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30th CONGRESS OF THE POLISH PHYSIOLOGICAL SOCIETY

September 24 – 26, 2026

KATOWICE, POLAND

BOOK OF PROGRAMME AND ABSTRACTS

Guest Editor:
Tomasz Sawczyn

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PREFACE

Dear Colleagues,

Physiology has always occupied a unique position among the biomedical sciences. By seeking to understand how living organisms function and adapt to continuously changing internal and external conditions, it provides a fundamental link between molecular and cellular mechanisms, the function of organs and systems, and the organism as a whole. At the same time, physiology remains closely connected with medicine and other health sciences, providing a foundation for understanding both health and disease.

Over the past decades, physiological research has undergone remarkable development. Classical experimental approaches have been complemented by molecular biology, advanced imaging, bioinformatics, engineering, and modern methods of functional assessment. Increasingly, physiology integrates knowledge obtained at different levels of biological organization and translates basic scientific discoveries into a better understanding of human health, disease mechanisms, physical performance, adaptation, and potential therapeutic strategies. This interdisciplinary character makes physiology one of the key fields connecting fundamental and clinical sciences.

The Congresses of the Polish Physiological Society have for decades provided an important forum for presenting new scientific findings, exchanging ideas, establishing collaborations, and discussing the future directions of physiological research. The XXX Congress of the Polish Physiological Society, held in Katowice on 24–26 September 2026, represents an important milestone in this tradition. Thirty congresses reflect not only the long history of Polish physiology, but also the continuity of a scientific community that has developed across generations while remaining open to new concepts, technologies, and research challenges.

The scientific programme of the present Congress reflects the remarkable diversity of contemporary physiology. It brings together researchers representing different areas of experimental and human physiology and creates an opportunity to discuss physiological mechanisms from the cellular and molecular level to the integrated responses of the entire organism. Particular emphasis has been placed on interdisciplinary exchange and on strengthening the links between basic research, clinical sciences, exercise and environmental physiology, and emerging areas of biomedical research.

Katowice and the Silesian academic community provide a particularly appropriate setting for such a meeting. The region combines a strong scientific and medical tradition with dynamic development and close cooperation between universities and research institutions. We hope that this environment will encourage not only the presentation of scientific results, but also open discussion, the exchange of experience, the establishment of new collaborations, and the involvement of young researchers who will shape the future of physiology.

The present Supplement of the *Journal of Physiology and Pharmacology* brings together the scientific contributions presented during the XXX Congress and provides a record of the questions, concepts, and research directions currently being explored within our community. We hope that these contributions will stimulate further discussion and inspire new scientific projects extending beyond the Congress itself.

On behalf of the Organizing Committee, I would like to express my sincere gratitude to all invited speakers, Session Chairs and Co-Chairs, members of the Scientific and Organizing Committees, authors of oral and poster presentations, and all Congress participants. I would also like to thank the institutions and partners whose scientific, organizational, and financial support made this meeting possible, as well as the Editors of the *Journal of Physiology and Pharmacology* for enabling the publication of this Congress Supplement.

I sincerely hope that the XXX Congress of the Polish Physiological Society will be remembered not only as a celebration of the long tradition of Polish physiology, but above all as a meeting that contributes to its future, by inspiring scientific discussion, creating new collaborations, and encouraging the next generation of physiologists to ask new questions about the mechanisms that govern life.

GUEST EDITOR

Tomasz Sawczyn
Katowice, 2026



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The Organizing Committee greatly appreciate the financial support of the 30th Congress of Polish Physiological Society in Katowice, funded by the following companies:



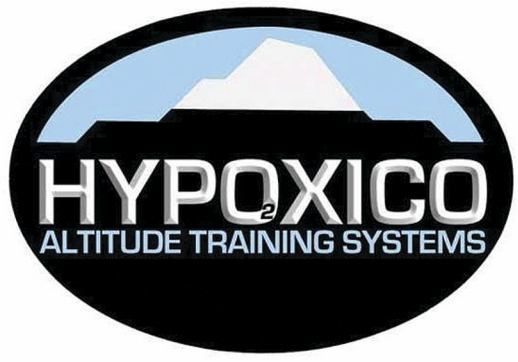












We would like to express our sincere gratitude to our Scientific Partners for their invaluable support, commitment, and contribution to the organization of this conference.

Your involvement and collaboration are greatly appreciated and have played an important role in making this event possible. Through our joint efforts, we are able to create a platform for the exchange of knowledge and experience, the presentation of the latest scientific achievements, and the development of new opportunities for collaboration.

Thank you for your trust, dedication, and continued support of initiatives that promote scientific excellence and foster cooperation within the academic and scientific community.

We are truly grateful to have you as our Scientific Partners and are delighted to share this special event with you.

With sincere appreciation,
The Conference Organizing Committee

30th CONGRESS OF THE POLISH PHYSIOLOGICAL SOCIETY
PROGRAMME

Thursday (September 24th, 2026)

9.30 – 11.15 General Assembly of Polish Physiological Society (Auditorium)
11.40 – 11.50 Opening Ceremony (Auditorium)
11.50 – 12.30 Opening Lecture 1: **EXTRACELLULAR VESICLES AND MICROBIAL SIGNALS AT THE FETOMATERNAL INTERFACE: NEW DIMENSIONS IN PREGNANCY PHYSIOLOGY?** M. Zygmunt (Department of Obstetrics and Gynecology , University of Greifswald, Germany), (Auditorium)
12.30 – 13.10 Opening Lecture 2: **PROTECTING THE BRAIN BARRIERS: EXERCISE AND THE NEUROBIOLOGY OF METHAMPHETAMINE TOXICITY.** M. Smolarz[1,2], A. Malecki[1], M. Nowacka-Chmielewska1, M. Toborek[1,2] ([1]Academy of Physical Education, Katowice, Poland; [2]Maria Sklodowska-Curie National Research Institute of Oncology, Gliwice Branch, Gliwice, Poland; [2]University of Miami, School of Medicine, Miami, USA), (Auditorium)
13.10 – 14.00 Lunch

ROOM A
14.00 – 15.25 **Session VI** – opening lecture and oral presentations (Part 1) **FUNCTION OF BLOOD. HEMOSTASIS**
15.30 – 16.00 Break
16.00 – 16.30 **Session VI** – oral presentations (Part 2) **FUNCTION OF BLOOD. HEMOSTASIS**
16.30 – 16.45 Poster session (Session VI)

ROOM B
14.00 – 15.30 **Session VII** – opening lecture and oral presentations (Part 1) **PHYSIOLOGY OF THE RESPIRATORY SYSTEM**
15.30 – 16.00 Break
16.00 – 16.30 **Session VII** – oral presentations (Part 2) **PHYSIOLOGY OF THE RESPIRATORY SYSTEM**

ROOM C
14.00 – 15.30 **Session X** – opening lecture and oral presentations (Part 1) **PHYSIOLOGY AND PATHOPHYSIOLOGY OF REPRODUCTION**
15.30 – 16.00 Break
16.00 – 16.45 **Session X** – oral presentations (Part 2) **PHYSIOLOGY AND PATHOPHYSIOLOGY OF REPRODUCTION**
16.45 – 17.30 Poster session (Session X)
17.45 – 18.45 Presentation provided by the sponsors
20.00 Formal dinner

Friday (September 25th, 2026)

ROOM A
8.30 – 10.10 **Session V** – opening lecture (1) and oral presentations (Part 1) **NEW TRENDS IN PHYSIOLOGY AND PATHOPHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM AND RENAL FUNCTION**
10.10 – 10.40 Break
10.40 – 12.45 **Session V** – opening lecture (2) and oral presentations (Part 2)

NEW TRENDS IN PHYSIOLOGY AND PATHOPHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM AND RENAL FUNCTION

12.45 – 13.30 Poster session (Session V)
13.30 – 14.30 Lunch
14.30 – 15.40 **Session II** – opening lecture and oral presentations (Part 1)
NEUROPHYSIOLOGY
15.40 – 16.00 Break
16.00 – 17.00 **Session II** – oral presentations (Part 2)
NEUROPHYSIOLOGY
17.20 – 18.10 Poster session (Sessions II)

ROOM B
8.30 – 10.00 **Session VIII** – oral presentations (Part 1)
ENDOCRINE REGULATIONS. CONTROL OF BODY WEIGHT
10.00 – 10.40 Break
10.40 – 11.10 **Session VIII** – oral presentations (Part 2)
ENDOCRINE REGULATIONS. CONTROL OF BODY WEIGHT
11.40 – 13.30 Poster session (Session VIII)
13.30 – 14.30 Lunch
14.30 – 15.40 **Session III** – opening lectures and oral presentation (Part 1)
EXERCISE PHYSIOLOGY
15.40 – 16.00 Break
16.00 – 17.00 **Session III** – oral presentations (Part 2)
EXERCISE PHYSIOLOGY
17.20 – 18.10 Poster session (Session III)

ROOM C
8.30 – 10.00 **Session IX** – opening lecture and oral presentations (Part 1)
GASTROINTESTINAL PHYSIOLOGY AND PATHOPHYSIOLOGY PANCREAS AND LIVER
10.00 – 10.40 Break
10.40 – 11.40 **Session IX** – oral presentations (Part 2)
GASTROINTESTINAL PHYSIOLOGY AND PATHOPHYSIOLOGY PANCREAS AND LIVER
11.40 – 12.40 Poster session (Session IX)
13.30 – 14.30 Lunch

Saturday (September 26th, 2026)

ROOM A
8.30 – 9.55 **Session I** – opening lecture and oral presentations (Part 1)
CELLULAR PHYSIOLOGY
10.00 – 10.30 Break
10.30 – 11.00 **Session I** – oral presentations (Part 2)
CELLULAR PHYSIOLOGY
11.00 – 12.45 Poster session (Session I)

ROOM B
8.30 – 9.45 **Session IV** – oral presentations
FATIGUE, ADAPTATION TO ENVIRONMENT, THERMOREGULATION
9.45 – 10.30 Break
10.30 – 11.30 Poster session (Session IV)

ROOM C
8.30 – 9.55 **Session XI** – opening lecture and oral presentations (Part 1)
ANIMAL PHYSIOLOGY AND PATHOPHYSIOLOGY

9.55 – 10.30 Break
10.30 – 12.30 **Session XI** – oral presentations (Part 2)
ANIMAL PHYSIOLOGY AND PATHOPHYSIOLOGY
12.30 – 13.45 Poster session (Session XI)
13.45 Closing ceremony

OPENING SESSION

Thursday (September 24th; 9.30 – 13.10) Auditorium

Chairman: Tomasz Brzozowski
President of the Polish Physiological Society
Department of Physiology, Faculty of Medicine,
Jagiellonian University Medical College, Krakow, Poland

Chairman: Stanislaw Okrasa
University of Warmia and Mazury in Olsztyn, Olsztyn, Poland

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Institute of Physiotherapy and Health Sciences
The Jerzy Kukuczka Academy of Physical Education in Katowice, Katowice, Poland

Chairman: Tomasz Sawczyn
Department of Physiology
Faculty of Medical Sciences in Katowice,
Medical University of Silesia, Katowice, Poland

DETAILED OPENING SESSION SCHEDULE

General Assebly of Polish Physiological Society (Thursday, September 24th, 9.30 – 11.15)

Opening ceremony (Thursday, September 24th, 11.40 – 11.50)

Opening lecture 1 (Thursday, September 24th, 11.50 – 12.30)

S0.L1 EXTRACELLULAR VESICLES AND MICROBIAL SIGNALS AT THE FETOMATERNAL INTERFACE: NEW DIMENSIONS IN PREGNANCY PHYSIOLOGY? M. Zygmunt (Department of Obstetrics and Gynecology, University of Greifswald, Germany).

Opening lecture 2 (Thursday, September 24th, 12.30 – 13.10)

S0.L2 PROTECTING THE BRAIN BARRIERS: EXERCISE AND THE NEUROBIOLOGY OF METHAMPHETAMINE TOXICITY. M. Smolarz[1,2], A. Malecki[1], M. Nowacka-Chmielewska[1], M. Toborek[1,2] ([1]Academy of Physical Education, Katowice, Poland; [2]Maria Sklodowska-Curie National Research Institute of Oncology, Gliwice Branch, Gliwice, Poland; [2]University of Miami, School of Medicine, Miami, USA; mtoborek@med.miami.edu).

S0.L1

EXTRACELLULAR VESICLES AND MICROBIAL SIGNALS AT THE FETOMATERNAL INTERFACE: NEW DIMENSIONS IN PREGNANCY PHYSIOLOGY?

M. ZYGMUNT

Department of Obstetrics and Gynecology, University of Greifswald, Germany; marek.zygmunt@med.uni-greifswald.de

The fetomaternal interface is a highly dynamic physiological environment where maternal and fetal tissues continuously exchange molecular signals to maintain a healthy pregnancy. While classical research has focused on endocrine regulation, angiogenesis, immune tolerance, and vascular adaptation, recent discoveries have identified extracellular vesicles (EVs) and microbial-derived signals as key mediators of intercellular communication. Placenta-derived EVs participate in the regulation of trophoblast function, vascular remodeling, immune responses, and maternal metabolic adaptation. At the same time, growing evidence suggests that bacterial products and microbial metabolites may influence placental physiology and pregnancy outcome, either through the maternal microbiome or infectious processes. These findings have expanded our understanding of the complex signaling networks operating at the fetomaternal interface. This lecture will review the evolution of our knowledge from classical concepts of placental physiology to emerging mechanisms involving extracellular vesicles and microbial communication. Particular emphasis will be placed on the role of these signaling pathways in normal pregnancy and their potential contribution to pregnancy complications such as preeclampsia, fetal growth restriction, and preterm birth. Finally, future perspectives for the use of EVs and microbial biomarkers as diagnostic tools and therapeutic targets in obstetric medicine will be discussed.

S0.L2

PROTECTING THE BRAIN BARRIERS: EXERCISE AND THE NEUROBIOLOGY OF METHAMPHETAMINE TOXICITY

M. SMOLARZ[1,2], A. MALECKI[1], M. NOWACKA-CHMIELEWSKA[1], M. TOBOREK[1,2]

[1]Academy of Physical Education, Katowice, Poland; [2]Maria Sklodowska-Curie National Research Institute of Oncology, Gliwice Branch, Gliwice, Poland; [2]University of Miami, School of Medicine Miami, USA; mtoborek@med.miami.edu

Methamphetamine (METH) misuse is associated with neuroinflammation and cognitive impairment; however, the molecular mechanisms underlying these effects and potential strategies for mitigating METH-induced neurotoxicity remain incompletely understood. We investigated the role of inflammatory signaling, with a particular focus on inflammasome activation, in METH-induced neurotoxicity. Prolonged METH exposure disrupted blood-brain barrier (BBB), induced systemic and hippocampal inflammation, including increased IL-1β expression and alterations in the inflammasome profile, and was accompanied by impaired spatial learning and memory, abnormal neurogenesis, and alterations in proteomic profiles. Importantly, these effects exhibited substantial sexual dimorphism. Pharmacological inhibition of inflammasome signaling attenuated several METH-induced inflammatory responses. Similarly, neutralization of IL-1 signaling using IL-1 Trap reduced hippocampal IL-1β levels and attenuated METH-induced cognitive deficits, further implicating IL-1β signaling and inflammasome as important mediators of METH-related cognitive dysfunction. In addition, physical exercise reduced METH-induced BBB dysfunction, neuroinflammation and neurogenesis impairment; however, its protective efficacy was dependent on the level of physical activity and varied between sexes. Collectively, these findings demonstrate that METH-induced cognitive decline involves coordinated inflammatory and neurogenic alterations, including inflammasome signaling, and that targeted pharmacological and behavioral interventions can mitigate these effects. The pronounced sex-dependent differences observed across METH exposure, inflammasome inhibition, and exercise responses highlight the importance of considering biological sex when developing targeted therapeutic and recovery strategies for METH misuse.

This study was supported by the National Science Centre (NSC) grant 2019/35/B/NZ7/03155 and a grant from the Jerzy Kukuczka Academy of Physical Education AWF/NF/ZB1/2024.

SESSION I

CELLULAR PHYSIOLOGY

Saturday (September 26th, 8.30 – 12.45) Room A

Chairman: Associate Professor Anna Gromotowicz-Poplawska
(Department of Biopharmacy and Radiopharmacy, Radiopharmacy Centre,
Medical University of Bialystok, Bialystok, Poland)

Vice-Chairman: Dr Michal Kowara
(Department of Experimental and Clinical Physiology,
Medical University of Warsaw, Warsaw, Poland)

DETAILED SESSIONS I SCHEDULE

Opening lectures (Saturday, September 26th, 8.30 – 9.10)

S1.L1 HOW CELLS BECOME MEDICINES: CELLULAR PHYSIOLOGY AS THE FOUNDATION OF ADVANCED THERAPIES. **A. Zimna[1], O. Suska[2], T. Kolanowski[1,2], N. Rozwadowska[1,2]** ([1]Institute of Human Genetics, Polish Academy of Sciences, Poznan, Poland; [2]FamiCordTX, Warsaw, Poland).

Oral presentations (Saturday, September 26th, 9.10 – 11.00)

S1.L2 *9.10 – 9.25* THE UTILIZATION OF CIRCULATING EXOSOMAL MICRORNA EXPRESSION PROFILING TO DISTINGUISH ISCHEMIC STROKE SUBTYPES BY ORIGIN. **D. Gendosz de Carrillo[1], O. Kocikowska[1], M. Rak[1], A. Wicher[1], J. Mi[2], M. Burek[2], A. Krzan[3], A. Lasek-Bal[3], H. Jedrzejowska-Szypulka[1]** ([1]Department of Physiology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland; [2]Department of Anesthesiology, Intensive Care, Emergency and Pain Medicine, Center of Operative Medicine, University Hospital Würzburg, Würzburg, Germany; [3]Department of Neurology, Faculty of Health Sciences, Medical University of Silesia, Katowice, Poland).

S1.L3 *9.25 – 9.40* WHEN FUSION GOES WRONG: THE ROLE OF UNCONVENTIONAL MYOSIN VI IN MYOGENESIS. **L. Lehka** (Laboratory of Molecular Basis of Cell Motility, Nencki Institute of Experimental Biology PAS, Warsaw, Poland).

S1.L4 *9.40 – 9.55* MACA EXTRACTS MODULATE DOXORUBICIN RESPONSE AND LIPID METABOLISM IN PROSTATE CANCER CELLS: IMPACT OF TUMOR MICROENVIRONMENT. **A.J. Olichwier[1], I.S. Kalita[1], M. Bruzgo-Grzybko[2], A. Gromotowicz-Poplawska[1,2]** (Radiopharmacy Centre, Medical University of Bialystok, Bialystok, Poland; Department of Biopharmacy and Radiopharmacy, Medical University of Bialystok, Bialystok, Poland).

9.55 – 10.30 Coffee break

S1.L5 *10.30 – 10.45* SPATIALLY RESOLVED LIPIDOMIC AND AMINO ACID HETEROGENEITY OF GOOSE SKELETAL MUSCLE FIBERS REVEALED BY TOF-SIMS. **M.E. Skalska[1,2], D. Wojtysiak[3]** ([1]Department of Medical Physics, M. Smoluchowski Institute of Physics, Faculty of Physics, Astronomy and Applied Computer Science, Jagiellonian University, Krakow, Poland; [2]Centre for Theranostics, Jagiellonian University, Krakow, Poland; [3]Department of Genetics, Animal Breeding and Ethology, University of Agriculture in Krakow, Krakow, Poland).

S1.L6 *10.45 – 11.00* A PHASE-AWARE POLARITY HOMEOSTAT: A UNIFIED MODEL OF CELLULARMEMBRANE POTENTIAL REGULATION COUPLING METABOLIC, BIOELECTRIC, AND SPATIAL DYNAMICS. **W.A. Pawlak[1], T. Buchner[2]** ([1]University of California, Berkeley, CA, USA; [2]Faculty of Physics, Warsaw University of Technology, Warsaw, Poland).

Poster Session (Saturday, September 26th, 11.00 – 12.45)

S1.P1 METABOLIC STRESS SIGNATURES OF CADMIUM TOXICITY REVEALED BY MASS SPECTROMETRY AND STABLE ISOTOPE TRACING. **A.A. Gonzalez-Ibarra[1], M. Arroyo-Negrete[1], W. Banaszuk-Krupa[2,3], B. Paziewska[4], A. Rivero-Muller[5], E. Yanez-Barrientos[1], J. Valverde Piedra[5], M. Tchorzewski[2], M. Hatzoglou[6], D. Krokowski[2]** ([1]University of Guanajuato, Guanajuato, Mexico; [2]Maria Curie-Sklodowska University, Lublin, Poland; [3]Doctoral School of Quantitative and Natural Sciences, Maria Curie-Sklodowska University, Lublin, Poland; [4]Medical University of Lublin, Lublin, Poland; [5]University of Life Sciences in Lublin, Lublin, Poland; [6]Case Western Reserve University School of Medicine, Cleveland, OH, USA).

S1.P2 CELL-MEMBRANE AND SECRETORY CHANGES IN ADIPOSE-DERIVED MESENCHYMAL STEM CELLS DURING LONG-TERM CULTURE. **M. Slotwinski[1,2], M. Skalska[1,2], M. Jez[1,2], E.L. Stepien[1,2]** ([1]Department of Medical Physics, M. Smoluchowski Institute of Physics, Krakow, Poland; [2]Centre for Theranostics, Jagiellonian University, Krakow, Poland).

S1.P3 THE ROLE OF THE TUMOR MICROENVIRONMENT IN PANCREATIC CANCER: COMPARISON OF 2D AND 3D CO-CULTURE MODELS. **M. Bruzgo-Grzybko[1], I.S. Kalita[2], A.J. Olichwier[2], N. Bielicka[1], E. Chabielska[1], A. Gromotowicz-Poplawska[1,2]** ([1]Department of Biopharmacy and Radiopharmacy, Medical University of Bialystok, Bialystok, Poland; [2]Radiopharmacy Centre, Medical University of Bialystok, Bialystok, Poland).

S1.P4 ECIS SCRATCH TEST AS A MODERN TOOL FOR STUDYING THE MIGRATION PROCESSES OF COLON CANCER CELLS (CT-26) EXPOSED TO LACCASE DERIVED FROM THE FUNGUS *CERRENA UNICOLOR*. **D. Sondej[1], M. Prendecka-Wrobel[1], D. Pigon-Zajac[1], M. Jaszek[2], D. Stefaniuk[2], A. Matuszewska[2], M. Rahnama-Hezavah[3], T. Malecka-Massalska[1]** ([1]Department of Human Physiology of the Chair of Preclinical Sciences, Medical University of Lublin, Lublin, Poland; [2]Department of Biochemistry and Biotechnology, Institute of Biological Sciences, Maria Curie-Sklodowska University, Lublin, Poland; [3]Department of Dental Surgery, Medical University of Lublin, Lublin, Poland).

S1.P5 NANOPLASTICS ENHANCE PRO-INFLAMMATORY RESPONSES IN HUMAN MICROGLIA. RELEVANCE TO NEURODEGENERATIVE DISEASE PROGRESSION. **E. Grzeda, N. Leciejewska, E. Pruszynska-Oszmalek, M. Sassek, M. Nowak, P. Juzwik, P. Kolodziejski** (Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland).

S1.P6 BRAIN EDEMA IS ASSOCIATED WITH INCREASED GENE EXPRESSION OF PROIFLAMMATORY FACTORS IN EXPERIMENTAL HYPONATREMIA. **M. Aleksandrowicz[1], P. Wencel[1], L. Przykaza[1], M. Popek[2]** ([1]Laboratory of Preclinical Research and Environmental Agents, Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland; [2]Department of Neurotoxicology, Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland).

S1.P7 LEAP-2 AS A REGULATOR OF MUSCLE CELL FUNCTION: A STUDY USING THE L6 CELL LINE. **M. Nowak[1], N. Leciejewska[1], P. Juzwik[1], E. Grzeda[1], A. Gorka[1], O. Sosinski[1], M. Gogulski[2], P. Kolodziejski[1]** ([1]Department of Animal Physiology and Biochemistry, Poznan University of Life Science, Poznan, Poland; [2]Department of Preclinical Sciences and Infectious Diseases, Poznan University of Life Sciences, Poznan, Poland).

S1.L1

HOW CELLS BECOME MEDICINES: CELLULAR PHYSIOLOGY AS THE FOUNDATION OF ADVANCED THERAPIES

A. ZIMNA[1], O.SUSKA[2], T. KOLANOWSKI[1,2], N. ROZWADOWSKA[1,2]

[1]Institute of Human Genetics, Polish Academy of Sciences, Poznan, Poland; [2]FamiCordTX, Warsaw, Poland; natalia.rozwadowska@igcz.poznan.pl

The advances in biotechnology have transformed cells from experimental models into therapeutic products. ATMPs (Advanced Therapy Medicinal Products) are innovative medicines based on genes, cells, or engineered tissues, developed to treat, prevent, or cure diseases. New therapeutic approaches, ranging from induced pluripotent stem cells (iPSCs) to genetically modified immune cells such as CAR-T cells, are being developed worldwide, and several therapies have already been approved by regulatory agencies. The transition from experimental models to medicinal products requires the establishment of new research platforms that define the pathways and key steps involved in ATMP design, evaluation, and approval. The talk will emphasize both the great therapeutic potential of ATMPs and the scientific and regulatory challenges that must be overcome before cells can be regarded as medicines.

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S1.L2

THE UTILIZATION OF CIRCULATING EXOSOMAL MICRORNA EXPRESSION PROFILING TO DISTINGUISH ISCHEMIC STROKE SUBTYPES BY ORIGIN

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By identifying novel molecular biomarkers, scientists hope to improve both the diagnostic capabilities and prognostic insights related to ischemic stroke and the disability that can follow. MiRNAs, circulating in the blood either freely or within exosomes, possess the capability to influence the progression of ischemia-induced brain damage at both localized and systemic levels. Their transfer between cells can differentially affect recipient cells. Several mechanisms can lead to ischemic stroke, and atrial fibrillation (AF) along with diabetes mellitus (DM) stand out as particularly common causes. We aimed to discover exosome-bound circulating miRNAs that could potentially characterize ischemic stroke patients originating from cardiac issues or diabetes mellitus. 72 individuals who had experienced an ischemic stroke (IS) were part of the study. Among them, 28 were identified as having a cardiac ischemic stroke, and 25 of the ischemic patients also had diabetes mellitus. Serum collected on days 1 and 10 post-IS was used to isolate exosomes. The levels of 48 distinct miRNAs, isolated from exosomes, were analyzed *via* qRT-PCR. Of the miRNAs that were analyzed, 5 demonstrated a link to IS and AF, while 2 showed a correlation with IS and DM. On day 1, the levels of miR-17-5p, 20a-5p, 23a-3p, 26b-5p, and 186-5p were elevated in both the IS and AF groups compared to the IS group alone. On day 10, this effect was not apparent; the IS and AF groups showed a significant decrease in levels over time, a trend not observed in the IS group. There was no link between miRNA expression in the IS and AF groups and hypertension or myocardial infarction. Reduced levels of these miRNAs in patients experiencing IS and AF were linked to unfavorable outcomes on the mRS scale at 10 days, 90 days, or both. On the first day after IS, the IS group with DM showed elevated levels of miR-181c-5p and miR-30b-5p compared to the IS group alone. However, this difference was not apparent by day 10. Patients who had an ischemic stroke and also had diabetes showed a better prognosis on the mRS scale at both 10 and 90 days post-treatment. The presence AF has been observed to elevate the likelihood of experiencing a stroke and also negatively influence the patient outcomes following IS, particularly in the group that exhibited both IS and AF; however, we found that the concurrent existence of DM contributed to a better prognosis for patients who had suffered an IS.

Study funding: MFWN, RW/59/2026.

S1.L3

WHEN FUSION GOES WRONG: THE ROLE OF UNCONVENTIONAL MYOSIN VI IN MYOGENESIS

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Skeletal muscle formation and regeneration depend on tightly coordinated myoblast differentiation and fusion into multinucleated myotubes. Although myogenesis has been extensively studied, the molecular mechanisms regulating these processes remain unclear. Our recent findings reveal, for the first time, that the loss of unconventional myosin VI (MVI) disrupts myotube formation. Myoblasts derived from MVI knockout mice (MVI-KO) form aberrant myotubes characterized by the spindle-like morphology and centrally positioned nuclei, suggesting impaired differentiation and/or fusion dynamics. Myoblast fusion is a critical step in myotube formation, which is controlled by a complex network of regulatory proteins. Recently, we observed that the expression levels of key fusogenic proteins, Myomaker and Myomerger, were significantly upregulated in differentiating MVI-KO myoblasts. These alterations in the fusion-related machinery were accompanied by elevated levels of reactive oxygen species (ROS) in MVI-KO myogenic cells. While the precise relationship between ROS and myoblast fusion remains unclear, emerging evidence suggests that excessive ROS level under the lack of MVI may regulate Myomaker expression. Taken together, the presented data indicate that MVI plays an important role in maintaining redox homeostasis during myogenic differentiation and may modulate myoblast fusion through ROS-dependent regulation of fusion machinery. We believe that this might be the result of impaired mitochondrial function in myogenic cells under MVI loss.

This work was supported by National Science Center, Poland (Grant number: 2022/47/D/NZ3/02737).

S1.L4

MACA EXTRACTS MODULATE DOXORUBICIN RESPONSE AND LIPID METABOLISM IN PROSTATE CANCER CELLS: IMPACT OF TUMOR MICROENVIRONMENT

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Tumor–stroma interactions critically shape prostate cancer progression and therapy response. Natural compounds with modulatory effects on tumor biology may influence these interactions, however, their activity within a physiologically relevant microenvironment remains poorly understood. This study evaluated whether maca (*Lepidium meyenii*) extracts: yellow (MY), red (MR), and black (MB), modulate doxorubicin (DOX) responses in prostate cancer cells, with emphasis on tumor-stroma interactions and metabolic regulation. Human prostate cancer 22Rv1 cells were treated with maca extracts under monoculture and co-culture with human dermal fibroblasts (HDFa), followed by assessment of cytotoxicity, apoptotic signaling, proliferation, and metabolic markers. In monoculture, maca extracts exhibited limited cytotoxicity, with MB exerting the strongest effect. Co-treatment with DOX did not enhance cytotoxicity or increase caspase-3 activation despite modulation of caspase-8, indicating altered apoptotic signaling. In contrast, co-culture markedly reduced DOX sensitivity, suggesting a protective role of fibroblasts and highlighting tumor microenvironment-driven resistance. Maca extracts induced extract-dependent effects on proliferation, with reduced proliferation at lower DOX concentrations and increased proliferation for MY and MR at higher doses. A consistent metabolic effect was reduced lipid droplet accumulation, particularly for MY, accompanied by increased ATGL levels, indicating enhanced lipolysis and metabolic reprogramming. In co-culture, maca extracts modulated key signaling pathways, including increased PI3K and PSMA levels and decreased FAP expression, while selected transcriptional regulators showed extract-dependent responses. Collectively, these results demonstrate that maca extracts act primarily as modulators of cancer cell metabolism and signaling rather than direct cytotoxic agents, and their activity is strongly dependent on microenvironmental context.

This research was funded by the Medical University of Bialystok, grant numbers: B.SUB.25.359 and B.SUB.26.376.

S1.L5

SPATIALLY RESOLVED LIPIDOMIC AND AMINO ACID HETEROGENEITY OF GOOSE SKELETAL MUSCLE FIBERS REVEALED BY TOF-SIMS

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Skeletal muscle tissue is characterised by substantial morphological, biochemical and metabolic heterogeneity, reflecting the presence of oxidative and glycolytic muscle fibre types. These fibre types differ in mitochondrial content, lipid metabolism, membrane composition, and amino acid turnover. Time-of-Flight Secondary Ion Mass Spectrometry (ToF-SIMS) enables label-free molecular imaging with high spatial resolution and may provide insights into the biochemical organisation of skeletal muscle fibres. This study aimed to investigate the spatial distribution of lipid- and amino acid-related ions in goose skeletal muscle (*pectoralis superficialis*) tissue and to identify molecular differences between distinct muscle fibre types. Prior histochemical staining enabled visualisation of heterogeneous muscle fibre composition and facilitated interpretation of molecular imaging results. generated for selected low- and high-mass ions associated with fatty acids, phospholipids, and amino acid-related fragments. Distinct localisation patterns were observed for several lipid-associated ions, including oleic acid FA(18:1) ($C_{18}H_{33}O_2^-$), linolenic acid FA(18:3) ($C_{18}H_{29}O_2^-$), palmitic acid FA(16:0) ($C_{16}H_{31}O_2^-$), and arachidonic acid-related fragments ($C_{20}H_{37}O_2^-$). Furthermore, phospholipid- and sphingolipid-related ions, including phosphatidylethanolamine and phosphatidylcholine fragments (PO_3^-, $PO_4H_2^-$, $C_{36}H_{69}PNO_6^-$, $C_{39}H_{75}PNO_8^-$), demonstrated heterogeneous spatial distributions within the analysed tissue sections. Regions enriched in phospholipid-related signals correspond to oxidative muscle fibres characterised by different membrane content of lipids. In addition, amino acid- and nitrogen-containing fragments, including $C_2H_3N_2O^-$, $C_4H_8N_2O_2^-$, and $C_3H_6N_2O_3^-$, exhibited distinct regional localisation, indicating metabolic differences between muscle fibre types. The observed molecular heterogeneity was reproducible across multiple analysed sections. The obtained results demonstrate that ToF-SIMS imaging enables discrimination of biochemically distinct regions within goose skeletal muscle tissue and may serve as a valuable tool for spatial metabolomic characterisation of muscle fibre heterogeneity. The presented approach provides new insight into lipid and amino acid organisation in avian skeletal muscle and may contribute to future studies on muscle physiology and metabolic specialisation.

S1.L6

A PHASE-AWARE POLARITY HOMEOSTAT: A UNIFIED MODEL OF CELLULARMEMBRANE POTENTIAL REGULATION COUPLING METABOLIC, BIOELECTRIC AND SPATIAL DYNAMICS

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In this submission we discuss the regulatory role of membrane potential (Vmem) as both subject and object. The electric state of the cell (the electrome) is often treated as a constant feature, under active control only in electrically excitable cells such as neurons and myocytes. Yet Vmem undergoes sustained depolarization in various pathological states, including cancer initiation and metabolic disease. In certain cases Vmem is an additional factor helping the cell maintain homeostasis, and one of the important features of the electrome is the ability to control Vmem itself - which may be intentionally increased to accelerate the cell cycle in embryonic development or wound healing. A further property of Vmem is its ability to couple the oscillations of neighboring cells, helping to organize the colony. This interaction is equivalent to chemical signaling and should be considered an important part of the metabolic network, as the ability of Vmem to directly affect gene transcription has also been demonstrated. Here we present a phase-aware polarity homeostat - a minimal model of cellular Vmem control. It comprises several regulatory loops that together form a composite controller structure. The regulatory circuit depends on the functional state of the cell, specifically its current Vmem and metabolic state perceived *via* ATP/AMPK status. It also incorporates the phase-locking value (PLV), a variable that senses the bioelectric state of neighbors and allows the cell to maintain a synchronized state. The actuation arm of the controller uses Kir4.1/HCN_2-based regulation of Vmem. The complex spatial and temporal dynamics defined above place the system in the family of reaction-diffusion models in which spatiotemporal patterns have been observed. We discuss the possibility of observing patterns richer than the known examples of metabolic waves. The homeostat provides a generic, unified computational model connecting bioelectric control logic to the metabolic state of the cell and to the electric state of its neighbors.

S1.P1

METABOLIC STRESS SIGNATURES OF CADMIUM TOXICITY REVEALED BY MASS SPECTROMETRY AND STABLE ISOTOPE TRACING

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Cadmium (Cd) is a toxic environmental metal that disrupts mitochondrial metabolism, redox balance and tissue homeostasis. This study characterised amino acid and polyamine changes induced by Cd (II) and evaluated candidate metabolic markers of toxicological stress. Mouse embryonic fibroblasts, HepG2 cells and zebrafish (*Danio rerio*) larvae were exposed to Cd (II). Primary amine metabolites were quantified by liquid chromatography-tandem mass spectrometry after derivatisation, and stable isotope tracing with ^{13}C-glucose, ^{15}N-ammonium chloride, ^{13}C-methionine and ^{15}N-isoleucine was used to assess carbon and nitrogen redistribution. Cd (II) reduced glutamate and aspartate concentrations and decreased glucose-derived ^{13}C incorporation into tricarboxylic acid cycle-associated amino acids, indicating impaired mitochondrial biosynthetic capacity. Exposure also activated the integrated stress response, increased LAT1-dependent amino acid exchange, promoted accumulation of branched-chain and aromatic amino acids, and enhanced ornithine decarboxylase-associated polyamine production. A consistent increase in α-aminobutyric acid across models, supported by isotope tracing, indicates disrupted amino acid homeostasis and identifies this metabolite as a candidate marker of Cd (II)-induced metabolic stress.

S1.P2

CELL-MEMBRANE AND SECRETORY CHANGES IN ADIPOSE-DERIVED MESENCHYMAL STEM CELLS DURING LONG-TERM CULTURE

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For many years, human adipose-derived mesenchymal stem cells (AdMSCs) have been considered a versatile and readily available tool in regenerative medicine. Recent studies have demonstrated that extracellular vesicles (EVs) secreted by AdMSCs also possess significant therapeutic potential. Nevertheless, prolonged in vitro expansion may negatively influence AdMSCs. Late-passage AdMSCs (P11) exhibited enlarged cell size. Oil Red O staining revealed altered intracellular lipid organization, characterized by a more dispersed distribution in the cytoplasm. Consistently, Time-of-Flight Secondary Ion Mass Spectrometry (TOF-SIMS) analysis demonstrated significant differences in lipid and amino acid profiles in late-passage cells. Interestingly, these changes were accompanied by enhanced extracellular vesicle (EV) secretion in prolonged cultures. In summary, these findings indicate that prolonged culture affects the morphological and biochemical characteristics of AdMSCs and may consequently influence properties of secreted EVs. Such changes may be critical for the application of EVs in radiovesicle-based imaging approaches and drug delivery systems.

S1.P3

THE ROLE OF THE TUMOR MICROENVIRONMENT IN PANCREATIC CANCER: COMPARISON OF 2D AND 3D CO-CULTURE MODELS

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Pancreatic ductal adenocarcinoma (PDAC) is one of the most aggressive solid malignancies, characterized by poor prognosis due to late diagnosis and the presence of a highly desmoplastic tumor microenvironment (TME). A major component of the TME are cancer-associated fibroblasts (CAFs), which arise from activated stromal fibroblasts and exhibit elevated expression of fibroblast activation protein (FAP), a protease involved in extracellular matrix remodeling and inflammatory regulation. CAFs influence cytokine secretion and tumor-stroma interactions, contributing to tumor progression and therapy resistance. This study aimed to develop and compare hybrid 2D and 3D co-culture models composed of PANC-1 pancreatic cancer cells and HDFa fibroblasts to better mimic the tumor microenvironment. Cell morphology, spheroid formation, and spatial organization were evaluated by microscopy, while radiotracer uptake was quantified using a gamma counter following incubation with FAPI-based radiotracers. The developed 3D models reproduced key *in vivo*-like features, including spatial cell organization, oxygen and nutrient gradients, as well as cell-cell and cell-matrix interactions that regulating proliferation and migration. Radiotracer uptake studies demonstrated increased accumulation of FAPI radiotracers in co-culture spheroids compared with monoculture systems, indicating enhanced FAP-related activity in the presence of fibroblasts. Overall, these findings highlight the potential of 3D co-culture systems as a valuable preclinical tool for improving the translational relevance of studies on tumor microenvironment-targeted diagnostics and therapies.

This research was funded by Medical University of Bialystok grant number B.SUB.26.609

S1.P4

ECIS SCRATCH TEST AS A MODERN TOOL FOR STUDYING THE MIGRATION PROCESSES OF COLON CANCER CELLS (CT-26) EXPOSED TO LACCASE DERIVED FROM THE FUNGUS *CERRENA UNICOLOR*

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Electric Cell Substrate Impedance Sensing *(*ECIS) is a modern method that visualizes changes in cellular electrical parameters in real time. Our previous studies demonstrated the effect of laccase isolated from the medicinal fungus *Cerrena unicolor* on Colon Cancer Cells (CT26). This time, migration and regeneration processes were examined using the ECIS scratch test. For this purpose, cell lines cultured on electrodes were exposed to specific current parameters. The wound thus created was recolonized by cells after some time. Differences were observed in the re-confluence of L929 and CT26 cells depending on the concentration of the fungal isolate used. In the case of the cancer cell line, there was no increase in impedance from a concentration of 2,5 µg/mL of laccase, whereas in the case of the L929 cell line, a significant effect was observed from a concentration of 250 µg/mL. This confirms the anticancer activity of laccase on the colon cancer cell line. The rate of refilling the resulting gap reflects the metastatic potential of the cells. Furthermore, this provides modern insight into the dynamics of migratory processes, providing a new perspective on the use of *in vitro* studies to better understand cell biology.

S1.P5

NANOPLASTICS ENHANCE PRO-INFLAMMATORY RESPONSES IN HUMAN MICROGLIA. RELEVANCE TO NEURODEGENERATIVE DISEASE PROGRESSION

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Neuroinflammation is increasingly recognized as a critical contributor to the development and progression of neurodegenerative diseases, including Alzheimer's disease (AD). Microglia, the resident immune cells of the central nervous system, play a key role in regulating inflammatory responses and maintaining neural homeostasis. Chronic microglial activation may contribute to neuronal dysfunction and neurodegeneration. Nanoplastics (NPs), emerging environmental contaminants capable of crossing biological barriers, may accumulate in the brain and induce oxidative stress and inflammatory signaling. However, their effects on human microglia under neuroinflammatory conditions remain insufficiently characterized. In the present study, human microglial HMC3 cells were exposed to nanoplastics, lipopolysaccharide (LPS), or combined NP+LPS treatment. Cell viability and metabolic activity were assessed using MTT and Neutral Red Uptake (NRU) assays, proliferation was evaluated by BrdU incorporation, and inflammatory gene expression was analyzed using quantitative PCR (qPCR).Exposure to NPs resulted in time- and treatment-dependent alterations in microglial activity. NP and NP+LPS treatment increased proliferative activity, particularly after 48 h of exposure, while NRU assay results suggested disturbances in lysosomal function and cellular metabolic homeostasis. qPCR analysis confirmed activation of pro-inflammatory pathways, with the most pronounced effects observed following combined NP and LPS exposure. These findings demonstrate that nanoplastics modulate human microglial function and may potentiate neuroinflammatory responses, supporting their potential involvement in mechanisms contributing to neurodegeneration.

The project is financed from the Polish State Budget funds allocated by the Minister of Science and Higher Education under the Science for the Development of Society program.

S1.P6

BRAIN EDEMA IS ASSOCIATED WITH INCREASED GENE EXPRESSION OF PROIFLAMMATORY FACTORS IN EXPERIMENTAL HYPONATREMIA

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Hyponatremia, defined as a decrease in the plasma Na^+ concentration below 135 mM belongs to the most common electrolyte abnormality in hospitalized patients. It is associated with neurological deficits, that are primarily linked to brain edema. Cerebral edema frequently triggers an inflammatory response, which may significantly affect the permeability of the blood–brain barrier (BBB). The aim of the present study was to evaluate the effect of acute (<48 h) and chronic (>2–3 days) hyponatremia on the development of brain edema, BBB integrity, and cortical gene expression of selected proinflammatory factors: interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), and inducible nitric oxide synthase (iNOS). The experiments were conducted in male Wistar rats using two models of hyponatremia: acute (6 h) and chronic (4 days). Acute hyponatremia was induced by subcutaneous (s.c.) injection of arginine vasopressin (AVP), a hormone involved in the pathogenesis of hyponatremia, or its synthetic analog desmopressin (dDAVP), combined with intraperitoneal (i.p.) water loading equivalent to 11% of body weight. Chronic hyponatremia was induced by continuous s.c. delivery of AVP or dDAVP using implanted ALZET mini osmotic pumps in combination with a liquid diet. Brain water content was assessed using the wet–dry method. BBB permeability was evaluated using sodium fluorescein. Gene expression levels of IL-1β, TNF-α, and iNOS in the cerebral cortex were measured by real-time PCR (RT-PCR). Plasma osmolality, Na^+, and Cl^- concentrations were analyzed using an electrolyte and blood gas analyzer. Brain water content increased in acute AVP- and dDAVP-induced hyponatremia (respectively to 79.52% ± 0.11, n=6 and to 79.40% ± 0.09, n=6 vs. 78.36% ± 0.11 in Sham, n=6, $p<0.001$). In contrast, no increase in brain water content was observed in the chronic hyponatremia groups. No BBB leakage was detected in either acute or chronic hyponatremia. In acute hyponatremia, a nearly two fold increase in IL-1β gene expression (n=10, $p<0.05$) and a six-fold increase in iNOS gene expression (n=10, $p<0.001$) were observed compared with controls. These changes were not present in chronic hyponatremia. TNF-α gene expression did not differ from control values in either acute or chronic hyponatremia. Brain edema observed in acute, but not chronic, hyponatremia correlates with increased gene expression of proinflammatory IL-1β and iNOS, without detectable changes in BBB permeability.

This study was supported by the grant 2021/43/D/NZ4/03304 from National Science Centre, Poland.

S1.P7

LEAP-2 AS A REGULATOR OF MUSCLE CELL FUNCTION: A STUDY USING THE L6 CELL LINE

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Liver-Enriched Antimicrobial Peptide 2 (LEAP-2), a 40-amino-acid peptide initially identified in the liver, was first described in 2003 (Krause *et al*., 2003). As an antimicrobial peptide, it forms part of the innate immune system and contributes to host defense against bacterial infections. However, a 2018 study (Ge *et al*., 2018) demonstrated that LEAP-2 also exerts important metabolic functions, acting as an endogenous antagonist of ghrelin and its receptor, GHS-R1a. Despite these advances, the influence of LEAP-2 on muscle cell biology remains poorly understood. Therefore, the present study aimed to evaluate the effects of LEAP-2 on muscle cell function, including viability, survival, and myogenic differentiation.The L6 myoblast cell line was used to assess the impact of LEAP-2 on key physiological processes related to cell viability and differentiation. Cell survival was evaluated using MTT and NRU assays. The effect of LEAP-2 on myogenic differentiation was examined at both early and late stages by analyzing the mRNA expression of stage-specific marker genes using real-time PCR. Our results indicate that LEAP-2 slightly enhances cell survival and modulates the differentiation program of L6 cells. Moreover, LEAP-2 altered the expression of genes associated with myogenic progression, suggesting a regulatory effect on the transition from myoblasts to myocytes. Overall, these findings demonstrate that LEAP-2 influences the differentiation processes and functional properties of L6 muscle cells. These results suggest that LEAP-2 may serve as a potential regulator of muscle cell function and could play a broader role in skeletal muscle physiology than previously recognized.

National Science Centre grant number 2021/41/B/NZ5/00446 and statutory funding of Faculty of Veterinary Medicine and Animal Science (506.558.09.00 Maria Nowak – grant for young scientists)

SESSION II

NEUROPHYSIOLOGY

Friday (September 25th, 14.30 – 18.10) Room A

Chairman: Associate Professor Tomasz Kowalczyk
(Department of Neurobiology, Faculty of Biology and Environmental Protection, University of Lodz, Lodz, Poland)

Vice-Chairman: Associate Professor Tomasz Blasiak
(Department of Neurophysiology & Chronobiology, Institute of Zoology and Biomedical Research, Jagiellonian University, Krakow, Poland)

DETAILED SESSION II SCHEDULE

Opening lecture (Friday, September 25th, 14.30 – 15.00)

S2.L1 EFFECTS OF BIASED OPIOID RECEPTORS AGONISTS ON OXYCODONE SEEKING AND CRAVING. **W. Solecki[1], A. Ciejka[2], M. Chodlewska[1], J. Duran[1], A. Kaczmarska[1], D. Krzysztofik[2], A. Nalepa[1], J .Furso[2], R. Przewlocki[2]** ([1]Department of Neurobiology and Neuropsychology Institute of Applied Psychology, Faculty of Management and Social Communication, Jagiellonian University, Krakow, Poland; [2]Maj Institute of Pharmacology Polish Academy of Science, Krakow, Poland).

Oral presentations (Friday, September 25th, 15.00 – 17.20)

S2.L2 *15.00 – 15.20* LATERALIZED SENSORY INPUT MODULATES NIGROSTRIATAL DOPAMINERGIC SIGNALLING AND ORIENTING BEHAVIOUR. **M. Marzec[1,2], K. Nowalinska[1], D. Moskal[1], A. Stankiewicz[1], U. Haidukou[1], T. Blasiak[1]** ([1]Department of Neurophysiology and Chronobiology, Institute of Zoology and Biomedical Research, Jagiellonian University, Krakow, Poland; [2]Doctoral School of Exact and Natural Sciences, Jagiellonian University, Krakow, Poland).

S2.L3 *15.20 – 15.40* A NOVEL RODENT MODEL OF MAJOR DEPRESSIVE DISORDER: LINKING HYPOTHELAMIC-PITUITARY-ADRENAL AXIS ABRUPTIONS TO FINDINGS FROM BOTH ELECTROENCEPHALOGRAPHIC AND BEHAVIORAL ANALYSES. **K. Pszczolkowska[1,2], B. Caban[1], R. Bocian[1], T. Kowalczyk[1]** ([1]Department of Neurobiology, Faculty of Biology and Environmental Protection, University of Lodz, Lodz, Poland; [2]University of Lodz Doctoral School of Exact and Natural Sciences, Lodz, Poland).

15.40 – 16.00 Coffee Break

S2.L4 *16.00 – 16.20* TITLE OF PRESENTATION: CHRONIC MILD STRESS SELECTIVELY DISRUPTS THE NUCLEAR FACTOR ERYTHROID 2-RELATED FACTOR 2-KOTHO AXIS IN THE RAT HIPPOCAMPUS BUT NOT FRONTAL CORTEX. **K. Dyndal[1], P. Panczyszyn-Trzewik[2], M. Sowa-Kucma[2,3]** ([1]Student's Science Club of Physiology "NEURON", Faculty of Medicine, Collegium Medicum, University of Rzeszow, Rzeszow, Poland; [2]Department of Human Physiology, Faculty of Medicine, Collegium Medicum, University of Rzeszow, Rzeszow, Poland; [3]Centre for Innovative Research in Medical and Natural Sciences, Collegium Medicum, University of Rzeszow, Rzeszow, Poland).

S2.L5 *16.20 – 16.40* LIVER-EXPRESSED ANTIMICROBIAL PEPTIDE 2 (LEAP2) AS A MODULATOR OF INFLAMMATION INDUCED IN MOUSE MICROGLIAL CELLS (SIM-A9). **A. Gorka, P. Juzwik, M. Nowak , O. Sosinski, N. Leciejewska, E. Grzeda, E. Pruszynska-Oszmalek** (University of Life Sciences, Poznan, Poland).

S2.L6 *16.40 – 17.00* THE EFFECT OF MYXOXANTHOPHYLLS ON THE CONDITION OF RETINAL GANGLION CELLS IN A RAT MODEL OF FRUCTOSE INDUCED METABOLIC SYNDROME. **Z. Kalinska[1], A. Babczynska[2], P. Zbik[3], P. Malec[3], M. Zych[4], I. Kaczmarczyk-Zebrowska[4], M. Pietrucha-Dutczak[1] (**[1]Chair and Department of Physiology, Medical University of Silesia, Katowice, Poland; [2]Institute of Biology, Biotechnology and Environmental Protection, Faculty of Natural Sciences, University of Silesia in Katowice, Katowice, Poland; [3]Department of Plant Physiology and Biochemistry, Faculty of Biochemistry, Biophysics and Biotechnology, Jagiellonian University, Krakow, Poland; [4]Department of Pharmacognosy and Phytochemistry, Faculty of Pharmaceutical Sciences in Sosnowiec, Medical University of Silesia, Katowice, Sosnowiec, Poland).

Poster Session (Friday, September 25th, 17.20 – 18.10)

S2.P1 RECENT ADVANCES IN UNDERSTANDING STRIATAL NEURONAL CIRCUITS AND THEIR ROLE IN MOTOR CONTROL AND PLASTICITY. **I. Asliozau (**Interdisciplinary Student Scientific Circle of Geriatrics and Internal Diseases, The Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Torun, Poland).

S2.P2 CHANGES IN THE PROFILE OF PRECONTACT ULTRASONIC VOCALIZATIONS (PVS) RELATED TO DIFFERENT SEXUAL CONTEXTS. **K. Gaweda, L. Ulloa-Bakowska, W. Bogacki-Rychlik, M. Bialy** (Department of Physiology and Pathophysiology, Center for Preclinical Research, Medical University of Warsaw, Warsaw, Poland).

S2.P3 POTENTIAL OF ISCHEMIC REACTION MODULATION *VIA* EMPAGLIFLOZIN. **O. Kocikowska[1,2], M. Rak[2], M. Koselski[2], M. Wierzchalek[3], M. Olejnik[3], B. Durda[2], N. Chojnicka[2], A. Huptas[2], K. Misterek[2], H. Dojka[2], W. Florek[2], P. Mosionek[2], C. Wawrzynosek[2], S. Student[1], M. Burek[4], H. Jêdrzejowska-Szypulka[2], D. Gendosz De Carrillo[2]** ([1]Biotechnology Centre, Silesian University of Technology, Gliwice, Poland; [2]Department of Physiology, Faculty of Medical Sciences, Katowice, Poland; [3]Students Scientific Club; [4]Department Anesthesiology, Critical Care, Emergency Medicine and Pain Management, Center of Surgical Medicine, Wurzburg, Germany).

S2.P4 MicroRNA-MEDIATED REGULATION OF NEUROVASCULAR UNIT ENDOTHELIAL RESPONSES TO ISCHEMIA. **M. Rak[1], O. Kocikowska[1,2], H. Jedrzejowska- Szypulka[1], D. Gendosz De Carrillo[1,3]** ([1]Department of Physiology, Faculty of Medical Sciences, Medical University of Silesia, Katowice, Poland; [2]Department of Engineering and Biology Systems, Faculty of Automatic Control, Electronics and Computer Science, Silesian University of Technology, Gliwice, Poland; [3]Department of Histology and Cell Pathology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Zabrze, Poland).

S2.P5 METABOLOMIC PROFILES OF RAT STRIATUM FOLLOWING LONG-TERM DEXAMETHASONE TREATMENT AND VITAMIN D3 SUPPLEMENTATION. **D. Korewo-Labelle[1], A. Bogucka[1], M. Karnia[2], D. Myslinska[2], M. Zabielska-Kaczorowska[1], K. Urbanowicz[3], J.J. Kaczor[2]** ([1]Department of Physiology, Medical University of Gdansk, Gdansk, Poland; [2]Department of Animal and Human Physiology, University of Gdansk, Gdansk, Poland; [3]Department of Biochemistry, Medical University of Gdansk, Gdansk, Poland).

S2.L1

EFFECTS OF BIASED OPIOID RECEPTORS AGONISTS ON OXYCODONE SEEKING AND CRAVING

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Opioid use disorder (OUD) is one of the most important crises of contemporary societies with limited effective pharmacological treatments. Opioid use leads to the development of OUD symptoms, including drug craving – a hallmark of OUD. Opioid craving has been associated with the activity of the mesocorticolimbic dopamine (DA) system. Recently, several biased opioid receptor agonists have been demonstrated to modulate opioid-induced behaviors, thus raising potential interest as a new candidates for OUD treatment. We aimed to evaluate effect of selected biased opioid receptors agonists treatment on mesocorticolimbic DA activity as well as oxycodone seeking and craving in rats. To measure DA release, we used fast scan cyclic voltammetry as well as *in vivo* fiber photometry in male Sprague-Dawley rats. To evaluate effects of biased opioid receptor agonists on oxycodone seeking we used oxycodone self-administration as well as several tests of oxycodone seeking and craving. Our results demonstrate that selected biased opioid receptor agonists effectively attenuates phasic DA release in the forebrain as well as decreases oxycodone seeking and craving. Such effects were not due to its potential sedative or dysphoric properties. We propose that administration of selected biased opioid receptor agonists leads to blunted dopaminergic responses to oxycodone conditional stimuli thus decreasing their incentive motivational effect on oxycodone seeking behavior.

National Science Centre Research grant no: UMO-2020/39/B/NZ7/03537

S2.L2

LATERALIZED SENSORY INPUT MODULATES NIGROSTRIATAL DOPAMINERGIC SIGNALLING AND ORIENTING BEHAVIOUR

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Midbrain dopaminergic (DA) neurons in the substantia nigra pars compacta (SNc) contribute to action selection through dopamine release in the dorsal striatum. Transient hemispheric asymmetries in nigrostriatal dopaminergic signalling have been proposed to bias directional behaviours, but the sensory pathways capable of generating such asymmetries remain poorly understood. Here, we examined whether spatially restricted sensory signals conveyed by the lateral superior colliculus (lSC) can modulate SNc dopaminergic activity and dopamine release in the dorsal striatum, thereby influencing orienting behaviour. Using viral tract-tracing approach in rats, we characterized the anatomical organization of the lSC › SNc › dorsal striatum pathway. To assess neuronal responses, in vivo extracellular recordings of SNc DA neurons were performed during pharmacological disinhibition of the lSC combined with unilateral visual stimulation. Dopamine dynamics in the dorsal striatum were monitored using fibre photometry, and the behavioural significance of the circuit was evaluated using pathway-specific optogenetic stimulation in a custom-designed orienting paradigm. Anatomical analyses revealed a topographically organized and lateralized pathway linking the lSC with SNc dopaminergic neurons projecting preferentially to the ipsilateral dorsal striatum. Unilateral visual stimulation evoked predominantly excitatory responses in SNc DA neurons. Althought firing responses did not display a robust hemispheric preference at single-neuron level, dopamine release within the dorsal striatum was significantly enhanced in the hemisphere contralateral to the stimulated eye. Moreover, selective optogenetic activation of the lSC › SNc pathway induced directional orienting biases in behaving animals. These findings indicate that asymmetric visual input can modulate nigrostriatal dopaminergic signalling through a defined lSC › SNc › dorsal striatum pathway. This circuit may provide a neural substrate through which spatial sensory signals bias orienting responses and action selection.

S2.L3

A NOVEL RODENT MODEL OF MAJOR DEPRESSIVE DISORDER: LINKING HYPOTHELAMIC-PITUITARY-ADRENAL AXIS ABRUPTIONS TO FINDINGS FROM BOTH ELECTROENCEPHALOGRAPHIC AND BEHAVIORAL ANALYSES

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Stress is an inevitable component of daily life, however, prolonged, or even single exposure to severe stress can induce significant neuroendocrine and behavioral dysfunctions. In vulnerable individuals, such alterations may contribute to the development of stress-related disorders, including post-traumatic stress disorder or major depressive disorder (MDD). The present study aimed to develop a novel rodent model of hypothalamic–pituitary–adrenal (HPA) axis dysregulation observed in MDD patients. This was achieved through pharmacological induction of chronic and combined chronic–acute stress, with simultaneous monitoring of behavioral activity and recording of electroencephalographic (EEG) activity from the posterior hypothalamic area (PHa) in freely moving rats undergoing open field (OF) test. Sixteen male Wistar rats were divided into equal experimental and control groups. The experimental group received continuous corticosterone (CORT) administration for 21 days and both groups underwent repeated OF testing. On the final day, an additional high dose of CORT was administered subcutaneously to induce an acute stress response. Behavioral activity and PHa EEG signals were recorded continuously during testing. Blood samples were collected on days 1, 10, 20, and 21 to assess plasma corticosterone levels. Temporal changes in corticosterone concentrations suggest adaptive activation of metabolic pathways regulating hormone levels. Behavioral alterations, including locomotor activity, time spent in the arena center, and changes in PHa EEG activity are discussed. The combined model of monitoring hormonal, behavioral and bioelectrical activities in stressful conditions could be a possible tool for investigating the neurobiological mechanisms and pharmacological targets of MDD.

S2.L4

TITLE OF PRESENTATION: CHRONIC MILD STRESS SELECTIVELY DISRUPTS THE NUCLEAR FACTOR ERYTHROID 2-RELATED FACTOR 2-KOTHO AXIS IN THE RAT HIPPOCAMPUS BUT NOT FRONTAL CORTEX

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Depressive disorder (DD) is increasingly recognized as a multifactorial condition that, beyond the neurotransmitter alterations, is also influenced by neuroinflammation and oxidative stress. Emerging evidence suggests that nuclear factor erythroid 2-related factor 2 (Nrf2) and its upstream regulator Klotho (i.e. key components of cellular antioxidant defense system), may play a critical role in the pathophysiology of DD by regulating redox homeostasis and neuronal vulnerability to stress. The present study aimed to evaluate the effects of chronic mild stress (CMS, a weel-validated animal model of depression), on Nrf2 and Klotho protein levels in the hippocampus (Hp) and frontal cortex (FCx) of rats. Brain tissue samples were collected from animals exposed to CMS for 7 weeks and from non-stressed controls. Protein concentrations were quantified using commercially available ELISA kits. Statistical analysis was performed using Graph Pad PRISM v.10.0 and an unpaired two-tailed t-test with Welch's correction. CMS exposure resulted in a significant reduction in both Nrf2 and Klotho levels in the hippocampus ($p=0.0029$ and $p=0.0087$, respectively), whereas no significant changes were observed in the frontal cortex ($p > 0.05$). These findings indicate that chronic stress selectively impairs the Nrf2-Klotho antioxidant axis in the hippocampus, highlighting its heightened vulnerability to stress-related oxidative dysregulation. This region-specific effect supports the critical role of the hippocampus in the neurobiological mechanisms underlying depression.

The study was partially supported by grants UMO-2016/21/B/NZ7/01623 and UMO-2016/23/N/NZ4/01337.

S2.L5

LIVER-EXPRESSED ANTIMICROBIAL PEPTIDE 2 (LEAP2) AS A MODULATOR OF INFLAMMATION INDUCED IN MOUSE MICROGLIAL CELLS (SIM-A9)

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Microglia constitute the primary population of immunocompetent cells in the central nervous system (CNS) and play a crucial role in maintaining homeostasis and regulating inflammatory responses. However, excessive microglial activation may lead to chronic inflammation, which is considered one of the key mechanisms underlying neurodegenerative diseases. In recent years, increasing attention has been paid to antimicrobial peptides (AMPs), which exhibit not only antimicrobial properties but also immunomodulatory activity. One such peptide is Liver-Expressed Antimicrobial Peptide 2 (LEAP2), which, in addition to its antibacterial activity, functions as an antagonist of the ghrelin receptor (GHSR). Previous studies suggest that LEAP2 may participate in the regulation of inflammatory processes; however, its effect on microglial activity remains poorly understood. The aim of this study was to evaluate the effect of LEAP2 on the modulation of inflammatory responses in mouse microglial cells (SIM-A9 cells line) under both physiological conditions and lipopolysaccharide (LPS) induced inflammatory conditions. The study focused on the analysis of pro-inflammatory gene expression, including TNFα, IL-1β, and IL-6, using quantitative PCR (qPCR), with GAPDH serving as the reference gene. In addition, the effect of LEAP2 on cell viability and proliferation of SIM-A9 cells under physiological and inflammatory conditions was evaluated. The obtained results indicate that LEAP2 affects the expression of the investigated pro-inflammatory cytokines in SIM-A9 cells under both physiological and LPS-induced inflammatory conditions. Furthermore, LEAP2 modulated cell viability and proliferation, suggesting its potential role in regulating microglial activity. These findings support the hypothesis that LEAP2 may act as an immunomodulatory factor in neuroinflammation. However, further studies are required to better understand the mechanisms underlying the action of this peptide in microglial cells.

S2.L6

THE EFFECT OF MYXOXANTHOPHYLLS ON THE CONDITION OF RETINAL GANGLION CELLS IN A RAT MODEL OF FRUCTOSE INDUCED METABOLIC SYNDROME

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Myxoxanthophylls are polar, glycosylated, and monocyclic carotenoids present in cyanobacteria. These carotenoids are gaining attention due to their remarkable antioxidant properties, attributed to their high number of conjugated double bonds. Such unique properties, similar to that present in lutein, make them potential biotechnology targets. In our research, we used a highly purified fraction of myxoxanthophylls obtained from crude biomass of *Arthrospira platensis* according to an original method developed at the Department of Plant Physiology and Biochemistry, Jagiellonian University in Krakow. The aim of the study was to examine whether myxoxanthophylls affect the survival and condition of retinal ganglion cells in a rat model of metabolic syndrome. The research was carried out on 60 male Wistar rats. Animals were assigned to six groups: C - control group; MS – metabolic syndrome group; S – spirulina (*Arthrospira* biomass*)* group, dose 200 mg/kg/day; M2 – myxoxanthophyll group, dose 2 mg/kg/day; M20 - myxoxanthophyll group, dose 20 mg/kg/day; DAPA – dapagliflozin group. Metabolic syndrome was induced by 10% fructose in drinking water for 12 weeks, tested substances were introduced 4 weeks before the end of the experiment. After euthanasia retinas were collected and processed for western blot analysis and immunohistochemistry. We observed that myxoxanthophylls, *Arthrospira* biomass and dapagliflozin decreased the expression of pro-inflammatory cytokines. The most beneficial effect was exerted by myxoxanthophylls in low dose and *Arthrospira* biomass. In conclusion, low-dose myxoxanthophylls may represent a new therapeutic option to protect retinal ganglion cells from damage in patients with metabolic syndrome.

S2.P1

RECENT ADVANCES IN UNDERSTANDING STRIATAL NEURONAL CIRCUITS AND THEIR ROLE IN MOTOR CONTROL AND PLASTICITY

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The striatum, the main input nucleus of the basal ganglia, plays a central role in motor control, action selection, habit formation, and motor learning. Recent research has significantly advanced our understanding of the complex neuronal circuits within the striatum, including the heterogeneity of spiny projection neurons (SPNs), the functional diversity of GABA-ergic interneurons, and the dynamic role of dopaminergic modulation in synaptic plasticity. This review aims to summarize the most important recent advances (2018–2025) in the field of striatal neuronal circuits, with particular emphasis on their implications for motor control and plasticity under physiological and pathological conditions. A literature review was conducted using PubMed, Google Scholar and Scopus. Original articles and reviews published between 2018 and 2025 were selected and analyzed, focusing on cortico-striatal plasticity, striatal microcircuitry, dopamine-dependent modulation, and the role of specific neuronal subpopulations. Recent studies have revealed high levels of cellular and functional heterogeneity within striatal circuits, including distinct roles of direct and indirect pathway SPNs as well as matrix and striosomal compartments. Key advances highlight the critical contribution of GABA-ergic interneurons and dopamine modulation to synaptic plasticity and motor learning. Furthermore, optogenetic, electrophysiological, and computational modeling studies emphasize the importance of precise circuit balance and timing for proper motor control in both health and disease states such as Parkinson's disease and dystonia. The last decade has brought substantial progress in deciphering the complexity of striatal neuronal circuits. These advances deepen our understanding of motor control and open new therapeutic perspectives for movement disorders. Further research integrating molecular, cellular, and behavioral levels is essential for translating these findings into clinical applications.

S2.P2

CHANGES IN THE PROFILE OF PRECONTACT ULTRASONIC VOCALIZATIONS (PVS) RELATED TO DIFFERENT SEXUAL CONTEXTS

K. GAWEDA, L. ULLOA-BAKOWSKA, W. BOGACKI-RYCHLIK, M. BIALY

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Our previous studies have shown that precontact ultrasonic vocalizations (PVs) emitted by male rats preceding copulatory behavior depend on sexual experience, the rewarding properties of sexual contact, and the level of sexual motivation. The present study examined the variability of PVs profiles related to different socio-sexual contexts. We analyzed PVs emission in two experimental set-ups: (1) before the copulation (COP), and at the end of the first postejaculatory period (copulation to one ejaculation); (2) before non-contact erection sessions (NCE), assessing sexual arousal in the absence of direct physical contact between males and females. COP PVs profiles of sexually experienced males were characterized by a high number of ultrasonic calls and the absence of low-frequency (22-kHz) ultrasonic vocalizations. In contrast, NCE PVs profiles contain fewer ultrasounds and include different low-frequency calls, i.e., mainly frequency-shifted vocalizations transitioning from ~45 kHz to 19–32 kHz. Surprisingly, pharmacological reduction of serotonin levels *via* a 5-HT1A autoreceptor agonist did not affect the occurrence of low-frequency calls during NCE-related PVs. These findings indicate that both the quantity and acoustic profile of PVs depend on sexual context, likely reflecting differences in sexual motivation and general arousal across types of sexual interactions.

S2.P3

POTENTIAL OF ISCHEMIC REACTION MODULATION *VIA* EMPAGLIFLOZIN

O. KOCIKOWSKA[1,2], M. RAK[2], M. KOSELSKI[2], M. WIERZCHALEK[3], M. OLEJNIK[3], B. DURDA[2], N. CHOJNICKA[2], A. HUPTAS[2], K. MISTEREK[2], H. DOJKA[2], W. FLOREK[2], P. MOSIONEK[2], C. WAWRZYNOSEK[2], S. STUDENT[1], M. BUREK[4], H. JEDRZEJOWSKA-SZYPULKA[2], D. GENDOSZ DE CARRILLO[2]

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Ischemic stroke (IS) is one of the leading causes of death and permanent disability worldwide. As the population ages, the incidence of the condition is rising, as age is the most significant, non-modifiable risk factor for IS. Current treatment relies on the earliest possible initiation of pharmacological and/or surgical therapy to restore blood flow in the area affected by ischemia. Treatment is not ideal, and over half of patients experience permanent complications and disability, which stems from the lack of effective neuroprotective therapies to support reperfusion therapy. Flozins are a group of drugs originally used in the treatment of diabetes by inhibiting the sodium-glucose cotransporter type 2 (SGLT2). In addition to their antidiabetic effects, these drugs–thanks to the phenomenon of drug repositioning–have found application in the treatment of cardiovascular diseases. A growing body of research suggests their potential in the treatment of central nervous system disorders, including complications of ischemic stroke. Our research focused on two main areas which we contrasted together. First, open access transcriptomics data analysis from GEO and ExpressArray data bases was analysed regarding flozin effect on genes involved in response to ischemia. For analysis we selected genes assigned to Gene Onthology ischemic response pathway (GO:0002931) and datasets involving empagliflozin (GSE118337, GSE191046, GSE206986, GSE243752, GSE285966, GSE155867, GSE255933). Second, we analysed samples from our *in vitro* ischemic model regarding chosen molecular targets. The obtained results showed that the molecular response to treatment is highly tissue- and model-dependent. No exact pattern of changes of genes involved in ischemic response pathway was observed. The obtained results provide a basis for further research into the empagliflozin administration in an ischemic stroke model, with a view to verifying the modulation of observed changes and their functional implications. It is crucial to determine whether the modulation of hypoxia induced pathway leads to allevating of damage and an improvement in neurological prognosis.

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S2.P4

MicroRNA- MEDIATED REGULATION OF NEUROVASCULAR UNIT ENDOTHELIAL RESPONSES TO ISCHEMIA

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The neurovascular unit (NVU) is essential for maintaining brain homeostasis and blood-brain barrier (BBB) integrity, while its disruption is a key feature of ischemic stroke (IS) and contributes to disease progression. MicroRNAs (miRNAs), as post-transcriptional regulators of gene expression, have emerged as important modulators of cellular responses under ischemia. This study aimed to identify miRNAs involved in endothelial responses during ischemia. Human brain endothelial cells were subjected to oxygen-glucose deprivation/reoxygenation (OGD/R) as an in vitro IS model. Based on experimental design and literature data, hyperacute (4 h) and acute (4 h + 20 h) phases were selected for next-generation sequencing. The analysis identified 3 significantly dysregulated miRNAs in the hyperacute phase and 12 in the acute phase. Among these, miR-193b and miR-365a were selected for further analysis due to their potential involvement in endothelial signaling and vascular stress responses. These findings indicate that miR-193b and miR-365a may play a role in NVU dysfunction under ischemic conditions and represent promising candidates for further investigation as therapeutic targets in ischemic stroke.

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S2.P5

METABOLOMIC PROFILES OF RAT STRIATUM FOLLOWING LONG-TERM DEXAMETHASONE TREATMENT AND VITAMIN D3 SUPPLEMENTATION

D. KOREWO-LABELLE[1], A. BOGUCKA[1], M. KARNIA[2], D. MYSLINSKA[2], M. ZABIELSKA-KACZOROWSKA[1], K. URBANOWICZ[3], J. KACZOR[2]

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Dexamethasone (Dex) is a steroid drug commonly used in medicine to treat inflammatory and autoimmune diseases. Prolonged Dex exposure significantly affects brain functions. The striatum, which regulates motor functions, cognitive processes, and emotional responses, is particularly sensitive to glucocorticosteroid action. Chronic Dex exposure may influence metabolic disorders and neurochemical changes within this structure. Increasing evidence suggests the neuromodulatory properties of vitamin D3 (VitD3). The study aimed to evaluate the effect of VitD3 supplementation during long-term Dex exposure on metabolic changes in the striatum of rats. The study lasted 28 days and was conducted on 17 male Wistar rats, randomly divided into three groups. These included two groups treated with Dex at a dose of 2 mg/kg/day with the addition of vegetable oil (Dex; n=5) or vitamin D3 600 IU/kg/day (Dex VD; n=6), respectively, and a control group treated with saline injection (Con; n=6). The content of specific receptors was measured by Western blot. Metabolic changes were assessed using targeted and untargeted liquid chromatography-mass spectrometry (LC-MS) metabolomics. We observed significant changes in glucocorticosteroid receptor content, glutamic acid/GABA ratio, redox-associated metabolites, and neurochemical dysregulation in both the Dex-treated group. However, VitD3 supplementation significantly attenuated these changes.

Research was financially supported by the project „Mlody Tworca Nauki“ Initiative of Excellence – Research University, Medical University of Gdansk, Poland.

SESSION III

EXERCISE PHYSIOLOGY

Friday (September 25th, 14.30 – 18.10) Room B

Chairman: Professor Jerzy A. Zoladz
(Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland)

Vice-Chairman: Associate Professor Hanna Drzymala-Celichowska
(Department of Biochemistry, Poznan University of Physical Education, Poznan, Poland)

DETAILED SESSION III SCHEDULE

Opening lecture Friday (September 25th 14.30 – 15.00)

S3.L1 SKELETAL MUSCLE OXIDATIVE METABOLISM IN MICROGRAVITY/DISUSE: BOTTLENECKS, ADAPTATIONS, INFERENCES ON HEALTH. **B. Grassi[1], G. Baldassarre[1], L. Gobbo[1], S. Perilli[2], F. Ferranti[2], L. Zuccarelli[1]** ([1]Exercise Physiology Laboratory, Department of Medicine, University of Udine, Udine, Italy; [2]Italian Space Agency, Rome, Italy).

Oral presentations Friday (September 25th 15.00 – 17.20)

S3.L2 *15.00 – 15.20* TIME REQUIRED FOR THE RESTITUTION OF EXTRA INITIAL FORCE IN FAST MOTOR UNITS OF THE RAT MEDIAL GASTROCNEMIUS MUSCLE. **H. Drzymala-Celichowska[1,2], J. Majerczak[3], K. Krysciak[1], J. Celichowski[1], J.A. Zoladz[3]** ([1]Department of Neurobiology, Faculty of Health Sciences, Poznan University of Physical Education, Poznan, Poland; [2]Department of Biochemistry, Faculty of Health Sciences, Poznan University of Physical Education, Poznan, Poland; [3]Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland).

S3.L3. *15.20 – 15.40* MUSCLE NITRITE CORRELATES WITH HIGH ENERGY PHOSPHATES IN RAT LOCOMOTORY MUSCLES. **J. Majerczak[1], L. Widacha[1], M. Grandys[1], R.T. Smolenski[2], J.A. Zoladz[1]** ([1]Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland; [2]Department of Biochemistry, Faculty of Medicine, Medical University of Gdansk, Gdansk, Poland).

15.40 – 16.00 Coffee break

S3.L4 *16.00 – 16.20* PREDICTING INDIVIDUAL READINESS FOR HYPOXIC TRAINING IN ELITE ATHLETES: INTEGRATING VENTILATORY RESPONSE AND FLOW-MEDIATED SKIN FLUORESCENCE (FMSF). **A. Stanek[1], P. Cieslak[2], T. Sawczyn[3]** ([1]Hypoxico Polska, Poland; [2]Centrum Formy CFO, Dabrowa Gornicza, Poland; [3]Department of Physiology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland).

S4.L5 *16.20 – 16.40* MECHANISMS OF THE TRAINING-INDUCED RESTING BRADYCARDIA IN HUMANS – THE OVERLOOKED ROLE OF ANDROGEN STATUS. **M. Grandys[1], J. Majerczak[1], M. Wozniak[1,2], J.A. Zoladz[1]** ([1]Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland; [2]Department of Angiology, Jagiellonian University Medical College, Krakow, Poland).

S3.L6 *16.40 – 17.00* ANALYSIS OF PHASE ANGLE (PhA) AND TOTAL BODY WATER (TBW) IN YOUNG ADULTS: A BIOELECTRICAL IMPEDANCE PILOT STUDY. **A. Panasiewicz[1], A. Blazowska[1], E. Kupczak[1], K. Michalczyk[2,3], M. Stojko[2], J. Poloczek[2], D. Gach[4], B. Bogielski[2], B. Skrzep-Poloczek[2,3]** ([1]Student Scientific Association of Physiology, Branch in Bielsko-Biala, Medical University of Silesia, Katowice, Poland; [2]Physiology Facility, Branch in Bielsko-Biala, Medical University of Silesia, Katowice, Poland; [3]Department of Physiology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland; [4]Scientific Research Facility, Branch in Bielsko-Biala, Medical University of Silesia, Katowice, Poland).

Poster Session Friday (September 25th 17.20 – 18.10)

S3.P1 SEX DIFFERENCES IN THE EXPERIENCE OF "RUNNER'S HIGH": ANALYSIS OF SUBJECTIVE SYMPTOMS IN THE CONTEXT OF ENDOCANNABINOID MECHANISMS. **K. Kufel, D. Czyz[1], J. Kulinska, A. Dittmann, O. Rak, M.J. Braczkowski** (University of Opole, Faculty of Medicine, Institute of Medical Sciences, Department of Physiology, Student Scientific Society of Physiology).

S3.P2 PHYSICAL ACTIVITY AND AIR POLLUTION: EXPLORING WAYS TO MINIMIZE THE HARMFUL EFFECTS OF AIR POLLUTION ON ATHLETES' HEALTH. **Z. Nieckarz[1], J.A. Zoladz[2]** ([1]Marian Smoluchowski Institute of Physics, Jagiellonian University, Krakow, Poland; [2]Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland).

S3.P3 THE BOOST OF FORCE IN UNFUSED TETANIC CONTRACTIONS AT VARIABLE INTERPULSE INTERVALS IN FAST MOTOR UNITS OF THE RAT MEDIAL GASTROCNEMIUS. **K. Krysciak[1], J. Celichowski[1], R. Raikova[2], K. Podgorski[1], H. Drzymala-Celichowska[1,3]** ([1]Department of Neurobiology, Poznan University of Physical Education, Poznan, Poland; [2]Institute of Biophysics and Biomedical Engineering, Bulgarian Academy of Sciences, Sofia, Bulgaria; [3]Department of Physiology and Biochemistry, Poznan University of Physical Education, Poznan, Poland).

S3.L1

SKELETAL MUSCLE OXIDATIVE METABOLISM IN MICROGRAVITY/DISUSE: BOTTLENECKS, ADAPTATIONS, INFERENCES ON HEALTH

B. GRASSI[1], G. BALDASSARRE[1], L. GOBBO[1], S. PERILLI[2], F. FERRANTI[2], L. ZUCCARELLI[1]

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Skeletal muscle oxidative metabolism and mitochondrial function represent "the last steps" in the pathway for O_2, from ambient air to oxidative phosphorylation. This metabolism is often neglected in studies dealing with the effects of microgravity/disuse on skeletal muscle, which mainly take into consideration muscle mass and muscle force. This is unfortunate, considering that all activities lasting longer than 1-2 minutes substantially rely on oxidative metabolism for ATP turnover. Apart from its direct effects on exercise tolerance, a decreased "cardiorespiratory fitness" determined by microgravity/disuse, as identified by the a decreased maximal O_2 uptake (VO_{2max}), is associated with profound negative consequences on the general health status of the subjects, such as decreased insulin sensitivity, "pro-inflammatory" condition, impaired endothelial function, mitochondrial dysfunction, altered function of the neuromuscular junction, altered redox status, increased oxidative stress. A VO_{2max} decrease during simulated microgravity/disuse has been described in "bed rest" studies, ranging from hours to a few months. These studies have allowed to identify "bottlenecks" along the O_2 transport and O_2 utilization pathway, mainly related to impaired cardiac function, reduced blood volume, impaired microvascular/endothelial function (attenuated hyperemic response in the common femoral artery during passive leg movements). These impairments would occur relatively early during exposure, whereas mitochondrial respiration would be significantly affected during longer exposures, after being relatively "spared" during shorter exposures, as demonstrated by our group both *in vivo* (by near-infrared spectroscopy) and *ex vivo* (mitochondrial respiration in isolated permeabilized muscle fibers). According to recent work from our group, short periods of simulated microgravity/disuse could have an interesting effect on skeletal muscle and whole body oxidative metabolism in resting conditions, that is a substantial decrease in resting muscle VO_2 and whole-body resting energy expenditure (REE). Even by being far less pronounced compared to the typical reduction of REE observed in obligate hibernating animals, the observed inactivity-related decrease in resting muscle VO_2 and REE, possibly aimed at preventing ATP accumulation or excessive ROS production, could mitigate numerous biological and logistic challenges of prolonged spaceflights, by lowering rates of crewmember consumable use (food, water, O_2) and CO_2 production.

Agreement N. 2025-10-HB.0 "BIOMEX", Italian Space Agency (Agenzia Spaziale Italiana, ASI).

S3.L2

TIME REQUIRED FOR THE RESTITUTION OF EXTRA INITIAL FORCE IN FAST MOTOR UNITS OF THE RAT MEDIAL GASTROCNEMIUS MUSCLE

H. DRZYMALA-CELICHOWSKA[1,2], J. MAJERCZAK[3], K. KRYSCIAK[1], J. CELICHOWSKI[1], J.A. ZOLADZ[3]

[1]Department of Neurobiology, Faculty of Health Sciences, Poznan University of Physical Education, Poznan, Poland; [2]Department of Biochemistry, Faculty of Health Sciences, Poznan University of Physical Education, Poznan, Poland; [3]Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland; drzymala@awf.poznan.pl

During tetanic contractions of fast motor units (MUs), an early increase in force (boost) is followed by a slight decline to a plateau (sag). The boost is present at the onset of activity but disappears during repeated contractions and can reappear after a period of rest. However, the background of the boost, especially the minimum time required to restore this effect, is unknown. In this study, single MUs were isolated and stimulated electrically. Three 504-ms tetanic contractions at 35 Hz were delivered once per second, and the series was repeated at intervals ranging from 90 to 2 s. As the intervals shortened, the boost progressively decreased, as reflected by a reduction in sag amplitude. In fast fatigue-resistant MUs, sag amplitude decreased by 25% and 50% at average intervals of 29.80 s and 16.84 s, respectively. In fast fatigable MUs, the corresponding intervals were 82.29 s and 54.07 s. These findings suggest that the physiological basis for the restitution of initial force (boost) is the kinetics of muscle energy status recovery following initial exercise.

This work was partially supported by project N43/DBS/000314 and created with the use of equipment cofinanced by the qLIFE Priority Research Area under the program "Excellence Initiative–Research University" at Jagiellonian University (06/IDUB/2019/94)

S3.L3

MUSCLE NITRITE CORRELATES WITH HIGH ENERGY PHOSPHATES IN RAT LOCOMOTORY MUSCLES

J. MAJERCZAK[1], L. WIDACHA[1], M. GRANDYS[1], R.T. SMOLENSKI[2], J.A. ZOLADZ[1]

[1]Chair of Exercise Physiology and Muscle Bioenergetics, Faculty of Health Sciences, Jagiellonian University Medical College, Krakow, Poland; [2]Department of Biochemistry, Faculty of Medicine, Medical University of Gdansk, Gdansk, Poland; joanna.majerczak@uj.edu.pl

It is well known that fast-twitch fibers are characterized by a higher content of high-energy phosphates (PCr, ATP) compared to slow muscles[1]. As we have shown in our recent studies[2], fast muscles are also characterized by higher nitrite (NO_2^-) content, which is stable metabolite of nitric oxide (NO). The main aim of the present study was to establish the relationship between high-energy phosphates and nitrite in the rats locomotory muscles. The levels of muscles metabolites were analyzed in the skeletal muscles of varied muscle fiber types (*Sol, MGS, MGF, TA*) using HPLC analysis in twelve adult male Wistar rats.We found a positive correlation between muscle NO_2^- and muscles content of PCr and ATP. These results suggest that at rest, muscle NO_2^- plays a role in maintenance of high-energy phosphates content in skeletal muscles.

This work was supported by the funds from National Science Centre 2017/27/B/NZ7/01976 awarded to Joanna Majerczak. The experiments were partially performed created with the use of equipment co-financed by the qLIFE Priority Research Area under the program “Excellence Initiative-Research University” at Jagiellonian University (number: 06/IDUB/2019/94).

S3.L4

PREDICTING INDIVIDUAL READINESS FOR HYPOXIC TRAINING IN ELITE ATHLETES: INTEGRATING VENTILATORY RESPONSE AND FLOW-MEDIATED SKIN FLUORESCENCE (FMSF)

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Athletes differ substantially in their physiological responses to hypoxia, making individualized prescription of hypoxic training difficult. This pilot study evaluated whether ventilatory behavior during progressive hypoxia combined with microvascular responses assessed by Flow-Mediated Skin Fluorescence (FMSF) can identify athletes better prepared for hypoxic training. Competitive athletes completed a two-day protocol. On Day A, ventilatory threshold (VT1) was determined during an incremental exercise test. Subsequently, participants underwent a 60-min intermittent hypoxia training session (5-min hypoxia, SpO_2 ~ 85%, alternating with 5-min normoxia). FMSF measurements were performed before and after hypoxia to assess changes in endothelial (ENDO), neurogenic (NEURO), myogenic (MYO), Flowmotion (FM), and Normoxia Oscillatory Index (NOI%) parameters. On Day B, athletes exercised at VT1 intensity while inspired oxygen concentration was progressively reduced to determine the Hypoxic Ventilatory Inflection and Hypoxia Tolerance Index (HTI). Marked inter-individual variability in microvascular responses was observed. Following hypoxia, many athletes demonstrated enhanced endothelial activity, suggesting improved vascular responsiveness. Significant associations were found between ventilatory and microcirculatory adaptations. Increased breathing frequency during hypoxia was associated with higher NOI% and lower myogenic contribution, whereas a greater increase in VE/VCO_2 was related to less favorable FM responses. HTI alone was not associated with FMSF-derived adaptations. These findings suggest that readiness for hypoxic training depends more on ventilatory economy and microvascular responsiveness than on hypoxia tolerance alone. The combined use of ventilatory assessment and FMSF may provide a practical, non-invasive tool for individualized hypoxic training prescription in athletes.

S3.L5

MECHANISMS OF THE TRAINING-INDUCED RESTING BRADYCARDIA IN HUMANS – THE OVERLOOKED ROLE OF ANDROGEN STATUS

M. GRANDYS[1], J. MAJERCZAK[1], M. WOZNIAK[1,2], J.A. ZOLADZ[1]

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There is evidence that testosterone may affects cardiac functions, including regulation of the ion channels expression. It seems, however, that in the debate – which has been ongoing for over 100 years – regarding the mechanisms underlying the training-induced resting bradycardia, the role of androgens has been overlooked. Therefore, in this investigation, we aimed to study the relationship between androgen status and resting heart rate (RHR) in a group of 87 healthy men aged 18 to 77 with varying levels of physical activity using multi-linear regression analysis. Venous fasting blood samples were taken to determine androgen status (testosterone, T; sex hormone-binding globulin, SHBG; dehydroepiandrosterone sulfate, DHAE-S; androstenedione, AE; dihydrotestosterone, DHT) and resting heart rate were monitored continuously using a 3-lead ECG (SphygmoCor system) in the supine position in a quiet, semi-darkened, and temperature-controlled room after a 20-minute rest period. It was demonstrated that free androgen index (FAI) and DHT were positively (both *rho*=0.25, *p*=0.02) whereas SHBG concentration negatively correlated with RHR (*rho*=-0.30, *p*=0.005). Moreover, there was a significant negative correlation between amount of physical activity and RHR (*rho*=-0.37, *p*=0.0004). Multivariate analysis showed that when adjusting for age, BMI and the amount of physical activity, individuals with lower FAI had lower resting heart rate (β=0.30, *p*=0.02, aR^2=0.22). We have concluded that a low resting heart rate – particularly common among endurance trained athletes – may be associated with lower androgen status, which sheds new light on the ongoing debate regarding the mechanisms underlying training-induced bradycardia.

The publication was created with the use of equipment co-financed by the qLIFE Priority Research Area under the program “Excellence Initiative-Research University” at Jagiellonian University (number: 06/IDUB/2019/94).

S3.L6

ANALYSIS OF PHASE ANGLE (PHA) AND TOTAL BODY WATER (TBW) YOUNG ADULTS: A BIOELECTRICAL IMPEDANCE PILOT STUDY

A. PANASIEWICZ[1], A. BLAZOWSKA[1], E. KUPCZAK[1], K. MICHALCZYK[2,3], M. STOJKO[2], J. POLOCZEK[2], D. GACH[4], B. BOGIELSKI[2], B. SKRZEP-POLOCZEK[2,3]

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Bioelectrical Impedance Analysis (BIA) measures resistance (R—fluid opposition) and reactance (Xc—cell membrane capacitance). Phase angle (PhA; normal range 5–7°) and total body water (TBW) reflect cellular integrity, nutritional status, and muscle quality, acting as markers of physical fitness rather than body fat predictors. The study aimed to analyze PhA and TBW% in young adults in relation to gender and body composition. The study included 49 medical students or candidates for medical studies (29 women, 20 men), aged 18–29, recruited from medical students of Medical University of Silesia, Branch in Bielsko-Biala or candidates for medical studies attending the Open House event in Branch in Bielsko-Biala in April, 2025. The exclusion criteria were: heart pacemaker, internal heart defibrillator, pregnancy or lactation, using drugs that change body fluids composition, substantial disability preventing accessing Tanita analyzer. Body composition, TBW% and PhA were assessed using Tanita BIA analyzer. Physical activity was assessed based on the International Physical Activity Questionnaire. 42.55% of responders reported high, 23.40% reported low, and 10.64% reported none physical activity. PhA was significantly higher in men than in women (6.52° *vs.* 5.43°). TBW% was significantly higher in men than in women (59% *vs*. 53.91%). PhA correlated strongly and positively with fat-free mass (FFM) and TBW. Higher values of PhA and TBW in men directly result from a higher percentage of muscle mass. BIA effectively evaluates cell membrane integrity in young adults. PhA and TBW% are sensitive, objective markers of metabolic status and tissue quality.

S3.P1

SEX DIFFERENCES IN THE EXPERIENCE OF "RUNNER'S HIGH": ANALYSIS OF SUBJECTIVE SYMPTOMS IN THE CONTEXT OF ENDOCANNABINOID MECHANISMS

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"Runner's high" is a state of euphoria and reduced pain perception occurring after physical exercise, primarily associated with activation of the endocannabinoid system. A key role is played by anandamide (AEA), an endogenous agonist of CB1 receptors, responsible for euphoric, anxiolytic, and analgesic effects. Experimental studies have demonstrated that the post-exercise increase in AEA concentration is significantly increased in women than in men, which may translate into sex differences in the subjective experience of "runner's high." The aim of the study was to analyze the characteristics and duration of the "runner's high" phenomenon among individuals regularly engaged in running, with particular emphasis on sex differences. A cross-sectional survey study was conducted in a group of 56 individuals who regularly practiced running. The frequency of "runner's high," its main symptoms, and its duration after exercise were assessed. Descriptive statistics were used for analysis. "Runner's high" was reported by 93% of participants. Significant sex differences were observed in its characteristics. Women more frequently reported both euphoria and reduced pain perception, whereas in men euphoria was the predominant symptom. Differences were also noted in duration – in women, the effect more often persisted for over 1 hour, while in men it was shorter, most commonly lasting 15–30 minutes. The obtained results indicate sex differences in the subjective experience of "runner's high," including both its qualitative characteristics and duration. The higher prevalence of the analgesic component and the longer duration of the effect in women may be associated with stronger activation of the endocannabinoid system, including a greater post-exercise increase in anandamide (AEA) levels. Due to its euphoric and analgesic effects, AEA may play a key role in the observed differences. Further studies incorporating direct biochemical measurements are required to confirm these associations.

S3.P2

PHYSICAL ACTIVITY AND AIR POLLUTION: EXPLORING WAYS TO MINIMIZE THE HARMFUL EFFECTS OF AIR POLLUTION ON ATHLETES' HEALTH

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It is well documented that fine particulate matters (PM), which are present in large quantities in polluted air, has a harmful effect on various tissues and organs of the human body (Chen *et al.,* 2017). We recently demonstrated that marathon running, by increasing minute ventilation, drastically increases the rate at which PM particles from inhaled air are deposited in runners' airways (Zoladz *et al.,* 2021). In the present poster we will present the latest methods for the precise measurement, monitoring, and analysis of air quality (particulate matter studies). The study will cover a broad spectrum of aerodynamic diameters of particulate matter, ranging from extremely small sizes of 5 nm up to 32 μm. For selected time periods, we will conduct a detailed analysis of temporal-size distribution spectrograms and temporal changes in the mass concentrations of PM1, PM2.5, and PM10. The research results will pertain to air quality at sports and recreational facilities located in Krakow. We will describe the characteristics of the measurement site and its surroundings.

The research for this publication has been supported by the budget of Anthropocene Priority Research Area (Earth System Science Core Facility Flagship Project) under the Strategic Programme Excellence Initiative at Jagiellonian University. The study were also partially performed with the use of equipment co-financed by the qLIFE Priority Research Area under the program "Excellence Initiative—Research University" at Jagiellonian University (number: 06/IDUB/2019/94).

S3.P3

THE BOOST OF FORCE IN UNFUSED TETANIC CONTRACTIONS AT VARIABLE INTERPULSE INTERVALS IN FAST MOTOR UNITS OF THE RAT MEDIAL GASTROCNEMIUS

K. KRYSCIAK[1], J. CELICHOWSKI[1], R. RAIKOVA[2], K. PODGORSKI[1], H. DRZYMALA-CELICHOWSKA[1,3]

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One of mechanisms modulating the force of unfused tetanic contractions of motor units (MUs) is boost (enhanced initial extra-efficient force production followed by sag - force decline), phenomenon strongly conditioned by blood circulation in the muscle. During voluntary movements, motoneuron interpulse intervals (IPIs) naturally vary. While boost and sag in unfused tetani are well-documented at constant frequencies, it remains unclear how IPI variability modulate the boost. Unfused tetanic contractions were recorded from fast fatigable (FF) and fast fatigue-resistant (FR) MUs of the rat medial gastrocnemius. Four protocols (mean frequency 35 Hz) were applied: one with constant IPIs and three with variable IPIs (28 ± 2 ms, ± 5 ms, and ± 7 ms). Additionally, the boost was also tested in contractions beginning with a doublet of stimuli at 5 ms interval, similar to discharge pattern of motoneurons frequently reported during their voluntary activity. Tetani were mathematically decomposed into successive twitch-like responses. The boost occurred exclusively in the first tetanic contraction of each set, persisting across all tested ranges of IPI variability in both MU types. Mathematical decomposition revealed that the underlying mechanisms of boost are pattern-independent. The boost phenomenon is a robust, pattern-independent property of fast MUs that occurs during irregular motoneuron discharge. This suggests that boost actively contributes to force development and neuromuscular regulation during natural, daily voluntary movements, particularly during short, powerful contractions. Effects of boost and doublet coexist and overlap which suggests that they have different mechanisms. The modulation of tetanic contractions by the boost and doublet varied depending on the type of MU and were stronger in FR MUs.

SESSION IV

FATIGUE, ADAPTATION TO ENVIRONMENT, THERMOREGULATION

Saturday (September 26th, 8.30 – 11.30) Room B

Chairman: Professor Pawel Zalewski
(Department of Exercise Physiology and Functional Anatomy, Collegium Medicum, Nicolaus Copernicus University in Torun, Torun, Poland)

Vice-Chairman: Professor Justyna Rogalska
(Department of Animal Physiology and Neurobiology;
Nicolaus Copernicus University in Torun, Torun, Poland)

DETAILED SESSION VI SCHEDULE

Oral presentations (September 26th, 9.00 – 9.45)

S4.L1 *9.00 – 9.15* AUTONOMIC MODERATION OF METABOLIC CARE EFFECTS ON SLEEP QUALITY TRAJECTORIES IN AGING: INSIGHTS FROM THE COPERNICUS STUDY. **A. Kujawska[1,2], S. Kujawski[1], Zalewski[3]** ([1]Department of Exercise Physiology and Functional Anatomy, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Torun, Bydgoszcz, Poland; [2]Cardiology and Cardiac Surgery Department, 10th Military Clinical Hospital with Polyclinic in Bydgoszcz, Bydgoszcz, Poland; [3]Department of Experimental and Clinical Physiology, Laboratory of Centre for Preclinical Research, Medical University of Warsaw, Warsaw, Poland).

S4.L2 *9.15 – 9.30* EVIDENCE FOR CHOLINERGIC MEDIATION OF TRP-DEPENDENT THERMOSENSORY RESPONSES IN AMERICAN COCKROACH FATIGUE, ADAPTATION TO ENVIRONMENT, THERMOREGULATION. **J. Maliszewska[1], M. Jankowska[1], M. Artkop[2], J. Rogalska[1]** ([1]Department of Animal Physiology and Neurobiology, Faculty of Biological and Veterinary Sciences, Nicolaus Copernicus University, Poland; [2]Department of Pathophysiology, Faculty of Pharmacy, Ludwik Rydygier Collegium Medicum, Poland).

S4.L3 *9.30 – 9.45* THE GUT, BRAIN AND FATIGUE AXIS IN ME/CFS AND LONG COVID: MICROBIOTA-RELATED MECHANISMS OF PERSISTENT FATIGUE AUTHORS. **H. Tabisz[1], S. Kujawski[1], J. Slomko[1], P. Zalewski[1,2]** ([1]Department of Exercise Physiology and Functional Anatomy, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Torun, Poland; [2]Department of Experimental and Clinical Physiology, Laboratory of Centre for Preclinical Research, Warsaw Medical University).

10.00 – 10.30 Coffee break

Poster Session (September 26th, 10.30 – 11.30)

S4.P1 DIAGNOSIS OF CHRONIC FATIGUE SYNDROME USING BEAT-TO-BEAT AUTONOMIC MEASUREMENTS. **S. Kujawski[1], H. Tabisz[1], K.J. Morten[2], A. Modlinska[1], J. Slomko[1], P. Zalewski[1,3]** ([1]Department of Exercise Physiology and Functional Anatomy, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Torun, Poland; [2]The Nuffield Department of Women's and Reproductive Health, University of Oxford, UK; [3]Laboratory of Centre for Preclinical Research, Department of Experimental and Clinical Physiology, Warsaw Medical University, Poland).

S4.P2 OPPORTUNITIES AND CHALLENGES OF CONTINUOUS MONITORING OF METABOLIC RATE AND HEAT STORAGE. A CONTINUOUS PARTIAL CALORIMETRY SYSTEM. **I. Ghafour[1], M. Petelczyc[1], S. Cygan[2], M. Zawiski[2], M. Szumilas[2], J. Zmigrodzki[2]** ([1]Warsaw University of Technology, Faculty of Physics, Poland; [2]Warsaw University of Technology, Faculty of Mechatronics, Warsaw, Poland).

S4.P3 EFFECTS OF WHOLE-BODY CRYOTHERAPY ON COGNITIVE FUNCTION IN CHRONIC DISEASES FATIGUE. ADAPTATION TO ENVIRONMENT. THERMOREGULATION. **A. Modlinska[1], H. Tabisz[1], S. Kujawski,[1] P. Zalewski[1,2]** ([1]Department of Exercise Physiology and Functional Anatomy, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Torun, Poland; [2]Department of Experimental and Clinical Physiology, Laboratory of Centre for Preclinical Research, Warsaw Medical University, Poland).

S4.P4 FATTY ACID COMPOSITION IN THE BROWN ADIPOSE TISSUE UNDER DIFFERENT CONDITIONS OF PHOTOPERIOD AND AMBIENT TEMPERATURE. **E. Bonda-Ostaszewska[1], A. Piotrowska-Niczyporuk[1], T.A. Bonda[2]** (Faculty of Biology, University of Bialystok, Bialystok, Poland; [2]Department of General and Experimental Pathology, Medical University of Bialystok, Bialystok, Poland).

S4.L1

AUTONOMIC MODERATION OF METABOLIC CARE EFFECTS ON SLEEP QUALITY TRAJECTORIES IN AGING: INSIGHTS FROM THE COPERNICUS STUDY

A. KUJAWSKA[1,2], S. KUJAWSKI[1], P. ZALEWSKI[1,3]

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Sleep-wake regulation in older adults is tightly coupled to autonomic balance, cerebral perfusion, and neurovascular homeostasis. Orthostatic hypotension (OH) reflects age-related autonomic/baroreflex decline and may alter physiological responsiveness to metabolic interventions. Whether autonomic dysfunction moderates the efficacy of specialized diabetes care on longitudinal sleep trajectories remains unexplored. In a 2-year longitudinal cohort of older adults (N=205), we tested whether OH status moderates the association between diabetologist-led care and change in self-reported sleep quality. Linear moderation models included an OH × Diabetologist interaction term and were adjusted for age, sex, and depressive symptoms. Conditional effects were probed via simple slopes analysis. A significant interaction emerged (β = -1.63, SE = 0.59, p = 0.0066, 95% CI [-2.80, -0.46]). Among participants without OH, diabetologist involvement predicted clinically meaningful sleep improvement (+1.71 points, $p < 0.001$). In contrast, among OH-positive individuals, this benefit was physiologically attenuated and statistically non-significant (+0.08 points, 95% CI [-0.83, 0.99], p = 0.86). Covariates were non-significant. The model explained 4.5% of variance (adj. R^2 = 0.0449), consistent with the multifactorial physiology of sleep regulation. Autonomic dysfunction, indexed by OH, constrains the sleep benefits of specialized metabolic care in aging. This suggests that hemodynamic/autonomic instability may override metabolic improvements, likely via impaired cerebral autoregulation, disrupted sleep-wake autonomic cycling, or altered neuroendocrine feedback. Integrating autonomic assessment and hemodynamic optimization into metabolic management may be necessary to restore sleep homeostasis in this subgroup. Future physiological studies should incorporate continuous autonomic markers (e.g., heart rate variability, baroreflex sensitivity) and polysomnographic endpoints to delineate underlying mechanisms.

S4.L2

EVIDENCE FOR CHOLINERGIC MEDIATION OF TRP-DEPENDENT THERMOSENSORY RESPONSES IN AMERICAN COCKROACH

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The perception of thermal stimuli in insects is mediated by Transient Receptor Potential (TRP) channels, which can be activated by temperature changes and specific chemical agonists. Although the involvement of TRP channels in insect thermosensation is well established, the downstream neural pathways underlying the integration of these signals in the cockroach nervous system remain poorly understood. This study aimed to elucidate the mechanisms linking TRP receptor activation to behavioral and neural responses in the American cockroach (*Periplaneta americana*), with a particular focus on nicotinic acetylcholine receptor (nAChR)-mediated signaling. Cockroaches were exposed to the TRP agonist capsaicin in the presence or absence of the nAChR antagonist MG624. Behavioral assays, including thermal preference and grooming behavior, were combined with extracellular recordings from the ventral nerve cord. Capsaicin induced a significant shift in thermal preference toward cooler environments, but this effect was abolished by nicotinic receptor blockade. Electrophysiological recordings showed that capsaicin reduced spontaneous neuronal firing, whereas MG624 increased baseline activity. Moreover, inhibition of α7-containing nAChRs prevented the capsaicin-induced suppression of neuronal activity, indicating that cholinergic signaling is required for this response. Collectively, these findings demonstrate that nicotinic cholinergic signaling is an essential component of the neural pathway mediating responses to thermo-TRP activation in cockroaches, providing new insights into the neurochemical basis of insect thermoregulation.

S4.L3

THE GUT, BRAIN AND FATIGUE AXIS IN ME/CFS AND LONG COVID: MICROBIOTA RELATED MECHANISMS OF PERSISTENT FATIGUE

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Persistent fatigue is a core symptom of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) and one of the most disabling manifestations of Long COVID. Both conditions include post exertional symptom exacerbation, cognitive complaints, autonomic imbalance and reduced tolerance to physiological stressors. Increasing evidence suggests that gut microbiota alterations may link immune activation, metabolic changes, intestinal barrier dysfunction and altered communication between the gut and the brain.Evidence from a systematic review on gut microbiota in ME/CFS and Long COVID was considered together with original ME/CFS microbiome analyses and pilot interventional observations. Microbiota composition was assessed using 16S rRNA sequencing. Clinical outcomes included fatigue severity, symptom burden and cognitive complaints. Diversity indices, taxonomic structure, ASV level differences and associations between microbial features and clinical outcomes were analyzed. ME/CFS and Long COVID showed partly overlapping microbiota alterations, including changes in taxa involved in inflammatory regulation, short chain fatty acid metabolism and gut barrier integrity. In ME/CFS, altered microbial composition and selected ASV level signatures were associated with fatigue related outcomes. Pilot observations after whole body cryotherapy combined with static stretching indicated that clinical changes may be accompanied by microbiome shifts, suggesting a contribution of microbial pathways to symptom modulation. Gut microbiota may represent a biologically relevant link between persistent fatigue, cognitive symptoms and impaired adaptation to physiological stressors in ME/CFS and Long COVID. These findings support further research on microbiota based biomarkers and interventions targeting fatigue dominant chronic conditions.

S4.P1

DIAGNOSIS OF CHRONIC FATIGUE SYNDROME USING BEAT-TO-BEAT AUTONOMIC MEASUREMENTS

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Background: Chronic Fatigue Syndrome (CFS) lacks reliable biomarkers, complicating diagnosis and delaying patient care. Autonomic nervous system (ANS) dysfunction is well-documented in CFS, yet objective diagnostic tools remain scarce. Methods: We conducted a prospective case-control study with 112 CFS patients and 61 healthy controls. High-frequency beat-to-beat cardiovascular and ANS parameters were recorded at rest using a Task Force Monitor. A novel sequential artificial intelligence pipeline was developed, combining a Transformer neural network with an XGBoost classifier trained on Transformer prediction errors. Six key predictors were selected: breathing frequency, stroke volume index (SV/FFM), high-frequency R-R interval (HF RRI), and beat-to-beat signed differences in heart rate, stroke volume, and diastolic blood pressure. Model performance was evaluated using Matthews correlation coefficient (MCC), accuracy, and ROC AUC, with SHapley Additive exPlanations (SHAP) for interpretability. Results: The combined Transformer-XGBoost model achieved exceptional subject-level diagnostic performance with an accuracy of 0.89, MCC of 0.79, and ROC AUC of 1.00. SHAP analysis revealed that lower HR differences and reduced HF RRI (indicating diminished cardiac vagal tone), higher dBP differences (reflecting elevated sympathetic vascular tone), and lower SV differences and SV/FFM values (indicating impaired cardiac hemodynamics) were strongly associated with CFS classification. Conclusions: This AI-driven pipeline demonstrates remarkable proficiency in objectively differentiating CFS patients from healthy controls using non-invasive beat-to-beat ANS measurements. The automated approach offers a fast, cost-effective, and highly accurate diagnostic tool that could significantly reduce diagnostic delays and improve clinical management of CFS.

Acknowledgements: We thank Prof Carmen Scheibenbogen, MD, for valuable feedback on the clinical context of this work.

S4.P2

OPPORTUNITIES AND CHALLENGES OF CONTINUOUS MONITORING OF METABOLIC RATE AND HEAT STORAGE. A CONTINUOUS PARTIAL CALORIMETRY SYSTEM

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Monitoring human metabolic rate (M) is used in the assessment of occupational safety, in rehabilitation, in the analysis of sports activity, and in the diagnosis of metabolic diseases. In practice M is determined by indirect calorimetry and estimated from models (e.g., Weir). The disadvantage of this approach is measurement limitations in free living conditions and lack of possibility to separate the exercise component. Using principles of heat balance principles, we developed a wearable device for continuous monitoring of metabolic rate (Continuous Partial Calorimetry system). Tests on nine healthy subjects (aged 28,67 ± 10,20) showed high percentage agreement (>95%) with gold standard in experiments performed in laboratory conditions. Additionally components of heat dissipation in human body were determined separately, focusing on radiation and convection. Measurement method combined with analytical model was used for heat storage estimation. Then mean body temperature was obtained showing significant increase for effort in relation to resting conditions. The latter parameter shows the effect of thermoregulation of human body in response to physical activity but can also be used to monitor the adaptation to the environment.

S4.P3

EFFECTS OF WHOLE-BODY CRYOTHERAPY ON COGNITIVE FUNCTION IN CHRONIC DISEASES

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Whole-body cryotherapy (WBC) involves brief exposure to extremely cold air (typically -110°C to -140°C) and has gained increasing interest as a therapeutic intervention beyond sports medicine. Cognitive dysfunction is a prevalent and debilitating symptom across numerous chronic diseases, including multiple sclerosis (MS), rheumatoid arthritis (RA), fibromyalgia, myalgic encephalomyelitis (ME)/chronic fatigue syndrome (CFS) and long COVID. For this reason, available scientific databases such as PubMed, Web of Science and Scopus were reviewed using the terms “whole-body cryotherapy,” “cryostimulation and cognition,” “cryotherapy and cognitive function,” “cryotherapy and chronic disease,” and “cold therapy and neuroinflammation” to analyze the effects of WBC on cognitive outcomes in chronic conditions. Current evidence indicates WBC may improve cognitive function through several interconnected mechanisms: reduction of systemic and neuroinflammation (decreased levels of pro-inflammatory cytokines such as IL-6 and TNFα), modulation of the autonomic nervous system, improvement of sleep quality, and reduction of pain and fatigue – all of which are factors known to impair cognitive performance. In patients with MS, WBC has been associated with improvements in attention and processing speed. In fibromyalgia and ME/CFS, studies report reductions in “brain fog” and mental fatigue following WBC series. In RA, improvements in mood and subjective cognitive complaints have been observed alongside decreased inflammatory markers. However, the available evidence remains limited by small sample sizes, heterogeneous study designs, and lack of standardized cognitive assessment protocols. WBC appears to be a promising non-pharmacological adjunct therapy for alleviating cognitive impairment in chronic diseases, though further well-designed randomized controlled trials are needed to establish optimal protocols and confirm long-term benefits.

S4.P4

FATTY ACID COMPOSITION IN THE BROWN ADIPOSE TISSUE UNDER DIFFERENT CONDITIONS OF PHOTOPERIOD AND AMBIENT TEMPERATURE

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Brown adipose tissue (BAT) plays an important role in thermoregulatory heat production. Thermoregulatory potential of tissue may change undergo photoperiod and temperature. The main energy component of BAT are fatty acids in triacylglycerol form and they are visible in the BAT cells as lipid droplets. The aim of this study was to investigate the effect of photoperiod and temperature on fatty acid composition and lipid droplets structure in BAT. Bank voles were held under short (SD) or long (LD) photoperiod in 20°C or 5°C, creating four experimental groups (LP20, SP20, SP5 and LP5). We found a significant reduction in total fatty acids content at low temperature (by 40% in LP5) and short photoperiod (by 50% in SP-20) with the greatest reduction in the SP5 group (60%). This effect was accompanied by both a total BAT mass depletion and a reduction of lipid droplet size seen on histological slides. Analysis of the fatty acid composition showed that as much as 99% of the fatty acids in BAT are long-chain fatty acids LC-FA (C13-C18). The largest fractions in this group are palmitic acid (C16:0) and stearic acid (C18:0). Their total relative share increases from 39% in LP20 group to 51-58% in remaining group. Thus these acids probably determine high energy potential of the BAT under low temperature and short photoperiod. A large, relatively constant fraction (25-27%) are monounsaturated C18 fatty acids (C18:1n9c, C18:1n9t). The content of linoleic acid (C18:3n6) decreases from 23% to 15%, 16% in SP groups, and up to 10% in LP5. Although VLC-FA (very long chain fatty acids, C ≥ 20) constitute only a small percentage of total amount of fatty acids (<1%), two interesting changes in their composition are visible: (1) low temperature and short photoperiod increase the contribution of VLV-FA in the total pool of fatty acids (from 0.65% in LP20 to 0.89% in SP5); (2) the proportion of unsaturated to saturated fatty acids increases from 47% in the LP20 group to 58-59% in the groups exposed to low temperature or/and short photoperiod. Increasing the proportion of unsaturated fatty acids can increase membrane fluidity and improve BAT efficiency.

SESSION V

NEW TRENDS IN PHYSIOLOGY AND PATHOPHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM AND RENAL FUNCTION

Friday (September 25th, 8.30 – 13:30) Room A

Chairman: Professor Barbara Malinowska
(Department of Experimental Physiology and Pathophysiology, Faculty of Pharmacy, Medical University of Bialystok, Bialystok, Poland)

Chairman: Professor Elzbieta Kompanowska-Jezierska
(Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland)

Chairman: Professor Agnieszka Cudnoch-Jedrzejewska
(Department of Experimental and Clinical Physiology, Laboratory of Centre for Preclinical Sciences, Medical University of Warsaw, Warsaw, Poland)

Vice-Chairman: Associate Professor Anna Pedzinska-Betiuk
(Department of Experimental Physiology and Pathophysiology, Faculty of Pharmacy, Medical University of Bialystok, Bialystok, Poland)

DETAILED SESSION V SCHEDULE

Opening lecture 1 Friday (September 25th, 8.30 – 9:10)

S5.L1 ENDOTHELIAL MITOCHONDRIAL DYSFUNCTION AND VASCULAR AGEING: IMPLICATIONS FOR NOVEL METABOLIC THERAPIES. **B. Kutryb-Zajac[1,2], M. Tarnawska[3], L. Wozniak-Mielczarek[4], R. Knapczyk[2,3], A. Parzuchowska[3], I. Walczak[1], A. Paterek[5], F. Rolski[5], P. Leszek[6], P. Blyszczuk[7,8], M. Maczewski[5], M. Hellmann[1]** ([1]Department of Biochemistry, Medical University of Gdansk, Gdansk, Poland; [2]Centre for Experimental Cardiooncology, Medical University of Gdansk, Gdansk, Poland; [3]Department of Cardiac Diagnostics, Medical University of Gdansk, Gdansk, Poland; [4]Department of Pediatric Cardiology and Congenital Heart Defects, Medical University of Gdansk, Gdansk, Poland; [5]Department of Clinical Physiology, The Centre of Postgraduate Medical Education, Warsaw, Poland; [6]Heart Failure, Transplantology and Mechanical Circulatory Support Department, National Institute of Cardiology, Warsaw, Poland; [7]Department of Clinical Immunology, Jagiellonian University Medical College, Cracow, Poland; [8]Department of Rheumatology, University Hospital Zurich, University of Zurich, Zurich, Switzerland).

Oral presentations Friday (September 25th, 9.10 – 10.10)

S5.L2 *9.10 – 9.25* CELLULAR SENESCENCE AND APOPTOSIS IN THE COURSE OF POST-MYOCARDIAL INFARCTION HEART FAILURE AND HIGH-FAT DIET**. K. Krauz, K. Czarzasta, Z. Zalewska, K. Momot, A. Cudnoch-Jedrzejewska** (Department of Physiology and Pathophysiology, Medical University of Warsaw, Warsaw, Poland).

S5.L3 *9.25 – 9.40* MODULATORY ROLE OF miR-425-5p IN THE TGF-β/SMAD PATHWAY IN POST-INFARCTION HEART FAILURE AS A POTENTIAL ANTIFIBROTIC TARGET NEW TRENDS IN PHYSIOLOGY AND PATHOPHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM. **M. Braczkowski[1], A. Wester[1], K. Kufel[2], D. Czyz[2], J. Kulinska[2], A. Dittmann[2], R. Braczkowski[3]** ([1]University of Opole, Faculty of Medicine, Institute of Medical Sciences, Department of Physiology; [2]Students Scientific Society of Physiology, University of Opole, Faculty of Medicine, Institute of Medical Sciences, Department of Physiology; [3]Silesia Academy, Katowice, Poland).

S5.L4 *9.40 – 9.55* STEAROYL-CoA DESATURASE REGULATES POSTNATAL HEART DEVELOPMENT. **V. Balatskyi, P. Dobrzyn** (Nencki Institute of Experimental Biology, Warsaw, Poland).

S5.L5 *9.55 – 10.10* ANALYSIS OF THE ROLE OF UNCONVENTIONAL MYOSIN VI IN THE HEART METABOLISM. **W. Ficerman-Kuta[1], O. Karatsai-Miaskowska[1], J. Nowak[1], V. Navrulin[2], P. Dobrzyn[2], J. Zurmanova[3], L. Lehka[1], D. Wojton[1], M. J. Redowicz[1]** ([1]Laboratory of Molecular Basis of Cell Motility, Nencki Institute of Experimental Biology PAS, Warsaw, Poland; [2]Laboratory of Molecular Medical Biochemistry, Nencki Institute of Experimental Biology PAS, Warsaw, Poland; [3]Department of Physiology, Faculty of Science, Charles University, Prague, Czech Republic).

10.10 – 10.40 Coffee break

Opening lecture 2 Friday (September 25th 10.40 – 11:20)

S5.L6 CRITICAL ROLE OF THE KIDNEY-BRAIN NEURAL INTERACTION IN PATHOPHYSIOLOGY OF CARDIORENAL SYNDROME: INSIGHT FROM STUDIES EMPLOYING SHR WITH VOLUME OVERLOAD INDUCED BY AORTO-CAVAL FISTULA. **Z. Honetschlagerova , M. Miklovic A.S. Kikerlova , J. Sadowski, Z. Vanourkova, P. Skaroupkova, S. Vackova, M. Taborsky, L. Cervenka** (Center for Experimental Medicine, Institute for Clinical and Experimental Medicine, Prague, Czech Republic).

Oral presentations Friday (September 25th 11.20 – 12:45)

S5.L7 *11.20 – 11.35* THE HEART UNDER PRESSURE IN PULMONARY HYPERTENSION: INSIGHTS FROM OUR STUDIES USING ISOLATED CARDIAC PREPARATIONS. **A. Pedzinska-Betiuk, J. Weresa, K. Ostapkowicz, K. Minczuk, P. Remiszewski, P. Ryszkiewicz, B. Malinowska** (Department of Experimental Physiology and Pathophysiology, Medical University of Bialystok, Bialystok, Poland).

S5.L8 *11.35 – 11.50* COMBINED CANNABINOID CB_1 RECEPTOR AND INOS INHIBITION RESTORES IMPAIRED VASCULAR FUNCTION IN EXPERIMENTAL RAT PULMONARY HYPERTENSION. **M. Kloza[1], A. Krzyzewska[1], H. Kozlowska[1], K. Minczuk[1], K.H. Majchrzak[1], P. Remiszewski[1], P. Ryszkiewicz[1], B. Malinowska[1], M. Baranowska, Kuczko[1,2]** ([1]Departmet of Experimental Physiology and Pathophysiology; [2]Department of Clinical Pharmacy, Medical University of Bialystok, Bialystok, Poland).

S5.L9 *11.50-12.05* THE ROLE OF UNCONVENTIONAL MYOSIN VI IN THE AORTA STRUCTURE AND FUNCTION. **J. Nowak, O. Karatsai-Miaskowska, W. Ficerman-Kuta, M.J. Redowicz** (Nencki Institute of Experimental Biology PAS, Laboratory of Molecular Basis of Cell Motility, Warsaw, Poland).

S5.L10 *12.05-12.20* QUANTITATIVE PHASE IMAGING REVEALS ALTERATIONS IN THE CELLULAR BEHAVIOUR, MIGRATION, AND MORPHOLOGY OF HUMAN PODOCYTES UNDER DIABETIC-MIMETIC CONDITIONS. **M. Szrejder, A. Piwkowska** (Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland).

S5.L11 *12.20-12.35* CD147 AS A MAJOR REGULATOR OF ENERGY HOMEOSTASIS IN PODOCYTES. **A. Kubiak, T. Kulesza, A. Piwkowska** (Institute of Experimental and Clinical Medicine of the Polish Academy of Sciences, Warsaw, Poland).

Poster Session Friday (September 25th, 12.45 – 13:30)

S5.P1 RISK OF CARDIOVASCULAR DISEASES (CVD), INCLUDING THE PROGRESSION OF ATHEROSCLEROSIS, IN PATIENTS EXPOSED TO MICRO- AND NANOPLASTICS. **D. Czyz[1], A. Dittmann[1], K. Kufel[1], J. Kulinska[1], D. Walas[1], E. Wozniak-Grygiel[2], M.J. Braczkowski[3], D. Soszynski[3], R.J. Buldak[4], W. Ochedzan–Siodlak[5], M. Gierlotka[6]** ([1]Student Scientific Society of Physiology, Department of Physiology, Institute of Medical Sciences, University of Opole, Opole, Poland; [2]Department of Histology, Institute of Medical Sciences, University of Opole, Opole, Poland; [3]Department of Physiology, Institute of Medical Sciences, University of Opole, Opole, Poland; [4]Department of Biochemistry and Laboratory Diagnostics, Institute of Medical Sciences, University of Opole, Opole, Poland; [5]Faculty of Chemistry and Pharmacy, University of Opole, Opole, Poland; [6]Clinical Department of Cardiology, University Clinical Hospital, Institute of Medical Sciences, University of Opole, Opole, Poland).

S5.P2 EFFECTS OF APELIN-13 ON HEMODYNAMIC ALTERATIONS AND OXIDATIVE STRESS MARKERS IN RAT MODEL OF CHRONIC DOXORUBICIN-INDUCED CARDIOTOXICITY. **W. Kosiorowska, K. Kaminska, K. Matusik, D. Sztechman, K. Buczma, H. Borzuta, K. Kasarello, A. Cudnoch–Jedrzejewska (**Department of Physiology and Pathophysiology, Medical Simulation Center, Medical University of Warsaw, Warsaw, Poland).

S5.P3 INCREASED LEVEL OF MIR-29B IN ACTIVELY SMOKING PATIENTS WITH CHRONIC CORONARY SYNDROME – A SMALL OBSERVATIONAL STUDY. **M. Kowara[1,3], M. Kopka[2], K. Kopka[2], R. Glowczynska[3], P. Barus[3], K. A. Sadowski[1], A. Cudnoch-Jedrzejewska[1]** ([1]Chair and Department of Experimental and Clinical Physiology, Medical University of Warsaw, Poland; [2]Department of Methodology, Medical University of Warsaw, Warsaw, Poland; [3]First Department of Cardiology, Medical University of Warsaw, Warsaw, Poland).

S5.P4 METABOLIC SYNDROME AND RS1544410 (BSMI) VDR POLYMORPHISM IN RELATION TO LIPID PROFILE AND ATHEROGENIC RISK IN OLDER ADULTS. **S. Michniewicz[1], M. Gibas-Dorna[2], K. Chmielowiec[3], B. Czyzniewski[2], E. Pruszynska-Oszmalek[4], P. Kolodziejski[4], M.T. Kowalski[5], A. Grzywacz[6], J. Chmielowiec[3]** ([1]Department of Humanization of Health Care and Sexology, University of Zielona Gora, Poland; [2]Department of Anatomy and Histology, University of Zielona Gora, Poland; [3]Department of Hygiene and Epidemiology, University of Zielona Gora, Poland; [4]Department of Animal Physiology, Biochemistry and Biostructure, Poznan University of Life Sciences, Poland; [5]Clinical Department of Cardiology, Multispecialty Hospital in Nowa Sol, Poland; [6]Independent Laboratory of Genetics and Behavioral Epigenetics, Pomeranian Medical University in Szczecin, Szczecin, Poland).

S5.P5 FATTY ACIDS SYNTHESIZED BY ELOVL3 HAVE DIFFERENT EFFECTS ON THE METABOLISM AND CONTRACTILITY OF HL-1 CARDIOMYOCYTES. **V. Navrulin, P. Dobrzyn** (Nencki Institute of Experimental Biology of the Polish Academy of Sciences, Warsaw, Poland).

S5.P6 MACHINE LEARNING-BASED INFORMATION FLOW ANALYSIS OF ECG SIGNALS FOR CONGENITAL LONG QT SYNDROME. **M. Ozimek, M. Andrzejewska-Ozimek, M. Petelczyc** (Faculty of Physics, Warsaw University of Technology, Warsaw, Poland).

S5.P7 MODEL-DEPENDENT RESPONSES TO CHRONIC TREATMENT WITH AMPK ACTIVATOR METFORMIN AND PERIHERAL CB_1 RECEPTOR ANTAGONIST JD5037 IN RAT PULMONARY HYPERTENSION. **P. Remiszewski, P. Ryszkiewicz, K. Minczuk, A. Pedzinska-Betiuk, M. Baranowska-Kuczko, B. Malinowska** (Department of Experimental Physiology and Pathophysiology, Medical University of Bialystok, Bialystok, Poland).

S5.P8 EFFECTS OF CHRONIC ADMINISTRATION OF PERIPHERAL HYBRID CANNABINOID CB_1R AND iNOS ANTAGONIST MRI-1867 ON RIGHT VENTRICULAR CONTRACTILE FUNCTION IN RAT PULMONARY HYPERTENSION. **J. Weresa, A. Pedzinska-Betiuk, K. Ostapkowicz, K. Minczuk, P. Remiszewski, P. Ryszkiewicz, B. Malinowska** (Department of Experimental Physiology and Pathophysiology, Medical University of Bialystok, Bialystok, Poland).

S5.L1

ENDOTHELIAL MITOCHONDRIAL DYSFUNCTION AND VASCULAR AGEING: IMPLICATIONS FOR NOVEL METABOLIC THERAPIES

B. KUTRYB-ZAJAC[1,2], M. TARNAWSKA[3], L. WOZNIAK-MIELCZAREK[4], R. KNAPCZYK[2,3], A. PARZUCHOWSKA[3], I. WALCZAK[1], A. PATEREK[5], F. ROLSKI[5], P. LESZEK[6], P. BLYSZCZUK[7,8], M. MACZEWSKI[5], M. HELLMANN[1]

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[6]Heart Failure, Transplantology and Mechanical Circulatory Support Department, National Institute of Cardiology, Warsaw, Poland, [7]Department of Clinical Immunology, Jagiellonian University Medical College, Krakow, Poland, [8]Department of Rheumatology, University Hospital Zurich, University of Zurich, Zurich, Switzerland; barbara.kutryb-zajac@gumed.edu.pl

Vascular ageing is increasingly recognized as a fundamental mechanism contributing to the development and progression of cardiovascular diseases. Recent evidence indicates that endothelial senescence, chronic low-grade inflammation (inflammaging) and mitochondrial dysfunction represent interconnected processes driving vascular remodeling, microvascular impairment and adverse cardiovascular outcomes. Our recent studies combine clinical investigations in patients with heart failure (HF), hereditary thoracic aortic disease (HTAD) and obesity with mechanistic analyses performed in primary human endothelial cells. Using fluorescence-based assessment of microcirculatory and metabolic function (Flow Mediated Skin Fluorescence, FMSF), proteomic profiling and advanced mitochondrial phenotyping, we identified profound alterations in endothelial homeostasis associated with ageing and cardiovascular pathology. Clinical studies demonstrated impaired microvascular and metabolic responses across the heart failure spectrum and in HTAD patients, accompanied by distinct circulating signatures of endothelial senescence and senescence-associated secretory phenotype (SASP). Proteomic analyses revealed dysregulation of biomarkers related to endothelial activation, inflammation, extracellular matrix remodeling and vascular ageing. In parallel, patient-derived coronary and aortic endothelial cells exhibited disease-specific mitochondrial dysfunction, altered bioenergetics, disturbed mitochondrial architecture and enhanced senescence-related signaling. Experimental studies indicate that contemporary metabolic therapies, including sodium-glucose cotransporter-2 inhibitors (SGLT2i) and incretin-based therapies, exert direct vascular protective effects. These interventions improve mitochondrial homeostasis, modulate endothelial metabolism, and attenuate molecular pathways associated with cellular senescence and inflammaging. Together, these findings support a novel paradigm in cardiovascular pathophysiology in which endothelial senescence and mitochondrial dysfunction constitute central determinants of vascular ageing and therapeutic responsiveness. Identification of integrated biomarker signatures linking endothelial ageing, mitochondrial health and microvascular function may facilitate precision medicine approaches and enable more effective monitoring of emerging metabolic therapies in cardiovascular disease.

S5.L2

CELLULAR SENESCENCE AND APOPTOSIS IN THE COURSE OF POST-MYOCARDIAL INFARCTION HEART FAILURE AND HIGH-FAT DIET

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Background: Myocardial infarction (MI) is a cause of heart failure (HF). Pathological cardiac remodeling after MI may be exacerbated by a high-fat diet (HFD). Cellular senescence has been shown to play a role in the pathogenesis of HF. Also, senescent cells become resistant to apoptosis. However, apoptosis itself may worsen the cardiac function during HF. The relations between these processes, especially during post-MI HF and HFD, remain insufficiently understood. Aim: To assess the effect of post-MI HF and HFD on apoptosis and selected markers of cellular senescence (Cdkn2a, Tp53) in the heart. Methods: The study was performed on 27 left ventricles obtained from male Sprague-Dawley rats fed an HFD or a normal-fat diet (NFD) for 12 weeks, starting at 4 weeks of age. At 12 weeks of age, the rats underwent ligation of the left coronary artery (NFD-HF, HFD-HF) or sham surgery (NFD-SO, HFD-SO). At 16 weeks of age, the tissues were collected for analysis. The ELISA method was used to measure plasma NT-proBNP concentrations. Tp53 and Cdkn2a mRNA expression was assessed by the qRT-PCR, whereas their protein levels - by Western blotting. Apoptosis was detected by TUNEL staining. Results: NT-proBNP plasma concentrations were higher in the HFD-HF and NFD-HF than in HFD-SO and NFD-SO, respectively. A two-way ANOVA revealed a significant interaction between the effect of the diet and post-MI HF on the number of TUNEL positive nuclei which was higher in NFD-HF in comparison to the NFD-SO, whereas such relationship was not observed among groups maintained on HFD. Only post-MI HF had a significant effect on Cdkn2a and Tp53 mRNA expression, and TP53 protein levels, whereas CDKN2A protein levels were affected only by the diet. Post-hoc test revealed that Tp53 and Cdkn2a mRNA expressions were higher in NFD-HF than in corresponding sham-operated animals. TP53 protein levels were higher in NFD-HF and HFD-SO than in NFD-SO and HFD-HF, respectively, whereas CDKN2A protein levels were the highest in HFD-HF. Conclusions: Coincidence of post-MI HF and HFD may promote stable cellular senescent state, over apoptosis, which seems to dominate in sham-operated animals on HFD.

S5.L3

MODULATORY ROLE OF MIR-425-5P IN THE TGF-B/SMAD PATHWAY IN POST-INFARCTION HEART FAILURE AS A POTENTIAL ANTIFIBROTIC TARGET NEW TRENDS IN PHYSIOLOGY AND PATHOPHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM

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Post-infarction heart failure is a major consequence of myocardial infarction and is associated with progressive adverse cardiac remodeling and increased mortality. Myocardial fibrosis, extracellular matrix remodeling and fibroblast activation contribute to deterioration of cardiac function despite guideline-directed therapy. One of the key molecular regulators involved in these processes is the TGF-β/Smad signaling pathway. Activation of TGF-β1 leads to phosphorylation of Smad2/3 proteins and regulation of genes associated with collagen synthesis, myofibroblast activation and extracellular matrix remodeling, thereby promoting pathological fibrosis and progression of heart failure. MicroRNAs are important post-transcriptional regulators of gene expression involved in cardiovascular remodeling. Among them, miR-425-5p has been identified as a potential modulator of fibrosis-related signaling pathways. Experimental studies suggest that altered miR-425-5p expression may affect TGF-β1 expression and Smad signaling activity, influencing fibroblast activation and myocardial remodeling. Despite increasing evidence regarding the role of microRNAs in cardiovascular disease, clinical data evaluating the association between miR-425-5p expression and TGF-β/Smad pathway activity in post-infarction heart failure remain limited. The relationship between miR-425-5p levels, profibrotic molecular signaling and clinical features of cardiac remodeling has not been fully elucidated. Better understanding of these mechanisms may support identification of novel biomarkers and future antifibrotic therapeutic targets in post-infarction heart failure.

S5.L4

STEAROYL-COA DESATURASE REGULATES POSTNATAL HEART DEVELOPMENT

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Stearoyl-CoA desaturase (SCD) is a key enzyme in the synthesis of monounsaturated fatty acids, and mice with knocked-out (KO) SCD1 or SCD4 gene exhibit protection against obesity-induced heart dysfunction. However, little is known about involvement of SCD in the regulation of postnatal heart maturation. We analyzed heart growth in SCD1 KO, SCD4 KO, and wild-type (WT) mice at 0-, 10-, and 21 days of age. Starting from day 10, the heart-to-body weight ratio was lower in SCD1 KO mice, whereas in SCD4 KO mice, this ratio was lower on day 21 compared to WT mice. Histological analysis of the hearts of 0-day-old mice revealed a decrease in trabecular myocardial thickness in SCD1 KO and SCD4 KO mice compared to WT mice, suggesting accelerated heart maturation. We then characterized the functional state of cardiomyocytes isolated from 0-day-old mice. The amplitude and time to peak of the spontaneous calcium wave were unchanged in KO cardiomyocytes, although SCD1 KO and SCD4 KO cardiomyocytes exhibited faster calcium reuptake. The level of Serca2a, a protein responsible for calcium reuptake, was increased in both SCD KO strains. SCD1 KO and SCD4 KO cardiomyocytes showed a higher amplitude of spontaneous beating and a shorter relaxation time, which is consistent with faster calcium clearance to the sarcoplasmic reticulum. Furthermore, neonatal SCD1 KO and SCD4 KO cardiomyocytes showed more developed sarcomeric organization than WT cardiomyocytes, which may account for the increased contractility of SCD KO cardiomyocytes. Thus, SCD1 and SCD4 may be involved in heart maturation, and their deletion accelerates the acquisition of the adult cardiomyocyte phenotype.

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S5.L5

ANALYSIS OF THE ROLE OF UNCONVENTIONAL MYOSIN VI IN THE HEART METABOLISM

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Unconventional myosin VI (MVI) has been identified as a contributor to cardiomyopathy (CM) as its lack leads to CM in *Snell's waltzer* mice, natural MVI knockouts (MVI-KO). Our group also demonstrated that MVI regulates skeletal muscle energy metabolism. As disturbances in lipid and glucose metabolism are associated with CM progression, we investigated MVI role in cardiac metabolism. Our studies were conducted on whole hearts of newborn (P0), 3-month- (3m) and 12-month-old MVI-KO and heterozygous (WT) mice. Thin-layer chromatography revealed elevated levels of cholesterol in P0 and 3m KO mice hearts and an increase in cholesteryl ester in P0 KO hearts. Despite this, the number and size of lipid droplets, visualized by transmission electron microscopy, were not changed in P0 KO hearts in comparison to control. Although Western Blot did not show changes in the levels of enzymes involved in lipid and glucose metabolism, we identified perilipin 2, ATP-dependent phosphofructokinase and L-lactate dehydrogenase as potential MVI binding partners. Our results indicate that lack of MVI promotes early cardiac cholesterol accumulation, without major alterations in glucose and lipid metabolism, suggesting altered cholesterol trafficking.

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S5.L6

CRITICAL ROLE OF THE KIDNEY-BRAIN NEURAL INTERACTION IN PATHOPHYSIOLOGY OF CARDIORENAL SYNDROME: INSIGHT FROM STUDIES EMPLOYING SHR WITH VOLUME OVERLOAD INDUCED BY AORTO-CAVAL FISTULA

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The first aim of the study was to assess the autoregulatory capacity of renal blood flow (RBF) and glomerular filtration rate (GFR) and the pressure-natriuresis relationship in spontaneously hypertensive rats (SHR) with aorto-caval fistula (ACF)-induced heart failure (HF), a model of HF-induced cardiorenal syndrome. The second aim was to evaluate the effects of total renal denervation (T-RDN; afferent and efferent renal nerves ablation) and selective renal afferent denervation (A-RDN) on autoregulation and pressure natriuresis. T-RDN and A-RDN were performed one week after ACF creation. Two weeks later, animals were prepared for acute studies evaluating RBF, GFR, urine flow (UF), and sodium excretion (UNaV) in response to stepwise changes in renal arterial pressure (RAP) induced by aortic clamping. Basal values of mean arterial pressure (MAP), RBF, UF, and UNaV were lower in ACF SHR than in sham-ACF SHR. In sham-ACF SHR, stepwise reductions in RAP decreased RBF and GFR, whereas in ACF SHR both remained unchanged. Stepwise reductions in RAP decreased UF and UNaV in sham-ACF SHR and ACF SHR, with greater decreases in sham-ACF SHR. In ACF SHR, neither T-RDN nor A-RDN altered MAP. Neither denervation increased basal RBF, but both increased basal UF and UNaV and improved the pressure-natriuresis relationship compared with innervated ACF SHR. In conclusion, in this model of high-output HF-induced cardiorenal syndrome, renal autoregulation is preserved and the pressure-natriuresis relationship is improved. Both T-RDN and A-RDN improve pressure-natriuresis relationship in ACF SHR, suggesting an important role of kidney-brain neural interaction in the pathophysiology of this condition.

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S5.L7

THE HEART UNDER PRESSURE IN PULMONARY HYPERTENSION: INSIGHTS FROM OUR STUDIES USING ISOLATED CARDIAC PREPARATIONS

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Pulmonary hypertension (PH) is a progressive and potentially life-threatening condition characterized by elevated pulmonary arterial pressure and increased vascular resistance. Disease development and prognosis are largely determined by the adaptive capacity of the overloaded right ventricle, making the heart not merely a passive responder but a central contributor to disease outcome. Understanding cardiac function, its adaptive limits, and its pharmacological responsiveness is therefore essential, highlighting the importance of focused studies on myocardial contractility in PH. A wide range of experimental tools is available for assessing myocardial function in the context of PH-induced changes, including measurements of right ventricular systolic pressure (RVSP) and echocardiographic evaluation of cardiac remodeling. An important and reliable method for assessing cardiac contractility in PH involves the use of isolated rat myocardial preparations (atria and papillary muscles). This approach enables evaluation of cardiac contractile activity under both basal conditions and following β-adrenoceptor stimulation, after which the contractile force of isolated right ventricular papillary muscles from rats with PH is typically reduced, reflecting impaired adaptive capacity of the heart. Moreover, our observations obtained using isolated myocardial preparations indicate that experimental variables may significantly influence the observed contractile responses, including initial body weight at the time of PH induction, sex, and the experimental model used.

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S5.L8

COMBINED CANNABINOID CB_1 RECEPTOR AND INOS INHIBITION RESTORES IMPAIRED VASCULAR FUNCTION IN EXPERIMENTAL RAT PULMONARY HYPERTENSION

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Pulmonary arterial hypertension (PAH) is a complex, multifactorial, and still incurable disease characterized by an imbalance between vasoconstrictor and vasodilator factors, leading to increased pulmonary vascular resistance and elevated right ventricular systolic pressure (RVSP). The aim of the study was to investigate the effects of chronic co-administration of JD5037 (a peripheral cannabinoid CB_1 receptor antagonist) and 1400W (an inducible nitric oxide synthase (iNOS) inhibitor) in two experimental rat models of pulmonary hypertension (PH): induced by Sugen/hypoxia (SuHx) and monocrotaline (MCT). RVSP in PH rats reached approximately 70 mmHg in both models. Functional studies were performed on isolated pulmonary segmental arteries using a wire myograph. Concentration-response curves analysis showed enhanced serotonin (5-HT)-mediated vasoconstriction in both PH models, and impaired ACh-induced endothelium-dependent relaxation only in the SuHx. 5-HT-induced vasoconstriction exhibited higher potency in the SuHx compared to the MCT, whereas acetylcholine (Ach)-induced endothelium-dependent relaxation did not differ between models. Monotherapy (JD5037 or 1400W) had no significant effect on the vascular function in each PH model, with the exception of JD5037, which reduced the vasoconstrictor response to 5-HT in the SuHx. Dual therapy (JD5037+1400W) (1) attenuated 5-HT-induced vasoconstriction and (2) improved Ach-induced relaxation in both PH models. Importantly, it also reduced RVSP in the MCT, while a downward trend in RVSP was observed in SuHx. These findings support a polypharmacological strategy targeting the cannabinoid CB_1 receptor and iNOS in PAH, demonstrating model-independent efficacy and suggesting advantages over single-target approaches, with further studies warranted.

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S5.L9

THE ROLE OF UNCONVENTIONAL MYOSIN VI IN THE AORTA STRUCTURE AND FUNCTION

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Unconventional myosin VI (MVI), a unique actin-based motor protein, is involved in skeletal and cardiac muscle functions. We previously showed that its loss leads to the age-progressing cardiomyopathy and impaired hindlimb muscle function and metabolism. We also demonstrated that MVI loss is associated with a decrease in vascular endothelial growth factor A (VEGFA) levels in the heart, indicating a possible MVI-associated impairment of vascularization and/or vessel function. Based on these observations, we decided to examine a role of MVI in aorta morphology and organization. We performed studies on the level of thoracic aorta of 3-month (3M) and 12-month (12M)-old MVI-KO (Snell's waltzer) and heterozygous (WT) mice. Morphometric analysis revealed age-dependent alterations in the aortic wall (from aortic stenosis to dilation). MVI is present both in the endothelium and smooth muscle compartments of the WT aortas and its level decreases with age, similarly to the cardiac muscle. Using transmission electron microscopy, we observed several changes in aorta structural compartments. Immunofluorescent staining of the aorta revealed an age-related accumulation of platelet/endothelial cell adhesion protein 1 (PECAM1) in the *tunica intima*, suggesting involvement of MVI in the synthesis and/or release of inflammatory mediators, hormones, and factors (including NO) regulating vascular contractile and relaxation activity. Furthermore, we showed that endothelial nitric oxide synthase (eNOS) is a potential MVI partner in the heart. Thus, these data indicate important role(s) of MVI in vascularization and blood vessel function, and further research is needed to elucidate mechanisms behind these observations.

S5.L10

QUANTITATIVE PHASE IMAGING REVEALS ALTERATIONS IN THE CELLULAR BEHAVIOUR, MIGRATION, AND MORPHOLOGY OF HUMAN PODOCYTES UNDER DIABETIC-MIMETIC CONDITIONS

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Diabetic kidney disease affects approximately one in three adults with diabetes and is the leading cause of end-stage renal disease requiring renal replacement therapy. Podocytes, key component of the glomerular filtration barrier, enwrap glomerular capillaries with their foot processes and play an essential role in blood filtration. Because this structure must continuously adapt to changes in filtration pressure, controlled podocyte motility appears to be critical for proper function. The aim of this study was to characterize how high glucose concentrations, mimicking diabetic conditions, affect the behaviour, motility, and morphology of immortalized human podocytes using quantitative phase imaging based on digital holographic microscopy (HoloMonitor, Phase Holographic Imaging AB, Lund, Sweden). Cells were analyzed under standard-glucose (11.1 mM) and high-glucose (30 mM) conditions. The results showed that exposure of podocytes to high glucose concentrations led to significant alterations in their motile and morphological properties. Kinetic motility analysis demonstrated reduced motility speed and accumulated distance, accompanied by increased migration distance. Moreover, single-cell tracking revealed that podocyte movement under hyperglycemic conditions was more directed compared with control cells. These changes in podocyte motility and morphology may indicate disruption of their normal function under hyperglycemic conditions, which may contribute to the development of diabetic damage to the glomerular filtration barrier.

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S5.L11

CD147 AS A MAJOR REGULATOR OF ENERGY HOMEOSTASIS IN PODOCYTES

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Diabetic kidney disease (DKD) remains a leading cause of reduced lifespan in patients with diabetes. Early pathological changes in DKD progression manifest as gradual glomerular hypertrophy and podocyte depletion, driven by chronic hyperglycemia and hyperinsulinemia. Metabolic alterations in podocytes under hyperglycemic (HG) conditions play a crucial role in dysfunction of the glomerular filtration barrier. It is hypothesized that CD147, a transmembrane glycoprotein and essential chaperone for monocarboxylate transporters (MCTs), may critically regulate the homeostasis of lactate and pyruvate as alternative energy substrates. However, the mechanisms underlying podocyte metabolic adaptation in diabetes remain elusive. Therefore, we investigated the roles of CD147 and MCTs in maintaining energy homeostasis in immortalized human podocytes under HG conditions, using a specific, orally active CD147 inhibitor (AC-73). This study demonstrates, for the first time, that human podocytes express MCT1, MCT4, and CD147 at both the mRNA and protein levels. The downregulation of CD147 observed under HG conditions and following AC-73 treatment correlated with reduced MCT expression. These concurrent changes highlight a tight functional link between MCTs and CD147 that directly influences lactate and pyruvate transport, as well as glycolysis and ATP production. Collectively, these findings elucidate the role of the CD147/MCT system in podocyte energy metabolism under diabetic conditions, providing novel insights into therapeutic targets for mitigating DKD.

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S5.P1

RISK OF CARDIOVASCULAR DISEASES (CVD), INCLUDING THE PROGRESSION OF ATHEROSCLEROSIS, IN PATIENTS EXPOSED TO MICRO- AND NANOPLASTICS

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The increasing global use of plastics poses a potential threat to human health through exposure to micro- and nanoplastic particles (MNPs), which may adversely affect multiple organ systems, including the cardiovascular system. The aim of this study was to summarize current knowledge regarding the risk of cardiovascular diseases (CVD), including atherosclerosis progression, associated with MNP exposure. A literature review of English-language publications from 2019–2025 was conducted using databases including PubMed, Google Scholar, PRIMO, and Scopus. Keywords included: microplastic, nanoplastic, cardiovascular, atherosclerosis, blood vessels, and human health. A total of 73 publications met the inclusion criteria and were analyzed. The circulatory system plays a crucial role in the transport of MNPs. Through the formation of protein coronas, MNPs may promote thrombus formation and increase levels of pro-inflammatory cytokines (IL-1, IL-1β, IL-6, IL-8, IL-18, TNF-α), leading to oxidative stress and redox imbalance. MNP exposure is also associated with mitochondrial dysfunction, glutathione depletion, lipid peroxidation, RBC hemolysis, and altered coagulation parameters (PT, APTT). These mechanisms may contribute to atherosclerosis progression and increase the risk of cardiovascular events, including acute coronary syndrome and ischemic stroke.Endothelial dysfunction, inflammation, and oxidative stress appear to be key mechanisms linking MNP exposure with cardiovascular diseases and atherosclerosis progression. However, further clinical studies are required to confirm the direct impact of MNPs on the human circulatory system.

S5.P2

EFFECTS OF APELIN-13 ON HEMODYNAMIC ALTERATIONS AND OXIDATIVE STRESS MARKERS IN RAT MODEL OF CHRONIC DOXORUBICIN-INDUCED CARDIOTOXICITY

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Anthracyclines remain a cornerstone of cancer therapy; however, their clinical use is limited by cardiotoxicity. Doxorubicin (DOX)-induced cardiac injury is manifested by progressive hemodynamic and electrophysiological disturbances, which are thought to result, at least in part, from increased oxidative stress, recognized as one of the key mechanisms underlying DOX-induced cardiotoxicity. Apelin-13 (APE-13) has emerged as a potential cardioprotective peptide. However, its effects on both hemodynamic alterations and specific oxidative stress markers remain poorly understood. Therefore, the aim of this study was to evaluate the effects of APE-13 on chronic DOX-induced hemodynamic disturbances and to determine whether its cardioprotective action may be associated with modulation of oxidative stress. Thirty-eight male Sprague-Dawley rats were divided into three groups and received intraperitoneal DOX injections once weekly for four consecutive weeks. Continuous administration of NaCl or APE-13 at a dose of 40 μg/kg body weight/day was achieved using osmotic pumps for 28 days. Electrocardiography and transthoracic echocardiography were performed at baseline and at the end of the experiment. In addition, 8-hydroxy-2'-deoxyguanosine (8-OHdG) and malondialdehyde (MDA) levels were determined as markers of oxidative DNA damage and lipid peroxidation, respectively. APE-13 prevented DOX-induced QT and QTc prolongation and improved left ventricular systolic parameters. Although the DOX-induced increase in MDA levels was not attenuated by APE-13, the reduced 8-OHdG levels observed following DOX treatment returned to values comparable to those of the control group after APE-13 administration. These findings suggest that APE-13 exerts cardioprotective effects against chronic DOX-induced hemodynamic disturbances and may selectively modulate oxidative stress pathways involved in anthracycline-induced cardiotoxicity.

S5.P3

INCREASED LEVEL OF MIR-29B IN ACTIVELY SMOKING PATIENTS WITH CHRONIC CORONARY SYNDROME – A SMALL OBSERVATIONAL STUDY

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Smoking cigarettes is an important risk factor of cardiovascular disease which influences on different molecular pathways related to endothelial cells, extracellular matrix and inflammation. This influence also results in altered microRNAs expression profile. In this study 5 miRNAs selected by miRTarBase 7.0 as down-regulators of atheroprotective proteins: miR-92a (of KLF2), miR-10b (of KLF4), miR-126 (of MerTK), miR-98 (of IL-10) and miR-29b (of TGFβ) and their plasma levels were compared between active smokers (n = 12) and non-smokers (n = 46) with chronic coronary syndrome (CCS) diagnosed upon coronary angiography. It was revealed that in CCS patients active smokers presented significantly increased miR-29b plasma levels than non-smokers (median values: 1.202 *vs* 0.721, p = 0.01). Baseline characteristics revealed no significant differences in age, sex, BMI, presence of hypertension, dyslipidemia and atherosclerosis burden (assessed by Gensini Score) between the study groups. However, there were more men in active smoker group comparing to non-smoker group (83% *vs* 54%, p = 0.07). Therefore, despite the lack of statistical significance, the multivariate analysis ANCOVA (adjusted for sex) was undertaken and significant difference in miR-29b expression between CCS smokers and non-smokers also occurred (p = 0.04). No differences between expression of miR-92a, miR-10b, miR-126 and miR-29b were found between active smokers and non-smokers with CCS. In conclusion, active smokers with diagnosed chronic coronary syndrome presented significantly higher miR-29b expression. Due to low number of patients and lack of mechanistic analysis the importance and role of this finding requires further studies.

S5.P4

METABOLIC SYNDROME AND RS1544410 (BSMI) VDR POLYMORPHISM IN RELATION TO LIPID PROFILE AND ATHEROGENIC RISK IN OLDER ADULTS

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The study evaluated associations between metabolic syndrome (MetS), the rs1544410 (BsmI) vitamin D receptor (VDR) polymorphism, and cardiometabolic risk factors in 95 residents of a Nursing and Treatment Facility. MetS was present in 40% of participants and was associated with a less favorable cardiometabolic profile, including higher triglyceride (TG) levels, waist-to-hip ratio, lower HDL-C concentrations, and increased Atherogenic Index of Plasma (AIP) (all $p < 0.05$).

Two-way ANOVA revealed significant effects of MetS on TG, HDL-C, and NEFA levels. The rs1544410 polymorphism was associated with TG and NEFA concentrations, and significant MetS × genotype interactions were observed for both parameters. Multiple regression analysis demonstrated that the AA genotype was independently associated with higher AIP ($\beta = 0.143$, $p = 0.044$).

In conclusion, the rs1544410 (BsmI) VDR polymorphism may influence triglyceride and NEFA levels and contribute to variability in atherogenic risk, particularly in the presence of metabolic syndrome. These findings highlight a potential gene–metabolic interaction relevant to cardiovascular risk in older adults.

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S5.P5

FATTY ACIDS SYNTHESIZED BY ELOVL3 HAVE DIFFERENT EFFECTS ON THE METABOLISM AND CONTRACTILITY OF HL-1 CARDIOMYOCYTES

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Both dietary and endogenously synthesized very-long-chain saturated fatty acids (FAs) are associated with development of heart failure and cardiomyopathy. ELOVL3, a member of the elongation of very long-chain fatty acid (ELOVL) family, catalyzes the synthesis of C20–C24 saturated and monounsaturated FAs. Unlike most lipogenic genes, *Elovl3* expression increases during postnatal heart maturation, which coincides with a marked increase in C20:0 content. This suggests that ELOVL3-derived FAs may contribute to metabolic remodeling in cardiomyocytes. The aim of this study was therefore to determine the effects of arachidic acid (C20:0) and *cis*-11-eicosenoic acid (C20:1), two products of ELOVL3, on lipid metabolism and functional properties of HL-1 cardiomyocytes. Both FAs increased the number of lipid droplets, while only C20:1 increased their size within the cells. At the transcriptional level, C20:0 decreased the expression of genes involved in FA synthesis and storage, while C20:1 decreased the expression of FA desaturases, while simultaneously increasing the expression of genes involved in FA elongation. In addition, C20:1, unlike C20:0, increased intracellular FA uptake. Both FAs also had differential effects on the expression of genes associated with the functional phenotype of cardiomyocytes. C20:0 increased the expression of *Mef2c* and *Myl7*, while decreasing the expression of *Myl2*, without affecting *Myh6*. In contrast, C20:1 decreased the expression of *Mef2c*, *Myl7*, and *Myh6*, without affecting *Myl2*. Functional analysis showed that C20:0 did not affect cardiomyocyte contraction parameters such the amplitude of beating, rising time, falling time, and both rising and falling slopes. C20:1 caused a shortening of falling time, suggesting a slightly faster relaxation phase, while the remaining contractile parameters of HL-1 cells remained unchanged. These results showed that ELOVL3, through the synthesis of C20:0 and C20:1, may contribute to the metabolic regulation of cardiomyocytes.

S5.P6

MACHINE LEARNING-BASED INFORMATION FLOW ANALYSIS OF ECG SIGNALS FOR CONGENITAL LONG QT SYNDROME

M. OZIMEK[1], M. ANDRZEJEWSKA-OZIMEK, M. PETELCZYC[1]

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Congenital long QT syndrome (LQTS) is associated with prolonged ventricular repolarization and increased risk of arrhythmia. The aim of this study was to assess whether entropy-based information flow measures extracted from ECG recordings can distinguish patients with LQTS from healthy controls. Beat-to-beat time series of the RR interval (i.e. the time interval between two consecutive heartbeats), QT interval (time interval between the onset of the Q wave and the end of the T wave), diastolic interval (DI, time interval between the end of T wave and the onset of the Q wave), and R-wave and T-wave amplitudes were obtained from nighttime Holter ECG recordings. Directed information transfer was estimated using transfer entropy and conditional transfer entropy using the ITS Toolbox developed by L. Faes. A subset of entropy-based features was used as input to supervised machine learning models. Random forest (RF) and support vector machine (SVM) classifiers were evaluated using repeated patient-level train-test splits. The RF model achieved a mean accuracy and mean sensitivity of >95%, and mean specificity of >90%. The SVM model achieved a mean accuracy, mean sensitivity and mean specificity of >90%. SHapley Additive exPlanations (SHAP) analysis was applied to improve model interpretability and quantify the contribution of entropy-based features to the classification. Multiclass classification performance for discrimination between LQTS subtypes will also be discussed. The results suggest that entropy-based information flow analysis of ECG signals may support non-invasive risk assessment in LQTS.

S5.P7

MODEL-DEPENDENT RESPONSES TO CHRONIC TREATMENT WITH AMPK ACTIVATOR METFORMIN AND PERIHERAL CB_1 RECEPTOR ANTAGONIST JD5037 IN RAT PULMONARY HYPERTENSION

P. REMISZEWSKI, P. RYSZKIEWICZ, K. MINCZUK, A. PEDZINSKA-BETIUK, M. BARANOWSKA-KUCZKO, B. MALINOWSKA

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Pulmonary hypertension (PH) is a disease characterized by elevated pulmonary arterial pressure leading to right heart failure. The AMPK activator metformin (MET) and the peripheral cannabinoid CB_1 receptor antagonist JD5037 have previously shown protective effects in a rat model of early-stage monocrotaline (MCT)-induced PH. The current study compared the effects of MET and JD5037 administered alone or in combination in two rat models of severe PH. Experiments were performed in male Wistar rats with PH induced either by (1) a single injection of MCT (60 mg/kg, s.c.) or (2) a single injection of SU5416 (20 mg/kg, s.c.) followed by 3 weeks of hypoxia (10% O2) and subsequent normoxia. From day 7 after MCT or day 35 after SU5416administration, rats received JD5037 (3 mg/kg), MET (100 mg/kg), their combination, or its vehicles by oral gavage once a day for 17-21 days. Invasive and non-invasive hemodynamic assessments were conducted. Both models produced comparable disease severity, although treatment efficacy differed between them. Combined therapy tended to attenuate PH more effectively in the SuHx model than in the MCT model. Interestingly, JD5037 monotherapy showed beneficial effects in MCT-induced PH but tended to worsen PH outcomes in SuHx rats. These findings demonstrate that therapeutic responses are model-dependent and highlight the importance of using multiple PH models to improve translational relevance.

The work was financed by National Science Centre, project 2021/41/B/NZ7/03757

S5.P8

EFFECTS OF CHRONIC ADMINISTRATION OF PERIPHERAL HYBRID CANNABINOID CB_1R AND INOS ANTAGONIST MRI-1867 ON RIGHT VENTRICULAR CONTRACTILE FUNCTION IN RAT PULMONARY HYPERTENSION

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Pulmonary hypertension (PH) is a characterized by elevated pulmonary artery pressure and right ventricular (RV) failure. We demonstrated beneficial effects of the peripheral cannabinoid CB_1 receptor (CB_1R) antagonist JD5037 in the monocrotaline (MCT)-induced PH. Selective inhibition of inducible nitric oxide synthase (iNOS) also exert cardioprotective effects in PH. Single-molecule dual-target strategies may offer pharmacological advantages over combination therapies based on separately administered selective ligands. Thus, the aim of the present study was to compare the effects of chronic oral treatment (17 days, starting on day 8 after PH induction with MCT) with the dual CB_1R/iNOS inhibitor (S)-MRI-1867 (10 mg/kg) and combined selective CB_1R and iNOS blockade (JD5037, 3 mg/kg + 1400W, 10 mg/kg) on contractile function of RV papillary muscles isolated from PH rats and normotensive controls. Although both treatments reduced RV systolic pressure (~30%), RV weight (~25%), and Fulton index (~26%), only treatment with (S)-MRI-1867 restored impaired β-adrenergic responsiveness in isolated papillary muscles, leading to a marked increase in isoprenaline-induced maximal positive inotropic response (E_{max}) of approximately 94% (expressed as % of basal values) compared with untreated PH rats. These findings indicate that single-molecule dual-target therapy provides superior functional benefits compared with combined selective CB_1R and iNOS inhibition, particularly in restoring β-adrenergic signalling in the PH-affected RV.

This work was supported by the National Science Centre, Poland (2021/41/B/NZ7/03757).

SESSION VI

FUNCTION OF BLOOD. HEMOSTASIS

Thursday (September 24th, 14.00 - 16.45) Room A

Chairman: Professor Boguslaw Machalinski
(Department of Hematology and Transplantology,
Pomeranian Medical University in Szczecin, Poland)

Vice-Chairman: Associate Professor Joanna Bartkowiak-Wieczorek
(Department of Physiology, Karol Marcinkowski University in Poznan, Poland)

DETAILED SESSION VI SCHEDULE

Opening lecture Thursday (September 24th, 14.00 – 14.40)

S6.L1 DISORDERS OF THROMBOPOIESIS: BRIDGING PHYSIOPATHOLOGY AND CLINICAL HEMATOLOGY. **E. Paczkowska** (Department of Hematology and Transplantology, Pomeranian Medical University in Szczecin. Poland).

Oral presentations Thursday (September 24th, 14.40 – 16.30)

S6.L2 *14.40 – 14.55* ERYTHROCYTE FUNCTIONAL PROPERTIES AND OXIDATIVE STRESS MARKERS IN CHILDREN VERSUS YOUNG ADULTS. **J. Radosinska[1], T. Jasenovec[1], N. Vrbjar[2]** ([1]Institute of Physiology, Faculty of Medicine, Comenius University in Bratislava, Bratislava, Slovakia; [2]Centre of Experimental Medicine, Institute for Heart Research, Slovak Academy of Sciences, Bratislava, Slovakia).

S6.L3 *14.55 – 15.10* BIODEGRADABLE HSA-HA NANOCAPSULES CO-DELIVERING ANGIOSTATIN AND THROMBOSPONDIN-1 AS A MULTITARGET STRATEGY AGAINST THE MULTIPLE MYELOMA MICROENVIRONMENT. **K. Luczkowska, M. Dabkowska, P. Stodolak, A. Szatanik, K. Pukacka, E. Pius-Sadowska, J. Machalinski, B. Machalinski.** (Department of General Pathology, Pomeranian Medical University, Szczecin, Poland).

S6.L4 *15.10 – 15.25* ANTIANGIOGENIC NANOTHERAPY: HARNESSING HUMAN SERUM ALBUMIN TO DELIVER ACTIVE ANTIANGIOGENIC PROTEINS. **I. Stukan[1], M. Dabkowska[1], A. Mietelska-Porowska[2], U. Wojda[2], A. Marshalak[1], B. Machalinski[1]** ([1]Pomeranian Medical University in Szczecin, Szczecin, Poland; [2]Nencki Institute of Experimental Biology PAS, Warsaw, Poland).

15.30 – 16.00 Coffee break

S6.L5 *16.00 – 16.15* ANALYSIS OF CHANGES IN VARIANT ALLELE FREQUENCY (VAF) OF MUTATED JAK2 V617F, NEUTROPHIL-TO-LIMPHOCYTE RATIO AND MIRNA EXPRESSION IN PATIENTS WITH POLYCYTHEMIA VERA DURING TREATMENT WITH ROPEGIFN-A2B. **M. Janowski[1], K. Luczkowska[1], A. Paczkowska[1], A. Dabrowska-Zimon[2], D. Zdrojewska[2], B. Machalinski[1,2], E. Paczkowska[1]** ([1]Department of General Pathology, Pomeranian Medical University, Szczecin, Poland; [2]Department of Hematology and Bone Marrow Transplantation, Pomeranian Medical University, Szczecin, Poland).

S6.L6 *16.15 – 16.30* VITAMIN D3 AND K2 IN MULTIPLE MYELOMA – A NOVEL VIEW ON NORMOXIC AND HYPOXIC MODULATION OF CELL BIOLOGY. **K. Luczkowska, P. Stodolak, P. Kulig, B. Machalinski** (Department of General Pathology, Pomeranian Medical University in Szczecin, Szczecin, Poland).

Poster Session Thursday (September 24th 16.30 – 16.45)

S6.P1 EFFECT OF PLASMA-TREATED ALGINATE HYDROGELS ON PORCINE LEUKOCYTES *IN VITRO*, POTENTIAL OF COLD ATMOSPHERIC PLASMA IN IMMUNE MODULATION**. J. Wessely-Szponder[1], G. Fornabaio[2,3], D. Nguyen Ngoc[1], B. Drzewiecka[1], C. Canal[2,3,4]** ([1]Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences, Lublin, Poland, [2]PlasmaMedLab-Biomaterials, Biomechanics and Tissue Engineering (BBT) Group, Department of Materials Science and Engineering and Institute for Research and Innovation in Health (IRIS), University Politecnica de Catalunya - BarcelonaTech (UPC), Barcelona, Spain; [3]Barcelona Research Centre in Multiscale Science and Engineering (CCEM), UPC, Barcelona, Spain; [4]Network Center for Biomedical Research in Bioengineering, Biomaterials and Nanomedicine (CIBER-BBN), Carlos III Health Institute (ISCIII)), Madrid, Spain.

Presentation provided by the Sponsor Thursday (September 24th 17.45 – 18.45)

S6.L1

DISORDERS OF THROMBOPOIESIS: BRIDGING PHYSIOPATHOLOGY AND CLINICAL HEMATOLOGY

E. PACZKOWSKA

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Megakaryopoiesis is an effectively coordinated process that sustains platelet production in the bone marrow. Platelets play an important role in process of blood coagulation. Preserving the physiological equilibrium of hemostasis, thereby preventing both bleeding and thrombosis, is essential for maintaining human health. Recent advances in molecular hematology have significantly expanded our understanding of the genetic and signaling mechanisms that regulate megakaryocyte proliferation, differentiation and function. Their dysregulation in hematologic disease is also a better-known process. This lecture will provide an overview of the molecular pathways changing megakaryocyte and platelet functioning, especially on driver mutations that promote aberrant proliferation and clonal expansion in myeloproliferative neoplasms. The interplay between oncogenic signaling, transcriptional regulation, and differentiation will be discussed in the context of altered platelet biology. These changes contribute to platelet dysfunction, thromboinflammation, and the risk of arterial and venous thrombosis. Finally, their implications for disease pathogenesis, risk assessment, and the development of therapeutic strategies will be discussed.

S6.L2

ERYTHROCYTE FUNCTIONAL PROPERTIES AND OXIDATIVE STRESS MARKERS IN CHILDREN VERSUS YOUNG ADULTS

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Human aging is characterized by a decline in hemorheological properties, Na,K-ATPase activity, and nitric oxide (NO) bioavailability. However, research has primarily focused on the age-related deterioration of erythrocyte properties in older individuals. This study investigated erythrocyte mechanical properties (deformability), nitric oxide (NO) production, Na,K-ATPase kinetics, and oxidative stress markers in healthy children (mean age 4.7 years) and young adults (mean age 22.2 years). Deformability was evaluated using erythrocyte filterability and ektacytometry under varying osmotic conditions, NO production was assessed via DAF-fluorescence, and Na,K-ATPase activity was measured at different NaCl concentrations. While enzyme activity increased with rising NaCl levels in both groups, the magnitude of this increase was lower in adults, coinciding with higher mean cell volume and reduced osmotic resistance. Additionally, NO production was decreased in adults, although erythrocyte deformability remained unchanged. Adult erythrocytes also showed a lower reduced-to-oxidized glutathione ratio and higher protein oxidation compared to those from children. These findings indicate that erythrocyte properties undergo measurable changes already in early adulthood, not only in old age; specifically, reduced Na,K-ATPase activity in young adults may contribute to increased cell volume and reduced osmotic resistance, potentially predisposing erythrocytes to further functional decline later in life. These pilot results may assist researchers in designing studies involving children and young adults by highlighting age-sensitive and age-independent erythrocyte parameters and demonstrating that detectable alterations in erythrocytes occur early even in healthy individuals.

Funds: APVV-24-0225, APVV-22-0154, and VEGA 1/0634/25.

S6.L3

BIODEGRADABLE HSA-HA NANOCAPSULES CO-DELIVERING ANGIOSTATIN AND THROMBOSPONDIN-1 AS A MULTITARGET STRATEGY AGAINST THE MULTIPLE MYELOMA MICROENVIRONMENT

K. LUCZKOWSKA, M. DABKOWSKA, P. STODOLAK, A. SZATANIK, K. PUKACKA, E. PIUS-SADOWSKA, J. MACHALINSKI, B. MACHALINSKI

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Background: Multiple myeloma (MM) is a plasma cell malignancy strongly influenced by interactions within the bone marrow niche, including angiogenesis, extracellular matrix remodeling, and chemokine signaling, which contribute to disease progression and therapeutic resistance. Therefore, strategies targeting both MM cells and the tumor microenvironment are highly needed. Aim: This study aimed to develop and evaluate multifunctional HSA-HA nanocapsules for co-delivery of angiostatin and thrombospondin-1 and assess their effects on MM cell proliferation and tumor–microenvironment-related pathways in vitro. Methods: U266 and MM1S MM cell lines were exposed to six treatment variants, including HSA-based nanoparticles, hyaluronic acid-coated nanoparticles, free antiangiogenic proteins, and fully formulated HSA-HA nanocapsules containing angiostatin and thrombospondin-1 (24 ng/mL each). Molecular effects were assessed using Luminex xMAP, automated capillary western blot (Jess), mRNA sequencing, and alamarBlue assay. Statistical analyses included one-way ANOVA with false discovery rate correction and Tukey post hoc test ($p.adj<0.05$). Results: Fully formulated HSA-HA nanocapsules showed the strongest antimyeloma activity, reducing viability by 63% in U266 ($p.adj<0.0001$) and 35% in MM1S cells ($p.adj=0.0445$). Treatment suppressed angiogenesis- and matrix remodeling-related factors, including VEGFA, MMP9, and MMP13, and altered chemokine signaling involving CXCL12, CXCL10, CXCL16, CXCL2, and CCL4. Transcriptomic analysis indicated modulation of pathways associated with proliferation, immune signaling, stress response, and tumor–microenvironment interactions. The HSA-HA carrier itself may additionally support intracellular delivery and CD44-mediated targeting. Conclusion: HSA-HA biodegradable nanocapsules co-delivering angiostatin and thrombospondin-1 demonstrated promising antimyeloma activity and may modulate key biological processes involved in MM progression, suggesting their potential as a multitarget therapeutic strategy requiring further mechanistic and in vivo validation.

S6.L4

ANTIANGIOGENIC NANOTHERAPY: HARNESSING HUMAN SERUM ALBUMIN TO DELIVER ACTIVE ANTIANGIOGENIC PROTEINS

I. STUKAN[1], M. DABKOWSKA[1], A. MIETELSKA-POROWSKA[2], U. WOJDA[2], A. MARSHALAK[1], B. MACHALINSKI[1]

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Pathological angiogenesis contributes to tumor progression and other vascular disorders. Human serum albumin (HSA), the most abundant plasma protein, may provide a physiologically compatible carrier due to its prolonged circulation time and interaction with endothelial albumin-handling pathways. We hypothesized that albumin-based nanocarriers may partially preserve native albumin-associated transport properties, thereby improving intravascular persistence and delivery of antiangiogenic cargo. Using complementary in vitro and in vivo models, we demonstrated that HSA-based nanocarriers interact with endothelial cells, retain biological activity and persist in circulation after intravenous administration. Fluorescent HSA nanoparticles and hyaluronic acid-coated HSA nanoparticles were internalized by both human umbilical vein endothelial cells (HUVEC) and bone marrow endothelial cells (BMEC-1), with distinct uptake profiles between the two endothelial models. BMEC-1 cells showed stronger FcRn-associated fluorescence, suggesting cell-type dependent engagement of albumin-handling pathways. Angiostatin-loaded formulations preserved functional antiangiogenic activity, affecting multiple parameters of endothelial tube formation in Matrigel assay, while no overt cytotoxicity toward endothelial cells was observed. Pre-incubation with excess free HSA significantly altered nanoparticle activity, supporting the involvement of physiological albumin-related mechanisms. Importantly, after a single intravenous administration in NOD-SCID-gamma mice, albumin nanocarriers remained detectable in circulation for up to 72 h, indicating sustained intravascular persistence. These findings suggest that harnessing these endogenous albumin-related pathways may support the development of longer-acting intravenous therapies targeting pathological angiogenesis.

S6.L5

ANALYSIS OF CHANGES IN VARIANT ALLELE FREQUENCY (VAF) OF MUTATED JAK2 V617F, NEUTROPHIL-TO-LIMPHOCYTE RATIO AND MIRNA EXPRESSION IN PATIENTS WITH POLYCYTHEMIA VERA DURING TREATMENT WITH ROPEGIFN-A2B

M. JANOWSKI[1], K. LUCZKOWSKA[1], A. PACZKOWSKA[1], A. DABROWSKA-ZIMON[2], D. ZDROJEWSKA[2], B. MACHALINSKI[1,2], E. PACZKOWSKA[1]

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Polycythemia Vera (PV) is a myeloproliferative neoplasm caused by a somatic JAK2 V617F mutation in a hematopoietic stem cell, leading to clonal abnormal hematopoiesis. Novel treatment with ropeginterferon alfa-2b (RopegINF-a1b) has been shown to reduce the allelic burden of the JAK2 mutation (variant allele frequency, VAF). The treatment preferentially suppresses neutrophils while preserving lymphocytes, leading to a significant reduction in the neutrophil-to-lymphocyte ratio (NLR). This reduction strongly correlates with suppression of the JAK2 VAF. An elevated NLR is associated with a higher VAF, an increased risk of venous thrombosis, and more aggressive or proliferative disease. Research efforts have increasingly focused on epigenetic mechanisms influencing gene expression. Several miRNAs have been identified as upregulated in PV patients compared with healthy volunteers. However, the previously published studies did not take into account the response to treatment. Here, we report changes in the expression of miRNAs in circulating leucocytes and analysis of correlation with VAF during subsequent months of treatment with RopegIFN-a2b. Material and methods: In this study, we recruited 15 patients with PV who started new treatment with RoperINF-a2b and 15 healthy volunteers. Peripheral blood samples were obtained from patients at different time points: i) at start of treatment, ii) after 3 and 12 months of treatment; and once from control group. Response to RopegINF-a1b treatment, required medication dose, and VAF level were assessed as well as blood parameters including neutrophil-to-lymphocyte ratio and hematocrit. RNA was isolated from leucocytes. Selected miRNAs in peripheral blood leucocytes were assessed using molecular methods. We compared expression levels of miRNAs at different time point to control group and to treatment start point. Results: All patients achieved disease control following interferon therapy. Approximately half of the patients required a dose of 100 µg, while the other half required 250 µg of the drug every two weeks. We observed at 3-month of treatment, significantly decreased VAF in majority of patients. At this time point, the mean reduction in VAF was 50%. We also observed changes in expression miRNAs at different time points. Conclusions: We found significant decreased VAF level in PV patients treated with RopegINF-a1b as well as differences in peripheral blood leucocytes. The observed changes seem to correlate with treatment response to RopegINF-a2b therapy. In our opinion, the changes observed after 3 months of treatment were particularly significant. These results suggests effectiveness of RopegINF-a1b treatment in decreasing JAK2 mutated allelic burden. The findings related to miRNA expression warrant further investigation. Considering the simplicity of the leukocyte-to-lymphocyte ratio, its use in the initial assessment of treatment response may have practical implications for patient monitoring in clinical practice. However, our observations require further in-depth studies at later stages of patients' treatment.

S6.L6

VITAMIN D3 AND K2 IN MULTIPLE MYELOMA – A NOVEL VIEW ON NORMOXIC AND HYPOXIC MODULATION OF CELL BIOLOGY

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Vitamin D3 and vitamin K2 are increasingly recognized for their immunomodulatory and potential anti-cancer properties, which may be particularly relevant in multiple myeloma (MM), where the tumor microenvironment strongly influences disease progression and therapeutic response. This study aimed to evaluate the molecular effects of 25-hydroxyvitamin D (25(OH)D) and menaquinone-7 (MK-7) in MM cell lines and compare responses under standard and bone marrow-like hypoxic conditions. U266 and MM1S MM cell lines were exposed for 48 h to 10 nM concentrations of 25(OH)D and MK-7 under normoxic (21% O_2, 5% CO_2) and hypoxic bone marrow-mimicking conditions (6% O_2, 5% CO_2) using the Avatar culture system. Cytotoxicity analysis confirmed non-toxic effects on fibroblasts. Transcriptomic (mRNA sequencing) and epigenetic (DNA methylation profiling) analyses were performed, followed by functional enrichment using DAVID. Distinct molecular responses depended on oxygen availability. Under normoxic conditions, treatment predominantly affected VEGF receptor signaling, intracellular signaling, and cytokine-related pathways, whereas hypoxia was associated with stronger regulation of cell cycle, DNA repair, and mitotic processes. Regulation of transcription by RNA polymerase II was suppressed under normoxia but activated during hypoxia. Hypermethylation of the MCOLN3 promoter under hypoxia was associated with reduced gene expression ($p<0.05$). Additionally, 312 significant correlations between DNA methylation and gene expression were identified under normoxic conditions, suggesting extensive epigenetic-transcriptional interactions. Vitamin D3 and MK-7 may influence molecular and epigenetic mechanisms in MM cells, while oxygen conditions strongly modulate treatment responses. These findings highlight the importance of physiologically relevant culture systems for improving translational relevance in MM research.

S6.P1

EFFECT OF PLASMA-TREATED ALGINATE HYDROGELS ON PORCINE LEUKOCYTES *IN VITRO*, POTENTIAL OF COLD ATMOSPHERIC PLASMA IN IMMUNE MODULATION

J. WESSELY-SZPONDER[1], G. FORNABAIO[2,3], D. NGUYEN NGOC[1], B. DRZEWIECKA[1], C. CANAL[2,3,4]

[1]Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences, Lublin, Poland, [2]PlasmaMedLab-Biomaterials, Biomechanics and Tissue Engineering (BBT) Group, Department of Materials Science and Engineering and Institute for Research and Innovation in Health (IRIS), University Politecnica de Catalunya - BarcelonaTech (UPC), Barcelona, Spain; [3]Barcelona Research Centre in Multiscale Science and Engineering (CCEM), UPC, Barcelona, Spain; [4]Network Center for Biomedical Research in Bioengineering, Biomaterials and Nanomedicine (CIBER-BBN), Carlos III Health Institute (ISCIII), Madrid, Spain; joanna.wessely@up.edu.pl

Cold atmospheric plasma (CAP) has gained significant attention in recent years as an innovative therapeutic approach with broad applications in various fields of medicine and veterinary medicine. CAP promotes tissue repair, offering promising applications in the treatment of chronic, hard-to-heal wounds and other skin disorders in humans and animals. Despite its widespread therapeutic use, some mechanisms responsible for CAP interactions with host cells remain unclear. In this study, we analyzed the response of leukocytes (blood-derived monocytes and neutrophils) isolated from porcine blood to stimulation with plasma-treated alginate hydrogels (PTH), based on metabolic activity, reactive oxygen and nitrogen species (RONS) generation, and neutrophil degranulation. The obtained results showed that PTH induces a controlled increase in nitrite and superoxide generation in both monocyte and neutrophil cultures, particularly at higher concentrations, without lytic cell death or significant loss of viability. We observed a slight decrease in neutrophil metabolic activity without increased degranulation, whereas monocyte metabolism remained unchanged. Our results indicate that therapeutic doses of CAP induce moderate oxidative stress, which can activate leukocytes while maintaining their functional stability. These results may pave the way for a more detailed characterization of the effects of CAP and PTH on immune modulation.

SESSION VII

PHYSIOLOGY OF THE RESPIRATORY SYSTEM

Thursday (September 24th, 14.00 – 16.30) Room B

Chairman: Associate Professor Bartlomiej Paleczny
(Department of Physiology and Pathophysiology, Faculty of Medicine,
Wroclaw Medical University, Wroclaw, Poland)

Vice-Chairman: Professor Jana Plekova
(Medical Education Support Center, Jessenius Faculty of Medicine,
Comenius University in Bratislava, Bratislava, Slovakia)

Vice-Chairman: Professor Andrea Calkovska
(Department of Physiology, Jessenius Faculty of Medicine,
Comenius University in Bratislava, Bratislava, Slovakia)

DETAILED SESSION VII SCHEDULE

Opening lectures Thursday (September 24th 14.00 – 15.00)

S7.L1. *14.00 – 14.30* (*including 10 min. discussion*) TRANSLATIONAL BIAS IN COUGH RESEARCH: INSIGHTS AND LIMITATIONS FROM ANIMAL MODELS. **J. Plevkova[1], T. Buday[1], Z. Biringerova[2], D. Mokra[3]** ([1]Medical Education Support Center, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, Martin, Slovakia; [2]Clinic of Anesthesiology and Intensive Medicine, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, Martin, Slovakia; [3]Department of Physiology, Jessenius Faculty of Medicine in Martin, Comenius University in Bratislava, Martin, Slovakia).

S7.L2 *14.30 – 15.00* (*including 10 min. discussion*) UNDERSTANDING THE PHYSIOLOGICAL ROLE OF PULMONARY SURFACTANT: THEN AND NOW. **A. Calkovska[1], M. Kolomaznik[2], P. Kosutova[2], J. Hanusrichterova[2], P. Mikolka[1]** ([1]Department od Physiology, Jessenius Faculty of Medicine, Comenius University, Martin, Slovakia; [2]Biomedical Centre Martin, Jessenius Faculty of Medicine, Comenius University, Martin, Slovakia).

Oral presentations Thursday (September 24th 15.00 – 16.30)

S7.L3 *15.00 – 15.15* RESPIRATORY FUNCTION AND EXERCISE CAPACITY IN JUNIOR ATHLETES ACROSS SPORT DISCIPLINES: CROSS SECTIONAL AND LONGITUDINAL ANALYSES. **S. Siatkowski, M. Sikora, O. Kwaskiewicz, A. Zebrowska** [1]Department of Clinical Physiology and Pathophysiology, Institute of Sport Sciences, Academy of Physical Education, Katowice, Poland).

S7.L4 *15.15 – 15.30* ASSOCIATION BETWEEN BONE MINERALIZATION AND RESPIRATORY MECHANICS IN HEALTHY OLDER WOMEN. **P. Gamza, M. Zielinska, M. Sikora, O. Lakomy** (Department of Clinical Physiology and Pathophysiology, Institute of Sport Sciences, Academy of Physical Education, Katowice, Poland).

15.30 – 16.00 Coffee break

S7.L5 *16.00 – 16.15* VENTILATORY RESPONSE TO HYPERCAPNIA IN A TRANSGENIC MOUSE MODEL OF ALZHEIMER'S DISEASE AFTER TREATMENT WITH FLUOXETINE AND REBOXETINE. **K. Andrzejewski[1], A. Wrzesien[1], M. Jampolska[1], M. Zaremba[2], I. Joniec-Maciejak[2], K. Kaczynska[1]** ([1]Department of Respiration Physiology, Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland; [2]Department of Experimental and Clinical Pharmacology, Centre for Preclinical Research, Medical University of Warsaw, Warsaw, Poland).

S7.L6 *16.15 – 16.30* TRANSIENT RESPIRATORY TESTS FOR THE EVALUATION OF PERIPHERAL CHEMOSENSITIVITY IN PRETERM-BORN ADULTS**. F. Jablonski[1], D. Bieganska[1], B. Ponikowska[1], B. Paleczny[1]** ([1]Department of Physiology and Pathophysiology, Wroclaw Medical University, Wroclaw, Poland).

Presentation provided by the Sponsor Thursday (September 24th 17.45 – 18.45)

S7.L1

TRANSLATIONAL BIAS IN COUGH RESEARCH: INSIGHTS AND LIMITATIONS FROM ANIMAL MODELS

J. PLEVKOVA[1], T. BUDAY[1], Z. BIRINGEROVA[2], D. MOKRA[3]

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Cough is a vital defensive reflex and a common clinical symptom, yet effective antitussive therapies remain limited. Animal models have been indispensable in elucidating the neurobiology and pharmacology of cough; however, significant translational gaps persist between preclinical findings and clinical outcomes. This abstract examines the issue of translational bias in cough research, focusing on insights derived from commonly used animal models. While these models have contributed to understanding airway sensory pathways, ion channel function, and neurotransmitter systems (e.g., TRP channels, purinergic signaling), important species-specific differences in airway innervation, receptor expression, and cough reflex regulation limit direct extrapolation to humans. For example, rodents such as mice lack a robust cough reflex, whereas guinea pigs more closely mimic human cough responses but still fail to fully replicate chronic cough phenotypes. Moreover, experimental conditions—such as anesthesia, acute stimulus models, and controlled environments—often fail to capture the complexity of human disease, including chronic inflammation, comorbidities, and central sensitization. Translational bias is further compounded by the reliance on endpoint measures (e.g., cough frequency) that may not reflect patient-relevant outcomes, as well as by publication bias favoring positive preclinical results. These limitations contribute to the high attrition rate of candidate antitussive therapies in clinical trials. Addressing these challenges requires a more integrative approach, including the development of refined animal models that better replicate chronic cough mechanisms, the incorporation of human-based experimental systems, and improved alignment between preclinical endpoints and clinical outcomes. Recognizing and mitigating translational bias is essential to accelerate the development of effective therapies for cough.

This work was supported by the Slovak Research and Development Agency under the Contract no. APVV-22-0052.

S7.L2

UNDERSTANDING THE PHYSIOLOGICAL ROLE OF PULMONARY SURFACTANT: THEN AND NOW

A. CALKOVSKA[1], M. KOLOMAZNIK[2], P. KOSUTOVA[2], J. HANUSRICHTEROVA[2], P. MIKOLKA[1]

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Pulmonary surfactant (PS) stabilizes the alveoli and small airways thereby prevents the lungs to collapse at the end-expiration. The importance of PS is not limited to reducing lung surface tension. In recent years, several studies focus on less perceived properties of surfactant components related to its role in the upper airways, or anti-inflammatory features and the relaxing effect on the airway smooth muscle. Surfactant research benefits from combination of numerous models, whose use will provide a relatively complex picture on the role of pulmonary surfactant. In series of studies, we addressed the effect of bacterial lipopolysaccharide (LPS) on the respiratory system, its interaction with PS and new treatment options for acute respiratory distress syndrome (ARDS). The most important results include the use of clinically relevant animal models to test and confirm the suitability of exogenous surfactant therapy, verified combinations with other substances and pointed out the risks[1]. The experiments focused on the relaxing effect of PS on the airway smooth muscle confirmed the involvement of leukotriene and histamine receptors in the contractile mechanisms of the airways after LPS exposure. The atomic force microscopy of airway smooth muscle cells revealed that the EP4 receptor for the relaxing prostaglandin PGE_2 may be involved in the mechanism of airway smooth muscle relaxation by surfactant through the interaction of the surfactant with the airway epithelium[2]. Moreover, study of the properties of endogenous surfactant allows to develop the preparations resistant to inhibition e.g.by plasma proteins[3] to treat surfactant deficiency in neonatology and other medical disciplines. Clearly, the role of PS extends far beyond the alveoli. Understanding the properties of the components of this complex substance and their interactions is of interest to physiology and essential to clinical medicine.

Supported by project VEGA 1/0097/23.

S7.L3

RESPIRATORY FUNCTION AND EXERCISE CAPACITY IN JUNIOR ATHLETES ACROSS SPORT DISCIPLINES: CROSS SECTIONAL AND LONGITUDINAL ANALYSES

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Background. The pulmonary system contributes to athletic performance through ventilatory capacity, airway mechanics and gas exchange. This study aimed to characterise respiratory function in junior athletes across sport disciplines, examine associations between pulmonary function and aerobic capacity, and evaluate three-year longitudinal changes in spirometric and oscillometric parameters. Methods: Spirometry, single- and multi-frequency forced oscillometry (8 Hz; 5–19 Hz), graded exercise test (aerobic capacity and lactate-threshold) were analysed. Group differences were tested with Welch t-tests, one-way ANOVA/Kruskal-Wallis tests, paired t-tests/Wilcoxon signed-rank tests and one-way repeated-measures ANOVA/Friedman tests; effect sizes were expressed as Cohen d, η^2 or partial η^2. Multiple linear regression was used to derive prediction equations and to model performance outcomes. Results: Sport disciplines did not reach significance for any spirometric or oscillometric variable. Athlete-specific multiple-regression equations explained 72–75% of the variance in VC, FVC and FEV1, 53% in PEF and 11% in FEV1/FVC. Spirometric volumes and flows were strongly correlated with VEmax ($r \sim 0.6–0.7$); airway resistance (R) was inversely associated with aerobic performance ($r \sim -0.25$). The three-year longitudinal subsample showed marked positive increase in FVC, FEV1, FEV1/FVC and PEF ($\eta^2p = 0.38–0.67$). Conclusions. The data confirm pronounced sex dimorphism in pulmonary function in junior athletes, justify the development of sport-specific reference equations, and demonstrate that both lung volume and airway mechanics are meaningful, statistically distinct contributors to performance.

S7.L4

ASSOCIATION BETWEEN BONE MINERALIZATION AND RESPIRATORY MECHANICS IN HEALTHY OLDER WOMEN

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Reduced bone mineral density (BMD) may affect thoracic mechanics and respiratory function, although early changes are often undetectable by standard spirometry. This study assessed relationships between bone status and respiratory mechanics in clinically healthy women over 60 years using the forced oscillation technique (FOT), spirometry, and plethysmography. Forty-seven women were divided into a control group with normal BMD ($n = 23$) and an osteopenia group ($n = 24$). Bone status was assessed by dual-energy X-ray absorptiometry (DXA), including BMD, bone mineral content (BMC), and T-score. Pulmonary function was evaluated using FOT, spirometry, and plethysmography. Associations were analyzed using Pearson correlations and linear regression. In the osteopenia group, trunk BMC correlated negatively with FOT reactance parameters: X5exp ($r = -0.88$, $p = 0.001$) and X11exp ($r = -0.92$, $p < 0.001$). Similar relationships were found for other DXA-derived parameters. FEV_1/FVC% correlated positively with trunk and spine BMC ($r = 0.56$, $p < 0.05$), whereas other spirometric indices and lung volumes showed no significant associations. In controls, spine and total BMD correlated with FEV_1/FVC% ($r = 0.59$, $p = 0.010$; $r = 0.58$, $p = 0.011$, respectively), RV%, and TLC%, while no significant correlations were observed for FOT parameters. Reduced bone mineralization in women over 60 may be associated with subtle alterations in passive respiratory mechanics, mainly reflected in oscillometric parameters. FOT may provide greater sensitivity for detecting early respiratory changes associated with bone status.

S7.L5

VENTILATORY RESPONSE TO HYPERCAPNIA IN A TRANSGENIC MOUSE MODEL OF ALZHEIMER'S DISEASE AFTER TREATMENT WITH FLUOXETINE AND REBOXETINE

K. ANDRZEJEWSKI[1], A. WRZESIEN[1], M. JAMPOLSKA[1], M. ZAREMBA[2], I. JONIEC-MACIEJAK[2], K. KACZYNSKA[1]

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Patients suffering from Alzheimer's disease (AD) display respiratory problems, which are still not fully comprehended. In our study we investigated hypercapnic ventilatory response (HCVR) in transgenic mouse model of AD (AβPP V717I) overexpressing AβPP. To reverse breathing changes, we used fluoxetine, a selective serotonin (5-HT) reuptake inhibitor, and reboxetine, a specific noradrenaline (NA) reuptake inhibitor, increasing the level of 5-HT and NA, respectively in the synaptic cleft. The ventilatory tests were performed with whole body plethysmography. Afterwards: hippocampus, brainstems and prefrontal cortex were collected to HPLC analysis of monoamines concentration. Normocapnic minute ventilation (MV) did not differ between APP+ and APP- mice. Only tidal volume (TV) showed higher values in AD mice. The ventilatory response to hypercapnia in APP+ mice was increased, as expressed by increased TV and MV. Fluoxetine treatment depressed resting ventilation and hypercapnic MV in APP+ and APP- mice restoring HCVR in APP+ mice to value represented by untreated sham mice. Reboxetine reduced MV in normoxia and during hypercapnia exposure in both groups, eliminating the significant difference between APP- and APP+ mice. HPLC analysis exhibited increased level of serotonin and its metabolite 5-HIAA in hippocampus of APP+ mice. The increased HCVR in APP+ mice indicate alterations in CO_2 chemoreception in brainstem respiratory control region. The compensatory effects of the drugs used suggest that 5-HT and NA neurotransmission play an important role in breathing regulation in AD. Fluoxetine and reboxetine, apart from their antidepressant effects may be an effective therapeutic strategy for treating breathing disorders in AD.

The study was funded by the National Science Centre 2022/45/B/00221

S7.L6

TRANSIENT RESPIRATORY TESTS FOR THE EVALUATION OF PERIPHERAL CHEMOSENSITIVITY IN PRETERM-BORN ADULTS

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Introduction: Preterm birth (<37 weeks of gestation) is associated with lifelong respiratory, cardiovascular and neural repercussions. Data on peripheral chemoreceptor (PCH) function are scarce and inconsistent, however, possibly due to methodological matters. Here, we used transient tests, which have been hypothesized to reveal the "pure" PCH response more accurately than sustained hypoxia protocols employed in previous studies. Methods: PCH sensitivity was assessed using two methods that relied on short-lasting manipulations of breathing air (breathing pure nitrogen or 13% CO_2 in air for transient hypoxia and the single-breath CO_2 method, respectively). Ventilatory responses: hypoxic (HVR, for transient hypoxia, [L/min/SpO_2%]), and hypercapnic (HCVR, for single-breath CO_2, [L/min/mmHg]) were calculated by linking either SpO_2 fall or end-tidal CO_2 rise to the corresponding increase in instantaneous minute ventilation. Hemodynamic responses to both tests (rise in heart rate and mean blood pressure, fall in total peripheral resistance) were calculated analogously. Results: 55 adults have been recruited prospectively to Preterm- (N=24, age: 23±3 y, BMI: 23.9±3.2 kg/m^2, 11 males), and Term-group (N=31, age: 24±5 y, Body Mass Index: 22.9±3.1 kg/m^2, 13 males). Preterm-born adults tended to have lower HVR (median with Q1 and Q3: 0.18 [0.12-0.25] vs. 0.24 [0.15-0.31] L/min/SpO_2%, p=0.09) and higher hypoxic HR-slope (mean±SD: 0.80±0.29 vs. 0.68±0.19 bpm/SpO_2%, p=0.07). Neither HCVR (Preterm vs. Term, median with Q1 and Q3: 0.10 [0.05-0.13] vs. 0.12 [0.09-0.16] L/min/mmHg, p=0.15) nor other hemodynamic parameters for hypoxic and hypercapnic tests differed significantly between the two groups (all p>0.25). Conclusions: Although preterm- and term-born adults did not differ significantly in the ventilatory or hemodynamic responses to acute hypoxic and hypercapnic stimulation, preterm-born adults tended to show lower HVR and greater tachycardic response to transient hypoxia. This pattern may suggest subtle alterations in PCH function that become apparent when the response is assessed with short-lasting, selective stimuli. Transient respiratory tests may therefore offer a useful approach for detecting PCH-related differences that could be obscured during prolonged hypoxic exposure.

SESSION VIII

ENDOCRINE REGULATIONS. CONTROL OF BODY WEIGHT

Friday (September 25th, 8.30 – 13.30) Room B

Chairman: Professor Magdalena Olszanecka-Glinianowicz
(Department of Health Promotion and Obesity Management,
Faculty of Medical Sciences, Medical University of Silesia in Katowice, Poland)

Vice-Chairman: Professor Pawel Antoni Kolodziejski
(Department of Animal Physiology and Biochemistry, Faculty of Veterinary
Medicine and Animal Science. Poznan University of Life Sciences, Poland)

Vice-Chairman: Associate Professor Anna Pedzinska-Betiuk
(Department of Experimental Physiology and Pathophysiology,
Faculty of Pharmacy, Medical University of Bialystok, Bialystok, Poland)

DETAILED SESSION VIII SCHEDULE

Opening lecture Friday (September 25th 8.30 – 9.15)

S8.L1 NEURONOSTATIN A PEPTIDE ENCODED BY SOMATOSTATIN GENE AS A NOVEL REGULATOR OF CARBOHYDRATE AND LIPID METABOLISM. **M. Skrzypski** (Department of Animal Physiology and Biochemistry, Faculty of Veterinary Medicine and Animal Science. Poznan University of Life Sciences, Poznan, Poland).

Oral presentations Friday (September 25th 9.15 – 11.25)

S8.L2 *9.15 – 9.30* LEAP2 REDUCES GLUCAGON SECRETION IN PANCREATIC α-CELLS AND ISLETS OF RODENTS. **O. Sosinski, N. Leciejewska, P. Juzwik, A. Gorka, M. Nowak, M. Sassek, P. Kolodziejski** (Department of Animal Physiology and Biochemistry, Faculty of Veterinary Medicine and Animal Science. Poznan, University of Life Sciences, Poznan, Poland).

S8.L3 *9.30 – 9.45* SPEXIN AS A REGULATOR OF PROLIFERATION AND ACTIVATION IN HUMAN SKIN FIBROBLASTS. **N. Leciejewska[1], P. Kolodziejski[1], O. Sosinski[1], E. Grzeda[1], A. Rekas-Dudziak[2], A. Lukomska[3], M. Sassek[1], E. Pruszynska-Oszmalek[1]** ([1]Department of Animal Physiology and Biochemistry, Faculty of Veterinary Medicine and Animal Science. Poznan University of Life Sciences, Poznan, Poland; [2]Medical Center Operowa, Lublin, Poland; [3]Department of Preclinical Sciences and Infectious Diseases, Poznan University of Life Sciences, Poznan, Poland).

S8.L4 *9.45 – 10.00* HEART FIBROSIS IS RETARDED BY OVERABUNDANT GLYCOSOAMINOGLYCANS ACCUMULATION IN DIABETIC CONDITION. **M. Drobnik[1], M. Galdyszynska[2], A. Kucner[2], P. Radwanska[2], W. Skierkowski[3], L. Piera[2], J.J. Szymanski[4], J. Drobnik[2]** ([1]Department of Nephrocardiology, [2]Department of Pathophysiology, [3]Department of Pathomorphology, [4]Central Scientific Laboratory, Medical University of Lodz, Lodz, Poland).

10.10 – 10.40 Coffee break

S8.L5 *10.40 – 10.55* EFFECTS OF AGE AND INSULIN RESISTANCE ON IGF-1 SIGNALLING: ASSOCIATION WITH CARDIOMETABOLIC RISK. **A. Zebrowska[1], K. Kempa[2], B. Hall[1]; (**[1]Department of Physiology and Pathophysiology, Academy of Physical Education, Katowice, Poland; [2]Institute of Sport Sciences Academy of Physical Education, Katowice, Poland).

S8.L6 *10.55 – 11.10* PHOENIXIN ATTENUATES INFLAMMATION IN 3T3-L1 ADIPOCYTES CO-CULTURED WITH MACROPHAGES. **J. Fiedorowicz, T. Wojciechowicz, J. Urbaniak, B. Wojtaszek, M. Krazek, M. Skrzypski** (Department of Animal Physiology and Biochemistry, Faculty of Veterinary Medicine and Animal Science, Poznan University of Life Sciences, Poznan, Poland).

Poster Session Friday (September 25th 11.40 – 13.30)

S8.P1 EFFECTS OF ELABELA-11 ON LIPOLYSIS, LIPOGENESIS, AND ADIPOKINE EXPRESSION AND SECRETION IN ISOLATED RAT ADIPOCYTES**. Z. Checinska-Maciejewska[1], O. Sosinski[2], N. Leciejewska[2], M. Sassek[2], H. Krauss[1], P. Kolodziejski[2], E. Pruszynska-Oszmalek[2] (**[1]Department of Medicine, Calisia University, Kalisz, Poland; [2]Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland).

S8.P2 CORD-BLOOD LEPTIN, BUT NOT INSULIN, IS INDEPENDENTLY ASSOCIATED WITH BIRTH WEIGHT AND THIGH CIRCUMFERENCE IN TERM NEONATES. **Z. Checinska-Maciejewska[1], M. Wojciechowska[2], A. Ciborek[1], H. Krauss[1]** ([1.]Department of Medicine, Calisia University, Kalisz, Poland; [2.]Department of Mother and Child Health, Poznan University of Medical Sciences, Poznan, Poland).

S8.P3 INTERACTION BETWEEN METABOLIC SYNDROME AND RS7975232 (BSMI) VDR POLYMORPHISM IN SHAPING METABOLIC AND ANTHROPOMETRIC TRAITS IN OLDER ADULTS. **S. Michniewicz[1], K. Chmielowiec[2], B. Czyzniewski[3], M. Gibas-Dorna[3] E. Pruszynska-Oszmalek[4], P. Kolodziejski[4], M.T. Kowalski[5], A. Grzywacz[6], J. Chmielowiec[3]** ([1]Department of Humanization of Health Care and Sexology, University of Zielona Gora, Poland; [2]Department of Hygiene and Epidemiology, University of Zielona Gora, Poland; [3]Department of Anatomy and Histology, University of Zielona Gora, Poland; [4]Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland; [5]Clinical Department of Cardiology, Multispecialty Hospital in Nowa Sol, Poland; [6]Independent Laboratory of Genetics and Behavioral Epigenetics, Pomeranian Medical University, Szczecin, Poland).

S8.P4 BAICALEIN CHANGES GLUCOSE METABOLISM AND ENDOCRINE FUNCTIONS OF MATURE RAT ADIPOCYTES. **K. Konieczna, T. Szkudelski, K. Szkudelska** (Poznan University of Life Sciences; Faculty of Veterinary Medicine and Animal Science; Department of Animal Physiology and Biochemistry; Poznan; Poland).

S8.P5 ASPROSIN STIMULATES PROLIFERATION OF 3T3-L1 CELLS VIA PKB/mTOR-DEPENDENT MECHANISM. **T. Wojciechowicz, M. Krazek, J. Fiedorowicz, A. Rak, M. Skrzypski** ([1]Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland; [2]Laboratory of Physiology and Toxicology of Reproduction, Institute of Zoology and Biomedical Research, Jagiellonian University, Krakow, Poland).

S8.P6 MOTS-C HAS LIMITED EFFECT ON ADIPOSE CELLS ACTIVITY**. M. Sassek, E. Pruszynska-Oszmalek, N. Leciejewska, E. Grzeda, M. Billert, P. Kolodziejski** (Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland).

S8.P7 EFFECTS OF NEURONOSTATIN ON METABOLIC PARAMETERS IN A MOUSE MODEL OF OBESITY**. M. Krazek, J. Fiedorowicz, P. Kolodziejski, E. Pruszynska-Oszmalek, T. Wojciechowicz, M. Skrzypski** (Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland).

S8.P8 POTENTIAL ANTI-INFLAMMATORY EFFECTS OF EPINECIDIN-1 IN 3T3-L1 CELLS. **P. Juzwik, N. Leciejewska, M. Nowak, A. Gorka, O. Sosinski, M. Sassek, E. Pruszynska-Oszmalek (**Department of Animal Physiology and Biochemistry, Poznan University of Life Sciences, Poznan, Poland).

S8.L1

NEURONOSTATIN A PEPTIDE ENCODED BY SOMATOSTATIN GENE AS A NOVEL REGULATOR OF CARBOHYDRATE AND LIPID METABOLISM

M. SKRZYPSKI

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Neuronostatin is a peptide encoded by the somatostatin gene. The biologically active form of neuronostatin is an amidated peptide consisting of 13 amino acids. Its biological effects are mediated through the activation of G protein-coupled receptor 107 (GPR107). Neuronostatin is widely expressed in the central nervous system and various peripheral tissues, including the pancreas, gastrointestinal tract, and adipose tissue. In the central nervous system, neuronostatin has been implicated in the regulation of nociception, depressive behaviors, and anxiety. Additionally, increasing evidence suggests that this peptide plays a role in regulating energy homeostasis by modulating food intake as well as glucose and lipid metabolism. Animal studies have shown that neuronostatin suppresses both appetite and water intake. Several studies have also reported that it stimulates glucagon synthesis and secretion while reducing circulating insulin levels. Moreover, our recent findings indicate that neuronostatin may be involved in regulating adipose tissue functions. In rodent white and brown preadipocytes, neuronostatin has been found to stimulate cell proliferation through the activation of protein kinase B (AKT). Conversely, we discovered that neuronostatin suppresses the differentiation of rat brown preadipocytes into mature fat cells. Furthermore, our research demonstrated that intraperitoneal administration of neuronostatin in mice with high-fat diet-induced obesity impairs insulin sensitivity and affects lipid metabolism and gene expression in both white and brown adipose tissue. In conclusion, these findings collectively suggest that neuronostatin may serve as a new modulator of glucose and lipid metabolism.

This work was financed by the National Science Centre (Poland) (Grant number 2021/43/O/NZ4/00275).

S8.L2

PET MICROPLASTIC EXPOSURE AFFECT THE ADRENAL CORTEX PROTEOME IN A PORCINE *IN VIVO* MODEL

A. KURZYNSKA[1], P. SZYMKOW[1], K. MIERZEJEWSKI[1], B.S. SAHIN[1], S.F. ZAIDI[1], A.S. KLINKBY[2], B. NAIR[2], A. ROGOWSKA-WRZESINSKA[2], I. BOGACKA[1]

[1]Department of Animal Anatomy and Physiology, Faculty of Biology and Biotechnology, University of Warmia and Mazury, Olsztyn, Poland; [2]Department of Biochemistry and Molecular Biology, University of Southern Denmark, Odense, Denmark; aleksandra.kurzynska@uwm.edu.pl

Polyethylene terephthalate (PET), widely used in food packaging, can release microplastic particles (MPs) during storage, which have been detected in human blood and tissues. Given their potential to disrupt physiological processes, this study investigated whether oral PET MP exposure alters the adrenal gland proteome in immature gilts. Adrenal glands from 7–8-month-old crossbred pigs (Large White × Polish Landrace; N=5/group) were analyzed following 4-weeks of oral exposure to PET MPs: control (empty capsules), low dose (0.1 g/day), and high dose (1 g/day). Proteins were extracted, digested with trypsin, and analyzed by LC–MS/MS (Evosep One–Orbitrap Astral, DIA). Data were processed in Spectronaut (directDIA), and differential protein abundance was assessed using PolySTest with Benjamini–Hochberg correction (FDR < 0.05). Exposure to PET MPs resulted in minimal proteomic changes at LD, with only one protein upregulated and one downregulated. In contrast, HD exposure led to substantial alterations, with 23 proteins upregulated and 115 downregulated. Differentially expressed proteins were predominantly associated with mitochondrial and endoplasmic reticulum function, protein synthesis, and exocytosis. These findings suggest that HD PET MP exposure disrupts adrenal cortex physiology by inducing mitochondrial and ER stress and impairing protein synthesis. Additionally, altered expression of exocytosis-related proteins may hinder the delivery of enzymes and lipids, potentially affecting hormone secretion.

The experiment and analyses is supported by the National Science Centre, Poland, grant no. 2021/43/B/NZ7/01093; The internship in Denmark is supported by the Reproductive Biology Society; MS and proteomics research at the University of Southern Denmark in Odense is supported by the Novo Nordisk Foundation through the INTEGRA research infrastructure (grant NNF20OC0061575) and by the Danish Agency for Higher Education and Science through PLATO: the Danish National Mass Spectrometry Platform for Proteomics and Biomolecular Imaging (grant 5229-00012B).

S8.L3

LEAP2 REDUCES GLUCAGON SECRETION IN PANCREATIC A-CELLS AND ISLETS OF RODENTS

O. SOSINSKI, N. LECIEJEWSKA, P. JUZWIK, A. GORKA, M. NOWAK, M. SASSEK, P. KOLODZIEJSKI

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Liver Enriched Antimicrobial Peptide 2 (LEAP2) is a peptide hormone identified in 2003 and recognized as an endogenous antagonist of the Growth Hormone Secretagogue Receptor (GHSR-1a). By inhibiting ghrelin binding to GHSR-1a, LEAP2 suppresses ghrelin activity and reduces food intake. Although recent studies have begun to elucidate the role of LEAP2 in energy metabolism, its relationship with glucagon regulation remains largely unexplored. The aim of this study was to investigate the effects of LEAP2 on glucagon production in murine α-cells and in isolated pancreatic islets from mice and rats.Cells and pancreatic islets were treated with increasing concentrations of LEAP2 (1, 10, and 100nM), followed by measurement of glucagon secretion as well as glucagon gene and protein expression. Our findings demonstrate that LEAP2 treatment leads to a significant reduction in glucagon secretion in both α-cells and isolated islets. We further confirmed the expression of LEAP2 and GHSR-1a in α-cells, pancreatic islets, and whole pancreas using qPCR, Western blotting, and immunofluorescence analyses. Finally, we show that LEAP2 stimulates α-cell proliferation without affecting apoptosis.

National Science Centre grant number 2021/41/B/NZ5/00446

S8.L4

SPEXIN AS A REGULATOR OF PROLIFERATION AND ACTIVATION IN HUMAN SKIN FIBROBLASTS

N. LECIEJEWSKA[1], PA. KOLODZIEJSKI[1], O. SOSINSKI[1], E. GRZEDA[1], A. REKAS-DUDZIAK[2], A. LUKOMSKA[3], M. SASSEK[1], E. PRUSZYNSKA-OSZMALEK[1]

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Spexin (SPX), also known as neuropeptide Q (NPQ), was first identified through bioinformatics analyses in 2007. SPX mRNA and protein have been detected in a wide range of tissues, including the nervous system, gastrointestinal tract and reproductive system, reflecting its broad tissue distribution. Recent studies suggest that SPX is involved in the regulation of various biological processes and may serve as a tumor biomarker in the diagnosis of basal cell carcinoma (BCC) and cutaneous malignant melanoma (CMM), where elevated SPX expression has been observed. The aim of this study was to evaluate the effects of SPX on normal human skin fibroblasts. Fibroblasts were isolated from human skin obtained during surgical procedures. Tissue samples were mechanically fragmented, pre-digested with collagenase (3 mg/mL, 15 min) and placed in culture dishes to allow spontaneous cell migration. After four days, residual tissue fragments were removed, and the cells were cultured until confluence. Immunofluorescence (IF) staining was used to confirm the presence of vimentin-positive cells and to demonstrate the basal expression of SPX, GALR2, and GALR3 in fibroblasts and skin sections. Fibroblasts were then incubated with SPX at concentrations of 10, 100 and 1000 nM for 24 hours. Cytotoxicity (MTT and NRU assays), proliferation (BrdU assay) and gene expression (qPCR) analyses were performed. Fibroblast migration, following mitomycin C treatment to inhibit proliferation, was assessed using a scratch assay after 48 hours of incubation with SPX. Real-time PCR was used to evaluate the expression of α-SMA and fibronectin as markers of early myofibroblast activation. The results indicate that SPX is a potent regulator of proliferative processes, promotes fibroblast activation toward a myofibroblast phenotype and enhances cell migration. These findings suggest that SPX may act as a potential modulator of skin fibroblast function.

S8.L5

HEART FIBROSIS IS RETARDED BY OVERABUNDANT GLYCOSOAMINOGLYCANS ACCUMULATION IN DIABETIC CONDITIONS

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Introduction. Collagen binding integrin, $\alpha 2\beta 1$ is involved in regulation of interleukin-6 (IL-6) secretion. This study is aimed at explanation whether glycation of collagen may modify release of IL-6 or collagen accumulation by human cardiac fibroblasts or diabetics heart. Materials and methods. This concept was tested on diabetic mice and human cardiac fibroblasts cultured on glycated collagen. Diabetes mellitus was induced by streptozotocin injections. In diabetics decreased body weight, increased glucose concentration and fructosoamine were detected. Results. In *cultures* on glycated by methylgloxal, collagen and in diabetic mice, glycosaminoglycans (GAG) accumulation is increased but collagen content is augmented or not influenced respectively. IL-6 concentration is not changed within the diabetic heart. Both extracellular and intracellular concentration of IL-6 and expression of its' gene are augmented in cultures on glycated collagen. Neither soluble nor attached to cell membrane IL-6 receptors density were influenced by collagen glycation. IL-6 membrane receptor density was similar in both groups of mice. GAG overabundance does not modify IL-6 but decrease collagen content within the cultures. Conclusions. Lack of fibrosis in diabetic mice is relevant to mechanism dependent on GAG overabundance. GAG decrease collagen content and inhibit expression of $\alpha 1$ chain of procollagen type I and III. Elevation of IL-6 secretion by glycated collagen is dependent on increase of this cytokine gene expression. The results excluded involvement of GAG excess or changes of tissue sensitivity to IL-6.

Acknowledgements: The study was supported by grant from Medical University of Lodz 564/5-000-00/564-20-050.

S8.L6

EFFECTS OF AGE AND INSULIN RESISTANCE ON IGF-1 SIGNALLING: ASSOCIATION WITH CARDIOMETABOLIC RISK

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Previous studies has demonstrated an age-related decline in circulating levels of insulin like growth factor-1 (IGF-1) levels and its cardioprotective effects. This study aimed to investigate the association between IGF-1 levels, its bbioavailability, and carbohydrate and lipid metabolism parameters with the occurrence of cardiometabolic risk factors. Methods: The IGF-1, glucose, insulin and lipid parameters levels were determined in a population-based sample of older Polish Caucasians aged 55 to > 90 years. To assess differences between groups stratified by age, sex, body mass status and insulin sensitivity, the repeated measures ANOVA was applied. Results: The statistical analysis revealed a significant effect of age ($p < 0.01$), sex ($p < 0.05$) and insulin resistance ($p < 0.01$) on IGF-1 concentrations. Serum IGF-1 levels were significantly lower in participants with HOMA-IR > 2.5 and was associated with higher atherosclerosis risk. Conclusions: Our findings indicate that age and insulin resistance significantly influence IGF-1 signalling and cardiometabolic risk in an older Polish Caucasian population. Importantly, the observed relationships varied between women and men across different age groups, highlighting potential sex-related differences in the metabolic and endocrine mechanisms associated with ageing.

S8.L7

PHOENIXIN ATTENUATES INFLAMMATION IN 3T3-L1 ADIPOCYTES CO-CULTURED WITH MACROPHAGES

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Peptide hormones play a key role in regulating diverse physiological processes, including modulation of the inflammatory response and immune homeostasis. During inflammation, endogenous peptides often exert anti-inflammatory effects by suppressing or modulating key inflammatory mediators and their activity. One of the peptide hormones with potential anti-inflammatory activity is phoenixin (PNX), a recently discovered neuropeptide identified through bioinformatic approaches. It is involved in diverse physiological processes, including reproductive and neurocognitive functions. Moreover, PNX regulates white and brown adipogenesis, but its role in inflammation associated with metabolic disorders such as obesity remains unclear. In this study we investigated whether PNX affects white adipocytes under inflammatory conditions resembling those observed in obesity. 3T3-L1 fibroblasts were differentiated into mature white adipocytes according to a standard protocol over an 8-day period. Mature adipocytes were co-cultured with lipopolysaccharide-stimulated (100 ng/ml, 24h) murine macrophages (RAW 264.7) in the presence or absence of PNX (10 and 100 nmol/l). Gene expression was assessed by qPCR. After 24 h of co-culture, PNX suppressed the mRNA expression of pro-inflammatory cytokines such as IL-6 and TNF-α at both concentrations 10 and 100 nmol/l. However, PNX did not alter the mRNA expression of MCP-1 and resistin. These results suggest that PNX can attenuate inflammation in 3T3-L1 adipocytes co-cultured with RAW 264.7 macrophages and may represent a potential therapeutic strategy for obesity-related metabolic and inflammatory disorders.

This research was funded by the National Science Centre, Poland (NCN), project no. 2022/45/B/NZ5/03453.

S8.P1

EFFECTS OF ELABELA-11 ON LIPOLYSIS, LIPOGENESIS, AND ADIPOKINE EXPRESSION AND SECRETION IN ISOLATED RAT ADIPOCYTES

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Elabela (ELA), an endogenous ligand of the apelin receptor, has anti-inflammatory and antioxidant properties, but its metabolic actions in adipose tissue remain insufficiently defined. This study evaluated the effects of ELA-11 (1, 10, 100, and 1000 nM) on lipolysis, lipogenesis, and leptin and adiponectin expression and secretion in isolated rat adipocytes. Adipocytes were isolated from perigonadal adipose tissue by collagenase digestion and incubated with ELA-11 for 2 or 6 h. Lipolysis was assessed by glycerol release, whereas lipogenesis was determined from incorporation of 14C-labelled glucose into lipids. Leptin and adiponectin secretion was measured by ELISA. The mRNA expression of both adipokines and their receptors was quantified by real-time PCR using the ΔΔCT method. ELA-11 inhibited lipolysis after 2 h ($p<0.01$) and 6 h ($p<0.05$). At 1000 nM, it reduced leptin and adiponectin mRNA expression ($p<0.05$) while increasing adiponectin receptor mRNA expression ($p<0.05$). Leptin secretion decreased after 6 h at 100 nM ($p<0.05$), and adiponectin secretion decreased after both incubation periods at selected concentrations ($p<0.01$). ELA-11 did not significantly affect lipogenesis. These findings indicate that ELA-11 modulates adipocyte function primarily by suppressing lipolysis and altering adipokine expression and secretion. Its role in adipose-tissue metabolism warrants further mechanistic investigation.

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S8.P2

CORD-BLOOD LEPTIN, BUT NOT INSULIN, IS INDEPENDENTLY ASSOCIATED WITH BIRTH WEIGHT AND THIGH CIRCUMFERENCE IN TERM NEONATES

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Birth weight is an overall measure of prenatal growth but does not reflect regional neonatal body composition. We assessed whether cord-blood leptin or insulin was independently associated with birth weight and regional circumferences in term neonates. This cross-sectional secondary analysis used 323 mother–newborn records. Cord-blood leptin and insulin were available for 196 and 195 neonates, respectively, and both hormones for 193. Associations with birth weight and head, chest, abdominal, thigh, and arm circumferences were examined using Spearman correlations and multivariable linear regression. Hormone concentrations were log-transformed. Models included both hormones and were adjusted for neonatal sex, gestational age, maternal pre-pregnancy weight, and gestational weight gain. HC3 standard errors and Benjamini–Hochberg false-discovery-rate correction were applied. Leptin correlated with birth weight and thigh circumference ($\rho=0.39$ for both; $q<0.001$), with weaker positive correlations for other circumferences. In adjusted models, leptin remained associated with birth weight (standardised $\beta=0.22$, 95% CI 0.09–0.36; $p=0.001$) and thigh circumference ($\beta=0.28$, 95% CI 0.12–0.43; $p<0.001$). Associations with head and abdominal circumference were weaker, while those with chest and arm circumference were non-significant. Insulin showed no independent association after adjustment. Cord-blood leptin was a more consistent endocrine correlate of neonatal size than insulin, particularly for birth weight and thigh circumference. The results support leptin as a marker of fetal adipose and peripheral-tissue accretion, but require prospective validation using direct body-composition measurements.

S8.P3

INTERACTION BETWEEN METABOLIC SYNDROME AND RS7975232 (BSMI) VDR POLYMORPHISM IN SHAPING METABOLIC AND ANTHROPOMETRIC TRAITS IN OLDER ADULTS

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The aim of the study was to investigate the associations between metabolic status, the rs7975232 polymorphism of the vitamin D receptor (VDR) gene, and selected anthropometric and biochemical parameters in residents of a nursing and treatment facility. The study included institutionalized older adults stratified into metabolic syndrome (MetS) and non-MetS groups. Anthropometric measurements, blood pressure, and biochemical parameters were assessed using standard methods, and VDR genotyping was performed. A two-way ANOVA model was applied to evaluate the effects of metabolic status, genotype, and their interaction.

Significant main effects of metabolic status were observed for key metabolic traits, including triglycerides, HDL-C, NEFA, and waist-to-hip ratio, indicating a less favorable metabolic profile in individuals with MetS. In contrast, most anthropometric parameters showed no strong genotype-dependent differences. The rs7975232 polymorphism demonstrated significant effects and interaction patterns predominantly in lipid-related traits. The most pronounced findings were observed for triglycerides and non-esterified fatty acids (NEFA), where genotype effects were dependent on metabolic status. Individuals carrying the AA genotype within the MetS group consistently exhibited the highest triglyceride and NEFA levels compared with other genotype–group combinations. In contrast, no genotype-related differences were observed within the non-MetS group. A weaker but similar interaction pattern was noted for total cholesterol, while no consistent effects were found for HDL-C or LDL-C. Anthropometric variables showed limited genotype dependence, with only isolated differences in abdominal circumference and no robust effects on other measures of body composition. In conclusion, the rs7975232 VDR polymorphism appears to modulate lipid metabolism in a metabolic status-dependent manner, with the strongest effects observed for triglycerides and NEFA. These findings suggest a potential gene–metabolic interaction in older institutionalized adults.

This work was financially supported as part of a project funded by the Marshal's Office of the Lubusz Voivodeship and the University of Zielona oora (Grant Agreement No. DRG.V.042.8.2024).

S8.P4

BAICALEIN CHANGES GLUCOSE METABOLISM AND ENDOCRINE FUNCTIONS OF MATURE RAT ADIPOCYTES

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Obesity is one of the most prevalent metabolic disorders of the 21st century and contributes to the development of numerous complications, including type 2 diabetes and cardiovascular diseases. Plant-derived polyphenols are increasingly recognized for the alleviation of metabolic disorders. One of them - baicalein, an aglycone of baicalin found in *Scutellaria baicalensis*, exhibits multiple biological properties; however, its role in adipocyte metabolism remains insufficiently explored. The aim of our study was to investigate the short-term effects of baicalein on glucose metabolism and endocrine function in isolated primary mature rat adipocytes. Adipocytes were isolated from epididymal adipose tissue of male Wistar rats. The intensity of lipogenesis and glucose oxidation in response to baicalein (100 mM) were assessed by conversion of C^{14} labeled glucose into cellular lipids. Glucose transport into cells was evaluated using C^{14} labeled 2-deoxy-D-glucose. Lactate concentration was measured using an enzymatic assay. Changes in mitochondrial membrane potential induced by glucose metabolism in adipocytes were assessed using fluorescence method using rhodamine-123. The concentration of adipokines secreted by cells was determined using ELISA. We demonstrated that baicalein enhanced insulin-stimulated lipogenesis and increased glucose oxidation without affecting glucose transport. Moreover, it reduced lactate release, indicating a shift toward aerobic metabolism. However, no effect of the compound was observed on the inner mitochondrial membrane potential. In the presence of insulin baicalein decreased secretion of leptin and adiponectin - key adipokines involved in the regulation of energy balance, glucose and lipid metabolism. The findings suggest that baicalein may modulate glucose metabolism and endocrine function of fat cells, however they indicate that baicalein should be carefully assessed as a compound with a beneficial effects on metabolic disorders.

S8.P5

ASPROSIN STIMULATES PROLIFERATION OF 3T3-L1 CELLS VIA PKB/MTOR-DEPENDENT MECHANISM

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Asprosin is a recently discovered adipokine that plays a significant role in glucose metabolism and energy homeostasis. Evidence suggests that altered levels of asprosin may be associated with obesity, insulin resistance, or type 2 diabetes. Additionally, research has shown that asprosin inhibits the browning of white adipocytes and promotes the differentiation of white preadipocytes into mature fat cells. However, the role of asprosin in regulating white preadipocyte proliferation remains unclear. Therefore, this study aimed to investigate the effects of asprosin on the proliferation of 3T3-L1 preadipocytes, a well-established model for white preadipocytes. In our experiments, 3T3-L1 cells were cultured in DMEM medium supplemented with 10% fetal bovine serum and antibiotics (streptomycin and penicillin). Cell proliferation was assessed using a 5-bromo-2'-deoxyuridine (BrdU) assay, and gene expression was determined by real-time PCR. Protein levels were evaluated through Western blot analysis. We found that asprosin at 100 nmol/l promoted 3T3-L1 cell proliferation after 24 hours of incubation. Additionally, asprosin continued to stimulate cell proliferation after 48 hours. This effect was further supported by increased expression of mRNA encoding genes involved in cell replication, such as Pcna (proliferating cell nuclear antigen) and c-myc (MYC proto-oncogene). We also observed that asprosin enhanced the phosphorylation of protein kinase B (PKB) and mammalian target of rapamycin kinase (mTOR). Importantly, asprosin failed to stimulate 3T3-L1 cell proliferation in the presence of phosphoinositide 3-kinase/PKB and mTOR inhibitors (LY294002 and rapamycin, respectively). These results demonstrate that asprosin stimulates 3T3-L1 cell proliferation via an PKB/mTOR-dependent pathway.

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S8.P6

MOTS-C HAS LIMITED EFFECT ON ADIPOSE CELLS ACTIVITY

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Mitochondria-derived peptides have recently emerged as important regulators of cellular metabolism and energy homeostasis. Among them, mitochondrial open reading frame of the 12S rRNA-c (MOTS-c) has attracted considerable attention due to its metabolic and cytoprotective properties. However, its role in adipocyte endocrine activity remains insufficiently understood. The aim of this study was to investigate the effects of MOTS-c on adipokine secretion, gene expression, and cellular metabolism in differentiated 3T3-L1 adipocytes. Differentiated 3T3-L1 adipocytes were cultured in vitro and incubated with MOTS-c under controlled experimental conditions. Leptin and adiponectin secretion levels were determined using enzyme-linked immunosorbent assay (ELISA). The expression of leptin, adiponectin, and their respective receptor genes was analyzed by quantitative PCR (qPCR). Cellular metabolic activity was assessed using the MTT assay, while cell viability and cytotoxicity were evaluated using the Neutral Red Uptake (NRU) assay. MOTS-c treatment significantly increased leptin secretion from 3T3-L1 adipocytes, while simultaneously reducing adiponectin secretion. Gene expression analysis revealed that MOTS-c did not affect leptin gene expression; however, it significantly downregulated adiponectin gene expression. No significant changes were observed in the expression of leptin or adiponectin receptor genes following MOTS-c treatment. Additionally, the MTT assay demonstrated increased metabolic activity in adipocytes exposed to MOTS-c, suggesting enhanced cellular metabolism. In contrast, the NRU assay showed no statistically significant changes in cell viability, indicating that MOTS-c did not exert cytotoxic effects under the experimental conditions applied. The present study demonstrates that MOTS-c modulates the endocrine activity of adipocytes by altering leptin and adiponectin secretion and suppressing adiponectin gene expression in 3T3-L1 cells. Furthermore, MOTS-c enhances cellular metabolic activity without affecting cell viability. These findings suggest that MOTS-c may play an important role in the regulation of adipocyte function and metabolic homeostasis, highlighting its potential relevance in obesity- and metabolism-related research.

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S8.P7

EFFECTS OF NEURONOSTATIN ON METABOLIC PARAMETERS IN A MOUSE MODEL OF OBESITY

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Neuronostatin (NST) is a peptide hormone encoded by the somatostatin gene. Its biological activity is mediated by the G protein-coupled receptor GPR107. Previous studies demonstrated that neuronostatin regulates energy homeostasis, regulates appetite, gastrointestinal motility. *In vitro* studies have shown that neuronostatin modulates adipogenesis in both white and brown adipose tissue, but its role in metabolism *in vivo* remain unknown. In this study, we investigated the effects of neuronostatin on insulin sensitivity, glucose tolerance and metabolic parameters in a mouse model of obesity. Mice were fed either a standard diet (SD) or a high-fat diet (HFD; 50% kcal from fat) for 8 weeks. Neuronostatin (100 ng/kg body weight, i.p.) was administered for 4 weeks. Body weight was monitored weekly, while glucose and insulin tolerance tests were performed in the final week. Metabolic parameters were measured using colorimetric assays and ELISA. HFD-fed mice showed increased body weight, impaired glucose tolerance, and reduced insulin sensitivity compared with SD mice. NST-treated obese mice showed impaired insulin sensitivity and increased circulating triacylglycerols. NST did not affect body weight, glucose, cholesterol, insulin, free fatty acids, or glucagon levels. These results suggest that neuronostatin may influence insulin sensitivity and lipid metabolism in obesity.

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S8.P8

POTENTIAL ANTI-INFLAMMATORY EFFECTS OF EPINECIDIN-1 IN 3T3-L1 CELLS

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Epinecidin-1 (Epi-1) is a fish-derived peptide with strong antibacterial and anti-inflammatory properties, representing a promising alternative in the face of increasing antibiotic resistance. In addition to its antimicrobial activity, Epi-1 modulates the immune response and reduces inflammation by decreasing the levels of pro-inflammatory cytokines, including TNF-α and IL-6. These properties are particularly important in the context of obesity, which is associated with chronic low-grade inflammation resulting from excessive cytokine production by adipose tissue. Therefore, Epi-1 is considered a potential tool in modern therapies for infections and chronic inflammatory diseases.The aim of the study was to evaluate the effects of epinecidin-1 (Epi-1) on the viability, proliferation, and expression of selected pro-inflammatory cytokines in 3T3-L1 cells differentiating into adipocytes under inflammatory conditions. The study was conducted using the murine fibroblast 3T3-L1 cell line differentiating into mature adipocytes. Inflammation was induced using lipopolysaccharides (LPS). Following incubation with Epi-1, cell proliferation was assessed using the BrdU assay, while cell viability was determined using MTT and NRU assays. Gene expression at the RNA level was analyzed by RT-PCR. Epi-1 exerted beneficial effects on the viability and proliferation of 3T3-L1 cells under inflammatory conditions. The peptide also altered the expression levels of pro-inflammatory cytokine genes. However, further studies are required to confirm the anti-inflammatory effects of Epi-1 in adipocytes.

S8.P9

MICROPLASTIC EXPOSURE ALTERS ADIPOSE TISSUE FUNCTION: INSIGHTS FROM A PORCINE *IN VIVO* STUDY

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Micro- and nanoplastics (MNPs), especially polyethylene terephthalate (PET), are emerging environmental pollutants that have been detected in animal and human tissues. MNPs exposure has been shown to cause oxidative stress, inflammation, and cellular metabolic disruption. Therefore, MNPs are associated with inflammatory and metabolic diseases. However, little is known about their effect on adipose tissue, an important metabolic and endocrine organ, or about the functional differences between distinct adipose depots. Adipose tissue exhibits functional variability, with subcutaneous fat being more protective and visceral fat being more metabolically active and strongly linked to metabolic diseases. To assess the involvement of PET MNPs in metabolic disorders, it is essential to comprehend how they impact these different depots. In this study, we used samples from 7–8-month-old crossbred Large White × Polish Landrace pigs, divided into three groups (N=3): control (C), receiving empty gelatine capsules; low dose (LD), receiving 0.1 g PET MPs/animal/day; and high dose (HD), receiving 1 g PET MPs/animal/day. The treatment was conducted over 4 weeks. Visceral and subcutaneous adipose tissues were excised, and total RNA was extracted for gene expression analysis using qRT-PCR. To evaluate functional differences between adipose depots in the experimental groups, markers associated with inflammation, oxidative stress, metabolism, and cellular stress were examined. These findings demonstrate that visceral fat tissue shows a more robust response in metabolic processes, due to downregulation of PPARγ in the LD group and FABP4 in the HD group. Although the response is less pronounced, inflammatory markers such as TNF-α and IL-1β mRNAs are likewise elevated in subcutaneous fat in the LD group. Additionally, alterations in metabolic parameters, such as decreased FABP4 mRNA expression in the HD group, indicate some disruption in lipid processing. PET MPs altered the expression of selected genes in both fat tissues, but the response in visceral fat tissue is more strongly linked to metabolic disorders due to its stronger correlation with elevated inflammation and decreased metabolism.

SESSION IX

GASTROINTESTINAL PHYSIOLOGY AND PATHOPHYSIOLOGY, PANCREAS AND LIVER

Friday (September 25th, 8.30 – 12.40) Room C

Chairman: Professor Tomasz Brzozowski
Department of Physiology, Faculty of Medicine,
Jagiellonian University Medical College, Krakow, Poland

Vice-Chairman: Professor Slawomir Kwiecien
Department of Physiology, Faculty of Medicine,
Jagiellonian University Medical College, Krakow, Poland

DETAILED SESSION IX SCHEDULE

Opening lecture Friday (September 25th, 8.30 – 9.15)

S9.L1 GUT MICROBIOTA AND INFLAMMATORY BOWEL DISEASE: FROM PATHOGENESIS TO NOVEL THERAPEUTIC OPPORTUNITIES. **P. Konturek[1], S. Andrei[1], W. Dieterich[2], Y. Zopf[2]** ([1]Department of Medicine 2, Thuringia-Clinic Saalfeld, Teaching Hospital of the University of Jena, Jena, Germany; [2]First Department of Medicine, University Erlangen, Nuremberg, Germany).

Oral presentations Friday (September 25th 9.15 – 11.40)

S9.L2 *9.15 – 9.30* METFORMIN TREATMENT DURING PREGNANCY AND LACTATION AFFECTS METABOLIC PROGRAMMING IN THE LIVER OF RAT OFFSPRING. **M. Slupecka-Ziemilska[1], M. Koperski[1], J. Polosak[2], J. Borkowska[3], M. Nowicka[1], M. Sulejczak[1], M. Wieczorek[1]** ([1]Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland; [2]Collegium Medicum, Cardinal Stefan Wyszynski University, Warsaw, Poland; [3]Centre of New Technologies, University of Warsaw, Warsaw, Poland).

S9.L3 *9.30 – 9.45* CROSSTALK BETWEEN THE GUT AND LIVER: EFFECTS OF HISTAMINE RECEPTOR BLOCKAGE ON HEPATIC OXIDATIVE STRESS IN EXPERIMENTAL COLITIS IN RATS. **B. Bogielski[1,2], K. Michalczyk[1], W. Gebski[1], K. Rozpedek[3], E. Szulinska[3], B. Tempka[1], A. Zorychta[4], E. Chelmecka[4], E. Kaczmar[5], P. Glodek[1], J. John[1], K. Nikiel[1], J. Poloczek[2], J. Jochem[1], K. Kiec-Kononowicz[6], D. Lazewska[6], D. Stygar[1], B. Skrzep-Poloczek[1]** ([1]Department of Physiology, Faculty of Medical Sciences in Zabrze, [2]Branch in Bielsko-Biala, [3]Institute of Biology, Biotechnology and Environmental Protection, Faculty of Natural Sciences, University of Silesia, Katowice, Poland; [4]Department of Medical Statistic, Faculty of Pharmaceutical Sciences in Sosnowiec, Medical University of Silesia, Katowice, Poland; [5]Department of Clinical Diagnostics, Faculty of Veterinary Medicine, University of Warmia and Mazury in Olsztyn, Olsztyn, Poland; [6]Department of Technology and Biotechnology of Drugs, Faculty of Pharmacy, Jagiellonian University Medical College, Krakow, Poland).

S9.L4 *9.45 – 10.00* UNCONVENTIONAL MYOSIN VI IS INVOLVED IN THE REGULATION OF LIPID METABOLISM IN LIVER AND BROWN ADIPOSE TISSUE. **O. Karatsai-Miaskowska[1], W. Ficerman-Kuta[1], L. Lehka[1], V. Navrulin[2], P. Dobrzyn[2], M.J. Redowicz[1]** ([1]Nencki Institute of Experimental Biology PAS, Laboratory of Molecular Basis of Cell Motility, Warsaw, Poland; [2]Nencki Institute of Experimental Biology PAS, Laboratory of Molecular Medical Biochemistry, Warsaw, Poland).

10.10 – 10.40 Coffee break

S9.L5 *10.40 – 10.55* THE SECRETORY HORMONE OF ADIPOCYTES, ADIPONECTIN, PREVENTS FORMATION OF EXPERIMENTAL ISCHEMIC GASTRIC LESIONS. **S. Kwiecien[1], Z. Sliwowski[1], D. Wojcik-Grzybek[1], M. Magierowski[2] , A. Danielak[3], M. Pinkas[3], U. Szczyrk[1], M. Strzalka[1], A. Ptak-Belowska[1], T. Brzozowski[1]** ([1]Department of Physiology, Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland; [2]Jagiellonian University Medical College, Faculty of Medicine, Center for Biomedicine and Interdisciplinary Sciences, Krakow, Poland; [3]Jagiellonian University Medical College, Doctoral School of Medical and Health Sciences, Krakow, Poland).

S9.L6 *10.55 – 11.10* ONE-YEAR FOLLOW-UP OF METABOLIC AND FIBROTIC BIOMARKERS IN SERUM AND LIVER TISSUE AFTER SURGICAL INTERVENTION. **M. Matyja[1], M. Kukla[2,3], E. Chelmecka[4], K. Michalczyk[5], B. Skrzep-Poloczek[5], R. Wasek[5], M. Michalski[6], J. Poloczek[7], D. Stygar[5]** ([1]2nd Department of General Surgery, [2]Department of Internal Medicine and Geriatrics, Jagiellonian University Medical College, Krakow, Poland; [3]Department of Endoscopy, University Hospital in Krakow, Krakow, Poland; [4]Department of Statistics, Department of Instrumental Analysis, Faculty of Pharmaceutical Sciences in Sosnowiec, Sosonowiec, Poland; [5]Department of Physiology, [6]Department of Histology and Cell Pathology, Faculty of Medical Sciences in Zabrze, Zabrze, Poland; [7]Branch in Bielsko-Biala, Medical University of Silesia, Katowice, Poland).

S9.L7 *11.10 – 11.25* HYDROGEN SULFIDE AND PERSULFIDATION AS A REDOX LANGUAGE OF GASTROINTESTINAL EPITHELIA: LESSONS FROM BARRETT'S ESOPHAGUS. **M. Magierowski** (Center for Biomedicine and Interdisciplinary Sciences, Jagiellonian University Medical College, Krakow, Poland).

S9.L8 *11.25 – 11.40* HYDROGEN SULFIDE ATTENUATES *HELICOBACTER PYLORI*-INDUCED GASTRIC FIBROBLAST ACTIVATION AND CAF-LIKE REPROGRAMMING**. G. Krzysiek-Maczka[1], A. Targosz[1], P. Bronowicka-Adamska[2], U. Szczyrk[1], M. Strzalka[1], H. Maczka[5], M. Wierdak[3], T. Brzozowski[1], J. Czyz[4] A. Ptak-Belowska[1]** ([1]Department of Physiology, Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland, [2]Chair in Medical Biochemistry, the Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland, [3]Department of Cell Biology, the Faculty of Biochemistry, Biophysics and Biotechnology, Jagiellonian University, Krakow, Poland, [4]Department of Endoscopic, Metabolic and Soft Tissue Malignancies Surgery of University Hospital, Jagiellonian University, Krakow, Poland; [5]Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland).

Poster session Friday (September 25th 11.40 – 12.40)

S9.P1 EEDBACK REGULATION OF PANCREATIC EXOCRINE SECRETION IN PIGS. **J.L. Valverde Piedra, S. Szymanczyk, K. Pierzynowska, S.G. Pierzynowski** ([1]Department of Pharmacology, Toxicology and Environmental Protection, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland; [2]Department of Animal Physiology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland; [3]Department of Biology, Lund University, Lund, Sweden).

S9.P2 CURCUMIN ACCELERATES HEALING OF EXPERIMENTAL COLITIS *VIA* REACTIVE OXYGEN SPECIES METABOLISM AND MODIFICATION OF GUT MICROBIOTA. **S. Kwiecien[1], Z. Sliwowski[1], D. Wojcik-Grzybek[1], T. Gosiewski[2], D. Salamon[2], A. Krawczyk[2], U. Szczyrk[1], A. Targosz[1], A. Ptak-Belowska[1], T. Brzozowski[1]** ([1]Department of Physiology, Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland; [2]Department of Microbiology Jagiellonian University Medical College, Faculty of Medicine, Krakow, Poland).

S9.P3 ASYMMETRIC DIMETHYLARGINIE (ADMA) AGGRAVATES ANTIOXIDATIVE PROPERTIES OF GASTRIC MUCOSA. **S. Kwiecien, D. Wojcik-Grzybek, Z. Sliwowski, U. Szczyrk, R. Pajdo, A Targosz, A. Ptak-Belowska, T. Brzozowski** (Department of Physiology, Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland).

S9.P4 INTERPLAY BETWEEN GLUTATHIONE, SUPEROXIDE DISMUTASE, NITRIC OXIDE AND PROSTAGLANDINS IN THE HEALING OF GASTRIC MUCOSA. **S. Kwiecien, Z. Sliwowski, U. Szczyrk, J. Cieszkowski, R. Pajdo, A. Ptak-Belowska, T. Brzozowski1, D. Wojcik-Grzybek** (Department of Physiology, Faculty of Medicine, Jagiellonian University Medical College, Krakow, Poland).

S9.L1

GUT MICROBIOTA AND INFLAMMATORY BOWEL DISEASE: FROM PATHOGENESIS TO NOVEL THERAPEUTIC OPPORTUNITIES

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Epidemiological studies show that the incidence of inflammatory bowel disease (IBD) is increasing rapidly due to different environmental factors. There is an increasing evidence that the perturbations in the intestinal microbiota are strongly linked to the pathogenesis of IBD. In the present study we analysed the gut microbiota composition in 40 patients with IBD (52,50 % females and 47,50% males) and 40 healthy controls using 16sRNA sequencing. The medium calprotectin value in IBD patients was 516 mg/g. Our study showed in IBD patients a significant decrease in alpha diversity indices (fisher divesrity, shannon diversity) as compared to healthy controls. b-diveristy (Bray curtis dissimilarity) demonstrated significant differences between the composition of gut microbiota in IBD patients vs healthy controls. At the phylum level a significant decrease of short chain fatty acids (SCFA) producing Firmicutes and increase of proinflammatory Proteobacteria was observed. At the family level, the overrepresentation of proinflammatory taxa such as Fusobacteriaceae, Enterobacteriaceae, Akkermansiaceae, Sutterellaceae und Bacteroidaceae was demonstrated. The abundance of SCFA producing Lachnospiraceae, Ruminococcaceae and Oscillospiraceae was significantly reduced. Conclusions: 1) patients with IBD show a profound intestinal dysbiosis; 2) The loss of short-chain fatty acid (SCFA)-producing species weakens the intestinal barrier, increasing gut permeability and promoting bacterial translocation across the intestinal epithelium into lamina propria. This also impairs the differentiation of Tregs, which are essential for immune tolerance. 3) The improvement of gut microbiota composition by special diets, probiotics, prebiotics, synbiotics or fecal microbiota therapy (FMT) play a crucial supporting role in the therapy of IBD.

S9.L2

METFORMIN TREATMENT DURING PREGNANCY AND LACTATION AFFECTS METABOLIC PROGRAMMING IN THE LIVER OF RAT OFFSPRING

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Metformin is commonly used to treat polycystic ovary syndrome (PCOS) and gestational diabetes (GDM). This study examines the effects of early life exposure to metformin on liver histology and metabolism in offspring, particularly in response to a high-fat diet (HFD). Female normoglycemic Sprague-Dawley rats were administered metformin during pregnancy and lactation. On postnatal day 79, their offspring were divided into groups and fed either a standard diet or a HFD for the following three consecutive weeks. Liver histology, AMPK and SIRT1 expression, and changes in body weight were assessed in 100-day-old male and female offspring separately. Maternal metformin treatment increased body weight and liver steatosis in HFD–fed offspring, compared to offspring fed HFD alone, without early-developmental exposure. However, no changes in AMPK and SIRT1 expression were observed. These findings suggest that early-life metformin exposure alters hepatic metabolic programming and may predispose offspring to diet-induced metabolic disturbances. The results highlight potential long-term risks of prenatal metformin use, particularly when combined with unhealthy dietary habits later in life.

This research was funded by National Science Centre, Poland. Grant no. 2022/46/E/NZ7/00286.

S9.L3

CROSSTALK BETWEEN THE GUT AND LIVER: EFFECTS OF HISTAMINE RECEPTOR BLOCKAGE ON HEPATIC OXIDATIVE STRESS IN EXPERIMENTAL COLITIS IN RATS

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Inflammatory bowel diseases are associated with persistent inflammation and extraintestinal complications, including redox homeostasis disturbances in the liver. Therefore the aim of the study was to examine the influence of histamine H1-H4 receptor blockage on hepatic oxidative stress (OS) in male Wistar rats subjected to 2,4,6-trinitrobenzenesulfonic acid-induced colitis. After 7 days of treatment with histamine receptor antagonists, animals were euthanized, and OS parameters were measured. Colitis was accompanied by an increase in α-esterase and a decrease β-esterase activity in the liver tissue. The treatment with H1 (cetirizine, 10 mg/kg, intramuscular [im]) or H3 (iodophenpropit, 10 mg/kg, im) receptor antagonists reduced β-esterase activity compared to control animals. In addition, cetirizine decreased superoxide dismutase activity and glutathione (GSH) concentrations. Treatment with the H2 receptor antagonist ranitidine (10 mg/kg, im) led to a decrease in malondialdehyde (MDA) concentration. Finally, H4 receptor blockade with JNJ7777120 hydrochloride (30 mg/kg, im) induced increases in GSH and MDA concentrations. In conclusion, histamine, acting *via* different types of receptors, is able to modulate differently hepatic OS responses in experimental colitis in rats.

S9.L4

UNCONVENTIONAL MYOSIN VI IS INVOLVED IN THE REGULATION OF LIPID METABOLISM IN LIVER AND BROWN ADIPOSE TISSUE

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Lipid metabolism plays a key role in the metabolic regulation, signal transduction, and energy supply under physiological and pathological conditions. Liver is responsible for coordinated uptake, synthesis, storage, and release of lipids. Brown adipose tissue (BAT), in turn, is responsible for the energy storage, and aids in the regulation of the fat and sugar metabolism. An imbalance in lipid metabolism has been associated with various pathologies such as hepatic lipogenesis, coronary atherosclerotic cardiopathy, obesity, and insulin resistance. We showed that unconventional myosin VI (MVI), a unique minus-end-directed motor protein, takes part in the regulation of the skeletal muscle metabolism. Loss of MVI resulted in accelerated lipolysis, especially in slow-twitch soleus (SOL) muscle. To further explore the mechanisms of MVI involvement in lipid metabolism, we analyzed the liver and BAT from *Snell's waltzer* mice (natural MVI knockouts, MVI-KO) during development (i.e. in newborns (P0), 3-month- and 12-month-old animals). We found that MVI-KO liver weight is reduced throughout the examined life span. Similar changes were observed for BAT of P0 mice. Analysis of the liver lipid content revealed that loss of MVI leads to increased levels of cholesterol, cholesterol ester and free fatty acids with concomitant decrease of triglycerides. To further characterize involvement of MVI in liver metabolism, we examined activity of several signaling pathways involved in lipogenesis de novo, lipolysis, protein synthesis, cellular respiration, and inflammation. This study confirms our suggestion of important role of MVI in in the regulation of lipid metabolism.

Statutory funds from the Ministry of Science and Higher Education to the Nencki Institute.

S9.L5

THE SECRETORY HORMONE OF ADIPOCYTES, ADIPONECTIN, PREVENTS FORMATION OF EXPERIMENTAL ISCHEMIC GASTRIC LESIONS

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Adiponectin is a peptide, produced by adipocytes, exhibiting beneficial metabolic action through lipids and carbohydrates metabolism stimulation, as well as anti-inflammatory action. In our investigations, we focus on the determination of adiponectin role in gastric ulcer healing, in of ischemia – reperfusion conditions (I/R). Acute damage of gastric mucosa make an important clinical problem. However the participation of ROS generation, lipid peroxidation products and involvement of NO and sensory afferent nerves in action of adiponectin remains unknown. The aim of our present investigations is explanation details of interplay between adiponectin and capsaicin – sensitive afferent nerves, endogenous production of NO, biological effect of ROS action, expressed by lipid peroxidation products and antioxidative system efficacy, genes expression for proinflammatory cytokines in action on gastric mucosa during I/R. Experiments were caried out on male Wistar rats and the area of gastric lesions was measured by planimetry. Colorimetric assays were used to determine gastric mucosal levels of malondialdehyde (MDA) and 4-hydroxynonenal (4-HNE), superoxide dismutase activity (SOD) and reduced glutathione level (GSH). High doses of capsaicin were used to inactivate sensory nerves. Results of our experiments exhibited protective role of adiponectin to gastric mucosa, revealed by decrease of gastric area and increase of gastric blood flow (GBF). Blockade of endogenous NO production reverses these effects, additional application of L-arginine restored protective effect of adiponectin. Capsaicin denervation also impeded beneficial action of adiponectin in I/R model, restored by application of CGRP. Adiponectin dose – dependently decreased MDA and 4-HNE, as well as proinflammatory cytokines and gastrin, with accompanied rise SOD, GSH, in I/R model. We concluded, that adiponectin, administered intravenously, exerted gastroprotective effect against ischemia/reperfusion – induced gastric lesions (I/R), through mechanism involving decrease of lipid peroxidation (MDA+4-HNE) increase of antioxidative factors (SOD, GSH), as well as *via* endogenous NO production and action capsaicin – sensitive afferent nerves.

S9.L6

ONE-YEAR FOLLOW-UP OF METABOLIC AND FIBROTIC BIOMARKERS IN SERUM AND LIVER TISSUE AFTER SURGICAL INTERVENTION

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Obesity is related to metabolic dysfunction-associated steatotic liver disease (MASLD) through complex interactions between adipose tissue, systemic inflammation, oxidative stress (OS), and hepatic metabolism. Adipokines and hepatokines may reflect metabolic and fibrotic remodeling following bariatric surgery. The study evaluated changes in circulating chemerin, apelin, and elabela concentrations and assessed their relationship with hepatic OS and fibrosis markers 12 months after bariatric surgery in 68 patients. Serum concentrations of chemerin, apelin, and elabela were measured before and 12 months after the procedure. Liver tissues sampled during surgery were analyzed for NOX1, NOX2, SIRT1, TGFβ1, THBS2, and chemerin concentrations. The analyses showed that 12 months after bariatric surgery, chemerin and apelin concentrations in serum significantly decreased, whereas elabela concentrations significantly increased. Liver tissues presented measurable expression of OS and fibrosis-related markers, including NOX1, NOX2, SIRT1, TGFβ1, THBS2, and chemerin. The reduction in circulating chemerin and apelin, accompanied by the increase in elabela concentrations, may reflect improvement in metabolic status and restoration of protective signaling pathways within the apelin–APJ system. Elabela appears to be a promising biomarker of metabolic recovery and may play a role in hepatic remodeling after bariatric surgery. The observed biomarker profile suggests substantial metabolic remodeling within the adipose tissue-liver axis, which induces significant post bariatric endocrine and metabolic changes extending beyond weight loss alone.

S9.L7

HYDROGEN SULFIDE AND PERSULFIDATION AS A REDOX LANGUAGE OF GASTROINTESTINAL EPITHELIA: LESSONS FROM BARRETT'S ESOPHAGUS

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Hydrogen sulfide (H_2S) is an endogenous gaseous mediator with a well-documented role in the regulation of redox homeostasis, inflammatory responses, and epithelial barrier integrity in the gastrointestinal (GI) tract. One of the key mechanisms underlying its biological activity is protein persulfidation (PSSH)–a reversible post-translational modification of thiol groups that protects proteins from irreversible oxidation and precisely modulates oxidative stress–dependent signaling pathways. Despite growing interest in the role of H_2S in gastrointestinal physiology and pathology, the significance of persulfidation in epithelial function regulation remains poorly understood. As a model for analyzing disturbances in H_2S signaling and persulfidation under conditions of chronic oxidative stress, we employed Barrett's esophagus, a premalignant metaplasia developing in the course of chronic gastroesophageal reflux disease. The application of a combined proteomic–persulfidomic approach to clinical material, complemented by experimental *in vivo* and *in vitro* studies, enabled the identification of early and progressive remodeling of the esophageal epithelial persulfidome. These changes involved numerous proteins associated with the regulation of inflammatory responses, lipid metabolism, and epithelial integrity, which were not detectable using conventional proteomic analyses. Pharmacological application of H_2S donors slowed the progression of pathological changes. The obtained results indicate that impaired H_2S bioavailability, including mitochondrial pool and H_2S-dependent persulfidation constitute a central redox mechanism regulating GI epithelial physiology and susceptibility to pathological reprogramming. Persulfidation emerges as a novel, functionally relevant level of epithelial biology regulation and a potential biomarker and therapeutic platform in GI diseases.

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S9.L8

HYDROGEN SULFIDE ATTENUATES *HELICOBACTER PYLORI*-INDUCED GASTRIC FIBROBLAST ACTIVATION AND CAF-LIKE REPROGRAMMING

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Early prevention of pathological changes underlying gastric cancer (GC) development is critical for limiting malignant progression. We previously showed that *Helicobacter pylori* (Hp) ($cagA^+vacA^+$) promotes GC development by reprogramming gastric fibroblasts toward a CAF-like phenotype capable of inducing aggressive, cancer stem cell (CSC)-related malignant transformation of $LGR5^+$ normal epithelial cells, with the NF-κB/STAT3 axis as a key mediator. Here, we investigated the protective role of hydrogen sulfide (H_2S) against Hp-induced fibroblast reprogramming. Normal human gastric fibroblasts were infected with Hp for 120 h and treated every 24 h with the fast-releasing H_2S donor NaHS (50 – 400 μM). Fibroblast activation markers, NF-κB/STAT3 signaling, Twist expression, sulfur metabolism, proliferation/viability, Hp adhesion, reinfection-induced activation, and Hp sensitivity to metronidazole were assessed. NaHS reduced Hp-induced fibroblast activation, suppressed the pro-inflammatory and pro-tumorigenic phenotype, and was associated with inhibition of the NF-κB/STAT3 axis, reduced Twist, FAP, FSP, IL-6, IL-8 and HGF expression, and modulation of sulfur metabolism while preserving sulfur-enzyme homeostasis. NaHS also partially restored fibroblast viability, reduced Hp adhesion at higher doses, limited reinfection-induced activation, and increased Hp sensitivity to metronidazole without compromising antibiotic efficacy. Preliminary observations with the slow-releasing donor GYY4137 suggested that sustained low-level H_2S exposure may also attenuate Hp-induced activation. Exogenous H_2S attenuates several components of Hp-induced fibroblast reprogramming and may help preserve fibroblast homeostasis by limiting NF-κB/STAT3-driven inflammatory signaling and sulfur-metabolic remodeling. These findings support further investigation of H_2S-based strategies, including slow-releasing donors, as potential approaches to restrain Hp-dependent stromal activation and malignant progression.

S9.P1

FEEDBACK REGULATION OF PANCREATIC EXOCRINE SECRETION IN PIGS

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Pancreatic exocrine secretion is regulated by feedback mechanisms linking the pancreas, bile flow, intestinal mucosa and luminal digestive conditions. In pigs, these mechanisms are particularly relevant because the porcine gastrointestinal tract provides a useful model for studying physiological control of digestion. The aim of this study is to characterise the main feedback pathways involved in the regulation of pancreatic juice secretion in pigs. Chronic experimental models allowed selective withdrawal and controlled reintroduction of pancreatic juice and bile into defined intestinal segments. Exposure of the duodenum and ileum to pancreatic juice suppressed pancreatic enzyme secretion, indicating that both proximal and distal intestinal sensing contribute to pancreatic feedback control. Intraduodenal pancreatin produced a biphasic inhibitory response, supporting the role of protease-dependent luminal regulation. Interruption of bile flow to the duodenum increased pancreatic protein output, whereas intraduodenal bile acid infusion attenuated pancreatic exocrine secretion. Prolonged bile deprivation was associated with sustained pancreatic hypersecretion and reduced biliary leptin output. These findings indicate that pancreatic secretion in pigs is not controlled by a single mechanism, but by interacting pathways involving pancreatic juice, bile acids, cholecystokinin-dependent signalling, distal intestinal feedback and bile-associated metabolic modulation. Together, they coordinate enzyme output with intestinal digestive demand and barrier conditions.

S9.P2

CURCUMIN ACCELERATES HEALING OF EXPERIMENTAL COLITIS *VIA* REACTIVE OXYGEN SPECIES METABOLISM AND MODIFICATION OF GUT MICROBIOTA

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Curcumin, a bioactive component of the *Curcuma longa* plant, exhibits antioxidant, anti-inflammatory, anti-proliferative and chemopreventive efficacy against various gastrointestinal (GI)-tract disorders but its role in pathogenesis of the inflammatory bowel diseases with respect to experimental colitis has been little studied. Important role in the maintenance of gut mucosa is played by reactive oxygen species. Novel investigations have evidenced important role of bacteria living inside GI tract, so called gut microbiota (gut flora). Changes of gut microbiota can affect functions of gut and participate in the gut damage healing. We compared the effect of vehicle and curcumin on the intensity of experimental colitis induced by TNBS (15 mg/kg *per rectum)* in rats. The macroscopic and microscopic damage and the colonic blood flow were determined. The plasma proinflammatory cytokines and SOD2 were analyzed by RT-PCR and Western blot. Lipid peroxidation products were assessed in colonic mucosa, as index of oxidative tissue damage. The composition in the intestinal microflora was determined by NGS in fecal samples. We evidenced decrease of lipid peroxidation products, increase of antioxidative factors and inhibition of inflammatory reaction. Curcumin-treated rats had a higher ratio of *Firmicutes* and *Bacteroidetes* and lower levels of *Proteobacteria* compared to vehicle-control TNBS rats. We concluded that curcumin accelerates the healing of TNBS-induced colitis due to its antioxidative and anti-inflammatory activities. Moreover, curcumin causes changes in the distribution and structure of intestinal microflora and may partially reverse changes in the diversity of gut microbiota in rat colitis.

S9.P3

ASYMMETRIC DIMETHYLARGINIE (ADMA) AGGRAVATES ANTIOXIDATIVE PROPERTIES OF GASTRIC MUCOSA

S. KWIECIEN, D. WOJCIK-GRZYBEK, Z. SLIWOWSKI, U. SZCZYRK,
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Integrity of the gastric mucosa depends upon a variety of factors, such as particularly maintenance of microcirculation and activity of antioxidizing factors, such as superoxide dismutase (SOD) and reduced form of glutathione (GSH). Nitric oxide (NO) is widely accepted as a major factor, involved in control of the gastric blood flow and in the maintenance of gastric mucosal integrity. Inhibition of NO-synthase (NOS) that causes decrease in local NO production, impairs gastric microcirculation and aggravates gastric lesions induced by noxious agents. In physiological conditions, NO is produced by NOS from L-arginine. In pathological conditions L-arginine may be involved in another methabolic pathway produces asymmetric dimethylarginine (ADMA).The aim of our present investigations is explanation details of interplay between ADMA and antioxidative system efficacy on gastric mucosa during stress. Experiments were caried out on male Wistar rats and the gastric lesions was measured by planimetry. Colorimetric assays were used to determine gastric mucosal activity of superoxide dismutase activity (SOD) and reduced glutathione level (GSH). Results of our experiments exhibited protective role of NO to gastric mucosa, revealed by decrease of gastric damage and increase of gastric blood flow. Blockade of endogenous NO production, by ADMA, reversed these effects, impeding SOD and GSH. Additional application of L-arginine restored protective effect antioxidative factors. Capsaicin denervation, reducing endogenous NO production, accelerated harmful action of ADMA on gastric mucosa, restored by application of CGRP. ADMA dose – dependently decreased SOD, GSH in I/R model. We concluded, that ADMA exerted noxious effects in stress – induced gastric lesions, through mechanism involving decrease of antioxidative factors (SOD, GSH), as well as *via* endogenous NO production and action capsaicin – sensitive afferent nerves.

S9.P4

INTERPLAY BETWEEN GLUTATHIONE, SUPEROXIDE DISMUTASE, NITRIC OXIDE AND PROSTAGLANDINS IN THE HEALING OF GASTRIC MUCOSA

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Glutathione is a peptide playing crucial role in the antioxidizing factors activity, such as superoxide dismutase (SOD) and involved in reduction of lipid peroxidation. Nitric oxide (NO) is involved in control of the blood flow in stomach and in the maintenance of gastric mucosal integrity, cooperating with prostaglandins, produced by enzyme cyclooxygenase (COX). Endogenous NO production is stimulated by activity of sensory afferent nerves. The aim of our investigations was to describe interplay between antioxidative system efficacy (GSH with SOD) after destruction of afferent neurons, blockade of COX and gastric damage healing during stress. Experiments were caried out on male Wistar rats and the gastric lesions was measured by planimetry. Colorimetric assays were used to determine reduced glutathione level (GSH), lipid peroxidation products concentration and gastric mucosal activity of superoxide dismutase (SOD). The afferent nerves was destroyed by high doses of capsaicin. COX was blocked by application of rofecoxib (inducible isoform) or SC-560 (constitutive isoform). Results of our experiments exhibited deleterious role of COX inhibition (regardless of the isoform of enzyme) on gastric mucosa healing, augmented by destruction of afferent fibers. Delaying of gastric damage healing was accompanied by depletion of GSH pool in tissues and reduction of SOD activity with increase of lipid peroxidation products concentration. We concluded, that interplay between GSH, SOD, COX-1. COX-2 and NO releasing from the afferent neurons played important role in the maintenance of the gastric mucosal barrier integrity and in acceleration of the healing of gastric damage in experimental stress conditions.

SESSION X

PHYSIOLOGY AND PATHOPHYSIOLOGY OF REPRODUCTION

Thursday (September 24th, 14.00 – 17.30) Room C

Chairman: Associate Professor Urszula Kosior-Korzecka
(Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland)

Vice-Chairman: Professor Anita Franczak
(Department of Animal Anatomy and Physiology, Faculty of Biology and Biotechnology, University of Warmia and Maury in Olsztyn, Olsztyn, Poland)

DETAILED SESSION X SCHEDULE

Opening lecture Thursday (September 24th, 14.00 – 14.40)

S10.L1 LABORATORY ANALYSIS OF SPERM QUALITY IN LIVESTOCK: OVERVIEW AND RECENT ADVANCES. **F.G. Savvulidi, M. Ptacek, L. Langerova** (Department of Animal Science, Faculty of Agrobiology, Food and Natural Resources, Czech University of Life Sciences (CULS) Prague, Czech Republic).

Oral presentations Thursday (September 24th, 14.40 – 16.45)

S10.L2 *14.40 – 14.55* AQUAPORINS IN BOVINE SPERM: PROMISSING TARGET FOR THE MALE REPRODUCTIVE STRATEGY. **K. Michalek[1], S. Zych[2], D. Gaczarzewicz[3], M. Marynowska[1], P. Oberska-Luszczewska[1], A. Syczewski[4], E. Mockus[5], E. Bartkiene[5]** ([1]Department of Physiology, Cytobiology and Proteomics; [2]Department of Microbiology and Biotechnology; [3]Department of Animal Reproduction Biotechnology and Environmental Hygiene, West Pomeranian University of Technology in Szczecin, Poland; [4]Genetic and Animal Husbandry, Mierzyn, Poland; [5]Institute of Animal Rearing Technologies, Veterinary Academy, Lithuanian University of Health Sciences, Kaunas, Lithuania).

S10.L3 *14.55 – 15.10* FUNCTIONAL CHARACTERIZATION OF SPERMATOZOA FROM WHITE AND YELLOW TURKEY (*MELEAGRIS GALLOPAVO*) SEMEN USING FLOW CYTOMETRY. **M. Slowinska, A. Mostek-Majewska, E. Gojlo, E. Drzewiecka, E. Liszewska, A. Bachmura, S. Waldowska, A. Ciereszko** (InLife Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Olsztyn, Poland).

S10.L4 *15.10 – 15.25* THE EFFECT OF EXTREMELY LOW-FREQUENCY ELECTROMAGNETIC FIELD ELF-EMF) ON THE EXPRESSION OF APOPTOSIS-RELATED PROTEIN, BAX AND BCL2 IN THE PORCINE MYOMETRIUM DURING PERI-IMPLANTATION PERIOD. **M. Khodadadi, A. Zmijewska, P. Wydorski, A. Franczak** (Department of Animal Anatomy and Physiology, Faculty of Biology and Biotechnology, University of Warmia and Mazury in Olsztyn, Poland).

15.30 – 16.00 Coffee break

S10.L5 *16.00 – 16.15* THE EFFECT OF PHOENIXIN ON STEROIDOGENIC ENZYME ABUNDANCE IN THE PORCINE ENDOMETRIUM DURING THE PERI-IMPLANTATION PERIOD - **M. Andrzejewska, M. Khodadadi, A. Franczak, A. Zmijewska (**Department of Animal Anatomy and Physiology, Faculty of Biology and Biotechnology, University of Warmia and Mazury in Olsztyn, Poland).

S10.L6 *16.15 – 16.30* ENDOCRINE AND BONE-REMODELING EFFECTS OF LACTOFERRIN AND α-KETOGLUTARATE IN ORCHIDECTOMIZED RATS - **O. Szeredko[1], N. Cybulska[1], R.P. Radzki[2], M. Bienko[2], S. Szymanczyk[2]** ([1]ISSS of Physiologists, Section of Regulatory Processes and Organ Physiology, [2]Department of Animal Physiology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Poland).

S10.L7 *16.30 – 16.45* RELATIONSHIP BETWEEN KISSPEPTIN-10, NEUROKININ B AND DYNORPHIN A IN THE COURSE OF NORMAL AND DELAYED PUBERTY IN EWES. **N. Szysiak[1], U. Kosior-Korzecka[1], M. Gregula-Kania[2], K. Patkowski[2], M. Fila[1], A. Junkuszew[2]** ([1]Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Poland, [2]Department of Animal Breeding and Agricultural Consulting, Faculty of Animal Sciences and Bioeconomy, University of Life Sciences in Lublin, Poland).

Poster Session Thursday (September 24th, 16.45 – 17.30)

S10.P1 LACTOFERRIN AND α-KETOGLUTARATE IMPROVE THE BIOCHEMICAL PROFILE IN ORCHIDECTOMIZED RATS. **N. Cybulska[1], O. Szeredko[1], M. Bienko[2], R. P. Radzki[2], S. Szymanczyk[2]** ([1]ISSS of Physiologists, Section of Regulatory Processes and Organ Physiology, [2]Department of Animal Physiology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Poland).

S10.P2 CHARACTERIZATION OF CIRCULATING MICRO-RNA PROFILES DURING PERIPARTURIENT PERIOD IN SHEEP. **M. Gregula-Kania[1], A. Hahaj-Siembida[2], K. Patkowski[1], A. Junkuszew[1], M. Niedospial[1], A. Nowakiewicz[2], K. Savvulidi Vargova[3], U. Kosior-Korzecka[4]** ([1]Department of Animal Breeding and Agricultural Consulting, Faculty of Animal Sciences and Bioeconomy, University of Life Sciences in Lublin, Poland; [2]Sub-Department of Veterinary Microbiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Poland; [3]Institute of Pathological Physiology, First Faculty of Medicine, Charles University, Prague, Czech Republic, [4]Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland).

S10.P3 VISFATIN (*Nampt*) siRNA INDUCED KNOCKDOWN EFFECT ON BLASTOCYST TRANSCRIPTOME, IMPLANTATION RATE AND OFFSPRING OVARIAN FUNCTION. STUDIES ON NORMAL WEIGHT AND OBESE MICE. **P. Kurowska[1], S. Honda[2], G. Tanaka[2], N Minami[2], A. Rak[1], S. Ikeda[1] (**[1]Laboratory of Physiology and Toxicology of Reproduction, Institute of Zoology and Biomedical Research, Jagiellonian University, Krakow, Poland; [2]Laboratory of Reproductive Biology, Graduate School of Agriculture, Kyoto University, Japan).

Presentation provided by the sponsors Thursday (September 24th, 17.45 – 18.45)

S10.L1

LABORATORY ANALYSIS OF SPERM QUALITY IN LIVESTOCK: OVERVIEW AND RECENT ADVANCES

F.G. SAVVULIDI, M. PTACEK, L. LANGEROVA

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Among the available analytical tools, computer-assisted sperm analysis represents a highly informative technique for motility assessment, enabling detailed quantification of parameters such as velocity and progressive movement. Advanced data-processing approaches, including clustering analysis, provide deeper insight into sperm subpopulations and motility heterogeneity. Ejaculate vorticity assessment, although still a subjective method, is a rapid and inexpensive test that provides valuable information about the fertilizing potential of the ejaculate. Flow cytometry enables high-throughput evaluation of sperm viability and function using classical dyes such as propidium iodide for the assessment of plasma membrane integrity and DNA fragmentation or condensation status, while alternative dyes (e.g., aniline blue) may also be used. However, flow cytometry has limitations, particularly the lack of direct visualization of fluorescence localization within cells, which may complicate the interpretation of assays such as those assessing oxidative stress using CellROX dyes. Functional assays, particularly the hypoosmotic swelling test, remain indispensable for evaluating sperm membrane functionality. In addition, predicting ejaculate freezability is of practical importance, and tests assessing sperm tolerance to dilution may provide useful indicators. Despite the great value of laboratory analyses, these methods provide only preliminary estimates of fertilizing potential and must be validated through field fertility trials, such as artificial insemination.

Acknowledgements: We thank Professor Szabolcs Tamás Nagy, Ph.D., from the Hungarian University of Agriculture and Life Sciences, for his valuable scientific cooperation and guidance.

S10.L2

AQUAPORINS IN BOVINE SPERM: PROMISSING TARGET FOR THE MALE REPRODUCTIVE STRATEGY

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In the light of the recent decline in male reproductive potential and the increase in male fertility disorders, identifying new factors that significantly influence reproductive processes has become particularly important. Analysis of the distribution and expression of aquaporins (AQPs) in spermatozoa may be a useful parameter for assessing the quality of frozen-thawed semen and evaluation of sperm fertilizing ability. To verify this statement, a study was conducted on cryopreserved semen samples from local Animal Breeding and Insemination Stations in the West Pomeranian region of Poland. A detailed analysis was conducted on samples from 29 bulls. Basic quality parameters of frozen-thawed sperm (total and progressive motility, viability, including Hos-test, mitochondrial activity, acrosome integrity, sperm morphology) were determined and utilized for semen categorization. The location and expression of individual AQPs were assessed using immunofluorescence and real-time PCR. The concentration of free amino acids (AA) and biogenic amines (BA), as well as fatty acid (FA) composition were assessed in spermatozoa and seminal plasma. The collected semen samples were classified into two groups: high (H) and low (L) sperm motility. The analysis showed that the percentage of spermatozoa with intense AQP3 staining in the midpiece, and diffuse AQP7 staining in the post-acrosomal region and midpiece, was significantly higher in the H group. Furthermore, AQP3, AQP7 and AQP8 expression showed positive correlations with multiple semen quality parameters and concentrations of selected AA and FA. The results indicate that the analysis of AQPs distribution in spermatozoa, particularly AQP3 and AQP7, may serve as a useful parameter for assessing the quality of cryopreserved semen in future studies.

This work was funded in whole by National Science Center, Poland, grant no. 2021/43/B/NZ9/00204.

S10.L3

FUNCTIONAL CHARACTERIZATION OF SPERMATOZOA FROM WHITE AND YELLOW TURKEY (*MELEAGRIS GALLOPAVO*) SEMEN USING FLOW CYTOMETRY

M. SLOWINSKA, A. MOSTEK-MAJEWSKA, E. GOJLO, E. DRZEWIECKA, E. LISZEWSKA, A. BACHMURA, S. WALDOWSKA, A. CIERESZKO

InLife Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Olsztyn, Poland; m.slowinska@pan.olsztyn.pl

Yellow semen syndrome (YSS) is a reproductive disorder in breeder male turkeys associated with reduced semen fertility and alterations in seminal plasma composition. Previous studies demonstrated extensive proteomic and metabolomic remodeling of YSS seminal plasma, suggesting altered reproductive tract secretory activity. However, the functional status of spermatozoa in YSS remains poorly understood. Therefore, the aim of this study was to characterize functional parameters of spermatozoa from normal white (WS) and YSS semen using flow cytometry (FC). Sperm viability, membrane fluidity, apoptosis, mitochondrial membrane potential (MMP), oxidative stress markers (ROS and NO), and DNA fragmentation were evaluated in 113 ejaculates. Sperm motility parameters were assessed using CASA system. Semen samples were classified as WS or YSS according to seminal plasma protein concentration (<20 mg/mL and >55 mg/mL, respectively). YSS semen showed lower sperm concentration and total sperm number and alterations in sperm motion characteristics including lower VAP, LIN, BCF and ALH values. Unexpectedly, FC analysis did not indicate impaired spermatozoa physiological status in YSS. In contrast, YSS spermatozoa showed higher viability and MMP together with lower apoptosis. No differences were observed in membrane fluidity, ROS+ and NO+ spermatozoa population, or DNA fragmentation. These findings indicate that despite altered kinetic parameters, YSS was not associated with deterioration of sperm functional status measured by FC.

Supported by a grant from the National Science Centre 2022/45/B/NZ9/01360.

S10.L4

THE EFFECT OF EXTREMELY LOW-FREQUENCY ELECTROMAGNETIC FIELD (ELF-EMF) ON THE EXPRESSION OF APOPTOSIS-RELATED PROTEIN, BAX AND BCL2 IN THE PORCINE MYOMETRIUM DURING PERI-IMPLANTATION PERIOD

M. KHODADADI, A. ZMIJEWSKA, P. WYDORSKI, A. FRANCZAK

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The extremely low-frequency electromagnetic field (ELF-EMF), ranging from 1 Hz to 300 Hz, is a growing concern in modern environments. It has been shown that ELF-EMF can disrupt biological processes in the uterus during early pregnancy and induce cellular stress responses, including alterations in apoptosis. Apoptosis is a tightly regulated process essential for tissue homeostasis and successful implantation, with BAX and BCL2 serving as key pro- and anti-apoptotic regulators, respectively. In this study, we investigated the effects of ELF-EMF exposure on the expression of BAX and BCL2 at the mRNA transcript and protein level in the porcine myometrium collected during the peri-implantation period. Slices (100 mg) of porcine myometrium were harvested on days 15-16 of pregnancy, pre-incubated in vitro for 2 hours, and subsequently exposed to the ELF-EMF (50 Hz, 8 mT, 2h). The control group tissue slices were not exposed to the ELF-EMF. After extracting RNA and protein, analysis was conducted on the genes and proteins involved in apoptosis regulation, namely BAX and BCL2. Each experimental group consisted of n=3 biological replicates. Our results (on n=3) indicate that the ELF-EMF treatment led to a significant decrease in protein abundance of BAX ($P \leq 0.05$), with no significant difference observed in BAX mRNA transcript abundance ($P > 0.05$). Furthermore, we observed a tendency towards increased mRNA transcript abundance and protein level of BCL2 in the ELF-EMF-treated myometrium. Our findings indicate alterations in the mRNA transcript and protein abundance of BAX and BCL2, which are key regulators of apoptosis. The observed significant decrease in BAX protein level, alongside a tendency for increased BCL2 mRNA transcript abundance and protein level, suggests a shift towards an anti-apoptotic state in the ELF-EMF-treated myometrium. These preliminary results document the significant impact of ELF-EMF on the regulation of apoptosis-related genes and necessitate further investigation into the effects of ELF-EMF exposure on the tissue.

The study was funded by UWM grant no.12.610.005.110 and partially by PRELUDIUM grant no. 2021/41/N/NZ9/01638 (NCN).

S10.L5

THE EFFECT OF PHOENIXIN ON STEROIDOGENIC ENZYME ABUNDANCE IN THE PORCINE ENDOMETRIUM DURING THE PERI-IMPLANTATION PERIOD

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The peri-implantation period is a critical stage for reproductive success, involving complex interactions between the embryo and the receptive endometrium. Phoenixin (PNX) has recently emerged as a novel regulator of these processes. Recent studies suggest that PNX may participate in the regulation of steroidogenesis, cell proliferation, and endometrial receptivity, thereby potentially influencing embryo implantation and early pregnancy maintenance. The aim of this study was to determine the effect of PNX on the abundance of steroidogenic enzymes in the porcine endometrium on days 15-16 of pregnancy. Endometrial tissue slices were preincubated (2h) and subsequently incubated (12h) with various doses of PNX (10 nM, 100 nM, 1 μM and control without treatment). Protein expression was analyzed using the Western blot method. The results indicate that PNX treatment modulates the abundance of key steroidogenic enzymes in the porcine endometrium. Specifically, Cyp11a1 abundance was significantly downregulated following treatment with 100 nM and 10 nM PNX, suggesting an inhibitory effect on the initial step of steroid hormone biosynthesis. In addition, HSD3B1 showed a significant decrease in abundance after exposure to the 10 nM dose of PNX. A 10 nM dose resulted in an approximately four-fold reduction in aromatase (CYP19A1/P450) abundance, whereas treatment with 100 nM PNX induced expression levels exceeding those observed in the control group. These findings suggest that PNX may regulate estrogen biosynthesis in a concentration-dependent manner and influence the balance of steroid hormone production in the porcine endometrium. Summarized, the obtained results support the hypothesis that PNX plays a regulatory role in endometrial steroidogenesis during early pregnancy and may contribute to maternal–embryo communication. Nevertheless, further studies are required to clarify the molecular mechanisms and signaling pathways underlying the PNX effect in porcine endometrium.

S10.L6

ENDOCRINE AND BONE-REMODELING EFFECTS OF LACTOFERRIN AND A -KETOGLUTARATE IN ORCHIDECTOMIZED RATS

O. SZEREDKO[1], N. CYBULSKA[1], R.P. RADZKI[2], M. BIENKO[2], S. SZYMANCZYK[2]

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Orchidectomy-induced hypogonadism is associated with endocrine dysregulation and impaired bone remodeling. As sex steroids, adrenal hormones, and the RANKL/OPG system regulate skeletal homeostasis, their disruption may promote bone loss. Lactoferrin and α-ketoglutarate (AKG) may modulate these responses under androgen-deficient conditions. Thirty-six 3-month-old male Wistar rats were maintained under standard laboratory conditions and assigned to sham-operated control or bilaterally orchidectomized groups. Following a 7-day recovery period, rats were treated daily for 60 days with physiological saline, lactoferrin (LF) (100 mg/kg BW), AKG (1 g/kg BW), or their combination [lactoferrin (100 mg/kg BW) + AKG (1 g/kg BW)]. Testosterone was administered once weekly at 7 mg/kg BW as a reference replacement therapy. At the end of the experiment, blood samples were collected for hormonal analyses. Orchidectomy induced a clear endocrine shift, characterized by reduced testosterone and estradiol, slightly decreased DHEA, and increased corticosterone. Testosterone replacement restored androgen status and lowered corticosterone to near-control levels. LF, AKG, and LF+AKG did not affect testosterone levels but increased DHEA and estradiol, with the strongest estradiol response after LF+AKG. AKG markedly increased corticosterone, whereas LF attenuated this response in the combined treatment. Orchidectomy increased the RANKL/OPG ratio, indicating enhanced osteoclastogenic signaling, while LF and LF+AKG shifted this ratio toward control values. LF-containing treatments also reduced EBF1, suggesting modulation of bone-remodeling pathways. In summary, combined LF+AKG treatment favorably modulated the endocrine and bone-related response to androgen deficiency, increasing DHEA and estradiol while shifting the RANKL/OPG balance toward a less osteoclastogenic profile.

S10.L7

RELATIONSHIP BETWEEN KISSPEPTIN-10, NEUROKININ B AND DYNORPHIN A IN THE COURSE OF NORMAL AND DELAYED PUBERTY IN EWES

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Kisspeptin (KiSS), neurokinin B (NKB) and dynorphin A (Dyn A) participate in the neuroendocrine regulation of reproductive system development and functioning. However, their role in the onset of sexual maturity and the pathomechanism of delayed puberty have not yet been fully elucidated. The aim of the study was to determine the changes in the kisspeptin-10 (KiSS-10), NKB and Dyn A concentration in blood plasma collected from ewe lambs during puberty, in relation to the first ovulation time, and to examine the relationship between these neuropeptides. Twenty-four ewe lambs were divided into two groups: originated from single (S; n=13) and from twin (T; n=11) pregnancies (the second one with higher risk of delayed puberty). To determine KiSS-10, NKB and Dyn A concentration, blood was collected from all sheep after completion of the 4th, 5th, 6th, 7th, 8th, 9th and 10th months of age (each time for 2 hours, every 15 minutes). S and T reached their first ovulations at 8 and 10 months of age, respectively. The results indicate a positive correlation between changes in plasma KiSS-10 and NKB concentrations with the age of the ewes, both in group S ($r = 0.86$) and in group T ($r = 0.89$). In turn, a negative correlation was found between KiSS-10 and Dyn A ($r = -0.55$ and -0.98 for groups S and T, respectively) and between NKB and Dyn A ($r = -0.89$ and -0.94 for groups S and T, respectively). The initiation of ovarian activity was associated with an increase in plasma KiSS-10 and NKB concentrations and reduced Dyn A levels in both groups, in the period immediately preceding the onset of reproductive activity (at 8 months of age in the S group and 10 months of age in the T group, respectively). Our results suggest that these neuropeptides may regulate the timing of reproductive activity onset in ewes.

S10.P1

LACTOFERRIN AND A-KETOGLUTARATE IMPROVE THE BIOCHEMICAL PROFILE IN ORCHIDECTOMIZED RATS

N. CYBULSKA[1], O. SZEREDKO[1], M. BIENKO[2], R.P. RADZKI[2], S. SZYMANCZYK[2]

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Orchidectomy-induced hypogonadism is an established model of androgen deficiency associated with metabolic imbalance, oxidative stress, and organ-specific dysfunction. Lactoferrin, an iron-binding glycoprotein with anti-inflammatory, antioxidant, and immunomodulatory properties, and α-ketoglutarate (AKG), a key tricarboxylic acid cycle intermediate involved in energy metabolism and redox regulation, may attenuate hypogonadism-related metabolic disturbances. The study involved 36 male Wistar rats, approximately 3 months old. The control group (CON-SHO) underwent a sham operation, whereas the experimental groups were subjected to bilateral orchidectomy (ORX) under isoflurane anesthesia. After a 7-day convalescence period, rats were treated daily for 60 days with physiological saline (CON-SHO and ORX), lactoferrin (100 mg/kg BW; ORX-LF), α-ketoglutarate (1 g/kg BW; ORX-AKG), or their combination (lactoferrin (100 mg/kg BW) plus AKG (1 g/kg BW); ORX-AKG-LF). Testosterone was administered once weekly at 7 mg/kg BW as a reference treatment. Blood samples were collected for biochemical analyses. Orchidectomy induced moderate metabolic and hepatobiliary alterations, including changes in the lipid profile, glucose concentration, ALT and ALP activities, and bilirubin indices. Testosterone partially attenuated these androgen-deficiency-related changes. Combined LF+AKG administration exerted the most evident effect on lipid metabolism, improving the lipid profile. Glucose concentration increased slightly in ORX rats and more markedly in the LF-, AKG-, and LF+AKG-treated groups. Moreover, LF+AKG reduced total bilirubin to values comparable to sham-operated controls. The increased ALP activity without a parallel GGT elevation suggests an extrahepatic (bone- or intestine-derived) origin of ALP rather than cholestatic liver injury. In summary, combined LF+AKG treatment showed the most favorable biochemical profile in orchidectomized rats.

S10.P2

CHARACTERIZATION OF CIRCULATING MICRO-RNA PROFILES DURING PERIPARTURIENT PERIOD IN SHEEP SESSION: PHYSIOLOGY AND PATHOPHYSIOLOGY IN REPRODUCTION

M. GREGULA-KANIA[1], A. HAHAJ-SIEMBIDA[2], K. PATKOWSKI[1], A. JUNKUSZEW[1], M. NIEDOSPIAL[1], A. NOWAKIEWICZ[2], K. SAVVULIDI VARGOVA[3],U. KOSIOR-KORZECKA[4]

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MiRNAs play important roles in regulating many pathways and essential biological processes, including innate immunity, inflammatory responses, and hematopoiesis, as well as cancer development. The objective of the present study, therefore, was to compare the plasma concentrations of mir146a and mir155 and in healthy ewes from 2 weeks before to 2 weeks after parturition.

Material and methods: Ewes were induced to ovulate using intravaginal sponges saturated with 20 mg fluorogestone acetate for 16 days. At 56–58 h after sponge removal, we performed laparoscopic intrauterine artificial insemination of all synchronized ewes. The blood samples were collected 14 days before parturition, a few hours after parturition, and 14 days after parturition. Total RNA, including microRNAs, was extracted from leucocytes using TRI reagent and 100 ng was reverse transcribed using the High-Capacity cDNA Reverse Transcription Kit with RNase In-hibitor (Thermo Fisher Scientific). TaqMan-based PCR with specific probes was performed on 7900HT Fast Real-Time PCR and QS7Pro instruments (both from Thermo Fisher Sci-entific). GAPDH and RNU44 were used as reference genes. The miRNA/mRNA expression was calculated by the 2ΔCT algorithm from target and reference CT values. MiR-146a functions in regulating pro-inflammatory immune responses by limiting excessive stimulation, thereby maintaining a homeostatic state of the immune system. miR-155 has potent regulatory functions in a wide range of immune cells, including T cells, B cells, and dendritic cells (DCs), by targeting specific mRNAs.

S10P3

VISFATIN (*NAMPT*) SIRNA INDUCED KNOCKDOWN EFFECT ON BLASTOCYST TRANSCRIPTOME, IMPLANTATION RATE AND OFFSPRING OVARIAN FUNCTION. STUDIES ON NORMAL WEIGHT AND OBESE MICE.

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Adipokines link metabolic status with reproductive function. Visfatin, increased in obesity, supports oocyte maturation, while our previous studies showed that its deficiency reduces blastocyst formation in obese mice. Here, we investigated the effects of *Nampt* silencing during early embryogenesis on blastocyst transcriptome, implantation, offspring number, and ovarian function in normal-weight and high-fat-diet-induced obese mice. *Nampt* was silenced by siRNA electroporation at the one-cell stage, and embryos were cultured to the blastocyst stage for RNA-seq analysis. Differentially expressed genes (DEGs; padj<0.05) were analyzed using gProfiler and DAVID. Implantation rate and offspring number were assessed, and ovaries from 17.5 dpc offspring were analyzed by real-time PCR for genes related to steroidogenesis, folliculogenesis/oogenesis, proliferation, and apoptosis (n=5). In normal-weight mice, *Nampt* silencing resulted in 73 upregulated and 24 downregulated genes associated with cell death, glycolysis, metabolism, development, cytoskeleton organization, and cell junctions, with enrichment of estrogen signaling, p53 signaling, and progesterone-mediated oocyte maturation pathways. In obese mice, only three DEGs were identified. *Nampt* silencing did not affect implantation rate or offspring number in normal-weight mice but altered ovarian gene expression in offspring, including increased *Bmp15* and *Cyp17a1* and decreased *Hsd3b* transcripts. These findings indicate that visfatin contributes to early embryonic development and may exert long-lasting effects on ovarian function in female offspring, while its limited transcriptomic impact in obesity suggests a potential compensatory role in reproduction under metabolic stress.

Funding: NCN ETIUDA and PAN-JSPS.

SESSION XI

ANIMAL PHYSIOLOGY AND PATHOPHYSIOLOGY

Saturday (September 26th, 8.30 – 13:45) Room C

Chairman Professor Ewa Tomaszewska
(Department of Animal Physiology, Faculty of Veterinary Medicine,
University of Life Sciences in Lublin, Lublin, Poland)

Vice-Chairman Associate Professor Marta Wojcik
(Department of Epizootiology and Clinic of Infectious Disease
Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland)

Vice-Chairman: Associate Professor Anna Pedzinska-Betiuk
(Department of Experimental Physiology and Pathophysiology,
Faculty of Pharmacy, Medical University of Bialystok, Bialystok, Poland)

DETAILED SESSION XI SCHEDULE

Opening lecture Saturday (September 26th 8.30 – 9:10)

S11.L1 *8.30 – 9.10* THE ROLE OF MITOCHONDRIA IN ANIMAL HEALTH AND DISEASE: NUTRACEUTICAL STRATEGIES FOR MITOCHONDRIAL PROTECTION. **V. Longo (**Institute of Agricultural Biology and Biotechnology, CNR, Pisa, Italy; Honorary Professor University of Life Sciences, Lublin).

Oral presentations Saturday (September 26th 9.10 – 12.30)

S11.L2 *9.10 – 9.25* AVERGE HORSE? AI HALLUCINATION? FLAWS IN EXERCISE PHYSIOLOGY MODELS. **A. Cywinska** (Nicolaus Copernicus University in Torun, Faculty of Biological and Veterinary Sciences, Department of Basic and Preclinical Sciences).

S11.L3 *9.25 – 9.40* THE EFFECT OF EXTRACELLULAR METABOLITES OF CANDIDA ALBICANS ON THE SECRETORY ACTIVITY OF OVINE PINEAL GLAND–*EX VIVO* STUDY. **A.P. Herman[1], J. Bochenek[1], K. Wojtulewicz[1], M. Sokolowski[1], O. Zawadzki[1], E. Kalinowska[2], A. Herman[2]** ([1]The Kielanowski Institute of Animal Physiology and Nutrition, Polish Academy of Sciences, Jablonna, Poland; [2]Chair of Drug and Cosmetics Biotechnology, Faculty of Chemistry, Warsaw University of Technology, Warsaw, Poland).

S11.L4 *9.40 – 9.55* DIETARY FAT COMPOSITION REGULATES SKIN LIPIDOMIC PROFILE. **J. Wisniewska[1], A. Bachmura[1], M. Kopcewicz[1], S. Machcinska-Zielinska[1], K. Walendzik[1], M. Radkiewicz[2], A. Jurgoski[1] (**[1]InLife Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Olsztyn, Poland; [2]Mass Spectrometry Facility, Institute of Biochemistry and Biophysics, Polish Academy of Sciences, Warsaw, Poland).

10.00 – 10.30 Coffee break

S11.L5 *10.30 – 10.45* EFFECTS OF CALORIC RESTRICTION AND AMPK INHIBITION ON HEMATOLOGICAL PARAMETERS IN RATS WITH METABOLIC SYNDROME. **T. Jasenovec[1], I. Szadvari[1], P. Balis[2], M. Kvandova[2], J. Radosinska[2]** ([1]Institute of Physiology, Faculty of Medicine, Comenius University in Bratislava, Slovakia; [2]Centre of Experimental Medicine, Slovak Academy of Sciences, Institute for Heart Research, Bratislava, Slovakia).

S11.L6 *10.45 – 11:00* SPATIALLY RESOLVED LIPIDOMIC AND AMINO ACID HETEROGENEITY OF GOOSE SKELETAL MUSCLE FIBERS REVEALED BY TOF-SIMS. **M.E. Skalska, D. Wojtysiak** ([1]Department of Medical Physics, M. Smoluchowski Institute of Physics, Faculty of Physics, Astronomy and Applied Computer Science, Jagiellonian University, Krakow, Poland; [2]Centre for Theranostics, Jagiellonian University, Krakow, Poland;[3]Department of Genetics, Animal Breeding and Ethology, University of Agriculture in Krakow, Krakow, Poland).

S11.L7 *11.00 – 11.15* FERMENTED WHEAT NUTRACEUTICAL ATTENUATES OXIDATIVE DAMAGE AND MODIFIES DETOXIFICATION ENZYMES IN RAT HEPATOCARCINOGENESIS MODEL. **B. Szymczak[1], M. Wojcik[2], L. Pozzo[3], A. Vornoli[3]** ([1]Sub-Department of Pathophysiology, Department of Preclinical Veterinary Sciences, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Poland; [2]Oncology Lab, Department of Epizootiology and Clinic of Infectious Diseases, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Poland; [3]Institute of Agricultural Biology and Biotechnology, National Research Council, Pisa, Italy).

S11.L8 *11.15 – 11.30* PET MICROPLASTIC EXPOSURE ALTERS THE ADRENAL CORTEX PROTEOME IN A PORCINE *IN VIVO* MODEL. **A. Kurzynska , P. Szymkow, K. Mierzejewski, B.S. Sahin, S. Zaidi, A.S. Klinkby, B. Nair, A. Rogowska-Wrzesinska, I. Bogacka.** ([1]Department of Animal Anatomy and Physiology, Faculty of Biology and Biotechnology, University of Warmia and Mazury in Olsztyn, Poland; [2]Department of Biochemistry and Molecular Biology, University of Southern Denmark, Odense, Denmark).

S11.L9 *11.30 – 11.45* PET MICROPLASTICS ALTER THE GLOBAL PROTEOMIC PROFILE OF THE PORCINE KIDNEY. **P. Szymkow[1], K. Mierzejewski[1], A. Kurzynska[1], M. Golubska[2], I. Galecka[1], J. Calka[1], P. Borrajo[3], M. Pazos[3], M. Carrera[3], I. Bogacka[1]** ([1]University of Warmia and Mazury in Olsztyn, Poland; [2] InLife Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Olsztyn, Poland; [3]Department of Food Technology, Institute of Marine Research (IIM), Spanish National Research Council (CSIC), Vigo, Spain).

S11.L10 *11.45 – 12.00* EFFECTS OF HIGH DIETARY HYBRID AND POPULATION RYE INCLUSION ON INFLAMMATORY-RELATED CYTOKINE CONCENTRATIONS IN FATTENING PIGS. **M. Batorski[1], S. Kowalik[1], J.L. Valverde Piedra[2], T. Schwarz[3]** ([1]Department of Animal Physiology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland; [2]Department of Pharmacology, Toxicology and Environmental Protection, University of Life Sciences, Lublin, Lublin, Poland; [3]Department of Animal Genetics, Breeding and Ethology, Faculty of Animal Sciences, University of Agriculture in Krakow, Krakow, Poland).

S11.L11 *12.00 – 12.15* PANCREATIC AND INTESTINAL RESPONSES TO RYE-BASED DIETS IN PIGS. **K. Bieda[1], P. Prokopiak[1], S. Szymanczyk[2], P. Dobrowolski[3], T. Schwarz[4], J.L. Valverde Piedra[5]**. ([1]University of Life Sciences in Lublin, Faculty of Veterinary Medicine, ISSS of Physiologists, Section of Regulatory Processes and Organ Physiology, Poland; [2]University of Life Sciences, Lublin, Faculty of Veterinary Medicine, Department of Animal Physiology, Poland; [3]Maria Curie-Sk[3]odowska University, Lublin, Department of Comparative Anatomy and Anthropology, Poland; [4]University of Agriculture, Krakow, Faculty of Animal Science, Department of Genetics, Animal Breeding and Ethology, Poland; [5]University of Life Sciences in Lublin, Faculty of Veterinary Medicine, Department of Pharmacology, Toxicology and Environmental Protection, Lublin, Poland).

S11.L12 *12.15 – 12.30* ASSESSMENT OF THE CYTOTOXIC RESPONSE OF HUMAN BJ FIBROBLASTS EXPOSED TO POLYSULFONE MEMBRANE EXTRACTS. **D. Nguyen Ngoc[1], P. Kazimierczak[2], T. Pereira-Riveros[3], D. Wojtowicz[4,5], E. Stodolak-Zych[4], J. Wessely-Szponder[1]** ([1]Sub-Department of Pathophysiology, Department of

Preclinical Veterinary Sciences, University of Life Sciences in Lublin, Lublin, Poland, [2]Department of Tissue Engineering and Regenerative Medicine, Medical University of Lublin, Lublin, Poland; [3]Laboratory of Molecular Microbiology & Antimicrobials, Department of Pathology and Experimental Therapeutics, Faculty of Medicine, University of Barcelona, Barcelona, Spain; [4]Department of Biomaterials and Composites, Faculty of Materials Science and Ceramics, AGH University of Krakow, Krakow, Poland, [5]Clinical Department of Anaesthesiology and Intensive Care, University Hospital, Krakow, Poland).

Poster Session Saturday (September 26th 12.30 – 13:45)

S11.P1 THE EFFECT OF DIET ON THE PROFILE OF GUT MICROBIOTA IN A MOUSE MODEL. **G. Swiderska-Kolacz[1], J. Czerwik-Marcinkowska[1], S. Zmorzynski[2], G. Wrobel[1], M. Trojak[1], E. Skowron[1], L. Stachera[1], E. Muszkat[1], S. Popek-Marciniec[2], A. Wojciechowska[3]** ([1]Institute of Biology, Jan Kochanowski University, Kielce, Poland; [2]Institute of Human Sciences, Laboratory of Genetics, Academy of Zamosc, Zamosc, Poland; [3]Department of Geobotany and Landscape Planning, Nicolaus Copernicus University, Torun, Poland).

S11.P2 DIETARY CASEIN ATTENUATES CADMIUM-INDUCED INTESTINAL INJURY IN RATS ANIMAL PHYSIOLOGY AND PATHOPHYSIOLOGY. **P. Prokopiak[1], K. Bieda[1], S. Szymanczyk[2], H. Kudhur[3], A. Chalabis-Mazurek[3], J.L. Valverde Piedra[3]** ([1]University of Life Sciences in Lublin, Faculty of Veterinary Medicine, ISSS of Physiologists, Section of Regulatory Processes and Organ Physiology, Lublin, Poland, [2]University of Life Sciences in Lublin, Faculty of Veterinary Medicine, Department of Animal Physiology, Lublin, Poland, [3]University of Life Sciences in Lublin, Faculty of Veterinary Medicine, Department of Pharmacology, Toxicology and Environmental Protection, Lublin, Poland).

S11.P3 DIET-RELATED INTESTINAL AND ENTERIC NERVOUS SYSTEM ADAPTATIONS IN MALLARD, TUFTED DUCK AND GREEN-WINGED TEAL. **L. Janicka[1], A. Dajnowska[1], C. Osiak-Wicha[1], K. Kras[1], M. Flis[2], K. Wozniak[1], M.B. Arciszewski[1]** ([1]Department of Animal Anatomy and Histology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland; [2]Department of Ethology and Wildlife Management, Faculty of Animal Sciences and Bioeconomy, University of Life Sciences in Lublin, Lublin, Poland).

S11.P4 ASSESSMENT OF ANGIOTENSIN II RECEPTORS AT1R, AT2R AND ENDOTHELIAL NITRIC OXIDE SYNTHASE IMMUNOREACTIVITY IN THE ABDOMINAL AORTA OF PIGLETS BORN β-HYDROXY-β-METHYLBUTYRATE (HMB) – SUPPLEMENTED SOWS. **M. Pawlowska-Olszewska, M. Morawski, I. Puzio, E. Tomaszewska** (Department of Animal Physiology, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Lublin, Poland).

S11.P5 COMPARATIVE PHARMACOKINETICS OF CANNABIDIOL AND ITS METABOLITES IN HORSES FOLLOWING OIL- VERSUS AQUEOUS-BASED ORAL DELIVERY. **A .Chlodowska, K. Sroda, A. Cywinska (**Nicolaus Copernicus University in Torun, Faculty of Biological and Veterinary Sciences, Department of Basic and Preclinical Sciences, Torun, Poland).

S11.P6 THE EFFECT OF ADROPIN ON THE LOCALIZATION OF AT1 AND AT2 ANGIOTENSIN RECEPTORS AND ADROPIN IN THE ABDOMINAL AORTIC WALL OF FEMALE RATS UNDER CONDITIONS OF OVARIECTOMY. **M. Pawlowska-Olszewska, M. Morawski, M. Wojcik, E.H. Predka, E. Tomaszewska, I. Puzio** (Department of Animal Physiology, Faculty of Veterinary Medicine, University of Life Sciences, Lublin, Poland).

S11.P7 AGE-RELATED DIFFERENCES IN THE DISTRIBUTION OF SELECTED POPULATIONS OF IMMUNOREACTIVE ENTEROENDOCRINE CELLS IN THE BOVINE ABOMASUM AND SMALL INTESTINE. **A. Dajnowska[1], L. Janicka[1], K. Wozniak[1], C. Osiak-Wicha[1], K. Kras[1], I. Luszczewska-Sierakowska[2], M.B. Arciszewski[1]** ([1]Department of Animal Anatomy and Histology, Faculty of Veterinary Medicine, University of Life Sciences, Lublin, Lublin, Poland; [2]Department of Correct Clinical and Imaging Anatomy, Faculty of Medical Sciences, Medical University of Lublin, Lublin, Poland).

S11.P8 MICROPLASTIC EXPOSURE ALTERS ADIPOSE TISSUE FUNCTION:INSIGHTS FROM A PORCINE *IN VIVO* STUDY. **S. Zaidi, A. Kurzynska, P. Szymkow, K. Mierzejewski, I. Bogacka** (University of Warmia and Mazury in Olsztyn, Faculty of Biology and Biotechnology, Department of Animal Anatomy and Physiology, Olsztyn, Poland).

S11.P9 A NONINVASIVE JACKETED SYSTEM FOR MONITORING CARDIORESPIRATORY PARAMETERS AND LOCOMOTOR ACTIVITY IN ANIMALS IN NON-CLINICAL RESEARCH: A NOVEL NON-INVASIVE TECHNOLOGY SUPPORTING REDUCTION AND REFINEMENT IN *IN VIVO* RESEARCH. **C. Simon[1], M. Wozniak[2]** ([1]R&D, ETISENSE SAS, Lyon, France; [2]ANIMALAB, distributor of animal models & lab equipment, Wysogotowo, Poland).

S11.L1

THE ROLE OF MITOCHONDRIA IN ANIMAL HEALTH AND DISEASE: NUTRACEUTICAL STRATEGIES FOR MITOCHONDRIAL PROTECTION

V. LONGO

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Mitochondria play a central role in animal physiology by regulating energy production, redox balance, calcium homeostasis, apoptosis, and cellular adaptation to metabolic stress. Mitochondrial dysfunction is increasingly recognized as a key mechanism involved in oxidative stress, inflammation, aging, reproductive impairment, metabolic disorders, and several pathological conditions affecting animal health. In this context, nutraceuticals and natural bioactive compounds have gained growing interest as potential modulators of mitochondrial function. Recent evidence highlights the biological relevance of dietary polyphenols in animals, not only as antioxidants, but also as compounds able to influence cellular signaling, mitochondrial metabolism, and endogenous defense systems. However, their effectiveness depends strongly on metabolism, bioavailability, tissue distribution, and interaction with the gut microbiota. In parallel, several natural mitochondria-targeting substances have been proposed as promising tools to preserve mitochondrial integrity by improving antioxidant defenses, reducing reactive oxygen species production, supporting mitochondrial membrane potential, and modulating pathways involved in mitochondrial biogenesis and quality control. Natural products may exert multifaceted effects on mitochondrial dysfunction through the regulation of oxidative phosphorylation, mitophagy, mitochondrial dynamics, inflammation, and apoptosis. These mechanisms suggest that nutraceutical strategies could contribute to maintaining mitochondrial health and preventing or attenuating disease processes in animals. A better understanding of dose-response relationships, species-specific metabolism, and in vivo efficacy is essential to translate these findings into practical applications in veterinary medicine, animal nutrition, and livestock production. Overall, nutraceutical modulation of mitochondrial function represents a promising approach to support animal health, improve resilience to stress, and counteract pathophysiological conditions associated with mitochondrial damage.

S11.L2

AVERGE HORSE? AI HALLUCINATION? FLAWS IN EXERCISE PHYSIOLOGY MODELS

A. CYWINSKA

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Exercise physiology has traditionally relied on the models describing changes that occur in response to certain types of exercise in certain group of animals or humans. Rodent models are commonly used for examining specific mechanisms in strictly controlled conditions on the most homogenous group. Investigations of the processes in equine, canine and human athletes subjeted to training and competitions were closer to the reality and allowed for more practical conclusions. Both types of studies require carefully selected study group, homogenous enough to detect important species-specific and exercise-specific issues. Currently, new trends for generalization and population-designed frameworks occured despite the fact that comparative evidences had cleary indicated the variations among real athletic groups. Moreover, artificial intelligence (AI) systems seem to assist these analyses. Although heplful, AI tools through hallucination-like synthesis may present statistically probable but physiologically inaccurate generalizations as universal truths. Large language models (LLMs) may further amplify these unjustified conclusions. Such actions pose a risk of ignoring biologically meaningful variation, particularly when comparative data are sparse or context-dependent. Thus, traditional group selection and source-grounded reasoning rather than probabilistic averaging still seem the best way for the research on exercise physiology, regardless of the species.

S11.L3

THE EFFECT OF EXTRACELLULAR METABOLITES OF *CANDIDA ALBICANS* ON THE SECRETORY ACTIVITY OF OVINE PINEAL GLAND – *EX VIVO* STUDY

A.P. HERMAN[1], J. BOCHENEK[1], K. WOJTULEWICZ[1], M. SOKOLOWSKI[1], O. ZAWADZKI[1], E. KALINOWSKA[2], A. HERMAN[2]

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C. albicans, the most clinically significant fungal species, can invade the central nervous system (CNS) and may contribute to neurodegenerative disorders associated with impaired melatonin secretion. Previous studies showed that inflammatory mediators can directly impair melatonin secretion in pituitary cells, while the presence of toll-like receptors (TLRs) in this gland suggests that its secretory activity may also be regulated by pathogen-associated molecular patterns. Therefore, the present study aimed to determine whether extracellular metabolites of *C. albicans* affect pituitary cells and melatonin secretion. *C. albicans* was cultured overnight and the collected culture supernatant (CA) was used in the concentrations of 5 or 10% v/v to treat ovine pineal gland explants incubated with norepinephrine (NE). After 3 h of incubation, melatonin concentration and gene expression were analyzed using HPLC and Real-Time PCR, respectively. Addition of *C. albicans* supernatant abolished NE-induced expression of the key melatonin synthesis enzymes aralkylamine N-acetyltransferase and hydroxyindole-O-methyltransferase, while 10% CA also inhibited NE-stimulated melatonin release in pineal explants. In contrast, CA did not affect the expression of genes encoding tryptophan hydroxylase and aromatic L-amino acid decarboxylase. Moreover, pineal explants expressed both TLR2 and TLR4 receptors, but *C. albicans* treatment increased only TLR2 gene expression at the higher supernatant concentration. Our study demonstrated that metabolites of *C. albicans* can impair pineal gland secretory activity, suggesting a potential mechanism underlying melatonin secretion disorders and highlighting the need for further *in vivo* studies on fungal effects on the neuroendocrine system.

S11.L4

DIETARY FAT COMPOSITION REGULATES SKIN LIPIDOMIC PROFILE

J. WISNIEWSKA[1], A. BACHMURA[1], M. KOPCEWICZ[1], S. MACHCINSKA-ZIELINSKA[1], K. WALENDZIK[1], M. RADKIEWICZ[2], A. JURGONSKI[1]

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Lipids play a crucial role in maintaining structure and function of the skin. Dietary fat composition influences skin lipid homeostasis and may affect skin physiology and barrier function. The aim of our study was to examine whether partial replacement of lard in Western diet (WD) with vegetable oils modulates the skin lipidome. C57BL/6 mice were subjected to an 8-week dietary intervention and assigned to four groups: normal, chowdiet (ND), lard-based WD (ctrl-WD), WD with 50% (by weight) rapeseed oil substitution (WD-Ro), or WD with 50% (by weight) palm oil substitution (WD-Pa). Lipidomics analysis identified 857 lipid species representing over 30 lipid classes across four major lipid categories: glycerolipids, glycerophospholipids, sphingolipids, and fatty acid-derived lipids. Among all experimental diets, WD-Ro induced the most extensive lipidomic remodeling, showing the highest number of significantly altered lipid species. The most increased lipids belonged to the triacylglycerols (TGs) class , whereas phosphatidylcholine (PC) were the most markedly decreased. Moreover, WD-induced lipidomic changes exhibited sex-dependent differences, with females showing more dynamic remodeling of skin lipid composition than males. Our findings highlight the importance of dietary fat composition as a regulator of skin lipid homeostasis and suggest that vegetable oils substitution may differentially influence skin metabolic adaptation.

Acknowledgements: This work was supported by the National Science Centre, Poland, Opus25 (2023/49/B/NZ9/01925).

S11.L5

EFFECT OF CALORIC RESTRICTION AND AMPK INHIBITION ON HEMATOLOGICAL PARAMETERS IN RATS WITH METABOLIC SYNDROME

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Introduction: Caloric restriction (CR) is a widely used intervention for reducing weight and improving metabolic syndrome (MetS) and its comorbidities. Its effects may involve AMPK, a central energy sensor downregulated under inflammatory and diabetic conditions. Commonly measured hematological parameters might represent accessible markers of MetS progression. This study examined the impact of CR in a high-fat diet-induced MetS model on complete blood count and erythrocyte (RBC) deformability, and evaluated whether these changes relate to AMPK activity using dorsomorphin as its inhibitor. Methods: 8-week-old male Wistar rats were divided into following 4 groups: Control, HFD (high fat diet for 10 weeks), HFD-CR (high fat diet for 6 weeks; standard diet with 20% CR for 2 weeks, and 35% CR for another 2 weeks) and HFD-CRd (HFD-CR group treated with dorsomorphin (2 x 1.5 mg/kg/week during CR). RBC deformability was assessed by ektacytometry. Results: A high-fat diet significantly decreased hematocrit, RBC count, and hemoglobin concentration, along with an increase in monocyte count, suggesting a tendency toward anemia linked to chronic inflammation. CR reverted these changes and improved RBC deformability at the higher shear stress levels. CR reduced both the count and percentage of neutrophils and increased lymphocyte percentage. However, these effects were not observed in the HFD-CRd group. In HFD-CRd group, derived inflammatory indices were also elevated. Conclusion: Although complete blood count parameters are not used for the direct diagnosis of MetS, some of them may provide a valuable insight into inflammatory and hematological alterations associated with MetS, which may aid in early detection, monitoring, and risk assessment. CR demonstrated normalization of several hematological parameters and RBC deformability with possible involvement of AMPK.

Supported by APVV-24-0225, APVV-22-0154, and VEGA 1/0634/25.

S11.L6

SPATIALLY RESOLVED LIPIDOMIC AND AMINO ACID HETEROGENEITY OF GOOSE SKELETAL MUSCLE FIBERS REVEALED BY TOF-SIMS

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Skeletal muscle tissue is characterised by substantial morphological, biochemical and metabolic heterogeneity, reflecting the presence of oxidative and glycolytic muscle fibre types. These fibre types differ in mitochondrial content, lipid metabolism, membrane composition, and amino acid turnover. Time-of-Flight Secondary Ion Mass Spectrometry (ToF-SIMS) enables label-free molecular imaging with high spatial resolution and may provide insights into the biochemical organisation of skeletal muscle fibres.

This study aimed to investigate the spatial distribution of lipid- and amino acid-related ions in goose skeletal muscle (*pectoralis superficialis*) tissue and to identify molecular differences between distinct muscle fibre types. Prior histochemical staining enabled visualisation of heterogeneous muscle fibre composition and facilitated interpretation of molecular imaging results. generated for selected low- and high-mass ions associated with fatty acids, phospholipids, and amino acid-related fragments. Distinct localisation patterns were observed for several lipid-associated ions, including oleic acid FA(18:1) ($C_{18}H_{33}O_2^-$), linolenic acid FA(18:3) ($C_{18}H_{29}O_2^-$), palmitic acid FA(16:0) ($C_{16}H_{31}O_2^-$), and arachidonic acid-related fragments ($C_{20}H_{37}O_2^-$). Furthermore, phospholipid- and sphingolipid-related ions, including phosphatidylethanolamine and phosphatidylcholine fragments (PO_3^-, $PO_4H_2^-$, $C_{36}H_{69}PNO_6^-$, $C_{39}H_{75}PNO_8^-$), demonstrated heterogeneous spatial distributions within the analysed tissue sections. Regions enriched in phospholipid-related signals correspond to oxidative muscle fibres characterised by different membrane content of lipids. In addition, amino acid- and nitrogen-containing fragments, including $C_2H_3N_2O^-$, $C_4H_8N_2O_2^-$, and C3H6N2O3$^-$, exhibited distinct regional localisation, indicating metabolic differences between muscle fibre types. The observed molecular heterogeneity was reproducible across multiple analysed sections. The obtained results demonstrate that ToF-SIMS imaging enables discrimination of biochemically distinct regions within goose skeletal muscle tissue and may serve as a valuable tool for spatial metabolomic characterisation of muscle fibre heterogeneity. The presented approach provides new insight into lipid and amino acid organisation in avian skeletal muscle and may contribute to future studies on muscle physiology and metabolic specialisation.

S11.L7

FERMENTED WHEAT NUTRACEUTICAL ATTENUATES OXIDATIVE DAMAGE AND MODIFIES DETOXIFICATION ENZYMES IN RAT HEPATOCARCINOGENESIS MODEL

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Chronic liver injury promotes hepatocarcinogenesis through persistent oxidative stress, impaired detoxification and altered cytochrome (CYP) P450-dependent bioactivation of xenobiotics. Nutraceuticals may influence these processes not by direct antitumor activity, but by modifying the pro-carcinogenic metabolic background of the liver. This study evaluated the effect of Lisosan G (LG), a fermented wheat-derived nutraceutical, on redox status and detoxification capacity in an experimental model of hepatocellular carcinoma (HCC). Wistar rats (n=90) were divided into control, partial hepatectomy (PH) and HCC groups and received standard diet or diet supplemented with 2.5% or 5% LG. HCC was induced by PH followed by exposure to N-nitrosodiethylamine in drinking water. Liver samples were processed for microsomal and cytosolic fraction isolation. The activities of ECOD, EROD, AnH, BqD, GST, DT-diaphorase, CAT and SOD were determined, and oxidative damage was assessed by hepatic MDA and protein carbonyl levels. LG showed no relevant effect in healthy liver, whereas in PH liver it increased selected microsomal monooxygenase activities and GST, indicating stimulation of adaptive metabolic responses during regeneration. In HCC, standard diet animals showed a pronounced decrease in ECOD, EROD, GST and SOD activity, elevation of AnH activity and the highest concentrations of MDA and protein carbonyls. LG supplementation increased ECOD and EROD activity, enhanced GST, catalase and SOD activity, reduced AnH activity and lowered lipid and protein oxidation markers. These findings support further evaluation of fermented nutraceuticals as modulators of liver redox physiology in conditions associated with increased HCC risk.

S11.L8

PET MICROPLASTIC EXPOSURE AFFECT THE ADRENAL CORTEX PROTEOME IN A PORCINE *IN VIVO* MODEL

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Polyethylene terephthalate (PET), widely used in food packaging, can release microplastic particles (MPs) during storage, which have been detected in human blood and tissues. Given their potential to disrupt physiological processes, this study investigated whether oral PET MP exposure alters the adrenal gland proteome in immature gilts. Adrenal glands from 7–8-month-old crossbred pigs (Large White × Polish Landrace; N=5/group) were analyzed following 4-weeks of oral exposure to PET MPs: control (empty capsules), low dose (0.1 g/day), and high dose (1 g/day). Proteins were extracted, digested with trypsin, and analyzed by LC–MS/MS (Evosep One–Orbitrap Astral, DIA). Data were processed in Spectronaut (directDIA), and differential protein abundance was assessed using PolySTest with Benjamini–Hochberg correction (FDR < 0.05). Exposure to PET MPs resulted in minimal proteomic changes at LD, with only one protein upregulated and one downregulated. In contrast, HD exposure led to substantial alterations, with 23 proteins upregulated and 115 downregulated. Differentially expressed proteins were predominantly associated with mitochondrial and endoplasmic reticulum function, protein synthesis, and exocytosis. These findings suggest that HD PET MP exposure disrupts adrenal cortex physiology by inducing mitochondrial and ER stress and impairing protein synthesis. Additionally, altered expression of exocytosis-related proteins may hinder the delivery of enzymes and lipids, potentially affecting hormone secretion.

The experiment and analyses is supported by the National Science Centre, Poland, grant no. 2021/43/B/NZ7/01093; The internship in Denmark is supported by the Reproductive Biology Society; MS and proteomics research at the University of Southern Denmark in Odense is supported by the Novo Nordisk Foundation through the INTEGRA research infrastructure (grant NNF20OC0061575) and by the Danish Agency for Higher Education and Science through PLATO: the Danish National Mass Spectrometry Platform for Proteomics and Biomolecular Imaging (grant 5229-00012B).

S11.L9

PET MICROPLASTICS ALTER THE GLOBAL PROTEOMIC PROFILE OF THE PORCINE KIDNEY

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It is estimated that over 10 billion tons of plastic waste have been produced, and this amount is expected to triple by 2060. In the environment, these wastes undergo fragmentation and degradation by many factors such as UV rays and mechanical stress. In the consequence, a new form of environmental pollution called microplastics (MPs, <5 mm) has emerged. To date, many studies have reported harmful effects of MPs on animals and humans. Moreover, recent reports suggest that the kidney may be sensitive to MPs due to its excretory function. Compared to other polymers, PET-MPs are rarely studied materials, although they are often identified in bottled water. In this study, we examined the influence of PET-MPs on the kidney proteomic profile of immature piglets. Approximately 8-week-old pigs were divided into a control (n=5) and two experimental groups (n=5 each). The experimental groups received a low dose (0.1 g/animal/day) or a high dose (1 g/animal/day) of PET-MPs for 4 weeks. At the end of the experiment, the animals were sacrificed. Total protein was extracted in RIPA buffer according to the manufacturer's instructions. Protein samples were analyzed by LC-MS/MS and proceeded using bioinformatic tools and statistical analysis. We identified 17 up-regulated and 3 down-regulated proteins. The proteins were related to metabolism (Ras GTPase-activating protein-binding protein 1, Carboxylic ester hydrolase, Glycine N-acyltransferase-like protein), transmembrane signaling systems (Guanine nucleotide-binding protein subunit gamma) and transmembrane transport (Protein kinase C and casein kinase substrate in neurons 3, Anion exchange protein, Calcium-transporting ATPase, V-type proton ATPase subunit). The data indicate that PET-MPs might interrupt membrane transport, particularly ion transport. Our results suggest that exposure to PET-MPs may alter kidney functions involved in maintaining water-electrolyte balance and osmotic tension.

S11.L10

EFFECTS OF HIGH DIETARY HYBRID AND POPULATION RYE INCLUSION ON INFLAMMATORY-RELATED CYTOKINE CONCENTRATIONS IN FATTENING PIGS

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Cereal grains are the main component of complete feed mixtures for pigs. Recently, thanks to modern varieties, rye has become a high-quality and economical feed component that can replace wheat or barley in the daily diet. Nutritional recommendations specify that rye should not constitute more than 20% of a pig's daily feed. This is due to a significantly higher concentration of antinutritional substances than in other cereals. The action of all these compounds is associated not only with limited nutrient absorption but also with possible intestinal inflammation. The aim of the study was to assess the effect of high concentrations of different rye varieties in feed mixtures on the concentrations of proinflammatory cytokines in pig blood plasma , namely IL-1β, IL-2, IL-6 and TNF. One hundred Polish Landrace pigs, equally male and female, were randomly assigned to four groups (n=25/group): HYB, POP, and their respective control groups. The animals were fed a grower diet for the first 40 days of fattening and a finisher diet during the last 32 days. Experimental diets contained 60% hybrid rye (HYB) or 60% population rye (POP) supplemented with multienzyme Enzyme G2G. Control diets contained 76% cereals (38% wheat and 38% barley). Blood samples were collected at ~100 kg body weight. The concentration of pro-inflammatory cytokines in blood plasma was determined using pig-specific ELISA kits. The study results showed that supplementing the diet with 60% rye did not significantly increase proinflammatory cytokine levels in either the POP or HYB groups, meaning it did not trigger an inflammatory response in the pigs. From a practical perspective, the results suggest the safe inclusion of higher doses of both types of rye in the pig diet.

S11.L11

PANCREATIC AND INTESTINAL RESPONSES TO RYE-BASED DIETS IN PIGS

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Rye is considered challenging in monogastric nutrition because its non-starch polysaccharides (NSPs), mainly arabinoxylans, may increase digesta viscosity and reduce nutrient availability. Modern rye cultivars and NSP-degrading enzymes may partly overcome these limitations. This study evaluated diets containing 60% population rye or 60% hybrid rye, with or without NSP-degrading enzymes, in relation to pancreatic enzyme activity, pancreatic morphometry and pro-inflammatory cytokines in intestinal homogenates of pigs. Population rye without enzyme supplementation increased trypsin activity, suggesting an adaptive response of the exocrine pancreas to greater digestive load. Enzyme supplementation reduced this effect, indicating lower pancreatic activation. Amylase activity remained unchanged in this dietary variant. In pigs fed 60% hybrid rye, amylase activity increased, whereas NSP enzymes reduced this response, suggesting lower pancreatic demand for carbohydrate hydrolysis. Trypsin activity was not markedly affected. Pancreatic morphometry did not differ significantly among groups, indicating functional rather than structural adaptation. The strongest local pro-inflammatory response, reflected by increased IL-6, TNF-α and IL-1β concentrations, was detected in the middle jejunum after population rye without NSP enzymes. Enzyme supplementation attenuated this response, suggesting beneficial modulation of the intestinal environment. These findings indicate that rye type and NSP enzymes modulate pancreatic function and intestinal immune responses in pigs.

S11.L12

ASSESSMENT OF THE CYTOTOXIC RESPONSE OF HUMAN BJ FIBROBLASTS EXPOSED TO POLYSULFONE MEMBRANE EXTRACTS

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Evaluation of cytotoxicity is an essential step in the biological assessment of biomaterials, particularly when carbon nanomodifiers are introduced to improve material properties. This study assessed the cytotoxic response of human BJ fibroblasts exposed to extracts of unmodified polysulfone membranes (PSU) and polysulfone membranes modified with carbon nanotubes (PSU-CNT), graphene oxide (PSU-GO), or graphite (PSU-GR). Extracts were prepared according to ISO 10993-12, and cytotoxicity was evaluated in accordance with ISO 10993-5. Cell viability was determined using the MTT assay, while Calcein AM staining was used for qualitative assessment of viable cells and morphology. All tested extracts maintained cell viability above the 70% non-cytotoxicity threshold, with mean values close to the control and no statistically significant differences between groups. Microscopic observations confirmed numerous viable cells with preserved morphology in all variants. These results indicate that, under the applied experimental conditions, unmodified and carbon nanomodified polysulfone membranes did not induce an evident cytotoxic response in human BJ fibroblasts. The findings support the use of preliminary cytotoxicity screening as a necessary step in the evaluation of modified biomaterials intended for further *in vitro* and *in vivo* biological studies.

S11.P1

THE EFFECT OF DIET ON THE PROFILE OF GUT MICROBIOTA IN A MOUSE MODEL

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The European Charter on the Fight against Chronic Non-communicable Diseases (2022) highlights the importance of healthy, unprocessed diets and modern nutritional strategies in preventing obesity, diabetes, and nutritional deficiencies. Gut microbiota plays a key role in metabolic regulation, and its disturbances are linked to obesity and type 2 diabetes. This study evaluates the effects of a high-fat diet and diabetes on metabolic disorders, oxidative stress, and gut microbiota to improve understanding of metabolic disease mechanisms. The study was conducted on 100 male Swiss mice, aged four weeks, which were kept under controlled environmental conditions. The animals were divided into three groups: LFD (low-fat diet), HFD (high-fat diet), and a diabetes model (LFD + streptozotocin/alloxan). Behavioural and physiological parameters (body weight, activity and food intake), metabolic tests (GTT and ITT) and selected blood biochemical parameters (triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), alanine aminotransferase (ALT) and aspartate aminotransferase (AST)) were assessed. Additionally, insulin and ghrelin levels (ELISA), oxidative stress markers and gut microbiota composition (PCR and 16S rDNA sequencing) were analysed. The detailed results of the study will be presented at the congress. These findings could improve our understanding of how a high-fat diet and diabetes affect metabolic disorders, oxidative stress and the gut microbiota. This could help us to understand the mechanisms behind the development of metabolic diseases.

S11.P2

DIETARY CASEIN ATTENUATES CADMIUM-INDUCED INTESTINAL INJURY IN RATS

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Cadmium (Cd) is a toxic environmental contaminant that may impair gastrointestinal integrity, as intestinal enterocytes are the first site of oral Cd uptake. Dietary proteins and their bioactive peptides, including casein-derived peptides, may modulate Cd toxicity through antioxidant, detoxifying, and metal-chelating properties. This study evaluated whether dietary casein supplementation attenuates cadmium-induced changes in intestinal morphology in young rats. Three-week-old Wistar rats were allocated by sex into four groups: Cd-exposed control, negative control, casein plus Cd, and casein-supplemented group. Cadmium was administered in drinking water at 19.4 mmol/L for 10 weeks, while experimental diets contained 11.25% bovine milk casein. Histomorphometric analysis was performed in the duodenum, mid-jejunum, ileum, and large intestine. Cadmium exposure was associated with impaired intestinal architecture, including reduced mucosal thickness, decreased villus height, thinning of the muscular layer, and increased crypt depth. Dietary casein partially improved mucosal morphology, increased villus height, and improved selected parameters of the intestinal wall. Casein supplementation also increased goblet cell size in the large intestine, suggesting improved mucus barrier support. These findings indicate that dietary casein may attenuate selected adverse effects of Cd exposure on intestinal morphology and mucosal protection. The results support its potential relevance as a nutritional modifier of gastrointestinal cadmium toxicity.

S11.P3

DIET-RELATED INTESTINAL AND ENTERIC NERVOUS SYSTEM ADAPTATIONS IN MALLARD, TUFTED DUCK AND GREEN-WINGED TEAL

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Introduction and aim. Ducks occupy diverse trophic niches, which may be reflected in the morphology and functional organization of their gastrointestinal tract. The Mallard, Tufted Duck and Green-Winged Teal represent distinct feeding strategies that impose different mechanical, absorptive and neuroregulatory demands on the intestine. The aim of this study was to compare the morphology of the small intestine and cecum, as well as the organization of HuC/D-immunoreactive enteric ganglia, in Mallard, Tufted Duck and Green-Winged Teal, and to determine whether these features reflect dietary specialization and ecological adaptation. Material and methods. The study included 18 adult wild ducks, six individuals from each species: Mallard (*Anas platyrhynchos*), Tufted Duck (*Aythya fuligula*) and Green-Winged Teal (*Anas crecca*). Samples of the duodenum, jejunum, ileum and cecum were collected post-mortem. Histomorphometric analysis included measurements of villus height and width, crypt depth and width, mucosal and submucosal thickness, muscular layer thickness, and the number of villi and crypts per 1 mm of mucosal surface. Immunohistochemistry with the pan-neuronal marker HuC/D was used to assess enteric ganglia. Results. Clear interspecies differences were observed in intestinal morphology and enteric nervous system organization. The Tufted Duck showed more developed intestinal muscle layers, particularly in the duodenum, ileum and cecum, as well as larger HuC/D-immunoreactive ganglia in selected intestinal segments, suggesting adaptation to mechanically demanding food. The Mallard displayed longer villi, deeper crypts and a more developed mucosal structure, indicating enhanced absorptive capacity associated with an omnivorous diet rich in plant material. In contrast, the Green-Winged Teal exhibited thinner intestinal walls and shorter or narrower villi, consistent with a more easily digestible diet based mainly on small aquatic invertebrates. Conclusion. The results indicate that intestinal morphology and enteric nervous system organization differ among wild duck species and may reflect dietary specialization and ecological adaptation. The applied histomorphometric and immunohistochemical methods proved useful in detecting subtle interspecies differences in the avian digestive system.

S11.P4

ASSESSMENT OF ANGIOTENSIN II RECEPTORS AT1R, AT2R AND ENDOTHELIAL NITRIC OXIDE SYNTHASE IMMUNOREACTIVITY IN THE ABDOMINAL AORTA OF PIGLETS BORN β-HYDROXY-β-METHYLBUTYRATE (HMB) – SUPPLEMENTED SOWS

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β-hydroxy-β-methylbutyrate (HMB) is a biologically active leucine metabolite known to influence cellular metabolism, protein synthesis and tissue development. The aim of the study was to evaluate the immunoreactivity of angiotensin II receptors type 1 and 2 (AT1R, AT2R) and endothelial nitric oxide synthase (eNOS) in the abdominal aorta of piglets born to sows supplemented with HMB during pregnancy. The study included piglets derived from Pulawska sows fed either a standard diet (control group, K) or a diet supplemented with HMB (0.2 g/kg body weight/day) between days 70 and 90 of gestation (HMB group). At 35 days of age, abdominal aorta samples were collected and processed using standard histological and immunohistochemical procedures. Immunoreactivity of AT1R, A2TR and eNOS was assessed semi-quantitatively in the tunica adventitia, tunica media and tunica intima. Piglets from the HMB group showed higher AT1R immunoreactivity in all layers of the vascular wall, particularly in the tunica adventitia and tunica intima, compared with the control group. Increased eNOS immunoreactivity was also observed in the HMB group, mainly in the tunica media and tunica intima. In contrast, AT2R immunoreactivity was lower in the tunica media of HMB piglets, while similar immunostaining intensity was observed in the tunica adventitia and tunica intima in both groups. The obtained results suggest that maternal HMB supplementation may affect the immunoreactivity of components associated with the renin-angiotensin system and endothelial function in the vascular wall of offspring.

S11.P5

COMPARATIVE PHARMACOKINETICS OF CANNABIDIOL AND ITS METABOLITES IN HORSES FOLLOWING ORAL ADMINISTRATION OF OIL AND AQUEOUS-BASED FORMULATIONS

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Cannabidiol (CBD) is a non-psychoactive phytocannabinoid that is increasingly being investigated in veterinary medicine for its potential anti-inflammatory, analgesic, and anxiolytic effects. The aim of this study was to compare the pharmacokinetic profile of CBD administered orally in oil and water-based formulations in horses. The horses (n=10) received CBD at a dose of 1 mg/kg body weight per os. The blood was drawn at T = 0.5, 1, 2, 3, 4, 5, 6, 12, and 24 hours. Plasma CBD concentrations were determined using ultra-high performance liquid chromatography-tandem mass spectrometry (UHPLC-MS/MS), and pharmacokinetic parameters were evaluated using non-compartmental analysis. Out of several analyzed metabolites, only one was found to be present in the plasma (7-COOH-CBD). Administration of CBD in the oil formulation resulted in higher systemic exposure compared to the water-based preparation ($AUC_{0\text{-}last}$ [min·µg·L^{-1}] = 1440.1 and 916.5, respectively). Oil- and water-based formulations reached peak plasma concentrations (4.42 and 2.22 µg·L^{-1}, respectively) at the same T_{max} (240 min). Consequently, the 7-COOH-CBD metabolite showed higher exposure and an earlier peak in the oil group ($AUC_{0\text{-}last}$ = 52427.5 min·µg·L^{-1}, C_{max} = 137.2 µg·L^{-1}, T_{max} = 240 min) than in the water-based one ($AUC_{0\text{-}last}$ = 30520.6 min·µg·L^{-1}, C_{max} = 78.3 µg·L^{-1}, Tmax = 360 min). The assessment of the metabolic ratio (MR) confirmed an intensive hepatic first-pass metabolism of CBD. Notably, the aqueous formulation exhibited a lower metabolic ratio than the oil carrier (31.4 vs. 37.1), indicating a lower total conversion of the parent drug, while the $T_{1/2}$ remained comparable between the groups (307.1 min and 272.9 min, respectively). These findings indicate that oil-based formulations increase systemic exposure to CBD in horses, whereas the novel water-based preparation alters absorption dynamics and successfully attenuates the immediacy of the hepatic first-pass effect.

S11.P6

THE EFFECT OF ADROPIN ON THE LOCALIZATION OF AT1 AND AT2 ANGIOTENSIN RECEPTORS AND ADROPIN IN THE ABDOMINAL AORTIC WALL OF FEMALE RATS UNDER CONDITIONS OF OVARIECTOMY

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Adropin (ADR) is an adipokine that plays a key role in maintaining metabolic homeostasis, particularly regarding body fat and lipid metabolism, by preventing obesity-related hyperinsulinemia and fatty liver disease through the regulation of lipid and glucose metabolism. The aim of the study was to assess the localization and immunoreactivity of ADR and angiotensin II receptors (AT1R and AT2R) in the abdominal aorta of female Wistar rats in the conditions of surgical elimination of the hormonal function of the gonads. The research was carried out on 40 female rats, which were intended for sham surgery (SHO, n = 20) or bilateral ovariectomy (OVX, n = 20). After 12 weeks, the animals were divided into 4 groups receiving i.p. physiological saline (SHO-PhS, n = 10; OVX-PhS, n = 10) or 2 µg/kg B.W. of ADR (SHO-ADR, n = 10; OVX-ADR, n = 10) every day for 8 wks. After euthanasia of the animals, fragments of the abdominal aorta were collected, fixed in formalin and subjected to histological and immunohistochemical procedures using antibodies for ADR, AT1R, and AT2R. A semi-quantitative method was used to assess immunoreactivity in the adventitia, media, and intima of the abdominal aorta. IHC studies revealed the presence of ADR, AT1R, and AT2R in all layers of the aortic wall. The highest ADR immunoreactivity was observed in the tunica adventitia and intima of the OVX-ADR group compared to the other groups. The highest AT1R immunoreactivity was observed in the adventitia in the SHO-ADR group and in the media in the OVX-ADR group. Higher reactivity was also observed in the intima in the SHO and OVX groups. The lowest AT1R immunoreactivity was confirmed in the intima of SHO-ADR and OVX-ADR groups. AT2R immunoreactivity was highest in the adventitia in the OVX group and slightly lower in the OVX-ADR group, as well as in the media and intima of the OVX group compared to the other groups. The results suggest that exogenous adropin may affect the immunoreactivity of AT1R, AT2R, and ADR in aortic wall in conditions of impaired hormonal homeostasis caused by ovariectomy.

S11.P7

AGE-RELATED DIFFERENCES IN THE DISTRIBUTION OF SELECTED POPULATIONS OF IMMUNOREACTIVE ENTEROENDOCRINE CELLS IN THE BOVINE ABOMASUM AND SMALL INTESTINE

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The gastrointestinal tract of ruminants is regulated by a complex endocrine network that coordinates digestive processes, nutrient absorption and metabolic homeostasis. Enteroendocrine cells produce numerous hormones involved in the regulation of secretion, motility and nutrient utilization. However, comparative data on the age-related distribution of enteroendocrine cell populations in different segments of the bovine gastrointestinal tract remain limited. The aim of this study was to compare selected enteroendocrine cell populations in the abomasum and small intestine of young and adult Holstein-Friesian bulls, and to determine whether their distribution reflects age-related functional maturation of the digestive system. The study included 12 male Holstein-Friesian cattle divided into two age groups: six young bulls aged 7–8 months and six adult bulls aged 20–24 months. Tissue samples were collected post-mortem from the abomasum, duodenum, jejunum and ileum. Immunohistochemical analysis was performed using antibodies against chromogranin A (CgA), gastrin, secretin, somatostatin (SOM), glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1). CgA was used as a general marker of enteroendocrine cells. Immunoreactive cells were quantified using standardized microscopic methods and the results were compared between age groups and gastrointestinal segments. Clear age-related and segment-specific differences were observed in the distribution of enteroendocrine cells. In the abomasum, CgA-immunoreactive cells were more numerous in young animals, whereas gastrin- and SOM-immunoreactive cells were more abundant in adult bulls. In the small intestine, CgA- and GIP-immunoreactive cells were generally more numerous in adult animals, with the highest values recorded in the duodenum. In contrast, secretin- and SOM-immunoreactive cells were more abundant in young bulls. GLP-1 showed a distinct age- and segment-dependent pattern: in young animals, the highest number of GLP-1-immunoreactive cells was observed in the ileum, whereas in adult animals the highest values were found in the jejunum. These findings indicate dynamic changes in the organization of the enteroendocrine system during postnatal development. The results demonstrate that the number and distribution of selected enteroendocrine cell populations differ between young and adult Holstein-Friesian bulls and vary among gastrointestinal segments. These changes may reflect functional maturation of the digestive tract and adaptation of endocrine regulation to age-related metabolic and digestive demands.

S11.P8

MICROPLASTIC EXPOSURE ALTERS ADIPOSE TISSUE FUNCTION: INSIGHTS FROM A PORCINE *IN VIVO* STUDY

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Micro- and nanoplastics (MNPs), especially polyethylene terephthalate (PET), are emerging environmental pollutants that have been detected in animal and human tissues. Exposure to MNPs has been shown to cause oxidative stress, inflammation, and cellular metabolic disruption. Therefore, MNPs are associated with inflammatory and metabolic diseases. However, little is known about their effects on adipose tissue, an important metabolic and endocrine organ, or about the functional differences between distinct adipose depots. Adipose tissue exhibits functional variability, with subcutaneous fat being more protective and visceral fat being more metabolically active and strongly linked to metabolic diseases. To assess the contribution of PET MNPs to metabolic disorders, it is important to understand how they affect these different depots. In this study, we used samples from 7–8-month-old crossbred Large White × Polish Landrace pigs divided into three groups (N = 3): control (C), receiving empty gelatine capsules; low dose (LD), receiving 0.1 g PET MPs/animal/day; and high dose (HD), receiving 1 g PET MPs/animal/day. The treatment was conducted for 4 weeks. Visceral and subcutaneous adipose tissues were collected, and total RNA was extracted for gene expression analysis using qRT-PCR. To evaluate functional differences between adipose depots in the experimental groups, markers associated with inflammation, oxidative stress, metabolism, and cellular stress were examined. The findings suggest that visceral adipose tissue exhibits a stronger metabolic response, reflected by the downregulation of PPARγ in the LD group and FABP4 in the HD group. Although the response was less pronounced, inflammatory markers such as TNF-α and IL-1β mRNAs were also elevated in subcutaneous adipose tissue in the LD group. Additionally, alterations in metabolic parameters, such as decreased FABP4 mRNA expression in the HD group, indicate disruption of lipid metabolism. PET MPs altered the expression of selected genes in both adipose depots; however, the response in visceral adipose tissue appeared more closely associated with inflammatory and metabolic alterations.

S11.P9

A NONINVASIVE JACKETED SYSTEM FOR MONITORING CARDIORESPIRATORY PARAMETERS AND LOCOMOTOR ACTIVITY IN ANIMALS IN NON-CLINICAL RESEARCH: A NOVEL NON-INVASIVE TECHNOLOGY SUPPORTING REDUCTION AND REFINEMENT IN *IN VIVO* RESEARCH

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External wearable jacketed telemetry has been used since 2008 in large animals (dogs, monkeys, pigs) in preclinical research. This technology has been miniaturized to enable non-invasive cardiorespiratory measurements in small mammals – rats (220–700 g) and marmosets (300–500 g) – for which no external monitoring system was previously available. The system combines a 3D accelerometer (activity), RIP bands (respiratory rate, tidal and minute volume) and ECG electrodes (heart rate, RR, PR, QRS and QT(c) intervals), with real-time Bluetooth data transmission. The aim of this poster is to demonstrate the ethical benefits of this solution based on the results of four use cases: safety pharmacology in rats (combined assessment of the central nervous system and respiratory system in the FOB test), circadian rhythm phenotyping under baclofen administration, treadmill exercise, and chronobiology in marmosets monitored over a 24-hour period while freely moving. In all cases, reliable physiological data were obtained without anesthesia, surgery or restraint of the animals. The solution supports the 3Rs principles: Reduction – through the animal's ability to serve as its own control and the combination of different physiological endpoints within a single study – and Refinement, by eliminating the need for surgery, restraint and isolation of animals, while remaining compliant with regulatory guidelines (ICH S7A).

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