Janusz Rak (*McGill University, Montreal*): From simplicity of oncogenic mutations to complexity of extracellular vesicle landscapes and their biological effects in cancer.

12:45 – 13:00 Coffee Break

13:00 – 14:45 Session "***Mathematical modelling of biological processes***"

(*Chairpersons*: Andrzej Świerniak & Marek Kimmel)

Marek Kimmel (*Rice University, Houston*): Houston to Gliwice and back.

Tomasz Lipniacki (*IPPT, Warszawa*): Dissecting antiviral immune response at the population and single-cell level

Adam Bobrowski (*Lublin University of Technology*): Existence and convergence of stochastic processes underlying a thin layer approximation of a coupled bulk-surface PDE

Marie Doumic (*INRIA, Paris*): Mathematical modelling of telomere dynamics

14:45 – 15:30 Lunch

15:30 – 17:15 Session "***New bioactive compounds and biomaterials***"

(*Chairpersons*: Magdalena Skonieczna & Anna Kasprzycka)

Radosław Kitel (*Jagiellonian University, Kraków*): Discovery and in vitro characterization of CLIP-2, a first-in-class small-molecule covalent inhibitor of PD-L1/PD-1 interaction.

Katarzyna Krukiewicz (*SUT, Gliwice*): Development of multifunctional biomaterials via electroactive surface engineering to modulate cell–material interactions.

Kristýna Valášková *(Tomas Bata University, Zlín)*: Cell-made scaffolds in tissue engineering: from controlled matrix assembly to mechanistically guided alignment.

Seweryn Gałecki *(SUT, Gliwice; UTSW, Dallas):* Seeing the unseen: expanding the boundaries of cellular imaging with Cy-ExM

17:15 – 17:45 **Poster Awards**, Scholarship Presentation, Closing

PROF. MIECZYSŁAW CHORĄŻY MEMORY LECTURE

To honor and commemorate Professor Chorąży, since 2021, the Gliwice Scientific Meetings conference has started with an *in-memoriam* keynote lecture.

Previous speakers:

2021 **Kari Hemminki** (*Charles University, Pilsen & DKFZ, Heidelberg*): Science and society.

2022 **Philip Maini** (*Wolfson Centre for Mathematical Biology, Oxford*): Can mathematical modelling help us understand cancer growth and progression?

2023 **Victor Ambros** (*University of Massachusetts, Worcester*) (Nobel Prize winner 2024): Genetic regulatory networks involving noncoding RNA.

2024 **Theresa L. Whiteside** (*University of Pittsburgh Medical Center, PA, USA*): Exosomes: the ubiquitous intracellular communication system in human health and disease.

2025 **Marc Henrik Baumann** (*University of Helsinki, Biomedicum, Helsinki*): All you need to know about Alzheimer's disease.

PROF. MIECZYSŁAW CHORĄŻY SCHOLARSHIP FOR YOUNG SCIENTISTS

Professor Mieczysław Chorąży was an eminent Man, an outstanding scientist, a soldier of the Warsaw Uprising, moral authority for generations of fellow researchers, and an exceptionally warm person.

To commemorate Him, the Association for the Support of Cancer Research has funded a scholarship for young researchers who seem to follow his path.

Previous Awardees:

2021 **Alexander Cortez**, PhD (*Maria Skłodowska-Curie National Research Institute of Oncology, Gliwice*)

2022 **Paulina Marona**, PhD *(Jagiellonian University, Kraków)*

2023 **Anna Sobiepanek**, PhD Eng. *(Warsaw University of Technology, Warsaw)*

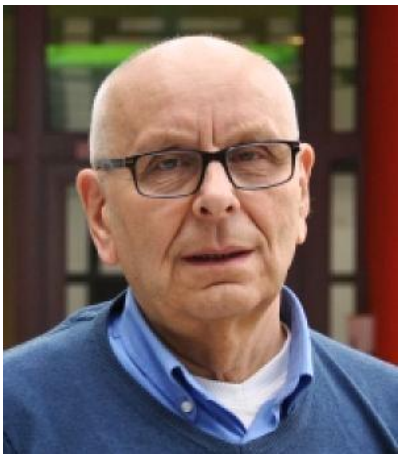
2024 **Marcelina Jureczko**, PhD Eng. *(Silesian University of Technology, Gliwice)*

2025 **Wojciech Szlasa**, MD (*Wrocław Medical University, Wrocław*)

Prof. Mieczysław Chorąży Memory Lecture 2026:

Prof. Jan Barciszewski, Ph.D.

Institute of Bioorganic Chemistry, Polish Academy of Sciences, Poznań, Poland



Prof. Jan Barciszewski is a distinguished Polish molecular biologist and biochemist at the Institute of Bioorganic Chemistry of the Polish Academy of Sciences (IBCH PAS) in Poznań, where he has been conducting research since 1973. He received his PhD in 1974, habilitation in 1985, and the title of Professor of Biological Sciences in 1993.

His research interests encompass the structure and function of nucleic acids, DNA and RNA biology, epigenetic regulation, and molecular mechanisms of disease. Prof. Barciszewski has published extensively in the field of molecular biology and bioorganic chemistry and has contributed significantly to the development of these disciplines in Poland.

He has authored and co-authored approximately 250 publications. Prof. Barciszewski is also an editor of the Springer book series RNA Technologies, which he has co-edited since its inception. The series comprises 15 volumes, covering various aspects of RNA structure, function, biology, and applications.

He is a member of the Polish Biochemical Society and has served on its governing board. He is also a member of the Committee of Molecular and Cell Biology of the Polish Academy of Sciences and was previously a member of the Committee of Biochemistry and Biophysics of the Polish Academy of Sciences.

Lecture Abstracts

Prof. Mieczysław Chorąży Memory Lecture

FROM GLIWICE TO THE WORLD: THE GREAT WORLD OF DNA. MIECZYSŁAW CHORĄŻY AND THE BREAKTHROUGHS OF MOLECULAR BIOLOGY

Jan Barciszewski

Institute of Bioorganic Chemistry, Polish Academy of Sciences, Poznań, Poland

This year marks the fifth anniversary of the death of Professor Mieczysław Chorąży. To some of us, he was a scientific mentor and doctoral supervisor; to all, he was an outstanding scientist of international renown, a visionary, and an exemplary role person. This naturally raises specific questions: what legacy did he leave behind, and for what should he be appreciated and remembered? We must acknowledge his achievements, his erudition, and the ease with which he navigated the realms of molecular and medical biology. To fully appreciate that, one must view his scientific body of work against the backdrop of the pivotal events that shaped the emergence and development of molecular biology. One might ask a simple question: what was on Mieczysław Chorąży's mind, what was he thinking about, when he arrived in Gliwice in the early 1950s as a young medical doctor. What did he know about cancer, DNA, genes, proteins, or the impact of the environment on humans? To understand M. Chorąży's scientific career, it can be divided into three periods and viewed against the backdrop of key developments in molecular biology such as the discovery of the DNA structure and the genetic code, the regulation of gene expression, and advances in chromatin research and molecular epidemiology. The first period spanned the years 1951–1959, during which he worked on his doctoral thesis. In the second period, lasting until 1963, M. Chorąży set out to acquire comprehensive knowledge and information regarding the fundamentals of molecular biology, particularly nucleic acids. He also prepared his habitation thesis. In the third period, extending from 1963 to 2021, he conducted independent studies at the Oncology Center in Gliwice.

Looking at that, one can conclude that Mieczysław Chorąży was not merely a passive recipient of global discoveries in molecular biology. He was able to translate them into new research questions concerning cancer, genome organization, and environmentally induced DNA damage. Three directions can be distinguished in his research:1) from cancer as a metabolic disorder to cancer as a disease of the cell; 2) from DNA as a chemical compound to DNA as a regulated genome; and 3) from molecular cancer research to the molecular assessment of environmental risks in humans.

When M. Chorąży began working at the State Cancer Institute in Gliwice in 1951, molecular biology was just emerging and taking its first steps. The double-helix model of DNA structure was proposed in 1953. In 1956, Francis Crick formulated the Central Dogma of Molecular Biology regarding the flow of information between nucleic acids and proteins, and in 1958, Malcolm Hoagland and Paul Zamecnik discovered tRNA and aminoacyl-tRNA synthetases. During that period, Mieczysław Chorąży conducted research on cancer cell metabolism, experimental chemotherapy, and the cellular effects of reactive oxygen species. This highly productive period was marked by the publication of three papers in Nature concerning experimental cancer therapies using hydrogen peroxide and chlorpromazine. The discovery of the DNA double helix structure induced a unique opportunity to ask a new question: did the fundamental abnormalities of cancer cells lie not only in metabolism but also in the genetic

material?. In M. Chorąży's view, the year 1953 was significant because that year received the double helix structure, and cancer, in turn, received a new mechanism for its characterization.

The years 1959–1963 marked the most significant period in M. Chorąży's scientific career. At the University of Wisconsin, he found an environment where DNA and RNA were ceasing to be merely objects of biochemical study and were instead becoming the language of biological information, and the relationship between base sequence and biological function could be experimentally investigated. In 1961, Marshall Nirenberg and Heinrich Matthaei demonstrated that poly-U directs the synthesis of polyphenylalanine and proved to be the first clearly defined link established between an RNA sequence and an amino acid. During this same period, M. Chorąży was conducting research at the Sloan-Kettering Institute in New York on metaphase chromosomes, their properties outside the cell, their uptake by mammalian cells, and the organization of genetic material within cancer cells. His publications from 1962–1963 on chromosome isolation and uptake demonstrated a clear shift from studying anti-cancer agents to investigating the genomic organization of cancer cells.

Upon returning to Gliwice from the USA in 1963, M. Chorąży established his own research program focused on the physical and functional organization of the genome. He focused on the interactions between DNA and acridine derivatives, the analysis of repetitive and inverted DNA sequences and the transcription of repetitive genomic fragments, as well as the study of DNA-chromatin protein complexes. M. Chorąży's research sought to answer a question left unaddressed by the central dogma: why is the same DNA information utilized differently depending on the cell's state?

M. Chorąży not only kept pace with global trends but also knew how to leverage them to address a local Silesian problem of global significance. This new concept emerged that chemical compounds cause cancer by modifying DNA and forming DNA adducts, inducing mutations, damaging chromosomes, and disrupting DNA repair, processes that ultimately lead to genomic instability. Advances in methods for labelling DNA adducts made it possible to measure airborne pollutant concentrations and assess their biological effects on the human body. To this end, M. Chorąży drew upon developments in molecular DNA biology, cytogenetic methods, DNA adduct research, mutagenicity testing, and molecular epidemiology, while also fostering international collaboration. Thanks to these efforts, environmental pollution in Upper Silesia was no longer characterized merely as a chemical or statistical issue. It was demonstrated as measurable damage to the genetic material of the local population. This work culminated in an article published in Nature in 1992, documenting molecular and genetic damage in individuals exposed to environmental pollution in Poland. M. Chorąży succeeded in linking four elements: the environment, chemical exposure, DNA damage, and cancer risk. This served as a model system for translational research, predating both the widespread adoption of the term "translational" and the establishment of a dedicated Center for Translational Research within the Institute of Oncology. M. Chorąży's greatest achievement lay not merely in applying modern methods to study pollution in Silesia, but in reframing an environmental issue as a problem of human molecular biology.

It must be noted that although M. Chorąży came to believe that DNA held the key to unlocking the mystery of cancer, DNA cannot be understood in isolation from the cell, the organism, or the environment. His career path took him from experimental cancer therapy—through research on chromosomes, DNA, and chromatin—to molecular environmental epidemiology. He demonstrated how global ideas could be creatively developed in Gliwice, and how a local issue facing Upper Silesia could evolve into a question of universal importance for biology and medicine.

Session I

„Heat Shock and cellular response to stress”

[L-I-1] TRIBUTE TO PROFESSOR ZDZISŁAW KRAWCZYK AND HEAT SHOCK PROTEINS

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Scientific discoveries do not end with the publication of an article. The most influential ones open new questions and set research directions that remain relevant as concepts and technologies evolve.

For the scientific community in Gliwice, Professor Zdzisław Krawczyk played a pioneering role by initiating research on heat shock genes in the 1980s. His work on the HSP70 gene family provided an early foundation for studying the regulatory mechanisms and function of these genes in cellular physiology and pathophysiology.

The significance of Professor Krawczyk's early achievements is particularly impressive when considering the context in which they were made. In Poland, in the late communist period, access to modern molecular biology technologies, reagents, scientific literature and opportunities for international cooperation was severely limited. Nevertheless, the research conducted in Gliwice addressed innovative topics within the rapidly developing field of molecular biology.

Professor Krawczyk's scientific vision laid the foundation for research programs that were expanded by his successors in the decades that followed. Katarzyna Lisowska contributed to the identification and molecular characterization of HSP70 genes; Wiesława Widłak investigated the regulation of heat shock gene expression and the role of heat shock factors (HSF); and Dorota Ścieglińska expanded research on the HSPA2 protein, analyzing its expression, regulation, and functions in both normal and cancer cells. Their work not only introduced new questions, technologies, and perspectives, but also reflects a genuine scientific curiosity regarding how cells utilize the heat shock response and heat shock proteins.

This lecture is a tribute to Professor Zdzisław Krawczyk, his scientific vision and his ability to inspire others to take on research challenges.

[L-I-2] KINETICS AND MECHANISMS OF TRANSCRIPTIONAL REPROGRAMMING DURING STRESS AND DIFFERENTIATION

Anniina Vihervaara

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Transcription drives differentiation and coordinates cellular responses. While mRNA expression in distinct cell types is well-characterized, the mechanisms that orchestrate RNA synthesis across genes and enhancers remain unclear. Here, I discuss our recent work that tracks transcription at nucleotide resolution and uncovers mechanisms that prime and time transcriptional reprogramming upon acute stress and differentiation. In human erythroid cells, a brief differentiation signal triggers slowly proceeding waves of transcription that propagate over cell divisions, and cause sustained changes in RNA synthesis, mRNA expression, and cell identity. The slow and sustained reprogramming during differentiation is primarily coordinated via Pol II initiation, and is profoundly distinct from stress responses that trigger instant and transient responses via Pol II pause-release. In differentiating late erythroid cells, NRF2, the potent trans-activator in oxidative stress, is stabilized without a detectable increase in reactive oxygen species. Consequently, the differentiating erythroid cells use the potent stress-induced trans-activator to establish tolerance for future, cell-type-specific stress occurring in the red blood cells. These results uncover drastically different strategies to coordinate transcriptional reprogramming upon acute stress and differentiation, and find lineage-specific and stress-induced factors to co-orchestrate erythroid differentiation, producing globin and antioxidant proteins before the cells enucleate to oxygen transporting erythrocytes.

[L-I-3] THE EPICHAPEROME MATRIX: A MULTISCALE NETWORK ORGANIZING TUMOR CELL STRESS RESPONSES

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Epichaperomes are stable, stress-induced supramolecular assemblies that integrate molecular chaperones, co-chaperones, signaling proteins, and client proteins. Although traditionally considered regulators of proteostasis, emerging evidence suggests that epichaperomes may also function as higher-order organizational structures that coordinate signaling and cellular adaptation in cancer. Here, we combine experimental characterization of plasma membrane-bound epichaperomes with a systems-level theoretical framework to propose the Epichaperome Matrix as a dynamic organizational network linking molecular interactions, membrane signaling, cellular motility, and stress adaptation.

Proteomic and network analyses identified a plasma membrane-bound epichaperome organized by membrane-associated Hsp70 (mHsp70) and enriched in chaperones, lipid-raft components, and regulators of cytoskeletal and RhoGTPase signaling. Glioblastoma-derived networks displayed increased connectivity, centrality, and modular organization, with enrichment of pathways associated with invasion and radioresistance. Functionally, isolated membrane epichaperome networks transferred signaling activity to recipient cells and enhanced migration and invasion through Rac1- and CDC42-dependent mechanisms. Pharmacological disruption of Hsp70 reduced network integrity and abolished the motility-promoting activity, whereas membrane lipid remodeling reprogrammed the network toward a tumor-suppressive phenotype and improved response to radiotherapy.

Building on these findings, the Epichaperome Matrix Theory conceptualizes epichaperomes as dynamic, nonequilibrium biomolecular matrices whose network connectivity, conformational dynamics, electrostatic properties, and membrane interactions may regulate molecular organization and transport. The model further proposes that intracellular, membrane-associated, and extracellular epichaperome assemblies could form functionally coupled networks across cellular and tissue scales. This framework generates experimentally testable predictions concerning network percolation, electrostatic organization, intercellular communication, and stress-dependent remodeling.

Together, these findings position the epichaperome not simply as a chaperone-associated protein complex, but as a potential multiscale organizational matrix that integrates proteostasis, membrane signaling, cytoskeletal regulation, invasion, and therapeutic resistance. Targeting or reprogramming this matrix may provide a new strategy for disrupting tumor adaptation and improving cancer therapy.

[L-I-4] BEYOND THE HEAT SHOCK RESPONSE: NON-CANONICAL ROLES OF HSF1 IN CELL SURVIVAL AND HORMONAL SIGNALING

Patryk Janus, Natalia Vydra, Agnieszka Toma-Jonik, Kinga Sarkowicz, Monika Bar, Wiesława Widłak

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Heat Shock Factor 1 (HSF1) is a key transcriptional regulator of the cellular response to proteotoxic stress. Its canonical function involves activating genes encoding heat shock proteins (HSPs), which maintain protein homeostasis and protect cells against stress-induced damage. However, accumulating evidence indicates that the biological activities of HSF1 extend beyond its well-established cytoprotective role, including the regulation of cell death, hormonal signaling, chromatin organization, and inflammatory and immune responses.

The dual role of HSF1 in the cellular response to heat stress, encompassing both cytoprotective and pro-apoptotic activities, has been recognized for decades. Our research has elucidated the molecular mechanism underlying the pro-apoptotic function of HSF1 in heat-sensitive cells. We demonstrated that activated HSF1 binds to regulatory regions of the PMAIP1 gene, which encodes the pro-apoptotic protein NOXA, thereby promoting its expression and triggering apoptosis. This mechanism operates independently of p53 and has been observed in murine and human cell models.

Another major focus of our research has been the non-canonical function of HSF1 in estrogen receptor α (ERα)-positive breast cancer cells. We demonstrated that, although HSF1 contributes to the activation of pro-inflammatory genes following heat shock, it also limits their excessive induction. Furthermore, we showed that HSF1 can be activated by 17β-estradiol through the MAPK signaling pathway, independently of proteotoxic stress. In this context, HSF1 regulates genes involved in cancer cell proliferation and function. Further investigations revealed a functional interplay between HSF1 and ERα in estrogen-dependent transcription. We also demonstrated that HSF1 influences ERα binding to chromatin and contributes to its three-dimensional organization, including the formation of chromatin loops associated with gene expression regulation, potentially involving interactions with the architectural protein CTCF. Consequently, HSF1 deficiency impairs cellular responses to estrogen and may enhance sensitivity to endocrine therapy.

Together, these findings establish HSF1 as a multifunctional regulator whose biological activities depend on the cellular context and the nature of the activating stimulus. Understanding the non-canonical functions of HSF1 provides new insights into the molecular mechanisms controlling cell fate, inflammatory responses, and hormone-dependent gene regulation in cancer. These findings may also inform the development of novel therapeutic strategies to modulate tumor responses to anticancer treatments.

Session II

„Genetic predispositions to cancer and targeted treatment ”

[L-II-1] GENETIC PREDISPOSITIONS TO CANCER AND TARGETED TREATMENT – HISTORICAL INTRO

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Familial clustering of cancers was noticed by clinicians in the late 19th century. In the sixties and early seventies of the 20th century, several familial cancer syndromes were discovered and partially characterized, e.g., Lynch syndrome (hereditary nonpolyposis colorectal cancer), Li-Fraumeni syndrome, and familial clustering of breast and ovarian cancers. However, the genetic mutations underlying these familial cancer syndromes were not known due to rudimentary knowledge of the human genome and cumbersome methods of DNA sequencing. The relevant genes were identified and sequenced only in the nineties; e.g., TP53 gene mutations responsible for Li-Fraumeni syndrome were identified in 1990, and mutations in BRCA1 and BRCA2 responsible for familial breast and ovarian cancers were identified in 1994 and 1995, respectively. From that time, the identification of mutation carriers was possible, which opened the possibility of clinical applications and genetic counselling. At the National Research Institute of Oncology in Gliwice (MSCI Gliwice), we started the research in this area in the late eighties, initially in the context of the analysis of DNA damage in individuals exposed to environmental pollution. We discovered seasonal variations in markers of DNA damage, which mirrored the variations in concentrations of markers of air pollution. Subsequently, these markers of DNA damage were examined in the context of functional genetic polymorphisms in genes linked to the metabolism of environmental pollutants. These frequent genetic variations modulate cancer risk to a small degree, frequently only in individuals heavily exposed to carcinogens. This is in strong contrast to rare genetic mutations in familial cancer genes, which result in very high cancer risk. Because NRIO treats a large number of patients with breast or thyroid cancers, in the middle of 90-ties we started the search for individuals with mutations in genes associated with these neoplasms. This was possible due to extensive experience of NRIO researchers in genetic analysis, the advent of automatic DNA sequencers, and close collaboration between biologists and clinicians. This work resulted in the identification of more than 1000 mutation carriers, mostly in BRCA1 and BRCA2 genes, including individuals with recurrent mutations, 5382insC and 9631delC, respectively. This enabled the use of fast screening tests for carrier identification, which was important before the era of next-generation sequencing. We also identified novel mutant variants of TP53 and frequent germline mutations in the RET proto-oncogene in patients with apparently sporadic thyroid medullary carcinomas. Currently, this type of genetic analysis is a part of routine clinical practice.

[L-II-2] GERMLINE GENETICS OF COLORECTAL CANCER

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Twin studies have estimated that heritability accounts for 35% of the variation in susceptibility to colorectal cancer (CRC) and Swedish Family-Cancer Database studies have shown that about 15% of CRC patients have first-degree relatives affected by the same malignancy. Linkage analysis in large CRC families have identified the main CRC predisposition genes, including the mismatch repair genes (*MSH2*, *MLH1*, *MSH6* and *PMS2*) that cause Lynch syndrome and *APC*, a regulator of the WNT signaling, that causes familial adenomatous polyposis (FAP). More recently, next generation sequencing, together with linkage analysis, has identified further predisposition genes, including polymerase proofreading genes, *POLE* and *POLD1*, and other genes related to DNA repair, TGFβ and WNT signaling pathways. Altogether germline mutations in the high-to-moderate penetrance genes explain only a few percent of familial clustering of CRC. Genome-wide association studies have further identified more than 200 common, low-penetrance variants, that in combination as a polygenic risk score, may be associated with equally high risk of CRC as the high-penetrance variants. Finally, epidemiological evidence shows that many cancers share familial risks and that genes predisposing to other hereditary cancers and syndromes may also predispose to CRC.

[L-II-3] Expression patterns of circulating miRNAs associated with DNA repair defects.

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Introduction: Identifying germline *BRCA1/2* mutation carriers is vital for reducing their risk of breast and ovarian cancer.

Material and methods: To derive a serum miRNA-based diagnostic test we used samples from 653 healthy women from six international cohorts: Brigham and Women's Hospital (BWH; Boston, MA; *N*=87), Dana-Farber Cancer Institute (DFCI; Boston, MA; *N*=200), a separate sample set from the Center for Cancer Genetics and Prevention at DFCI (CCGP; Boston, MA; *N*=162), Tata Medical Center (DGO; Kolkata, India; *N*=20), Pomeranian Medical University (IHCC; Szczecin, Poland; *N*=52), and University of Pennsylvania (UPenn; Philadelphia, PA; *N*=132). The group included 350 (53.6%) with *BRCA1/2* mutations and 303 (46.4%) *BRCA1/2*-wild-type. All individuals were cancer-free before and at least 12 months after sampling. In parallel, to validate the findings, confirm the validity of miRNAs associated with BRCA-MT status, we prospectively recruited 34 women at the Medical University of Lodz. In that group we collected plasma samples and isolated lymphocytes and neutrophils to quantify their potential contribution to the circulating miRNA landscape. MiRNA sequencing was performed in that group using the same protocol as in the primary group.

Results: RNA-sequencing followed by differential expression analysis identified 19 miRNAs significantly associated with *BRCA* mutations, 10 of which were ultimately used for classification: hsa-miR-20b-5p, hsa-miR-19b-3p, hsa-let-7b-5p, hsa-miR-320b, hsa-miR-139-3p, hsa-miR-30d-5p, hsa-miR-17-5p, hsa-miR-182-5p, hsa-miR-421, hsa-miR-375-3p. The final model achieved 82.35% accuracy, 84.51% sensitivity and 79.39% specificity on the original training set. The training AUC ROC was 0.89 (95%CI: 0.87-0.93). In an independent validation group, sensitivity equalled 93.88% and specificity 80.72%. Mutated gene, menopausal status or having preemptive oophorectomy did not affect classification performance. In the Polish cohort, results were replicated in plasma. Correlation between the fold changes of miRNAs detected in both sets was $r=0.73$ $p<0.0001$ between the two sets, confirming the validity of the approach and miRNA selection. Differences between BRCA-MT and BRCA-WT miRNA profiles quantified within neutrophils and lymphocytes did not yield significant results. Fold changes of miRNAs quantified in the cells did not correlate with plasma levels ($r=-0.0136$ for neutrophils vs plasma and $r=-0.1253$ for lymphocytes $p>0.15$).

Conclusions: Circulating microRNAs may be used to identify *BRCA1/2* mutations in patients at high risk of cancer, offering an opportunity to reduce screening costs. Contribution of the circulating blood cells to the miRNA profile of BRCA-MT is negligible, suggesting their tissue origin. This newly developed approach supplements our previous effort by providing a means to identify individuals at elevated risk for ovarian cancer who require careful monitoring and may be advised to undergo risk-reduction surgery.

[L-II-4] Selenium and arsenic in relation to cancer risk among BRCA1 pathogenic variant carriers.

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Women with pathogenic BRCA1 variants have a substantially increased risk of breast and ovarian cancer. Strategies to reduce cancer risk beyond prophylactic surgery remain limited. Our goals was to evaluate whether selenium and arsenic blood concentrations are associated with incident cancer risk among BRCA1 pathogenic variant carriers participating in a dietary intervention study. Participants in the intervention group received dietary recommendations aimed at reducing selenium and arsenic intake based on repeated blood measurements obtained at baseline and during follow-up. During a mean follow-up of 55.0 months, 23 incident cancers were diagnosed in the intervention group, including 19 breast cancers and 4 cancers at other sites. Selenium concentrations in the lowest quartile were associated with increased cancer risk compared with quartiles 2 through 4 (hazard ratio [HR], 3.53; 95% CI, 1.48–8.41; P =0.004). Low arsenic concentrations were also associated with higher cancer risk, although this association was not statistically significant (HR, 1.80; 95% CI, 0.77–4.24; P =0.17). The strongest association was observed for combined selenium and arsenic concentrations below these thresholds (HR, 3.93; 95% CI, 1.58–9.80; P = 0.003). We conclude that low selenium concentrations, particularly when combined with low arsenic concentrations, were associated with increased cancer risk among BRCA1 pathogenic variant carriers.

[L-II-5] TARGETED GENOMIC PROFILING OF MELANOMA USING LIQUID BIOPSY

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Introduction: Genomic profiling of cancer using next-generation sequencing (NGS) has been extensively investigated for therapy selection, prediction and monitoring of treatment response across various cancer types, including melanoma. However, its clinical implementation remains limited. Here, we present our results of targeted ctDNA profiling in 138 patients with melanoma treated with palliative immunotherapy or targeted therapy.

Materials & methods: cfDNA (cell-free DNA) obtained from liquid biopsies of 138 patients with stage III or IV melanoma was analysed. For 50 patients, tumor DNA obtained from standard tissue biopsies was also analysed to assess concordance between tissue and liquid biopsy-based profiling. cfDNA was isolated using the QIAamp Circulating Nucleic Acid Kit (Qiagen). DNA libraries were prepared using a custom panel (30 genes) and the Ion AmpliSeq HD library kit. Sequencing and data analysis were performed using the Ion Torrent NGS platform, dedicated software, and Alamut Visual Plus. Copy number variation analysis was performed using the ONCOCNV v7.0 and ExomeDepth v1.1.15 software. Statistical analyses were performed in R (v4.5.2), utilizing multiple correspondence analysis for profiling and Fisher's exact test for pairwise comparisons. Associations between genomic alterations and treatment response were assessed using univariable and multivariable logistic regression adjusted for potential confounders (applying Firth's penalized regression for rare mutations). Statistical significance was defined as $p < 0.05$.

Results: The analysis identified 234 variants in plasma samples from 104 patients (75%) and 152 variants in tumour tissue samples from 48 patients (96%), covering all genes included in the panel with variant allele frequency (VAF) ranging from 0.1% to 86%. Variants were classified as pathogenic, likely pathogenic, or variants of uncertain significance (VUS-LP). The most frequently mutated genes were BRAF, NRAS, NF1, ARID2, TP53, and the TERT promoter. Concordance analysis demonstrated a 63% overlap in detected variants between liquid biopsy and tumor tissue samples. In 33 patients, two or more liquid biopsy samples were profiled before treatment and at disease progression, revealing increases or decreases in the VAFs of altered genes and/or the emergence of new variants. Statistical analyses of associations between genomic profiles and treatment response are ongoing.

Conclusions: Genomic profiling of melanoma tumor tissue and liquid biopsy samples is a useful approach for identifying genomic alterations and, in some cases, monitoring genomic evolution during treatment. The potential utility of our 30-gene NGS panel for predicting treatment response requires further investigation in larger patient cohorts.

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Session III

„Radiobiology and DNA damage/repair ”

[L-III-1] DNA DAMAGE AND RADIOBIOLOGY – GLIWICE PERSPECTIVE

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DNA damage and repair became important research areas at the Department of Tumour Biology (Institute of Oncology in Gliwice) in the early 1980s, forming an integral part of broader investigations into the role of environmental factors in cancer risk. Initiated by Prof. Mieczysław Chorąży and his team, this research closely complemented the emerging field of molecular epidemiology. Throughout the 1980s and 1990s, the Department hosted an Environmental Mutagenesis Laboratory, whose key researchers included Dr Grażyna Motykiewicz, Dr Ewa Grzybowska, Dr Jadwiga Michalska and Dr Jan Szeliga. The team employed a broad range of analytical techniques to investigate the effects of air pollution on genomic damage in the inhabitants of Silesia, as well as in occupationally exposed workers. The group maintained extensive international collaborations, particularly with Prof. Frederica Perera of Columbia University and Prof. Kari Hemminki of the Karolinska Institute. These partnerships led to numerous international research projects and publications documenting the role of environmental pollutants in cancer risk (including a landmark paper published in Nature in 1992).

One of the analytical methods introduced in Gliwice during this period was the ^{32}P-postlabelling technique, which enabled highly sensitive detection of DNA damage caused by chemical agents (so-called DNA adducts). This technique was subsequently adopted by Dr Joanna Rzeszowska's team to investigate DNA damage induced by chemical carcinogens, including 2-acetylaminofluorene and benzo[a]pyrene, in experimental model systems. This work led to original studies on the role of chromatin structure and nuclear scaffold structures in the formation and repair of DNA damage. In 1997, the DNA Topology Laboratory, headed by Prof. Joanna Rzeszowska, merged with the independent Radiobiology Laboratory, headed by Dr Maria Wideł, and a group of young clinician-radiotherapists led by Dr Rafał Tarnawski to establish the Department of Experimental and Clinical Radiobiology (Prof. Rzeszowska became its head). The Department continued its activities until 2010 and investigated numerous aspects of the molecular and cellular responses to ionising radiation and other DNA-damaging agents. Research topics included DNA damage-binding (DDB) proteins and their role in DNA damage recognition, genetic variants in DNA repair pathways, the effects of antioxidants on radiation-induced DNA damage, the radiation-induced bystander effect, and mathematical modelling of cellular responses to radiation, the latter in collaboration with the Silesian University of Technology.

Some of these research programmes were continued by Prof. Rzeszowska and Dr Wideł at the Biotechnology Centre of the Silesian University of Technology, where they moved in 2010. Other research directions evolved at the Institute of Oncology. Between 2010 and 2025, Prof. Piotr Widłak's team at the Clinical Proteomics Laboratory investigated systemic responses to ionising radiation at the proteome and metabolome levels, the role of NF-κB in radiation-induced sterile inflammation, and the role of extracellular vesicles in radiation-induced signalling. In addition, between 2020 and 2025, a Clinical Radiobiology Laboratory led by Prof. Dorota Słonina operated at the Institute of Oncology, whose research focused on low-dose hyper-radiosensitivity.

DNA damage and repair, together with radiobiology, became recurring themes at the Gliwice Scientific Meetings. Between 1997 and 2021, these topics featured in sessions held almost every year, encompassing nearly 140 lectures in total. The speakers included many internationally recognised experts in the field, among them Dr Leon H. Mullenders, Dr Margaret Zdzienicka, Dr Carmel Mothersill, Dr Nicolas Foray and Dr Kai Rothkamm.

[L-III-2] FROM DNA DAMAGE TO LEUKAEMIA DEVELOPMENT

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To improve health risk estimates and radiological protection for low dose and dose-rate exposures of ionising radiation (IR) encountered in occupational, medical, and public/emergency situations, further research is required. Epidemiological studies provided clear evidence for increased leukaemia incidence following IR exposure even at low doses with acute myeloid leukaemia (rAML) being the most prevalent. IR is a recognised mutagen and a carcinogen. Following irradiation, DNA damage occurs in hematopoietic stem and progenitor cells (HSPCs) in the bone marrow via direct energy transfer or indirectly through water radiolysis, which generates destructive Reactive Oxygen Species (ROS). While it causes a range of DNA lesions, radiation's hallmark is double-strand breaks (DSBs), some being complex clustered. If cells cannot repair the damage, they trigger apoptosis or senescence, which can lead to severe tissue toxicity. While accurate repair via Homologous Recombination preserves genetic integrity, the main DSBs repair pathway is the error-prone Non-Homologous End Joining creating permanent genetic deletions or chromosomal translocations; the consensus is that it leads to the formation of abnormal fusion oncogenes or disabling tumour suppressors in surviving, mutated HSPCs which gain a growth advantage, leading to clonal expansion over the following year (s) culminating in leukaemia diagnosis. However this has not been mechanistically fully demonstrated. Animal studies greatly contribute to improve the understanding of rAML. Murine rAML feature both hemizygous chromosome 2 deletions and point mutations (R235) within the haematopoietic regulatory gene Spi1. New pathways without Spi1 R235 where the decrease in Spi1 gene expression is negatively correlated with Spi1 promoter DNA methylation at specific CpG sites were identified. Moreover, we generated mouse models to track preleukemic cells in vivo to reveal the sequence of molecular events and identify the cell(s) of origin. A new model carrying a hemizygous Spi1 R235C point mutation was developed which confers hypersensitivity to rAML. The increased rAML sensitivity and shortened latency of this model allow to quantify rAML risk at low doses/dose-rates, otherwise prohibited by the high numbers of animals to reach statistical significance. Finally, the data generated allowed to rethink the role of IR in radiation leukaemogenesis, acting as an accelerator of AML development in this model rather than initiator through mutations. Increased risks of leukaemia following radiation exposure may be almost entirely attributable to a few predisposed individuals. If it is the case, there are major implications for how to think about radiation induces AML. While there is presently nothing that can be done to remove the mutated cells that accumulate in normal tissues, a promotion/acceleration stage that influences the rate of cancer development, can potentially be inhibited, hence preventing AML development.

[L-III-3] INFLUENCE OF RADIATION PARAMETERS ON RADIATION-INDUCED INFLAMMATION IN DIFFERENT PRIMARY HUMAN CELL LINEAGES.

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Background: Inflammation is a physiological immune response triggered by infectious agents or physical trauma, including ionizing radiation (IR). Inflammation is largely controlled at the transcriptional level after activation of the NF-kB transcription factor. IR–induced genotoxic stress also activates the Ataxia Telangiectasia Mutated (ATM)-dependent cellular DNA damage response (DDR), which orchestrates the transcriptional reprogramming in irradiated cells after activation of the p53 transcription factor. The interplay between NF-kB and p53 activities plays a major role in cell fate decisions through the induction of inflammatory and stress response genes, respectively. Irradiation may evoke different NF-kB and p53 responses in cells of different lineages.

We compared the induction of inflammatory and DNA damage responses in different primary human cells types exposed to different types (X-rays, particles) and doses/delivery of radiation, and the impact on the modulation of the immune microenvironment of irradiated cells.

Materials and Methods: We analysed by RT-qPCR the expression of genes under the control of p53 or NF-kB after exposure of primary blood cells, fibroblasts, keratinocytes and endothelial cells (ECs) to low and high doses of X-rays, protons and ^{12}C-ions. Cytokine secretion in culture media was measured by multiplexed FACS analysis. Modulation of the microenvironment was assessed by conditioned culture media transfer.

Results: High dose X-rays or ^{12}C-ions induces an early and transient inflammatory response in endothelial cells, not observed in blood cells, fibroblasts or keratinocytes. However, the transcriptional control of genes coding for different inflammatory factors becomes uncoordinated in ECs exposed to ^{12}C-ions of different energies. Different responses are observed in irradiated primary fibroblasts exposed to low doses of X-rays, protons and ^{12}C-ions.

Conclusion: Exposure to X-rays or particles of different LET differentially affect radiation-induced inflammation patterns in different cell lineages.

This work was supported by LABEx PRIMES (Grant n°ANR-11-LABX-0063), EDF CPPHE, GGP V3 -101 “Sciences du Vivant”, and EU’s EURATOM research and innovation program under the grant agreement 101061037 through the PIANOFORTE partnership.

[L-III-4] RADIOTHERAPY-INDUCED MODULATION OF THE URINARY MICROBIOME IN PATIENTS WITH PROSTATE CANCER

Dorota Gabryś[1], Ewelina Sibińska[2], Fernanda Monedeiro[3], Wioletta Miśta[1], Kinga Karoń [1] Adrian Arendowski[2], Piotr Fijałkowski[2], Monika Pietrowska[1], Jolanta Mrochem-Kwarciak[1], Anna Jędrzejewska[1], Ewa Telka[1], Małgorzata Rabsztyn[1], Donata Gräupner[1], Roland Kulik[1], Paweł Pomastowski[2], Michał Złoch[2]

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During radiotherapy for prostate cancer, adverse side effects induced in the urinary tract can impact a patient's quality of life. Although RT-induced urinary side effects are well-recognized complications of the treatment, changes in the urinary microbiome during and after RT remain insufficiently characterized. The aim of this study was to characterize the urinary microbiome using MALDI-TOF MS and assess its changes over time in patients undergoing RT for prostate cancer.

Blood samples for biochemical analyses and urine samples for microbiological assessment were collected at six time points: before gold fiducial marker implantation (T1), at the beginning of RT (T2), at the end of RT (T3), and 1, 4, and 7 months after RT completion (T4, T5, and T6, respectively). This research was conducted under grant NSC2020/39/B/NZ7/02733. Changes in microbial species composition were analyzed in relation to biochemical parameters measured in blood and urine.

A total of 1,773 microbial isolates were identified, with microorganisms detected in 89% of urine samples and 79% of samples exhibiting a polymicrobial composition. The most abundant group comprised members of the genus *Staphylococcus*, accounting for 51.6% of all isolates and represented by 24 species, including *S. epidermidis* (14%), *S. hominis* (10.3%), and *S. haemolyticus* (8.8%). *Micrococcus* (9.1%) was another relatively abundant genus, followed by *Enterococcus* (7.6%), *Kocuria* (5.6%), *Corynebacterium* (5.4%), and *Streptococcus* (2.2%).

Microbial diversity decreased at the end of RT and subsequently recovered during the seven-month follow-up period. Genera such as *Actinomyces, Corynebacterium, Staphylococcus,* and *Streptococcus* were significantly associated with the study time course, whereas *Kocuria rhizophila* showed a statistically significant increase in abundance over time ($p = 0.045$). A significant association was observed between the number of detected microbial species and sampling time (Fisher's exact test, $p = 5 \times 10^{-4}$; Pearson's chi-squared test, $p = 0.001$). Two phases of change were identified. First, the total number of species (TNS) increased from 51 at T1 to 62 at T2 and subsequently decreased to 53 at T3. Second, a considerable increase in the biodiversity of the urinary microbiota composition was noted within 1–7 months after RT, with the peak of the increase in species incidence at T5, with 87 different species. The prevalence of bacterial species was strongly associated with urinary glucose concentrations, with a weaker association observed for blood glucose levels. The presence of *Staphylococci* was associated with higher tPSA levels.

RT for prostate cancer is associated with dynamic changes in the urinary microbiome, characterized by an initial reduction in species diversity at the end of treatment, followed by an increase during subsequent follow-up. Glucose concentrations in urine and blood were significantly associated with microbial composition, suggesting that host metabolic factors may contribute to RT-associated changes in the urinary microbiome. These findings highlight the interplay between RT, host metabolism, and urinary microbiota and may support further investigation.

Session IV

„Clinical Proteomics”

[L-IV-1] CLINICAL PROTEOMICS – GLIWICE PERSPECTIVE

Piotr Widłak[1] and Monika Pietrowska[2]

[1]Medical University of Gdańsk, Gdańsk, [2]Institute of Genetics and Animal Biotechnology, Jastrzębiec, Poland

Clinical proteomics emerged as a coherent research field at the Institute of Oncology in Gliwice in 2007. That year, our team published its first review article on the subject and launched its first research grant dedicated to cancer proteomics. A major milestone in the development of the team was the acquisition of its first mass spectrometer, the UltrafleXtreme MALDI-TOF/TOF instrument, purchased with funding from the Silesian Bio-Farma project. Initially, the instrument was used for serum peptidome profiling and was subsequently employed for molecular tissue imaging.

The team's early projects focused on analyzing the endogenous serum peptidome as a potential diagnostic and prognostic biomarker in cancer. The scope of its research soon expanded to include the characterization of the human body's response to ionizing radiation and the monitoring of radiation-induced responses in patients undergoing radiotherapy. Key partners during this period included colleagues from the Institute of Bioorganic Chemistry in Poznań (Prof. Maciej Stobiecki, Dr Łukasz Marczak and Dr Anna Wojakowska), who introduced our team to the field of mass spectrometry; researchers from the Silesian University of Technology (Prof. Joanna Polańska and Prof. Andrzej Polański), who developed unique computational algorithms for our applications; and clinical collaborators (Professors Ryszard Tarnawski, Rafał Suwiński, Leszek Miszczyk and Krzysztof Składowski), whose involvement enabled access to valuable biological specimens.

Another important stage in the team's development was the implementation of advanced, specialised tools for MALDI mass spectrometry imaging of tissues. This was made possible through collaboration with Dr Andreas Römpp, Dr Corinna Henkel and Prof. Malcolm Clench, as well as participation in a European COST Action. In addition to proteomic analyses, the team also explored the use of mass spectrometry-based serum metabolomics to identify biomarkers for the early detection of lung cancer, in collaboration with colleagues from the Medical University of Gdańsk (Prof. Witold Rzyman and Prof. Rafał Dziadziuszko), and to investigate biomarkers of breast cancer risk, in collaboration with Prof. Tone Bathen and colleagues at the Norwegian University of Science and Technology in Trondheim. The team's most recent research area was the proteomics of extracellular vesicles. This field became a major focus of Prof. Monika Pietrowska's research and was developed largely through her collaboration with Prof. Theresa Whiteside of the University of Pittsburgh.

The Clinical Proteomics Laboratory was headed by Prof. Piotr Widłak until 2021, when he moved to the Medical University of Gdańsk. Prof. Monika Pietrowska subsequently led the laboratory until 2026, when it was closed and she moved to the Institute of Genetics and Animal Biotechnology in Jastrzębiec. Over its 20 years of activity, research conducted by the Clinical Proteomics Laboratory and its scientific collaborations resulted in 72 publications, which have received more than 1,400 citations.

The laboratory's extensive international collaborations also enabled the team to invite research partners from around the world to dedicated proteomics and metabolomics sessions at the Gliwice Scientific Meetings (GSM). Since 2007, these sessions have been held nine times, in 2007, 2009, 2012, 2013, 2018, 2020, 2021, 2023 and 2026, featuring more than 60 speakers in total. Some of these sessions were co-organized with the Polish Proteomics Society, whose founding members included members of our team.

[L-IV-2] ANALYZING THE DYNAMICS OF MICROGLIA DURING DEVELOPMENT IN HEALTH AND DISEASE: A FUNCTIONAL OMICS APPROACH.

Maciej Łałowski

Department of Gene Expression, Institute of Molecular Biology and Biotechnology, Faculty of Biology, Adam Mickiewicz University, Poznań, Poland; Biochemistry/Developmental Biology and HiLIFE, Meilahti Clinical Proteomics Core Facility, University of Helsinki, Finland.

Part I: Microglia are the main drivers of inflammation in the central nervous system. They are known to respond to both dietary changes and psychological stress, notably by modifying their production of cytokines and relationships among the brain parenchyma. Psychological stress increases the risk for psychiatric disorders, often triggering central nervous system inflammation driven by microglia. Because the ketogenic diet (KD) exhibits anti-inflammatory properties, we investigated its impact on stress responses. Adult male mice fed a control diet or KD underwent 10 days of repeated social defeat (RSD) and were classified as susceptible (SUS) or resistant (RES). Mice on the KD showed a higher trend toward the RES phenotype. Morphological and electron microscopy analyses in the ventral hippocampus CA1 revealed distinct microglial alterations, with KD-fed animals exhibiting reduced cellular stress markers. Furthermore, SUS mice showed decreased microglial-synaptic contacts and a distinct hippocampal lipidomic profile compared to controls. Together, these unique metabolic and microglial adaptations demonstrate novel mechanisms by which KD enhances psychological stress resilience.

Part II: Microglia are CNS innate immune cells that migrate toward injuries, but the role of the fractalkine receptor (CX3CR1) in this process remains unclear. Proteomic profiling of *Cx3cr1*-deficient murine cortex predicted altered RHO-signaling, hinting at cytoskeletal dysregulation. Two-photon *in vivo* imaging after a cortical laser lesion revealed that *Cx3cr1*-deficient microglia exhibited enhanced migration, as well as increased length and velocity of processes extending toward the injury. Normal migration in *Ccr2*-deficient mice confirmed this accumulation was independent of monocyte-derived macrophages. Thus, CX3CR1 modulates microglial migration toward CNS injury, representing a potential therapeutic target.

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[L-IV-3] MULTI-MODAL MASS SPECTROMETRY IMAGING TO INVESTIGATE DRUG-INDUCED RESPONSE

Laura Cole

Sheffield Hallam University, Sheffield, United Kingdom

A Multimodal Mass Spectrometry Imaging (MSI) approach has been used to investigate the chemotherapy drug-induced response of a Multicellular Tumour Spheroid (MCTS) 3D cell culture model of osteosarcoma (OS). The work addresses the critical demand for enhanced translatable early drug discovery approaches, by demonstrating robust spatially resolved molecular distribution analysis in tumour models following chemotherapeutic intervention. Advanced high-resolution techniques were employed to assess the interplay between metabolic and cellular pathways in response to chemotherapeutic intervention.

[L-IV-4] FROM SIMPLICITY OF ONCOGENIC MUTATIONS TO COMPLEXITY OF EXTRACELLULAR VESICLE LANDSCAPES AND THEIR BIOLOGICAL EFFECTS IN CANCER

Janusz Rak

McGill University, Montreal, Canada

Oncogenic transformation and rise of cancer cell populations entails a built-in juxta/paracrine program that orchestrates formation of the tumour microenvironment (TME) and impacts systemic dimensions of the disease. This remarkable transition from a solitary mutational hit (or their small number) to expansion of complex multicellular networks results, at least in part, from deregulation of the soluble and particulate cancer cell secretome, the latter epitomised by extracellular vesicles and particles (EVPs). EVPs are small cellular fragments either containing lipid membranes (vesicles) or membrane-free multimolecular aggregates (particles), which are released to pericellular milieu in response to intracellular or external cues. Several studies documented the impact of oncogenic pathways on the landscape of EVPs. This is particularly apparent in glioblastoma (GBM) where oncogenic mutation of epidermal growth factor receptor (EGFRvIII) reprograms EVP biogenesis, molecular content and biological activity, including the EVP-mediated extracellular export of EGFRvIII oncogene itself. Using this mechanism EGFRvIII-driven mesenchymal human glioma stem cells (MES-GSCs) transmit EGFRvIII to endothelial cells triggering non-angiogenic, transformation-like responses leading to formation of dysmorphic, enlarged blood vessels (vasectasia) resistant to antiangiogenic agents. Paradoxically, while the expression of EGFRvIII on the surface of cancer cells is often demonstrably high and uniform, only a fraction of their derived EVPs carry EGFRVIII oncoprotein, while the same cells may also shed a diverse repertoire of EGFRvIII-negative EVPs enriched in other cargo, such as proteasome, or nuclear histones. The enrichment of certain fractions of EVPs in histones is of interest as their mutant forms, such as H3K27M oncohistone H3 may act as oncogenic drivers in their own right, particularly in a subset of pediatric GBM-like tumours known as diffuse midline gliomas (DMGs). Interestingly, the epigenome deregulating H3K27M mutation also orchestrates complex changes in the EVP repertoire of DMG cells, including the release of chromatin particles carrying H3K27M oncoprotein itself. Thus, oncogenic mutations exert multifaceted and consequential effects on the particulate secretome of cancer cells resulting in the onset of abnormal cell-cell communication, changes in the TME and entry of molecularly informative biomarkers (driver mutations) into biofluids, with important implications for liquid biopsy diagnostics.

Session V

„Mathematical modelling of biological processes”

[L-V-1] HOUSTON TO GLIWICE AND BACK.

Marek Kimmel

Rice University, Houston, USA; Silesian University of Technology, Gliwice, Poland

The talk is a historical mini-review of mathematical models of cell cycle dynamics that were authored and co-authored by Marek Kimmel. It will start from the stochastic point process approach in the 1980s, and then continue to the differential and integral equation models conceived jointly with Ovide Arino and supported by Zbigniew Darzynkiewicz' experiments. An important episode was the work on the book "Branching Processes in Biology" jointly with David Axelrod. It will also refer to optimal control and other work with Colleagues from Gliwice and other places in Poland, such as Lublin or Warsaw. Finally, it will cover more recent work based on single-cell genome dynamics and disease models of cancer and leukemias. This work is ongoing.

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[L-V-2] DISSECTING ANTIVIRAL IMMUNE RESPONSE AT THE POPULATION AND SINGLE-CELL LEVEL

Maciej Czerkies, Zbigniew Korwek, Wiktor Prus, Paulina Koza, Frederic Grabowski, Ilona Kosiuk, Joanna Jaruszewicz, Marek Kochańczyk and Tomasz Lipniacki

Institute of Fundamental Technological Research, Warsaw, Poland

The innate immune system translates pathogen-induced signals into cell-fate decisions throughout the infected population. We demonstrate that feedback interactions between the transcription factor pathways IRF3, NF-κB, and STAT lead to switch-like responses to a viral analog, poly(I:C), in contrast to pulse-like responses to bacterial LPS. Poly(I:C) activates both IRF3 and NF-κB, which is required for the induction of interferon β (IFNβ) expression. IFNβ activates STAT signaling and triggers an antiviral state in bystander cells by upregulating positive-feedback components including RIG-I, PKR, and RNase L (1).

Next, we showed that in cells transfected with poly(I:C), transcriptional STAT activity is terminated due to depletion of the IFNβ receptor, IFNAR. Two ISGs, RNase L and PKR, not only hinder the replenishment of IFNAR but also suppress negative regulators of IRF3 and NF-κB, thereby promoting IFNβ transcription. We incorporated these findings into a comprehensive mathematical model of innate immunity. By coupling signaling through the IRF3/NF-κB and STAT1/2 pathways with the activity of RNase L and PKR, the model explains how poly(I:C) switches the transcriptional program from STAT1/2-induced to IRF3/NF-κB-induced, turning IFNβ-responding cells into IFNβ-secreting cells (2).

In the context of RSV infection, we showed that the mutual antagonism between RSV and innate signaling leads to dichotomous responses at the single-cell level. RSV blocks innate responses at three levels: by inhibiting IRF3 activation, interferon synthesis, and STAT1/2 activation. In turn, proteins coded by ISGs inhibit the synthesis of viral RNA and viral proteins. The consequence of these mutual inhibitions is a lack of cells that simultaneously express viral proteins and IFNβ (3).

Overall, our analysis indicated that innate signaling components that correlate at the population level may be anticorrelated at the single-cell level.

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[L-V-3] EXISTENCE AND CONVERGENCE OF STOCHASTIC PROCESSES UNDERLYING A THIN LAYER APPROXIMATION OF A COUPLED BULK-SURFACE PDE

Adam Bobrowski

Lublin University of Technology, Lublin, Poland

We present a system of coupled bulk-surface partial differential equations (BS-PDEs), describing changes in concentration of certain proteins (Rho GTPases) in a living cell. These proteins, when activated, are bound to the plasma membrane where they diffuse and react with the inactive species; inactivated species diffuse inside the cell cortex; these react with the activated species when they are close to the plasma membrane. For our case study, we model the cell cortex as an annulus, and the plasma membrane as its outer circle.

Mathematically, the aim of the paper is twofold: Firstly, we show the master equation for the changes in concentration of Rho GTPases is the Kolmogorov forward differential equation for an underlying Feller stochastic process, and, in particular, the related Cauchy problem is well-posed. Secondly, since the cell cortex is typically a rather thin domain, we study the situation where the thickness of the annulus modeling the cortex converges to 0. To this end, we note that letting the thickness of the annulus to 0 is equivalent to keeping it constant while increasing the rate of radial diffusion. As a result, in the limit, solutions to the master equation lose dependence on the radial coordinate and can be thought of as functions on the circle. Furthermore, the limit master equation can be seen as describing diffusion on two copies of the circle with jumps from one copy to the other.

[L-V-4] MATHEMATICAL MODELLING OF TELOMERE DYNAMICS

Marie Doumic-Jauffret

Institut national de recherche en informatique et en automatique (INRIA), Paris, France

Telomeres are specific DNA sequences at the ends of chromosomes that act as caps to protect them from degradation and fusion with other chromosomes. During DNA replication, they are only partially replicated. Their progressive erosion ultimately causes replicative senescence and is linked with aging and tumor suppression. In this talk, I will review recent results and open problems on mathematical models for telomere shortening and re-elongation, both with stochastic branching processes and their deterministic limits. Mathematical analysis and quantitative comparison with data from *Saccharomyces cerevisiae* reveal selection forces within a population and the sources for heterogeneity in individual cell fates.

Session VI

„New bioactive compounds and biomaterials”

[L-VI-1] DISCOVERY AND *IN VITRO* CHARACTERIZATION OF CLIP-2, A FIRST-IN-CLASS SMALL-MOLECULE COVALENT INHIBITOR OF PD-L1/PD-1 INTERACTION

Radoslaw Kitel[1], Agnieszka Maslanka[1,2], Wiktor Uzar[1,2], Damian Muszak[1], Piotr Wilk[3], Bozena Skupien-Rabian[3,4], Urszula Jankowska[3,4], Lukasz Skalniak[1]

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The interaction of programmed death receptor (PD-1) with its ligand (PD-L1) represents a major immune checkpoint that cancer cells exploit to evade immune surveillance. Targeting either PD-1 or PD-L1 with therapeutic monoclonal antibodies (mAbs) has transformed cancer treatment in the recent decade. However, only a small portion of patients (10-30%) develop durable responses, and current therapies are associated with the risk of immune-related adverse events (iRAEs). Consequently, a large medical need remains for safe PD-L1/PD-1 inhibitors.

In response to these challenges, we discovered CLIP-2, a potent covalent small-molecule inhibitor that targets PD-L1 and impairs its interaction with PD-1. CLIP-2 acts as a cross-linking inhibitor of PD-L1, leading to irreversible dimerization. Mechanistically, CLIP-2 induces irreversible PD-L1 dimerization through covalent engagement of Y123. This activity is retained in the cellular environment and translates into robust T-cell activation in a reporter-based assay. Notably, CLIP-2 does not exert significant toxicity to cancer cells or T-cells, thereby outperforming currently available small-molecule reversible PD-L1 inhibitors.

In summary, CLIP-2 is a first-in-class small-molecule covalent PD-L1 inhibitor with activity in cell-based assays and therefore emerged as a valuable lead compound for the development of a new class of therapeutics targeting the PD-L1/PD-1 immune checkpoint.

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[L-VI-2] DEVELOPMENT OF MULTIFUNCTIONAL BIOMATERIALS VIA ELECTROACTIVE SURFACE ENGINEERING TO MODULATE CELL–MATERIAL INTERACTIONS

Katarzyna Krukiewicz

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Electroactive materials offer exciting opportunities for the development of multifunctional biomaterials capable of actively modulating cell–material interactions. Among these, conducting polymers combine tunable physicochemical properties with electrical functionality, making them promising candidates for advanced biomedical applications, particularly in neural tissue engineering and implant surface modification. However, achieving the desired biological response while maintaining biocompatibility and preventing bacterial colonization remains a major challenge.

Over the past decade, the Bioelectrochemistry Group at the Silesian University of Technology has advanced the development of electroactive biomaterials for biomedical applications, with a particular focus on engineering multifunctional surfaces to control biological responses. This lecture will present selected achievements illustrating how electrochemical surface engineering and complementary polymer modification strategies can tailor biomaterial interfaces to meet specific therapeutic needs. Examples will include hybrid conducting polymer films designed to promote neurite outgrowth and neural–electrode integration; curcumin-loaded electroactive coatings combining antibacterial activity with improved biocompatibility; bioactive polymer coatings developed to mitigate brain scarring around cranial implants; and heparan sulfate-mimetic coatings that provide neuroprotective and neurotropic effects at neural electrodes. These studies demonstrate complementary strategies for modulating neuronal responses, promoting favourable cellular interactions, limiting adverse host reactions, and addressing implant-associated infections. Particular attention will be given to the interplay between surface properties, electrical functionality, cytocompatibility, antimicrobial activity, and selective cellular responses, as well as the challenges of translating multifunctional interfaces into practical biomedical applications.

Ultimately, this work demonstrates the potential of rational surface engineering to transform biomaterials from passive structural components into active interfaces capable of influencing biological processes and supporting next-generation bioelectronic and regenerative therapies.

Acknowledgements. The research of the Bioelectrochemistry Group is supported by the National Science Centre, Poland [2021/42/E/ST5/00165; 2023/49/B/ST4/02942; 2024/06/Y/ST5/00217], the Polish Agency of Academic Exchange [BPN/BFR/2024/1/00011/U/00001] and the National Centre of Research and Development [LIDER15/0024/2024].

[L-VI-3] CELL-MADE SCAFFOLDS IN TISSUE ENGINEERING: FROM CONTROLLED MATRIX ASSEMBLY TO MECHANISTICALLY GUIDED ALIGNMENT

Kristýna Valášková[1], Lukáš Maňas[1,2], Małgorzata Adamiec-Organiściok[3,4], Leona Mahelová[2], Magdalena Skonieczna[3,4] and Petr Humpolíček[1,2]

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The development of scaffolds that reproduce the structural and biochemical complexity of native tissues remains a major challenge in tissue engineering. Cell-made scaffolds (CMSs), generated through the self-assembly of extracellular matrix (ECM) by living cells, offer a biologically derived alternative to conventional materials. Their potential is based on the maintenance of ECM architecture during processing and the control of its organisation to deliver appropriate structural cues to cells. This study aims to develop fibroblast-derived CMSs, evaluate their suitability for revitalisation, and investigate mechanical stimulation as a strategy for inducing anisotropic tissue alignment.

Human dermal fibroblasts (HDFs) were cultured for eight weeks in conditions that promoted the deposition of the ECM to produce cohesive CMSs. The resulting sheets were then devitalised and decellularised, after which their structural integrity, ECM preservation and cellular removal were evaluated using histological, protein and gene expression analyses. To assess their suitability for cellular revitalisation, the decellularised CMSs were seeded with H9c2 cardiomyoblasts. Meanwhile, mechanical stimulation was applied to the scaffolds during their cultivation in a custom chamber with an actuated membrane area of 25 mm² that provided deformation to encourage the directional organisation of the fibroblasts and their ECM.

Long-term HDF culture produced cohesive sheets that could be handled and processed. The analyses confirmed ECM structure preservation and effective cellular component removal following decellularisation. In another reseeding experiment with a different cell line, CMSs supported H9c2 adhesion and proliferation, resulting in elongated cellular morphology. Mechanical stimulation in the custom chamber induced pronounced alignment of fibroblasts, indicating the potential for mechanically guided anisotropy in cell-made scaffolds.

These findings suggest that CMSs are versatile platforms that combine cell-derived matrix cues with the potential for controlled structural organisation. Controlled membrane deformation was found to promote directional fibroblast alignment, indicating that the custom stimulation system provides mechanical cues capable of guiding cellular organisation within developing CMSs. This approach provides a foundation for creating anisotropic CMSs for tissue engineering. However, further quantitative analysis of alignment and functional validation is required to determine how mechanically conditioned CMSs can be tailored to specific applications in tissue engineering.

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[L-VI-4] SEEING THE UNSEEN: EXPANDING THE BOUNDARIES OF CELLULAR IMAGING WITH CY-EXM

Seweryn Gałecki[1,2], Kevin M. Dean [1]

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A cell cannot be fully understood by visualizing just four molecular targets at a time. Its biological complexity lies not merely in the identity of its individual components, but in their intricate spatial relationships across molecular and organelle scales. Yet, conventional fluorescence microscopy remains constrained by diffraction and spectral overlap, limiting our ability to visualize multiple biological targets at super-resolution across the entire 3D cellular volume.

In this talk, I will present Cyclically Multiplexed Expansion Microscopy (Cy-ExM), an approach integrating cryopreservation, physical sample expansion, and iterative immunolabeling to reconstruct cellular architecture in three dimensions, mapping up to 20 molecular targets with ~70 nm precision. I will demonstrate its applications in revealing the nanoscale organization of cytoskeletal networks and intracellular organelles across diverse biological models, including mouse fibroblasts, human breast cancer cells, Ewing sarcoma cell colonies, and cutaneous squamous cell carcinoma sections. Quantitative analyses of organelle architecture, including the Golgi apparatus, alongside assessments of structural stability and epitope preservation across repeated imaging cycles, highlight the method's reproducibility and analytical potential.

Looking beyond individual cells, I will discuss the opportunities and challenges of extending Cy-ExM to highly multiplexed imaging of complex tissues and organs, paving the way toward comprehensive nanoscale atlases with applications in oncology and neuroscience.

Poster Abstracts

Poster sessions

I: Molecular and Cellular Biology

II: Disease OMICS

III: Bioinformatics and Mathematical Modeling

IV: Biomaterials and Applied Biotechnology

V: Bioactive Compounds and Medicinal Chemistry

VI: New Diagnostic and Therapeutic Approaches

Posters marked with an asterisk (*) will be presented during the:

Young Scientist poster session

Session I: Molecular and Cellular Biology

[I-1] EFFECT OF MELATONIN ON DOXORUBICIN-INDUCED APOPTOSIS IN ADIPOSE-DERIVED STEM CELLS

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Adipose-derived stem cells (ADSCs) represent a promising cell source for regenerative therapies due to their accessibility, proliferative capacity, and multilineage potential. However, exposure to cytotoxic agents may markedly impair their survival and regenerative properties. Doxorubicin (DOX) exerts potent anti-cancer activity but also damages non-cancerous cells, also stem cells. Melatonin (MEL) has attracted attention as a potential protective compound because of its antioxidant properties.

This study aimed to evaluate whether MEL modifies DOX-induced cytotoxicity in ADSCs, with particular emphasis on cell viability and apoptosis.

The experiments were performed using Adipose-Derived Mesenchymal Stem Cells (PCS-500-011, ATCC). The experiments were performed using the following experimental groups: (1) control – untreated ADSCs (C), (2) melatonin (MEL) – ADSCs treated with melatonin alone, (3) doxorubicin (DOX) – ADSCs treated with doxorubicin alone, (4) DOX + MEL – ADSCs pretreated with melatonin followed by doxorubicin exposure, (5) DOX + LUZ – ADSCs treated with the MT1/MT2 receptor antagonist luzindole prior to doxorubicin exposure, (6) DOX + MEL + LUZ– ADSCs pretreated with melatonin in the presence of the MT1/MT2 receptor antagonist luzindole, followed by DOX exposure. Cellular viability was assessed using cytotoxicity assays, whereas apoptosis-related protein expression was evaluated by Western blotting.

Results showed that DOX markedly impaired ADSC viability and metabolic activity, reducing ATP, decreasing total cellular protein, and mitochondrial activity. MEL alone did not adversely affect ATP content or mitochondrial activity and increased total protein content compared with control cells. However, MEL pretreatment did not restore DOX-induced cytotoxicity. Western blot analysis demonstrated pronounced activation of the intrinsic apoptotic pathway following DOX treatment, characterized by increased cleaved CASP-9 and cleaved CASP-3 together with elevated p53 expression. Addition of MEL to DOX did not suppress caspase activation and was associated with prominent cleavage of both caspase-9 and caspase-3.

Under the investigated experimental conditions, MEL did not provide substantial protection against DOX-induced cytotoxicity in ADSCs. Luzindole modified the cellular response to DOX and MEL, including changes in metabolic activity and apoptosis-related protein expression. It suggests that melatonin receptor-dependent signaling may contribute to the observed effects.

[I-2] LOMEFLOXACIN AS A POTENTIAL PHOTOSENSITIZER IN CHERENKOV RADIATION-ENHANCED RADIOTHERAPY (RECA)

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Radiotherapy remains one of the main treatment modalities for cancer, yet its efficacy may be limited by locoregional recurrence. Cherenkov radiation-enhanced photoactivation (RECA) is a promising strategy for increasing tumour cell radiosensitivity. Clinical megavoltage photon beams generate Cherenkov radiation in tissues, including a UV component. Its use to activate a photosensitizer may enhance the cytotoxic effects of radiotherapy.

This study aimed to evaluate lomefloxacin, a clinically used antibiotic with phototoxic properties, as a photosensitizer in RECA. In vitro experiments were performed using the human FaDu hypopharyngeal squamous cell carcinoma line and the A253 salivary gland carcinoma line. Cells were treated with lomefloxacin (200 μM) and subsequently irradiated with a 6 MV X-ray photon beam at 100 MU/min, using either a single dose of 1 x 10 Gy or a fractionated regimen of 3 x 2.5 Gy. Cell viability and death were assessed using Annexin V/7-AAD, DNA fragmentation by TUNEL, mitochondrial membranę depolarization, proliferative capacity by CellTrace, cell-cycle distribution and DNA damage based on γH2AX levels. Analyses were performed by flow cytometry.

FaDu cells, radiotherapy combined with lomefloxacin produced the strongest apoptotic and cytotoxic response after 3 × 2.5 Gy, reflected by increased Annexin V/7-AAD and TUNEL positivity, reduced proliferative capacity and decreased clonogenic survival. The combined treatment also induced changes in cell-cycle distribution, including a reduction in the G1 population, an increase in the sub-G1 fraction and elevated γH2AX levels. After 1 × 10 Gy, radiotherapy alone induced an effect similar to that seen with the combination of lomefloxacin and radiation therapy, indicating no marked additional effect of lomefloxacin under this irradiation regimen. In A253 cells, radiotherapy alone induced a strong cell-death and γH2AX response after both irradiation regimens. However, the addition of lomefloxacin more effectively inhibited proliferation, particularly after 3 × 2.5 Gy, and enhanced DNA fragmentation. After 1 × 10 Gy, the combination mainly intensified cell-cycle alterations, including a reduction in G1 and increases in the sub-G1 and G2/M fractions.

These findings indicate that lomefloxacin has radiosensitizing potential in the RECA model. However, the magnitude and character of this effect depend on the cell line and irradiation regimen. Clinically available drugs with phototoxic properties may represent a promising group of compounds for further studies aimed at enhancing radiotherapy efficacy.

[I-3] LINKING CELL-MIMETIC MEMBRANE PROPERTIES WITH CELLULAR RESPONSES: LANGMUIR MONOLAYERS AND IN VITRO CELL CULTURES ACROSS DIFFERENT EXPOSURE ROUTES

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The lipid composition of a cell membrane shapes its interactions with substances encountered through different exposure routes. Here, we combine Langmuir monolayers with in vitro cell cultures to examine the effects of selected substances at three levels: individual membrane lipids, multicomponent model membranes, and membranes of living cells. The applicability of this approach will be illustrated by examples involving polyphenols (oral exposure), triclosan (TCS; dermal exposure), and ozone (inhalation exposure).

A central feature of this approach is the use of both single-component and multicomponent lipid monolayers. Single-component systems help identify which lipids interact most strongly with a given substance, whereas mixtures selected to reflect the membrane composition of the cell lines under study reveal how these interactions change in a more complex lipid environment. Varying the proportions of saturated and unsaturated lipids further clarifies the contribution of lipid composition. Changes in surface pressure–area isotherms provide information on monolayer organization and physicochemical properties, including the mean molecular area and the static surface compression modulus. These parameters are considered alongside biochemical readouts from cell cultures, such as cell viability.

Our cellular models focus particularly on cancer cell lines. Comparing lipid-level responses with changes in cancer cell viability may help identify membrane interactions relevant to the cellular effects of the studied substances. This combined approach also provides a basis for further investigation of their potential role in processes associated with cancer progression. Together, the two models offer a framework for connecting changes at lipid interfaces with cellular responses across distinct exposure routes.

[I-4] Robust Activation of p53 as an Innovative Strategy to Enhance Cancer Cell Sensitivity to Fas Receptor Ligand (FasL)

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The tumor suppressor p53 activates the pro-apoptotic gene FAS, encoding the death receptor for Fas ligand (FasL). However, FasL alone induces limited apoptosis in cancer cells. We previously demonstrated that pretreatment with actinomycin D and nutlin-3a (ActD+Nut3a), which can be replaced by its derivative idasanutlin (ActD+Ida), markedly sensitizes cancer cells to FasL-induced apoptosis. Exposure to a low FasL concentration (20 ng/mL) for only 5 hours eliminates most cancer cells. This effect depends strictly on TP53 status and has been observed across diverse cancer types, while being markedly reduced in normal human fibroblasts and epithelial cells. This study aimed to evaluate ActD+Ida pretreatment followed by FasL exposure in an *in vivo* xenograft model and patient-derived tumor organoids.

BALB/c nude mice bearing human A549-luc2 lung cancer xenografts were divided into four groups: control, FasL alone, ActD+Ida, and ActD+Ida followed by FasL. Treatment was administered twice, and tumor growth was monitored. Tumors were analyzed by hematoxylin and eosin staining and immunohistochemistry to assess tissue architecture, cell death (TUNEL assay), vascularization, and immune cell infiltration. Patient-derived colorectal, pancreatic, and gastric tumor organoids with known TP53 status were treated, and cytotoxicity was assessed using the ATPlite assay.

In the A549-luc2 xenograft model, ActD+Ida followed by FasL significantly inhibited tumor growth and induced extensive tumor cell death, with a high proportion of TUNEL-positive cells. In patient-derived organoids, cytotoxic activity was observed in the gastric cancer model, whereas no comparable effect was detected in the tested colorectal and pancreatic models.

ActD+Ida pretreatment may enhance FasL-induced apoptosis *in vivo*. However, heterogeneous responses in patient-derived organoids highlight the need to identify molecular determinants of treatment sensitivity. TP53 status may help guide patient selection, but larger studies are needed to establish its predictive value. This strategy could support targeted therapeutic approaches for selected patients with TP53-wild-type tumors.

[I-5] TGF-B1–3 AND TGFBR1–3 IN EPITHELIAL OVARIAN CANCER: COORDINATED EXPRESSION, DIVERGENT METHYLATION AND THE DISTINCT BEHAVIOUR OF TGFBR3

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The role of transforming growth factor beta (TGF-β) in epithelial ovarian cancer depends on disease stage: in early, well-differentiated tumours it limits cell proliferation, whereas in advanced disease it promotes epithelial–mesenchymal transition, remodelling of the tumour stroma and escape from immune surveillance. It remains unclear whether all three ligands (TGF-β1, TGF-β2, TGF-β3) and all three receptors (TGFBR1, TGFBR2, TGFBR3) change in the same way during this shift, and whether methylation of their promoters is reflected in transcript and protein levels; these questions have not previously been addressed simultaneously within a single patient cohort.

We examined tissue from 109 women, comprising 40 patients who received surgery followed by platinum-based chemotherapy (group A), 13 patients managed with surgery only (group B) and 56 women without oncological disease (group C). For each of the six pathway components, transcript levels were quantified by RT-qPCR ($2^{-\Delta\Delta Ct}$ method with ACTB and GAPDH as reference genes), protein concentrations by ELISA, and promoter methylation status by methylation-specific PCR (MS-PCR). Transcript and protein levels of TGFB1, TGFB2, TGFB3, TGFBR1 and TGFBR2 increased progressively from group C to group B and were highest in group A (Kruskal–Wallis $p<0.05$ for all measures, $p<10^{-10}$ for most). TGFBR3 was the only component to show the opposite trend, decreasing towards group A (protein, Kruskal–Wallis $p=6.5\times10^{-11}$). Transcript and protein levels were positively correlated for all six genes (Spearman $\rho=0.44$–0.85), and the twelve mRNA and protein parameters distinguished tumour from control tissue with AUC values between 0.644 and 0.958. Three separate methylation patterns emerged. The TGFBR3 promoter was methylated in all group A tumours compared with only 8.9% of control samples (χ^2 $p=4.9\times10^{-18}$), consistent with the classical inverse association between methylation and reduced expression. TGFB2 displayed a paradoxical pattern, with promoter methylation increasing in parallel with its expression ($p=0.002$). For TGFB1, TGFB3, TGFBR1 and TGFBR2, methylation frequency did not differ systematically between groups. Within group A, TGFBR3 was the only marker whose transcript and protein levels were significantly elevated in platinum-resistant compared with platinum-sensitive tumours ($p=0.0009$ and $p=0.017$, respectively).

Taken together, these findings indicate that the TGF-β ligand–receptor axis is activated in a coordinated yet heterogeneous manner as disease progresses from benign tissue to ovarian cancer requiring chemotherapy. TGFBR3 differs from the remaining five components in the direction of its dysregulation, its link to promoter hypermethylation and its association with platinum resistance, which makes it the most mechanistically distinctive member of the panel and a promising candidate for further evaluation as a biomarker of chemoresistance.

[I-6] EZH2 TARGETING INDUCES SENESCENCE-ASSOCIATED PHENOTYPIC REMODELING AND MODULATES TUMOR–STROMAL SIGNALING IN LUNG ADENOCARCINOMA

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Introduction/Rationale: Enhancer of zeste homolog 2 (EZH2), the catalytic subunit of polycomb repressive complex 2 (PRC2), is frequently overexpressed in lung adenocarcinoma (LUAD) and contributes to transcriptional programs supporting tumor proliferation and cellular plasticity. EZH2 may also be involved in the regulation of cellular senescence, which can alter tumor cell-derived signals and influence stromal cell responses. However, the effects of EZH2 targeting on senescence-associated changes in LUAD cells and their potential impact on stromal fibroblast activation remain insufficiently understood.

Methods: In this study, we investigated two distinct EZH2-targeting strategies in A549 human lung adenocarcinoma cells: catalytic inhibition with EPZ6438 (5 μM) and targeted degradation using the PROTAC-like degrader MS1943(4 μM). Western blotting was performed to assess the protein levels of EZH2 and the cell cycle regulators p16 and p21, while senescence-associated β-galactosidase (SA-β-gal) staining was used to quantify β-galactosidase-positive cells. RT-qPCR was performed to assess the expression of key inflammatory genes associated with senescence-associated secretory phenotype (SASP), namely CXCL8, CCL2, and IL6. Subsequently, primary murine lung fibroblasts were exposed to conditioned media from treated A549 cells and fibroblast activation was evaluated by immunofluorescence staining for alpha-smooth muscle actin (αSMA) and collagen type I alpha 1 (COL1A1).

Results: EZH2 targeting increased p16 and p21 protein levels and the proportion of SA-β-gal-positive cells, with more pronounced effects following MS1943 treatment. These changes were accompanied by an increased expression of SASP-related inflammatory genes. Conditioned media from treated A549 cells increased αSMA and COL1A1 staining in primary murine lung fibroblasts, with the strongest effects observed following exposure to conditioned media from MS1943-treated cells.

Conclusion: EZH2 targeting induces senescence-associated changes in lung adenocarcinoma cells and modulates tumor cell-derived signals associated with fibroblast activation. Importantly, MS1943 elicited more pronounced effects than EPZ6438, highlighting differences between catalytic inhibition and targeted EZH2 degradation. These findings warrant further investigation into the contribution of non-canonical EZH2 functions to tumor cell plasticity and tumor-stromal signaling.

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[I-7] SMALL EXTRACELLULAR VESICLES IN THE MODULATION OF FERROPTOSIS

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Ferroptosis is an iron-dependent form of regulated cell death characterized by the accumulation of reactive oxygen species (ROS) and lipid peroxidation. Its regulation is closely linked to iron homeostasis and glutathione (GSH) metabolism. Ferroptosis has gained increasing attention for its role in cancer biology and its interactions with cellular metabolic pathways.

Small extracellular vesicles (sEVs) are nanosized structures, approx. 30-150 nm in diameter, produced by all cell types, and involved in intercellular communication through the transfer of proteins, nucleic acids, and lipids. In cancer, tumor-derived extracellular vesicles (TEX) can promote tumor progression by modifying the tumor microenvironment, stimulating angiogenesis, suppressing immune responses, and influencing cellular metabolism. Emerging evidence also suggests that extracellular vesicles can modulate ferroptosis by regulating iron transport, antioxidant defenses, and lipid peroxidation. By enhancing glutathione-dependent GPX4 activity or activating the NRF2 pathway, these vesicles may protect recipient cells against oxidative damage [1]. Tumor-derived vesicles have also been associated with ferroptosis resistance in several cancer types, including gastric cancer [2].

Despite growing interest in the relationship between extracellular vesicles and ferroptosis, the links between ferroptotic cell death and vesicular molecular cargo remain poorly understood. This work aims to investigate these interactions and explore how extracellular vesicle composition relates to ferroptosis. A better understanding of these mechanisms may provide new insights into cancer biology and support the development of therapeutic strategies that exploit ferroptosis to target tumor cells.

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[I-8] ESRP2 AS A REGULATOR OF CISPLATIN SENSITIVITY IN BLADDER CANCER CELLS IN IN VITRO AND IN VIVO MODELS

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Background: Variability in response to cisplatin remains a significant challenge in the treatment of bladder cancer. However, the mechanisms determining cancer cell sensitivity to therapeutic stress are still poorly understood. ESRP2 is a regulator of the epithelial phenotype and cellular plasticity, suggesting its potential role in shaping treatment response.

The aim: The aim of this study was to evaluate the effect of ESRP2 on the sensitivity of bladder cancer cells to cisplatin and to determine whether this effect is observed in both *in vitro* and *in vivo* conditions.

Materials and methods: Cell viability analysis was performed in bladder cancer cells with overexpression of ESRP1 and ESRP2 in response to increasing concentrations of cisplatin. Additionally, a colony formation assay was performed to assess the clonogenic potential of the cells following cisplatin treatment. Subsequently, the impact of ESRP expression on treatment response was evaluated in a mouse model with subcutaneously implanted tumor cells.

Results: Cells overexpressing ESRP2 demonstrated increased sensitivity to cisplatin compared to controls, as evidenced by reduced viability in in vitro assays. This effect was more pronounced than that observed for ESRP1. In the in vivo model, tumors expressing ESRP2 exhibited a stronger response to cisplatin treatment, resulting in inhibited tumor growth. Differences between ESRP1 and ESRP2 suggest distinct roles of these proteins in regulating therapy response.

Conclusions: ESRP2 enhances the sensitivity of bladder cancer cells to cisplatin and may serve as an important regulator of the response to therapeutic stress. The findings suggest that ESRP2-mediated changes in cellular state influence the ability of cancer cells to survive treatment.

[I-9] HSPA1 VS. HSPA2: DISTINCT ROLES IN EPIDERMAL DIFFERENTIATION AND INFLAMMATORY REACTIONS

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Keratinocytes serve as the fundamental building material of the epidermis, establishing a physical and immunological barrier between the human body and the external environment. Heat shock proteins (HSPs) act as molecular chaperones regulating proteome stability by supervising protein folding and degradation. The HSPA (Hsp70) family comprises 12 highly homologous proteins predominantly exhibiting cytoprotective roles. Although HSPA1 and HSPA2 share substantial structural similarity and intracellular localization, their distribution across epidermal layers differs distinctly. HSPA1 is present throughout all strata (with highest concentrations in the spinous and granular layers), whereas HSPA2 is predominantly localized to the basal layer. Furthermore, these proteins diverge functionally: HSPA1 acts primarily as a cytoprotectant, while HSPA2 contributes significantly to tissue homeostasis.

Previous investigations by our team highlighted the involvement of HSPA2 in epidermal immune responses, yet literature regarding HSPA1's specific role in epidermal differentiation and maturation remains scarce. We hypothesized that HSPA1 and HSPA2 fulfill distinct biological functions within the epidermis, though functional redundancy cannot be entirely dismissed. This study aimed to perform a comparative analysis of both HSPA proteins in keratinocytes by evaluating their impact on epidermal differentiation, maturation, and inflammatory regulation. Utilizing a reconstructed human epidermis (RHE) model – a 3D co-culture of fibroblasts and CRISPR-Cas9-edited keratinocytes lacking HSPA1 or HSPA2 expression (HSPA1-KO, HSPA2-KO) – we assessed morphological and molecular alterations. Immunohistochemical (IHC) staining for epidermal maturation markers (filaggrin and involucrin) and progenitor cell marker (p63) was conducted to evaluate epidermal proliferation and differentiation. The contribution of these proteins to inflammatory processes was quantified via cytometric analysis of media collected from RHE cultures under standard and pro-inflammatory conditions. IHC findings revealed that HSPA2-deficient RHE constructs were thinner and exhibited reduced expression of keratinocyte differentiation markers relative to controls. Conversely, the absence of HSPA1 induced no observable changes in marker protein localization or expression levels. Cytometric profiling further demonstrated that HSPA2 deficiency leads to elevated baseline secretion of interleukins and interferon under standard conditions – an effect absent in the HSPA1-deficient model. In conclusion, HSPA1 and HSPA2 perform divergent functions within the human epidermis. HSPA2 is actively engaged in differentiation and maturation processes, serving as a critical pillar for tissue homeostasis and participating in inflammation regulation. Conversely, the contribution of HSPA1 appears minor, potentially reflecting functional redundancy within the HSPA family and compensation by alternative chaperones.

[I-10] THE EFFECTS OF MICROPLASTICS AND NANOPLASTICS ON THE CENTRAL NERVOUS SYSTEM AND BIOLOGICAL BARRIERS AT THE CELLULAR LEVEL: POTENTIAL NEUROBIOLOGICAL RISKS

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Microplastics and nanoplastics represent a significant environmental concern and a potential threat to human health. Due to their small size and diverse physicochemical properties, these particles can enter the human body through various routes, including ingestion and inhalation. This literature review aimed to summarize current knowledge of their physicochemical properties, routes of entry into the body, distribution within tissues, and potential effects on biological barriers and the central nervous system (CNS). Particular attention was given to the blood–brain barrier (BBB), the blood–cerebrospinal fluid barrier (BCSFB), the intestinal barrier (IB), and the blood–placental barrier (BPB). The review covered in vitro studies using cellular models, in vivo studies involving animal models, and research investigating the presence of microplastics and nanoplastics in human tissues. Potential mechanisms of cellular toxicity, including oxidative stress, inflammatory responses, and cell death pathways, were also examined, alongside studies exploring possible associations with cognitive impairment and neurodegenerative diseases, including dementia. Current research provides evidence of potential effects of micro- and nanoplastics on biological barriers and the CNS. However, further studies are needed to better understand the underlying mechanisms and their possible implications for human health.

[I-11] SNAIL-REGULATED SMALL RNAS AS MEDIATORS OF RHABDOMYOSARCOMA PROGRESSION AND CELL-TO-CELL COMMUNICATION

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Rhabdomyosarcoma (RMS) is a common pediatric soft tissue mesenchymal tumor associated with significant morbidity and mortality. It arises from impaired differentiation of myogenic progenitors. The SNAIL transcription factor is a crucial regulator of RMS development and modulates the expression of several microRNAs in RMS, including miR-28-5p, miR-28-3p, and miR-193a-5p.

This study investigated whether SNAIL-regulated small RNAs contribute to RMS progression and intercellular communication. To assess the effects of SNAIL silencing on the small-RNA transcriptome, we performed next-generation sequencing in RMS cells. This analysis revealed significant changes in the expression of several small nucleolar RNAs (snoRNAs), identifying new candidates for mechanistic studies and functional investigation in RMS.

Additionally, we investigated the extracellular function of SNAIL-regulated miRNAs. We demonstrated that RMS cells secrete miRNAs via extracellular vesicles, and that tumor-derived miR-193a-5p is internalized by endothelial cells, altering their function and suggesting a role in tumor-driven vascularization.

To establish the intracellular functional role of miR-28-5p, RMS cells were transfected with miR-28-5p mimics. Overexpression of miR-28-5p upregulated myogenic regulatory factors, suppressed RMS cell proliferation, and altered cell cycle progression, and inhibited migration. It also decreased chemotaxis and Matrigel invasion toward FBS and HGF.

In conclusion, our findings identify novel snoRNAs for further investigation in RMS and indicate that endothelial uptake of tumor-derived small RNAs may contribute to RMS progression. Furthermore, we establish miR-28-5p as an important regulator that inhibits proliferation and migration while promoting the myogenic differentiation of RMS cells.

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[I-12] AGO2-ASSOCIATED CIRCRNAS IN T-CELL ACUTE LYMPHOBLASTIC LEUKEMIA: FUNCTIONAL INVESTIGATION REVEALS ONCOGENIC CIRCN4BP2L2

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Circular RNAs constitute an emerging area of intensive research in cancer. In T-cell acute lymphoblastic leukemia (T-ALL), an aggressive hematologic malignancy characterized by clonal proliferation of T-cell precursors, the function of most aberrantly expressed circRNAs is not yet understood. To identify circRNAs acting as miRNA sponges, we performed AGO2 immunoprecipitation and RNA sequencing (AGO2-RIP-seq) in four T-ALL cell lines. Our analysis revealed circRNAs consistently enriched in the AGO2-bound fraction, highlighting a new resource for T-ALL research. Notably, circRNAs were more enriched (7.3%) compared to linear RNAs (3%). For functional investigation, we focused on the most abundant AGO2-bound circRNAs that were also found to be dysregulated in T-ALL patients. Knockdown of circN4BP2L2 revealed its ability to promote cell proliferation and confer resistance to apoptosis in T-ALL cell lines, in addition to modulating key T-ALL pathogenic pathways. Analysis of gene expression of T-ALL patients disclosed peculiar features of T-ALL with high circN4BP2L2 expression. A marked overlap was observed between differentially expressed genes upon knockdown of circN4BP2L2 in vitro and the profiles of circN4BP2L2 stratified T-ALL cases. This work provides new insights into the circRNA-mediated regulatory networks driving this malignancy and presents circN4BP2L2 as a key RNA shaping oncogenic phenotype in T-ALL.

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[I-13] IMPACT OF MIR-146A SILENCING ON MELANOMA VIABILITY AND RADIATION RESPONSE

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MicroRNAs (miRNAs) are short, non-coding RNA molecules that control gene expression at the post-transcriptional level. Depending on the tissue and cellular context, they can act as either oncogenes or tumor suppressors. In radiotherapy, ionizing radiation (IR) damages the DNA of cancer cells, and the cellular miRNA profile directly influences this radiation response.

This study investigates the functional significance of miR-146a in melanoma and its impact on radiosensitivity. We exposed Me45 melanoma cells to 4 Gy of IR. IR induced a G2/M cell cycle arrest at 8 h and increased apoptosis rates at 24 h compared to non-IR controls. Next, we performed small RNA sequencing at 4 and 12 h post-IR to assess early transcriptomic changes. The analysis revealed sustained alterations in three specific transcripts: miR-1260, miR-933, and a novel sequence, chrM:11954-11969. Notably, miR-146a was the most highly expressed miRNA in both control and irradiated Me45 cells. We confirmed these high levels in three melanoma cell lines (Me45, WM1552c, and WM793b) using RT-qPCR. To evaluate the role of miR-146a in melanoma, we inhibited its expression via lentiviral transduction using a miR-Zip-146a vector. A scrambled sequence (SCR) served as a negative control. Both vectors co-expressed a GFP reporter, allowing us to track the transduced cells by flow cytometry in a GFP competition assay. The suppression of miR-146a caused a progressive decline in the GFP-positive Me45 population compared to co-cultured wild-type cells. This decline was not observed when the SCR vector was used. This confirms that miR-146a promotes melanoma cell viability. This may be due, at least in part, to the observed higher percentage of apoptotic cells in Me45 cells with inhibited miR-146a. However, when we investigated its role in the radiation response, exposing the miR-146a-depleted Me45 cells to 4 Gy of IR produced no significant changes in apoptosis compared to the control groups.

In summary, silencing miR-146a severely impairs melanoma viability. Further research will be focused on miR-146a target genes involved in cell survival.

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[I-14] CONCENTRATION OF DICKKOPF AND SECRETED FRIZZLED-RELATED PROTEINS IN DIFFERENT LOCATION OF NON-SMALL CELL LUNG CANCER

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In 2024, lung, bronchus, and trachea cancers were the most frequently diagnosed globally, with a leading role in cancer-related deaths. Secreted frizzled-related proteins (SFRPs) and the Dickkopf protein family (DKKs) are key regulators of the Wnt signalling pathway, which is linked to tumour progression.

The aim of this study was to evaluate the concentration of SFRPs and DKK proteins in tumour and margin tissues obtained from 65 patients with primary Non-Small Cell Lung Cancer (NSCLC) in location in right and left lungs. An enzyme-linked immunosorbent assay was used to measure the protein concentrations.

No statistically significant differences were observed in TNM classification, grading, smoking status, age, and sex in location of the tumour in the left or right lung. However, there were significant differences in the concentration of SFRP1, SFRP2, and SFRP5 in tumour between the right and left lung, with no difference in margin. For DKK proteins, there were differences in tumour for DKK-1 protein and in margin for DKK-2 protein between locations.

Differences in the concentrations of the studied proteins could be affected by the location of the tumour due to the varying influence of the surrounding area. Analysed proteins in different locations do not influence tumour progression in different way.

[I-15] When the Antioxidant Shield Breaks: Genetic Context as a Determinant of Ferroptosis Vulnerability in Lung Cells

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Cells undergo ferroptosis when lipid peroxidation outpaces their antioxidant defences, yet both normal and malignant cells differ widely in how easily they reach this threshold. Protection depends mainly on GPX4, the NRF2-driven transcriptional response and the thioredoxin system, but it remains unclear how p53 status and the loss of GPX4 modify this protection in the lung epithelium.

To address this, we compared two lung cancer cell lines, A549 (p53 +/+) and H1299 (p53 −/−), with non-transformed bronchial epithelial BEAS-2B cells in three variants: wild-type, CRISPR control and GPX4 knockout. Ferroptosis was triggered with erastin at 5 and 10 μM for 24 h. Cell viability was determined by the MTT assay, and the mRNA levels of NRF2, TRX, TXNRD1 and IRP2 were measured by RT-qPCR.

The three systems responded to erastin in clearly different ways. A549 cells were only partially affected, and this tolerance was accompanied by higher expression of NRF2 and TXNRD1, suggesting that compensatory antioxidant mechanisms were switched on. In H1299 cells, viability dropped more strongly and the pattern of redox gene expression was altered, which points to a reduced ability to cope with ferroptotic stress. The most dramatic loss of viability was observed in GPX4-knockout BEAS-2B cells, which additionally failed to induce NRF2- and TRX-related responses, indicating that without GPX4 the remaining defences cannot be mobilised effectively. Changes in IRP2 expression suggest that iron metabolism also contributes to the final outcome.

Taken together, our results indicate that vulnerability to ferroptosis in lung epithelial cells is set by the combination of p53 status, GPX4 function and the capacity to activate antioxidant signalling. In this view, genetic background determines how much adaptive reserve a cell can draw on when the shield is under attack, and thus whether it survives or dies. These observations provide a rationale for exploiting ferroptosis as a selective therapeutic approach in lung cancer.

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[I-16] N-GLYCOSYLATION PROFILES OF EXTRACELLULAR VESICLES DERIVED FROM RENAL CANCER CELLS AND REFERENCE NON-MALIGNANT EPITHELIAL CELLS

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Background: Extracellular vesicles (EVs) participate in intercellular communication and transport glycoproteins that may reflect the molecular characteristics of their cells of origin. Alterations in N-glycosylation are associated with cancer development and may influence protein sorting, vesicle interactions, and cellular responses. Characterizing EV-associated N-glycans may therefore provide insight into molecular changes accompanying renal cancer.

Objective: This study aimed to characterize the N-glycan composition of EVs released by renal cancer cells and identify differences relative to EVs derived from non-malignant renal epithelial cells.

Methods: EVs were isolated from conditioned media of Caki-2 papillary renal carcinoma cells and non-malignant HREpC renal epithelial cells. Vesicular preparations were characterized using transmission electron microscopy, nanoparticle tracking analysis, and immunoblotting for selected EV-associated markers. N-glycan composition was analyzed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). Individual glycan structures and major glycan groups were evaluated to identify differences in their relative abundance.

Results: A total of 44 N-glycan compositions were identified across the analyzed renal cell-derived EV samples, including 40 structures in Caki-2-derived EVs and 44 in HREpC-derived EVs. Comparative analysis of individual structures revealed increased abundance of oligomannose glycans H8N2, H9N2, and H10N2 in EVs released by renal carcinoma cells. Analysis of major glycan groups also demonstrated an increased relative abundance of oligomannose-type N-glycans and a reduction in hybrid-type structures in cancer-derived EVs. Furthermore, overall sialylation was decreased, with lower abundance of selected sialylated structures, including H5N4E2, H6N5E1L2, and H6N5E2L1. These findings indicate that renal carcinoma-associated changes involve both the distribution of major N-glycan groups and the abundance of individual glycan structures.

Conclusions: Renal carcinoma-derived EVs exhibit increased oligomannose content and reduced hybrid glycosylation and sialylation. These findings provide a basis for further investigation of EV-associated glycosylation in renal cancer.

[I-17] THE SIGNIFICANCE OF NADPH OXIDASES (NOX) AND THE NADP+/NADPH BALANCE IN THE OXIDATIVE RESPONSE AND SURVIVAL OF HCT116 COLORECTAL CANCER CELLS

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Oxidative stress is an imbalance between the production of reactive oxygen species (ROS) and the capacity of antioxidant systems to neutralise them. It plays an important role in cancer pathogenesis and in regulating cellular susceptibility to ferroptosis, an iron-dependent form of cell death driven by membrane lipid peroxidation. NADPH oxidases (NOX) occupy a special place among ROS sources, because they generate free radicals deliberately at the expense of oxidising NADPH to NADP+. The same cofactor is essential for both major antioxidant shields of the cell. Glutathione reductase (GSR) uses NADPH to regenerate glutathione (GSH) from its oxidised form (GSSG), and thioredoxin reductase (TRXRD) uses it to restore reduced thioredoxin. Excessive NOX activity may therefore deplete the reducing pool, impair GPX4 function and increase the susceptibility of cancer cells to ferroptosis.

HCT116 colorectal cancer cells were treated with erastin, an inhibitor of the cystine/glutamate antiporter (system xC−), at 10 and 15 μM. The response was assessed at successive time points up to 24 h. Metabolic activity was measured with the MTS assay, ROS levels with the DCFH-DA probe, and lipid peroxidation with the BODIPY-C11 probe. The GSH/GSSG ratio was determined by a bioluminescent assay. Expression of the FSP1, TFRC and DUOX2 genes was analysed by RT-qPCR. In parallel, a five-dimensional deterministic kinetic model of the antioxidant network was developed based on mass-action kinetics. The model comprises coupled glutathione and thioredoxin modules fuelled by a shared NADPH pool.

Erastin caused a transient, statistically significant decrease in metabolic activity at 3 h and 12 h, to about 80–84% of control. Cytosolic ROS levels, in contrast, remained close to control throughout the experiment. The most pronounced effect was a late, drastic collapse of the GSH/GSSG ratio. At 18 h it fell from about 30 in control cells to 0.37–0.64 in treated cells, and at 24 h a dose dependence was observed. This was accompanied by a trend towards increased lipid peroxidation at 24 h. FSP1 expression was also markedly reduced; FSP1 provides a glutathione-independent line of defence against ferroptosis that acts in parallel to GPX4. Expression of DUOX2, a member of the NOX family, followed a biphasic pattern: early induction at 1–3 h, followed by strong silencing in the late phase. The model reproduced a physiological redox steady state (GSH/GSSG ≈ 58, TRXred/TRXox = 4.0) and identified the NADPH pool as the factor limiting the capacity of both antioxidant shields.

These results point to a membrane-associated, ferroptotic character of the HCT116 response to erastin. They support the hypothesis that NOX-related NADPH consumption increases cellular susceptibility to ferroptosis, although they require confirmation in biological replicates. The model provides a starting point for simulating the effects of NOX overactivity and glutathione depletion.

[I-18] THE POTENTIAL FOR EXOSOME USE IN DIAGNOSTICS AND TREATMENT OF MALIGNANT BRAIN GLIOBLASTOMA

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Stage IV glioblastoma (GB), previously known as glioblastoma multiforme (GBM) is the most frequently diagnosed and the most aggressive primary brain tumor. GB cells are strongly resistant to temozolomide (TMZ) and radiotherapy and its infiltrative growth makes full tumor resection unfeasible. In recent years, many studies revealed the possible potential of exosomes in diagnosis and treatment of tumors that are resistant to the standard treatment options. Exosomes are small extracellular vesicles that carry biologically active particles (cargo). They participate in intracellular communication and tumor's microenvironment (TME) formation. The use of exosomes will have a crucial role in the development of new diagnostic methods for glioblastoma and drug delivery. It is mainly because most of the chemotherapeutic agents, like TMZ do not pass brain-blood-barrier (BBB) well.

[I-19] Exploring the Prognostic Interplay Between FGFR2 Expression and the Stromal Molecular Landscape in Breast Cancer

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Keywords: BC breast cancer; CAFs cancer-associated fibroblasts; TME tumour microenvironment

Background: Stromal signalling may influence the biological and prognostic relevance of fibroblast growth factor receptor 2 (FGFR2) in breast cancer. This study investigated whether stromal molecular profiles identify prognostic subgroups and modify the association between tumour FGFR2 expression and patient outcomes.
Methods: Laser capture microdissection was used to isolate stromal tissue from 44 invasive breast carcinomas of no special type. Expression of a custom panel of 47 FGFR2-related stromal genes was assessed using the NanoString nCounter platform, while tumour FGFR2 expression was evaluated immunohistochemically using the H-score. Genes associated with progression-free survival (PFS) in Cox models accounting for FGFR2 expression and molecular subtype were selected for k-means clustering. Associations with survival and interactions between FGFR2 and stromal profiles were examined in the entire cohort and the estrogen receptor-positive (ER-positive) subgroup.
Results: Two stromal clusters were identified. Cluster 1 (n = 15), characterized by higher CCL5, FAP, HDGF and S100A4 expression, was associated with poorer outcomes. Cluster 2 (n = 29), characterized by higher COL1A1, CXCL8, FBLN1, HSPG2, MMP14 and SRGN expression, was associated with more favourable survival. The clusters did not differ significantly in ER, PR, or HER2 status, lymph node involvement, or histological grade. The strongest prognostic association was observed for PFS in ER-positive patients (HR = 6.853, 95% CI: 1.80–26.10; p = 0.005) which was comparable to stage of the disease (cluster: HR = 6.853, 95%CI 1.80-26.10, p=0.005; stage I/II vs III: HR=1.15, 95%CI 0.042-0.514, p=0.003). Higher FGFR2 expression showed a borderline association with poorer outcome in this subgroup. However, no significant FGFR2–cluster interaction was detected (p = 0.590).
Conclusion: Our findings strongly suggest that stromal composition may provide important information when addressing one of the key clinical questions in luminal breast cancer: why do some patients experience disease recurrence despite otherwise favorable tumor characteristics?

[I-20] MTARC2 Loss Reprograms Lipid Metabolism and the Transcriptomic Landscape in Colorectal Cancer

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Background: The mitochondrial amidoxime-reducing component (mARC) complex, comprising MTARC1, MTARC2, Cyb5R and Cyb5b, is anchored to the outer mitochondrial membrane and reduces N-oxygenated functional groups. MTARC2 deficiency enhances fatty acid oxidation and protects mice against diet-induced obesity, while human MTARC1 variants protect against chronic liver disease. MTARC2 expression is dysregulated in cancer, with reduced protein levels in colorectal cancer (CRC), previously attributed to promoter hypermethylation. We investigated how MTARC2 loss affects CRC metabolism and tumorigenic potential.

Methods: MTARC2 was knocked out by CRISPR in SW48, HT29 and RKO cells, with protein loss confirmed by mass spectrometry. RNA-Seq, proliferation assays and xenografts in immunodeficient mice assessed transcriptomic and growth effects. MTARC2 protein and promoter methylation were evaluated in matched tumor and normal colonic mucosa from 100 CRC patients using immunohistochemistry and MeDIP/pyrosequencing. LC–MS lipidomics compared parental and MTARC2-KO SW48 cells under 10% and 1% FBS conditions; NMR metabolomics assessed additional metabolic changes.

Results: MTARC2 loss had no consistent effect on proliferation or xenograft growth across cell lines, although SW48 xenografts showed increased tumor volume. RNA-Seq revealed dysregulation of glycolysis, glucose and carbohydrate metabolism, and lipoprotein assembly and clearance. CRC tissues showed reduced MTARC2 protein, inversely associated with tumor stage, without significant differences in promoter methylation. MTARC2-KO SW48 cells exhibited 269 altered lipid species: 34 increased and 235 decreased, predominantly glycerophospholipids, glycerolipids and sphingolipids. Serum deprivation further reduced 84 species, mainly acylcarnitines and ceramides, and increased four unsaturated phospholipids. NMR revealed lower carnitine, choline and UDP-N-acetylglucosamine levels, indicating altered energy, lipid and nucleotide metabolism.

Conclusions: MTARC2 loss induces substantial transcriptomic and metabolic reprogramming without consistently affecting CRC cell proliferation. Protein reduction in CRC tissues and altered glycolytic and lipid pathways suggest that MTARC2 modulates the CRC metabolic landscape. Extensive lipid remodeling, intensified by serum deprivation, suggests a shift toward a more catabolic state. NMR findings are consistent with impaired mitochondrial fatty acid transport and membrane phospholipid turnover. Together, these results suggest that MTARC2 downregulation contributes to metabolic reprogramming during CRC progression without detectable promoter methylation changes, warranting investigation of alternative epigenetic and post-transcriptional mechanisms.

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[I-21] ALIX DEFICIENCY REMODELS THE PROTEOMIC CARGO OF SMALL EXTRACELLULAR VESICLES RELEASED BY MELANOMA CELLS

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Keywords: ALIX; high-resolution mass spectrometry; melanoma; proteomic cargo; small extracellular vesicles

Small extracellular vesicles (sEV) released by melanoma cells contribute to intercellular communication, tumor progression, immune regulation, and remodeling of the tumor microenvironment. ALIX is an ESCRT-associated protein involved in endosomal membrane remodeling, multivesicular body formation, and extracellular vesicle biogenesis. However, its contribution to selective incorporation of proteins into melanoma-derived sEV remains incompletely understood. This study aimed to determine how ALIX deficiency affects the proteomic composition of sEV released by melanoma cells and to identify ALIX-associated molecular pathways potentially involved in vesicular cargo sorting.

sEV were isolated from conditioned media of wild-type (A375-WT) and ALIX-knockout (A375-ALIX-KO) melanoma cells, with three biological replicates per group. Protein composition was investigated using LC–MS/MS-based proteomics. Quantitative comparisons were complemented by principal component analysis, hierarchical clustering, and a detection-based analysis of proteins consistently identified in WT-derived sEV but undetected in ALIX-KO-derived preparations. Functional enrichment analyses were performed using Gene Ontology, Kyoto Encyclopedia of Genes and Genomes and Reactome databases.

Proteomic profiling revealed substantial differences between sEV preparations derived from A375-WT and A375-ALIX-KO cells. A total number of 375 proteins were identified in all preparations: 208 were common for both groups, and 167 were unique for A375-WT. The obtained dataset was further analyzed using qualitative and quantitative approach. The former approach revealed 6 proteins which exhibited significantly higher abundance in ALIX-KO-derived sEV after multiple-testing correction (FDR<0.05), namely: histone H3.4, purine-rich element-binding protein B, ribosomal protein P2 (RPLP2), amino acid transporter SLC7A5, histone H2B type 1-M (H2BC13), and tetraspanin CD81.

To conclude, ALIX deficiency profoundly alters the proteomic landscape of sEV released by A375 melanoma cells. The loss of detectable proteins associated with endosomal sorting, ESCRT-dependent processes and cellular adhesion, together with increased abundance of selected proteins, suggests that ALIX contributes to regulation of vesicular protein cargo composition. These findings highlight ALIX as an important factor associated with molecular heterogeneity of melanoma-derived sEV and provide a basis for investigating the functional consequences of altered vesicle cargo for melanoma progression and tumor-microenvironment communication.

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[I-22] FUNCTIONAL AND PROTEOMIC PROFILES OF CD3(+) PLASMA-DERIVED SMALL EXTRACELLULAR VESICLES DIFFERENTIATE CANCER PATIENTS FROM HEALTHY DONORS

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Keywords: cancer plasma; high-resolution mass spectrometry; melanoma cell-derived sEVs (MTEX); rewiring of CD3(+) sEV; small extracellular vesicles (sEV)

Plasma of cancer patients contains a heterogeneous mix of small extracellular vesicles (sEV) derived from tumor cells (tumor-derived extracellular vesicles, TEX) and various tissue-resident and circulating nonmalignant cells, including immune cells. Separation from plasma of CD3(+)sEV that are products of T lymphocytes allows for assessments of their molecular and functional characteristics in addition to enabling studies of the TEX-enriched CD3(−)sEV fraction. Emerging data suggest that T lymphocytes in melanoma (and other cancers) are reprogrammed by the tumor and acquire functional characteristics that differentiate them from T cells in healthy individuals. As T cells in the tumor-bearing hosts frequently have compromised immune competence, the examination of T cell-derived CD3(+)sEV, which mimic parent T cells, could inform about the phenotypic and functional status of patients' T cells.

In the presented study, we applied the immune capture strategy to separate CD3(+)sEV from the plasma of melanoma patients (MPs). Blood samples were obtained from 10 consenting healthy donors (HDs) and 10 melanoma patients treated at the UPMC Hillman Cancer Center. Vesicles were isolated from the precleared plasma by ultrafiltration followed by size exclusion chromatography.

The functional studies performed suggest that CD3(+)sEV obtained from MPs have acquired some functional attributes that are usually associated with MTEX, including pro-tumor effects such as promotion of tumor growth and suppression of T cells or fibroblast migration. The obtained results support our hypothesis that in MPs, T cells are reprogrammed by MTEX produced by the tumor and acquire the ability to produce and release CD3(+)sEV that differ functionally from CD3(+)sEV of HD's and recapitulate certain functional features of MTEX. Comparison of mass spectrometry-based proteomic profiles of CD3(+)sEV isolated from the plasma of MPs and HDs revealed differences, especially in the abundance of species involved in the function of the immune system, including increased levels of immunoregulatory proteins in CD3(+)sEV in MPs (e.g. CD36, CD47, and CD59). On the other hand, among proteins downregulated in MPs' CD3(+)sEV were heat shock proteins, which are general stimulators of immune response. Moreover, among downregulated processes associated with proteins discriminating MPs from HDs was the response to pathogens. Overall, the obtained data suggest that CD3(+)sEV may be potentially valuable for molecular, functional, and metabolic monitoring of T cells in patients with melanoma, and likely in patients with other cancers.

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[I-23] MITOCHONDRIAL METABOLISM OF GLYCEROL AS A REGULATOR OF T CELL ACTIVATION

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In recent years major advances have been made in our understanding how cellular metabolism controls the function of T cells. However, the role of glycerol metabolism in that respect still poses an unresolved question.

We have previously established that mitochondrial reactive oxygen species (mtROS) are crucial for T cell activation. T cell receptor (TCR)-induced mtROS are indispensable for the full transcriptional response of activated T cells. Interestingly, TCR triggering activates mitochondrial glycerol-3-phosphate dehydrogenase (GPD2), which seems to play a major role in mtROS generation.

Obesity-associated chronic inflammation leads to diabetes and cancer and is mediated by infiltrating, hyperactivated T cells. Interestingly, the interstitial space of adipose tissue of the obese is an environment rich in glycerol. Thus, it remains to be tested, whether GPD2-mediated mitochondrial metabolism of extracellular glycerol regulates T cell (hyper-) activation. Here, we present early results in favor of this hypothesis.

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[I-24] EXPLORING SMALL EXTRACELLULAR VESICLE-ASSOCIATED PROTEINS AS POTENTIAL BIOMARKERS OF CHRONIC KIDNEY DISEASE

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Introduction: The assessment of chronic kidney disease (CKD) relies largely on clinical metrics, including estimated glomerular filtration rate (eGFR), which is used to evaluate renal function. Nevertheless, there is still a need for molecular biomarkers that reflect disease-related mechanisms and enable more precise differentiation between CKD stages. Although mass spectrometry-based proteomics allows comprehensive analysis of serum proteins, its application is complicated by the wide concentration range of circulating proteins, particularly the predominance of highly abundant species. Small extracellular vesicles (sEVs) offer a promising alternative source of proteomic information, as they may carry proteins originating from specific tissues. Despite extensive research on serum and tissue proteomes in CKD, the potential of sEV-associated proteins as a source of disease-related biomarkers remains insufficiently investigated.

Materials and methods: Serum samples were obtained from three study groups comprising 10 healthy kidney donor candidates, 10 patients with stage G2-G4 chronic kidney disease, and 10 individuals with end-stage kidney disease receiving hemodialysis (CKD G5D). Samples were collected at the Medical University of Gdańsk and approved by the institutional ethics committee (approval no. KB/201/2024). Small extracellular vesicles were isolated by size-exclusion chromatography and characterised according to the MISEV2023 guidelines. The sEV proteome was then analysed using liquid chromatography-mass spectrometry (LC-MS/MS).

Results: Matched serum and sEV samples were analysed by LC-MS/MS to investigate their potential for distinguishing protein-level alterations associated with CKD. Quantitative proteomic profiling revealed 555 proteins in serum, of which 124 were found to be differentially abundant across the experimental groups. In sEV analysis identified 775 proteins, of which 119 showed differential abundance.

In serum, proteins identified as differentially abundant were mostly well-characterised circulating proteins, such as apolipoprotein A4, alpha-1-microglobulin, and serpin F2. Proteomic analysis of sEV revealed changes in alterations in sEV-associated transmembrane proteins that were not readily detectable in serum by MS. These included several integrins and intercellular adhesion molecule 1 (ICAM1), an integrin-binding protein involved in cell adhesion and signaling. Previous studies have documented changes in both integrins and ICAM1 in CKD, including in renal tissue.

Conclusions: These findings highlight the potential of sEV proteomics as a complementary approach for identifying molecular signatures of CKD.

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[I-25] FOXM1 ACTIVATION AND B-CATENIN SIGNALING REMODELING DRIVE CLEAR CELL RENAL CELL CARCINOMA PROGRESSION

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Keywords: ccRCC**,** Wnt/β-catenin, FOXM1, MCPIP1, molecular transition

Introduction Clear cell renal cell carcinoma (ccRCC) is the most common subtype (75%) of renal cancer and remains a major cause of mortality among patients with advanced kidney cancer. The highly vascularized tumor and its significant phenotypic diversity makes it so difficult to treat effectively. While the histological grade remains a crucial predictor of tumor aggressiveness and patient outcomes, the precise mechanisms driving the transition between these grades remain unknown. Identifying transcriptional changes across individual grades may therefore help to define events associated with the development of more aggressive disease. Dysregulation of Wnt/β-catenin signaling has been associated with aggressive tumor behavior and β-catenin activity depends not only on its abundance but also on its phosphorylation status and intracellular localization. Among transcriptional regulators associated with β-catenin signaling, FOXM1 has emerged as an important mediator of proliferation and tumor aggressiveness implicated in ccRCC progression.

Material and method To investigate the molecular mechanisms of ccRCC development and progression, we used *in vivo*, *in vitro* models and samples from patients. We conducted a comprehensive analysis of crucial genes and proteins in the Wnt/β-Catenin pathway using viral vectors, the Silencer siRNA system, Western blot, microarray analysis, quantitative PCR, immunofluorescence, and immunohistochemistry staining.

Results and discussion We analyzed the early molecular changes activated by the developing neoplastic process in ccRCC patient tissues and during tumor progression. Transcriptomic and biochemical analysis of 60 patient-derived tumor specimens showed the largest changes between Grade II and Grade III tumors. To map downstream transcriptional targets of activated β-catenin, we evaluated the expression profiles of genes associated with the CTNNB1 promoter encoding β-catenin. Microarray analysis identified FOXM1 as the transcriptional regulator most strongly and consistently associated with advanced disease. We further show that ccRCC progression is associated not with a uniform increase in β-catenin abundance but with changes in the activity and intracellular distribution of different β-catenin pools. In addition, we identified MCPIP1 as a negative regulator of progression-associated β-catenin signaling. MCPIP1 limited the accumulation of signaling-active β-catenin variants, reduced nuclear β-catenin localization, and suppressed the FOXM1-driven transcriptional program, whereas loss of MCPIP1 RNase activity promoted activation of the FOXM1/β-catenin axis.

Conclusion The progression of ccRCC is characterized by transcriptional alterations in which MCPIP1-dependent regulation of β-catenin modulates FOXM1-associated pathways characteristic of advanced disease stages.

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[I-26] SHARED TRANSCRIPTOMIC ALTERATIONS ASSOCIATED WITH EXTRACELLULAR MATRIX ORGANIZATION IN ENDOMETRIAL AND OVARIAN CANCER

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Introduction: Endometrial and ovarian cancers are major gynecological malignancies characterized by heterogeneous molecular alterations. Identifying shared transcriptional changes may provide insights into biological processes associated with these tumors.

Methods: Differential gene expression analysis was performed using GEPIA2 and transcriptomic data from TCGA/GTEx. Endometrial carcinoma (UCEC) and ovarian serous cystadenocarcinoma (OV) were analyzed separately using ANOVA, with thresholds of $|\log_2 FC| \geq 2$ and q-value ≤ 0.05. Genes showing concordant expression changes in both malignancies were selected for functional enrichment analysis using g:Profiler, Gene Ontology Biological Process, and Reactome.

Results: A total of 699 unique genes showing concordant differential expression were identified. Functional enrichment analysis revealed significant overrepresentation of the extracellular matrix organization pathway (Reactome; adjusted $p = 4.17 \times 10^{-14}$), involving 42 genes from the analyzed dataset. Five selected genes showed decreased expression in both malignancies: DCN ($\log_2 FC$: −5.254 in UCEC; −4.393 in OV), COL6A1 (−3.796; −2.963), COL6A2 (−3.843; −3.033), COL6A3 (−3.866; −2.634), and DDR2 (−3.513; −2.833).

Conclusions: The findings indicate shared transcriptional alterations involving extracellular matrix-associated genes in endometrial and ovarian cancers. These observations warrant further investigation, particularly regarding the contribution of tumor microenvironment composition to the observed expression patterns.

[I-27] GRADE-DEPENDENT DYSREGULATION OF OXIDATIVE STRESS-RELATED MICRORNAS IN DEGENERATIVE INTERVERTEBRAL DISC DISEASE

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Introduction: Oxidative stress is increasingly recognized as a key driver of intervertebral disc degeneration (IVDD), and post-transcriptional regulation by microRNAs (miRNAs) may modulate the underlying oxidative stress-related gene network. This study aimed to characterize the expression of oxidative stress-related miRNAs across degeneration grades and pain intensity in IVDD.

Material and Methods: The study included 200 patients with lumbosacral IVDD undergoing microdiscectomy and 100 postmortem control samples without spinal pathology. Degeneration severity was classified using the Pfirrmann scale (grade 2, n=32; grade 3, n=45; grade 4, n=117; grade 5, n=6), and pain intensity was assessed with the Visual Analog Scale (VAS 3–10). Candidate miRNAs targeting oxidative stress-related genes were identified in silico using the miRDB and miRanda platforms (confidence score >90) and subsequently validated experimentally by RT-qPCR, normalized to a small-RNA reference control.

Results: RT-qPCR validation confirmed significant, grade-dependent dysregulation of four oxidative stress-related miRNAs. hsa-miR-3163 and hsa-miR-196a-1-3p were consistently downregulated across all Pfirrmann grades relative to controls, with the lowest expression in grade 4, whereas hsa-miR-665-3p and hsa-miR-4686 were upregulated across all grades, peaking in grades 3–5. Correlation analysis showed that miRNA expression was more strongly related to structural degeneration than to pain: hsa-miR-4686 correlated positively with Pfirrmann grade ($\rho = 0.632$, $p < 0.001$), while hsa-miR-3163 ($\rho = -0.383$, $p < 0.001$) and hsa-miR-196a-1-3p ($\rho = -0.444$, $p < 0.001$) correlated negatively; these associations were confirmed by regression analysis (all $p < 0.001$). In contrast, associations with VAS were weak, reaching significance only for hsa-miR-196a-1-3p ($\rho = 0.147$, $p = 0.038$) and, on regression, for hsa-miR-665-3p ($\beta = -0.021$, $p = 0.048$).

Conclusions: Oxidative stress-related miRNAs, particularly hsa-miR-3163, hsa-miR-196a-1-3p, hsa-miR-665-3p and hsa-miR-4686, are significantly dysregulated in IVDD in a grade-dependent manner and correlate more strongly with structural degeneration severity than with subjective pain intensity, supporting their potential role as post-transcriptional regulators and biomarkers of disc degeneration.

[I-28]* THE EFFECT OF CO-CULTURING NORMAL AND CANCER INTESTINAL CELLS ON THE SPATIAL ORGANIZATION OF 3D SPHEROIDS

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Three-dimensional (3D) cell culture models allow for a more physiological recapitulation of tissue architecture and intercellular interactions compared to traditional 2D cultures. In this study, spheroids formed by the co-culture of the normal intestinal epithelial cell line CCD 841 CoN and the colorectal cancer cell line HCT116 were utilized and compared with spheroids composed exclusively of cancer cells.

Evaluations were conducted after 3 and 6 days of culture based on the morphological analysis of the structures and the expression of occludin (OCDN)—a key protein required for the proper functioning of tight junctions and the maintenance of epithelial barrier integrity. Distinct differences in the spatial organization of the spheroids and occludin expression levels were observed between the mixed and monoculture models. The presence of normal cells influenced the structural ordering of the spheroids as well as properties related to barrier function.

These findings indicate that the co-culture of distinct cell types within 3D models serves as a valuable research tool, enabling more realistic modeling of the tissue microenvironment and the analysis of interactions occurring between normal and neoplastic cells.

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Young Scientist poster session

[I-29]* ROLE OF IRON IN FERROPTOSIS PATHWAY IN KERATINOCYTE CELLS WITH GPX4 ENZYME KNOCKOUT

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Iron homeostasis and antioxidant defence are closely linked to cellular susceptibility to ferroptosis. This study investigated how GPX4 knockout affects iron homeostasis in HaCaT keratinocytes exposed to erastin.

Wild-type (WT) and CRISPR/Cas9-generated GPX4-knockout (KO) HaCaT cells were treated with 5 or 10 μM erastin for 24 h. Transcript levels of genes involved in iron uptake, storage, and export (TFRC, FTH, SLC40A1, and DMT1) were assessed by RT-qPCR. Reactive oxygen species levels were also evaluated. Cellular iron deposits were visualised using Prussian blue staining, and intracellular iron was additionally assessed using FerroOrange staining and confocal microscopy.

In WT cells, TFRC expression decreased with increasing erastin concentrations. In GPX4 KO cells, TFRC expression was not detected following treatment with 5 μM erastin but increased at 10 μM, alongside cellular iron accumulation. DMT1 expression decreased at both concentrations in both cell lines, with a less pronounced reduction in GPX4 KO cells. SLC40A1, encoding the iron exporter ferroportin, was upregulated in WT cells following erastin treatment but remained unchanged in GPX4 KO cells. GPX4 knockout altered the expression of iron-homeostasis markers without affecting cell viability under the conditions tested. IRP2 expression increased in WT cells but was suppressed in GPX4 KO cells, indicating distinct responses of the iron-regulatory system.

These findings suggest that GPX4 deficiency modifies the response of iron uptake and export pathways to erastin in HaCaT keratinocytes. The changes observed in GPX4 KO cells were associated with iron accumulation despite preserved viability. Further assessment of lipid peroxidation and rescue with ferroptosis inhibitors is needed to determine whether these alterations contribute to ferroptotic susceptibility.

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Young Scientist poster session

[I-30]* CELL STATE CLASSITICATION DURING FERROPTOSIS INDUCTION USING A DECISION TABEL

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Ferroptosis is a specific type of regulated cell death that is strictly dependent on iron levels and characterized by the massive accumulation of reactive oxygen species (ROS) and membrane lipid peroxidation. Understanding the complex regulatory mechanisms underlying this process is of crucial importance in cell biology research, oncology, and the analysis of therapy resistance phenomena. The main goal of this study was to develop and preliminarily validate a decision-making model based on a network of genes and redox state indicators, designed for the precise classification of cellular condition under escalating oxidative stress and ferroptosis induction. The research focused primarily on key cellular antioxidant defense mechanisms as well as enzymatic and non-enzymatic sources of reactive oxygen species generation. The analyzed network incorporated, among others, cascades responsible for the reduction of lipid hydroperoxides, transmembrane transport systems critical for the synthesis of intracellular redox buffers, and master transcription factors coordinating the stress response. Furthermore, the model included reductive pathway enzymes, superoxide dismutases neutralizing free radicals, the total pool of low-molecular-weight antioxidants, and the activity of ROS-generating oxidases. The decision table methodology was employed to construct the decision rules. This approach enables clear and targeted mapping of complex cellular states in response to varying doses of ferroptosis-inducing agents and allows for structuring continuous biological measurements into a system of decision levels. The obtained results indicate that the expression patterns of genes involved in maintaining redox homeostasis exhibit non-linear relationships with the level of oxidative stress, enabling effective classification and differentiation of death-sensitive cells from those activating protective mechanisms. The developed decision model serves as a flexible analytical tool with high application potential, supporting the prediction of cancer cell responses to pro-oxidant treatments and deepening the understanding of redox network dynamics.

Young Scientist poster session

[I-31]* USE OF THE BODIPY™ C11 PROBE TO ASSESS LIPID PEROXIDATION IN CELLS

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Lipid peroxidation is the process of oxidation of the lipid components of cell membranes, associated with the action of reactive oxygen species and an imbalance in the cell's oxidative-antioxidant balance. Excessive lipid peroxidation can lead to membrane damage and plays a significant role in ferroptois, a regulated form of iron-dependent cell death. Assessing lipid peroxidation is therefore a key component of research into oxidative stress and the mechanisms of ferroptois. One method used to monitor this process is the BODIPY™ C11 fluorescent probe, whose spectral properties change upon oxidation. When combined with confocal microscopy, it enables the visualization of changes occurring within cells.

The aim of this study is to analyze the literature on the use of the BODIPY™ C11 probe to assess lipid peroxidation in cells and to discuss the potential applications of this method in ferroptoxis research. Particular attention was paid to the use of erastin, which induces ferroptosis by disrupting the cystine transport system, and ferrostatine, which inhibits lipid peroxidation and limits the processes associated with this form of cell death.

This poster discusses the fundamentals of the lipid peroxidation mechanism, the significance of reactive oxygen species, and the role of lipid oxidation in ferroptosis. It also presents the properties of the BODIPY™ C11 probe and the potential applications of confocal microscopy for detecting changes in fluorescence. The analysis was related to planned experimental studies using two cell lines: Caco-2, derived from colorectal adenocarcinoma, and CCD 841 CoN, isolated from normal colon tissue. The experiments will involve comparing control cells with cells treated with erastin, ferrostatin, and a mixture of both compounds, followed by staining with the BODIPY™ C11 probe. Microscopic imaging is scheduled for 1 and 24 hours after treatment to allow for the assessment of changes over time.

A review of the literature indicates that BODIPY™ C11 can be used to monitor lipid peroxidation in living cells, and confocal imaging allows for the recording and analysis of changes in the fluorescence signal. The use of this probe, in combination with appropriately selected experimental conditions, makes it possible to compare changes in lipid oxidation levels across different cell treatment conditions. This literature review provides the theoretical basis for interpreting the results of planned experiments and evaluating the suitability of this method for studies on processes related to ferroptosis.

Young Scientist poster session

[I-32]* EXPRESSION OF TIGHT JUNCTION GENES IN 2D AND 3D CELL CULTURES UNDER OXIDATIVE STRESS

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Spheroids are three-dimensional cellular aggregates that form in an environment preventing cell adhesion to the substrate. Their structure and functionality faithfully reflect the in vivo tissue environment. Unlike 2D cultures, 3D models are characterized by a high level of cell-cell and cell-extracellular matrix interactions. Furthermore, physiological gradients of nutrient and oxygen concentrations develop within spheroids.

Classic two-dimensional cultures force cells to grow as a flat monolayer, which forces changes in their metabolism and gene expression. As a result, 2D cells exhibit greater sensitivity to external substances, making them a less reliable research model compared to three-dimensional structures.

The aim of the study was to compare the cellular response to oxidative stress in two culture models: a classic 2D culture and 3D spheroids. HCT116 cell line cultures were conducted in parallel and treated with erastin to induce oxidative stress. Material for analysis was collected at three time points: at 1, 6 and 24 hours after exposure. Subsequently, the expression level of selected genes associated with tight junctions was examined. This study aimed to determine the mechanisms by which tight junctions proteins participate in the signal transduction associated with oxidative stress, as well as to investigate their role in the formation and stabilization of complex three-dimensional structures.

The study proved that 3D models are more resistant to stress factors, correlating with the higher complexity of such systems.

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Young Scientist poster session

[I-33]* DYNAMICS OF FERROPTOSIS AND FERRITINOPHAGY MARKER GENE EXPRESSION IN CELLULAR MODELS

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Ferroptosis is an iron-dependent form of regulated cell death characterized by the accumulation of lipid peroxides in cellular membranes. Ferritinophagy, the lysosomal degradation of ferritin, a major intracellular iron-storage protein, may contribute to ferroptosis by increasing the labile iron pool, thereby facilitating lipid peroxidation. However, the temporal patterns of expression of genes associated with these processes, and the extent to which these patterns vary across cellular models, remain insufficiently understood.

This study aimed to characterize the temporal changes in the expression of selected genes associated with ferroptosis and ferritinophagy in HCT116 cells, following treatment with erastin, a commonly used ferroptosis inducer. Samples were collected at selected time points after the treatment. RNA was subsequently extracted, and gene expression was assessed using reverse-transcription quantitative PCR (RT-qPCR).

The preliminary results indicate time-dependent changes in the expression of investigated genes following erastin treatment, highlighting potential differences in their transcriptional dynamics. Overall, this study may contribute to a better understanding of the molecular links between iron homeostasis, redox imbalance, and the regulation of ferroptosis-associated processes.

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Young Scientist poster session

[I-34]* EFFECTS OF GLIADIN ON TIGHT JUNCTION ORGANISATION IN A THREE-DIMENSIONAL INTESTINAL EPITHELIAL MODEL

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Intestinal barrier integrity depends on the proper organisation of tight junctions between epithelial cells. Alterations in the structure and localisation of tight junction proteins may contribute to increased epithelial permeability and inflammatory responses. Three-dimensional organoid cultures provide a platform for investigating these processes in a spatial context and modelling selected mechanisms of intestinal barrier damage relevant to coeliac disease.

This project aims to establish and characterise a 3D intestinal organoid model to assess the effects of gliadin on tight junction organisation, epithelial morphology and ultrastructure. The study will include optimisation of culture conditions and gliadin exposure, followed by comparisons between exposed organoids and untreated controls. The distribution of tight junction proteins, including ZO-1, occludin and claudins, will be assessed by immunofluorescence using confocal microscopy. Morphological and ultrastructural changes will be examined using transmission electron microscopy (TEM) and serial block-face scanning electron microscopy (SBF-SEM). Imaging analyses will be complemented by RT-qPCR assessment of selected genes involved in epithelial barrier organisation and inflammatory responses.

Integration of imaging and molecular data will help determine whether gliadin exposure is associated with changes in intercellular junction organisation and the architecture of 3D structures. The resulting model may provide a foundation for further studies of the epithelial effects of gliadin and intestinal barrier dysfunction. Conducted within a project-based learning (PBL) framework, the project combines cell biology, bioimaging and data analysis while developing students' research skills through collaboration between the Silesian University of Technology and the University of Silesia.

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Young Scientist poster session

[I-35]* INVESTIGATION OF THE FORMATION OF ACTIVE REGULATORS FOR NUCLEAR SIGNALIZATION USING SYNTHETIC PROXIMITY-BASED SYSTEMS

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Studying protein biosynthesis requires an understanding of the process, which is characterized by complex regulatory mechanisms. Translation involves the formation of multi-component complexes, specifically those composed of multiple protein subunits, whose production and delivery to appropriate cellular compartments are governed by feedback loops, whether positive or negative. While ribosomes serve as the site of translation, full initiation of the process requires additional elements, such as protein translation factors, messenger RNA (mRNA), polymerases, metal ion cofactors, and the amino acids necessary for synthesizing new polypeptide chains. Although translation is subject to multi-level regulation, research has increasingly focused on the inhibition of this process, particularly following the discovery of small, 22-nucleotide miRNAs. Translation can be inhibited by blocking the assembly of the ribosomal complex (e.g., through the inhibition of initiation sites, a mechanism employed by certain naturally occurring toxins), by degrading mRNA (via cleavage by specific nucleases), or through the formation of the RNA-induced silencing complex (RISC). This multi-protein system incorporates miRNA and nuclease-like enzymatic activities; by interacting with mRNA through interference, it forms a structurally and spatially complex hybrid, a miRNA:mRNA:protein assembly, within the RISC complex. This triggers a cascade of events leading to the reversible or permanent inhibition of translation. Translation inhibition itself underlies various physiological processes, such as tissue remodeling during embryogenesis or the suppression of protein production, effectively silencing expression, to regulate basal metabolism. Conversely, dysregulation of translation contributes to pathological changes that underpin numerous diseases, including tumorigenesis. Research into translation inhibition requires first inducing the process and subsequently identifying all components of the RISC silencing complex using advanced in vitro molecular techniques. One of the latest methods is the Proximity Ligation Assay (PLA) system, which enables the simultaneous observation of multiple protein complexes, and their formation within cells without the need for cell destruction, prior fixation, or labeling. These studies can be conducted *in vivo* (or in live-cell cultures) in real time, allowing for the simultaneous qualitative and quantitative detection of specific generated signals.

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Young Scientist poster session

[I-36]* ESTABLISHMENT AND CHARACTERISATION OF THREE-DIMENSIONAL GLIOMA MODELS DERIVED FROM CELL LINES

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Three-dimensional (3D) cell cultures enable the investigation of tumour cell behaviour in a spatial context that cannot be fully reproduced in conventional monolayer cultures. Glioma spheroids provide an experimental platform for studying the formation, growth and maintenance of multicellular tumour structures.

This project aims to establish and characterise 3D glioma models derived from the human U-87 and LN-229 cell lines. The study focuses on spheroid formation, morphological development, growth, structural stability and cell viability during culture under standard conditions.

Microscopic monitoring will be used to evaluate spheroid size, shape, compactness and integrity at successive time points. Viability measurements will complement morphological assessment, allowing the identification of culture conditions that support the maintenance of viable 3D structures. Comparative analysis of the two cell lines will assess potential differences in their capacity to form and sustain spheroids and support the development of a reproducible experimental protocol.

The resulting models are intended to provide a foundation for subsequent studies of glioma biology and responses to experimental treatments. Conducted within a project-based learning framework, the study also integrates practical training in cell culture, microscopy, quantitative image analysis and the interpretation of experimental data.

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Young Scientist poster session

[I-37]* Analysis of Cancer Cell Responses to Regulated Cell Death in 2D and 3D Models Using Cell Imaging

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Regulated cell death (RCD) plays an essential role in maintaining organismal homeostasis, while dysregulation of its mechanisms may contribute to cancer development and progression. Ferroptosis has attracted particular research interest as an iron-dependent form of cell death associated with increased lipid peroxidation and disruption of cellular redox homeostasis. Understanding the molecular mechanisms underlying this process and the roles of the proteins involved may provide further insight into cancer cell responses to RCD-inducing factors and help identify potential directions for the development of anticancer strategies.

The selection of an appropriate cellular model is another important aspect of cancer research. Although widely used, two-dimensional (2D) cultures do not fully reproduce tissue architecture, cell–cell interactions, or the heterogeneity of the tumor microenvironment. Three-dimensional (3D) models, including spheroids, better reproduce selected features of tumor organization, which may influence cellular responses to the induction of regulated cell death.

This study aims to review current knowledge of the molecular mechanisms underlying RCD, with particular emphasis on ferroptosis, and to discuss the relevance of 2D and 3D culture models for investigating cancer cell responses. The review also addresses approaches to identifying and analyzing protein markers associated with these processes using molecular biology techniques, immunofluorescence microscopy, and bioinformatics tools.

The review provides a scientific basis for a research project involving the analysis of data from previous experiments and the comparison of selected protein expression profiles in 2D and 3D models. Planned activities include validation of selected markers using Western blotting with densitometric analysis and immunofluorescence microscopy. Bioinformatic analyses will complement the experimental work by identifying potential regulatory nodes within RCD pathways and examining the relationship between model architecture and cellular response profiles. The results are expected to provide a basis for further investigations into the mechanisms of regulated cell death and the applicability of three-dimensional models in cancer biology research.

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[I-38]* mRNA Expression of Ferroptosis-Suppressive Genes Across Breast Cancer Subtypes and Their Systemic Reflection After Cryoablation

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Introduction: Ferroptosis, an iron-dependent form of regulated cell death, is increasingly implicated in breast cancer biology, but its transcriptional regulation across molecular subtypes remains incompletely characterized. This study evaluated mRNA expression of ferroptosis-suppressive genes in breast cancer tissue by microarray with RT-qPCR validation, and assessed whether these changes are mirrored systemically in peripheral blood following localized tissue injury.

Material and Methods: Tumor tissue and matched non-neoplastic tissue were obtained from 405 breast cancer patients across five molecular subtypes: luminal A (n=130), luminal B HER2− (n=100), luminal B HER2+ (n=96), non-luminal HER2+ (n=36) and triple-negative breast cancer (TNBC, n=43). Genome-wide mRNA profiling was performed by microarray (HG-U133_A2, Affymetrix), and six subtype-independent ferroptosis-related genes (SLC7A11, GPX4, FTH1, NQO1, NFE2L2, SQSTM1) were validated by RT-qPCR. In an independent cohort of 34 patients with breast fibroadenoma undergoing cryoablation, peripheral blood mRNA levels of the same six genes were measured by RT-qPCR before the procedure and at 30–60 minutes, 8–12 hours, 48–72 hours, 7 days, 1 month and 3 months afterward.

Results: All six genes were significantly upregulated at the mRNA level in tumor tissue across every subtype relative to control (all $p < 0.05$; log2FC ranges: SLC7A11 +3.42 to +4.76, GPX4 +3.18 to +4.21, FTH1 +3.21 to +4.39, NQO1 +3.32 to +4.51, NFE2L2 +3.02 to +4.11, SQSTM1 +3.21 to +4.43, from luminal A to TNBC), with RT-qPCR confirming the direction and magnitude of these changes and the highest expression consistently seen in non-luminal HER2+ and TNBC tumors. Following fibroadenoma cryoablation, peripheral blood mRNA levels of the same six genes rose transiently, peaking at 8–12 hours post-procedure (fold-change 1.66–2.03 vs baseline, all $p < 0.05$), remained significantly elevated at 48–72 hours, and returned to baseline by 7 days to 3 months.

Conclusions: A core panel of six ferroptosis-suppressive genes is consistently upregulated at the mRNA level across all breast cancer molecular subtypes, with the highest expression in the most aggressive subtypes, and is transiently and reproducibly mirrored in peripheral blood after localized tissue injury, supporting its potential as an RT-qPCR-based biomarker panel for ferroptosis-related tumor and systemic responses.

Young Scientist poster session

[I-39]* REDOX BALANCE AND SUBGROUP-DEPENDENT VARIABILITY DURING CYCLOSPORINE A THERAPY IN PSORIASIS VULGARIS

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Introduction: Oxidative stress contributes to the pathogenesis of psoriasis vulgaris, and cyclosporine A (CsA) therapy may restore systemic redox balance, but whether this effect is uniform across comorbidity subgroups and predicts clinical response is unclear. This study assessed redox balance dynamics during CsA therapy, their modulation by smoking, diabetes and body mass index (BMI), and their relationship with clinical response.

Material and Methods: In a prospective cohort of 37 adults with moderate-to-severe psoriasis vulgaris receiving 12-week CsA therapy, the serum copper-to-zinc (Cu/Zn) ratio, total antioxidant status (TAS), total oxidant status (TOS) and oxidative stress index (OSI=TOS/TAS×100) were assessed spectrophotometrically at baseline, day 42 and day 84 and analyzed by repeated-measures ANOVA with Tukey post-hoc testing. Serum element concentrations were additionally compared by smoking status, diabetes and BMI category at each timepoint. Patients achieving PASI75 at day 84 (responders, 29/37, 78.4%) were compared with non-responders (8/37, 21.6%) for redox trajectories.

Results: The Cu/Zn ratio decreased progressively from 2.87 to 1.78 to 0.89 ($p<0.0001$). TAS increased from 0.85 to 1.05 to 1.32 mmol/L ($p<0.0001$; day 0 vs day 42 $p=0.009$, day 0 vs day 84 $p<0.001$), while TOS decreased from 28.0 to 20.0 to 14.0 µmol H2O2 equivalent/L ($p<0.001$; day 0 vs day 84 $p=0.003$) and OSI fell from 3294.1 to 1904.8 to 1060.6 ($p<0.0001$; day 0 vs day 42 $p=0.014$, day 0 vs day 84 $p<0.001$). Element concentrations (notably Fe, Mg, Ca, Na and K) differed significantly by smoking status, diabetes and BMI category at nearly every timepoint ($p<0.0001$ for most comparisons), whereas Cu and Zn differences were smaller and less consistent; smokers, diabetic and obese patients showed greater baseline abnormalities and more variable normalization, with obese patients maintaining persistently higher Cu. Responders showed a greater OSI reduction than non-responders (3311→972, -70.6% vs 3235→1479, -54.3%; $p=0.049$) and a larger TAS increase (0.84±0.15→1.39±0.21 vs 0.87±0.16→1.16±0.19 mmol/L; $p=0.033$).

Conclusions: CsA therapy induces a substantial improvement in systemic redox balance in psoriasis vulgaris, with the magnitude of biochemical normalization modulated by smoking, diabetes and obesity and closely linked to clinical response, supporting redox indices as candidate mechanistic biomarkers of treatment efficacy.

Young Scientist poster session

Session II: Disease OMICS

[II-1] MicroRNAs Potentially Regulating UBA2, GLO1, STATH and TUFT1 in Ovarian Cancer and Drug Resistance

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Introduction: Drug resistance remains a major obstacle in the management of ovarian cancer. The genes UBA2, GLO1, STATH and TUFT1 have been implicated in this process, but the microRNAs (miRNAs) potentially regulating their expression have not been systematically characterized. This study evaluated candidate regulatory miRNAs for these four genes in ovarian cancer.

Material and Methods: The study included 98 women: group A (n=36; surgery plus first-line chemotherapy, stage I–IV ovarian cancer), group B (n=12; surgery alone, type I ovarian cancer) and a control group C (n=50; surgery for non-oncological indications). Candidate miRNAs targeting UBA2, GLO1, STATH and TUFT1 were identified *in silico* using the miRDB and miRanda databases and ranked by target score; their expression in tumour versus control tissue was assessed using the GeneChip miRNA 2.0 microarray, in parallel with mRNA (RT-qPCR) and protein (ELISA) analysis of the same genes.

Results: Bioinformatic prediction identified hsa-miR-133a-3p (target score 98) and hsa-miR-133b (98) for UBA2; hsa-miR-561-5p (90) for GLO1; hsa-miR-137-3p (97) and hsa-miR-580-3p (80) for STATH; and hsa-miR-1233-3p (86) and hsa-miR-2052 (94) for TUFT1. Six of these seven miRNAs were significantly overexpressed in ovarian cancer tissue relative to controls ($p < 0.05$), with a consistent direction of change in both group A and group B; only hsa-miR-561-5p was significantly downregulated in both groups. Integrated mRNA-miRNA-protein analysis showed that for UBA2, GLO1 and STATH the direction of miRNA change accompanied concordant changes at both mRNA and protein level in groups A and B versus controls, whereas TUFT1 showed a discordant pattern in group B, with protein and miRNA overexpressed but mRNA downregulated.

Conclusions: A consistent panel of six overexpressed miRNAs (miR-133a-3p, miR-133b, miR-137-3p, miR-580-3p, miR-1233-3p, miR-2052) and one downregulated miRNA (miR-561-5p) potentially regulates the drug-resistance-associated genes UBA2, GLO1, STATH and TUFT1 in ovarian cancer, largely irrespective of treatment extent, supporting their role as candidate post-transcriptional regulators and biomarkers in ovarian cancer.

[II-2] EXPLORING EMPAGLIFLOZIN-MEDIATED MODULATION OF ISCHEMIC RESPONSES

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Ischemic stroke (IS) remains a major cause of mortality and long-term disability worldwide. Population ageing further increases its burden, highlighting the need for effective therapeutic strategies. Current treatment focuses on rapid restoration of cerebral blood flow through intravenous thrombolysis and/or mechanical thrombectomy. However, substantial neurological impairment may persist despite reperfusion, underscoring the need for complementary neuroprotective approaches.

Sodium-glucose cotransporter 2 (SGLT2) inhibitors, commonly known as flozins, were initially developed for the treatment of type 2 diabetes and subsequently gained indications in cardiovascular and renal disease. Growing preclinical evidence also suggests their potential relevance to central nervous system disorders, including ischemic brain injury.

This study combined two complementary approaches to investigate the potential of empagliflozin to modulate molecular responses associated with ischemia. First, publicly available transcriptomic data from the Gene Expression Omnibus (GEO) and ArrayExpress repositories were examined for treatment-associated changes in genes annotated to the Gene Ontology biological process "response to ischemia" (GO:0002931). Second, selected molecular targets were evaluated in samples obtained from our in vitro ischemia model, allowing comparison with the transcriptomic findings.

The transcriptomic analyses identified significant empagliflozin-associated changes in several ischemia-response genes, with substantial variation across experimental settings. ANGPTL4, EGR1, and DDAH1 were significant in the overall EMPA analysis. Subgroup analyses identified EGR1 and DDAH1 in vitro, and ANGPTL4, ALKBH5, and HIPK2 in vivo. Tissue-specific analyses additionally identified ANGPTL4 and CXCL12 in cardiac datasets, ANGPTL4 in kidney datasets, and VEGFA in vascular datasets. These findings suggest that empagliflozin-associated modulation of ischemia-related gene expression depends on tissue and experimental context, rather than following a uniform pattern across models. The identified genes provide candidates for further experimental investigation to determine whether these molecular changes contribute to reduced ischemic injury.

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[II-3] COMPREHENSIVE INVESTIGATION OF TUMOR RENEWAL-ASSOCIATED FEATURES IN FUSION-POSITIVE RHABDOMYOSARCOMA USING SPATIAL TRANSCRIPTOMICS AND SNAI1 GENE-FOCUSED ANALYSIS

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Recurrence of fusion-positive rhabdomyosarcoma (FPRMS) is associated with poor prognosis, highlighting the need for therapies selectively targeting tumor renewal-related genes or cell types. Recent single-cell RNA-seq studies identified FPRMS cell states resembling muscle development stages ("FP-progenitor", "proliferative" and "differentiated") and the "ground" state with no clear expression pattern. Interestingly, a "neuronal" cell state was also observed. "FP-progenitor", "differentiated" and "neuronal" were proposed as contributing to recurrence. Likewise, our recent analysis of bulk RNA-seq dataset showed upregulation of "neuronal" state genes in recurrent FPRMS cases. SNAI1 transcription factor and membrane proteins VANGL2, FGFR3, CD44, MET, CXCR4 and CD133 have been previously suggested as linked to cancer stem cells (CSC) which, canonically, give rise to recurrent tumors.

Our goal was to identify and map FPRMS cell states in patient-derived xenograft (PDX) tumors using spatial transcriptomics. By analyzing the expression of potential CSC markers, we aimed to indicate cell states associated with relapse. Finally, we intended to evaluate the relationship between SNAI1, the expression of other potential CSC markers, and tumor renewal capacity.

We used 12 FPRMS PDX samples and the RH30 cell line. Orthotopic xenografts were established in NSG mice. Spatial transcriptomics was performed on 4 PDX tumors using 10X Genomics Visium HD. Annotation was based on published cell state marker genes and visual assessment of spatial distribution of the clusters. SNAI1 was silenced in RH30 cells using shRNA lentiviral vectors. Expression of proposed CSC surface markers was measured by flow cytometry. Tumor renewal capacity was assessed as time to engraftment.

Visium HD analysis identified cell states that, according to their expression patterns and spatial distribution, could be best described as "fibroblast-like", "muscle-like", "neuronal", "proliferative" and "ground". "Fibroblast-like" cells localized along the fibrovascular septa. "Muscle-like" cells grouped together, forming distinct patches on a tissue section, surrounded by "ground" and "proliferative" areas. "Neuronal" cells were scarce in 3 samples, while dominant in one, where they localized towards the outer parts of the tumor. All analyzed CSC markers were upregulated in "muscle-like" state. SNAI1, VANGL2 and CD44 were upregulated in "neuronal", while FGFR3, CXCR4, MET and CD133 - in "ground" cells. SNAI1 silencing in RH30 cells increased CD44 expression, reduced FGFR3 expression, and significantly impaired the engraftment capacity.

We demonstrate the spatial hierarchy of FPRMS cell states and further support "muscle-like" and "neuronal" as associated with recurrence. Our results also support SNAI1 as a potential therapy target.

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[II-4] SPATIAL REMODELING OF THE TUMOR-IMMUNE MICROENVIRONMENT BY NEOADJUVANT CHEMORADIOTHERAPY IN NSCLC

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The combination of chemoradiotherapy and immune checkpoint inhibition (ICI) in unresectable stage III NSCLC yields markedly divergent outcomes depending on treatment sequencing: consolidation durvalumab following chemoradiotherapy confers significant survival benefits, whereas concurrent administration fails to improve progression-free or overall survival. This paradox implicates radiation-induced remodeling of the tumor-immune microenvironment as a key, yet poorly understood, determinant of immunotherapy response.

We applied Opal multiplex immunofluorescence (AKOYA Biosciences) to resected NSCLC following neoadjuvant chemotherapy (CHT, n=16), chemoradiotherapy (CRT, n=22), or primary resection (n=10), with matched pre-treatment biopsies in a subset. A six-plex panel (CD8, CD4, FOXP3, CD20, CD68, PanCK) was quantified in QuPath to profile the immune composition across tissue compartments and classify the tumor ecosystem by KNN into inflamed, excluded, and cold phenotypes. In a subset of samples from 18 patients (6 PR, 6 CT and 6 CRT, matched for clinical characteristics) we performed mIF and matched spatial transcriptomics (Visium) on adjacent FFPE slide, registered via hull-based rigid registration in 36 tissue microarray cores (2 cores/sample).

CRT-treated tumors showed selective depletion of CD20+ B cells at the invasive margin. FOXP3+ regulatory T cell density also differed significantly across treatment groups ($p=0.02$), while CD8+ effector infiltration was preserved following CRT and CHT. After quality control, 160,000 cells from 27 cores (mIF) and 4,083 spots from 31 cores were available for downstream analysis. Integration with spatial transcriptomics revealed treatment-specific decoupling of immune activation and spatial engagement: in CT cores, IFNγ pathway scores correlated negatively with with mIF-derived CD8-to-cancer cell distance ($R=-0.86$, $p=0.024$), while in CRT cores the correlation was inverse (Spearman $R=0.73$, $p=0.031$). These findings suggest cytotoxic treatment preferentially remodels humoral and immunoregulatory compartments at the tumor-stroma interface, revealing spatial immune barriers relevant to subsequent immunotherapy response.

[II-5] dSTRIDE-RAD51 REVEALS GENOTYPE-SPECIFIC MICRORNA CORRELATES OF HOMOLOGOUS RECOMBINATION ACTIVITY IN BREAST TISSUE AND PLASMA

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Background: Germline pathogenic variants impairing homologous recombination (HR) may alter the repair of DNA double-strand breaks (DSBs) in breast tissue. We examined RAD51 engagement at DSB sites in situ, measured by dSTRIDE-RAD51, and its association with tissue and circulating microRNAs (miRNAs) in mutation carriers (MT) and wild-type women (WT).

Methods: dSTRIDE-RAD51 foci per nucleus were quantified on tissue microarrays and normalized to the MDA-MB-436 control (MT: 23 women, 34 cores, including 16 healthy carriers and 7 carriers with cancer contributing tumor, tumour-adjacent and tumour-distant cores; WT: 12 women, 17 cores). Small RNA sequencing was performed on matched FFPE tissue and on plasma of carriers (21 women). Associations were tested with partial Spearman correlation adjusted for tissue type (plasma: disease status and sample batch) and linear mixed models with patient as random effect, including a dSTRIDE-RAD51 × genotype interaction. Correlations are reported as partial Spearman coefficients (r).

Results: In MT, dSTRIDE-RAD51 differed between tissue types ($p = 0.006$) and was highest in tumors (1.70-fold vs tumour-distant, $p = 0.005$; 1.51-fold vs healthy, $p = 0.03$), whereas no tissue effect was observed in WT ($p = 0.45$). Tumors of carriers showed 1.97-fold higher dSTRIDE-RAD51 than WT tumors ($p < 0.001$; genotype × tissue interaction $p = 0.008$). In healthy breast tissue, dSTRIDE-RAD51 did not differ between carriers and WT women (1.23-fold, $p = 0.19$). In FFPE tissue, miR-125b-1-3p correlated positively with dSTRIDE-RAD51 in both genotypes (WT $r = 0.64$, MT $r = 0.39$; pooled model $p < 0.001$). Two other miR-125 family members (miR-125a-5p, miR-125b-2-3p) showed the same direction in both genotypes. miR-1180-3p was the strongest carrier-associated miRNA ($r = 0.55$; 2.1-fold change per doubling of dSTRIDE-RAD51, $p = 0.01$), and miR-941 and miR-149-5p showed significantly stronger associations in MT than in WT (interaction $p < 0.05$). In WT, miR-574-3p ($r = 0.83$) and miR-320a-3p ($r = 0.78$) passed FDR < 0.05, and miR-214-3p ($r = 0.79$) and miR-27a-5p ($r = 0.68$) were also WT-specific. In carriers, miR-126-3p, miR-338-3p and miR-335-5p were inversely associated with dSTRIDE-RAD51 in both tissue ($r = -0.43$ to -0.46) and plasma ($r = -0.52$, $p = 0.02$). This agrees with known HR-suppressive functions of miR-335 (targeting the resection factor CtIP) and miR-139-5p (repressing DNA repair networks; tissue $r = -0.49$).

Conclusions: Breast tumors of mutation carriers show increased RAD51 engagement at DSBs. miR-1180-3p and miR-125b-1-3p emerged as tissue correlates of this HR activity, while miR-126-3p, miR-338-3p and miR-335-5p are candidate circulating markers. These exploratory findings require validation in larger, independent cohorts.

[II-6] FATTY ACID METABOLISM AS POTENTIAL METABOLIC DRIVER OF T-ALL RELAPSE: INSIGHTS FROM BULK AND SINGLE-CELL RNA-SEQ

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T-cell acute lymphoblastic leukemia (T-ALL) is a subtype of the most prevalent pediatric malignancy called acute lymphoblastic leukemia (ALL). T-ALL arises from immature T-cells in thymus. T-ALL accounts for approximately 15% of pediatric ALL cases. Current T-ALL treatment relies on intensive multi-drug chemotherapy with often severe side effects, yet fails in approximately 20% of children due to resistance or relapse, which accounts for most T-ALL-related deaths. Unraveling molecular complexity of T-ALL relapse pose a scientific problem and an unmet need in pediatric oncology. Uncovering the mechanisms governing the 'fitness' of T-ALL cells, especially, the mechanisms which can be therapeutically exploited, is of particular interest. The emergence of single-cell RNA sequencing, allows for more in-depth studies of the mechanism of relapse.

We generated a transcriptomic datasets of T-ALL pediatric patients including bulk mRNA-seq of 87 diagnostic T-ALL samples, stratified by disease recurrence status, and scRNA-seq of 9 T-ALL pediatric patients, with paired diagnosis and relapse samples from 5 of them. Identification of differentially expressed genes (DEGs), followed by QL-F test based gene set enrichment analysis (GSEA) of both datasets, using the following databases: KEGG and MSigDB consistently showed involvement of DEGs in cellular metabolic pathways, especially regarding fatty acid (FA) metabolism.

Our bulk mRNA-seq data analyses suggest that diagnostic samples of patients who later experience relapse are characterized by distinct FA metabolic phenotype. With potential increased uptake of fatty acid and concurrent depletion of pathways related to FA catabolism, potentially leading to esterification of FA and subsequent storage in lipid droplets, serving as energy reservoirs.

Further analyses of scRNA-seq data reveal that T-ALL relapse may be closely dependent on intensification of catabolism of FA. GSEA results indicate an enrichment of pathways related to mitochondrial and peroxisomal oxidation of FA, with concurrent enrichment of pathways related to autophagy of membranous organelles and lipophagy, potentially to meet the increased FA demand.

So far, metabolic studies of T-ALL have primarily focused on oxidative phosphorylation and glutamine metabolism, both regulated by NOTCH1 signaling, whose activating mutations are common in this malignancy. Another central metabolic regulator, mTOR, integrates signals with nutrient availability and controls metabolic processes including lipid metabolism. Interestingly, mTOR signaling, which is also commonly altered in T-ALL (evident also in our dataset), regulates FA metabolism. Together, our findings challenge current approaches to T-ALL metabolic studies and highlight a potential role of fatty acid metabolism for relapse development.

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[II-7] STEPWISE MIRNA CHANGES ACROSS THE ENDOMETRIUM, ENDOMETRIOSIS AND ENDOMETRIOSIS ASSOCIATED OVARIAN CANCER

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Introduction: Endometriosis is a precursor of endometrioid (EOC) and clear cell ovarian carcinoma (CCOC), but miRNA changes associated with malignant transformation remain poorly characterised. We profiled miRNAs across benign, endometriotic and malignant tissues and validated findings in independent datasets.

Methods: Small RNA sequencing was performed on 83 tissues: fallopian tube (FT, n=12), healthy endometrium (EH, n=13), ovarian endometrioma (OE, n=12), peritoneal (PE, n=7) and deep infiltrating endometriosis (DIE, n=11), EOC (n=10), CCOC (n=10) and high-grade serous carcinoma (HGSOC, n=8). Host and target genes were assessed by random-effects network meta-analysis of 10 GEO datasets (698 samples: DIE 88, EEM 109, PE 76, OE 40, EH 44, CCOC 65, EOC 91, HGSOC 163, FT 22) using netmeta.

Results: Endometriotic lesions gained miR-21-5p (OE vs EH: log2FC 1.71, FDR<0.001) and miR-146a-5p (log2FC 1.94, FDR=0.002), whose targets were enriched in interferon-γ, TNFα/NF-κB and Toll-like receptor signalling and epithelial–mesenchymal transition. Conversely, miR-503-5p decreased across OE, PE and DIE (log2FC −2.08 to −2.29, FDR<0.01), alongside miR-10b-5p and miR-130b-5p, implicating PI3K, insulin and IGF1 receptor signalling. The imprinted DLK1–DIO3 cluster was upregulated across endometriosis subtypes, particularly PE (41/69 miRNAs, FDR=9×10^{-28}).

Compared with endometriosis, EOC and CCOC gained the epithelial miR-200 family (miR-200b-3p: EOC vs OE, log2FC 4.60, FDR=2×10^{-11}) and miR-183/182/96 cluster (miR-182-5p: log2FC 4.56, FDR=2×10^{-11}), alongside miR-7-5p and miR-203a-3p, associated with VEGF signalling and actin cytoskeleton regulation. Carcinomas lost stromal miR-143-3p (EOC vs DIE: log2FC −2.95, FDR=1.5×10^{-5}), miR-145-5p and miR-125b-5p, targeting PI3K–AKT/mTOR pathways. Losses also involved DLK1–DIO3 and H19/IGF2 loci, including miR-675-3p (log2FC −2.48, FDR=0.005). miR-885-5p was selectively elevated in CCOC (vs OE: log2FC 6.86, FDR=2.5×10^{-12}), whereas miR-34b/c loss was restricted to HGSOC (miR-34c-5p vs FT: log2FC −4.92, FDR=2.7×10^{-7}). Independent transcriptomic data showed increased MEG3 in endometriosis and decreased H19 and DIO3 in carcinomas, with concordant direction in 20/21 significant miRNA–host gene pairs (p<0.001).

Conclusions: Endometriosis and ovarian carcinoma show distinct miRNA profiles. Endometriotic lesions gain the inflammation-associated miR-21-5p and miR-146a-5p and lose miR-503-5p and miR-10b-5p, which target PI3K and IGF1 receptor signalling. Malignant transformation is marked by gain of the epithelial miR-200 family and miR-183/182/96 cluster and loss of the stromal miR-143/145. miR-885-5p and loss of miR-34b/c emerge as candidate markers of clear cell and high-grade serous carcinoma, respectively. Changes in miRNAs of the imprinted DLK1-DIO3 and H19/IGF2 loci were confirmed by their host genes in independent transcriptomic data, pointing to imprinting deregulation as a candidate mechanism of malignant transformation.

[II-8] COMPARATIVE EVALUATION OF TRANSCRIPTOMIC, CELL-COMPOSITION AND MULTIOMIC CLASSIFIERS FOR RELAPSE AND TREATMENT RESISTANCE PREDICTION IN PEDIATRIC T-ALL

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Acute T-cell lymphoblastic leukemia (T-ALL) is an aggressive hematologic malignancy characterized by significant molecular heterogeneity. Despite advances in treatment, relapse and treatment resistance remain serious clinical challenges associated with poor treatment outcomes. The aim of this study is to develop and compare prognostic models for relapse and/or treatment resistance in pediatric T-ALL using diagnostic samples, as well as to evaluate whether cellular deconvolution of bulk RNA-seq data can provide prognostic value comparable to approaches based on gene expression and multi-omics features.

The study cohort included 62 pediatric patients with T-ALL for whom data were available from whole-genome sequencing (WGS), RNA sequencing (RNA-seq), miRNA sequencing (miRNA-seq), and DNA methylation. Patients were clinically classified based on disease relapse, treatment resistance, and the occurrence of either of these events. All datasets underwent quality control and feature cleaning. WGS features were aggregated at four levels, with the highest level based on mutation signatures from the COSMIC database. Feature selection was performed using Boruta algorithm, followed by classification using the Random Forest method. For the deconvolution-based approach, scRNA-seq data from six paired samples collected at diagnosis and relapse, as well as six unpaired samples, were analyzed using the Louvain Community Detection method, which identified 13 cellular clusters. A signature matrix was constructed by calculating the average expression profiles for the 4,500 genes with the highest discriminatory power across all clusters. Subsequently, the CIBERSORTx Fractions module was used to deconvolute the bulk RNA-seq samples, generating a matrix of estimated cluster proportions for each patient. These fractions were used as features in a Random Forest classifier without feature selection. In all approaches, classes were balanced by reducing the number of samples, and model evaluation was performed using "leave-one-out" cross-validation (LOOCV). Classifiers based on RNA sequencing and multivariate classifiers followed the same classification workflow.

Cellular deconvolution demonstrated promising prognostic value, approaching the effectiveness of multi-omic classification, based solely on bulk RNA-seq data. These results suggest that representing transcriptomic profiles as estimated cellular fractions may allow for the capture of biologically relevant information related to treatment outcomes, offering a potential alternative to more complex and data-intensive multiomic approaches. However, the effectiveness of models based on deconvolution may depend on the design of signatures, the definition of clusters, and the robustness of fraction estimates. Therefore, further methodological improvement and validation in independent cohorts are necessary to determine their overall usefulness and clinical utility, particularly in predicting relapse.

[II-9] ALTERATIONS IN THE N-GLYCOME OF EXTRACELLULAR VESICLES DERIVED FROM PROSTATE CANCER CELLS

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Background: Protein glycosylation is involved in cellular regulation and may undergo substantial remodeling during prostate cancer development. Extracellular vesicles (EVs) carry glycoproteins that may reflect the characteristics of their parental cells and contribute to intercellular communication. Analysis of EV-associated N-glycans may help characterize prostate cancer-related molecular changes and identify potential candidates for future biomarker research.

Objective: This study aimed to investigate the N-glycan composition of EVs released by prostate adenocarcinoma cells and compare their glycosylation profiles with those of EVs derived from non-malignant prostate epithelial cells.

Methods: EVs were isolated from conditioned media of PC-3 prostate adenocarcinoma cells and non-malignant HPrEC prostate epithelial cells. EV preparations were characterized using transmission electron microscopy, nanoparticle tracking analysis, and immunoblotting for selected EV-associated markers. N-glycan profiles were analyzed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). The analysis included individual glycan structures and major glycan groups, with assessment of oligomannose, hybrid-type, and complex-type glycans, fucosylation, and sialylation.

Results: A total of 57 N-glycan compositions were identified across the prostate-derived EV samples, including 53 structures in PC-3-derived EVs and 33 in HPrEC-derived EVs. Individual glycan analysis revealed increased abundance of oligomannose structures H5N2, H8N2, H9N2, and H10N2 in cancer-derived EVs, whereas H7N2 was less abundant. The hybrid-type structure H6N3 also showed increased abundance. At the group level, PC-3-derived EVs exhibited an increased relative abundance of oligomannose-type glycans and fucosylated structures. In contrast, the relative abundance of complex-type N-glycans and overall sialylation was reduced, including α2,6-linked sialylated structures. Several individual fucosylated glycans were enriched in cancer-derived EVs, while multiple structures carrying α2,3- and/or α2,6-linked sialic acid showed decreased abundance. These results demonstrate changes in both the composition and distribution of major N-glycan groups associated with prostate cancer-derived EVs.

Conclusions: Prostate cancer-derived EVs show increased oligomannose content and fucosylation, accompanied by reduced complex glycosylation and sialylation. These alterations warrant further investigation in prostate cancer glycomics and biomarker research.

[II-10]* ANTIOXIDANT DEFENCE AGAINST FERROPTOSIS: DISTINCT ROLES OF GPX4, FSP1 AND PRDX1

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Ferroptosis is an iron-dependent form of regulated cell death characterised by excessive peroxidation of polyunsaturated fatty acids within membrane phospholipids. A major protective mechanism is the glutathione–glutathione peroxidase 4 (GSH–GPX4) pathway, which reduces phospholipid hydroperoxides and maintains redox homeostasis. However, the resistance of some cells to ferroptosis despite reduced GPX4 activity suggests that alternative antioxidant mechanisms contribute to cellular protection.

This work aims to compare antioxidant defence mechanisms, focusing on the FSP1–CoQ10 pathway and peroxiredoxin 1 (PRDX1), and to examine their potential contributions to limiting lipid peroxidation independently of GPX4 activity.

Ferroptosis suppressor protein 1 (FSP1) is a well-characterised regulator that acts in parallel with GPX4. By using reducing equivalents to regenerate ubiquinol, FSP1 supports the neutralisation of lipid radicals and limits the propagation of membrane lipid peroxidation. PRDX1 represents a distinct antioxidant mechanism, contributing to the removal of hydrogen peroxide and other peroxides and to the regulation of redox homeostasis. Its activity is coupled to the thioredoxin–thioredoxin reductase–NADPH system. Unlike FSP1, PRDX1 may influence ferroptosis through both GPX4-independent mechanisms and indirect modulation of NRF2–GPX4 signalling, depending on the cellular model.

This comparison highlights that protection against ferroptosis involves a network of complementary antioxidant systems extending beyond GPX4. The interplay between these mechanisms may contribute to differences in cellular susceptibility to oxidative stress and ferroptosis induction.

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Young Scientist poster session

[II-11]* ASSESSING THE USE OF SODIUM TRANSPORT ASSOCIATED GENE EXPRESSION CHANGES AS POTENTIAL EFFECT MARKERS FOR EMPAGLIFLOZIN TREATMENT IN ISCHEMIC STROKE THERAPY

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Empagliflozin is a reversible inhibitor of sodium–glucose cotransporter 2 (SGLT2), used to treat type 2 diabetes, heart failure, and chronic kidney disease. Preclinical studies suggest that it may attenuate cerebral ischemia-reperfusion injury. Given the contribution of disrupted sodium homeostasis to ischemic brain oedema, changes in sodium transport-related gene expression may provide clues to its potential protective mechanisms.

The aim of this research was to identify genes most susceptible to significant expression changes after the administration of empagliflozin. Identification of such genes could prove useful in further research regarding the use of empagliflozin in stroke therapy.

A meta-analysis was conducted using established bioinformatic analysis methods. A total of 32 potential datasets were gathered from Gene Expression Omnibus (GEO) and ArrayExpress (search query: “Empagliflozin” or “EMPA”). Given the limited number of brain-specific studies, we included 17 datasets spanning multiple species (*Homo sapiens, Rattus norvegicus, Mus musculus,* and *Danio rerio*) and diverse tissues and cell types, including heart, lung, liver, smooth muscle, brain, and endothelial cells. Differential gene expression analysis was subsequently performed, retaining genes with at least a two-fold ($|\log_2 FC| > 1$) expression change, and a p-value < 0.05. Genes associated with sodium ion transport (pathway GO:0006814) were then sequestered from the remaining data. Next, Robust Rank Aggregation (RRA) was performed, and significantly altered genes were evaluated based on the magnitude of their expression changes (RRA p_adj <0.05).

Our analysis identified 16 genes whose expression was significantly altered in response to empagliflozin. Of these, two genes have shown consistent changes across datasets; CNNM4 was upregulated, whereas YWHAH was downregulated. Several other genes have previously been associated with cerebral ischemia, including increased expression of EDNRB, SLC6A8, and DOT1L, increased WNK1 protein levels in hypertensive rats, decreased SCN1B expression, and reduced AKT3 protein levels following experimental stroke. These overlapping responses identify candidate molecular links between cerebral ischemia and empagliflozin exposure, warranting further investigation in stroke-specific models. An example of such candidate target is CNNM4; its expression is sensitive to empagliflozin, and the protein it encodes is abundantly found in brain tissue (microglia, astrocytes, and neurons). These findings suggest that CNNM4 is a possible link between empagliflozin treatment and ischemic response; however, this link needs to be verified in stoke-model.

Young Scientist poster session

Session III: Bioinformatics and Mathematical Modeling

[III-1] MICROHOMOLOGY-DRIVEN GENOMIC ALTERATIONS AS PROGNOSTIC BIOMARKERS: IN A PAN-CANCER ANALYSIS

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Microhomology-mediated end joining (MMEJ) is a highly mutagenic pathway for double-strand break (DSB) repair that relies on short homologous sequences. Often associated with template switching, MMEJ generates distinct genomic alterations, including microhomology-flanked deletions, insertions, and multinucleotide variants (MNVs). Because homologous recombination deficiency (HRD) can increase the reliance on MMEJ as an alternative DSB repair pathway, characterization of MMEJ-associated alterations may provide insight into the mechanisms underlying genomic instability in cancer. Given the high prevalence of HRD, ovarian cancer represents an important model for studying the genomic consequences of MMEJ. To assess whether these patterns extend beyond ovarian cancer, we further investigated MMEJ-associated alterations across multiple tumor types in a pan-cancer analysis.

To identify and characterize MMEJ-associated alterations, we developed the MHDetect algorithm, which classifies these variants using whole-genome sequencing (WGS) and whole-exome sequencing (WES) data. Comparison of the two methods showed that the limited genomic coverage of WES affects the reliable detection and quantification of these variants, supporting WGS as the preferred approach for their characterization.

We then investigated how Loss of Function (LoF) alterations affect microhomology-associated deletion burden and assessed their prognostic significance across an ovarian cancer (OV) cohort and a TCGA Pan-Cancer dataset. In the OV cohort, increased MMEJ-associated burden was not significantly associated with canonical BRCA1/2 alterations but was significantly associated with loss of the tumor suppressors RB1 and CCDC122. The pan-cancer analysis identified additional genes, including FDX1 and PDE8B, whose inactivation was associated with increased accumulation of MMEJ-associated deletions across multiple tumor types. In OV, a higher MMEJ-associated deletion burden was also associated with longer overall survival.

Our results identify specific genetic alterations associated with increased MMEJ activity and demonstrate the potential prognostic relevance of MMEJ-associated mutational burden in cancers. These findings provide a foundation for personalized therapies.

[III-2]* THE IMPACT OF BATCH EFFECT ON DATA CLUSTERING IN CT SCREENING STUDIES

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Introduction: Lung cancer is leading cause of mortality among all cancers. Diagnosis is significantly hampered because the disease does not present typical symptoms in its early stages. In Poland, most cases are diagnosed at an advanced stage. Thus, low dose screening programmes are developed, but the resulting large volumes need automated analysis. As the data between centres may differ substantially, the transferability of DL methods can be limited. We compare two screening datasets to analyse the potential batch effect in nodule radiomic features.

Materials and methods: Two LDCT cohorts were used: Pilot Pomeranian Lung Cancer Screening Program, conducted in 2009-2011 among smokers (928 scans), and the DUKE Lung Cancer Screening dataset, comprising 1613 scans acquired between 2015 and 2021. Lungs and bronchovascular bundles were segmented using RONALD. Then, lesions were segmented via region growing, using the datasets' annotations and parenchyma masks. Radiomic features of the changes were extracted, and nodules were clustered using k-means, with cluster number chosen via Silhouette score and clinical and batch concordance measured by the Adjusted Rand Index (ARI). Distributional differences were assessed using Maximum Mean Discrepancy (MMD2) with RBF kernel.

Results: K-means clustering with k=7 showed near-chance agreement with malignancy labels (ARI= -0.003), while cohort labels are more explained by underlying data structure (ARI=0.2204). Quantitative evaluation of distribution shifts using MMD^2 revealed a substantial batch effect between the two cohorts (DUKE vs. Pomeranian: MMD^2 = 0.1997). In contrast, the distance between clinical classes across the datasets was markedly lower (Malignant vs. Benign: MMD^2 = 0.0121).

Conclusions: The results indicate that batch effects substantially influenced the radiomic features, with differences between cohorts likely reflecting both technical and clinical sources of variation. In particular, differences in data acquisition equipment and dosage may have contributed to the observed batch effect, while variations in the definition and classification of early-stage cancer across countries may have introduced additional inconsistencies in clinical labelling.

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Young Scientist poster session

[III-3] GENOME-WIDE CHARACTERIZATION OF GENETIC ASSOCIATIONS AND THEIR STRENGTH IN TYPE 1 DIABETES

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Genome-Wide Association Study (GWAS) is a statistical method designed to identify genetic variants that show statistically significant associations with the susceptibility to a disease under study. This method is particularly useful in research on diseases whose genetic basis have not yet been fully understood. An example of such a disease is type 1 diabetes (T1D).

The dataset for this study included 3,824,482 genetic variants obtained from 2,598 patients and 4,240 control samples in the Ukrainian population. Following the quality control assessment of the data, 865,009 genetic variants were obtained. In the subsequent stage, linkage disequilibrium pruning procedure was implemented to reduce redundancy in the genetic information by excluding variants that were correlated ($r^2>0.2$). This procedure yielded a set of 176,031 variants, which was then used for principal component analysis without the necessity of removing these variants from the association analysis. 10 principal components that define the main patterns of genetic variation among the study participants, were used in the GWAS model.

The results were analyzed with respect to the effect of association with the disease for each variant, calculated using the odds ratio (OR) adjusted for the case-control disproportion. In this study OR ranged from 0.163 to 4.725. In total, among the risk variants (OR>1), 3,077 presented at least small effect size (OR>1.386). The vast majority (1,916) was located on chromosome 6, while the remaining variants were evenly distributed across the other chromosomes. The situation was analogous for protective variants (OR<1). 2,679 of these reached at least small effect size (OR<0.722), with the majority (1,334) being again located on chromosome 6. This finding is consistent with the biological mechanisms underlying type 1 diabetes, in which genes belonging to the Major Histocompatibility Complex on this chromosome have been identified as the most susceptible to T1D.

A comparison of the GWAS results with those reported by one of the largest studies on T1D (Robertson et al.) revealed 23 shared variants, none of which reached the established OR effect size threshold for the small effect category. This finding may suggest that within the analyzed population, the estimated effects of these variants were relatively negligible.

The results obtained in this study indicate that GWAS analyses are a very effective method for identifying variants statistically associated with a disease, and the reported odds ratios can be used to assess the actual strength of this effect. It is also important to study diverse populations and analyze them separately, which allows for a better characterization of genetic associations.

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[III-4] MATHEMATICAL MODELLING OF EPITHELIAL–MESENCHYMAL TRANSITIONS WITH A TREATMENT PROTOCOL

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Epithelial-to-Mesenchymal Transition (EMT) and its reverse, Mesenchymal-to-Epithelial Transition (MET), are important biological processes influencing cancer progression, particularly tumor invasiveness and metastasis. Based on Celià-Terrassa et al. (2018), we propose a mathematical model describing the interactions between the main components of these transitions, including TGF-beta signaling, the miR-200/ZEB feedback loop, and E-cadherin expression, a marker of the epithelial phenotype. The poster presents the model scheme and the basic dynamics of its two main subsystems: the first describing TGF-beta in its free and bound forms, and the second describing the production and degradation of miR-200, ZEB, and E-cadherin. A key feature of the second subsystem is the possibility of bistability and hysteresis, which depend on the level of TGF-beta entering the subsystem (Szafrańska-Łęczycka et al. (2026)). We focus on how these dynamical properties may influence transitions between epithelial and mesenchymal phenotypes and their implications for cancer treatment. In particular, we introduce therapy protocols involving Galunisertib (LY2157299 monohydrate), a TGF-beta receptor I inhibitor, and investigate how targeting TGF-beta signaling may alter tumor-cell phenotype and potentially influence treatment response (Herbertz et al. (2015)).

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[III-5] MRI-Based Radiomic Assessment of Tumor Heterogeneity Following Local Paclitaxel Delivery via PLGA Nanofibers in a Mouse Model of Breast Cancer

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Mice are widely used as preclinical animal models in cancer research to investigate tumor growth, progression, and responses to therapeutic interventions. Magnetic resonance imaging (MRI) is not routinely used in all preclinical studies involving mice due to its high cost, limited availability and specialized technical requirements. Consequently, tumor growth and treatment response are often assessed using alternative methods, such as caliper-based tumor measurements and histological analysis. However, these approaches provide limited information about internal tumor structure, vascularization, and changes over time.

The study included 12 mice that were injected with aggressive 4T1 breast tumor cells subcutaneously (2×10^5 cells in 100 µL PBS, lower flank). Eight days post-injection, poly(L,L-lactide-co-glycolide) (PLGA 50:50) nanofiber implants were introduced directly into the tumor tissue in two variants (n = 6 per group): blank PLGA 50:50 nanofibers (control group) and paclitaxel-loaded PLGA 50:50 nanofibers (PLGA 50:50 PTX, 122 µg). Each mouse underwent magnetic resonance imaging (MRI) prior to implant insertion (baseline) and at 4, 8, and 13 days post-implantation. MRI was performed using a Bruker 9.4 T micro-imaging system. The imaging protocol consisted of relaxometry studies (T_1 and T_2), diffusion-weighted imaging (DWI), and dynamic contrast-enhanced T_1-weighted imaging (DCE-MRI). Internal structure of tumors was compared between timepoints and groups using radiomic features. Radiomics from the dynamic images were extracted separately using 11 model-free features, that were describe the pharmacokinetic changes in each point in the space.

Statistical analysis of the extracted features involved three complementary approaches: the Student's t-test or Mann–Whitney U test was used to assess differences between individual features at each time point; the Chow test was applied to compare the average rates of change between the two groups; and ANCOVA was used to evaluate differences in feature trajectories over time and predict feature values at later time points. All p-values were adjusted for multiple comparisons using the Benjamini–Hochberg procedure.

The analysis of individual features revealed no statistically significant differences. However, both the Chow test and ANCOVA models identified significant differences in the increments of dispersion in the areas under individual dynamic points. Additionally, the Chow test identified a correlation derived from T2 maps as significant. The observed changes in area variability reflect differences in pharmacokinetic profiles, potentially indicating alterations in tumor heterogeneity over time caused by a medicine application.

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[III-6] STATISTICAL IDEALIZATION OF MULTICHANNEL PATCH-CLAMP RECORDINGS

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Ion channel activity provides a vital functional readout of cancer cell electrophysiology and cellular responses to experimental perturbations. Patch-clamp recordings may capture one or several active channels, making reliable identification of functional states essential for quantitative analysis.

Channel currents are often idealized as binary signals, with "0" representing a closed channel and "1" an open channel, enabling analysis of dwell-time distributions. threshold may suffice for simple recordings, but noise, baseline drift, subconductance states, and variable open-state conductance complicate state assignment, particularly when several channels are active simultaneously. These challenges motivate alternative segmentation schemes.

Existing analytical methods have distinct requirements: Hidden Markov Models use specified kinetic schemes; the multiscale methods considered here require recording-filter characterisation; and deep-learning frameworks require labelled training data during development. We propose a statistical approach based on differences in variance within and across current states. The tool operates without predefined gating models or neural-network training and retains transparent, adjustable parameters. Segmentation can represent intermediate current levels when they satisfy the statistical and amplitude criteria.

The signal-processing workflow includes baseline correction, noise estimation, segmentation, removal of small events, and merging adjacent levels. The baseline is estimated from local minima of absolute current and subtracted from the recording. Noise is estimated from local variance distributions. Segmentation evaluates differences between mean currents relative to their uncertainties; merging depends on step amplitude and the variance of combined segments. The program estimates a single-channel current amplitude from transition sequences and uses it to assign the fitted signal to discrete levels and estimate open-channel counts.

The workflow was successfully executed on a broad set of experimental patch-clamp recordings. The tool generated baseline-corrected signals, fitted idealized traces, segment statistics, and dwell-time distributions across complete recordings. Traces exhibiting severe baseline drift required targeted parameter adjustments beyond default heuristics, delineating operational boundaries for automated runs.

This framework provides an inspectable, low-barrier foundation for quantifying single- and multi-channel channel behavior in healthy and cancer cells. Systematic benchmarking against synthetic ground-truth traces and independently annotated datasets validates the workflow's accuracy in brief-event detection, confirms its resilience to recording-filter distortions, and demonstrates substantial execution speed advantages over existing deconvolution platforms.

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[III-7] EXPLORING THE TUMOR MICROENVIRONMENT THROUGH SPATIAL TRANSCRIPTOMICS WITH GRAPH NEURAL NETWORKS

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Background: Pancreatic ductal adenocarcinoma (PDAC) is characterized by a complex tumor microenvironment (TME), in which neural invasion (NI) is associated with aggressive disease progression and poor prognosis. Spatial transcriptomics (ST) provides an opportunity to investigate the spatial organization of the TME and its relationship with NI. This study aimed to evaluate the potential of graph neural networks (GNNs) for predicting NI status in PDAC.

Methods: Visium ST data were integrated with single-cell and single-nucleus RNA sequencing data to estimate cell-type composition. Tissue sections were represented as graphs, with nodes corresponding to spatial spots and edges reflecting their spatial proximity. Different node features and edge-weighting strategies were evaluated using graph convolutional networks to classify NI status.

Results: The best-performing configurations achieved a balanced accuracy of 0.941, with the highest-performing combination of Harmony-corrected transcriptomic embeddings and cell-type composition-based edge weights yielding 0.941 ± 0.034. The corresponding sensitivity and specificity were 0.963 and 0.920, respectively. Analysis of learned representations revealed associations with ductal cells and cancer-associated fibroblasts, suggesting the relevance of tumor–stromal organization in NI.

Conclusions: These findings demonstrate the potential of graph-based spatial transcriptomics analysis for predicting neural invasion in PDAC, although independent validation is required to establish model generalizability.

[III-8] PRE-PROCESSING WHOLE-SLIDE HISTOPATHOLOGY IMAGES WITH HISTOKIT FOR ENDOMETRIAL CANCER GRADE CLASSIFICATION

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The advent of Whole Slide Imaging (WSI) has enabled the creation of digital equivalents of glass slides containing stained tissue specimens, facilitating the development of automated image analysis methods based on image processing and machine learning. However, several challenges still hinder their reliable implementation in clinical practice. One is artifacts introduced at various stages of slide preparation and scanning. In addition, WSI analysis pipelines typically involve multiple repetitive preprocessing steps that need to be performed consistently across datasets.

In this work, we present a practical use case for HistoKit, a publicly available package that provides an integrated workflow for WSI preprocessing and quality control. The workflow begins with tissue segmentation using the GaMRed algorithm, followed by quality control based on an improved version of the state-of-the-art artifact segmentation method GrandQC. HistoKit enables the segmentation of multiple artifact classes, including out-of-focus regions, tissue folds, incorrectly exposed regions, foreign objects, air bubbles, tissue edges, and marker traces of different colors. The package further supports stain normalization using the Macenko, Vahadane, and Reinhard methods, image augmentation, and multithreaded patch extraction. We also introduce an interactive dashboard for fast visualization of artifact segmentation maps, analysis of artifact distributions across the dataset, and identification of low-quality slides.

We demonstrate the workflow on a cohort of 59 hematoxylin and eosin-stained endometrial cancer WSIs representing three histological grades, obtained from the Department of Pathomorphology at Jagiellonian University. Following HistoKit quality control, artifact-free patches were extracted and processed using the slide-level foundation models TITAN and PRISM 2 within the TRIDENT framework. The resulting slide-level embeddings were used for tumor grade classification with a fully connected neural network. Model performance was evaluated using classification metrics over 50 iterations of Monte Carlo cross-validation. In an additional experiment, nuclei segmentation with the Hover-Net model was performed without excluding regions identified as artifacts to investigate the effect of artifacts on downstream algorithms.

The presented experiments demonstrate practical applicability of HistoKit for processing new cohorts without modifying the source code or developing custom preprocessing scripts. In particular, out-of-focus regions affected downstream image analysis: HoVer-Net failed to correctly segment cell nuclei in severely blurred tissue areas. These observations highlight the importance of systematic WSI quality control and data preprocessing.

[III-9] DOMAIN GENERALIZATION FOR DEEP LEARNING MODELS BASED ON WHOLE EXOME SEQUENCING DATA

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Background: Whole-exome sequencing (WES) is a valuable source of genetic information widely adopted in genome-wide association studies, genetic biomarker identification, and personalized medicine. A crucial step in WES data preparation is target enrichment, which enables the isolation of specific genomic regions of interest. To date, many brands offer various target enrichment kits that differ in the gene panels they provide. Differences among enrichment panels introduce additional sources of technical variation and result in shifts in domain representation, especially in studies that collect data from multiple sources. The goal of this study is to construct a domain generalization framework for deep learning-based prediction of breast cancer age at onset, thereby reducing kit-related technical variation in gene-level genomic representations.

Methods: The collected WES data consist of 9 datasets from several data sources, obtained using 8 different target enrichment kits from Agilent, Nimblegen (Roche), and IDT. The data were processed using the pipeline described in [1], excluding the gene-level imputation step. The final data representation consists of gene-level scores and clustered samples of sequencing kits with similar characteristics. The model used for predictions was a multi-layer perceptron (MLP) with an auxiliary domain-generalization autoencoder (AE) branch operating on the intermediate MLP representations. The MLP branch was assessed using the correlation concordance coefficient (CCC), while the AE was forced to minimize the Kullback-Leibler divergence between pre-identified batch-related clusters. Moreover, in AE, Huber loss was used to preserve biological information. To assess the potential benefits of the AE branch for domain generalization, the local inverse Simpson's index was used.

Results: Preliminary findings demonstrated the feasibility of simultaneous learning of age at onset and domain generalization. The regression model achieved CCCs of approximately 0.962 and 0.573 on the training and validation sets, respectively. Domain generalization analysis of the LISI index, comparing latent and high-dimensional representations, indicated improved mixing among initially separated domain clusters, suggesting a more unified domain representation.

Conclusions: The proposed dual-branch architecture provides a promising conceptual framework to handle a source of variation specific to target enrichment kits in heterogeneous WES data. Our preliminary results indicate the feasibility of reducing domain differences through latent representation learning. Further investigations will focus on assessing other advanced generalization strategies, among others, adversarial learning and meta-learning.

1. Jarosz, L., Ochocki, M., Merta, J., Pusztai, L., & Marczyk, M. (2026). A Practical Workflow for Correcting Kit-Specific Effects in Whole-Exome Sequencing Data. Methods and Protocols, 9(5), 125.

[III-10] MODELLING FERRITINOPHAGY DYNAMICS: AN ODE MODEL OF THE KEAP1–NRF2/HERC2–NCOA4 AXIS FOR ERASTIN-INDUCED IRON STRESS

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Introduction: Ferritinophagy is the selective autophagic breakdown of ferritin by the NCOA4 receptor. It releases stored iron into the labile iron pool (LIP), and too much of it can push cells into ferroptosis. Existing models rarely link oxidative stress sensing to autolysosomal iron release.

Aim: To build and test a deterministic model that connects the KEAP1–NRF2 cascade with the HERC2–NCOA4 autolysosomal switch.

Methods: We built a system of 10 ODEs covering NRF2, the active and inactive forms of KEAP1, ROS, HERC2, NCOA4, the NCOA4–ferritin complex, ferritin, ferroportin and the LIP. The LIP is the central feedback node. Kinetic parameters came from the literature. As reference data, HCT116 cells were treated with 10 μM erastin and followed for 1–48 h with RT-qPCR (NFE2L2, FTMT, NCOA4), FerroOrange and DCFH-DA.

Results: The model reproduced the main experimental trends. KEAP1 was quickly inactivated, NRF2 accumulated with damped oscillations, and ferritin and ferroportin rose. Around 3 h, both the LIP wave and the ROS signal fell, which matched the experiments. Using RT-qPCR fold changes directly as multipliers of synthesis rates made the system numerically unstable. This shows that mRNA levels cannot be translated linearly into rates of protein synthesis.

Conclusions: The model can serve as a virtual laboratory for estimating homeostatic stability thresholds qualitatively. Two next steps are to add saturation of NRF2 binding at ARE promoter sites and to calibrate the model with protein-level (Western blot) data.

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[III-11] From Experimental Data to Mathematical Modeling of MIR155HG Products Dynamics

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The MIR155 Host Gene (MIR155HG) generates multiple functional products, including the oncogenic microRNA miR-155, the long non-coding RNA BIC (lncRNA-155), and the micropeptide miPEP155. While miR-155 is an established contributor to B-cell lymphoma (BL) pathogenesis and a potential therapeutic target, the regulation and biological significance of the other two products remain poorly understood. Understanding the dynamics of these products is particularly relevant when considering therapeutic strategies targeting miR-155, as changes in its production may be accompanied by alterations in other products derived from the same locus.

This study focuses on developing a mathematical framework to characterize MIR155HG product biogenesis and support the investigation of their regulatory relationships. A system of ordinary differential equations (ODEs) is being developed to represent five molecular species: unspliced MIR155HG transcripts, spliced lncRNA-155 in the nucleus and cytoplasm, mature miR-155, and miPEP155. Model parameterization integrates information from published studies with experimental measurements. Quantitative RT-PCR (qRT-PCR) data from BL cell lines under basal conditions are being processed to characterize transcript abundance and support the estimation of model parameters. Previously obtained data following ionizing radiation exposure provide an additional reference for exploring changes in MIR155HG expression under stress conditions.

Preliminary experimental observations indicate that MIR155HG-derived products exhibit distinct responses to ionizing radiation, with increased levels of pri-miR-155 and spliced lncRNA-155, while unspliced transcript levels remain comparatively stable. These observations provide a biological basis for investigating whether a model incorporating multiple MIR155HG-derived species can capture their potentially different regulatory dynamics.

The ongoing work aims to establish a quantitative framework for studying MIR155HG biogenesis under basal and stress conditions. Integration of experimental data and subsequent model analysis will support the generation of hypotheses concerning the regulation of individual products and their potential relevance to B-cell lymphoma biology.

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[III-12] AUTOMATED ANALYSIS OF HIGH-FREQUENCY ULTRASOUND IMAGES OF HEALTHY SKIN ACROSS DIVERSE ANATOMICAL SITES

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High-frequency ultrasound (HFUS) enables non-invasive, high-resolution visualization of skin architecture. This study proposes an end-to-end framework integrating deep learning-based quality assessment, automated layer segmentation, and multi-parametric feature extraction for 75 MHz HFUS imaging.

A cohort of 44 healthy participants aged 18-45 underwent HFUS imaging at 4 face locations. Before imaging, age, sex, height, weight and skin phototype of all participants was recorded, as well as questionnaires about their sun-protection habits, caffeine consumption, stress and skin treatment history. Image quality was screened using a pretrained VGG16 network. Accepted images were subjected to automated skin layer segmentation using a TransUNet architecture pretrained on inflammatory skin diseases. The model extracted 3 distinct skin layers: epidermis, subepidermal low-echoic band (SLEB) and dermis. Based on the segmentation results, a comprehensive set of quantitative features describing morphology, echogenicity, intensity, thickness, and texture was calculated. Finally, statistical analyses were performed to evaluate variations in skin parameters across sex, age, BMI, stress level, skin phototype and reported sun-protection habits.

Across all participants, the SLEB layer became more uneven with age, and the proportion of pixels with medium echogenicity increased and the dermis thickened with age across the analyzed sites. There was no significant correlation with participants' BMI. Sex-based analysis revealed that women were characterized by significantly higher skin echogenicity in both the epidermis and dermis, as well as lower epidermal roughness. No sex-related differences were observed regarding the thickness of individual skin layers. The analysis revealed no statistically significant associations between skin features and phototype, sun protection habits, or substance use.

The proposed automated framework demonstrates reliable performance in quality control and multi-layer segmentation for 75 MHz HFUS. This approach provides a robust, objective foundation for comprehensive dermatological mapping and inter-demographic analysis.

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[III-13] In Silico Profiling of SHH and Group 3 Medulloblastoma Molecular Subgroups

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Introduction: Medulloblastoma is a heterogeneous pediatric brain tumor which comprises four molecular subgroups: WNT, SHH, Group 3 and Group 4. Besides protein-coding genes, non-coding RNAs, including circular RNAs (circRNAs), may contribute to tumor biology through interactions with RNA-binding proteins (RBPs). This in silico study aimed to characterize mRNA and circRNA expression in SHH and Group 3 medulloblastoma compared with normal cerebellum and to explore potential circRNA–RBP interactions.

Methods: RNA-sequencing data were downloaded from GEO dataset GSE203174. This set consists of both mRNA (81 medulloblastoma, 5 normal cerebellum samples) and circRNA data (65 medulloblastoma, 4 normal cerebellum samples). Sequencing depth was assessed, followed by a principal component analysis (PCA). Differential expression analysis using DESeq2 compared primary SHH (19 mRNA, 19 circRNA samples) and primary Group 3 (15 mRNA, 9 circRNA samples) medulloblastoma samples with normal cerebellum (5 mRNA, 4 circRNA samples). Differentially expressed transcripts were defined by p adjusted < 0.05 and |log2FC| > 1 (circRNA) or |log2FC| > 2 (mRNA). For both SHH and Group 3, mRNA and circRNA expression data were integrated with CLIP-seq-derived circRNA-RBP interactions obtained from Cancer-Specific CircRNA Database 2.0. Gene Ontology (GO) enrichment analysis was performed with clusterProfiler package. All analyses were performed in R (version 4.5.3).

Results: GO enrichment analysis of differentially expressed mRNAs revealed enrichment in biological processes across SHH and Group 3 subgroups associated mainly with neural development and neuronal signaling, respectively. The SHH circRNA-RBP network comprised 670 interactions involving 161 upregulated circRNAs spliced from 135 host genes along with their respective 11 RBPs: FBL, LIN28B, IGF2BP1, CBX7, IGF2BP3, RPS5, MSI1, YBX3, VIM, NPM1 and EZH2. The Group 3 network comprised 233 interactions involving 55 upregulated circRNAs spliced from 46 host genes and 10 associated RBPs: FBL, IGF2BP1, IGF2BP3, LIN28B, ELAVL3, KHDRBS2, ALYREF, MSI1, VIM and CBX7. Several of these RBPs are involved in RNA stabilization and RNA metabolism associated processes.

Conclusions: Differential expression analysis of mRNA and circRNA revealed marked differences between examined medulloblastoma subgroups and normal cerebellum. The predicted network points to the importance of RNA metabolic processes associated with the upregulation of circRNAs in medulloblastoma. Further experimental validation is needed to confirm these interactions and establish their functional relevance in medulloblastoma biology.

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[III-14] GENOMIC MUTATION DYNAMICS AND SITE FREQUENCY SPECTRUM SIGNATURES OF EMERGING SARS-COV-2 SUBVARIANTS

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Rapid evolution of SARS-CoV-2 and the emergence of subsequent subvariants have underscored the need to develop methods for the early identification of evolutionary changes in viral populations. In our study, we used the data from the GISAID database, comprising nearly 15 million SARS-CoV-2 genome sequences, to analyze the dynamics of mutations associated with variants of concern (VOCs). We observed that mutations defining new evolutionary lineages can appear in characteristic patterns, and their frequencies change over time, which may reflect selection processes occurring within the viral population.

The results of the site frequency spectrum (SFS) analysis revealed characteristic shifts in the cumulative mutation frequency distribution, manifested as local discontinuities in its tails. This phenomenon was linked to the presence of a group of mutations co-occurring over a specific period of time. In subsequent observation stages, the discontinuities shifted toward higher mutation frequencies, consistent with the increasing prevalence of the mutations forming these groups. This pattern was identified retrospectively during the analysis of Delta variant evolution and may provide insights into the dynamics of emerging subvariants and their potential competitive advantage over parental variants.

These findings suggest that analyzing the tails of the cumulative SFS can serve as a useful complement to monitoring SARS-CoV-2 evolution and as a potential source of early warning signals for the emergence of new viral sublineages. Additionally, we developed a model to quantitatively describe the dynamics of the observed transition process. This approach can support the identification of viral lineages at an early stage of frequency growth, although the detected signals require further verification as well as additional epidemiological and evolutionary analyses.

[III-15] COOPERATIVE ION CHANNEL GATING: SIGNAL SIGNATURES, FUNCTIONAL CONSEQUENCES, AND HYSTERESIS

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Ion channels shape membrane electrical signaling by switching between conducting and nonconducting states. Although channel gating is commonly independent, in some biological systems neighboring channels may interact, enhancing or suppressing cluster currents relative to independent gating and potentially producing gain- or loss-of-function-like effects. Signatures and functional consequences of cooperative gating remain, however, poorly understood.

Building on our published random-walk model, we simulated clusters of two to five channels exhibiting positive, negative, or no inter-channel coupling. At a fixed membrane potential, we generated 20 independent realizations per condition, each comprising 25000 transitions between cluster conductance states, across a broad range of activation-driving values. We quantified open probability (P_o), the mean number of open channels (NP_o), aggregate-state dwell times, and statistical and entropy-based descriptors. Machine-learning methods (CatBoost and 1D-CNN) were also used to classify coupling regimes using segments of aggregate current or sequences of aggregate-state dwell times, with additional descriptors where applicable.

Coupling of channels within a cluster modulated current magnitude, gating kinetics, and entropic properties. Preliminary classification results suggest that coupling regimes can be distinguished from aggregate signals, with performance depending on signal representation and descriptor selection. We extended the model to periodic depolarization, incorporating charge-dependent relaxation and inheritance of channel states between cycles. The resulting hysteresis depended on both coupling type and the ratio of stimulation period to cluster relaxation time; stimulation before complete relaxation yielded pronounced history-dependent responses. We discuss these findings in the context of empirical characteristics of BK channels in U87-MG glioblastoma cells, KATP channels expressed in HEK293 cells, epilepsy-associated Slack mutants studied in Xenopus laevis oocytes and neuronal models, and neuromorphic nanopore systems.

Together, our results identify inter-channel cooperativity and competing timescales as potential determinants of cluster-level gain- or loss-of-function-like effects and memory-like electrical responses. The proposed analytical framework provides adaptable tools for interpreting multichannel recordings across biological and synthetic systems.

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[III-16] Mathematical Modeling to Extend the Measurement Range of the Anti-SARS-CoV-2 QuantiVac ELISA (IgG) Assay Above the Highest Calibrator: A Preliminary Study

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Introduction: In the QuantiVac assay, results exceeding the highest calibrator (120 RU/ml; 384 BAU/ml) require re-testing after dilution, which increases workload and reagent consumption. The aim was to estimate the extent to which a result could be obtained by extrapolating the calibration curve with an acceptable margin of error.

Methods: A retrospective analysis was conducted on 12 runs of the Anti-SARS-CoV-2 QuantiVac ELISA (IgG) test (487 samples; 373 samples re-tested after dilution). The results were obtained during research conducted as part of the Cardio-Vascular Intervention for Multimorbidity Prevention project (CV-IMP; 2024/ABM/01/00058). Results extrapolated above 120 RU/mL were compared with results obtained after dilution in leave-one-plate-out cross-validation on a log10 scale, using a priori acceptance criteria (bias and CV ≤20%, total error ≤30%). The effective dilution factor was determined using samples with undiluted results falling within the curve's range. Extrapolation was performed by linear extension of the last calibration segment (Cal2–Cal1), a plate-specific four-parameter logistic (4PL) model and a joint 4PL/5PL model across plates, and spline interpolation as well as function-basis approximation were assessed as alternatives. Computations were performed in MATLAB (R2025b).

Results: The effective dilution factor of the re-test dilution (nominal 1:1010) was 62.6 (95% CI 58.3–66.7), indicating an approximately 60% overestimation of post-dilution results; these readings (1.4–2.6 RU/mL; n = 21) fell below the manufacturer's verified linear range (6–120 RU/mL). After correction, linear extension of the last calibration segment met all criteria up to 160 RU/mL (bias −0.9%, CV 12.7%, total error 13.6%; n = 36). Above 160 RU/mL acceptance was not robust to the uncertainty of the dilution factor, and above 200 RU/mL no model met the criteria (increasing bias of the linear extension and CV >20% for the 4PL/5PL models). Therefore, 62 (12.7%) samples could be calculated using the extended curve up to 160 RU/mL.

Conclusions: Preliminary data indicate that the measurement range can be extended to 160 RU/ml (512 BAU/ml), which would avoid approximately 13% of repeat tests. The results require confirmation in a serial-dilution experiment with duplicate measurements.

[III-17] IN SILICO ANALYSIS OF MIR-193B AND MIR-365A TARGET GENES REVEALS POTENTIAL LINKS TO ATRIAL FIBRILLATION AND CARDIOEMBOLIC STROKE

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Atrial fibrillation (AF) is a major risk factor for cardioembolic ischemic stroke. Despite advances in diagnosis, identifying the cardioembolic origin of ischemic stroke remains challenging, highlighting the need for novel molecular biomarkers. MicroRNAs (miRNAs) are post-transcriptional regulators of gene expression that may influence both cardiac electrophysiology and the endothelial response to ischemic injury. This study aimed to investigate the potential relevance of miR-193b and miR-365a to AF-associated cardioembolic stroke by examining their predicted target genes and associated signaling pathways.

miR-193b and miR-365a were selected based on miRNA sequencing results obtained from human brain endothelial cells exposed to ischemic conditions. Potential target genes were predicted using the miRDB and DIANA bioinformatic databases, and overlapping genes were retained for further analysis. Enrichment analysis was performed using STRING and ShinyGO, based on the KEGG and Gene Ontology databases. Enriched pathways and biological processes were considered statistically significant at a false discovery rate (FDR) < 0.05.

Functional enrichment analysis of predicted miR-193b target genes highlighted the PI3K–Akt, MAPK, Ras, ErbB, and Hippo pathways, which may be involved in cellular signaling and structural remodeling processes relevant to AF. For miR-365a, pathways associated with cholinergic signaling and mTORC1 were identified, suggesting potential links to cellular regulation and cardiac electrophysiology. Notably, KCNJ2, KCNJ3, and KCNH2, which encode potassium channel subunits involved in cardiac membrane potential and repolarization, were identified among the predicted target genes. These findings suggest that miR-193b and miR-365a may be associated with molecular mechanisms involved in atrial electrical remodeling and AF, potentially providing a link between miRNA-mediated regulation and cardioembolic ischemic stroke.

In conclusion, miR-193b and miR-365a may represent candidate regulators of molecular processes relevant to AF and cardioembolic ischemic stroke. Their predicted target genes and associated signaling pathways warrant further investigation to clarify their potential role in cardiac electrical remodeling and their value as biomarkers of stroke etiology.

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[III-18]* Comparison of Semantic Similarity and Expert-Curated Refinement of Molecular Pathway Grouping Tools in GO Enrichment Analysis for Ischemic Stroke Research

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Ischemic stroke represents a critical medical challenge characterized by complex pathophysiology, where microRNAs such as miR-9-3p, miR-9-5p, and miR-129-5p act as post-transcriptional regulators responding to reperfusion therapy. To elucidate underlying cellular mechanisms, modern bioinformatics relies on pathway enrichment analysis utilizing databases such as Gene Ontology (GO), KEGG, Hallmark, and Panther, fed by target gene lists retrieved from miRDB under strict confidence thresholds. However, standard post-analysis grouping tools remain unadapted to the neurobiological context of stroke. Conventional semantic similarity algorithms Wang, Resnik, Lin, Rel, and Jiang implemented in GOSemSim combined with parent term reduction tools (such as rrvgo) generate substantial background noise, because they cluster biological processes based purely on mathematical topology and ontology graph structures, ignoring real pathological relevance. Consequently, automated platforms like ShinyGO and clusterProfiler frequently group metabolic, general signaling, and vascular repair processes in a manner detached from actual stroke pathology.

To address these limitations, we evaluated existing clustering methods and, in collaboration with domain experts, introduced a custom dictionary-based grouping pipeline implemented in R. This rule-based system utilizes advanced data frame transformations from dplyr and precise pattern matching from stringr, operating on specialized biological dictionary curated by experts. As a result, our approach enables deterministic mapping of GO terms to core pathological processes of stroke (including neuroinflammation, oxidative stress, apoptosis, structural integrity, and vascular repair), operating independently of parent term hierarchies.

For comparative assessment and visual presentation, enrichment results were aggregated and visualized using ggplot2 and treemapify. Comparative treemaps and boxplots of term-level significance distributions illustrated differences between conventional grouping approaches and our custom framework. The dictionary-based approach organized enriched terms into predefined categories relevant to stroke pathophysiology, facilitating interpretation of processes otherwise distributed across multiple groups. Incorporating $-\log10$(p-values) highlighted more statistically significant terms within these categories, while the original enrichment statistics were retained. Statistical significance was interpreted separately from biological or clinical relevance. Furthermore, owing to fully modular architecture in the dictionary approach allows for seamless adaptation and revision to other biomedical domains or multi-omic contexts, laying a scalable foundation for future bioinformatics developments. Overall, this integrated framework provides a transparent, robust, reproducible, and biologically sound alternative that significantly enhances data interpretability, intuitive visual assessment, and workflow rigor.

Young Scientist poster session

[III-19]* AUTOMATIC STENOSIS DETECTION IN CORONARY VESSELS

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Coronary artery disease is one of the most lethal conditions worldwide, with stenosis, which is an abnormal narrowing of vessels being the direct cause of severe medical complications. Detecting it effectively may be the difference between severe complications and life saving treatment. In this study, we present a method for automatic detection and localization of candidate stenosis in coronary vessels, which is a fusion of standard computer vision and deep learning-based approach. The first step of the proposed method is the segmentation of coronary vessels. To achieve better results, three different deep learning methods had been used and compared basing on performance metrices and generalization ability, where classical U-Net outperformed Attention U-Net and dual branch U-Net architecture. The next step was extracting skeletons, which are the centerlines of the binary masks obtained from previous segmentation. To obtain the skeletons, Zhang-Suen and Lee thinning algorithms have been used, with Lee method producing branches with less spurious points, giving the cleaner versions of received output. The last step of the pipeline based on detecting the stenosis itself. It used classical computer vision methods and based on obtaining the diameter of the vessel along its length using distance transform. The stenosis regions were detected by comparing the diameters of the neighboring points along the centerline and looking for sudden drop of the width of an inspected vessel. The final results were satisfactory in each of those three steps and are a proof that mixing classical and deep learning methods can be crucial in correct assessment of complex tasks related to medicine.

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Young Scientist poster session

[III-20]* PROFILING DNA METHYLATION PATTERNS IN ACUTE MYELOID LEUKEMIA USING UNSUPERVISED MACHINE LEARNING

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Introduction: Acute Myeloid Leukemia (AML) is a disease, in the development of which a massive role is played by DNA methylation - a process crucial for gene expression control. Finding hidden patterns in DNA methylation profiles helps to better understand the AML background and can improve risk stratification and personalized treatments.

Methods: In this study, unsupervised machine learning was used to group AML patients according to their methylation profiles. The analyzed data came from The Cancer Genome Atlas (TCGA) project and were downloaded from the Genomic Data Commons database. The data were cleaned by removing sex chromosomes, imputing missing values, and transforming them to adjust statistical distributions. Different dimensionality reduction methods - such as Principal Component Analysis (PCA) and Gaussian Mixture Model (GMM) - were used to isolate the most variable CpG sites. The data were clustered using algorithms - such as Partitioning Around Medoids (PAM) and hierarchical clustering with several different distances (Euclidean, Maximum, Canberra, Pearson, and Spearman). The clustering was carried out on three datasets: the complete one, the subset restricted to the most significant CpG sites according to PCA, and according to GMM. The cluster quality was checked using UMAP projections and heatmaps.

Results: This comparison showed that Spearman and Canberra distances were unsuccessful in dividing all three datasets, while PAM and hierarchical clustering with Euclidean, Pearson, and Maximum distances generated consistent consensus matrices in at least one case. The optimal number of clusters was set to 4 or 5 for every dataset and clustering algorithm. Division of GMM-filtered data resulted in clearer clusters on the heatmaps, which implies that the analysis performed only on the most variable methylation loci amplified the biological signal and silenced the technical noise. The best division was created by PAM and hierarchical clustering with Euclidean or Pearson distances. Clustering revealed 4 to 5 stable groups of AML patients visible on UMAP, while heatmaps showed clusters of similar methylation profiles with hyper- and hypomethylation in certain regions. The best visual representations were provided by hierarchical clustering with Euclidean distance performed on data filtered by GMM and hierarchical clustering with Pearson distance performed on data filtered by PCA.

Conclusions: Dimensionality reduction was crucial for improving cluster separation, highlighting the importance of selecting the most informative CpG sites. The choice of clustering algorithm and distance metric also strongly influenced the results. These findings indicate that methylation-based clustering may contribute to the characterization of AML heterogeneity, although further investigation is required.

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Young Scientist poster session

[III-21]* DEEP LEARNING CLASSIFICATION OF CORONARY ARTERY TREES IN ANGIOGRAPHIC IMAGES AS A STEP TOWARDS INDIVIDUAL VESSEL CLASSIFICATION

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Introduction: Precise analysis of coronary arteries is important for the automatic assessment of coronary anatomy. Designed process includes three different tasks. First one is image classification into LCA (left coronary artery) and RCA (right coronary artery). Second one is vessel segmentation, and last one classification of individual arteries. Image classification is the first step and provide the basis for further vessel classification. However, this task is challenging due to anatomical differences and the complex branching of coronary arteries but the deep learning methods can help automate image analysis.

Methods: Three CNN architectures, ConvNeXt V2[1], EfficientNet V2S[2] and ResNet-50[3], were used for image classification into LCA and RCA. Each network has different structure and approach to image processing. ConvNeXt V2 is a modern CNN architecture designed to improve feature extraction using updated convolutional blocks. EfficientNet V2S aims to balance model performance and computational cost. ResNet-50 uses residual connections, which help the network learn more complex features and make training easier. The models were trained on the CADICA[4] dataset using 5-fold cross-validation and on-the-fly data augmentation. Their performance was evaluated using accuracy, precision, recall, F1-score, error rate, specificity, and the receiver operating characteristic curve (ROC) with the area under the curve (AUC).

Results: The best-performing model for binary classification of coronary angiography images into LCA (left coronary artery) and RCA (right coronary artery) was EfficientNet V2S, trained using on-the-fly data augmentation. It achieved an accuracy of 0.90 and an AUC of 0.97 on an independent test set. The other models also showed good classification performance.

Conclusions: In conclusion, EfficientNet V2S achieved the best classification performance, while ResNet-50 obtained the lowest results. On-the-fly data augmentation improved the performance of all three models, showing its importance for coronary artery image classification.

Future tasks: Future work will focus on improving classification performance using boosting methods or bootstrap resampling. Another step will be to develop separate segmentation models for LCA and RCA separately.

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[III-22]* CHARACTERIZING BREAST CANCER CLONAL ARCHITECTURE USING DIFFERENT COMPUTATIONAL APPROACHES

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Intratumor heterogeneity is a characteristic feature of cancer that influences tumor progression, metastasis, and response to therapy. It results from the presence of genetically distinct cell populations within the same tumor. Identifying these populations from sequencing data can provide insights into tumor evolution, clonal relationships, and differences between distinct tumor regions. Reconstructing tumor clonal architecture is therefore essential for understanding tumor evolution, although different computational methods may produce varying results due to their underlying assumptions.

We aimed to characterize breast cancer clonal architecture by identifying mutation clusters corresponding to potential tumor clones and subclones using PyClone-VI [1] and comparing the results with those obtained using the DECODE [2] tool. PyClone-VI groups mutations based on allele read counts, copy number information, and estimated tumor content, while DECODE identifies clonal and subclonal clusters based on variant allele frequency (VAF) distributions. The analysis included samples from primary tumors and lymph node metastases, with CITUP [3] applied to PyClone-VI results to explore potential phylogenetic relationships between tumor sites. Additionally, synthetic data generated using TEMULATOR [4] were used to test the methods on simulated datasets with characteristics and clonal complexity comparable to those of the real data.

The comparison focused on the number and composition of identified clusters, the consistency of mutation assignments, and the overlap between mutations identified by the two methods. Preliminary results revealed differences between PyClone-VI and DECODE in the number and composition of mutation clusters, with PyClone-VI generally identifying more clusters in the analyzed samples. The overlap between mutations assigned by the two methods also varied, highlighting the importance of considering methodological differences when interpreting inferred clonal structures.

Together with the phylogenetic relationships explored using CITUP, these results provide complementary insights into breast cancer clonal architecture and potential relationships between primary tumors and lymph node metastases, and show how the choice of computational approach may influence the interpretation of tumor evolution.

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Young Scientist poster session

[III-23]* FUNCTIONAL SCALING OF GBK CHANNEL ACTIVITY MODULATED BY 17B-ESTRADIOL AND PROGESTERONE IN HUMAN GLIOBLASTOMA CELLS

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Gliomas occur more frequently in men than in women, suggesting that sex-related biological factors, including steroid hormones, may contribute to glioma development and progression. However, the mechanisms through which sex hormones influence glioma biology remain poorly understood. In this study, we investigated the effects of 17β-estradiol (E2) and progesterone (P) on large-conductance voltage- and Ca^{2+}-activated potassium channels (gBK) in human U87-MG glioblastoma cells. These channels have been implicated in glioblastoma cell proliferation and migration, and their expression is elevated in malignant gliomas compared with nonmalignant brain tissue. Our preliminary electrophysiological observations revealed an unusually high occurrence of multichannel activity in U87-MG cells, motivating us to investigate hormone-dependent changes in both individual gBK channel activity and the collective gating behavior of multichannel ensembles.

U87-MG cells were exposed to E2 or P at selected concentrations ranging from ng/mL to µg/mL for different durations (<1, 6, 24, or 48 h). Channel activity was assessed using single- and multichannel patch-clamp recordings obtained at fixed membrane potentials under different hormonal conditions and corresponding controls. We analyzed open-state probabilities, current amplitudes associated with distinct conductance states, and the temporal characteristics of transitions between channel ensemble states. These parameters were evaluated as functions of hormone concentration, exposure duration, and the number of active channels within individual membrane patches. To investigate potential inter-channel interactions, native multichannel recordings were compared with synthetic multichannel traces generated by superimposing independent single-channel recordings, providing a reference model of independent channel gating.

Our findings indicate that E2 and P modulate gBK channel activity in a concentration- and time-dependent manner, leading to either enhanced or suppressed channel activity under different experimental conditions. Importantly, hormone-dependent alterations in multichannel gating dynamics suggest that sex steroids may influence not only individual channel function but also the collective behavior of gBK channel ensembles. These observations provide new insights into the hormonal regulation of gBK channels in glioblastoma and raise the possibility of inter-channel interactions contributing to the observed functional responses.

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Young Scientist poster session

[III-24]* TEXT EMBEDDINGS FOR PATHWAY ENRICHMENT IN OMICS DATA

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Pathway enrichment analysis is widely used bioinformatical step in omic data analysis, which use the statistical inference. However, classical approaches rely on gene-pathway associations without considering the semantic relationships between their functional descriptions. Gene set analysis based on text embedding may offer alternative approach to analyzing pathways.

Text embeddings of human genes descriptions from NCBI and descriptions of 271 KEGG pathways were generated using five models: BioBERT, MedCPT, PubMedBERT, SapBERT, text-embedding-3-small. Differentially expressed genes (DEGs) were identified from microarray and RNA-seq datasets available through the GSEABenchmarkeR package. For each dataset, a DEGs and pathway semantic matrix was exact, while semantic similarity was calculated using cosine distance. Next, the similarity matrix for each dataset was used to calculate enrichment scores (ES) using two different approaches. The first approach involved summing cosine similarity scores between differentially expressed genes (DEGs) and KEGG pathways. The second approach employed z-score standardization, using genes without statistically differential expression as the background reference. The resulting one-dimensional ES vectors were then subjected to clustering using k-means (KM), Gaussian mixture models (GMM), and hierarchical clustering (HC).

The best-performing configuration combined summation-based ES, text-embedding-3-small embeddings, and GMM clustering. When evaluated against MalaCards-derived reference annotations, this configuration achieved a mean Matthews correlation coefficient (MCC) of 0.12, sensitivity of 0.63 and specificity of 0.53 across the evaluated datasets.

Embedding based pathway enrichment analysis provides an alternative framework for interpreting omics data by incorporating semantic relationships between gene and pathway descriptions. Future research should investigate alternative ES calculations and strategies to improve agreement with reference pathway annotations.

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Young Scientist poster session

[III-25]* EVALUATING LANGUAGE MODEL EMBEDDINGS IN A MEDICAL TEXT CLASSIFICATION TASK

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Introduction: Text classification is a fundamental task in Natural Language Processing (NLP) that assigns predefined labels to textual data based on its content. For specialized tasks, using field-specific language representations may improve classification results. This study evaluates the effectiveness of medical language representations for medical text classification by comparing a medical-domain language model with a general-purpose model, both fine-tuned on the same domain-specific dataset.

Methods: Two language models were evaluated: (i) Polish-distilroBERTa and (ii) Polish-distilroBERTa-medical. A reduced version of a medical dataset obtained from mostwiedzy.pl, consisting of 2664 records assigned to six categories with a slightly imbalanced class distribution, was used. The dataset was stratified by class and split into training, validation, and test sets in a 70:15:15 ratio. The best hyperparameter configuration was selected based on the highest validation macro-F1-score on the validation set. We then evaluated model performance on the test set using accuracy and macro-averaged precision, recall, and F1-score. We used confusion matrices to identify the most often misclassified classes.

Results: For all evaluated metrics, Polish-distilroBERTa-medical outperformed Polish-distilroBERTa on the test set, achieving a macro-F1-score of 0.818. At the class level, the medical model performed better on most classes. The confusion matrices revealed that both models struggled to distinguish between “Pathomorphological examination” and “Oncological examination” classes.

Conclusions: The results suggest that domain-specific language representations may improve performance in medical text classification compared with general-purpose representations. With further improvements and additional comparisons, the Polish-distilroBERTa-medical model may prove useful for medical text classification. Future work will refine hyperparameter optimization to improve model performance, compare additional models, and integrate an XAI module to visualize how medical terms influence model predictions.

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Young Scientist poster session

Session IV: Biomaterials and Applied Biotechnology

[IV-1] CELLULAR RESPONSES AND CALCIUM SIGNALLING AT GABA-LOADED PEDOT BIOELECTRODES

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Neurological disorders are among the leading causes of disability and premature mortality in Europe and are frequently associated with impaired neurotransmission involving glutamate, γ-aminobutyric acid (GABA), dopamine, or acetylcholine. Current therapeutic strategies rely mainly on systemic drug administration, which often results in non-specific distribution, limited targeting precision, and adverse side effects. Neurotransmitter-loaded conducting polymers offer a promising means of delivering biologically active molecules directly at the bioelectrode in response to electrical stimulation.

This study investigated the cytocompatibility and cellular signalling responses associated with GABA-loaded poly(3,4-ethylenedioxythiophene) (PEDOT) coatings. PEDOT coatings were fabricated by electrochemical deposition onto electrode substrates. Their effects on SH-SY5Y neuroblastoma and U87 glioblastoma cultures were evaluated using resazurin-based metabolic assays and morphological assessment. The ability of the coatings to modulate cellular signalling was assessed by fluorescence calcium imaging to quantify intracellular Ca^{2+} transients following electrically triggered GABA release.

Cells cultured on PEDOT:GABA coatings exhibited a moderate reduction in metabolic activity compared with PEDOT controls, while preserving normal morphology and viability. Calcium imaging of U87 cultures revealed reproducible, stimulus-dependent intracellular Ca^{2+} fluctuations in response to electrical stimulation, confirming their capacity to modulate cellular signalling pathways. These responses were detected only upon electrical stimulation and were absent under non-stimulated conditions, indicating that stimulation of the GABA-loaded interface was associated with changes in cellular calcium signalling.

Together, these findings support the cytocompatibility of PEDOT:GABA coatings under the conditions tested and demonstrate stimulus-dependent cellular responses at the bioelectrode. The preservation of cellular morphology, alongside measurable calcium signalling responses, supports further investigation of neurotransmitter-loaded conducting polymers for spatially and temporally controlled neuromodulation. Establishing the specific contribution of released GABA and validating these responses in physiologically representative neural models will help clarify their biological and therapeutic potential.

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[IV-2] WINNING THE "RACE FOR THE SURFACE": CURCUMIN-LOADED PEDOT COATINGS WITH ENHANCED BIOCOMPATIBILITY AND ANTIBACTERIAL CAPABILITIES

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The "race for the surface" refers to the critical period immediately following the implantation of medical devices, where host cells and bacteria compete to colonize the implant's surface. Despite decades of medical advancement, perioperative infections remain a major clinical challenge. To address this issue, we investigated the immobilization of curcumin within a poly(3,4-ethylenedioxythiophene) (PEDOT) coating - a conductive polymer known for its biocompatibility - aiming to impart antibacterial properties without compromising the host cell integration.

PEDOT coatings were electrochemically synthesized in three distinct environments, with and without curcumin. The modified surfaces were comprehensively characterized regarding their electrochemical behaviour (cyclic voltammetry, electrochemical impedance spectroscopy), physicochemical properties (wettability, roughness, SEM), and biological performance. Specifically, we evaluated the coatings' impact on NHDF cell viability (AlamarBlue assay) and Pseudomonas aeruginosa colonization (Live/Dead analysis).

The results demonstrated that while the inclusion of curcumin slightly reduced the layer's overall conductivity, the impedance profiles remained well below the critical 1 MΩ threshold required for high-quality signal recording. Furthermore, curcumin significantly altered the surface roughness and wettability, leading to enhanced biocompatibility and reduced bacterial viability. Crucially, the addition of curcumin also improved the physical adhesion of the PEDOT layer to the substrate, successfully addressing a well-known limitation of this conducting polymer.

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[IV-3] FROM NANOTUBE ARCHITECTURE TO CELL RESPONSE: CARBON NANOTUBE BUCKYPAPERS AS NEURAL BIOINTERFACES

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Carbon nanotube (CNT)-based buckypapers (BPs) are promising bioelectronic interfaces because of their conductivity, porous high-area architecture and interaction with living cells [1]. Effective bioelectrodes should combine electrochemical performance with biological response [2]. Thus, understanding how CNT architecture and surface features affect both aspects is essential for rational design [3].

BPs prepared from single-walled carbon nanotubes (SWCNTs) and multi-walled carbon nanotubes (MWCNTs) differing in dimensions, purity, and surface modification were investigated. Electrochemical properties were assessed by cyclic voltammetry (CV) and electrochemical impedance spectroscopy (EIS). Biological response of SH-SY5Y neuroblastoma and U-87 glioblastoma cells was evaluated by resazurin assay and cell area, circularity, and fractal dimension.

CNT architecture markedly affected electrochemical behaviour. Charge storage capacity (CSC) decreased with increasing scan rate, indicating ion accessibility within the porous CNT network. Selected SWCNT- and short, thin MWCNT-based BPs showed high CSC at low scan rates, whereas oxidation-modified CNTs had lower CSC despite improved wettability. Thus, wetting alone was insufficient; CNT organisation, electronic connectivity and pore accessibility were crucial.

Biological response depended on material composition and cell type. Short, thin MWCNT-based BPs promoted cell spreading and complex morphology, especially in U-87 cells, whereas a hydrophobic commercial BP induced a compact phenotype with higher circularity, lower fractal dimension and smaller cell area. In several cases, morphology did not match metabolic activity, showing that both readouts are complementary. Differences between SH-SY5Y and U-87 cells confirmed model-dependent interactions.

Overall, CNT BP performance as bioelectrodes cannot be predicted from a single property. CNT geometry, surface chemistry, wettability, porous architecture and electrochemical accessibility collectively shape the biointerface. Combining electrochemical characterisation with quantitative cell analysis may support rational optimisation of CNT-based materials for bioelectronics.

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[IV-4] COVALENT IMMOBILIZATION OF ANTIBODY ON POLY(N,N-DIPHENYL-1,4-PHENYLENEDIAMINE) AND ITS APPLICATION IN AN ELECTROCHEMICAL THYROGLOBULIN SENSOR

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Differentiated thyroid cancer is increasingly common, and long-term surveillance relies on highly sensitive thyroglobulin (Tg) measurement to detect recurrence. Tg is a 660-kDa glycoprotein used as a key biomarker, with healthy levels ranging from 3 - 40 ng/mL and cancer-associated levels typically exceeding 40 ng/mL. Currently, Tg assays are mostly optical, expensive, and unsuitable for point-of-care testing. Therefore, a robust electrochemical biosensor capable of low-level Tg detection in a portable, rapid, and affordable format is urgently needed. Developing such a sensor requires a conductive electrode material that supports covalent antibody grafting without compromising electrochemical performance. To address this, we synthesized the aniline derivative N,N-diphenylbenzene-1,4-diamine (DPB) monomer. DPB provides a rigid aromatic backbone and a structurally stable framework, without swelling and shrinkage during repeated redox cycling. Enhanced π–π stacking in DPB facilitates charge transport and improves film stability. The resulting polymer, p-DPB, offers a robust platform for antibody and enzyme immobilization, making it attractive for biosensing applications. Here, p-DPB was employed as the substrate for covalent Tg-antibody immobilization *via* Schiff-base chemistry. Electropolymerization and subsequent glutaraldehyde grafting were evaluated using cyclic voltammetry (CV) and electrochemical impedance spectroscopy (EIS). Possible reactions between p-DPB thin films and glutaraldehyde were examined by FTIR, CV, and EIS. Antibody immobilization on the p-DPB surface was confirmed through confocal imaging and immunoreactivity studies. Eventually, AbTg-p-DPB/screen printed carbon electrode successfully employed in thyroglobulin biomarker sensor.

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[IV-5] EVALUATING NANOGEL PERMEABILITY ACROSS HUMAN BBB CO-CULTURE MODEL

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Introduction: There are many brain diseases, and one of the challenges with their treatment is effective drug delivery through the blood-brain barrier (BBB). BBB is a highly selective, very tight barrier that is permeable to lipophilic substances. One of the solutions is the use of nanogel carriers with the sufficient charge, shape and size that could effectively reach their target and cross BBB. The use of an in vitro model of the BBB makes it cost-effective and faster to test the ability of applied nanoparticles to cross the barrier than to conduct in vivo animal studies. By using the BBB's in vitro model, it is possible to estimate which carriers have the best chance of success in further stages of testing. A wide range of BBB models has been established, from simple monoculture systems to more sophisticated co-culture and 3D models. Among the available BBB models, Transwell-based co-culture systems offer a practical and cost-effective alternative to more complex experimental models.

Aim: Synthesis of a fluorescently labeled nanogel and evaluation of its penetration through Transwell-based co-culture BBB.

Methods: A nanogel was synthesized via photoinitiated free radical polymerization using a reverse miniemulsion technique and then conjugated to Cy5. The permeability of the nanogel across a cell-free membrane with 0.4 μm pores coated with fibronectin and poly-L-lysine was assessed as a control. An in vitro co-culture BBB model compromising immortalized human endothelial cells and astrocytes was used to evaluate the penetration ability of the nanogel. Nanogel flux was assessed at 2, 4, 6, and 8 h, while cells were fixed at 30 min, 2 h, and 6 h for further analysis. The nanogel cytotoxicity was assessed with MTS assay.

Results and conclusions: The nanogel was non-cytotoxic and internalized by BBB cells. Its concentration in the basolateral compartment increased over time, suggesting time-dependent transport across the BBB model.

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[IV-6] VALORIZATION OF STRAW INTO BIOCHAR FOR AMELIORATION OF SANDY SOIL

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Straw is an abundant agricultural residue that can be converted into biochar for the sustainable management of salt affected soils. This pot based study evaluated the potential of straw derived biochar to ameliorate sandy soil and support mung bean growth. Alkaline biochar was produced using pyrolyser and incorporated into sandy soil. The experiment followed a completely randomized design with replicated treatments comprising different levels of seawater induced salinity and biochar application rates. Changes in key soil properties, including pH, electrical conductivity, and the availability of sodium, potassium, and calcium, were assessed. Seed germination, chlorophyll content, plant height, root length, and other growth related parameters were also examined to determine the influence of biochar on plant responses to salt stress. Biochar application improved soil conditions, nutrient availability, seed germination, and plant growth under low-to-moderate salinity. A relatively low biochar application rate produced the most favorable response, whereas its effectiveness declined with increasing salinity and higher application rates. These findings demonstrate that the performance of biochar depends on both the amendment rate and the severity of salt stress. Overall, the appropriate application of straw derived biochar could provide a sustainable approach for improving the agricultural suitability of coastal sandy soils while promoting the beneficial utilization of crop residues.

[IV-7] STRATEGIES TO ENHANCE BIOGAS PRODUCTION FROM FOOD WASTE THROUGH CHEMO-MICROWAVE PRETREATMENT

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Food waste (FW), owing to its high organic content, represents a promising substrate for sustainable biofuel production. However, the complex structure of organic matter limits its biodegradability and conversion efficiency during anaerobic digestion. Although conventional pretreatment methods can improve biomass solubilization, their high energy requirements and limited effectiveness often restrict their practical application. This study investigates the effect of chemo-microwave pretreatment (CMP) to improve food waste solubilization and promote biogas production while reducing energy consumption. Initially, microwave pretreatment (MWP) was applied to facilitate the breakdown of organic compounds and increase chemical oxygen demand (COD) solubilization. The combined CMP process achieved greater COD solubilization and lower microwave energy consumption than microwave pretreatment alone. The improved solubilization of organic matter also contributed to higher biogas yields compared with the microwave alone and untreated food waste.

[IV-8] ELECTROSPUN MATERIALS FOR DRUG DELIVERY SYSTEMS INTENDED FOR UROLOGICAL APPLICATIONS

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Electrospinning is a method for producing fibrous polymer materials characterized by high porosity and a large specific surface area; due to their resemblance to the natural extracellular matrix, they find application in medicine, including tissue engineering, tissue regeneration, and in case when they are loaded with drug- they can find also application in controlled drug release (as a drug delivery systems). The technique involves producing fine fibers by subjecting a polymer solution (which can be enriched with drug molecules) to a strong electric field, which stretches the material into thin strands. This technology holds particular promise in oncology, where electrospun materials can facilitate the local delivery of anticancer drugs, potentially helping to reduce the risk of disease recurrence associated with incomplete tumor removal and metastasis. The formation and morphology of the fibers depend on the various parameters such as: solution properties, process parameters, and ambient conditions. These parameters strongly influence not only morphology of the fibers but the properties of the whole electrospun system, including release profile.

In our study we have developed different, drug-loaded electrospun implants with diverse architecture. For the bioresorbable implants we used electrospun materials obtained with biodegradable polymers which we loaded with different anticancer agents. Developed implants are flexible and can be used for a loco-regional strategy of treating various cancers. It is particulary interesting since the idea of local delivery has become attractive strategy and drug delivery systems are constantly being developed. The example of such systems is: mitomycin C-based hydrogel (UGN-101; Jelmyto) or erdafitinib implant (TAR-210) which has entered clinical trial phase (1–2). These examples illustrate the development of diverse local drug delivery platforms tailored to the anatomical location of the tumor and the properties of the active substance employed.

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[IV-9] CONDUCTIVE POLYPHENYLENEDIOXYTHIOPHENE-N-HYDROXYSUCCINIMIDE GRAFTED ANTIBODY FOR THE IMMUNOSENSOR APPLICATION

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Immunosensors are in high demand for the timely diagnosis of diseases through bioelectrochemical approaches. However, grafting bioreceptors (antibodies) onto electrode surfaces without harsh chemical treatments is desirable to preserve their biological activity. In this context, we designed and synthesized a highly conductive dioxythiophene-based material, 2,5-dioxopyrrolidin-1-yl 1,3-dimethylbenzo[b]thieno[3,4-e][1,4]dioxine-6-carboxylate (PheDOT-NHS), incorporating phenylenedioxythiophene (PheDOT) and N-hydroxysuccinimide (NHS) as an electrode material. The poly phenylenedioxythiophene-N-hydroxysuccinimide enable readily graft with antibody amine group without damaging its activity. PheDOT-NHS aims to establish effective interfaces between synthetic organic materials and living systems by leveraging their electrical and optoelectronic properties. The resulting material exhibits limited water solubility and excellent electrical conductivity, alongside the potential to form covalent bonds with amine-containing biomolecules. These complementary properties make PheDOT-NHS a promising candidate for the development of functional bioelectronic interfaces. This work establishes a molecular design strategy for integrating electronic functionality and chemical reactivity into organic materials for bioelectronic applications.

[IV-10] EFFECT OF COMONOMER TYPE AND RATIO ON THE PHYSICOCHEMICAL PROPERTIES OF ZWITTERIONIC COPOLYMERS

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Introduction and Aim: Sulfobetaine-based polymers have attracted interest due to their unique ionic structure, which influences their hydration, thermal behaviour and interactions with aqueous environments. Copolymerization with non-zwitterionic monomers offers a strategy to tailor these properties by adjusting copolymer composition. This study investigates the physicochemical characteristics of two previously synthesized series of zwitterionic copolymers comprising sulfobetaine methacrylate (SBMA) and either 2-hydroxyethyl methacrylate (HEMA) or methyl methacrylate (MMA). The copolymers feature linear or star-shaped architectures, with SBMA molar fractions (F) ranging from 0.22 to 0.89 in the SBMA-HEMA series and from 0.24 to 0.90 in the SBMA-MMA series. The study aims to elucidate the effects of comonomer type, composition, and polymer architecture on the thermal, solution, and surface properties of the resulting materials, considering their potential use in future biomedical applications.

Methods: Differential scanning calorimetry (DSC) was used to determine the glass transition temperatures (Tg) of the polymers. The critical micelle concentration (CMC) was determined using fluorescence spectroscopy and goniometry. Cloud point temperature (TCP) was investigated in water or phosphate-buffered saline (PBS), depending on the copolymer composition, using UV-Vis spectroscopy coupled with a Peltier temperature control system. Water contact angle (WCA) measurements were performed to determine the surface characteristics of the polymer films.

Results and Conclusions: Tg is a key parameter affecting polymer chain mobility, material stability, and the drug release behavior of polymer-based delivery systems. DSC analysis confirmed the influence of polymer composition and architecture on the thermal properties of the obtained zwitterionic polymer matrices, including variations in their Tg. Copolymers with HEMA showed lover Tg (130-161°C) compared to copolymers with MMA (151-184°C). TCP depended on polymer composition and architecture, being observed only for homopolymers and copolymers with the lowest MMA content. MMA-rich polymers required NaCl or PBS for sufficient solubility. The CMC was determined by nonlinear regression analysis and was evaluated only for the P(SBMA-co-MMA) copolymers. The WCA increased with increasing HEMA and MMA content in the copolymers. The lowest WCA (29.4°) was recorded for the linear homopolymer, whereas the highest value (63.3°) was observed for the star-shaped P(SBMA-co-MMA) copolymer with a 1:3 molar ratio. The next stage of this research will involve the development of drug-loaded micellar systems and polymer conjugates with folic acid analogue, focusing on their potential application in transdermal drug delivery platforms.

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[IV-11]* GRAPHENE QUANTUM DOT-SUPPORTED GOLD NANOPARTICLES FOR THE ELECTROCHEMICAL DETECTION OF HYDROGEN PEROXIDE

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Gold nanoparticles (AuNPs) are nanoscale gold structures that exhibit unique optical, electronic, catalytic, and physicochemical properties, making them highly attractive for applications in medical diagnostics, drug delivery, catalysis, and biosensing. In general, AuNPs are synthesized through the reduction of gold ions (Au^{3+}) to metallic gold (Au^0), followed by the nucleation and growth of gold atoms into nanostructured particles. Various reducing agents, including sodium borohydride, citrate, and ascorbic acid, have been widely employed to facilitate this process, while stabilizing or capping agents are commonly used to control particle size and prevent aggregation. For electrode-based applications, AuNPs can be fabricated directly on conductive substrates through electrodeposition or via electroless deposition and seed-mediated growth approaches, enabling the formation of well-adhered nanostructured surfaces with enhanced electrochemical performance.

In this work, AuNPs were directly deposited onto a graphene quantum dot (GQD)-modified glassy carbon electrode through a self-deposition process driven by the inherent reducing properties of GQDs, eliminating the need for external reducing agents. Dodecyltrimethylammonium bromide was utilized as a shape-directing agent to regulate nanoparticle growth and morphology. The resulting GQDs/AuNPs nanocomposite was characterized by spectroscopic, microscopic, and electrochemical techniques. The fabricated electrode exhibited excellent electrocatalytic activity toward hydrogen peroxide (HP) reduction and enabled its sensitive determination by amperometric sensing after optimization of the fabrication conditions. Furthermore, the practical applicability of the sensor was demonstrated by analyzing human serum samples using the standard addition method. The sensor also exhibited satisfactory selectivity, reproducibility, and storage stability, highlighting its potential for reliable HP detection in complex biological matrices.

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Young Scientist poster session

[IV-12]* ULTRA-SENSITIVE GQDS/AUNPS/POLYPYRROLE IMMUNOSENSOR FOR CA-125 BIOMARKER DETECTION

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Ovarian cancer, known as the "silent killer", is one of the most difficult cancers to detect due to its asymptomatic nature in the early stages. Clinical diagnosis predominantly relies on the CA-125 antigen as the primary biomarker, with normal baseline levels below 35 U/mL, which can surge up to 1340 U/mL in aggressive cases. Given that routine testing methods lack the sensitivity required for early-stage detection, there is a need for constructing rapid, simple, and ultra-sensitive diagnostic tools.

In this study, a nanocomposite immunosensing platform was fabricated using a glassy carbon (GC) electrode modified with graphene quantum dots (GQDs), gold nanoparticles (AuNPs), and a conducting polymer layer (a polypyrrole derivative). The sensing interface was prepared by deposition of GQDs, non-electrochemical growth of AuNPs, and electropolymerization of pyrrole-3-carboxylic acid (Py-3-COOH). Free carboxyl groups on the polymer surface enabled covalent immobilization of specific anti-CA-125 antibodies via EDC/NHS coupling. The modified platform surface was thoroughly characterized, and CA-125 antigen detection was evaluated using cyclic voltammetry and electrochemical impedance spectroscopy.

The experimental data verified that the PPy-3-COOH layer provides a stable platform for immobilizing antibodies. Detailed electrochemical analyses allowed for the determination of a full calibration curve across the target concentration range. Furthermore, the immunosensor exhibited exceptional signal reproducibility, robust polymer film stability, and selectivity, underscoring its applicability for possible clinical diagnostics.

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Young Scientist poster session

[IV-13] EFFECTS OF PHOTOPERIOD LENGTH ON GROWTH AND BIOMASS PRODUCTION OF THREE DUCKWEED SPECIES AT A CONSTANT DAILY LIGHT INTEGRAL

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Duckweeds have considerable potential for biomass production and wastewater treatment, but their cultivation requires appropriate lighting conditions. The daily light integral (DLI) quantifies the total amount of photosynthetically active radiation received per day. However, equal daily light inputs delivered through different combinations of photoperiod and light intensity may not support equivalent growth. This study evaluated the responses of three duckweed species to different photoperiods at a constant DLI.

Lemna minor, Landoltia punctata, and Spirodela polyrhiza were cultivated in Steinberg medium for 14 days under photoperiods of 4, 8, 12, 16, 20, and 24 h of light per day. Photosynthetic photon flux density was adjusted inversely to photoperiod duration, ranging from 900 to 150 μmol m^{-2} s^{-1}, to maintain a DLI of 12.96 mol m^{-2} d^{-1}. Relative growth rates (RGRs) based on frond number and surface area were determined after 7 days, while dry biomass was measured after 14 days.

The 4 h photoperiod produced the lowest mean RGRs in all species: 0.208, 0.184, and 0.181 d^{-1} based on frond number and 0.235, 0.165, and 0.153 d^{-1} based on surface area for L. minor, L. punctata, and S. polyrhiza, respectively. The corresponding maximum RGRs were 0.331, 0.270, and 0.347 d^{-1} for frond number and 0.352, 0.253, and 0.336 d^{-1} for surface area. In L. minor and L. punctata, neither RGR differed significantly among photoperiods of 8–20 h. Continuous illumination reduced both RGRs relative to 12–20 h in L. minor and 8–20 h in L. punctata. In S. polyrhiza, frond number-based RGR did not differ significantly among 12–24 h, whereas surface area-based RGR at 20 h significantly exceeded all other treatments. Dry biomass was also lowest at 4 h, averaging 0.053, 0.049, and 0.096 g for L. minor, L. punctata, and S. polyrhiza, respectively. In L. minor, the highest mean dry biomass occurred at 24 h (0.495 g), with no significant differences among 16–24 h. In L. punctata, dry biomass at 16 and 20 h (0.370 and 0.399 g, respectively) was significantly higher than in all other treatments. In S. polyrhiza, the highest mean dry biomass occurred at 24 h (0.797 g) and significantly exceeded values at 4–16 h but did not differ significantly from 20 h.

A constant DLI did not ensure equivalent duckweed growth or biomass production. Longer photoperiods combined with lower light intensities generally supported better performance than the shortest photoperiod. However, continuous illumination offered no consistent advantage across species and endpoints. Optimizing duckweed cultivation therefore requires species-specific selection of photoperiod and light intensity, while distinguishing early growth responses from final biomass yield.

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[IV-14] DESIGNING THE SURFACE ARCHITECTURE OF TITANIUM VIA PLASMA ELECTROLYTIC OXIDATION WITH ORGANIC-INORGANIC PARTICLES

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Titanium and its alloys are commonly used for designing the bone implants, however the bioinertness of titanium oftentimes limits osseointegration. Plasma Electrolytic Oxidation is a tool allowing us to tailor the titanium surface by developing porous, ceramic layers doped with functional ions and compounds (containing e.g. Ca and Sr). While calcium is the main element building the bone tissue, strontium is well known for promoting osteogenesis and hindering osteoclast activity. In this study innovative approach was taken for formation of the pectin-based particles with inorganic compounds (CFP) as a part of solution for anodizing process.

The PEO coatings were formed on titanium employing two electrolytes: aqueous solution of 0.1M K2HPO4, and the same solution supplemented with 5 g/L of CFP. To investigate the voltage influence on the process, three values of voltage peak–to–peak were employed (430 V, 450 V, 500 V). All the samples were processed using constant current density of 100 mA/cm2. The morphological, internal and crystallography characteristics were analyzed using scanning electron microscopy (Hitachi, 10kV SEM), coupled with EDS (QUANTAX, Brucker), optical profilometry and X-ray diffraction (EMPYREAN, Cu Kα).

The topography and internal structure of the obtained coatings were highly dependent on the employed voltage. High voltage of 500 V induced high energy plasma discharges resulting in formation of micro-cracks on the surface, but with high roughness (Sa = 0.71 μm). In contrast, the addition of pectin-based CFP acted as a thermal buffer, yielding more compact internal architecture (coating thickness ranged from 3.72 – 5.64 μm) and changing the surface morphology. EDS scans revealed that the content of strontium in the coating ranged from 0.18 % at. under 430 V – 0.31 % at. under 500 V. XRD analysis confirmed the crystallization of anatase as the main titanium oxide phase, featured by the complex titanium-potassium phosphates. Crucially, optimal value of voltage was found to be 450 V where the strongest reflexes of anatase and crystalline phosphates were recorded.

The result of this study indicates on possibility of using pectin-based particles in PEO process, which is a novel approach for electrolyte design for obtaining coatings with new chemical composition with biofunctional properties.

[IV-15]* MXENE FUNCTIONALIZED ENZYMATIC SENSOR FOR SENSITIVE DETECTION OF PHENOLS

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Enzyme grafting onto electrode surfaces faces challenges such as limited stability, poor reproducibility, and reduced storage activity. To address these issues, we investigated a two-dimensional chitosan@MXene composite as an electrode material for enzyme immobilization. MXenes are highly conductive materials with tunable multilayer-to-few-layer sheet structures, exhibiting excellent electrical conductivity, hydrophilicity, and the potential for further functionalization. These properties make them attractive for applications in drug delivery, biosensing technologies, antimicrobial systems, and bioimaging.

In this study, Ti3C2Tx MXene was synthesized through selective etching of the aluminum layer from Ti3AlC2 (MAX phase). The resulting MXene contains surface functional groups such as –OH, –O, and –F, providing a suitable platform for further modification with chitosan and enzyme immobilization. The model enzyme used in this work was tyrosinase (Tyr), an oxidoreductase containing two copper ions in its active site. Tyrosinase plays a key role in phenolic sensing due to its selective catalysis of the oxidation of phenolic compounds into quinones. These electroactive products can be reduced at the electrode surface, producing measurable current changes that enable the detection of environmentally relevant phenolic pollutants.

The formation of the chitosan@MXene composite and the covalent immobilization of Tyr on chitosan@MXene were confirmed by X-ray photoelectron spectroscopy, while surface microstructures were analyzed using scanning electron microscopy. The resulting Tyr–chitosan@MXene enzyme electrode demonstrated a wide detection range for phenolic derivatives (catechol) from 10 to 348.6 μM, high sensitivity (0.116 μA μM-1), and a low detection limit (0.35 μM).

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Young Scientist poster session

[IV-16]* SELF-ASSEMBLY OF NONIONIC AND ZWITTERIONIC AMPHIPHILIC POLYMERS: PHYSICOCHEMICAL CHARACTERIZATION AND COMPUTER-ASSISTED IMAGE ANALYSIS

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Amphiphilic polymers can spontaneously self-assemble into micellar nanostructures in aqueous environments, offering promising opportunities for the development of functional nanomaterials with potential biomedical applications. Understanding how polymer chemical structure influences micellization behavior is essential for the rational design of nanoscale systems with controlled properties. In particular, comparing nonionic and zwitterionic amphiphilic polymers may provide valuable insights into the role of polymer composition and intermolecular interactions in self-assembly processes.

The aim of this project is to investigate the relationships between polymer chemical structure, critical micelle concentration (CMC), and the size and morphology of the resulting micellar assemblies. An additional objective is to develop computational tools supporting quantitative analysis of polymer nanostructures.

To achieve these objectives, aqueous solutions of selected nonionic and zwitterionic amphiphilic polymers will be prepared over a range of concentrations. CMC values will be determined using appropriate physicochemical techniques, including spectroscopic, fluorescence-based, tensiometric, or conductometric methods. Dynamic light scattering (DLS) will be employed to determine the hydrodynamic diameters and size distributions of the formed assemblies. Their morphology, dimensions, and aggregation behavior will be further investigated using scanning electron microscopy (SEM), transmission electron microscopy (TEM), and/or atomic force microscopy (AFM). In parallel, computational image-processing methods, including image segmentation, object detection, particle size measurement, and morphological classification, will be implemented to develop a prototype for semi-automated microscopy image analysis.

The expected outcomes include a better understanding of structure–property relationships governing polymer micellization and the development of a computational approach enabling more systematic and reproducible characterization of self-assembled polymer nanostructures.

[IV-17]* IN VITRO ASSESSMENT OF MICROPLASTIC AND SYNTHETIC GLITTER TOXICITY IN HUMAN EPITHELIAL CELLS

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Microplastics (MPs), defined by the European Food Safety Authority (EFSA) as a heterogeneous mixture of polymeric particles ranging in size from 0.1 μm to 5 mm, represent one of the most serious contemporary environmental pollutants. The widespread presence of MPs in cosmetic products (e.g., in the form of glitter) poses a risk of direct exposure to the human body. The small particle size facilitates tissue penetration, which can induce an inflammatory response and exert cytotoxic effects.

The aim of this study was to evaluate the degree of cytotoxicity of industrial microplastics and synthetic glitter on human cell lines in vitro, including normal colon epithelial cells (CCD 841 Con), bronchial epithelial cells (BEAS-2B), and skin keratinocytes (HaCaT).

The effect of the tested particles on cell viability and metabolism was evaluated using the MTS assay across all tested cell lines (CCD 841 Con, BEAS-2B, HaCaT). Oxidative stress levels were determined by measuring reactive oxygen species (ROS) concentrations. Additionally, gene expression of key inflammatory mediators (IL6 and IL8) was analyzed.

A statistically significant, concentration-dependent reduction in cell viability was demonstrated across the evaluated cell populations (CCD 841 Con, BEAS-2B, and HaCaT). Exposure to the tested materials also led to a marked increase in intracellular ROS levels and upregulation of pro-inflammatory cytokine expression.

The obtained results indicate significant cytotoxic potential of microplastics and synthetic glitter toward human intestinal, respiratory, and skin epithelial cells. Induction of oxidative stress and activation of pro-inflammatory pathways represent key mechanisms underlying the cellular toxicity of the investigated particles, highlighting a potential threat to tissue barrier homeostasis (intestinal, pulmonary, and cutaneous) and the immune system.

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Young Scientist poster session

[IV-18]* DESIGNING DEDICATED MICROFLUIDIC CHIPS FOR SPECIALIZED BIOMEDICAL RESEARCH ON CELLS AND CELL CYCLE ANALYSIS

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Researching complex cellular processes, such as drug internalization and cell cycle progression, is complicated and requires controlling many variables. For this reason, traditional methods for assessing drug penetration and cell cycle dynamics, such as spectrofluorometry or flow cytometry, provide data averaged across the entire cell population and are less precise because they cannot observe process dynamics over time at the single-cell level. However, microfluidic systems overcome this limitation. With them, researchers can precisely set and replicate substance concentrations, contact time, and hydrodynamic conditions across experiments. Importantly, cells cultured under flow conditions exhibit different morphology and metabolic activity than those in conventional cultures, making the model more biologically relevant. These factors can also directly influence drug-transport kinetics and cell-cycle regulation. Unfortunately, despite their many advantages, microfluidic systems are very complex to design and manufacture. The design process itself is important and requires coordination and knowledge sharing among the entire research team. This study showcases an improved process of designing microfluidic chips based on rapidly changing experimental parameters and manufacturing limitations that was developed by our team.

Initially, sequential handoffs between experimentalists, 3D designers, and makers delayed design changes. We streamlined this by merging design and fabrication, introducing cross-team members, and jointly defining parameters, materials, sensors, and equipment upfront. 3D models are analyzed early to plan production steps and resolve constraints before making test chips. Using a 40W CO2 laser (glass, PMMA) and punched PDMS, we iterated from cracking engraved glass (1x20 mm chambers) and unevenly ablated 5 mm PMMA to a uniform stack: glass with CO2 strips, cut 0.5 mm PMMA channels, 4 mm PMMA, and PDMS ports. Fluidic tests led to 1.2 mm chamber spacing for dual-strip imaging and 15x7 mm hexagonal chambers (from 8x15 mm) to prevent air bubbles. Finally, UV-gluing needles prevented port leaks, while pressing glass-PMMA separately from PDMS protected the photosensitive strips before testing.

In conclusion, good, fast communication is necessary when designing microfluidic chips for rapidly evolving experiments. However, the research team's understanding of limitations and proven design choices and manufacturing methods, as well as having a single dedicated person responsible for accepting and adapting the design, are critical for successful and efficient microfluidic chip production.

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Young Scientist poster session

[IV-19]* DEVELOPMENT OF A DUAL LASER MANUFACTURING STATION FOR MULTIMATERIAL PROTOTYPING OF MICROFLUIDIC CHIPS WITH ADDITIVE AND SUBTRACTIVE MANUFACTURING

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Microfluidic chips require the precise fabrication of complex micro-scale elements to function reliably. Contemporary designs often integrate multiple materials, multi-level overlapping microchannels, and internal sensors. Coupled with strict biocompatibility and sterility standards, these demands challenge conventional single-process additive or subtractive methods. Building on the Biotechnology Center's laser processing technology and expertise, we expanded our custom laser workstation with a dedicated additive manufacturing subsystem. This study examines the hardware architecture, software integration, and operational workflow of this multimode system, evaluating its advantages for advanced microfluidic fabrication and experimental research.

Previously, we developed a custom laser workstation integrated with an Olympus microscope for micron-scale patterning via laser ablation. While effective for standard prototypes, it's strictly subtractive nature limited geometric complexity. To overcome this, we upgraded the setup for laser-based additive manufacturing *via* selective photolithography. Although initial parameter tuning allowed the primary ablation laser to cure photoresists like SU-8, the resolution was insufficient for intricate micro-features. Since photolithography requires much lower optical power than ablation, we adapted the microscope's scanning laser as a secondary source for high-resolution additive curing, reserving the primary laser for bulk ablation and large-area structuring. Mounted opposite the primary source, the scanning laser enables simultaneous dual-sided processing - ablating the substrate on one side while additively curing structures on the other. Custom control software with advanced path-planning algorithms unifies the dual-laser hardware, optimizing exposure times and alignment accuracy. Fabricated prototype chips demonstrated high structural fidelity, complex geometries, and seamless, single-step integration of functional electronic components, such as sensing microelectrodes.

In conclusion, combining subtractive laser ablation with high-precision photolithography in one optical workstation bridges the gap between micro-machining and 3D micro-printing. By eliminating multi-station alignment errors and reducing fabrication steps, this hybrid system enables the rapid, cost-effective production of multi-material, sensor-integrated microfluidic devices tailored to demanding bioengineering applications.

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Young Scientist poster session

[IV-20]* NATURAL POLYMER-BASED HYDROGELS AS CARRIERS OF BIOACTIVE SUBSTANCES IN ANTICANCER THERAPY

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The widespread use of hydrogels in biotechnology - including drug delivery systems, tissue engineering, food technology, cosmetics, and environmental remediation - necessitates the development of production methods that yield materials with specific physicochemical and mechanical properties [1]. To this end, composite systems comprising two or more polymer components, subjected to single- or multi-stage cross-linking, are frequently employed. The resulting three-dimensional network ensures the hydrogel's structural integrity and enables it to swell in water. Due to the presence of numerous functional groups and structural diversity, natural polymers offer extensive possibilities for hydrogel modification, allowing for the creation of advanced materials with unique and enhanced properties.

This paper presents hydrogel materials derived from natural polymers and composite materials fabricated using various cross-linking techniques. Chitosan, alginate, and hyaluronic acid were selected as the polymers, while glutamic acid, carboxymethylcellulose, and glycerophosphate were used as cross-linking agents. Hydrogels capable of encapsulating active substances used in cancer therapy were developed, enabling the sustained release of the biologically active compound at the tumor site. Additionally, hybrid systems consisting of a porous inorganic carrier and a hydrogel were obtained; these systems facilitate transport to cancer cells and the subsequent release of the active substance from a prodrug form. The developed bioencapsulation systems may find application in the encapsulation of proteins, including enzymes and biologically active biomolecules.

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Young Scientist poster session

[IV-21]* THE SIGNIFICANCE OF MICRO- AND NANOPLASTIC PARTICLE SIZE ON THEIR INTERACTION WITH HUMAN CELLS IN VITRO

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The growing environmental pollution caused by minute plastic fragments currently poses a severe challenge to public health. Microplastics (particles ranging from 0.1 μm to 5 mm in diameter) and even smaller nanoplastics are the products of multi-stage degradation - including physical, chemical, and biological processes - of commonly used polymers such as polystyrene and polypropylene. Due to their miniature dimensions, these contaminants (originating from packaging, construction materials, and textiles) easily permeate water, food, and air. A crucial factor determining their impact on living organisms is their physicochemical profile, predominantly particle size.

The primary objective of this work is to evaluate how the size scale of micro- and nanoplastic particles dictates their effects on human cells in in vitro models. Based on a comprehensive review of scientific literature, the potential biological mechanisms underlying these interactions were analyzed. The gathered data was used to demonstrate a direct correlation between the dimensions of polymeric particles and the nature and intensity of their impact on the human body at the cellular level.

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Young Scientist poster session

[IV-22]* FINITE ELEMENT ANALYSIS OF MANDIBULAR CONDYLAR NECK FRACTURE RISK UNDER DIFFERENT LOADING DIRECTIONS

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Mandibular condylar fractures are clinically significant due to the complex geometry of the condyle and the mechanical loads transmitted through the mandible during mastication. Finite element analysis (FEA) can be used to investigate stress distribution and identify regions that may be susceptible to fracture under different loading conditions. Accurate evaluation of local stress concentrations requires suitable mesh refinement and consideration of the loading direction.

The aim of this study was to evaluate stress distribution in the mandible under different loading directions and identify regions with a higher risk of fracture. A three-dimensional mandibular model was analyzed using the finite element method in Siemens NX (Simcenter 3D). A mesh convergence study was conducted using element sizes of 0.30, 0.25, and 0.20 mm to evaluate the effect of mesh refinement on the calculated stress values. A load of 700 N was applied in three directions: lateral, oblique, and straight. The resulting stress distributions were compared to determine the influence of loading direction and identify critical regions with potential fracture risk.

The results showed that the mandibular condylar neck was particularly sensitive to the applied loading and was identified as a critical region for potential fracture. Stress distribution differed depending on the loading direction, indicating that the direction of force application should be considered when evaluating the structural integrity of the mandible.

The findings show that FEA can help identify regions of the mandible that are susceptible to fracture under different loading conditions. This study provides a basis for further investigation of mandibular condylar fracture mechanisms and may contribute to the evaluation of load-bearing capacity in mandibular implant design.

Keywords: Finite element analysis (FEA); Mandibular condyle; Condylar neck fracture; Loading direction; Mesh convergence; Stress distribution; Biomechanics.

Young Scientist poster session

[IV-23]* TITLE: NUMERICAL STRESS MODELING USING FEM IN PATIENT-SPECIFIC DESIGN OF REMOVABLE COMPLETE DENTURES

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Frequent denture fractures during mastication have financial consequences for patients and are time-consuming for dentists. Currently, CAD/CAM technology is increasingly being used in the design and manufacture of removable complete dentures. However, there is a lack of methodology for studying the stress state using the finite element method (FEM) in complete dentures in accordance with engineering principles and analyzing the effect of finite element division on stress values.

The hypothesis of this work was that it is possible to numerically assess the stress state in a patient-specific denture during load transfer on an anatomical foundation. A three-dimensional model of a lower complete denture was designed in a dental program (Exocad), and FEM analysis was performed in FEM software (Nx Siemens) with various mesh densities (element sizes: 0.8 mm, 0.5 mm, and 0.3 mm). A scenario of symmetric loading of a denture on molar teeth was simulated, with vertical forces (100 N) and oblique forces of 140 N at a 45-degree angle. The soft foundation had a Young's modulus of E=5 MPa, and the denture material was typically E=2 GPa.For oblique forces in the anterior region, the nominal bending stress was increased but significantly lower than the flexural strength of the denture material. Only around the anatomical transitions between teeth and gingival grooves were concentrations close to the fatigue strength range.

The study demonstrated that with a refined mesh in finite element analysis (FEA), it was possible to predict nominal bending stresses and concentrations around anatomical irregularities. This provides a basis for the design of patient-specific dentures based on engineering durability criteria.

Young Scientist poster session

[IV-24]* ASSESSMENT OF THE INFLUENCE OF ENVIRONMENTAL CONDITIONS AND TIME ON THE DEGRADATION PROCESS OF CROSSLINKED POLY(GLYCEROL SEBACATE) (PGSU) IN THE CONTEXT OF BIOMEDICAL APPLICATIONS

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Biomaterials play a significant role in modern biomedical engineering, finding applications in implantology, tissue engineering, and controlled drug delivery systems, among others. The dynamic development of this field is associated with the growing demand for biocompatible and biodegradable materials capable of supporting tissue regeneration and gradually breaking down within the body's environment.

The research focused on a biodegradable polymeric biomaterial - poly(glycerol sebacate) (PGS). The synthesis and crosslinking of the material were performed, and subsequently, its degradation process was analyzed under various environmental conditions and as a function of time. As part of the study, qualitative and quantitative analyses were conducted, including the evaluation of changes in the mass, chemical structure, and surface morphology of the samples.

The obtained results demonstrated a significant impact of the degradation environment and exposure time on the properties of the investigated biomaterial. The observed changes confirm the ability to control the degradation process of the tested biopolymer, indicating its high potential for application in tissue engineering and other biomedical systems requiring materials with controlled biodegradability.

Young Scientist poster session

Session V: Bioactive Compounds and Medicinal Chemistry

[V-1] ETOPOSIDE-LOADED CROSS-LINKED MICROSPHERES – FORMULATION AND CHARACTERIZATION

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Microspheres are versatile drug delivery systems that enable the encapsulation and protection of a wide range of therapeutic agents, as well as the controlled release of active substances. Drugs can be physically dispersed within the polymer matrix or chemically bonded to it. The latter approach requires the presence of suitable functional groups within the polymer structure. Furthermore, functional groups exposed on the microsphere surface can be used to modify their physicochemical properties, such as hydrophilicity, or to attach targeting ligands that facilitate the selective delivery of drugs to specific cells or tissues.

Cross-linked microspheres with a stable, highly developed porous structure constitute an interesting group of polymer microspheres. Depending on their properties—such as chemical structure, particle size, and mechanical and thermal strength—these cross-linked microspheres find wide-ranging applications, including as carriers for controlled drug release, supports for enzyme immobilization, catalysts, and porous substrates for protein synthesis.

The aim of this study was to prepare and characterize polymer–drug systems in the form of microspheres based on poly(ester-anhydrides) containing allyl groups in their side chains, cross-linked via a free-radical-initiated thiol–ene reaction. The microspheres were obtained by emulsion (O/W) solvent evaporation method. Etoposide, an anticancer drug used in the treatment of small-cell lung cancer, was encapsulated in polymer carriers.

For obtained polymer-drug systems, parameters such as encapsulation efficiency and drug loading were determined. The encapsulation efficiency and drug loading were dependent on the particle size and the starting amount of the drug.

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[V-2] FUCOSYLTRANSFERASE INHIBITORS - DESIGN, SYNTHESIS AND EVALUATION OF BIOLOGICAL ACTIVITY

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The fucosylation of glycans and glycoconjugates plays an important role in numerous biological processes, including cell adhesion, angiogenesis, the immune response, cancer progression and metastasis formation. Fucosyltransferases (FUTs) are enzymes belonging to the glycosyltransferase class (EC 2.4.1.) that catalyze the transfer of L-fucose units from a donor substrate (GDP-β-L-fucose) to an acceptor substrate (an oligosaccharide, glycopeptide, or glycolipid). Thirteen distinct fucosyltransferases have been identified in the human genome and are classified into subtypes based on the glycosidic linkages they form [1,2].

Among all fucosyltransferases, fucosyltransferase 8 (FUT8), which catalyzes core fucosylation of glycoproteins, plays a significant role in cancer progression, immune evasion and the development of drug resistance. Therefore, FUT8 represents a promising therapeutic target. However, the development of selective inhibitors of this enzyme remains a major challenge due to similarities in substrate specificity among individual fucosyltransferases [2,3].

Based on literature data and previous studies, novel compounds with potential inhibitory activity against FUT8 were designed. As part of this study, 2-fluorinated sugar derivatives were synthesized and subsequently conjugated with derivatives of mucochloric acid containing either a furan-2(5H)-one or a 2H-pyrrol-2-one scaffold. The resulting compounds were subjected to a preliminary evaluation of their biological activity using a selected cell line characterized by FUT8 overexpression.

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[V-3] TARGETING IRON METABOLISM IN GLIOBLASTOMA: ANTICANCER POTENTIAL OF TERPYRIDINE–THIOSEMICARBAZONE DERIVATIVES

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Cancer remains a major global health challenge, highlighting the need for novel therapeutic strategies. Cancer cells exhibit an increased demand for iron and altered iron homeostasis, which support their proliferation and contribute to their iron-dependent phenotype. Consequently, targeting iron metabolism represents a promising approach to inhibiting tumor growth [1,2].

TST derivatives are a novel class of iron chelators combining thiosemicarbazone (TSC) and terpyridine moieties, both characterized by strong iron-binding properties. Their incorporation into a single molecular structure offers potential for the development of new anticancer agents. This study investigated the cytotoxic activity, iron-chelating properties, and potential mechanisms of action of TST derivatives in glioblastoma models. Spectrophotometric analyses confirmed the ability of the investigated compounds to chelate iron ions.

Cytotoxicity was initially evaluated in two-dimensional (2D) cultures of human glioblastoma cell lines, including T98G, U251, LN229, and LN18. The most active derivatives were subsequently examined in a three-dimensional (3D) U251 model, providing a more physiologically relevant representation of the tumor microenvironment [3]. Additionally, blood–brain barrier (BBB) studies were performed to assess the potential of these compounds for central nervous system drug delivery. The experiments yielded promising results, further supporting their potential application in glioblastoma treatment.

To investigate the mechanisms underlying their cytotoxic effects, reactive oxygen species (ROS) generation and the expression of selected iron-related proteins were analyzed. TST treatment increased ROS levels in the 3D model, suggesting the involvement of oxidative stress in their anticancer activity. Furthermore, changes in the expression of divalent metal transporter 1 (DMT1), mitoferrin 1 (MFRN1), and frataxin (FXN) indicated disturbances in cellular iron homeostasis.

Overall, these findings demonstrate the promising anticancer potential of TST derivatives against glioblastoma. Their iron-chelating activity, induction of oxidative stress, modulation of iron-related proteins, and encouraging BBB results provide a strong foundation for further investigation. However, additional studies are required to clarify their mechanisms of action and evaluate their selectivity toward cancer cells.

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[V-4] INVESTIGATING THE MECHANISM OF ACTION OF NOVEL SCHIFF BASES AS TARGETED THERAPEUTICS FOR NON-SMALL CELL LUNG CANCER AND GLIOBLASTOMA

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Schiff bases, also known as imines, were first obtained by Hugo Schiff in 1864. They are formed by the condensation of primary amines with carbonyl compounds, i.e. aldehydes or ketones. Although traditionally employed as dyes, catalysts and polymer stabilizers, these compounds are now attracting growing attention in pharmacology and molecular biology. Their diverse biological profile includes antiproliferative, antimicrobial and anti-inflammatory effects [1]. Of particular interest are structures in which the imine group is conjugated with aromatic heterocycles such as quinoline or coumarin. Such fragments strongly shape the physicochemical characteristics of the molecule, providing suitable lipophilicity, the ability to chelate metal ions and distinctive optical behaviour. As a result, Schiff bases are increasingly recognized as promising cellular stains and diagnostic probes. Owing to their capacity to bind zinc ions, they can act as "smart" sensors responsive to both pH and metal ions, enabling dynamic, real-time tracking of processes taking place inside cells [2-3].

In the present study, we investigated a new series of Schiff bases built on a quinazolone scaffold. Their antiproliferative potential was screened against a panel of cancer cell lines, among them the non-small cell lung cancer (NSCLC) lines PC-9 and A549 as well as glioblastoma (GBM) line U251. The cytotoxicity data identified compound SXIII as particularly active towards PC-9 cells, additionally compounds SI and SII were active towards U251 cells. These derivatives were therefore selected for detailed mechanistic studies aimed at elucidating their mode of action. We focused mainly on their influence on cell cycle progression, as well as on the expression of key proteins that regulate the cell cycle. Importantly, treatment with SXIII also led to marked reorganization of the cytoskeleton. Taken together, these findings highlight the studied Schiff bases as promising leads for the development of novel anticancer agents, especially for NSCLC and GBM therapy.

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[V-5] SULFONATED STYRYLQUINAZOLINES BLOCK AKT/MTORC1-DEPENDENT TRANSLATIONAL SIGNALING AND ARREST EPITHELIAL CANCER CELLS IN MITOSIS

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Aberrant cell cycle control and persistent activation of the PI3K/Akt pathway drive the progression of epithelial malignancies such as lung and colorectal carcinomas, highlighting the value of small molecules capable of targeting pro-survival signaling and mitotic regulation at the same time. Our group has recently investigated a series of sulfonated styrylquinazoline derivatives, assessing their antiproliferative activity in epithelial cancer models together with their impact on intracellular signaling, cell cycle progression, and apoptotic cell death. Analogues bearing a chlorine substituent on the quinazoline scaffold inhibited the growth of colon, lung, breast, and pancreatic cancer cells at submicromolar concentrations, while showing selectivity over normal human fibroblasts. In colon cancer and non-small cell lung cancer (NSCLC) cells, the most potent derivatives caused accumulation of cells in the G2/M phase. This was accompanied by a pronounced reduction in Akt phosphorylation at Ser473 and by impaired mTORC1-mediated translational signaling, as reflected in decreased phosphorylation of p70 S6 kinase and 4E-BP1. In parallel, the compounds altered the expression of key cell cycle regulators, modulating cyclin and Aurora A levels and upregulating GADD45, a pattern indicative of sustained mitotic arrest that may involve interference with microtubule dynamics. Ultimately, exposure to the compounds triggered extensive apoptosis, marked by cleavage of caspase-9, which points to engagement of the intrinsic pathway, as well as cleavage of PARP-1. Taken together, our results position sulfonated styrylquinazolines as potent and selective agents that suppress Akt-dependent survival and translational programs, enforce mitotic arrest, and induce apoptotic cell death, supporting their further investigation in genetically diverse cancer models.

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[V-6] PHYTOCHEMICAL PROFILING OF PROPOLIS EXTRACT AND ITS EFFECTS ON CELL VIABILITY AND APOPTOSIS IN COLORECTAL CANCER CELLS

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Introduction: Propolis is a natural resinous substance produced by honey bees from plant-derived materials, characterized by a complex chemical composition rich in bioactive compounds, including polyphenols. Owing to its diverse biological activities, including antioxidant, anti-inflammatory, and anticancer effects, propolis has attracted considerable interest as a potential source of compounds with therapeutic properties.

Aim of the study: The aim of this study was to characterize the phytochemical profile of a propolis extract (PE) and subsequently evaluate its effects on cell viability and its ability to induce apoptosis in two human colorectal cancer cell lines, HCT116 and SW480.

Materials and methods: The effects of PE on the viability of HCT116 and SW480 colorectal cancer cells were evaluated using the MTT assay after 24, 48, and 72 h of exposure to PE at concentrations ranging from 12.5 to 400 µg/mL. 5-Fluorouracil (5-FU) at concentrations of 50 and 100 µM was used as a reference cytotoxic agent. The half-maximal inhibitory concentration (IC_{50}) was calculated from the concentration–response curves. Apoptosis and cell membrane integrity were assessed by flow cytometry using the FITC Annexin V Apoptosis Detection Kit.

Results: UHPLC-DAD analysis revealed that the PE was rich in polyphenolic compounds, predominantly hydroxycinnamic acids (2234.57 mg/100 g DW), particularly p-coumaric acid (1322.82 mg/100 g DW) and ferulic acid (615.68 mg/100 g DW). Among the identified flavonoids, kaempferol (122.99 mg/100 g DW) and apigenin (116.05 mg/100 g DW) were the most abundant. PE reduced cell viability in a concentration-dependent manner, with IC_{50} values after 48 h of 40 µg/mL for HCT116 and 55 µg/mL for SW480, indicating higher sensitivity of HCT116 cells. Flow cytometric analysis confirmed a reduction in the proportion of viable cells and an increase in early apoptosis in both cell lines. At 75 µg/mL PE, the proportion of viable cells decreased to 44% in HCT116 and 39% in SW480, while early apoptotic cells increased to 30% and 36%, respectively. The effects of 75 µg/mL PE on cell viability and apoptosis were similar to those observed following treatment with 50 µM 5-FU.

Conclusions: The PE reduced cell viability and induced apoptosis in HCT116 and SW480 colorectal cancer cells, with HCT116 cells showing greater sensitivity based on IC_{50} values. These findings suggest that PE is a promising source of bioactive compounds for further investigation of their potential anticancer activity against colorectal cancer.

[V-7] EFFECTS OF HIPPOPHAE RHAMNOIDES LEAF EXTRACTS ON CELL VIABILITY AND CELL DEATH IN HORMONE-DEPENDENT AND TRIPLE-NEGATIVE BREAST CANCER

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Introduction: Breast cancer (BC) is one of the most commonly diagnosed malignancies worldwide, indicating the urgent clinical need for continuous therapeutic innovation to overcome drug resistance and treatment-related toxicity. One promising approach involves investigating plant-derived extracts containing various bioactive compounds that may inhibit tumor progression while potentially exhibiting more favorable safety profiles. The plant deserving special attention in the context of chemoprevention is sea buckthorn (SB) (Hippophae rhamnoides). Studies indicate that various parts of this plant, including its leaves, may reduce BC cell viability and induce apoptosis.

Aim of the study: This study aimed to evaluate the total polyphenol content, antioxidant capacity, and anticancer activity of two sea buckthorn leaf extracts (SBLEs) prepared using 60% (SBLE60) and 70% (SBLE70) ethanol. Moreover, the effects of these extracts on BC cell viability and their ability to induce apoptosis were investigated.

Materials and Methods: The MTT assay was conducted after 24, 48, and 72 hours with SBLE60 and SBLE70 to check their impact on the viability of triple-negative MDA-MB-231 and hormone-dependent MCF7 breast cancer cells. The extracts were tested across a wide concentration range of 50-1000 μg/mL. 5-fluorouracil (50 and 100μM) was used as a positive control. Based on the MTT results, two concentrations of each extract were chosen for each cell line for further flow cytometric analysis using the FITC Annexin V Apoptosis Detection Kit I to assess apoptosis and necrosis following treatment with the tested extracts.

Results: Phytochemical analysis showed that SBLE60 exhibited higher total phenolic (30,040 vs. 27,203 mg GAE/100 g) and flavonoid contents (5,333 vs. 4,307 mg QE/100 g), as well as higher ABTS radical scavenging activity (319,835 vs. 238,603), than SBLE70. Both extracts caused a significant, time- and concentration-dependent decrease in the viability of both cell lines. Based on IC50 values obtained after 48 h of treatment, MCF7 cells were more sensitive to both extracts (150 and 153 μg/mL for SBLE60 and SBLE70, respectively) than MDA-MB-231 cells, which required higher extract concentrations to achieve a 50% reduction in viability (440 and 382 μg/mL for SBLE60 and SBLE70, respectively). Flow cytometric analysis also showed a significant decrease in cell viability following treatment with the extracts, accompanied by a significant increase in the percentage of apoptotic and necrotic cells compared with the control.

Conclusions: The results show that the extract can induce different effects depending on the BC subtype. Nevertheless, both SBLE60 and SBLE70 exhibit promising chemopreventive potential. In triple-negative BC, SBLE70 demonstrates greater cytotoxic activity than SBLE60.

[V-8] UNLEASHING NOVEL ALKOXYLATED FISETIN DERIVATIVES TO DRIVE NON-CANONICAL CELL DEATH IN HEAD AND NECK CANCERS

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Head and neck cancer (HNC) is a therapeutically challenging cancer what underscores the need for new chemical agents that selectively induce programmed cell death. Fisetin, a naturally occurring flavonoid, exhibits promising anticancer activity but displays limited proapoptotic efficacy and cell type selectivity. Here, we report the synthesis and comprehensive evaluation of alkoxylated fisetin derivatives (propynyloxy, 2-butynyloxy, and butyloxy) with improved physicochemical and biological properties.

In silico analyses revealed favourable drug-like characteristics, with propynyloxy derivatives displaying optimal oral bioavailability, enhanced predicted absorption and increased electrophilic potential. Functional assays across multiple HNC cell lines (CAL27, UCSF-OT, FaDu, A-253) demonstrated that fisetin derivatives significantly reduce cancer cell viability at low micromolar concentrations comparing to normal fibroblasts, indicating improved selectivity. Enhanced cytotoxicity of fisetin derivatives was associated with increased apoptosis. Moreover, cytotoxicity occurred independently of canonical DNA damage responses with no induction of γH2AX or p53. Time-lapse imaging revealed a distinct mechanism of action for these derivatives. They increased the frequency of mitotic entry, but cells subsequently failed to complete cytokinesis, which led to chromatin condensation and cell death. In contrast, fisetin primarily induced a cytostatic arrest at the G2/M phase and activated ERK1/2 signaling. Surprisingly, the propynyloxy derivative caused upregulation of cyclin D1 and p21 expression.

To summarize, these findings demonstrate that targeted alkoxylation of fisetin changes its activity to enhancing anticancer efficacy through non-genotoxic mechanisms. Propynyloxy modification induces a unique mechanism involving disruption of mitosis and selective induction of apoptosis in HNC.

[V-9] PLANTAGO LANCEOLATA AND XANTHIUM STRUMARIUM AERIAL-PART EXTRACTS MODULATE OXIDATIVE AND INFLAMMATORY RESPONSES OF ASPERGILLUS FUMIGATUS-CHALLENGED MACROPHAGES: HPLC PROFILING, CYTOTOXICITY AND ANTIFUNGAL POTENTIAL

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Ethnopharmacological relevance: *Plantago lanceolata* L. (*Plantaginaceae*) is a European remedy for cough and inflammation of the throat and mouth. *Xanthium strumarium* L. (*Asteraceae*) is used in traditional Chinese medicine to treat rhinosinusitis and allergic rhinitis. Whether these plants influence how the host responds to the airborne mould Aspergillus fumigatus has not been investigated.

Aim of the study: To profile ethanolic extracts from the aerial parts of both species. We also aimed to determine whether the extracts are safe for epithelial cells and macrophages. Finally, we assessed how they affect macrophage oxidative and cytokine responses to *A. fumigatus* and whether they act directly on the fungus.

Materials and methods: The phytochemical composition was characterised by UV–Vis spectroscopy and a validated RP-HPLC method. Antioxidant capacity was measured with the DPPH, FRAP and CUPRAC assays. Cell viability was tested in HaCaT keratinocytes, BEAS-2B bronchial epithelial cells and RAW 264.7 macrophages. In RAW 264.7 cells stimulated with PMA or *A. fumigatus* conidia, ROS generation was recorded by luminol-enhanced chemiluminescence. TNF-α and IL-10 release was quantified by ELISA. Conidial germination and hyphal growth were assessed microscopically.

Results: Aucubin (11.63 mg/g) and verbascoside (7.93 mg/g) dominated the *P. lanceolata* extract. Dicaffeoylquinic acid (0.78 mg/g) was the main quantified compound in *X. strumarium*. *P. lanceolata* did not reduce macrophage viability at concentrations up to 2 mg dry plant equivalent (DPE)/mL. *X. strumarium*, by contrast, was cytotoxic from 0.5 mg DPE/mL. At non-toxic concentrations, both extracts inhibited PMA-triggered ROS production. *X. strumarium* reduced TNF-α secretion by fungus-pulsed macrophages by about 50%, with no change in IL-10. Only *X. strumarium* inhibited hyphal growth, and only at 1–2 mg DPE/mL. *P. lanceolata* had no antifungal effect.

Conclusions: Both extracts attenuate the oxidative burst of macrophages. *X. strumarium* additionally limits TNF-α release induced by *A. fumigatus*. These results partly support the traditional use of both plants in inflammatory airway conditions. Direct anti-Aspergillus activity is marginal and occurs only at concentrations toxic to host cells.

[V-10] HYPERICIN AS A NATURAL PHOTOSENSITIZER: INFLUENCE OF LIPOSOMAL FORMULATION ON OXIDATIVE STRESS AND PHOTOCYTOTOXICITY IN BREAST CANCER CELLS

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Natural bioactive compounds are attracting interest as potential therapeutic agents owing to their diverse biological activities. Photodynamic therapy (PDT) is a treatment approach that involves the activation of a photosensitizing compound by light of an appropriate wavelength, resulting in the generation of reactive oxygen species (ROS) and subsequent cellular damage. Hypericin, a naturally occurring photosensitizer found in Hypericum perforatum, exhibits promising light-dependent cytotoxic activity. However, its hydrophobicity, poor aqueous solubility, and tendency to aggregate limit its potential biomedical applications. Liposomal encapsulation may help overcome these limitations by improving the delivery of natural photosensitizers, modulating their biological activity, and expanding their potential therapeutic applications. This study investigated the effects of passive and active liposomal loading strategies on hypericin-mediated PDT in human breast cancer cells. Hypericin was incorporated into liposomes either passively, within the lipid bilayer, or through an active loading strategy involving copper(II) sulfate. MCF-7 and MDA-MB-231 cells were incubated with free or liposomal hypericin and subsequently exposed to orange light (approximately 590 nm; 7.2 J/cm²). Cytotoxicity in the absence and presence of irradiation was evaluated using the MTT assay, while intracellular ROS-associated fluorescence was assessed by flow cytometry. Under the tested conditions, the liposomal formulations showed limited dark cytotoxicity, whereas irradiation reduced cell viability. The MTT assay revealed cell line-dependent differences in photocytotoxicity, with lower IC_{50} values observed in MDA-MB-231 cells compared to MCF-7 cells, indicating greater sensitivity of MDA-MB-231 cells to hypericin-mediated PDT. Photodynamic treatment increased intracellular ROS-associated fluorescence, with responses differing between cell lines and hypericin formulations. In MDA-MB-231 cells, actively loaded liposomes yielded a higher ROS-associated fluorescence signal than passively loaded liposomes. These findings indicate that the loading approach may influence the cellular effects of hypericin-mediated PDT. Overall, liposomal hypericin demonstrated light-dependent cytotoxic activity in breast cancer cell models, supporting further investigation of formulation-dependent differences in photodynamic response.

[V-11] LIPOSOMAL CURCUMIN AND RUTHENIUM(II) COMPLEX AS A PHOTOSENSITIZER FOR PHOTODYNAMIC THERAPY OF SQUAMOUS CELL CARCINOMA AND MELANOMA: AN IN VITRO AND IN OVO CAM STUDY

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Photodynamic therapy (PDT) is a promising approach to cancer treatment that combines a photosensitizing agent with light of an appropriate wavelength to induce selective cytotoxic effects. However, the therapeutic potential of photosensitizers may be limited by poor aqueous solubility, insufficient bioavailability, and non-specific distribution. Liposomal delivery systems may help overcome these limitations and improve the effectiveness of PDT. Curcumin, a natural compound with photosensitizing properties, and ruthenium(II) complexes represent promising candidates for the development of novel anticancer strategies. This study aimed to investigate the comparison of the photodynamic efficacy of free curcumin and of liposome-encapsulated curcumin and ruthenium(II) complex against squamous cell carcinoma and melanoma cell lines and to explore the underlying mechanisms of their anticancer activity.

Methods: The phototoxic effects of the investigated substances were evaluated *in vitro* using human melanoma MUG-Mel2 and squamous cell carcinoma SCC-25 cells, with non-malignant cells included as controls. The cytotoxic response was assessed using the MTT assay following photosensitizer exposure and irradiation. Flow cytometry was used to investigate cell death and ROS generation. Additional analyses were investigated using the chick chorioallantoic membrane (CAM) model to evaluate their antitumor activity *in ovo*.

Results: A comparison of free curcumin and the liposomal curcumin-ruthenium(II) complex revealed no cytotoxic effect of the complex without irradiation. Furthermore, differences in the responses of the tested cancer cell lines and non-malignant control cells indicated cell-line-dependent effects. Apoptosis and ROS generation increased following treatment compared with the control groups. The results obtained using the CAM model were consistent with the *in vitro* findings.

Conclusions: This study explores free curcumin and liposomal curcumin with a ruthenium (II) complex as potential photosensitizing formulations for PDT of skin cancer. Combining *in vitro* cellular assays with the innovative CAM model may provide complementary evidence regarding their antitumor efficacy and biological mechanisms of action, supporting further preclinical evaluation of the liposomal formulation of curcumin with a ruthenium (II) complex.

[V-12]* SPILANTHOL ENDOPEROXIDE: BIOLOGICAL ACTIVITY OF A NOVEL DERIVATIVE FROM ACMELLA OLERACEA

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Spilantol, a naturally occurring compound, exhibits a broad spectrum of biological activity. It is commonly used in cosmetology, but due to its good cellular penetration and ability to cross the blood-brain barrier, interest in spilanthol's pharmacological potential has been growing in recent years. However, spilanthol is rapidly metabolized and eliminated from the body, which limits its therapeutic potential.

In this study, we investigated the biological activity of a cyclic spilanthol derivative (SPLE), which is expected to have a longer half-life in the body. We conducted the studies on human cancer cell lines with wild-type p53 (HCT 116wt) and with a defective p53 gene (HCT 116 p53-/-). After incubating the cells for 24 hours with various concentrations (0–1000 μM) of the cyclic spilanthol derivative, cell viability was assessed using the CCK-8 assay. The effect of SPLE on the cell cycle was examined using a propidium iodide-based flow cytometry assay.

The results showed that the biological activity of SPLE depends on the status of the p53 protein. In p53 -/- HCT 116 cells, the observed cytotoxic effect is stronger than in wt HCT 116 cells. This indicates the anticancer potential of SPLE.

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Young Scientist poster session

[V-13]* EVALUATION OF THE CYTOTOXIC POTENTIAL OF NEXT-GENERATION PHOTOSENSITIZERS IN THREE-DIMENSIONAL SPATIAL MODELS OF HUMAN TUMORS

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Photodynamic therapy (PDT) represents a rapidly evolving field of non-toxic, targeted oncological strategies; however, its clinical efficacy in treating aggressive central nervous system tumors remains limited. A major challenge in the treatment of gliomas and neuroblastoma is the hindered transport of active molecules deep into the tumor structure and the presence of local hypoxia. Conventional 2D cell models, lacking complex tissue architecture, fail to fully replicate actual diffusion barriers, often leading to an overestimation of the therapeutic potential of new molecules during preclinical studies. Addressing these limitations, this study focuses on the use of spatial cell models (3D spheroids) to evaluate the properties and biological efficacy of next-generation photosensitizers, with particular emphasis on diclofenac derivatives. This three-dimensional system enables a reliable representation of tumor tissue structure, including oxygen and nutrient concentration gradients and the physical constraints on active substance transport. Microscopic analysis involves assessing the penetration and accumulation of the tested compounds within the spheroid structures, as well as identifying the molecular pathways of cell death induced by irradiation-triggered oxidative stress. Comparing the biological responses of 2D and 3D systems allows for a precise assessment of how tissue architecture influences cancer cell sensitivity and the selectivity of the treatment relative to normal cells. The obtained results represent a significant step in evaluating the suitability of new multifunctional systems for PDT, aligning with the trends of personalized medicine and computational oncology.

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Young Scientist poster session

Session VI: New Diagnostic and Therapeutic Approaches

[VI-1] INTERFACIAL ENGINEERING OF MXENE@CU-MOF HYBRIDS FOR ENHANCED ELECTROCHEMICAL HYDROGEN PEROXIDE DETECTION IN CELL ENVIRONMENTS

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Reactive oxygen species (ROS), particularly hydrogen peroxide (H2O2), play a significant role in cell signaling, tumor progression, and oxidative stress responses in cancer. Reliable detection of H2O2 in the cancer cell microenvironment is therefore essential for studying cancer-associated redox processes. However, electrochemical monitoring in biological systems requires sensing interfaces that are sensitive, stable, and cytocompatible. In this study, a MXene@Cu-MOF hybrid was developed as an electrochemical platform for H2O2 detection. The hybrid forms a porous three-dimensional architecture in which MXene nanosheets uniformly coat and separate the copper-based metal organic frameworks (Cu-MOF) crystals, thereby limiting aggregation while facilitating electron transfer and electrolyte accessibility. Structural and spectroscopic characterization confirmed the formation of an integrated heterostructure featuring interfacial chemical interactions and electronic coupling. Electrochemical measurements revealed that the H2O2 reduction current obtained with the hybrid was approximately three times higher than that observed with Cu-MOF alone. Under optimized conditions, the sensor exhibits a linear response range of 0.5 μM to 9 mM, a detection limit of 0.3 μM, and excellent selectivity against common electroactive interferents. The hybrid also demonstrates superior reproducibility and enhanced storage stability, retaining 78% of its initial response after six weeks. Furthermore, the observed cell viability reached 84.2 ± 8.9% with the hybrid, compared to 69.6 ± 9.5% with Cu-MOF alone. The platform was also evaluated for H2O2 quantification in U87 glioblastoma cell suspensions, yielding recovery rates between 97.2% and 104.7% and relative standard deviations ranging from 1.0% to 4.6%. These results demonstrate the potential of the developed sensor for the reliable quantification of H2O2 in environments containing cancer cells. This approach thus represents a promising analytical tool for studying processes associated with oxidative stress in the context of cancer biology.

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[VI-2] ANTINUCLEAR ANTIBODY PROFILES IN PATIENTS TREATED WITH HYMENOPTERA VENOM IMMUNOTHERAPY: A CROSS-SECTIONAL STUDY USING AN EXTENDED LINE IMMUNOASSAY

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Background: The clinical significance of antinuclear antibody (ANA) reactivity in patients treated with venom immunotherapy (VIT) requires careful interpretation. We aimed to characterize antigen-specific immunoblot profiles and describe clinical characteristics.

Methods: This cross-sectional analysis included 32 patients treated with Hymenoptera VIT. Samples were analysed using EUROLINE ANA Profile 23 (IgG; EUROIMMUN). Intensity data were available for 22 specificities. Signals were classified as negative (0–5), borderline (6–10), positive + (11–25), positive ++ (26–50), or strongly positive +++ (>50). Borderline reactivity was reported separately. No systematic paired pre-VIT measurements or control groups were available.

Results: The group comprised 16 women and 16 men (50.0% each); age was 52.2 ± 12.9 years (mean ± SD; range 22–73). Positive antigen-specific reactivity was observed in 21 patients (65.6%), borderline reactivity only in ten (31.3%), and an entirely negative profile in one (3.1%). Of the 21 positive profiles, 17 (81.0%) showed a single positive specificity and four (19.0%) showed multiple positive specificities. Of 26 positive antigen-specific reactions, 20 (76.9%) were graded +, three ++ and three +++. The most frequent reactivities were anti-Mi-2β (7/32; 21.9%), anti-RP11 (5/32; 15.6%) and anti-DFS70 (4/32; 12.5%). Anti-DFS70 was the sole positive specificity among the 22 analysed in four patients; three also showed borderline reactivity to other antigens. One anti-DFS70-positive patient had reported atopy and previous grass pollen immunotherapy. Among the 21 patients with positive profiles, reported VIT involved wasp venom in 17, honeybee venom in two and dual-venom treatment in one; venom details remained unconfirmed in one patient. Three had completed VIT. Biologic therapy for asthma and previous methotrexate use were each reported in one patient. Two had reported antibody positivity around VIT initiation in 2022, without verified pre-treatment timing.

Conclusions: Positive profiles were predominantly monospecific, with most reactions graded +. Interpretation requires HEp-2 indirect immunofluorescence and clinical correlation. Anti-DFS70 positivity alone neither establishes nor excludes systemic autoimmune rheumatic disease. These cross-sectional findings do not establish VIT-induced seroconversion or causality. A prospective controlled study with pre-treatment measurements is planned.

[VI-3] Optimization of Aminophenylboronic Acid Anchoring Conditions on Amino-Functionalized UiO-66 MOF for Oriented Antibody Immobilization

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Oriented immobilization of antibodies (Abs) is crucial for achieving highly sensitive immunosensing platform because traditional methods like EDC/NHS crosslinking target primary amines and are distributed all over the Ab-including the antigen-binding Fab region. Those random coupling often blocks or sterically hinders the Ab from capturing its target. Boronic acid chemistry enables the highly efficient, site-specific and oriented immobilization of Abs by forming stable boronate ester with carbohydrate moieties present on the Fc regions of Abs. Unlike random immobilization-which often blocks the antigen-binding sites, boronic acids selectively bind to the carbohydrate chains natively present on the Ab's Fc region, leaving the Fab fully exposed and active to bind with target antigen. This oriented immobilization strategy enhances the sensitive and stable binding of target antigen thereby lowers the limit of detection of the biosensor. In the present study, this boronic acid chemistry was executed over amine-functionalized UiO-66 metal-organic frameworks (MOFs) for the sensitive detection of thyroglobulin antigen (Tg) a highly specific tumour marker for thyroid cancer. Zirconium (Zr) based UiO-66 MOF has emerged as promising candidate for electrochemical sensing applications due to their tuneable porosity, high surface area, chemical stability, abundant Zr active sites and catalytic properties. In the present study, amine-functionalized UiO-66 (UiO-66-NH_2) were synthesized via solvothermal methods from zirconium tetrachloride and 2-aminoterephthalic acid in DMF. Then 2 mgmL-1 of the MOF was dropcasted on screen-printed-carbon electrode (SPC) and 5% glutaraldehyde was used as the crosslinking agent to couple MOF-NH2 and 4-aminophenyl boronic acid (APBA). The present study focuses on the various parameters such as the concentration and pH of APBA and incubation time to reach maximum anchoring of BA moieties on SPC/MOF to sensitively target thyroglobulin antigen (Tg), a highly specific tumour marker for thyroid cancer. Following the coupling of APBA, 50 µg of anti-Tg Ab were immobilized on the surface exposed with Fab region to sensitively target various concentrations of Tg. In each case, the electrochemical impedance spectra were recorded to measure charge transfer resistance (Rct).

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[VI-4] Elevated Capillary Uric Acid Levels in Adults Without Diagnosed Gout: Preliminary Results of a Screening and Educational Study in the Opole Region

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Introduction: Hyperuricemia is the principal risk factor for gout and may remain asymptomatic for years. Affected individuals may therefore be unaware of it. Community-based screening combined with health education provides an opportunity to identify elevated levels and inform participants about their significance. This preliminary analysis aimed to estimate the proportion of adults without diagnosed gout from the Opole region with elevated screening uric acid levels.

Methods: This observational, cross-sectional study included adult residents of Opole Voivodeship, Poland, recruited during community events between September 26 and October 8, 2026. Volunteers without previously diagnosed gout were included after providing written informed consent. Participants completed a questionnaire on diet, alcohol consumption, comorbidities, family history of gout, acute joint pain episodes and awareness of hyperuricemia and gout. Uric acid concentrations were measured in finger-prick capillary whole blood using the UASure II portable electrochemical analyzer (Apex Biotechnology Corp.). Elevated levels were defined using commonly applied thresholds: ≥7.0 mg/dL in men and ≥6.0 mg/dL in women. Participants with elevated results were advised to undergo venous blood confirmation and consult a primary care physician. Descriptive statistics were used; 95% confidence intervals were calculated using the Wilson method, and groups were compared using the Mann-Whitney U test, Spearman's correlation and Fisher's exact test.

Results: The analysis included 83 participants: 65 women (78.3%) and 18 men (21.7%). Median age was 57 years (IQR: 22-69; n=81), with two predominant groups: <30 years (n=30) and ≥50 years (n=46). Median uric acid was 4.3 mg/dL (IQR: 3.6-5.4); 10 results (12.0%) were below the analyzer's lower limit (3.0 mg/dL). Uric acid did not differ significantly between women and men (4.3 vs 4.8 mg/dL; p=0.19) and was not significantly correlated with age (ρ=0.19; p=0.087). Elevated levels were found in 9 participants (10.8%; 95% CI: 5.8-19.3%): 7 women (10.8%) and 2 men (11.1%). They were more frequent in participants aged ≥50 years than in those <30 years (15.2% vs 3.3%). Awareness of the relationship between elevated uric acid levels and gout was reported by 56.1% of respondents, while 35.8% knew that elevated levels may occur without joint pain. Among 81 respondents, alcohol and red meat were the most frequently identified dietary risk factors, followed by sugar-sweetened drinks and seafood.

Conclusions: In this volunteer sample, approximately one in ten adults without previously diagnosed gout had elevated capillary uric acid levels requiring laboratory confirmation. Continued recruitment, particularly of older adults and men, will allow assessment of associated risk factors and awareness of hyperuricemia and gout. Voluntary participation and point-of-care capillary measurement limit the generalizability of these preliminary findings.

[VI-5] LIPOSOMAL CO-DELIVERY OF AURORA A AND EGFR INHIBITORS FOR COMBINATION THERAPY OF LUNG CANCER

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Non-small cell lung cancer (NSCLC) remains a leading cause of cancer related deaths worldwide. Standard treatment involves the administration of epidermal growth factor receptor tyrosine kinase inhibitors (EGFR-TKIs). A limitation of conventional therapies is the development of drug resistance in NSCLC cells.

The aim of the study was to evaluate the cytotoxicity of selected compounds and their potential synergistic effect on the PC-9 cell line. Cytotoxicity was assessed using the colorimetric MTS assay after 72 hours of incubation. The strongest synergistic effect was observed with the combination of afatinib and alisertib; this may result from the simultaneous inhibition of the EGFR signaling pathway and the disruption of mitotic regulation through the blockade of Aurora A kinase.

To enhance the therapeutic effect, each of the selected drugs (afatinib and alisertib) was encapsulated in liposomal carriers. The liposome preparation method involved lipid film hydration, extrusion, and functionalization with hyaluronic acid. Our study also involved characterizing the physicochemical properties of the obtained liposomal carriers and assessing their stability in culture medium and during storage. Subsequently, PC-9 cells were treated with drug-loaded liposomes to evaluate their therapeutic efficacy.

Our results indicate that rationally designed combinations of inhibitors targeting key signaling pathways and agents inducing cell cycle arrest facilitated by liposomal drug delivery systems may represent a promising therapeutic strategy for treating EGFR-dependent NSCLC.

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[VI-6] MODULATION OF THE pH GRADIENT IN THE TUMOUR MICROENVIRONMENT AS A STRATEGY FOR ENHANCING THE EFFICACY OF CHEMOTHERAPY

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Cancer treatment has remained one of the major challenges in modern medicine and scientific research. Due to the high heterogeneity of cancer cells, developing a universal and effective anticancer therapy is still a difficult task. Nevertheless, tumour cells share several common characteristics, including uncontrolled proliferation, resistance to apoptosis, evasion of immune responses, the ability to form metastases, and an acidic extracellular environment. The reduced pH of the tumour microenvironment is mainly associated with lactate production resulting from glycolysis, known as the Warburg effect. Lactate is transported out of tumour cells by monocarboxylate transporters (MCTs), contributing to extracellular acidification.

Lactate accumulation in the tumour microenvironment plays a significant role in tumour progression. It promotes angiogenesis, suppresses immune cell activity, and contributes to resistance to anticancer treatments. In contrast to the acidic extracellular environment, tumour cells maintain a relatively higher intracellular pH than normal cells. This altered pH gradient can reduce the effectiveness of conventional chemotherapeutic agents. Many anticancer drugs, including doxorubicin, exhibit weakly basic properties. Under acidic extracellular conditions, these drugs become protonated, which limits their ability to cross cell membranes and enter tumour cells. Therefore, modifying the tumour pH gradient may improve the therapeutic efficacy of weakly basic anticancer drugs.

In this study, human breast cancer cells were treated with monocarboxylate transporter (MCT) inhibitor to block lactate transport and decrease intracellular pH. Simultaneously, the acidic extracellular environment was neutralised using alkaline substance capable of buffering lactic acid. The cells were subsequently exposed to doxorubicin, a weakly basic anticancer drug. Cell viability and intracellular drug accumulation were then assessed to investigate the effects of these treatments on the cellular response to doxorubicin.

[VI-7] THERAPEUTIC STRATEGIES BASED ON NANOFORMULATIONS CONTAINING GOLD(I) COMPOUNDS, TARGETING THE METABOLISM OF COLORECTAL CANCER CELLS

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Colorectal cancer (CRC) remains one of the leading causes of cancer-related mortality worldwide, with metabolic plasticity contributing to tumor progression and resistance to therapeutic stress. Cancer cells can exploit both aerobic glycolysis (the Warburg effect) and mitochondrial oxidative phosphorylation to maintain energy production under changing environmental conditions. Targeting complementary metabolic and redox vulnerabilities may therefore represent a promising strategy to enhance anticancer efficacy.

In this study, we investigated a combination of a gold(I) compound ("A") and a monocarboxylate transporter inhibitor ("B") as a potential therapeutic approach against CRC. Compound A inhibits thioredoxin reductase (TrxR), an enzyme frequently overexpressed in cancer cells. TrxR inhibition disrupts cellular redox homeostasis, promotes reactive oxygen species (ROS) accumulation, and may consequently induce oxidative stress and apoptosis. Compound B targets monocarboxylate transporters involved in lactate efflux, leading to intracellular lactate accumulation, cytoplasmic acidification, disruption of pH homeostasis, and impaired energy metabolism. Simultaneous targeting of redox balance and metabolic adaptation may enhance the cytotoxic effects of both compounds.

To overcome the pharmacokinetic limitations and systemic toxicity associated with free compounds, emulsomes were developed as nanocarriers for their coordinated delivery. Emulsomes are vesicular systems consisting of a solid lipid core surrounded by one or more phospholipid layers, providing enhanced capacity for the encapsulation of lipophilic compounds. The obtained formulations were characterized in terms of their physicochemical properties, stability, and encapsulation efficiency over a two-month period. Their biological activity was evaluated in the LS180 and SW620 colorectal cancer cell lines and compared with the effects of the corresponding free compounds.

The results indicate that combining compounds A and B at an equal molar ratio leads to a stronger cytotoxic effect than compound A alone. Importantly, incorporation of compound A into emulsomes further increased its anticancer activity compared with the free gold(I) compound, while simultaneous encapsulation of both compounds resulted in an even more pronounced effect. Empty emulsomes showed no detectable cytotoxicity under the tested conditions, supporting their potential as a safe delivery platform.

Overall, these findings support the potential of emulsome-based co-delivery of metabolic and redox-targeting agents as a promising strategy for colorectal cancer treatment. The observed differences between free compounds, single-drug formulations, and combined nanoformulations highlight the potential of this approach and provide a basis for further optimization. The full set of biological and physicochemical results is presented on the poster.

[VI-8] PEDOT: PSS-BASED EUTECTOGELS AS PRINTABLE CONDUCTIVE MATERIALS FOR ELECTROCHEMICAL IMMUNOSENSING

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Eutectogels are emerging soft materials that combine the tunable physicochemical properties of deep eutectic solvents (DES) with the structural characteristics of polymeric gels. Their integration with conductive polymers offers promising opportunities for developing printable materials for electrochemical biosensing. In this study, PEDOT: PSS eutectogels incorporating a choline chloride–lactic acid (ChCl – LAC) deep eutectic solvent was developed and systematically investigated by varying the PEDOT weight ratio (40%. 50%, 60%, 70% wt). The obtained Eutectogels were characterized in terms of morphology, rheological behaviour, electronic conductivity, and electrochemical properties to identify an optimal composition for printable biosensing platforms. Scanning electron microscopy revealed porous, interconnected structures following freeze-drying, while rheological measurements demonstrated shear-dependent flow behaviour suitable for extrusion-based printing. The PEDOT:PSS ratio significantly influenced the electrical and electrochemical properties of the eutectogels. Among the investigated formulations, the eutectogel containing 50 wt.% PEDOT:PSS exhibited the highest electronic conductivity, reaching 89.2 ± 29.7 S cm^{-1}, and the lowest impedance across the investigated frequency range. The optimized formulation was subsequently employed to develop a redox-active electrochemical immunosensor for tumour necrosis factor-α (TNF-α), an important pro-inflammatory cytokine associated with inflammatory and autoimmune disorders. The resulting sensor demonstrated efficient electron transfer and promising analytical performance, with a limit of detection of 10.2 pg mL^{-1}. These findings establish PEDOT:PSS/DES eutectogels as versatile conductive materials with tunable rheological and electrochemical properties, offering potential for printable electrode architectures and next-generation electrochemical biosensing applications.

[VI-9] TARGETING SNAI1 IN RHABDOMYOSARCOMA: ANTISENSE OLIGONUCLEOTIDES AS PROMISING THERAPEUTIC AGENTS

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SNAI1 transcription factor regulates E-cadherin repression during epithelial-mesenchymal transition (EMT) and its dysregulation drives tumor progression in rhabdomyosarcoma (RMS).

Current RMS therapies remain suboptimal, necessitating novel therapeutic strategies. Our previous studies have shown that targeting SNAI1 in rhabdomyosarcoma may be a promising approach for future therapies.

Antisense oligonucleotides (ASOs) have gained attention for their potential use as a precision therapy against tumors. Their mechanism of action offers targeted gene silencing with potential advantages over traditional small molecules and antibodies, making them an attractive option for innovative treatment strategies.

The present study aims to explore the potential of ASOs targeting SNAI1 as therapeutic agents for RMS.

We designed ASOs targeting 5' and 3' sequences of SNAI1 mRNA and evaluated their therapeutic efficacy in alveolar rhabdomyosarcoma (Rh30 cell line) and patient-derived xenograft cells. We used extreme serial dilution assay (ELDA) to estimate the frequency of RMS stem cells.

ASO-mediated SNAI1 knockdown significantly reduced cell viability and spheroid formation capacity. Using extreme limiting dilution assay (ELDA), we demonstrated that SNAI1 targeting decreased RMS stem cell frequency, establishing the critical role of SNAI1 in maintaining the cancer stem cell pool.

Our findings validate ASOs targeting SNAI1 as a promising therapeutic approach for rhabdomyosarcoma and provide mechanistic insight into SNAI1's function in RMS stem cell maintenance.

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[VI-10]* DEVELOPMENT OF AN AFFORDABLE PROTOTYPE FOR ELECTROCHEMICAL MEASUREMENTS USING MULTICHANNEL BIOLOGICAL SENSORS FOR POINT-OF-CARE DIAGNOSTICS

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Miniaturized electrochemical measurement systems offer opportunities for developing affordable analytical devices for point-of-care (POC) applications. This study presents a prototype electrochemical measurement system based on the ESP32 microcontroller and its preliminary evaluation using acetaminophen as a model analyte. Its significance for the future development of a biosensor platform capable of multichannel measurements is also discussed.

The prototype enables programmable control voltage (VSET) waveforms, current signal recording, and wireless data transmission via Wi-Fi. Six samples with nominal paracetamol concentrations of 0, 50, 100, 150, 200, and 250 µg/mL were analyzed. Each recording consisted of 512 data points collected during a bidirectional control voltage sweep from 3.3 V to 0 V and back to 3.3 V. Data analysis in MATLAB (R2026b) included accounting for the direction of the waveform, signal smoothing, background correction, and a preliminary assessment of concentration-dependent responses. Numerical simulations were used separately to develop signal processing and visualization procedures and were not treated as experimental results.

The recorded signals demonstrated the prototype's ability to acquire real-time data under the test conditions. Differences were observed in the samples' responses: however, no clear correlation with concentration was established. Therefore, the method for the quantitative determination of acetaminophen has not yet been validated. Further optimization of the measurement circuits, electrodes, signal processing, and measurement repeatability is necessary.

The engineering and analytical experience gained forms the basis for the next project, which focuses on a multichannel platform for electrochemical biosensors for POC diagnostics. Future work will include a measurement architecture for three analytes using the EmStat Pico MUX16 platform, integrated software for measurement and control, and analytical validation. This interdisciplinary approach combines analytical chemistry, biotechnology, and automation to develop miniaturized diagnostic systems.

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Young Scientist poster session

[VI-11] POTENTIAL OF LATENCY-ASSOCIATED MYCOBACTERIUM TUBERCULOSIS PEPTIDE POOLS FOR THE IMMUNODIAGNOSIS OF LATENT TUBERCULOSIS INFECTION: A LONGITUDINAL STUDY

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Background: Interferon-gamma release assays (IGRAs) are widely used to detect Mycobacterium tuberculosis infection, but they cannot reliably distinguish latent tuberculosis infection (LTBI) from active tuberculosis. Antigens associated with different phases of M. tuberculosis infection, particularly latency-associated antigens, may provide additional information on infection status and persistence of cellular immune responses. We evaluated novel M. tuberculosis-derived peptide pools during a six-month follow-up of individuals with LTBI.

Methods: Thirteen individuals with LTBI who were positive by QuantiFERON-TB Gold Plus (QFT-Plus) at baseline were followed for six months. At baseline and follow-up, whole-blood samples were tested using the QFT-Plus and an in-house assay containing different combinations of newly selected M. tuberculosis peptides. Experimental antigens were evaluated as 5-peptide pools, including pools enriched for latency-associated antigens, reactivation, and replication. Antigen-specific interferon-gamma (IFN-γ) production was measured after stimulation, and longitudinal responses were compared with QFT-Plus results.

Results: At six-month follow-up, 11 of 13 participants (84.6%) remained positive by QFT-Plus, whereas two participants (15.4%) reverted from positive to negative. Mean TB1-Nil and TB2-Nil responses declined during follow-up. Among the experimental peptide pools, responses to RD- and RPF-derived peptides were generally low, whereas several pools containing DosR-associated latency antigens elicited stronger and more consistent IFN-γ responses. Notably, one participant who reverted to a negative QFT-Plus at follow-up remained responsive to selected DosR-containing peptide pools, suggesting persistence of latency-associated cellular immune responses despite loss of reactivity in the conventional assay.

Conclusions: DosR-associated peptide pools showed greater immunoreactivity than RD- and RPF-based pools in this preliminary longitudinal cohort and may represent promising components of an LTBI-focused immunodiagnostic test. Their ability to detect IFN-γ responses in an individual who reverted to a negative commercial IGRA further supports investigation of latency-associated antigens as complementary targets to conventional RD1-based assays for detection of TB infection.

[VI-12] ESTABLISHMENT OF A RELIABLE AND CLINICALLY RELEVANT IN VIVO BLADDER CANCER MODEL

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Bladder cancer is one of the most commonly diagnosed malignancies worldwide and remains a significant clinical challenge due to its high recurrence rate and potential for disease progression. Despite advances in diagnosis and treatment, the development of effective therapeutic strategies requires a better understanding of tumor biology and reliable preclinical research models. *In vivo* models of bladder cancer play a crucial role in investigating tumor development, assessing disease progression, and evaluating the efficacy of potential therapeutic interventions under physiological conditions. However, the establishment of such models is challenging, as different induction methods may vary in tumor formation efficiency, reproducibility, and the extent to which they reflect the characteristics of human bladder cancer. Therefore, the systematic comparison of available induction approaches is essential to identify a reliable and reproducible experimental model suitable for further preclinical investigations. This study aimed to establish an *in vivo* model of bladder cancer and compare different tumor induction methods in terms of their effectiveness and suitability for experimental research.

In this study an *in vivo* bladder cancer model was developed using 4 different agents to disrupt the protective glycosaminoglycan layer of the bladder mucosa (22% EtOH, 0,1mM AgNO3, 0,125% Trypsin, 0,01% Poly-L-lysine) beforeintravesical implementation of cancer cells into the bladder. Tumor development and progression were monitored using IVIS imaging system. At chosen time points (7th and 14th days after implementation), bladders were harvested, frozen, sectioned, and subjected to H&E and fluorescence staining for histological assessment of tumor formation and tissue characteristics.

Intravital tumor imaging revealed comparable bioluminescence signal intensities across the experimental groups on day 7, whereas control mice showed only background-level signals. By day 14, substantial differences in signal intensity and variability between individual animals were observed across the groups. The greatest tumor growth was observed in the group in which ethanol was the damaging agent, but tumor growth varied. The poly-L-lysine group demonstrated consistent tumor establishment in all animals, accompanied by lower inter-group variability compared with the other induction protocols.

Subsequent histological examination of H&E-stained sections enabled the assessment of tissue architecture and tumor morphology. Immunofluorescence staining for Ki-67 and luciferase was performed to evaluate tumor cell proliferation and confirm the presence of luciferase-expressing tumor cells.

Poly-L-lysine pretreatment resulted in successful bladder tumor formation in all animals, with consistent tumor sizes and no excessive growth. These findings support its use as a reliable agent for establishing a reproducible in vivo bladder cancer model.

[VI-13] SALIVARY TGF-B1–3 AND TGF-BRI–III CONCENTRATION PROFILE IN ADULTS WITH ACTIVE DENTAL CARIES

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Introduction: Dental caries remains one of the most prevalent chronic diseases worldwide. Transforming growth factor-beta (TGF-β) isoforms and their receptors are known to modulate oral immune and inflammatory responses. The aim of this study was to evaluate salivary concentrations of TGF-β1, TGF-β2, TGF-β3 and their receptors (TGF-βRI, TGF-βRII, TGF-βRIII) in adults with active dental caries of varying severity, compared with caries-free controls.

Material and Methods: The study included 75 patients aged 20–45 years (mean 32.6 ± 7.1) with active dental caries (DMFT ≥ 5), diagnosed according to WHO criteria. Patients were stratified by caries intensity into three subgroups: B1A (n = 25, DMFT 5.0–8.9, low), B1B (n = 25, DMFT 9.0–13.9, moderate) and B1C (n = 25, DMFT > 13.9, high). The control group (K, n = 50) comprised caries- and periodontal disease-free healthy adults aged 20–45 years (mean 33.8 ± 4.7). Unstimulated saliva samples were centrifuged (3000 rpm, 30 min, 4°C) and supernatants stored at –70°C until analysis. Salivary concentrations of TGF-β1, TGF-β2, TGF-β3, TGF-βRI, TGF-βRII and TGF-βRIII were determined by sandwich ELISA (Invitrogen and Assay Genie kits) according to the manufacturers' protocols, with absorbance read at 450 nm (Tecan M200PRO). All assays were performed in triplicate.

Results: Salivary TGF-β1 increased progressively with caries severity, from 165 ± 42 pg/ml in controls to 248 ± 53 pg/ml in B1A and 270 ± 70 pg/ml in B1C ($p < 0.05$), with B1C significantly higher than both K and B1A. A similar pattern was seen for TGF-β2, rising from 420 ± 110 pg/ml (K) to 550 ± 140 pg/ml (B1C, $p < 0.05$). In contrast, TGF-β3 declined with disease progression, from 310 ± 75 pg/ml (K) to 180 ± 50 pg/ml (B1C, $p < 0.05$). Among receptors, TGF-βRI increased markedly from 2.15 ± 0.55 ng/ml (K) to 3.70 ± 0.80 ng/ml (B1C, $p < 0.001$), while TGF-βRII showed only a non-significant upward trend ($p = 0.08$). TGF-βRIII decreased from 510 ± 160 pg/ml (K) to 290 ± 120 pg/ml (B1C, $p < 0.01$). All significant differences were confirmed by post-hoc analysis between B1C and both K and B1A.

Conclusions: Salivary TGF-β1, TGF-β2 and TGF-βRI increase, while TGF-β3 and TGF-βRIII decrease, with increasing severity of dental caries, suggesting an imbalance in the TGF-β signalling pathway associated with caries progression and its potential role as a salivary biomarker panel of disease severity.

[VI-14] mRNA Expression of TGF-β1, TGF-β2 and TGF-β3 Distinguishes WHO Grade 1 from Grade 2 Meningioma

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Introduction: Meningiomas are the most common primary intracranial tumors, and their biological behavior varies considerably between WHO CNS5 grade 1 and grade 2 lesions. Transforming growth factor-beta (TGF-β) isoforms have been implicated in tumor progression across several central nervous system neoplasms. This study aimed to compare the mRNA expression profile of TGF-β1, TGF-β2 and TGF-β3 between WHO grade 1 and grade 2 meningiomas.

Material and Methods: This prospective, cross-sectional observational study was conducted between October 2022 and December 2024 at two neurosurgical departments in Krakow, Poland. Of 273 patients undergoing elective meningioma resection, 154 met eligibility criteria (histopathologically confirmed WHO CNS5 grade 1 or 2 meningioma, age ≥18 years, written informed consent); patients with concurrent neoplasm, emergency surgery, non-diagnostic imaging, insufficient tissue quality or grade 3 disease were excluded. The final cohort comprised 124 grade 1 (80F/44M, mean age 60.1 ± 8.9 years) and 30 grade 2 meningiomas (21F/9M, mean age 59.9 ± 11.2 years), with no significant between-group difference in age ($p = 0.92$) or sex distribution ($p = 0.72$). Total RNA was extracted from snap-frozen tissue (TRIzol, RNeasy Mini Kit with on-column DNase I digestion) and quality-checked (A260/A280 ≥ 1.8, RIN ≥ 7.0). TGF-β1-3 mRNA expression was quantified by RT-qPCR (SensiFast SYBR No-ROX One-Step Kit) using GAPDH and ACTB as reference genes, with relative expression calculated by the 2-ΔΔCt method (fold-change relative to grade 1).

Results: Quantitative RT-qPCR analysis revealed distinct expression patterns among the three TGF-β isoforms when grade 2 meningiomas were compared to grade 1 tumors. TGF-β2 and TGF-β3 were both markedly upregulated in grade 2 specimens, with fold-change values of 5.681 and 5.231, respectively, indicating an approximately 5-fold increase in mRNA abundance relative to the grade 1 reference group. TGF-β1 expression, in contrast, was dramatically reduced, with a fold-change of 0.039, reflecting near-complete transcriptional suppression in grade 2 tumors compared to grade 1. All three comparisons reached statistical significance ($p < 0.05$).

Conclusions: WHO grade 2 meningiomas show a distinct, isoform-specific TGF-β mRNA signature relative to grade 1 tumors, with marked upregulation of TGF-β2 and TGF-β3 and near-loss of TGF-β1 expression. These findings suggest that the TGF-β pathway is differentially engaged with increasing meningioma grade and may warrant further evaluation as a molecular grading biomarker.

[VI-15] Longitudinal Changes in Core Inflammatory Mediators (PTGS2, TNF, IL-6, CCL2) Following Microdiscectomy

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Introduction: Chronic low back pain associated with intervertebral disc degeneration involves activation of neuroinflammatory pathways, with the core mediators PTGS2 (COX-2), TNF, IL-6 and CCL2 driving peripheral sensitization and pain persistence. This study evaluated longitudinal mRNA and serum protein changes in these mediators following microdiscectomy.

Material and Methods: In this prospective longitudinal study, 86 patients undergoing microdiscectomy for lumbosacral disc degeneration (32F/54M, mean age 50.45 ± 8.87 years) and 84 healthy controls were enrolled; the groups did not differ in age ($p = 0.62$) or sex distribution ($p = 0.91$). Peripheral blood was collected preoperatively and at 3 and 6 months postoperatively. mRNA expression of PTGS2, TNF, IL6 and CCL2, part of an 18-gene panel, was quantified by RT-qPCR ($2^{-\Delta\Delta Ct}$ method, normalized to ACTB/GAPDH), and the corresponding serum protein concentrations were measured by ELISA. Disc degeneration severity was graded using the Pfirrmann classification (grades III-V).

Results: At baseline, mRNA and protein levels of all four mediators were significantly elevated in patients versus controls (all $p < 0.001$) and increased progressively with Pfirrmann grade. Serum concentrations decreased significantly at each postoperative time point (pre > 3 months > 6 months, all $p < 0.001$): PTGS2 fell from 10.84 ± 2.58 to 4.81 ± 1.31 ng/mL, TNF from 246.68 ± 71.11 to 108.55 ± 34.12 pg/mL, IL-6 from 19.62 ± 6.38 to 8.59 ± 2.86 pg/mL, and CCL2 from 561.31 ± 158.14 to 245.06 ± 70.88 pg/mL, yet all remained significantly above control values (2.93 ± 0.54 ng/mL, 41.87 ± 10.95 pg/mL, 2.93 ± 1.25 pg/mL and 140.78 ± 37.12 pg/mL, respectively; $p < 0.001$ at every time point). mRNA fold-changes followed a parallel trajectory across Pfirrmann grades III-V, with the highest baseline expression and the largest postoperative decline observed in grade V patients. Among the full 18-target panel, these four core inflammatory mediators showed the strongest mRNA-protein correlations (Pearson $r = 0.87$-0.91, all $p < 0.001$).

Conclusions: PTGS2, TNF, IL-6 and CCL2 are markedly and concordantly upregulated at both the mRNA and protein level in discogenic pain, decline progressively after microdiscectomy, but remain elevated relative to controls at 6 months, indicating persistent subclinical inflammation and supporting their potential as biomarkers for monitoring postoperative recovery.

[VI-16] PLASMA MMP1, MMP3, MMP9, MMP13 AND TIMP1–3 CONCENTRATIONS ACROSS ORTHOBIOLOGIC THERAPIES FOR SACROILIAC JOINT OSTEOARTHRITIS

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Introduction: Matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) regulate extracellular-matrix turnover in osteoarthritis, but the sacroiliac joint (SIJ), an underrecognised source of chronic low back pain, has rarely been studied with this panel. This study evaluated plasma MMP1, MMP3, MMP9, MMP13 and TIMP1-3 concentrations in SIJ osteoarthritis and their response to three orthobiologic therapies.

Material and Methods: In this prospective, single-centre, non-randomised cohort study, 90 patients with SIJ osteoarthritis received autologous conditioned serum (Orthokine, n=30), platelet-rich plasma (PRP, n=30) or hyaluronic acid (HA, n=30), and were compared with 30 disease-free reference volunteers. Plasma MMP1, MMP3, MMP9, MMP13, TIMP1, TIMP2 and TIMP3 were quantified by ELISA at baseline and on days 7, 28, 90 and 180. Baseline between-group differences were assessed by ANCOVA (adjusted for age, BMI, CRP and sex); within-group changes and time × treatment interactions were tested with mixed-effects models on log2-transformed concentrations and the Friedman test.

Results: At baseline, plasma MMP1, MMP3, MMP9, MMP13 and TIMP1 were all significantly higher, and TIMP2 and TIMP3 significantly lower, in the osteoarthritis groups than in the reference group (all adjusted $p \leq 0.008$; e.g. MMP1 42.5–45.3 vs 14.0 ± 2.0 ng/mL; MMP13 3486.6–3622.9 vs 779.8 ± 102.2 pg/mL; TIMP2 2427–2475 vs 3051.4 ± 369.9 pg/mL). Over follow-up, MMP1, MMP3, MMP9 and MMP13 decreased significantly in every treatment group (Friedman $p < 0.001$ throughout), e.g. MMP1 falling to 30.7–37.0 ng/mL and MMP13 to 2541.7–3058.8 pg/mL by day 180, with a significant time × treatment interaction for all four analytes (all $p < 0.001$). Plasma TIMP1 also showed a significant, though smaller, time × treatment interaction ($p = 0.045$), whereas TIMP2 and TIMP3 did not change significantly over time ($p = 0.403$ and $p = 0.113$). A composite MMP:TIMP balance index likewise showed a significant time × treatment interaction ($p < 0.001$).

Conclusions: SIJ osteoarthritis is associated with a distinct plasma MMP:TIMP imbalance relative to disease-free controls. All three orthobiologic therapies reduced plasma MMP1, MMP3, MMP9 and MMP13, but the magnitude and the resulting MMP:TIMP balance differed significantly by treatment, while TIMP2 and TIMP3 remained largely unresponsive, supporting further evaluation of this panel as a treatment-response biomarker in SIJ osteoarthritis.

[VI-17] PLASMA MMP1, MMP3, MMP9, MMP13 AND TIMP1–3 CONCENTRATIONS ACROSS ORTHOBIOLOGIC THERAPIES FOR KNEE OSTEOARTHRITIS

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Introduction: Matrix metalloproteinases (MMPs) and their tissue inhibitors (TIMPs) govern cartilage matrix turnover in knee osteoarthritis (OA). This study evaluated plasma MMP1, MMP3, MMP9, MMP13 and TIMP1-3 concentrations in knee OA relative to a disease-free reference group and their response to three intra-articular orthobiologic therapies.

Material and Methods: In this prospective, single-centre, non-randomised cohort study, 90 patients with knee osteoarthritis received autologous conditioned serum (Orthokine, n=30), platelet-rich plasma (PRP, n=30) or hyaluronic acid (HA, n=30), and were compared with 30 disease-free reference volunteers. Plasma MMP1, MMP3, MMP9, MMP13, TIMP1, TIMP2 and TIMP3 were quantified by ELISA at baseline and on days 7, 28, 90 and 180. Baseline between-group differences were assessed by ANCOVA (adjusted for age, BMI, CRP and sex); within-group changes and time × treatment interactions were tested with mixed-effects models on log2-transformed concentrations and the Friedman test.

Results: At baseline, plasma MMP1, MMP3, MMP9, MMP13 and TIMP1 were all significantly higher in the OA groups than in the reference group (adjusted differences: MMP1 +30.7 ng/mL, MMP3 +3.4 ng/mL, MMP9 +6.6 ng/mL, MMP13 +2728.8 pg/mL, TIMP1 +1697.2 pg/mL; all $p < 0.001$), whereas TIMP2 and TIMP3 were significantly lower (−569.0 and −1035.2 pg/mL respectively; both $p < 0.001$). Over follow-up, plasma MMP1, MMP3, MMP9 and MMP13 decreased significantly in every treatment group (Friedman $p < 0.001$ throughout), with a significant time × treatment interaction for all four (MMP1 $p = 0.002$; MMP3 $p < 0.001$; MMP9 $p = 0.002$; MMP13 $p < 0.001$). Plasma TIMP1 also showed a significant time × treatment interaction ($p = 0.004$), while TIMP2 and TIMP3 did not ($p = 0.397$ and $p = 0.223$). For MMP13, model-adjusted estimates were significantly lower in the Orthokine and PRP groups than in the HA group at both day 90 and day 180 (Holm-adjusted $p < 0.001$), and for TIMP1, the HA group had significantly higher estimates than Orthokine and PRP at day 180 (both $p \leq 0.005$). A composite MMP:TIMP balance index also showed a significant time × treatment interaction ($p < 0.001$).

Conclusions: Knee osteoarthritis is associated with a distinct plasma MMP:TIMP imbalance relative to disease-free controls. All three orthobiologic therapies reduced plasma MMP1, MMP3, MMP9 and MMP13, but the magnitude, particularly for MMP13 and TIMP1, differed significantly by treatment, while TIMP2 and TIMP3 remained largely unresponsive, supporting further evaluation of this panel as a treatment-response biomarker in knee osteoarthritis.

[VI-18] MicroRNA-Mediated Regulation of IL-2, MTOR and COL1A1 by Tacrolimus in LPS-Stimulated Retinal Pigment Epithelial Cells

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Introduction: The PI3K/AKT/mTOR signaling pathway drives inflammatory and fibrotic responses in human retinal pigment epithelial (H-RPE) cells implicated in proliferative vitreoretinopathy (PVR). Tacrolimus, a calcineurin inhibitor with antifibrotic properties, may act partly through microRNA (miRNA)-mediated post-transcriptional regulation of this pathway. This study characterized miRNA changes accompanying tacrolimus modulation of PI3K/AKT/mTOR-related transcripts in inflamed H-RPE cells.

Material and Methods: Primary H-RPE cells were exposed to lipopolysaccharide (LPS, 1 μg/mL, 6h) to model inflammation, to tacrolimus alone (10 ng/mL, 6/12/24h), or to LPS followed by tacrolimus (6/12/24h), and compared with untreated controls. mRNA and miRNA expression were profiled using HG-U133_A2 and GeneChip miRNA 2.0 microarrays (Affymetrix), respectively, and an in silico mRNA-miRNA target prediction analysis was performed to identify regulatory miRNAs for differentially expressed PI3K/AKT/mTOR-pathway transcripts (all changes $p < 0.05$).

Results: LPS-induced upregulation of IL-2 (log2FC up to +3.89 ± 0.43 by 24h of combined treatment) was accompanied by significant downregulation of its predicted regulators hsa-miR-462 (−2.19 ± 0.51), hsa-miR-27a-5p (−3.29 ± 0.42) and hsa-miR-3163 (−2.34 ± 0.43) under LPS alone (target scores ≥ 90); tacrolimus co-treatment reversed this suppression, restoring all three miRNAs to positive log2FC values (up to +4.01 ± 0.45 for miR-27a-5p at 24h). MTOR induction under LPS+tacrolimus (log2FC up to +3.76 ± 0.91) mirrored a similar pattern for hsa-miR-3182 and hsa-miR-1271-5p, both initially suppressed by LPS (log2FC −2.87 to −3.09) and progressively restored by tacrolimus (up to +4.56 ± 0.65 and +3.76 ± 0.91, respectively). COL1A1 upregulation after co-treatment coincided with recovery of the anti-fibrotic hsa-miR-29a-3p, which fell sharply under LPS (−3.98 ± 0.87) but rose to +2.91 ± 0.34 by 24h of tacrolimus co-treatment.

Conclusions: Tacrolimus restores the expression of inflammation- and fibrosis-associated miRNAs (miR-462, miR-27a-5p, miR-3163, miR-3182, miR-1271-5p and miR-29a-3p) that are suppressed under LPS-induced inflammatory stress in H-RPE cells, in inverse coordination with their target transcripts IL-2, MTOR and COL1A1, supporting a miRNA-mediated mechanism for its antifibrotic effect in proliferative vitreoretinopathy.

[VI-19] mRNA Expression of Selenoprotein- and Redox-Related Genes in Endometrial and Ovarian Cancer

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Introduction: Endometrial cancer (EC) and ovarian cancer (OC) share inflammatory and oxidative mechanisms, but the role of selenoprotein-related pathways remains poorly defined. This study evaluated mRNA expression of selenium-related and inflammation-associated genes in EC and OC by RT-qPCR.

Material and Methods: Fifty women with endometrioid EC (grades G1–G3) and 20 controls, together with 41 women with epithelial OC divided into group A (n=28, cytoreductive surgery plus adjuvant chemotherapy) and group B (n=13, surgery alone), and 25 controls, were enrolled. Relative mRNA expression of 14 selenium-related and inflammation-associated genes (TXNRD1, GPX1, GPX3, GPX4, SELENOP, SELENOS, SEPHS2, MSRB1, SELENOK, DIO1–3, NFKB1, JUN, FOS) was quantified by RT-qPCR using the $2^{-\Delta\Delta Ct}$ method.

Results: In EC, most genes showed grade-dependent upregulation, with the largest increases in G3 tumours for TXNRD1 (fold-change 2.394 ± 0.386 vs control), MSRB1 (2.641 ± 0.421), FOS (2.600 ± 0.296) and SELENOS (2.278 ± 0.307; all ANOVA $p < 0.0001$). SELENOP showed a distinct non-linear pattern, decreased in G1 (0.772 ± 0.278) before rising sharply to 2.476 ± 0.308 in G3 (G1 vs G3 $p < 0.0001$). In OC, most transcripts were significantly higher in group A than in group B, e.g. TXNRD1 (3.462 ± 0.379 vs 2.228 ± 0.368) and SELENOK (3.168 ± 0.293 vs 1.712 ± 0.232; both $p < 0.001$), whereas SELENOP again showed an inverse pattern, higher in group B (2.586 ± 0.290) than group A (2.208 ± 0.425, $p = 0.006$). SEPHS2, DIO1 and NFKB1 did not differ significantly between the OC subgroups (all $p > 0.05$).

Conclusions: Selenoprotein- and redox-related genes are upregulated at the mRNA level in both endometrial and ovarian cancer, with expression tracking tumour grade in EC and treatment burden in OC, while SELENOP consistently shows an atypical, inverse regulatory pattern relative to the other selenoproteins, warranting further investigation as a distinct molecular marker.

[VI-20] HAEMATOLOGIC TOXICITY PATTERNS DURING DEFINITIVE CHEMORADIOTHERAPY FOR CERVICAL CANCER: LEUKOPENIA, MONOCYTOSIS AND ANAEMIA

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Introduction: Definitive chemoradiotherapy (dCRT) for cervical cancer (CC) causes substantial haematologic toxicity, but the dynamics of the complete blood count and leukocyte differential beyond composite indices such as NLR, PLR, LMR and SII remain incompletely described. This study characterized treatment-related changes in blood cell counts and the leukocyte differential during dCRT.

Material and Methods: In a prospective cohort of 106 women with FIGO stage IB–IVA CC undergoing dCRT, peripheral blood counts and leukocyte differential were obtained one day before treatment and within three days after completion of teletherapy. Paired pre–post changes in white blood cell (WBC), red blood cell (RBC), haemoglobin, haematocrit and platelet counts, and in relative neutrophil, lymphocyte, monocyte, eosinophil and basophil fractions, were assessed using the Wilcoxon signed-rank test. The eosinophil-to-neutrophil ratio (ENR) and eosinophil-to-lymphocyte ratio (ELR) were additionally derived.

Results: WBC count fell sharply after teletherapy (median 8.32 to 4.03 × 10^9/L, n=72, $p < 0.0001$), accompanied by significant reductions in platelet count (310 to 238 × 10^9/L, $p < 0.0001$), RBC count (4.23 to 3.58 × 10^{12}/L, $p < 0.0001$), haemoglobin (11.45 to 10.70 g/dL, $p = 0.0004$) and haematocrit (36.35% to 32.95%, $p = 0.0001$). Within the leukocyte differential (n=44), the relative monocyte fraction rose markedly, from 7.85% to 11.95% ($p < 0.0001$), while eosinophils showed a borderline decrease (2.45% to 1.95%, $p = 0.053$) and basophils did not change significantly ($p = 0.37$). The eosinophil-to-neutrophil ratio decreased significantly after teletherapy (0.037 to 0.031, $p = 0.035$), whereas the eosinophil-to-lymphocyte ratio did not change significantly ($p = 0.28$).

Conclusions: dCRT for cervical cancer induces a broad haematologic response comprising leukopenia, thrombocytopenia and anaemia, together with a relative shift within the leukocyte differential toward monocytosis and a reduced eosinophil-to-neutrophil ratio. Distinct from the previously reported tumour-volume-driven changes in composite inflammatory indices in this cohort, these findings highlight relative monocytosis and the eosinophil-to-neutrophil ratio as additional candidate markers of treatment-related haematologic stress warranting further evaluation.

[VI-21] Serum Trace Element and Electrolyte Dynamics During Cyclosporine A Therapy in Psoriasis Vulgaris: Association with Clinical Response

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Introduction: Psoriasis vulgaris is associated with disturbances in systemic mineral and electrolyte homeostasis, but the dynamics of these parameters during cyclosporine A (CsA) therapy and their relationship with clinical response remain incompletely characterized. This study evaluated serum trace element and electrolyte dynamics during 12-week CsA therapy in psoriasis vulgaris and their correlation with disease severity.

Material and Methods: In a prospective cohort of 37 adults with moderate-to-severe psoriasis vulgaris receiving 12-week CsA therapy, serum copper (Cu), zinc (Zn), magnesium (Mg), calcium (Ca), iron (Fe), sodium (Na) and potassium (K) were quantified by atomic absorption spectrometry at baseline, day 42 and day 84. Sex-stratified differences were assessed, and Pearson correlations between element concentrations and Psoriasis Area and Severity Index (PASI) were calculated at each timepoint.

Results: Serum Cu decreased progressively (1750±210 to 1230±180 to 850±170 μg/L; $p<0.0001$), while Zn (610±85 to 690±80 to 960±90 μg/L), Mg (16200±1150 to 18550±1250 to 20800±1050 μg/L), Ca (81200±4100 to 89000±3600 to 97500±3500 μg/L) and Fe (48±9 to 68±11 to 85±12 μg/L) increased significantly toward reference ranges (all $p<0.0001$). Na and K concentrations did not change significantly over treatment ($p=0.321$ and $p=0.235$, respectively). Sex-stratified analysis showed a more pronounced decline in Cu and Fe among women, whereas Zn, Mg and Ca increased slightly more in men. Correlations between element concentrations and PASI were weak and non-significant at baseline, but by day 84 serum Mg correlated significantly with PASI ($r=0.733$, $p=0.016$), with a similar non-significant trend for Ca ($r=0.612$, $p=0.060$).

Conclusions: Twelve-week CsA therapy in psoriasis vulgaris is accompanied by progressive normalization of serum Cu, Zn, Mg, Ca and Fe, with electrolytes Na and K remaining stable, and mild sex-related differences in trajectory. The emergence of a significant Mg-PASI correlation only at treatment end suggests serum Mg as a candidate biomarker for monitoring clinical response to CsA, warranting validation in larger cohorts.

[VI-22]* 17β-Estradiol Regulates the Expression Levels of BK Subunits in Glioblastoma Cells in a Concentration-Dependent Manner

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Introduction: Potassium channels (BK) regulate physiological processes in both healthy and diseased cells. Due to their high expression levels, BK channels are particularly important for the functioning of nerve cells. In glioblastoma cells, the overexpression of BK channels is associated with the rate of tumor progression and sensitivity to therapy. Differences in incidence glioblastoma between the sexes suggest that sex hormones, including 17β-estradiol (E2), may influence glioma cell behavior.

BK channels consist of α subunits and regulatory β and γ subunits. Studies conducted primarily on mouse cells have shown that E2 can affect the expression levels of BK channel subunits or influence the function of BK channels by directly interacting with the proteins that make up the channel. In this study, we investigated the effect of physiological (0.0018 μg/ml) and pharmacological (1.8 μg/ml) concentrations of estradiol on the expression of BK channel subunits in human U-87 MG cells.

Methods: The study was conducted using U-87 MG cells incubated for 6 h with 17β-estradiol. After treatment, total RNA was isolated, and the expression of gBK α and β subunits was determined by RT-qPCR using GAPDH as a reference gene. The protein expression levels of the examined BK channel subunits were assessed by Western blotting, using GAPDH as a reference protein for normalization of the results.

Results: The mRNA expression analysis revealed increased transcript levels of the β1 and β2 BK channel subunits following treatment with E2. Western blot analysis showed E2-concentration-dependent changes in the expression levels of both the alpha subunit and the regulatory subunits, including the β3 subunit, for which no association between its expression level and E2 levels has been demonstrated to date.

Conclusion: The results indicate that 17β-estradiol regulates BK channel subunit expression in U-87 MG cells at both the mRNA and protein levels, suggesting that E2 inhibitors and synthetic derivatives have therapeutic potential.

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[VI-23]* THE EFFECT OF PROGESTERONE ON THE EXPRESSION LEVELS OF BK CHANNEL SUBUNITS IN U87-MG CELLS

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Introduction: Glioblastoma is an aggressive type of brain cancer that is diagnosed more frequently in men than in women. The sex-dependent incidence of glioblastoma suggests a link between levels of sex hormones, including progesterone (P), and development of glioblastoma. BK ion channels also play a significant role in the development and progression of glioblastoma. BK channels are composed of α subunits and regulatory β and γ subunits. These subunits influence the conformational properties of BK channels, thereby enhancing their sensitivity to Ca^{2+} and modulating their activity. The literature indicates that progesterone can bind to β subunits, modulating the activity of BK channels. Although progesterone, in complex with its receptor, can act as a transcription factor, little is known about the regulation of BK channel subunit expression by progesterone. Therefore, the aim of this study was to investigate the effect of P on the expression levels of BK channel subunits.

Methods: The study was conducted using U-87 MG cells incubated for 6 h with P at concentrations of 0,25 or 25 μg/ml. After treatment, total RNA was isolated, and the expression of BK α, β and γ subunits were determined by RT-qPCR using GAPDH as a reference gene. The protein expression levels of the examined BK channel subunits were assessed by Western blotting, using GAPDH as a reference protein for normalization of the results.

Result: The mRNA expression analysis revealed increased transcript levels of the β1 and β2 BK channel subunits following treatment with P. Western blot analysis showed P-concentration-dependent changes in the expression levels of the regulatory subunits, with no significant effect on the expression level of α subunit.

Conclusion: The results indicate that progesterone regulates the expression of BK channel subunits in U-87 MG cells at both the mRNA and protein levels, suggesting that synthetic progesterone derivatives may have therapeutic potential.

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[VI-24]* NEXT-GENERATION PHOTOSENSITIZERS IN THREE-DIMENSIONAL BRAIN TUMOR MODELS- IN VITRO STUDIES OF INTRACELLULAR TRANSPORT AND ACCUMULATION

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Glioblastoma and neuroblastoma represent brain tumors characterized by an exceptionally difficult localization and resistance to conventional treatment methods, driving the search for innovative therapeutic strategies such as photodynamic therapy (PDT). Traditional, flat monolayer cell cultures (2D) fail to accurately reflect the complexity of the tumor microenvironment, including oxygen gradients, nutrient availability, and transport barriers. The primary objective of this study is to expand current oncological knowledge by developing and optimizing advanced 3D spheroid models of glioblastoma and neuroblastoma using U87, SH-SY5Y, and normal HMC3 cell lines to evaluate the effectiveness of next-generation photosensitizers.

The project focuses on a detailed analysis of the transport, penetration, and accumulation processes of active compounds deep within three-dimensional structures. A key aspect of the research involves elucidating the mechanisms of cellular response to the phototherapeutic agent, with particular emphasis on signal transduction pathways associated with oxidative stress and the processes leading to programmed cell death. By systematically comparing results obtained from classical 2D models with those from 3D spatial models, the biological variability of tumors and the drug release efficiency from the engineered spatial systems are evaluated. The expected results will not only assess the true potential of novel photosensitizers in combating brain tumors, but will also contribute to the development of modern approaches in computational oncology and personalized medicine.

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Young Scientist poster session

[VI-25]* SHARED BIOMARKERS AND BIOLOGICAL PATHWAYS REGULATED BY MIR-9-3P MIR-9-5P, AND MIR-129-5P AFTER REPERFUSION IN ISCHIEMIC STROKE: AN IN SILICO ANALYSIS

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BACKGROUND: Ischemic stroke remains a major cause of mortality and long-term disability, while early diagnosis and prediction of clinical outcome remain challenging. Peripheral blood is one of the most accessible biological materials in stroke patients, and circulating microRNAs (miRNAs) are promising biomarkers due to their stability in blood. However, although miRNA levels can be measured in serum or plasma, expression of their downstream genetic targets in affected brain tissue cannot be routinely assessed from blood samples. Therefore, we focused on circulating protein products of miRNA-regulated genes, which may indirectly reflect stroke-related molecular processes and become detectable following tissue injury and blood-brain barrier disruption.

THE AIM: To identify circulating protein targets and biological pathways regulated by miR-9-3p, miR-9-5p, and miR-129-5p after reperfusion in ischemic stroke and to prioritize candidates with potential biomarker utility.

MATERIALS AND METHODS: The miRDB database identified 3,932 predicted target genes of the three miRNAs. A Google Scholar search combining individual targets with "blood," "plasma," and "serum" was performed to identify corresponding proteins investigated in circulating samples. Non-human studies, proteins unrelated to neurological or cardiovascular pathology, and studies limited to peripheral blood mononuclear cells were excluded. Studies using quantitative methods, including ELISA, AlphaLISA, Western blotting, and mass spectrometry, were prioritized. This screening yielded 59 proteins measured directly in serum or plasma. Protein-protein interaction and functional enrichment analyses were performed using STRING, leading to prioritization of 11 proteins based on functional connectivity and relevance to ischemic stroke.

RESULTS: The final set comprised NRG1, SEMA4D, SESN3, NTN1, REST, RYR2, MBP, HOMER1, EFEMP1, DPYSL2, and NCAM1. STRING analysis showed that these proteins are involved predominantly in processes related to nervous system development, axon guidance, neurogenesis, synaptic organization, and neuronal signaling. Network analysis also indicated that several candidates may be functionally connected indirectly through first-shell interactors rather than by direct protein-protein interactions.

CONCLUSIONS: This in silico analysis identified 11 circulating protein candidates potentially linking altered miRNA expression with molecular processes relevant to ischemic stroke. Circulating protein products may provide a clinically accessible approach to indirectly assess molecular alterations that cannot be readily evaluated in affected brain tissue. The identified candidates are being validated in appropriately selected study groups to assess their diagnostic and prognostic relevance, with further prospective validation required before clinical implementation.

FUNDING: This study was funded by MNiSW, grant no. SKN/SP/67537/2026.***Young Scientist poster session***

[VI-26]* COMPARATIVE ANALYSIS OF SUBCORTICAL STRUCTURE VOLUMES IN PARKINSON'S DISEASE PATIENTS AND A NORMATIVE GROUP USING FREESURFER AND FSL

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Neurodegenerative diseases, such as Parkinson's disease (PD), may induce changes in subcortical brain structures that can be investigated using magnetic resonance imaging (MRI). Automated segmentation of brain structures and the extraction of radiomic features enable an objective characterization of these structures, potentially supporting disease assessment and monitoring. However, radiomic features may vary depending on the segmentation method used, and PD may further influence the degree of agreement between methods.

The aim of this study was to compare volumes extracted from subcortical brain structures segmented using FreeSurfer and FMRIB Software Library (FSL) and to determine whether agreement between methods differed between PD and healthy controls (HC).

The analysis was conducted on T1-weighted brain MRI scans from 94 healthy participants and 70 patients with Parkinson's disease. Twelve regions of interest (six bilateral subcortical structures: thalamus, caudate, putamen, pallidum, hippocampus and amygdala) were segmented using FreeSurfer and FSL, and the volume of each structure was extracted using PyRadiomics. Agreement between methods was assessed separately in the PD and HC groups using the intraclass correlation coefficient (ICC). Differences in ICC between groups were tested with a permutation test, and absolute percentage differences between methods were compared with the Mann–Whitney U test. PD–HC volume differences were analyzed separately for each method. P-values were FDR-corrected (Benjamini–Hochberg).

Agreement between FreeSurfer and FSL volumes was poor in all structures in both groups. FreeSurfer volumes were generally larger than FSL volumes. ICC was lower in PD than in HC in all structures, although the difference was significant after correction in only a few structures. The choice of method also influenced PD–HC comparisons: volume differences between PD and HC were detected in different structures depending on the segmentation tool.

Agreement between FreeSurfer- and FSL-derived volumes was generally limited and lower in patients with PD than in HC. Moreover, the choice of segmentation method changed whether PD-related volume differences were detected, so the volumes from the two tools are not interchangeable and the method should be reported and harmonized in PD imaging studies.

Young Scientist poster session

[VI-27]* FERROPTOSIS-RELATED PROTEINS IN BREAST CANCER TISSUE AND THEIR SYSTEMIC RESPONSE TO FIBROADENOMA CRYOABLATION

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Introduction: Ferroptosis is an iron-dependent form of regulated cell death driven by lipid peroxidation and counteracted by antioxidant and iron-buffering proteins, many of which are controlled by the transcription factor NRF2. It remains unclear whether these proteins differ across breast cancer molecular subtypes and whether they can be detected systemically after localized tissue injury.

Aim: To quantify six ferroptosis-related proteins (SLC7A11, GPX4, FTH1, NQO1, NFE2L2/NRF2 and SQSTM1/p62) in breast cancer tissue and to evaluate the time course of their serum concentrations after fibroadenoma cryoablation.

Material and methods: Tumor and matched non-neoplastic tissue were obtained from 405 patients with T1N0M0 breast cancer representing five molecular subtypes: luminal A, luminal B HER2−, luminal B HER2+, non-luminal HER2+ and triple-negative breast cancer (TNBC). The second cohort comprised 34 women with fibroadenoma treated with cryoablation (IceCure ProSense™). Their serum was collected before the procedure (T0) and 30–60 min, 8–12 h, 48–72 h, 7 days, 1 month and 3 months afterwards. Protein concentrations were determined by ELISA, and interactions among the proteins were analyzed using the STRING database.

Results: All six proteins were significantly elevated in tumor tissue compared with control tissue ($p<0.05$), with levels increasing progressively from luminal A to TNBC. SLC7A11 rose from 0.84 ng/mL in control tissue to 4.48 ng/mL in TNBC, GPX4 from 28.6 to 118.4 ng/mL, FTH1 from 214.9 to 742.1 ng/mL and NQO1 from 182.9 to 731.9 pg/mL, with similar patterns for NRF2 and SQSTM1. The proteins formed a densely connected interaction network (15 edges; PPI enrichment $p=1.11\times10^{-16}$) associated with glutathione metabolism and the response to oxidative stress. After cryoablation, serum concentrations of all proteins increased significantly within 30–60 min, peaked at 8–12 h and remained elevated at 48–72 h. Elevated levels persisted at 7 days for all proteins except SLC7A11, and returned to baseline at 1 and 3 months.

Conclusions: Breast cancer shows coordinated, subtype-graded overexpression of NRF2-related proteins that protect against ferroptosis, with the highest levels in aggressive subtypes. Localized tissue injury induces a transient and measurable increase of these proteins in serum, suggesting their potential as minimally invasive indicators of tissue stress, although they should not be regarded as specific markers of ferroptosis.

Young Scientist poster session

[VI-28]* DOWNREGULATION OF FERROPTOSIS-RELATED MICRORNAS ACROSS MOLECULAR SUBTYPES OF BREAST CANCER

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Introduction: Ferroptosis is an iron-dependent form of regulated cell death driven by lipid peroxidation, and resistance to it may support breast cancer progression and therapy failure. MicroRNAs (miRNAs) regulate iron metabolism and redox homeostasis at the post-transcriptional level, but their contribution to ferroptosis-related signaling across breast cancer molecular subtypes remains poorly characterized.

Aim: To assess the expression of miRNAs predicted to regulate key ferroptosis-related genes in five molecular subtypes of breast cancer.

Material and methods: The study included 405 patients with T1N0M0 breast cancer: luminal A (n=130), luminal B HER2− (n=100), luminal B HER2+ (n=96), non-luminal HER2+ (n=36) and triple-negative breast cancer (TNBC; n=43). Tumor tissue and matched non-neoplastic tissue from the surgical margin were collected. miRNA expression profiling was performed using GeneChip miRNA 2.0 arrays (Affymetrix). miRNA–target interactions were predicted with TargetScan and miRDB, and target scores above 80 were considered reliable. Statistical significance was set at $p<0.05$.

Results: Four miRNAs predicted to target ferroptosis suppressors were significantly downregulated in all breast cancer subtypes compared with control tissue: hsa-miR-1297 and hsa-miR-26a-5p, both targeting SLC7A11, hsa-miR-18b-3p targeting NQO1, and hsa-miR-28-5p targeting NFE2L2 (NRF2). The direction of change was consistent across subtypes, whereas its magnitude was subtype-dependent and generally greatest in aggressive tumors. Expression of miR-26a-5p decreased progressively from luminal A (log2FC −2.34) to TNBC (log2FC −3.67). miR-18b-3p showed the strongest downregulation in non-luminal HER2+ tumors (log2FC −3.87) compared with luminal A tumors (log2FC −2.11), and miR-28-5p reached its lowest levels in non-luminal HER2+ (log2FC −3.87) and TNBC (log2FC −3.81) samples. Downregulation of miR-1297 was moderate and comparable across subtypes (log2FC from −1.19 to −1.89).

Conclusions: Breast cancer is characterized by consistent loss of miRNAs regulating the SLC7A11/glutathione axis and the NRF2-dependent antioxidant pathway. Reduced post-transcriptional repression may favor a ferroptosis-resistant, cytoprotective phenotype, particularly in HER2-positive and triple-negative tumors. This mechanism most likely acts as a secondary layer reinforcing NRF2-driven signaling rather than as its primary driver, and requires functional validation. ***Young Scientist poster session***

[VI-29] GLIO-SMART: A ROBUST IMMUNE–NEURONAL TRANSCRIPTIONAL AXIS STRATIFIES DIFFUSE GLIOMAS ACROSS GRADES

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Immune checkpoint blockade has shown little benefit in diffuse glioma, partly because patients are not selected by the immune state of their tumour microenvironment (TME). It is unclear whether immune-associated transcriptional states stay stable across grades and molecular classes. This study aimed to identify such a signature and test its robustness across grades and gene panels.

RNA-seq and clinical data were analysed for 670 TCGA diffuse gliomas: 509 lower-grade gliomas (LGG) and 161 glioblastomas (GBM). Genes were selected from expression data and literature and grouped into two TME panels. Six analyses were run, combining three cohorts (integrated, LGG-only, GBM-only) with each panel. In every run, consensus clustering of variance-stabilised, cohort-corrected TME gene expression, supported by non-negative matrix factorisation, identified two reproducible TME subgroups. Differential expression was assessed on raw counts using DESeq2 (Benjamini–Hochberg FDR < 0.05, $|\log_2 FC| > 1$), and after harmonising cluster labels across runs, $\log_2$ fold-change profiles were compared by Spearman correlation.

Each run yielded 2,048–3,256 differentially expressed genes; 954 were significant in all six runs, 98.8% with consistent direction. Fold-change profiles correlated strongly between integrated and LGG runs (Spearman $\rho = 0.94$–0.98) and moderately with GBM runs ($\rho = 0.48$–0.67). Patients in the inflamed ("hot") cluster showed enrichment for myeloid and inflammatory associated genes (CD163, CD14, PTPRC, CCL2–CCR2, CHI3L1, POSTN, SAA1) and over-expressed CD274 (PD-L1), as well as PDCD1LG2 (PD-L2). Patients in the immune-silent ("cold") cluster had tumours characterised by neuronal and synaptic associated genes (CARTPT, CAMK2A, SLC12A5, NEUROD6). Subgroups followed molecular class: 92% of IDH-mutant, 1p/19q-codeleted tumours were immune-silent, whereas most IDH-wild-type and IDH-mutant non-codeleted tumours were inflamed. The axis remained detectable, though weaker, within GBM.

Diffuse gliomas lie along a robust immune–neuronal transcriptional axis that spans grade and aligns with molecular classification. At the inflamed pole, myeloid recruitment co-occurs with PD-L1/PD-L2 expression. This signature defines a distinct TME state and provides candidate markers for validation in outcome- and treatment-annotated cohorts.



AUTENTIKOS

The band **AUTENTIKOS** was formed in August 2015 in Mikołów. It consists of five musicians with many years of stage experience: Arkadiusz Kuś - founder and drummer, Marcin Tomaszewski - bassist, Rafał Cieślak and Tomasz “Tiziano” Czerw - guitarists, and Łukasz Kwapisiewicz - pianist. The vocalist and lyricist is Sebastian Giebel, who is a professor of medicine and a hematologist in his daily life.

In 2018, the first album by AUTENTIKOS, titled "Audiotherapy", was released. In April 2019, the band performed at the concert studio of Polish Radio Katowice, while simultaneously recording material for their first live album, "Maria Curie". In December 2022, under the patronage of Radio Katowice, their second studio album, "Fejm", was released. The songs, covering a variety of musical styles - from danceable funk to hip-hop, poetic ballads, punk, and progressive rock - reached the top spots on the charts of "Polish Radio Katowice" as well as Warsaw's "Radio dla Ciebie". Lyrically, they reflect contemporary reality, sometimes seen through a distorted, pastiche mirror from the singer's perspective. The common denominator is the honesty of the message, which became the reason behind the choice of the band's name. Currently, the band is recording material for their third studio album in collaboration with the choir of the Song and Dance Ensemble "Śląsk".

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Czym się zajmujemy?

Zajmujemy się badaniami biomateriałów oraz materiałów na zastosowania biomedyczne, projektowaniem stanowisk badawczych, a co więcej - popularyzacją nauki.

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JAK DZIAŁAMY?

Realizacja projektów: Stale realizujemy projekty, które pomagają nam rozwijać naszą wiedzę oraz budować dorobek naukowy. Każdy student ma możliwość realizowania projektu o tematyce, która najbardziej wpasowuje się w jego zainteresowania.

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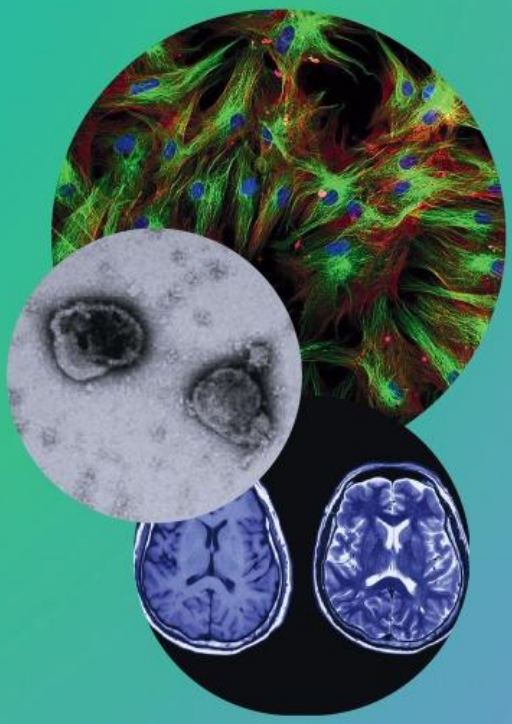
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