1st National Scientific Conference of Medical Analytics Students of the University of Rzeszów „Carpatia Diagnostica”

ABSTRACT BOOK

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Welcome message

Ladies and Gentlemen,

The 1st National Scientific Conference of Medical Analytics Students of the University of Rzeszów "Carpatia Diagnostica" took place on 29 May 2026, organized by medical analytics students affiliated with the College of Medical Sciences, University of Rzeszów.

"Carpatia Diagnostica" was a national scientific and educational event organized by students of the University of Rzeszów on an unprecedented and prestigious scale. Established to celebrate Laboratory Diagnostician Days, the conference integrated the community of laboratory diagnosticians, students, doctoral candidates, and young researchers from all over Poland, all united by their passion for medicine, modern laboratory diagnostics, and biomedical sciences.

The conference program featured a rich scientific and training agenda, including student oral and poster sessions divided into thematic panels. Participants also had the opportunity to attend expert lectures delivered by distinguished professors, specialists, and recognized authorities in the field of laboratory medicine. Furthermore, the event successfully combined the scientific conference with specialized training certified by the National Chamber of Laboratory Diagnosticians (KIDL).

This Abstract Book contains all scientific abstracts presented during Carpatia Diagnostica 2026.
The authors are solely responsible for the content and grammatical correctness of their abstracts.

Sabina Galiniak

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Title of presented paper: Assessment of lipoprotein (a) concentration as an independent risk marker in patients with chest pain

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Type of the paper: Original paper

Introduction and aim. Lipoprotein (a) [Lp(a)] is an established, genetically determined cardiovascular risk factor. In acute states, it may be crucial to determine whether its concentration correlates with the extent of myocardial injury and whether it exhibits variability during the acute phase of an incident. To assess the correlation between Lp(a) concentration and cardiac troponin I (*c*TnI) levels, and to analyze the stability of Lp(a) during the initial phase of hospitalization in patients presenting with chest pain.

Material and methods. The study included 116 patients (mean age 72±16 years, 53% female) with chest pain admitted to the hospital emergency department. Lp(a) concentration was measured using an immunoturbidimetric method on a Cobas c503 analyzer (Roche, Basel). In a subgroup of 10 patients, *c*TnI and Lp(a) levels were determined at the time of admission (T0) and 2 hours (T2) after the start of hospitalization to compare the dynamics of both parameters. Statistical analysis was performed using Statistica software.

Results. The group was divided into tertiles based on *c*TnI levels. Median *c*TnI was 87 ng/L (Q1: 54; Q3: 331), and Lp(a) was 16 mg/dL (Q1: 5; Q3: 69). No correlation was found between Lp(a) and *c*TnI (p>0.05). Significantly higher Lp(a) levels were observed in patients >70 years old (p=0.002), with a median of 26.00 mg/dL (IQR: 6.99–138.00) compared to 9.13 mg/dL (IQR: 1.73–31.00) in younger subjects. The highest percentage of Lp(a) results >30 mg/dL was recorded in the first *c*TnI tertile (51.3%). Dynamic analysis (n=10) showed an increase in *c*TnI (mean 102.4 ng/L) while Lp(a) remained stable (change of 5 mg/dL).

Conclusion. The lack of correlation between Lp(a) and *c*TnI confirms the distinct biological roles of both markers. In patients with elevated Lp(a) concentrations, coronary incidents may manifest and be detected at relatively lower concentrations of released troponins. It was demonstrated that even in the face of an acute cardiac incident, Lp(a) remains a stable parameter, independent of troponin I dynamics. This indicates the reliability of its measurement as early as the first hour of hospitalization, allowing for a precise assessment of the patient's risk profile in acute states. Higher Lp(a) concentrations observed in geriatric patients may result from correlations with inflammatory states and the degree of renal impairment.

Keywords. atherosclerosis, cardiovascular risk, chest pain, lipoprotein (a), troponin I

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Title of presented paper: Subclinical liver function impairment in patients with hbsag(-)/anti-hbc(+) serological profile

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Type of the paper: Original paper

Introduction. The AST to platelet ratio index (APRI) and fibrosis-4 index (FIB-4) markers have gained widespread use in assessing the severity of liver fibrosis due to their non-invasive nature and relatively low cost. Their role is crucial in the early diagnosis of chronic liver diseases, which can remain asymptomatic even in advanced stages. These parameters are based on routine laboratory tests: APRI utilizes AST activity and platelet count (PLT), while FIB-4 additionally incorporates the patient's age and ALT activity. Despite numerous advantages, the interpretation of results should account for the limitations of these methods (e.g., the dependence of APRI on local AST reference ranges) and, in cases of ambiguity, require verification through invasive procedures. Aim To evaluate the degree of liver fibrosis in patients with an HBsAg(-)/anti-HBc(+) serological profile using non-invasive biochemical markers.

Material and methods. The study group consisted of 167 patients (age range 25–96 years, median: 66.5) with an HBsAg(-)/anti-HBc(+) profile, hospitalized at the Central Clinical Hospital (CSK UCK WUM). Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were measured spectrophotometrically (Cobas c503, Roche). HBsAg and anti-HCV were determined qualitatively via electrochemiluminescence immunoassay (ECLIA, Cobas e801, Roche). Platelet count (PLT) was determined using impedance or fluorescence methods (Sysmex XN series).

Results. In the majority of patients, the biochemical and morphological components of the indices were within reference ranges (median: ALT 26 U/L, AST 29 U/L, PLT 212,000/μL). Spearman's rank correlation analysis revealed a statistically significant, strong positive correlation between APRI and FIB-4 ($r_s=0.8464$; $p<0.0001$). Using established clinical cut-off points for APRI, significant fibrosis was ruled out (result ≤0.5) in 68% of the study group. Results suggesting advanced fibrosis or cirrhosis were observed in 21.5% of patients.

Conclusion. Patients with an HBsAg(-)/anti-HBc(+) serological profile represent a heterogeneous group regarding the degree of liver injury. The application of APRI and FIB-4 indices enables the detection of subclinical fibrosis in this population, which is of key importance in the prevention of complications such as cirrhosis and hepatocellular carcinoma (HCC).

Keywords. APRI, FIB-4, anti-HCV, anti-HBc, HBsAg

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Title of presented paper: Do professional volleyball players experience stress? – Quantification of 8-isoprostane as a marker of oxidative stress using GC-MS

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Type of the paper: Original paper

Introduction and aim. 8-isoprostane is a product of non-enzymatic peroxidation of arachidonic acid and is considered a reliable biomarker of oxidative stress. This study aimed to assess changes in oxidative stress in response to prolonged intensive training in professional athletes.
Material and methods. Ten female professional volleyball players were included in the study. Blood samples were collected before and after a 10-week intensive training period. Plasma was separated, purified, and 8-isoprostane was isolated using immunoaffinity column chromatography. Quantification was performed using gas chromatography-mass spectrometry (GC-MS).
Results. A Wilcoxon signed-rank test was performed ($p=0.028$). The median concentration of 8-isoprostane at baseline was 0.06 ng/mL and it increased significantly to 0.196 ng/mL after the training period. The effect size was large ($r=0.69$).
Conclusion. An increase in plasma 8-isoprostane concentration suggests a relationship between intensive professional training and elevated oxidative stress. These findings indicate the need for strategies to reduce oxidative stress in athletes.
Keywords. immunoaffinity column chromatography, GC-MS, 8-isoprostane, oxidative stress

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Title of presented paper: Preparation and validation of an in vitro HFLS-N and HFLS-RA fibroblast-like synoviocyte model for inflammatory and functional studies using all-trans retinoic acid (ATRA)

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Type of paper: Original paper

Introduction and aim. Rheumatoid arthritis (RA) is a chronic autoimmune disease in which fibroblast-like synoviocytes (FLS) contribute to persistent inflammation and destruction of cartilage and bone. These cells demonstrate increased inflammatory, migratory, and invasive activity, making them an important in vitro model for evaluating compounds that may modulate inflammatory processes. The aim of this study was to establish and preliminarily validate an in vitro model using HFLS N and HFLS-RA cells and to evaluate the effects of different ATRA concentrations on cell viability and metabolic activity using the MTT assay. An additional objective was to select an appropriate ATRA concentration for further analyses.

Material and methods. HFLS-N and HFLS-RA cells were cultured under standard in vitro conditions and seeded into 96-well plates. After 24 hours, HFLS-N cells were stimulated with LPS (1 μg/ml) to induce an inflammatory response. Subsequently, ATRA was added to all experimental groups: HFLS-N, HFLS-N+LPS, and HFLS-RA at concentrations of 0.5, 1, 5, 10, and 50 μM. After 24 hours, cell viability and metabolic activity were assessed using the MTT assay.

Results. The MTT assay enabled evaluation of the effects of different ATRA concentrations on the metabolic activity of HFLS-N, HFLS-N+LPS, and HFLS-RA cells. LPS stimulation allowed the development of an induced inflammatory model that could be compared with both normal and rheumatoid synoviocytes. The results demonstrated a relationship between ATRA concentration and cell viability in all experimental groups. The 10 μM concentration was selected for further analyses because it did not significantly reduce cell viability and enabled comparison of cellular responses between groups.

Conclusion. The developed in vitro model using HFLS-N, HFLS-N+LPS, and HFLS-RA cells may serve as a useful tool for investigating the effects of ATRA on fibroblast-like synoviocytes under normal, inflammatory, and rheumatoid conditions. The MTT assay enabled preliminary assessment of ATRA effects on cell viability and allowed selection of 10 μM as the optimal concentration for further inflammatory and functional analyses.

Keywords. all-trans retinoic acid, ATRA, fibroblast-like synoviocytes, HFLS-N, HFLS-RA

Funding. This work was funded under the program of the Minister of Science and Higher Education entitled 'Student Scientific Clubs Create Innovations,' project no. SKN/SP/657673/2026. The amount of project funding was PLN 68,400.00, and the total project value was PLN 69,400.00.

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Title of presented paper: Drug-cell-gene: preliminary studies on the effects of methotrexate in rheumatoid arthritis models using the MTT assay

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Type of work: Original paper

Introduction and aim. Rheumatoid arthritis (RA) is a chronic autoimmune disease in which fibroblast-like synoviocytes (FLS) play a key pathogenic role. Under inflammatory conditions, FLS exhibit aggressive properties leading to synovial hyperplasia and cartilage destruction. Methotrexate (MTX), a dihydrofolate reductase (DHFR) inhibitor, remains the gold standard in RA treatment due to its antiproliferative and anti-inflammatory effects. The aim of this study was to evaluate the cytotoxicity profile of MTX in an in vitro FLS model and to determine the optimal concentration for further functional studies.
Material and methods. The experiment was performed using HFLS-N and HFLS-RA cell lines. Inflammation was induced in selected HFLS-N cultures using LPS (1 μg/mL). Cells were incubated for 24 hours with MTX at concentrations ranging from 0.01 to 50 μM. Cell viability was assessed using the MTT assay.
Results. MTX caused a decrease in cell viability in HFLS-N and HFLS-N+LPS lines to approx. 68-76%. The survival rate of the pathological HFLS-RA line remained at 93–116% regardless of the MTX concentration used (0.01–50 μM). This indicates an early resistance phenomenon or compensatory metabolic hyperactivity of rheumatoid cells in response to acute cellular stress.
Conclusion. The results are preliminary, did not confirm the inhibitory effect of MTX on HFLS-RA cells in the MTT assay. Due to the lack of a clear dose-response relationship and experimental limitations, it is necessary to repeat the experiment with an extended exposure time.
Keywords. fibroblast-like synoviocytes, MTX, HFLS-N, HFLS-RA, MTT assay, RA

Funding. This work was funded under the program of the Minister of Science and Higher Education entitled 'Student Scientific Clubs Create Innovations,' project no. SKN/SP/657673/2026. The amount of project funding was PLN 68,400.00, and the total project value was PLN 69,400.00.

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Title of presented paper: EVALI - the epidemic of the 21st century

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Type of the paper: Review

Introduction and aim. EVALI (e-cigarette or vaping product use-associated lung injury) has become a significant public health concern of the 21st century reflecting the rapid global rise in the use of e-cigarettes and vaping products. This review aims to synthesise current knowledge of the epidemiology, pathomechanisms, clinical features, and risk factors of EVALI with a particular focus on the role of vitamin E acetate and patterns of product use.
Literature search. A systematic review of the literature was conducted using the PubMed database, with "EVALI" employed as the search term. The analysis included peer-reviewed articles in English that focused on epidemiology, clinical presentation, pathophysiology, and associated risk factors of EVALI.
Analysis of the literature. Particular focus was placed on patient demographics, substance use patterns (particularly those involving THC-containing products), types of devices used, frequency of exposure, and the sources of these vaping products. The analysis also examined the proposed mechanisms of lung injury, with particular attention paid to the role of vitamin E acetate in disrupting pulmonary surfactant function, leading to the accumulation of lipid-laden macrophages, and inducing inflammatory lung injury. Analysis of the available data indicates that EVALI mainly occurs in young men who regularly use vaping products containing THC, frequently alongside nicotine. A considerable number of patients report using these products on a daily or almost daily basis. Vitamin E acetate, frequently used as a thickening agent in illicit THC-containing liquids, has been identified as a major contributor to lung injury. Upon inhalation, it impairs surfactant function, facilitates the accumulation of lipid-laden macrophages, and may produce toxic compounds such as ketene when heated, resulting in chemical pneumonitis and acute respiratory distress. Clinical presentations vary widely, ranging from coughing, chest pain, and dyspnea to severe respiratory failure, necessitating hospitalisation.
Conclusion. EVALI represents a significant and preventable public health issue, clearly indicating that e-cigarettes should not be regarded as a safe alternative to conventional tobacco smoking. The development of standardised clinical guidelines is crucial for the enhancement of early detection, ensuring accurate diagnosis, and supporting effective management of EVALI.
Keywords. EVALI, e-cigarette, inhalation toxicity, lung injury, vaping

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Title of presented paper: Genetic mutations and cancer risk – where is the line?

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Type of the paper: Review

Introduction and aim. Cancer is one of the leading causes of death worldwide, and its development is closely associated with the accumulation of genetic alterations driving neoplastic transformation. In cancer patients, two distinct genomes can be identified: the constitutional genome, inherited from parents and present in most cells, and the tumor genome, characterized by high variability resulting from genomic instability. The aim of this study is to present the relationship between genetic mutations and cancer development, with particular emphasis on mutations predisposing to breast cancer and their significance in diagnosis, prevention, and therapy.

Literature search. This study is a narrative review based on an analysis of the available scientific literature concerning genetic mutations associated with cancer risk and modern molecular diagnostic methods. Particular attention was paid to high-risk genes, including BRCA1 and BRCA2, and the application of DNA sequencing techniques in identifying hereditary cancer predisposition.

Analysis of the literature. Genetic mutations may lead to the activation of oncogenes or inactivation of tumor suppressor genes, resulting in disruption of cell cycle control, DNA repair mechanisms, and apoptosis. In breast cancer, mutations in BRCA1 and BRCA2 significantly increase the risk of disease and influence diagnostic, preventive, and therapeutic decisions. Increasingly, multigene panel testing and next-generation sequencing are being used instead of single-mutation analysis, allowing for a more comprehensive assessment of cancer risk.

Conclusion. Genetic mutations play a crucial role in cancer development and are a key element of modern molecular diagnostics. Their detection enables assessment of individual risk, implementation of appropriate preventive strategies, and selection of targeted therapies. Advances in molecular biology significantly enhance the potential of personalized medicine in oncology.

Keywords. apoptosis, breast cancer, cancer risk

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Title of presented paper: One step away from diabetes: insulin resistance in the light of modern diagnostics

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Type of the paper: Review

Introduction and aim. Insulin resistance is a condition characterised by reduced tissue sensitivity to insulin and is one of the main factors contributing to the development of type 2 diabetes. The aim of this paper was to discuss the mechanisms of insulin resistance, its risk factors, and modern diagnostic methods. The paper presents the most important risk factors associated with the development of insulin resistance, such as overweight and obesity, physical inactivity, poor diet, genetic factors, and hormonal disorders. The role of insulin in regulating carbohydrate metabolism and the impact of chronic hyperinsulinaemia on the development of metabolic disorders are also discussed.

Literature search. Publications on the pathophysiology, risk factors, and diagnostic methods of insulin resistance were reviewed, with particular emphasis on current evidence and laboratory markers.

Analysis of the literature. The development of insulin resistance is associated with genetic and environmental factors, such as obesity, physical inactivity, and an unhealthy diet. Early detection of these disorders enables the implementation of appropriate prevention and treatment strategies. The paper pays particular attention to laboratory markers used to assess tissue sensitivity to insulin.

Conclusion. Insulin resistance is a significant health problem associated with the development of metabolic diseases. Early diagnosis may reduce the risk of developing type 2 diabetes and its complications.

Keywords. diabetes diagnosis, insulin, insulin resistance, type 2 diabetes

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Title of presented paper: Diagnosis of mitochondrial diseases in the age of genomics: challenges and new opportunities

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Type of the paper: Review

Introduction and aim. Mitochondrial diseases constitute a diverse group of genetically determined disorders resulting from abnormalities in the respiratory chain. Mutations may affect both mitochondrial DNA and nuclear genes, leading to a broad spectrum of clinical manifestations and considerable diagnostic challenges. The aim of this study is to present current diagnostic methods for mitochondrial diseases and assess the role of genomic techniques in clinical practice.
Literature search. This review is based on an analysis of recent scientific publications from 2020 to 2025 available in PubMed, Springer Nature, and Nature Reviews Neurology. Clinical studies, observational studies, and review articles concerning the molecular diagnosis of mitochondrial diseases were included.
Analysis of the literature. This paper discusses the application of modern genomic methods in the diagnosis of mitochondrial diseases, with particular emphasis on whole-exome sequencing and whole genome sequencing. WES and WGS enable comprehensive assessment of both mitochondrial DNA and nuclear genes associated with mitochondrial function. Their use increases the likelihood of identifying pathogenic variants, including variants with low levels of heteroplasmy. The role of transcriptomic analysis, including RNA-seq, is also discussed, as it may support the interpretation of genetic variants and reveal expression abnormalities that are not detectable by standard DNA analysis.
Conclusion. Modern genomic technologies significantly improve the accuracy of mitochondrial disease diagnosis. However, their proper interpretation requires a multidisciplinary approach and the integration of clinical, biochemical, and molecular data.
Keywords. mitochondrial diseases, molecular diagnostics, WES, WGS, RNA-seq

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Title of presented paper: Tumor-infiltrating lymphocytes in cancer immunotherapy

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Type of the paper: Review

Introduction and aim. Tumor-infiltrating lymphocytes (TILs) are immune cells located within the tumor microenvironment that recognize tumor-associated antigens. They consist predominantly of CD8+ cytotoxic T lymphocytes capable of directly eliminating malignant cells. The antigen specificity of these cells has provided the foundation for the development of personalized adoptive cell therapies in oncology. This poster aims to present an overview of the complete process of TIL-based therapy, including contemporary laboratory techniques used for TIL isolation, expansion, and clinical application.
Literature search. This work is based on a narrative literature review conducted using the PubMed database.
Analysis of the literature. TIL-based immunotherapy is primarily applied in the treatment of solid tumors because of their high molecular and cellular heterogeneity. The therapeutic process consists of several stages, including surgical collection of tumor tissue, isolation of TILs, ex vivo expansion of lymphocytes, assessment of their reactivity against tumor antigens, and subsequent clinical monitoring of treatment response.
Conclusion. Tumor-infiltrating lymphocytes represent a promising strategy in the immunotherapy of solid malignancies. Ongoing advances in TIL isolation procedures, expansion protocols, and treatment conditions continue to improve the safety and efficacy of this therapeutic approach.
Keywords. tumor- infiltrating lymphocytes, solid tumors, immunotherapy

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Title of presented paper: Short-term exposure to a 50 hz extremely low-frequency electromagnetic field (ELF-EMF) leads to ros-mediated DNA damage in gynecological and urological cancer cells in vitro

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Type of the paper: Review

Introduction and aim. Extremely low-frequency electromagnetic fields (ELF-EMF) are generated by, among other things, power lines and electrical devices that people use daily. This paper analyzes the results of research presented in the publication, where the aim of the study was to examine the short-term in vitro effects of ELF-EMF exposure on the formation of reactive oxygen species, the expression levels of genes and proteins involved in the cellular response to DNA damage, and the occurrence of epigenetic modifications in the studied cell lines.
Literature search. The analysis covered the paper from the PubMed database entitled "Short-Term Exposure to a 50 Hz Extremely Low-Frequency Electromagnetic Field (ELF-EMF) Leads to OS-Mediated DNA Damage in Gynecological and Urological Cancer Cells In Vitro" by G. Betlej et. all reported on short-term exposure of HeLa, ES-2, and DU145 cell lines to ELF-EMF, in which ROS levels, gene expression, and protein expression were assessed. In the analyzed work, the authors conducted studies on in vitro cultures of three cell lines: HeLa, ES-2, and DU-145. The cell lines were tested in a sinusoidal, continuous electromagnetic field with a frequency of 50 Hz and a magnetic induction of 1.3 mT for 15 and 30 minutes. Reactive oxygen species were detected using a dihydroethidium probe. To examine protein levels and localization, the authors used immunofluorescence. They performed RT-PCR to determine the expression of the studied genes (including NFE2L2, XRCC5, XRCC6, and POLβ).
Analysis of the literature. The analyzed study demonstrated that exposure to ELF-EMF for 30 minutes induces increased ROS production and changes in the levels of DDR and epigenetic modification factors in the studied cells. The strongest effect of ELF-EMF treatment was documented in ES-2 cells, where the increased ROS levels were accompanied by a significant regulation of XRCC5 gene expression and increased levels of several other proteins. They also demonstrated that ELF-EMF induces a delayed cellular response, manifested by a rapid increase in reactive oxygen species levels 24 hours after the end of exposure.
Conclusion. Based on this literature review, short-term 50 Hz electromagnetic field therapy could prove to be a promising treatment strategy for urinary and gynecological cancers.
Keywords. electromagnetic field, DNA damage, reactive oxygen species

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Title of presented paper: Advanced technologies for isolation and characterization of circulating tumor cells as a foundation of modern liquid biopsy

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Type of the paper Review

Introduction and aim. Circulating tumor cells (CTCs) play a key role in the metastatic process and represent a promising, minimally invasive biomarker in liquid biopsy. Due to their extremely low concentration in blood, their effective detection remains challenging. This study aims to summarize current knowledge on CTC biology and modern isolation and analysis techniques.
Literature search. Relevant publications were identified in PubMed, Scopus, and Web of Science using keywords related to circulating tumor cells, liquid biopsy, isolation methods, and molecular characterization.
Analysis of the literature. The paper is based on a review of recent literature concerning molecular mechanisms of CTC survival and technological advances in their detection. Current approaches include physical enrichment methods, immunomagnetic separation (e.g., EpCAM-based) and microfluidic systems such as CTC iChip. Molecular characterization involves scRNA-seq, ddPCR, NGS and protein profiling. Results/Description of the case CTC clusters show significantly higher metastatic potential than single cells and exhibit high phenotypic plasticity, including EMT, which may hinder their detection. Changes in CTC number and molecular profile correlate with treatment response and may precede imaging-based evaluation.
Conclusion. CTCs are important prognostic biomarkers with applications in precision oncology. Their use enables real-time therapy monitoring and development of personalized treatment strategies. Combining CTC and ctDNA analysis offers improved diagnostic value, while further technological progress is needed to enhance detection efficiency.
Keywords. circulating tumor cells, liquid biopsy, microfluidics, metastasis, biomarkers

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Title of presented paper: The interferon regulatory factors family – key regulators of immunity, cellular homeostasis, and human diseases

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Type of the paper: Review

Introduction and aim. The interferon regulatory factor (IRF) family plays a key role in regulating the immune response, influencing both innate and adaptive immunity. This review aims to analyze the role of IRF genes in maintaining immune homeostasis by controlling the expression of interferons and numerous genes involved in inflammatory and antiviral responses, with a particular focus on how their functions are highly context-dependent and may exhibit protective or pathogenic effects depending on cell type and stimuli.
Literature search. The analysis covered scientific publications from electronic databases, including the PubMed database, focusing on the role of the IRF family in the immune system. The selection of literature included studies evaluating the molecular mechanisms of specific IRF genes (such as IRF1, IRF2, IRF3, IRF4, and IRF5) and their impact on cell differentiation, cytokine production, and disease pathogenesis.
Analysis of the literature. In autoimmune diseases, IRFs regulate T-cell differentiation and function, including the balance between Th17 and Treg cells, the disruption of which leads to chronic inflammation. For example, IRF4 and IRF5 modulate inflammatory responses by influencing cytokines such as IL-6 and IL-17, which promotes the development of autoimmune and inflammatory diseases. In the context of viral infections, IRFs are key regulators of the antiviral response, particularly through the induction of type I interferons, where mutations or deficiencies in genes such as IRF1 or IRF3 lead to impaired interferon production and increased susceptibility to viral infections. In tumors, IRF genes exhibit a bidirectional effect: they can inhibit tumor growth by supporting immune surveillance, for example when loss of IRF2 leads to reduced expression of MHC class I molecules and allows cancer cells to evade T cell responses, or promote tumor progression by modulating the tumor microenvironment.
Conclusion. In summary, IRF genes play a central role in regulating the immune response and constitute a key component of the pathogenesis of autoimmune, viral, and neoplastic diseases. Their pleiotropic effects and ability to modulate both protective and pathological responses make them a promising therapeutic target and biomarker in precision medicine.
Keywords. chronic inflammation, interferon regulatory factors, immune response, tumor microenvironment, viral infections

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Title of presented paper: The effect of repetitive transcranial magnetic stimulation on the miRNA profile in Alzheimer's disease

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Type of the paper: Review

Introduction and aim. Repetitive transcranial magnetic stimulation (rTMS) is a modern, non-invasive therapeutic method that can slow cognitive decline in patients with Alzheimer's disease (AD). This effect is linked to mechanisms of synaptic plasticity, in which microRNA plays a key role. The aim of the study was to evaluate the impact of rTMS on microRNA expression, which may serve as potential therapeutic biomarkers for the treatment of AD.

Literature search. The in vitro study used human SH-SY5Y neuroblastoma cells differentiated into neurons and treated with amyloid-β oligomers. A dosimetric model was applied to replicate the rTMS field used in patients within an in vitro environment. The clinical trial involved 20 patients with Alzheimer's disease and 10 healthy controls. The rTMS therapy consisted of 15 sessions targeting the precuneus. miRNA levels were determined using real-time qPCR with TaqMan assays. RNA isolation was performed using miRNeasy kits, and sample purity was confirmed by absorbance measurements.

Analysis of the literature. rTMS significantly decreased the expression of miR-26b, miR-125b, miR-181c, and miR-146a, restoring their levels to levels similar to those of the control group. In Alzheimer's patients prior to treatment, significant increases in miR-26b and miR-30b, and a decrease in miR-125b, were observed compared with the healthy control group. Correlation analysis showed that lower levels of miR-25 were significantly associated with cognitive improvement, as assessed by the ADAS-Cog scale. ROC curve analysis revealed that miR-25 achieved an area under the curve value of 0.950, enabling the differentiation between treatment responders and non-responders. No significant changes in BDNF levels were observed following rTMS application.

Conclusion. The obtained results suggest that rTMS influences specific microRNAs in patients with AD. In particular, miR-25 represents a promising direction for further research and may serve as a useful biomarker of rTMS efficacy and a prognostic marker of cognitive improvement.

Keywords. Alzheimer's disease, microRNA, miR-25, repetitive transcranial magnetic stimulation

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Title of presented paper: How can a single gene disrupt metabolism?

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Type of papier: Review

Introduction and aim. Inborn errors of metabolism constitute a diverse group of monogenic disorders in which a mutation in a single gene leads to a dysfunction of a key protein, most often an enzyme involved in a specific metabolic pathway. The consequences of such a mutation are cascading in nature and may include the accumulation of toxic substrates, a deficiency of essential reaction products, and a disruption of cellular energy balance. The aim of this study was to present the mechanisms through which a mutation in a single gene can lead to the disruption of entire metabolic pathways and the development of systemic diseases.
Literature search. This is a review article based on an analysis of the available scientific literature on inborn errors of metabolism. The PubMed and Scopus databases were searched, focusing on publications from 2020–2025, including clinical, observational, and review studies.
Analysis of the literature. Particular attention was devoted to selected amino acid disorders, such as maple syrup urine disease, tyrosinemia, and phenylketonuria, in which acute or chronic poisoning occurs due to unmetabolized substrates or byproducts. The importance of early diagnosis and available therapeutic options was also emphasized.
Conclusion. Inborn errors of metabolism demonstrate that a single-gene mutation can trigger a cascade effect, leading to dysfunction of metabolic pathways, cells, organs, and the organism as a whole.
Keywords. amino acid disorders, metabolism, gene, phenykoketonuria, syrup urine disease, tyrosinemia