About a Conference

The conference is dedicated to the memory of the Polish biochemist Professor Jerzy Popinigis, the initiator and organizer of these conferences in the 1990s. It's one of the largest international conferences in Central and Eastern Europe on free radicals and exercise biochemistry. The speakers at the conference are world-class scientists from Canada, the United States, Hungary, Estonia, Germany, Japan, Italy and Poland. The conference program will feature four key sessions exploring the role of free radicals in biology, sports, nutrition, and medicine.

Contents

I Session: Oral presentations

O.01

Lipoproteins and oxidative stress in obesity

Tiziana Bacchetti

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Human obesity, the most common nutritional disorder worldwide, is a well-established risk factor for type 2 diabetes mellitus and cardiovascular disease. Among the metabolic disturbances associated with obesity, increased oxidative stress and altered plasma lipoprotein profiles are frequently observed. These include reduced levels of high-density lipoprotein cholesterol (HDL-C) and higher levels of low-density lipoprotein cholesterol (LDL-C). Oxidized and dysfunctional lipoproteins have been also demonstrated in obesity, and it has been reported that HDL from obese subjects are characterized by impaired antioxidant and anti-inflammatory properties.

Abdominal adiposity is associated with a state of low-grade chronic inflammation. The connection between lipoproteins and adipocytes represents the potential mechanism underlying metabolic disturbance in obesity. In individuals of normal weight, adipose tissue helps preserve cardiovascular health by maintaining both HDL-C levels and HDL functions. However, in obesity, adipose tissue morphology and function are altered, impairing its regulatory role and contributing to metabolic imbalance Therefore, adipose tissue has emerged as a potential target for both the prevention and treatment of lipoprotein-related metabolic disorders in obesity.

A central strategy for prevention remains lifestyle modification, particularly through dietary changes and physical activity, which can positively influence several metabolic parameters, including plasma lipoprotein levels and functionality.

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O.02

N-homocysteinylation of ferritin cause impairment in endothelial iron metabolism- the role of Akt signalling pathway

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Research Background:
Hyperhomocysteinemia is an independent risk factor for neurodegeneration and cardiovascular disease. While iron is implicated in this condition, its molecular interactions remain unclear.

Objectives:
This study aimed to investigate the effects of homocysteine (Hcy) and its reactive derivative, homocysteine thiolactone (HcyT), on iron metabolism in Human Umbilical Vein Endothelial Cells (HUVEC) and neuronal cell line SH-SY5Y.

Methodology:
HUVEC and SH-SY5Y cell lines were treated with various concentrations of Hcy and HcyT over different time points. The study assessed cytotoxicity and examined changes in signaling pathways related to iron metabolism and homocysteinylation.

Results:
Hcy and HcyT exposure led to a marked increase in ferritin L and H subunits, correlating with elevated total and labile iron pools (LIP) in HUVEC cell line but not in SH-SY5Y. In HUVEC HcyT-treated cells, iron export proteins such as ferroportin and APP were upregulated. Iron chaperones PCBP1 and PCBP2 showed significant dysregulation, while transferrin receptor levels decreased, suggesting a protective mechanism against iron overload. Cells pre-treated with H_2O_2 were more sensitive to HcyT toxicity, confirming an iron-dependent cell death mechanism. Notably, HcyT induced N-homocysteinylation of ferritin H, likely impairing its function and contributing to the observed ferritin and LIP increase. Finally, changes in ferritin levels triggered by Hcy were shown to be regulated through the Akt-FOXO3a signaling pathway.

Conclusions:
Hcy and HcyT disrupt iron homeostasis in endothelial cells, with HcyT causing ferritin H modification and iron accumulation, contributing to endothelial dysfunction. These findings provide new insights into iron-related mechanisms in hyperhomocysteinemia.

O.03

Obesity, global syndemia and urban health

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President Italian Obesity Network

Obesity is a chronic complex disease defined by excessive fat deposits that can impair health. The inflammation is the link betwen obesity, type 2 diabetes, cancer and heart disease. The starting point are the hypertrophic white adipocytes that die by pyroptosis and pyroptosis is a proinflammatory programmed cell death. The OBS/REDOX model contributes to a unifying theory for the global rise in obesity. Obesity, undernutrition, and climate change represent the Global Syndemic. They constitute a synergy of epidemics, because they co-occur in time and place, interact with each other and have common underlying societal drivers. Obesity and diabetes have complex relations with cities. Air pollution is the characteristic of cities most frequently associated with disease. In 2005 Christopher Wild introduced "the exposome" as an innovative concept that includes environmental exposure and concomitant biological responses of the people. The exposome is the set of external stressors such as lifestyle, social ecosystem, physical factors and includes what we eat and do, our experiences, and where we live and work. The chemical exposome is an integral part of the exposome concept. The exposome induces biological responses at every level (epigenetic, transcriptome and proteome). Cities, but especially the suburbs, can cause health problems with a strong impact, caused by air and noise pollution, road accidents, the adoption of unhealthy lifestyles and the spread of infectious agents in overcrowded conditions. We need to aim for urban regeneration. Planetary health is a new concept. It refers to the interdependent health of humans and the environment, recognizing that the two are inseparable and that the health of one is intricately linked to the health of the other. The Urban Health Rome Declaration defines the strategic aspects to improve health in cities. Following this Declaration we are trying to carry out a project entitled Live Well Valnerina.

O.04

The effect of swim training and exercise-mimics treatment on life prolongation and skeletal muscle protection in Amyotrophic Lateral Sclerosis mice

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Amyotrophic lateral sclerosis (ALS) is an incurable, chronic neurodegenerative disease accompanied by body mass and skeletal muscle mass reduction. These changes were related to mitochondria dysfunction and impairment in both aerobic and anaerobic energy metabolism, as well as increased oxidative stress. Among all therapies studied in the mouse model of ALS, one of the most effective is swim training, which has been shown to prolong lifespan and ameliorate the reduction in skeletal muscle mass. Swim training also maintained mitochondrial function and lowered oxidative stress.

Therefore, the main goal of this study was to determine the effects of swim training and exercise-mimics treatment, as well as a combination of these treatment options (hybrid therapy), on the lifespan and motor function of ALS mice. In the study, we used transgenic mice with the G93A mutation in the SOD1 gene. The therapy (five times per week) began at the symptomatic stage (96 ± 4 days of age) and continued until the mice inability to continue training or death. During the animals' lives (since 70 days of age), we examined the body and tibialis anterior muscle mass, motor function, and grip strength.

Median survivals were 128 days of age in the sedentary mice, 139 days in the exercise-mimic group, and 134.5 days in the swimming group. Hybrid therapy did not induce prolongation of ALS mice's life, but we observed even a shortening of it. All therapies that result in lifespan prolongation also ameliorate the reduction in skeletal muscle mass and improve grip strength.

This study was supported by grant from National Science Centre, Poland (2021/43/D/NZ7/00862).

O.05

Boosting the effect of exercise on mitochondria to enhance muscle health with advancing age

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Regulation of skeletal muscle health involves the coordinated activity of cellular organelles. Mitochondria produce energy for muscle contraction and are increased in muscle by the adoption of a regular exercise program. In contrast, mitochondria can be reduced within the cell by extended periods of muscle disuse, and with age. This decline in mitochondria occurs when they become dysfunctional, and it is mediated by the process of mitophagy in which they are delivered to lysosomes for degradation. The terminal step of mitophagy involves the fusion of autophagosomes containing defective mitochondria with the lysosome for degradation and recycling.

Our previous work has shown that chronic exercise leads to improved mitochondrial function and content, and this therefore obligates less need to remove dysfunctional mitochondria via the mitophagy pathway. This occurs even though exercise can induce coincident increases in lysosomal degradation capacity. We have recently sought to investigate how alternative treatments can boost both the mitochondrial and lysosomal improvements induced by exercise. These include nutraceutical treatments such as sulforaphane, urolithin and resveratrol, as well mitochondrial transplant therapy. These treatments have the potential to augment exercise effects, particularly in compromised conditions such as aging muscle, to help preserve muscle health. This presentation will focus on results that illustrate the effectiveness of these treatments in pre-clinical models.

O.06

NAD and Aging Skeletal Muscle: Merit of Supplementation

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Nicotinamide adenine dinucleotide (NAD) is a versatile chemical compound serving as a coenzyme in metabolic pathways and as a substrate to support the enzymatic functions of sirtuins (SIRTs), poly (ADP-ribose) polymerase-1 (PARP-1), and cyclic ADP ribose hydrolase (CD38). Under normal physiological conditions, NAD+ consumption is matched by its synthesis primarily via the salvage pathway catalyzed by nicotinamide phosphoribosyltransferase (NAMPT). However, aging and muscular contraction enhance NAD+ utilization, whereas NAD+ replenishment is limited by cellular sources of NAD+ precursors and/or enzyme expression. This speech will briefly review NAD+ metabolic functions, its roles in regulating cell signaling, mechanisms of its degradation and biosynthesis, and major challenges to maintaining its cellular level in skeletal muscle. The effects of aging, physical exercise, and dietary supplementation on NAD+ homeostasis will be highlighted based on recent literature.

Keywords: aging; exercise; NAD+, mitochondria; skeletal muscle; sirtuin

O.07

Chronic Stress: Free Radicals, Mitochondrial Dysfunction, and the Fight for Muscle Integrity

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Chronic psychological stress is increasingly recognized as a systemic factor contributing to both skeletal muscle and brain dysfunction. Sustained hypothalamic–pituitary–adrenal axis activation and prolonged glucocorticoid (GC) exposure promote redox imbalance, mitochondrial impairment, and tissue-specific degeneration in a rat model involving prolonged stimulation of the bed nucleus of the stria terminalis (BST). Chronic stress induces distinct, fiber-type-dependent alterations in skeletal muscles and early signs of hippocampal vulnerability.

In skeletal muscle, changes in 11β-HSD1/2 levels, GR/MR protein content, and local GC activation were observed in both slow-twitch (soleus, SOL) and fast-twitch (extensor digitorum longus, EDL) fibers. While SOL exhibited partial compensatory responses, including elevated citrate synthase activity, increased PGC-1α, and IGF-1 content. In contrast, EDL demonstrated enhanced oxidative damage, reduced GR signaling, and impaired mitochondrial function.

Parallel experiments showed that chronic stress also impacts hippocampal metabolism, leading to reduced mitochondrial enzyme activity, altered BDNF-Akt signaling pathways, and oxidative damage to lipid and protein. Vitamin D_3 treatment partially prevented stress-induced changes in both systems by supporting mitochondrial function and redox balance.

Taken together, these findings suggest that skeletal muscle and the hippocampus respond to chronic stress through overlapping but tissue-specific mechanisms. Both serve as active targets of endocrine and redox disturbances, with region-dependent susceptibility. Strategies enhancing mitochondrial resilience, such as vitamin D3 treatment, may help preserve functional integrity under prolonged stress.

O.08

Melatonin: a free scavenger and skin aging molecule

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The skin, being the largest organ in the human body, is exposed to the environment and suffers from both intrinsic and extrinsic aging factors. The skin aging process is characterized by several clinical features such as wrinkling, loss of elasticity, and rough-textured appearance. This complex process is accompanied with phenotypic and functional changes in cutaneous and immune cells, as well as structural and functional disturbances in extracellular matrix components such as collagens and elastin. Because skin health is considered one of the principal factors representing overall "well-being" and the perception of "health" in humans, several anti-aging strategies have recently been developed. Thus, while the fundamental mechanisms regarding skin aging are known, new substances should be considered for introduction into dermatological treatments. Herein, melatonin as a potential "aging neutralizers" is enclosed. Melatonin (*N*-acetyl-5-methoxytryptamine), an evolutionarily ancient derivative of serotonin with hormonal properties, is the main neuroendocrine secretory product of the pineal gland. It regulates circadian rhythmicity and also exerts anti-oxidative, anti-inflammatory, immunomodulatory, and anti-tumor capacities. There is a summarizing disturbances within skin aging, research advances on the molecular mechanisms leading to these changes including mitochondrial disturbances, and the impact of the melatoninergic anti-oxidative system controlled by melatonin, targeting the prevention or reversal of skin senescence.

O.09

A novel approach for the assessment of glutathione index (GSH/GSSG) in capillary blood samples

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The ratio of reduced to oxidized glutathione (GSH/GSSG), also known as the glutathione index, serves as a critical biomarker of oxidative stress and redox balance in clinical and translational research. However, accurate measurement of this index remains technically challenging, particularly in capillary blood samples due to the high reactivity and instability of glutathione species. Conventional venous blood processing is often incompatible with decentralized or point-of-care testing. Dried blood spots (DBS), a minimally invasive sampling method, offer logistical advantages but introduce additional analytical complexities such as incomplete recovery of analytes, matrix effects, and potential oxidation during drying.

We present a novel workflow for reliable quantification of GSH and GSSG in dried capillary blood spots samples. The method integrates optimized sample collection, immediate stabilization with thiol-protecting reagent with targeted LC-MS/MS analysis with isotope-labeled internal standards to ensure accurate and reproducible measurements. Key innovations include the prevention of artifactual GSH oxidation during the drying process and enhanced extraction efficiency for both GSH and GSSG from DBS matrices.

Validation studies demonstrate excellent linearity, sensitivity, and robustness of the method across a physiologically relevant range of the glutathione index. Furthermore, the approach enables consistent analysis of samples stored at room temperature, facilitating remote monitoring and large-scale population studies.

This novel DBS-based method for glutathione index assessment holds significant promise for advancing redox biology research and supporting clinical applications in oxidative stress monitoring and personalized health.

O.10

Muscle and Brain, One and the Same: Synergies Between Physical and Cognitive Performance in Aging

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A growing body of evidence supports a powerful synergy between physical and cognitive performance. This study explores how structured physical activity promotes neurophysiological changes that not only enhance muscular strength and endurance but also protect and improve cognitive function.

Far from being isolated systems, muscle and brain operate in deep physiological interdependence—united through common biochemical pathways, energetic demands, and adaptive responses. Regular exercise enhances oxygen transport efficiency, cardiac output, and mitochondrial function. These changes support not just muscular performance but also increase arousal, cerebral blood flow, and neuro-supportive molecule synthesis such as BDNF, Irisin, Cathepsin B, and/or Lactate. Simultaneously, exercise upregulates neurotransmitter systems including dopamine, serotonin, noradrenalin, and acetylcholine. These changes contribute to improved memory, attention, executive function, and emotional regulation.

Of particular interest is how moderate to high-intensity interval training (HIIT) can activate regions like the left dorsolateral prefrontal cortex (DLPFC), crucial for cognitive control, without triggering harmful stress responses. This supports neurogenesis, synaptic plasticity, and resilience against age-related cognitive decline.

In clinical populations, exercise is increasingly recognized as a key non-pharmacological intervention for mitigating the impact of chronic conditions such as cardiovascular disease, obesity, and neurodegeneration. Understanding these shared mechanisms between muscle and brain opens promising avenues for integrated therapeutic strategies. Our study highlights the importance of promoting exercise not only for physical fitness but as a scientifically grounded tool to sustain cognitive vitality and quality of life across the lifespan.

O.11

Role of vitamin D and its derivatives in regulation of oxidative stress

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Vitamin D is a naturally occurring hormone responsible for the proper functioning of the musculoskeletal, nervous, immune, and skin systems. Therefore, the worldwide prevalence of vitamin D deficiency has a significant impact on our health. The primary source of vitamin D is the human skin exposed to sunlight. Vitamin D, which is biologically inert, requires two steps of activation through hydroxylation in the liver and kidneys. The final product, calcitriol, is a fully functional hormone that binds to the nuclear receptor VDR and modulates the expression of hundreds of genes. It is well-established that calcitriol is responsible for calcium-phosphate homeostasis; however, its pleiotropic nature suggests the existence of alternative pathways activated by vitamin D. Vitamin D deficiency or dysregulation of vitamin D signaling pathways is a common feature associated with dysmetabolic diseases, including obesity, metabolic syndrome, and diabetes, as well as many types of cancer. Curiously, it appears that one of vitamin D's primary evolutionary functions was to scavenge reactive oxygen species yet before its receptor existed. Interestingly, process of formation of vitamin D from 7-dehydrocholesterol is associated with ROS scavenging while an excess of UV results in photodegradation of this powerful molecule and further neutralisation of ROS. Furthermore, vitamin D metabolism and activity are strongly associated with mitochondria, where reactive oxygen species are generated.

Vitamin D is an important supplement that could be used in therapy. However, its potential clinical use may be limited due to its hypercalcemic effect at high doses. Thus, many low-calcemic analogs of vitamin D have been synthesized and tested.

Several studies have shown that vitamin D analogs could be effective against cancer, including melanoma. The anticancer activity of vitamin D and its analogs results from its impact on mitochondria. Our recent studies on basal cell carcinoma and melanoma cells have shown that calcitriol, affects mitochondrial ultrastructure, network, membrane potential and bioenergetics. This effect appears to be primarily facilitated by the genomic activity of vitamin D. However, a direct impact on mitochondrial ion channels has also been postulated, recently. Vitamin D activates a cellular response against reactive oxygen species (ROS) and ultraviolet radiation, as well as stimulating DNA repair.

Summarizing, despite its VDR-driven regulation of cellular homeostasis, vitamin D appears to be strongly associated with mitochondria through both, genomic pathways, a direct impact on mitochondrial bioenergetics and ROS scavenging. The pleiotropic activity of vitamin D and its derivatives could be used not only to prevent diseases associated with energy imbalance, such as diabetes or cancer, but also to treat them, especially as an adjuvant.

O.12

Mitochondria - Microbiome cross talk: is it ROS mediated?

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Wild and PGC-1 alpha overexpressed mice in the skeletal muscle with and without exercise training were used to study the possible relationship between mitochondrial network and microbiome. PGC-1 alpha levels significantly increased in the skeletal muscle and the colon, which is near the microbiome. Still, we could not detect other significant differences in cell signaling and mitochondria-related proteins in the colon. On the other hand, the H2O2 mitochondrial production of the colon decreased in the overexpressed group. The relative abundance of several bacterial species differed between wild and PGC-1 alpha overexpressed groups, suggesting a shift in the microbiome milieu to cope with increased metabolism, enhanced usage of short-chain fatty acids, and better endurance capacity. Ten weeks of exercise training has less significant effects on the microbiome of PGC-1 alpha overexpressed mice, probably due to the already modified microbiome to handle exercise-induced metabolic challenges. The results suggest a cross-talk between mitochondria and microbiome, independent of redox signaling.

O.13

Effects of the α7 nicotinic acetylcholine receptor in skin

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Background: The α7 nicotinic acetylcholine receptors (nAChR) belongs to the familiy of neurotransmitter-gated ion channels with acetylcholine or nicotine as ligands. The α7nAChR is not only expressed by neural tissues but also in the skin in cell types including epidermal keratinocytes, sebocytes and dermal fibroblasts (HDF).
Objectives: Translational research focusing on the exploitation of the α7nAChR in dermatology aims to reveal if this receptor could be a promising target for the treatment of skin diseases.
Methodology: To adress the question in vitro methods such as quantitative RT-PCR, immunofluorescence, histology, Western Immunoblotting and oxidative stress detection were utilized. Mouse models of experimentally induced fibrosis were used to confirm the obtained data.
Results: Transforming growth factor (TGF)-β1-mediated profibrotic responses of HDF could be counteracted by α7nAChR agonists. In accordance, α7nAChR agonists were effective in several models of experimentally induced skin fibrosis. Mechanistically, the antifibrotic effect of the α7nAChR in HDF was mediated via the redox-sensitive factor JunB. Moreover, TGF-β-induced mitochondrial OXPHOS dysfunction was antagonized by α7nAChR agonists. In epidermal keratinocytes that are crucially involved in the pathogenesis of inflammatory skin diseases α7nAChR agonists could suppress ultraviolet light-induced production of proinflammatory cytokines. Especially the reduction of oxidative stress involved in dermal photo aging points towards a potential of α7nAChR agonists in the prevention of extrinsic skin aging. Thus, translational research targeting the α7nAChR in skin may lead to the development of new treatment and prevention modalities against fibrosclerotic and inflammatory skin diseases as well as skin aging.

O.14

Molecular mechanisms regulating brain-derived neurotrophic factor (BDNF) expression in health and pathology

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One of the key regulators of neuronal survival and synaptic connectivity is brain-derived neurotrophic factor (BDNF), a member of the neurotrophin family. BDNF promotes neuronal survival and differentiation and regulates activity-dependent synaptic plasticity. Decreased BDNF and its receptor TrkB levels and activity are implicated in various pathologies, including neuropsychiatric and neurodegenerative disorders. Neural plasticity, the ability of the nervous system to adapt structurally and functionally in response to stimuli, underlies memory and long-term behavioural changes. Neuronal activity leads to a rise in intracellular calcium, ultimately leading to changes in transcription of specific target genes. Neuronal activity-regulated genes play a crucial role in the formation of neuronal plasticity, and dysregulation of this process gives rise to various nervous system disorders. BDNF is among the best-studied activity-regulated genes and polymorphisms of the BDNF gene are associated with impairments in human cognition. Physical exercise and intermittent fasting, on the other hand, protect synaptic and neuronal structure and function, with increased BDNF as a major mediator of exercise-induced enhancements in cognitive function. Overview of molecular mechanisms regulating BDNF expression in health and pathology will be presented and possible means of enhancing BDNF levels discussed.

O.15

Metabolic alterations and enhanced ROS in patients with rare and ultra-rare diseases

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My group is investigating metabolic alterations and disrupted redox homeostasis that could be the result of mitochondrial dysfunction and enhanced ROS production in rare and ultra-rare diseases, Neurodegeneration with Brain Iron Accumulation (NBIA) and PACS2-related disorder respectively. We try to associate observed metabolic abnormalities with the clinical phenotype of patients to propose a possible treatment strategy.

A primary rare disease of interest in our research is NBIA (formerly known as Hallervorden-Spatz disease) encompasses a group of rare inherited disorders that share the clinical features of an extrapyramidal movement disorder accompanied by excessive iron deposition in the deep basal ganglia nuclei of the brain. The prevalence of NBIA is quite low (1–3 cases/1 000 000 population). Among 11 NBIA subtypes, especially in the case of mitochondrial membrane protein-associated neurodegeneration (MPAN) caused by mutation in *C19orf12* gene, molecular mechanism underlying MPAN is still not fully understood. What is more important, MPAN seems to be the most common and dominant NBIA subtype in Poland.

Another disease we are working on is PACS2-related disorder, which is an ultra-rare disease with about 32 cases reported in the literature and approximately 100 in total diagnosed worldwide. Two mutations in the *PACS2* gene (c.625 G > A and p.Glu211Lys) are responsible for the development of this syndrome. PACS2 (phosphofurin acidic cluster sorting protein 2) is a multifunctional cytosolic membrane trafficking protein that plays an important role in modulation of ER and mitochondria communication. We hypothesize that alterations at the level of mitochondria-ER contact sites are key contributors to the pathogenesis of PACS2 syndrome.

We believe that our study may indicate which processes or proteins might serve as potential pharmacological targets for NBIA-MPAN and PACS2-related disorder.

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O.16

Why is myoglobin an essential protein for exercise performance?

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Myoglobin (Mb) is expressed in vertebrate cardiac, skeletal and smooth muscle cells and reversibly binds O_2 by its haem residue (Hendgen-Cotta *et al.,* 2014). It is well established that Mb can store oxygen as well as can also facilitate its diffusion to mitochondria (for review see, e.g., Wittenberg & Wittenberg, 2003). Current studies demonstrated that Mb plays a role of a myocardial O2 sensor and also scavenges $NO^{\bullet}$ in its oxygenated state, whereas under hypoxia deoxygenated Mb acts as an $NO^{\bullet}$ producer, which contributes to cardiomyocyte $NO^{\bullet}$ signaling (for a review see Hendgen-Cotta *et al.,* 2014). Following this concept it has been recently proposed by Clanton (2019) that Mb might play a similar role in the skeletal muscle during exercise as a PO_2-dependent catalytic switch for the oxidation/reduction reaction of $NO^{\bullet}$ and NO_2-. These aforementioned functional properties of Mb suggest its potentially important role during exercise, but surprisingly some influential studies using myoglobin knockout mice (Mb-/-) have questioned its importance for exercise performance (Garry *et al.,* 1998; Gödecke *et al.,* 1999). During this lecture a new data from our recent experiments involving Mb-/- in relation to control mice (Mb+/+) will be presented (Zoladz *et al.,* 2024). The main outcome of this study was that myoglobin is of vital importance for maximal oxygen uptake ($V\square O_2max$) and maximal running performance as well as we have shown why the previous studies have failed to prove such a role of myoglobin when using the $Mb^{-/-}$ mouse model (Zoladz *et al.,* 2024).

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II Session: Poster Contest

PC.01

Per and polyfluoroalkyl substance (PFAS) exposure as a potential factor influencing clinical features of polycystic ovary syndrome (PCOS)

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Background study: PCOS is a heterogeneous endocrinopathy, the leading endocrine disorder among women of reproductive age, characterized by anovulatory menstruation, biochemical or clinical evidence of hyperandrogenism, and polycystic ovary on transvaginal ultrasound. Women with this syndrome are associated with insulin resistance, leading to metabolic consequences like T2DM and hypertension, which together predispose them to the development of cardiovascular diseases. The pathogenesis remains unclear, with some evidence suggesting that endocrine disruptors might be a risk factor.

Objective: To determine, using Ultra-high performance liquid chromatograph-tandem mass spectrometer (UHPLC-MS/MS), the serum concentration of PFAS in women with PCOS and the control group, and to establish if there is a potential link between PFAS exposure and the clinical course or features of PCOS

Methodology: 360 selected females aged 18- 40 years, with 200 diagnosed with PCOS, and 160 control group. Blood samples were taken between the 6th and 10th day of their menstrual cycle for the following analysis: fasting glucose, lipid profile, hormonal assay using electrochemiluminescence immunoassay, and PFAS analysis using UHPLC-MS/MS

Results: Hormonal results: Elevation of LH, FSH, PRL, insulin, androgens, and decreased levels of SHBG, E2 among the PCOS group compared to the control group.

Serum PFAS: Decreased levels in the PCOS group compared to the control group

This possibly points out that there might be a possible interaction of PFAS with implicated PCOS hormones, and it can be a feasible way of PFAS potentiating PCOS clinical features.

Keywords: PCOS, PFAS, T2DM, CVD, endocrine disruptor

PC.02

Blood prooxidant-antioxidant balance and cortisol pre- and post-surgery in patients with benign parotid gland tumours: A preliminary study

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Background: The majority of parotid gland tumors are benign e.g. pleomorphic adenoma (PA) and Warthin's tumor (WT). Oxidative stress from a biomedical point of view is significantly important because of its association with a various types of cancer. Oxidative stress has been implicated in the origin and development of various cancers including parotid cancers.

Objectives: The main objective of the study was to evaluate blood prooxidant-antioxidant balance indicators and cortisol levels in patients with benign parotid gland tumors before and after tumor removal.

Methodology: Patients (n=20) diagnosed with PA (n=14) and WT (n=6) after histopathological verification and computed tomography (CT) were qualified for surgical treatment. Blood for biochemical tests was collected before and after surgery. The activities of the antioxidant enzymes (SOD, CAT and GPx), the non-enzymatic antioxidant (GSH), oxidative stress markers (MDA and TOS) and cortisol concentration (Cor) were determined in the blood.

Results: Following surgical intervention, no statistically significant alterations were detected in the activities of antioxidant enzymes. A significant postoperative increase in serum TOS was observed ($p<0.05$), whereas both plasma MDA levels and blood GSH concentrations demonstrated a significant decrease. Cortisol (Cor) levels remained unchanged, with no statistically significant differences observed postoperatively.

Conclusions: The findings of our study indicate that, despite a pronounced reduction in MDA concentrations, a significant increase in TOS was observed in patients following salivary gland excision. This paradoxical increase in oxidative stress may be attributed to a concomitant and significant decline in GSH levels in patients undergoing tumor resection. Nevertheless, further studies are warranted to elucidate the underlying mechanisms and validate these observations.

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PC.03

Effect of combined therapy of vitamin d3 and sodium butyrate on neuroprotection and mitochondrial dynamism in an experimental model of ischemic stroke

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Introduction: Ischemic stroke (IS) remains a leading global cause of death and long-term disability. Although previous studies have highlighted the neuroprotective effects of vitamin D3 (VD) and sodium butyrate (SB), the role of mitochondria in these effects, particularly in aged rats, remains unclear. This study aimed to investigate the neuroprotective efficacy of a combined VD and SB treatment administered within the first hour post-IS induction in aged rats. Additionally, we aimed to explore the impact of this therapy on mitochondrial function and oxidative stress.

Methods: To determine the effects of combined therapy of VD and SB on IS, twenty male Wistar rats (18–20 months old) were randomly divided into five groups: sham, stroke-control, VD-stroke, SB-stroke, and VD&SB-stroke. IS was induced via endothelin-1, followed by treatment administration within the first hour. Rats were treated with SB (300 mg/kg, i.p.) and VD (150 μg/kg). Brain samples were collected on the fifth day post-stroke.

Results: Our results demonstrate that the combined VD and SB therapy effectively inhibited mitochondrial fission, as evidenced by reduced Drp-1 and MFF protein content, while preserving fusion processes through maintained MFN2 and pDrp-1 content. Cytochrome c oxidase activity remained comparable to that of the sham group. The concentration of sulfhydryl groups was significantly higher compared to the stroke-control group.

Conclusion: These results suggest that early post-stroke intervention using combined VD and SB has neuroprotective effects, likely mediated through the normalization of mitochondrial dynamics, energy metabolism, and reduction of oxidative stress. This therapeutic strategy may offer promising potential for neuroprotection following ischemic stroke in the elderly.

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PC.04

Effects of amyotrophic lateral sclerosis progression on mitochondrial bioenergetic in mice spinal cord and skeletal muscle

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Amyotrophic lateral sclerosis (ALS) is a progressive, incurable neurodegenerative disease accompanied by muscle atrophy. During the development of ALS abnormalities occur in mitochondrial morphology and physiology, decreasing electron transport chain efficiency and increasing reactive oxygen species generation.

The goal of this study was to investigate how ALS affects the metabolism of skeletal muscle and spinal cord at different stages of disease.

All experiments were performed on transgenic male SOD1-G93A mice, which were divided into 3 groups. Sacrificed: before the first symptoms, at the onset, and at the terminal stage of the disease. We collected quadriceps muscle and spinal cord samples from mice killed by cervical dislocation. Next, we prepared a 10% tissue homogenate, and used it to perform high-resolution respirometry measurements using Oxygraph-2k. Digitonin was used to permeabilize cell membranes. Measurements were made according to the substrate-uncoupler-inhibitor titration protocol, which allows us to evaluate respiratory chain activity.

Our research shows a reduction in aerobic metabolism in skeletal muscle as the disease progresses. The activity of complex I is significantly reduced (62%, $p = 0.0011$) in terminal mice compared to before symptoms. A 26% reduction ($p = 0.0130$) was also observed in OCE CI value of terminal mice, suggesting that diminished OXPHOS coupling efficiency depended on complex I. At the same time, we haven't noticed significant differences in the spinal cord energy metabolism.

Our results confirm that development of ALS leads to significant deterioration of mitochondrial energetics; however, we observed changes only within skeletal muscle.

This study was supported by grant from National Science Centre, Poland (2020/39/B/NZ7/03366).

PC.05

Redox imbalance modulates antioxidant activity in an in vitro glutathione-depletion model of human colon cancer (HT29) cells

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While antioxidants are widely recognized for their protective roles against oxidative stress, recent findings suggest that their effects may be dependent on cellular redox status. Reactive oxygen species (ROS) not only act as harmful oxidants but also function as crucial signaling molecules, raising concerns about indiscriminate antioxidant use under pathological conditions.

This study aimed to evaluate how glutathione (GSH) depletion affects the efficacy of exogenous antioxidants using an in vitro redox-imbalanced model. First human colon cancer cells (HT29) were treated with buthionine sulfoximine (BSO), an inhibitor of GSH synthesis. Cytotoxicity of BSO was assessed by MTT assay, and genotoxicity by comet assay. Next, intracellular GSH levels after BSO treatments were measured using HPLCMS/MS. Based on this results concentration of BSO and treatment times were selected for further research. Cellular antioxidant activity (CAA assay) was evaluated for selected antioxidants in normal HT29 cells and in GSH-depleted model.

BSO treatment at concentrations ≥50 μM caused dose- and time-dependent cytotoxicity and significantly decreased intracellular GSH levels (up to 75% after 24 h). Partial GSH recovery was detected after 72 h. Genotoxic effects of BSO were minimal. CAA assay revealed that most antioxidants, including vitamin C and polyphenols, had reduced activity under GSH-deficient conditions. Interestingly, naringenin, NAC, and p-coumaric acid displayed pro-oxidant effects, while Trolox retained stable antioxidant capacity regardless of redox status.

These results highlight the crucial role of redox balance in shaping antioxidant function. Under oxidative stress, certain antioxidants may exert harmful effects, reinforcing the need for redox-sensitive therapeutic strategies.

PC.06

Evaluation of the antioxidant capacity of rose aerial parts

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Botanists estimate that there are over 200 species and more than 20.000 varieties of roses. In cosmetics, rose hydrosols are known for their soothing and regenerative properties and are obtained through the distillation of rose petals. Rose essential oils are also valued for their anti-inflammatory, calming, and aromatherapeutic effects.

The aim of the study was to obtain alcoholic extracts from the aerial parts of Rose (petals, buds, leaves) using Soxhlet extraction and to analyze the antioxidant potential of the obtained extracts using the DPPH radical method. The extracts were analyzed using gas chromatography coupled to mass spectrometry in order to identify valuable chemical compounds with antioxidant activity.

Four five-percent extracts were prepared from aerial parts of rose using 96% ethanol as the solvent. The extraction process lasted 2 hours. Spectrophotometric analyses at a wavelength of 517 nm using the

purple DPPH radical enabled the determination of high antioxidant activities in the alcoholic extracts from the aerial parts of Rose. The antioxidant potential was as follows: fresh Rose petals – 4.14±0.02

mg TEAC/g, fresh Rose leaves – 3.93±0.01 mg TEAC/g, dried Rose petals – 4.2±0.03 mg TEAC/g and dried Rose buds – 4.23±0.01 mg TEAC/g.

The aerial parts of the Rose, such as fresh petals and leaves, as well as dried petals and buds have presented high antioxidant potential. Analyses performed using GC-MS method confirmed that alcoholic extracts from the aerial parts of the Rose contain compounds having antioxidant properties such as quinic acid, 1,2,3-benzenetriol, 4H-pyran-4-one, 2,3-dihydro-3,5-dihydroxy-6-methyl-, phytol, γ-sitosterol and eugenol.

Keywords: rose, antioxidant potential, DPPH, GC-MS method.

PC.07

Assessment of Oxidative Stress Indices and Total Phenolics Concentrations in Children with Autism Spectrum Disorder

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Background: Autism spectrum disorder (ASD) is a severe neurodevelopmental disorder in early childhood characterized by impairment in communication and behavior. Recent research is focused on oxidative stress as a potential pathomechanism of children with ASD functioning.
Objectives: The aim of the study was to analyze selected indicators of oxidative stress in children with autism spectrum disorder (ASD) and their typically developing peers.
Methodology: A total of 77 children participated in the study. The study comprised 47 children diagnosed with ASD and 30 neurotypical children who were included as typically developing (TDs). The concentration of the total antioxidative potential was determined by the Ferric Reducing Ability of Plasma (FRAP) method. Moreover, an assessment of concentration of thiobarbituric acid reactive substances (TBARS) and total phenols was carried out. Moreover, parents of children with ASD fulfilled the Autism Treatment Evaluation Checklist (ATEC).
Results: Our results showed no statistically significant differences between the mean concentrations of FRAP, TBARS and total phenols in both groups. Analysis of the correlation of the obtained results of the biochemical test showed a significant dependence of age and mean levels of total phenols ($r = 0.3499$) and TBARS ($r = 0.4888$) in ASD boys. A positive statistical correlation was observed between age and mean levels of total phenols ($r = 0.5398$) in typically developing boys.
Conclusion: There are no significant differences in the oxidative stress and total phenols concentration in the studied groups. Children's age and gender are among the factors influencing total phenols and TBARS concentration.

PC.08

Investigating Oxidative Stress Responses in PACS-2 Syndrome

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Mitochondria-endoplasmic reticulum contact sites (MERCs) are microdomains critical for metabolite transfer and ion flux between organelles, regulating processes like phospholipid biosynthesis, calcium signaling, mitochondrial metabolism and apoptosis. Moreover, both mitochondria and endoplasmic reticulum are sites of ROS production, therefore the communication at MERCs participates to the diffusion of the harmful effects of ROS. Phosphofurin acidic cluster sorting protein 2 (PACS-2) localizes to MERCs, modulating these functions. A missense mutation (E209K) in *PACS-2* causes PACS-2 syndrome, an ultra-rare genetic disorder characterized by early-onset developmental and epileptic encephalopathy with cognitive impairment. The disease's pathogenesis is unknown, though metabolic dysfunction and oxidative stress are hypothesized.
This study investigated the impact of the *PACS-2* E209K mutation on reactive oxygen species (ROS) production and oxidative damage repair. ROS levels were significantly elevated in fibroblasts from PACS-2 patients compared to healthy controls, suggesting increased oxidative stress. Despite elevated ROS, no significant oxidative damage was detected, potentially indicating active cellular repair mechanisms. Comparative proteomic analysis revealed no significant alterations in antioxidant enzymes. However, changes in the levels of proteins involved in oxidative damage repair were observed. These findings suggest that while PACS-2 mutation may be associated with elevated ROS production, repair mechanisms may mitigate oxidative damage in fibroblasts of PACS-2 patients.

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PC.09

Beneficial effects of quercetin on glucose metabolism and oxidative status in fructose-fed rats. Surprising results of fructose-methylcellulose combination

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A general increase in calorie consumption, particularly from refined carbohydrates including fructose, is clearly correlated with a concerning rise in metabolic syndrome. This study aimed to enhance insulin sensitivity in animals fed with fructose by administering the flavonoid quercetin (QCT). After consuming a 10% fructose solution for 9-weeks, the fructose fed rats received QCT (20 mg/kg/day in 1% methylcellulose given by gavage) for additional 6-weeks. The frucose-control group of rats received fructose plus the methylcellulose vehicle. The fructose-non-consuming groups of animals received tap water with methylcellulose and tap water with QCT in methylcellulose, respectively. The most significant finding from the QCT treatment was the normalization of fructose solution consumption, bringing it down to levels similar to that of drinking water. Additionally, QCT supplementation notably reduced plasma glucose levels and the HOMA-IR in the fructose-consuming rats. Surprisingly, the ingestion of fructose did not lead to an increase in plasma uric acid, thiobarbituric acid reactive substances, nitrotyrosine, or advanced glycation end product´s fluorescence. Instead, a reduction in these parameters was observed. Additionally, we found increased protein expression of Nrf2, Keap1, and SOD2 in the kidneys of rats fed with fructose or in combination with QCT. These results suggest that QCT supplementation reduces fructose consumption and increases insulin sensitivity. Furthermore, the combination of methylcellulose with fructose appears to have uric acid-lowering, antioxidant, and anti-glycation effects. This implies that methylcellulose may shift fructose to an alternative pathway with a partially positive impact on metabolism. We speculate that the interaction between fructose and methylcellulose mimics that of fructose with fiber found in unprocessed fruits.

This study was supported by APVV-20-0421, VEGA 2/0105/24, VEGA 2/0148/22, Grant No: 451-03-9/2021-14/200017.

Keywords: Quercetin; Fructose-rich diet; Methylcellulose; Glucose metabolism; Uric acid; TBARS

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PC.10

Antioxidant Capacity and Oxidative Stress Response to a Paleolithic Diet in Professional Athletes

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Research background: The impact of alternative diets on oxidative stress in athletes depends on their composition. Nutrient-rich diets may enhance antioxidant defenses, while restrictive ones can increase oxidative damage. This study compared plasma oxidative stress markers and antioxidant capacity in professional handball players following a Paleolithic diet.
Objective: Twenty-five professional handball players were assigned to two groups for an 8-week intervention: an experimental group (n = 14) following a Paleolithic diet (PD) and a control group (n = 11) adhering to a rational, balanced diet (CD).
Methods: Based on individual energy and nutrient requirements, normoenergetic all-day food rations were planned for each athlete and prepared by a catering company. Total oxidant status (TOS) and total antioxidant status (TAS) were measured at baseline and after 4 and 8 weeks using enzymatic assays. The oxidative stress index (OSI) was calculated as the TOS-to-TAS ratio.
Results: During the experiment, TAS in the PD group ranged from 374- 380 μmol/L, TOS from 196- 222 μmol/L, and OSI from 0.51- 0.56, with no significant differences compared to the CD group. The PD group consumed more antioxidant vitamins (C, E, A), while selenium, iron, zinc, and copper intake levels were similar. Manganese intake was higher in the CD group.
Conclusion: The Paleolithic diet maintained a high antioxidant status and moderate oxidative stress, while the OSI remained low, likely attributable to the diet's rich antioxidant content. Both diets demonstrated very high nutritional quality, although they differed significantly in macronutrient composition and in the intake of specific antioxidant micronutrients.

PC.11

Omega-3 Index and Its Impact on Selected Parameters of Muscle Strength in Athletes – A Randomized Controlled Trial

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Background: Omega-3 fatty acids—eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)—are essential nutrients that play a key role in modulating inflammatory processes, muscle regeneration and adaptation to physical exertion. The Omega-3 Index (O3I), defined as the percentage of EPA + DHA in erythrocyte membranes relative to total fatty acids, is a reliable marker of the body's omega-3 status.
Objective: To determine whether a 12-week strength-training programme change O3I in athletes and whether these changes correlate with selected muscle-strength parameters.
Methods: In a 12-week randomized controlled trial, 34 participants trained three 3x/week (36 sessions) and 11 served as a non-exercise control group. Participants (18–30 years, < 1 year training experience, ≤ 3 sessions week) followed an identical programme with equal volume and mean intensity (%1-RM). O3I was measured before and after the intervention, and strength was assessed with a Kistler force plate (Fmax, Frel, F150–250 ms) and a Biodex system (maximal torque of knee and elbow flexors/extensors at 95° and 135°).
Results: O3I was similar in both groups and below the recommended level. Only a weak correlation was found between O3I and gains in selected strength parameters: a slight increase in maximal knee and elbow torque coincided with higher O3I, but the relationship was weak. Other strength indices did not change significantly.
Conclusions: O3I in the training group did not differ from the control group and remained below the norm in all participants, indicating the need for greater EPA and DHA intake to achieve optimal O3I.
Relationship between O3I and muscle strength parameters needs further analysis.

PC.12

Multimodal assessment of the energy metabolism and oxidative stress in coronary microvascular endothelial cells during heart failure

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Background Endothelial dysfunction plays a crucial role in the development of heart failure (HF), contributing to the pathogenesis of coronary artery atherosclerosis and coronary microvascular dysfunction. The disrupted function of endothelial cells (ECs) is characterized by impaired energy metabolism and heightened oxidative stress. Elevated levels of reactive oxygen species (ROS) disrupt the balance of nitric oxide (NO) production, a critical regulator of vascular tone, inflammation, and cellular homeostasis.
Objectives Assessment of the energy metabolism and oxidative stress in coronary microvascular endothelial cells isolated from human hearts.
Methods The study utilized primary endothelial cells isolated from human left ventricular myocardium obtained from healthy donors excluded from use for transplant surgery (n=5) and heart failure patients with reduced ejection fraction (HFrEF, n=5). Experiments were conducted at the 3rd, 4th, and 5th passages. Endothelial cell bioenergetics were evaluated by analyzing mitochondrial respiration and glycolysis using the Seahorse XFp metabolic analyzer. Mitochondrial morphology was assessed using transmission electron microscopy (TEM). The MitoSox assay was used to determine mitochondrial ROS.
Results In endothelial cells isolated from both healthy and failing human hearts, we observed a progressive decline in mitochondrial function and oxidative phosphorylation over time of cell culture. Moreover, mitochondrial function is less efficient in ECs from HF. ECs from healthy hearts compensate for mitochondrial dysfunction by increasing the rate of glycolysis. Microscopy has shown that mitochondria from failing hearts are characterized by poorly formed cristae, a less dense matrix, and ballooning of the mitochondrial membrane. Mitochondrial ROS are elevated in the ECs from failing hearts, reflecting their pathological state.

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PC.13

Targeting Oxidative Stress in Monocytes and Macrophages Through Bioactive Compounds

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Bioactive compounds offer several benefits to human health by altering metabolic pathways and acting as antioxidants and anti-inflammatory agents. Inflammation is often described as a double-edged sword- it is essential for managing various defence mechanisms, but when uncontrolled, it can have detrimental effects. Reactive oxygen species (ROS) are considered key signalling molecules that regulate the initiation and progression of inflammation. In this study, we aimed to identify an eco-friendly approach to counteract the negative consequences of inflammation. We used bioactive compounds- chlorogenic acids, oleuropein, tomatine, and tyrosol from biological waste and investigated their effects on the modulation of inflammation. The impact of these compounds on redox balance was evaluated in U-937 and THP-1 monocytic cell lines. Following differentiation, cells were treated with the bioactive compounds, and changes in their morphological architecture and cellular integrity were assessed using confocal microscopy.

Cytotoxicity assays indicated no adverse effects in the presence of the bioactive compounds. Furthermore, the expression of reference genes, as well as inflammatory and anti-inflammatory markers, was evaluated via western blot analysis. Notably, treatment with the bioactive compounds led to a downregulation of the anti-inflammatory marker IL-4 and a reduction in myeloperoxidase (MPO) expression. Additionally, some compounds promoted the suppression of lipoxygenase-5 (LOX-5) expression, which is directly involved in the generation of inflammatory mediators. Overall, our findings demonstrate that bioactive compounds derived from biological waste exhibit anti-inflammatory properties and could serve as potential candidates for the treatment of inflammatory diseases and for use in nutraceutical products.

PC.14

Effect of doxorubicin on proteins involved in autophagy in HEK293 cells

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Background: Doxorubicin (DOX) is a widely used anticancer drug known to induce oxidative stress and cell death through various mechanisms, including reactive oxygen species (ROS) generation, DNA-adduct formation, topoisomerase II inhibition, and disruption of Ca^{2+} homeostasis. Autophagy, a catabolic process crucial for cellular homeostasis, degrades protein aggregates and damaged organelles. Oxidative stress can trigger autophagy, which plays a dual role—either protective or contributing to cell death.

Aim: To investigate the influence of DOX on proteins involved in autophagy regulation in HEK293 cells.

Methods: HEK293 cells were exposed to varying concentrations of DOX for different durations. Protein expression levels were analyzed using Western blot, while cytotoxicity was assessed by MTT assay.

Results: DOX exposure led to changes in autophagy marker expression. Our previous data showed that prolonged exposure to 2,5 μmol/L DOX upregulated Atg5, Atg12, LC3A/B, and Beclin-1. The current study found that even a 3-hour treatment with 2,5 μmol/L DOX increased Atg5 levels. Short-term exposure also elevated LC3A/B, Atg5, and Atg3, with the most pronounced effects at 1 μmol/L DOX.

Conclusion: The results point to an important role of proteins involved in autophagy processes in mechanisms of DOX action on cells, and the effects of DOX were for some autophagy markers dependent on the concentration of used DOX and the duration of cell exposure.

Key words: autophagy, doxorubicin, oxidative stress

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PC.15

Chaenomeles superba as a Rich Dietary Source of Proanthocyanidins and Antioxidants: A Comparative Study of 20 Cultivars

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Dietary antioxidants play a key role in maintaining redox balance in the human body and in the prevention of oxidative stress-related diseases. Quince fruits (*Chaenomeles* spp.) are known as a rich source of flavonoids and terpenoids, which exhibit a range of health-promoting properties, including antidiabetic, anti-inflammatory, and antioxidant activities. *Chaenomeles superba* is a hybrid of Chinese (*C. speciosa*) and Japanese (*C. japonica*) quince that is cultivated both ornamentally and for its fruit. Numerous cultivars have been developed through intra-hybrid crosses to improve fruit size, horticultural traits, and bioactive potential. The aim of this study was to evaluate the antioxidant potential of 20 cultivars of *C. superba* using a multi-method approach and to identify promising cultivars for future cultivation, with the well-known Latvian cultivar 'Cido' serving as a reference. All plants were grown under the same environmental conditions and obtained from a local plantation near Gdańsk (Straszyn, Poland). Antioxidant activity was assessed using three complementary methods: ABTS and DPPH spectrophotometric assays, HPTLC-bioautography (ABTS and DPPH), and HPLC with post-column ABTS derivatization. Total antioxidant capacity was expressed as Trolox Equivalent Antioxidant Capacity (TEAC). The results indicate considerable variation in the composition of individual antioxidants depending on the *Chaenomeles* cultivar. In spectrophotometric assays, the extract from cultivar EII23 exhibited the highest radical scavenging potential. Among the main antioxidant compounds identified were flavan-3-ol derivatives, including procyanidin B2 and C1. These findings underscore the nutritional potential of *C. superba* and demonstrate the value of integrated antioxidant screening methods for selecting superior cultivars for cultivation.

PC.16

The 8 days of only-water fasting reduce serum TMAO concentration not in all subjects

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Research background:
Trimethylamine-N-oxide (TMAO) is formed as an oxidation product of trimethylamine catalyzed by the action of flavin monooxygenases in the liver. It is a metabolite produced by the gut microbiota from dietary precursors such as choline and carnitine. Currently, evidence suggests that elevated TMAO levels may contribute not only to cardiovascular disease risk but also to the pathogenesis of neurodegenerative diseases, including Alzheimer's disease.
Objectives:
The main objective of this project is to evaluate whether an 8-day water-only fasting intervention will influence TMAO concentration in a group of 18 individuals.
Methodology:
The study involved an 8-day water-only fasting protocol. All subjects underwent an exercise test on a cycloergometer before and after the intervention. Blood and urine samples were collected before and after the test from all participants. The samples were analyzed for various biochemical parameters, including TMAO concentration, using liquid chromatography-mass spectrometry.
Results:
After the intervention, more than 70% out of the total study group showed a measurable decrease in plasma TMAO concentration. Likewise, most of the subjects also showed decreases in urinary TMAO levels per gram of creatinine. One of them started with a very high level of TMAO concentration (almost 10 times higher than the reference level); his final value was lower but still very high. Physical exercise did not cause any significant changes.
Conclusions:
Fasting is effective in reducing TMAO concentration, while single exercise had no effects. Additional studies are needed to explain why fasting did not reduce TMAO in some subjects.

PC.17

SGLT2 inhibitors combined with exercise attenuate renal damage in an experimental model of diabetic nephropathy

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Background: Diabetic kidney disease (DKD) treatment remains a significant clinical challenge; however, despite the recently demonstrated benefits of SGLT2 inhibitors, the residual risk of progression is still present, and strategies involving combined interventions may provide additional benefits.
Objectives: to evaluate the effects of sodium-glucose cotransporter 2 inhibitor (SGLT2i) empagliflozin (35 mg/kg/day) and moderate-intensity physical training (50-min sessions every 48 h at 60–80% of maximal velocity), both individually and in combination, on DKD progression.
Methodology: Male C57BL/6J mice exposed to a high-fat diet and a single dose of streptozotocin (STZ) (40 mg/kg) were assigned to four groups (n=12/group): Control, SGLT2i, physical training (PhTr), and SGLT2i+PhTr. Mice were evaluated over 25 weeks. Body mass, caloric intake, blood glucose, glomerular morphology, neutrophil gelatinase-associated lipocalin (NGAL) expression, oxidative stress markers, inflammatory cytokines, and inflammasome-pyroptosis axis were evaluated.
Results: At week 25, the SGLT2i+PhTr group exhibited significantly lower body mass compared with the Control group. SGLT2i and SGLT2i+PhTr demonstrated the most pronounced effects on blood glucose level reduction. All treatments improved glomerular morphology and reduced NGAL expression. SGLT2i treatment decreased oxidative stress markers in kidney tissue, whereas the combined therapy modulated inflammatory cytokines in skeletal muscle. Despite unchanged inflammasome components, all interventions reduced gasdermin D (GSDMD) immunoexpression. These findings highlight distinct renal- and musclespecific responses to SGLT2i and PhTr. Both single and combined interventions demonstrated distinct effects in experimental DKD, underscoring the potential benefits of a multimodal therapeutic approach.

Keywords: Oxidative stress; SGLT2i; Obesity; Physical training; Diabetic Kidney Disease.

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PC.18

Effects of fermentation and storage on the antioxidant activity of white cabbage (*Brassica oleracea* L. var. capitata L.)

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Fermented cabbage (*sauerkraut*) is a traditional Polish food product with significant cultural, nutritional, and economic importance. Poland ranks among the leading global producers and exporters of sauerkraut, with a strong tradition of natural fermentation without preservatives or pasteurization. Fermentation is a dynamic process in which native compounds degrade, and transform, which affects both nutritional and health-promoting properties of the final product.
This study aimed to evaluate changes in antioxidant activity and composition during fermentation of white cabbage and subsequent storage. Total antioxidant activity (TAA) in cabbage extracts and fermentation brines was assessed using ABTS, DPPH, and Folin-Ciocalteu assays, which reflect the overall capacity to scavenge free radicals. To gain deeper insight, HPTLC coupled with bioautographic derivatization was applied to identify specific antioxidant compounds, while HPLC enabled detailed profiling and quantification.
Results showed that raw cabbage had the highest TAA, which decreased already when plant material was being prepared for fermentation. Shredding a cabbage mechanically disrupts plant cells, releasing enzymes, while salting induces osmosis, removing water-soluble antioxidants. After fermentation began, antioxidant activity dropped further by approximately 50% and remained stable throughout storage. Chromatographic analyzes revealed degradation of native antioxidants and formation of new radical-scavenging compounds during fermentation. Interestingly, antioxidants accumulation in the fermentation brine resulted in nearly twice the activity compared to raw cabbage juice.
These findings highlight the complex transformations of antioxidants during fermentation and demonstrate the value of chromatographic techniques in tracking compound-specific changes. Understanding these processes supports the development of functional fermented foods with enhanced health-promoting properties.

PC.19

Melatonin scavenges UVR-induced free radicals and alterations in mitochondria targeting the prevention of skin aging

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Cellular senescence is an irreversible growth arrest that occurs as a result of different damaging intrinsic and extrinsic stimuli, including DNA damage, telomere shortening and dysfunction or oncogenic stress. Human skin, the largest organ of the body, provides a physical barrier against harmful microbes, toxins, and protects from ultraviolet radiation (UVR). Increasing evidence suggests that senescent cells accumulate in chronologically aged and photoaged skin; and may contribute to age-related skin changes and pathologies. Skin health is considered one of the principal factors representing overall "well-being" in humans. Thus, there is an imperative to consider melatonin's regulatory activity on cellular senescence of the skin.

Melatonin, an evolutionarily ancient derivative of serotonin with hormonal properties, is the main neuroendocrine secretory product of the pineal gland. It regulates circadian rhythmicity and exerts anti-oxidative, anti-inflammatory, immunomodulatory, and anti-tumor capacities.

Herein, in vitro studies using human epidermal keratinocytes exposed to UVB (50 mJ/cm^2), we noticed that melatonin attenuates altered cell survival ratio, and affects expression of senescence markers (p53, p16, γH2AX, IL-6). Moreover, melatonin ameliorates UVB-induced oxidative stress and depolarization of mitochondrial transmembrane potential (mtΔΨ) indicating the mitochondrial dysfunction-associated senescence (MiDAS). In vivo studies have been performed using C57BL/6 mice treated subcutaneously biennially with melatonin (10 mg/kg) and changes in skin aging has been substantially ameliorated followed by melatonin-treated mice compared to control ones. These data enclose changes within skin aging and the impact of the melatoninergic anti-oxidative system controlled by melatonin, targeting the prevention or reversal of skin senescence.

PC.20

Chemiexcitation-induced melanogenesis in SK-MEL-1 cells following UVA

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Melanogenesis is a complex cellular process that serves as a protective mechanism against UV radiation by increasing melanin production in the skin. While the stimulatory effect of UVB radiation on melanogenesis is well documented, mainly via direct DNA damage, UVA radiation presents a different biological challenge. UVA radiation does not cause direct DNA damage, yet it may still induce melanogenesis through alternative mechanisms, chemiexcitation, a process involving electronically excited species generated by oxidative reactions. During chemiexcitation, triplet excited carbonyls formed via oxidative reactions can transfer energy to DNA bases, leading to the formation of cyclobutane pyrimidine dimers (CPDs) in the absence of direct UVB-induced photodamage. These "dark CPDs" can activate the p53 pathway, which plays a key role in initiating melanogenesis through transcriptional upregulation of melanogenic factors. This study explores the potential of UVA-induced chemiexcitation to initiate melanogenesis in SK-MEL-1 cells. The cells were exposed to varying doses of UVA radiation, and changes in the expression of key regulatory proteins, including p53, MITF, TYRP1, TYRP2, and tyrosinase, were analyzed using western blotting. In parallel, melanin content was quantified spectrophotometrically. Preliminary data indicate that UVA exposure modulates the expression of melanogenesis-related proteins and leads to increased melanin production, suggesting a chemiexcitation contributes to melanogenesis. These findings support the hypothesis that melanogenesis can be initiated independently of direct DNA damage caused by UVB and provide new insight into the molecular effects of UVA radiation.

PC.21

Effects of Dimethyl Fumarate and Vitamin D3 on Oxidative Stress and Cognitive Function in a Rat Model Mimic Alzheimer's Disease Symptoms

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Introduction: Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by memory loss and cognitive decline. Oxidative stress is considered one of the main contributors to AD pathogenesis. Vitamin D3 (VitD3) and dimethyl fumarate (DMF) exhibit promising neuroprotective properties against oxidative stress and cognitive deterioration in AD. Their combined application may offer a synergistic approach to mitigating AD-related neurodegeneration and behavioral impairments.

Our study aimed to evaluate the effects of vitamin D3 and dimethyl fumarate treatment on cognitive function and markers of oxidative stress in a rat model mimic sporadic AD symptoms, induced by intracerebroventricular (ICV) injection of streptozotocin (STZ).

Methods: Male Wistar rats aged 3-4 months (n=50) were assigned to five groups: STZ (induced symptoms without treatment, n=10), VEH (vehicle control, n=10), VITD (STZ+VitD3, n=10), DMF (STZ+DMF, n=10), and COMBO (STZ+VitD+DMF, n=10). Treatments were administered orally for 90 days. Spatial memory was assessed using the Morris Water Maze. Oxidative stress markers (GSH, GSSG, 8-isoprostanes, SH groups) were measured in the supernatant of the hippocampus. Vitamin D metabolites were analyzed in plasma with LC-MS/MS.

Results: STZ administration induced cognitive deficits and oxidative stress damage. Both VitD3 and DMF improved memory performance and attenuated oxidative stress. The combined treatment showed the most pronounced effects, including reduced lipid peroxidation, normalized GSSG/GSH ratio, and an increased concentration of 3-epi-25(OH)D3.

Conclusion: Our findings provide insight into the mechanisms underlying the neuroprotective combined action of VitD3 and DMF, as well as their potential application in the treatment of Alzheimer's disease.

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PC.22

The Effect of 5-Aminoimidazole-4-Carboxamide Ribonucleotide Treatment on Lifespan and Functional Tests in a Mouse Model of ALS

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Amyotrophic lateral sclerosis (ALS) is an incurable neurodegenerative disease. ALS progression is related to mitochondria dysfunction, which may trigger a cascade of molecular events leading to the degeneration and death of motor neurons and muscle atrophy. 5-Aminoimidazole-4-Carboxamide Ribonucleotide (AICAR) is a potential therapeutic agent due to its ability to induce mitochondrial biogenesis and enhance skeletal muscle strength. This study investigated the impact of AICAR treatment initiated after disease onset on lifespan and motor function in a mouse model of the human form of ALS disease.

SOD1-G93A mice (n=16) served as an ALS model, while wild-type SOD1 mice (n=4) served as controls. Half of the ALS mice received AICAR, while the other half received a placebo. Weight measurements, Rota-Rod testing, and grip-strength tests were systematically conducted. Gastrocnemius muscle samples were collected at terminal disease stages to assess atrophy.

AICAR significantly improved survival, increasing lifespan by 13% ($p=0.0110$). However, it did not affect body weight, grip strength, or muscle atrophy compared to the placebo group. AICAR influenced the time from symptom onset to Rota-Rod test failure (extending it from 12 to 21 days; $p=0.0192$) and death (extending it from 27 to 41 days; $p=0.0001$) in ALS compared to the placebo mice. ALS mice exhibited a significantly reduced body weight ($p=0.0002$), muscle mass ($p=0.0002$), and muscle atrophy index ($p=0.0012$) compared to control mice. AICAR appears to slow ALS progression without terminating it.

PC.23

Ferroptosis in MPAN: a key driver of neurodegeneration in NBIA or a misinterpretation of iron and redox homeostasis dysregulation?

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Mitochondrial membrane protein-associated neurodegeneration (MPAN) is one of 11 subtypes of neurodegeneration with brain iron accumulation (NBIA), a group of rare diseases. MPAN is caused by mutations in the *C19orf12* gene, and its pathomechanism still remains poorly understood. Although the exact function of C19orf12 remains unknown, accumulating evidence suggests its involvement in lipid metabolism and redox homeostasis. Both, iron overload and oxidative stress are known triggers of ferroptosis, a form of cell death characterized by iron-dependent lipid peroxidation. For this reason, ferroptosis could be considered as a key mechanism driving neurodegeneration in MPAN and associated with the pathogenesis of this disease. However, so far there is no strong experimental evidence supporting this hypothesis.

To verify this hypothesis, our study aimed to evaluate the relevance of ferroptosis in cells carrying *C19orf12* mutations. We analyzed ferroptosis-related markers in fibroblasts derived from 18 MPAN patients and 6 healthy donors. Reactive oxygen species (ROS) levels were measured using fluorescence-based probes. Lipid peroxidation was assessed *via* Western blot detection of (4-HNE)-modified proteins. Moreover, we analyzed the proteomic profile of key ferroptosis-related proteins, both pro- and anti-ferroptotic, and correlated their levels with clinical severity scores. Our results provide new insights into ferroptosis-related mechanisms in MPAN and contribute to a deeper understanding of the MPAN pathomechanism.

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PC.24

Time-Restricted Eating Modulates Kynurenine Pathway and Bone Metabolism Markers in Older Adults

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Background: Time-restricted eating (TRE) is a nutritional intervention with emerging potential to modulate oxidative stress-related metabolic pathways. The kynurenine pathway, closely linked to redox balance, and bone-derived hormones such as osteocalcin are promising biomarkers for assessing the effects of dietary interventions in ageing populations.

Objectives: To evaluate the impact of a 12-week TRE intervention on plasma kynurenic acid (KYNA) levels and osteocalcin concentrations in women aged 60+.

Methodology: Fifteen women aged 60+ participated in a 12-week TRE programme, limiting daily food intake to a 10-hour window. Plasma levels of kynurenic acid (KYNA) were quantified pre- and post-intervention using validated isotope dilution liquid chromatography–tandem mass spectrometry (ID-LC-MS/MS). Serum osteocalcin concentrations were measured using a chemiluminescence immunoassay (CLIA). Statistical paired t-tests and Pearson correlation analyses were conducted.

Results: TRE resulted in a statistically significant reduction in KYNA levels (6.29 ± 1.13 vs 5.83 ± 1.03, $\Delta=-0.46$, $p=0.001$). Osteocalcin levels showed an increasing trend post-intervention (22.37 ± 4.89 vs 23.79 ± 5.73, $\Delta=1.43$, $p=0.08$). Importantly, the change in KYNA levels was significantly correlated with post-intervention osteocalcin concentrations ($r=0.53$, $p=0.04$).

Conclusions: These preliminary results suggest that TRE may beneficially modulate kynurenine pathway metabolites and bone turnover markers in older women. Further studies with larger cohorts would confirm these findings and explore the underlying interactions.

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Keywords: time-restricted eating, kynurenine pathway, older adults, LC-MS/MS

PC.25

Mediterranean diet bioactives induce Nrf2 signaling and attenuate oxidative stress in steatotic HepG2 cells

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The Mediterranean diet (MedDiet) is widely regarded as one of the healthiest dietary patterns and has been shown to exert Antioxidant Capacity Antioxidant Capacity in metabolic disorders, including obesity and metabolic dysfunction-associated steatotic liver disease (MASLD). MedDiet is particularly rich in bioactive compounds, such as polyphenols, which possess well-documented antioxidant properties. Considering that oxidative stress has been shown to play a role in the pathogenesis and progression of MASLD, dietary strategies targeting excessive free radicals may offer promising therapeutic potential.
This study evaluated the protective effects of MedDiet-derived bioactive compounds against oxidative stress during steatosis and to investigate whether these effects are mediated via the NRF2 signaling pathway.
HepG2 cells were pretreated for 72 hours with either individual compounds or selected mixtures, followed by a 48-hour co-incubation with the compounds and a free fatty acid (FFA) mixture to induce steatosis. After treatment, we assessed key cellular parameters, with a particular focus on ROS production, NRF2 pathway activation, lipid accumulation and cellular antioxidant activity.
Our results demonstrate that mixtures of bioactive compounds exhibit enhanced efficacy in mitigating excessive free radical generation. The superior effectiveness of these combinations compared to individual constituents indicates potential synergistic interactions. These findings support the hypothesis that bioactives derived from the Mediterranean diet may exert protective effects under steatotic conditions.

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PC.26

Popular culinary spices and their antioxidant properties

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Research Background: Increased exposure to internal and external oxidative stressors in modern life highlights the importance of antioxidant-rich diets. Spices, traditionally used in many cultures, are known not only for their flavor-enhancing properties but also for their potential health benefits.
Objectives: The aim of this study was to compare the antioxidant activity of ethanolic extracts obtained from selected culinary spices.
Methodology: Ethanolic extracts (5%) from dried oregano, cumin, rosemary, allspice, and thyme were prepared using Soxhlet extraction with 96% ethanol for two hours. The chemical composition of each extract was analyzed by gas chromatography coupled to mass spectrometry (GC-MS). Antioxidant activity was assessed using the DPPH free radical scavenging assay, with absorbance measured on a Shimadzu UV-1280 spectrophotometer.
Results: The extracts contained key bioactive compounds such as thymol (oregano, thyme), carvone and limonene (cumin), eucalyptol (rosemary), and eugenol derivatives (allspice). Antioxidant activity (%RSA) was highest for allspice, followed by oregano, rosemary (both over 80% RSA), thyme and cumin at the end.
Conclusion: Most tested spices thanks to polyphenols content exhibited strong antioxidant activity, particularly allspice, oregano, and rosemary. Herbs not only give a unique taste and aroma to dishes, but are rich in biologically active compounds that have a beneficial effect on the human body. These findings support the regular inclusion of diverse spices in the diet, both to maintain culinary traditions and to enhance the intake of natural antioxidants, which are essential for maintaining oxidative balance and overall health.

Key words: antioxidants, free radicals, DPPH, spices, GC-MS

PC.27

Coenzyme Q10 as an Ergogenic Aid for Long-Distance Runners – Is it a Breakthrough or a Myth? A Pilot Study

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Coenzyme Q10 (CoQ10) is a mitochondria-rich compound involved in energy resynthesis in the electron transport chain. Adequate levels of CoQ10 are crucial for physical performance during exercise, when energy demand increases. Recent findings have revealed that supplementation with CoQ10, up to a dose of 300 mg per day, enhanced anaerobic capacity, though not aerobic capacity. We, therefore, hypothesised that a higher dose of the supplement is required to improve aerobic capacity in long-distance runners. Six amateur-trained male long-distance runners (age: 41.5 ± 3.6 years; VO_2peak: 54.5 ± 4.0 $ml \cdot kg^{-1} \cdot min^{-1}$; 10 km PB: 39:48 ± 3:14) participated in a randomized, double-blind, placebo-controlled, crossover trial. Participants received either 800 mg/day of CoQ10 (ubiquinone) or a colour-matched maltodextrin as a placebo for four weeks, after which a crossover followed a four-week washout period. At each visit, blood was collected, and VO2peak and running economy (RE) were assessed using a running test until volitional exhaustion on a motorised treadmill with simultaneous gas analysis. Plasma CoQ10 levels were analysed using an HPLC-MS/MS method. CoQ10 supplementation significantly increased plasma CoQ10 levels ($p < 0.001$), but no significant differences were found between conditions for %ΔVO2peak ($p = 0.455$) or %ΔRE ($p = 0.147$). Four weeks of CoQ10 supplementation increased plasma levels but did not improve the aerobic capacity of long-distance runners. The present results did not confirm our hypothesis but are consistent with previous studies that questioned the ergogenic properties of CoQ10.

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PC.28

How Aerobic and Resistance Acute Interval Exercise Shapes Exerkine Profiles in Older Adults

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Background: High-Intensity Interval Exercise (HIE) improves physical and cognitive func-tions in young individuals, yet its molecular effects in older adults remain underexplored. Exercise-induced signaling molecules—exerkines—are known to mediate neuroplasticity and systemic adaptation, potentially contributing to cognitive health in aging. This study investigates the acute impact of different HIE modalities on circulating exerkines in elderly individuals.

Aim: To assess how a single session of aerobic (HIE) and resistance-based (HICE) high-intensity interval exercise influences exerkine concentrations among older adults.

Methods: Fifty-two older adults (mean age: 69.3 ± 3.4 years; 11 men, 41 women) were ran-domly assigned to either a HIE or HICE group. The HIE protocol consisted of eight 60-second cycling intervals at 80–90% HRmax with 30-second passive rests. HICE involved eight bodyweight exercises with 30-second rest intervals. Venous blood samples were col-lected immediately before and after exercise to measure concentrations of cortisol, copeptin, BDNF, cathepsin B, irisin, klotho, clusterin, and Lac-Phe.

Results: HIE significantly increased cathepsin B, klotho, and Lac-Phe. HICE led to eleva-tions in copeptin, BDNF, cathepsin B, klotho, and Lac-Phe. Notably, cathepsin B, klotho, and Lac-Phe were elevated across both exercise modalities.

Conclusion: Acute aerobic and resistance-based HIE elicit distinct but overlapping exerkine responses in older adults. These molecular changes may underlie the neuroprotective and metabolic benefits of interval training in aging populations.

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PC.29

The Neuroprotective Potential of Coenzyme Q10 via the Kynurenine Pathway in Long-Distance Runners: A Pilot Study

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Tryptophan is primarily degraded by the kynurenine pathway (KP), which produces several neuroactive metabolites. These include the neuroprotective picolinic acid (PA), the neurotoxic quinolinic acid (QA), and the homeostasis-dependent 3-hydroxyanthranilic acid (3-HAA).

The appropriate balance of these metabolites remains unexplored, particularly under conditions of increased metabolic stress, such as those experienced by endurance athletes. However, the impact of metabolically active compounds with redox-regulating properties, such as coenzyme Q10 (CoQ10), on the KP is poorly understood. This study aimed to assess the effects of CoQ10 supplementation on selected KP metabolites in amateur long-distance runners. Eleven male (age: 41.5 ± 3.6 years old; body mass: 78.5 ± 10.6 kg; VO2peak: 52.5 ± 6.7 ml·kg^{-1}·min^{-1}) amateur long-distance runners took part in a randomized, double-blind, placebo-controlled, crossover trial. Each participant received either 800 mg/day of CoQ10 (ubiquinone) or a colour-matched maltodextrin as a placebo for four weeks, followed by a four-week washout period and subsequently a crossover. Blood samples were collected before and after each intervention and analysed using the HPLC-MS/MS method. CoQ10 levels increased significantly after CoQ10 supplementation (post hoc $p < 0.001$).

A statistically significant increase in ΔPA levels ($p = 0.034$) was observed between experimental and control conditions, with no significant change in Δ3-HAA ($p = 0.954$) or ΔQA ($p = 0.373$) levels. This study revealed, for the first time, that CoQ10 supplementation may modulate the KP. These findings suggest that CoQ10 potentially promotes neuroprotection.

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PC.30

Changes in blood prooxidant-antioxidant balance after marathon in middle-age men

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Research background:
Prolonged and intense physical exercise induces metabolic stress and enhances the generation of reactive oxygen species (ROS) as a consequence of inflammation triggered by microinjuries in both skeletal muscles and the myocardium.
Objectives: The aim of our study was to determine the effect of a long-distance run on the levels of selected biomarkers related to disturbances in prooxidant-antioxidant balance and to damage of skeletal muscles and the myocardium in middle-aged male runners.
Methodology: The study included 16 marathon runners who completed the marathon distance during the Silesia Marathon held on October 6, 2024. Participants underwent basic anthropometric measurements before and after the run. Venous blood samples were collected from the cubital vein at three time points: at rest, immediately after the run, and 24 hours post-run. The collected biological material was analyzed for biochemical markers reflecting the prooxidant-antioxidant balance (SOD, CAT, GPx, GSH, UA, MDA, PerOx, TAS) as well as indicators of skeletal muscle (CK, LDH) and myocardium injury (CK-MB, cTnI, TNNT2, BNP).
Results: Based on the obtained results, no statistically significant differences were observed in the blood prooxidant-antioxidant balance. However, a statistically significant increase in UA concentration was noted at 24 hours post run ($p<0.01$) compared to rest and post run ($p<0.01$). A statistically significant increase in LDH activity was also observed after exercise ($p<0.001$) and at 24 hours ($p<0.01$) compared to rest. CK levels were significantly higher after the run ($p<0.001$) and at 24 hours ($p<0.001$) compared to baseline. The results showed no significant changes in CK-MB, cTnI, TNNT2, BNP.
Conclusions: Despite the fact that the physical exercise associated with the ultramarathon did not disturb the blood prooxidant-antioxidant balance nor significantly elevate cardiac stress biomarkers in the blood of middle-aged men, it nevertheless resulted in substantial microdamage to muscle fibers.

PC.31

Dynapenia and redox balance in geriatric patients- does sex play a role?

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RESEARCH BACKGROUND Dynapenia is an age-associated decline in muscle strength that leads to bad outcomes, decreased functional capacity and increased mortality.
OBJECTIVES The study aimed to measure total oxidative status (TOS), total antioxidative status (TAS) and evaluate the extent of oxidative stress in the serum of older patients with low muscle strength, exploring the role of oxidative stress in dynapenia, dependent on sex.
METHODOLOGY The study was performed in geriatric ward patients over 60 years of age, who were able to take part in the functional assessment. Dynapenia was diagnosed if grip strength was <27kg in men, and <16 kg in women. TOS and TAS were assayed in the serum. The severity of oxidative stress was expressed as oxidative stress index (OSI).
RESULTS 134 geriatric ward patients (73.9% of women, mean age 79.1 ± 7.3 years) took part in the study. Dynapenia was observed in 37.3% of cases, with similar prevalence in women (35.4%) and men (42.9%). The analysis revealed that redox imbalance correlated with dynapenia, but only in women. However, the logistic regression analysis identified OSI as a significant determinant of dynapenia increasing its probability by 60% (odds ratio=1.6; 95% CI, 1.11 - 2.31), while sex was found to be a non-significant factor.
CONCLUSIONS The study results have confirmed that oxidative stress can be a risk factor for dynapenia. Assessment of redox status using OSI may have clinical value in evaluating the probability of dynapenia independently of sex.

PC.32

Irisin, BDNF and redox balance in geriatric dynapenia

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RESEARCH BACKGROUND Irisin and brain-derived neurotrophic factor (BDNF) are two substances regarded as potential biomarkers for sarcopenia. However, the mutual interaction between them is unclear.
OBJECTIVES The study aimed to assess the serum concentration of irisin and BDNF in older patients and their correlation with markers of sarcopenia and redox homeostasis parameters.
METHODOLOGY Patients over 60 years old in the geriatrics ward who were able to participate in a functional assessment were included. Dynapenia was diagnosed if the time of the 5 Times Sit-to-Stand Test was greater than 15 seconds. Irisin, BDNF, and redox homeostasis parameters (total oxidative status, TOS; total antioxidative status, TAS) were assayed in the serum. The ratio of irisin to BDNF was mathematically calculated. The severity of oxidative stress was expressed as the oxidative stress index (OSI).
RESULTS 110 geriatric ward patients (72.7% women, mean age 78.2 ± 7.1 years) participated in the study. BDNF concentration was significantly lower, while the irisin/BDNF ratio and OSI were substantially higher in patients with dynapenia. No correlation was observed with irisin. BDNF correlated negatively with irisin and irisin/BDNF ratio. TOS, TAS, and OSI correlated negatively with BDNF and positively with irisin. 5TSST results correlated negatively with BDNF and positively with the irisin/BDNF ratio, TAS, and OSI.
CONCLUSIONS A lower concentration of BDNF and a higher irisin/BDNF ratio in patients
with dynapenia may indicate lower BDNF expression in aging muscles, despite adequate
irisin concentration. Higher levels of oxidative stress parameters can also play a role in sarcopenia development.

PC.33

Novel *in vitro* model of C19orf12 deficiency – a promising tool to study pathogenesis of NBIA-MPAN

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Neurodegeneration with Brain Iron Accumulation (NBIA) encompasses a group of rare genetic disorders characterized by progressive neurological decline and abnormal iron accumulation in specific regions of the brain. Key recognized NBIA subtypes include pantothenate kinase-associated neurodegeneration (PKAN), PLA2G6-associated neurodegeneration (PLAN), beta-propeller protein-associated neurodegeneration (BPAN), and mitochondrial membrane protein-associated neurodegeneration (MPAN). This study focuses on MPAN, a form linked to pathogenic variants in the *C19orf12* gene, which is one of the most prevalent NBIA subtypes diagnosed within the Polish population.

Analysis of fibroblasts from MPAN patients revealed alterations in cellular and mitochondrial metabolism, including a reduced proliferation rate, decreased mitochondrial oxygen consumption rate, and increased ROS levels. Recognizing patient heterogeneity (even among those with the same mutation) and varying disease severity influencing the measured parameters in patients' fibroblasts, we generated an HEK293-T cell line lacking C19orf12 protein. This *in vitro* model allows for further investigation of MPAN-related dysfunctions, for instance mitochondrial respiratory chain activity and its potential link to altered redox homeostasis.

C19orf12 protein-deficient cells represent a promising model that mirrors defects observed in patient-derived fibroblasts, thereby offering a valuable tool for studying the molecular mechanisms underlying MPAN pathology.

The study is co-financed from the state budget from the Education and Science Ministry program entitled "Science for Society". Project number NdS/537386/2021/2022, the amount of co-financing 1 900 000 PLN, total value of the project 1 900 000 PLN. Poland

III Session: Poster presentation

P.01

Metabolomic profiles of dietary modifications during intermittent fasting in dyslipidemia across both genders.

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Background: Amino acids and their derivatives play an integral role in the synthesis of structural and regulatory elements in organisms. Pathologies such as dyslipidemia may alter the blood pattern of these compounds. Improving endothelial function and modulation of metabolism by intermittent fasting (IF) and a diet enriched with nuts may favor the regulation of blood flow, improve physical performance and function of organs, and delay the progression of dyslipidemia and atherosclerosis.
Objective: This study aimed to evaluate changes in plasma concentrations of amino acid-related metabolites in IF and a diet enriched with nuts in a search for potential biomarkers and mechanisms of the disease.
Methods: Male and female dyslipidemia ApoE-/-LDLR-/- mice were treated with the diet enriched with macadamia and pecan nuts under ad libitum feeding conditions or under conditions of alternating fasting (24 hours) and feeding (24 hours) for 12 weeks. Metabolic changes were evaluated in the serum through targeted liquid chromatography–mass spectrometry (LC-MS)-based metabolomics and subsequent statistical analysis.
Results: Targeted metabolomic profiling revealed distinct alterations in amino acid metabolite patterns. A total of 22 of 40 compounds showed statistically significant differences in concentrations between IF and control groups (e.g., 1-methyl histidine – 111.90% increase, creatinine – 8.27% increase, SDMA – 6.48% increase). A decrease of 34.03% in the concentration of L-NMMA was statistically significant for the addition of nuts in the diet. These findings highlight the importance of dietary choices in modulating biochemical pathways that could affect cardiovascular health. Further research is needed to elucidate the specific mechanisms by which these dietary modifications influence redox balance and overall cardiovascular function.

P.02

Mood improvement is acompanied with altered tryptophan metabolism when endurance exercise is paired with time-restricted eating in healthy adults.

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The combined effects of time-restricted eating (TRE) and endurance training on tryptophan (TRP) metabolism and psychological well-being in healthy individuals have not been comprehensively investigated. This study aimed to explore whether alterations in anxiety levels following such an intervention are associated with changes in serum kynurenine metabolites.
A total of 71 participants completed a 12-week endurance training program while adhering to a 10-hour daily TRE window. Participants were divided into two groups: older adults (OE-TRE) and younger individuals (YE-TRE). Assessments conducted at baseline and post-intervention included body composition, inflammatory markers, serum kynurenines, and psychological indices (State-Trait Anxiety Inventory, Mood Adjective Checklist).
Significant reductions in body weight and BMI were observed in both groups. In the OE-TRE group, anthranilic acid (AA) levels increased, whereas a decrease was noted in the YE-TRE group. Overall, 67% of participants experienced a reduction in anxiety, which was associated with lower levels of 3-hydroxykynurenine (3-HK). The intervention also increased the xanthurenic acid (XANA) + picolinic acid (PA)/3-HK ratio in the OE-TRE group and decreased TNF-alpha levels. Baseline differences between groups in the 3-HAA/3-HK and XANA+PA/3-HK ratios were no longer evident after the intervention. Moreover, an increase in 3-HAA was accompanied by a reduction in GP130 protein levels.
These findings suggest that the combination of endurance exercise with TRE leads to beneficial changes in body composition, psychological state, systemic inflammation, and TRP metabolism. In particular, shifts in 3-HK concentration appear to play a pivotal role in mediating the anxiolytic and health-promoting effects of the intervention.

P.03

Effect of Physical Prehabilitation on Mitochondrial Function and Oxidative Stress in Rat Kidneys Following Hepatic Ischemia-Reperfusion Injury

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Background: Hepatic ischemia-reperfusion (HI/R) injury is a common adverse effect of liver surgery that can lead to kidney injury due to hypoperfusion, increased reactive oxygen species production, and systemic inflammation, contributing to progressive renal failure. Considering the beneficial effects of exercise on mitochondrial function, oxidative stress, and inflammation, we examined the effect of aerobic exercise prehabilitation on renal mitochondrial function and oxidative stress following HI/R.

Objective: Investigate the effect of different aerobic exercise intensities on renal mitochondria biogenesis and oxidative stress markers following HI/R injury.

Methods: Twenty-five male Wistar rats were randomly divided into five groups (n=5 per group): sedentary control, voluntary physical activity, and three aerobic exercise groups subjected to light (60% VO_{2max}), moderate (75% VO_{2max}), or high (90% VO_{2max}) intensity exercise. Exercise protocols were implemented on a treadmill 5 d/week over 12 weeks. Intensity was determined after a maximal incremental test with indirect calorimetry (Columbus Instruments). Following the intervention, animals underwent 45 minutes of partial hepatic ischemia and 24 hours of reperfusion, after which they were euthanized and kidneys collected. Markers of mitochondrial biogenesis, dynamics, and oxidative stress were analyzed in kidney lysates. Protein levels were determined via Western blot and dot blot.

Results: Exercise prehabilitation of varying intensities reduced the expression of mitochondrial fusion markers MFN1 ($p = 0.0355$) and MFN2 ($p = 0.0048$) compared to sedentary controls. There was no effect of exercise on markers of mitochondrial biogenesis or oxidative stress markers such as 3-nitrotyrosine, 4-hydroxynonenal, and carbonyl proteins.

Conclusion: Results indicate that prehabilitation aerobic exercise reduced kidney mitochondrial fusion markers after HI/R while having no significant effect on oxidative stress markers.

Research was funded by the Research Travel Grants Program (RTG), Initiative of Excellence – Research University (IDUB), Ministry of Science and Higher Education

P.04

Fat Under the Radar: Fat Intake and Relative Energy Deficiency in Sport (RED-S) Risk in Top-Performing Female Amateur Triathletes

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Background/Objectives: Triathlon combines swimming, cycling, and running into one continuous endurance event. The sport's high energy cost—both in training and racing—makes meeting triathletes' nutritional needs particularly challenging. When daily energy intake is insufficient, triathletes enter a state of low energy availability (LEA). Prolonged LEA can progress to Relative Energy Deficiency in Sport (RED-S), a syndrome for which female athletes are especially prone because of sex-specific hormonal and physiological factors (Mountjoy, 2024).

Fat is the most energy-dense macronutrient, making it an attractive fuel for heavy training. Sports nutrition guidelines emphasize how much fat athletes should eat, but the fats they choose matter just as much. We therefore re-analyzed dietary fat intake in top-performing female amateur triathletes from our previous study (Langa, 2025), examining both quantity and quality in relation to RED-S risk.

Methods: Our sample comprised 20 top-performing female triathletes who regularly raced at quarter-, half-, full-, or double-Ironman distances. Their mean training load was 11 ± 3.8 h/week, and their training experience averaged 5.5 ± 2.5 years. Dietary intake and exercise volume were recorded in consecutive 3-day food and training diaries. Triathletes also completed the Low Energy Availability in Females Questionnaire (LEAF-Q), which stratified them into low-risk (L-LEA, n = 10) and high-risk (H-LEA, n = 10) groups for RED-S.

Results: Total fat intake did not differ between groups. Compared with the L-LEA group, triathletes in the H-LEA group consumed less saturated fatty acids (SFA: 26.8 ± 9.0 vs 34.0 ± 14.7 g/d) and more n-6 polyunsaturated fatty acids (n-6 PUFA: 12.5 ± 4.0 vs 9.0 ± 2.3 g/d). Mean daily intakes of the long-chain n-3 PUFA eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) were similarly low in both groups (EPA: 70 ± 130 mg/d, DHA: 220 ± 380 mg/d), well below current sport nutrition guidelines of 500–600 mg/d combined EPA + DHA to avoid deficiency and 1000–2000 mg/d during heavy training, inflammation, injury, or immobilization to protect lean tissue.

Conclusions: Suboptimal intake of n-3 PUFA may blunt their sport-specific benefits—greater endurance and cardiovascular efficiency during aerobic exercise, lower oxygen cost, enhanced immune support, sharper cognition, and faster recovery. When an insufficient n-3 PUFA intake is combined with a high n-6 : n-3 PUFA ratio, the metabolic and inflammatory burdens of LEA can worsen. Together, these findings show that nutrition plans for female triathletes must address not only the quantity but also the quality of dietary fat.

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P.05

Unveiling the secrets of the *exo-xis* region: a key player in Shiga toxin phage φ24B induction and host genes expression under oxidative stress condition

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Research background
Enterohaemorrhagic *Escherichia coli* (EHEC) strains, which harbor temperate lambdoid bacteriophages carrying Shiga toxin genes (Stx-phages), are responsible for severe infections, including bloody diarrhea, hemolytic uremic syndrome (HUS), and even death. One of the well-studied Stx-phages is $\varphi 24_B$, whose toxin production is triggered when the phage enters the lytic cycle under oxidative stress conditions. The genome of φ24B, like other lambdoid phages, contains the *exo-xis* region, the precise function of which remains unclear.
Objectives
This study aimed to investigate the impact of deleting the entire exo-xis region on phage development and host gene expression during oxidative stress induction.
Methodology
E.coli MG1655 strains lysogenic for phage φ24B and a derivative with of the *exo-xis* region deletion were treated with hydrogen peroxide to induce prophage activation. The effects on phage development and host gene expression were analyzed using microarray and metabolomics assays.
Results
The results revealed that the deletion of the *exo-xis* region delayed prophage induction. Furthermore, significant differences in host gene expression were observed, particularly in pathways related to arginine metabolism, protein biosynthesis, and iron transport. Deletion of the *exo-xis* region led to reduced expression of these genes.
Conclusions
The *exo-xis* region is crucial for the proper development of Stx phages under oxidative stress conditions. Our results suggest that gene products from *exo-xis* region modulate host gene expression, hightlighting its role in phage-host interaction and viral lifecycle regulation.

P.06

Antioxidant potential of lactic acid bacteria isolated from fermented beetroot products

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Oxidative stress, caused by an imbalance between reactive oxygen species (ROS) and antioxidant defenses, is implicated in the development of numerous chronic diseases, including cardiovascular disorders, neurodegenerative conditions, and cancer. Fermented vegetables, such as beetroot (*Beta vulgaris*), are gaining attention as functional foods due to their rich content of antioxidant compounds and beneficial microbiota.
This study aimed to evaluate the antioxidant potential of lactic acid bacteria (LAB) isolated from spontaneously fermented beetroot products. A total of 18 LAB strains were isolated and identified through 16S rRNA sequencing. Their radical scavenging capacity and reducing power were assessed through standard *in vitro* assays (DPPH, ABTS).
The results revealed substantial variability in antioxidant profiles among the strains. Differential performance in the ABTS and DPPH assays suggests the involvement of distinct antioxidant mechanisms. Several isolates (e.g., *Weissella cibaria* FBU24, *Lactiplantibacillus plantarum* FBU44, *L. plantarum* BK1, and *Enterococcus hirae* FBUW44) surpassed the reference strain in at least one assay, underscoring their potential functional relevance.
These findings suggest that LAB from fermented beetroot may serve as natural sources of antioxidants and could contribute to the development of functional foods with potential health-promoting effects. Further studies, including cell-based assays and *in vivo* models, are warranted to confirm and better understand the antioxidant mechanisms and biological relevance of these strains.

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P.07

Impact of Acute Sleep Deprivation Induced by Violent Video Game Exposure on Cognitive Function and Physiological Responses in College-Aged Males: A Pilot Study

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[*]These authors contributed equally to this work.

Background: Sleep is a fundamental biological process essential for cognitive restoration and physiological balance. Sleep deprivation (SD) is known to impair brain regions such as the prefrontal cortex, hippocampus, amygdala, and thalamus, leading to deficits in attention, memory consolidation, and executive functioning. In young adults, including university students, one of the growing contributors to insufficient sleep is the excessive use of video games, particularly those featuring violent content. The overstimulation caused by such games may amplify the adverse effects of SD on brain function and systemic physiology.
Objective: The present study investigated the immediate impact of an all-night session of violent video game play on selected cognitive and physiological parameters in a sample of young adults.
Methods: Sixteen male university students took part in an eight-hour nighttime gaming session involving violent video games. Cognitive assessments targeting executive function, attention, and memory were performed both before and after the session. Physiological indicators, including blood pressure (systolic and diastolic), heart rate, blood glucose, and lactate levels, were also recorded. Additionally, mood and emotional states were evaluated using validated psychological questionnaires.
Findings: The overnight gaming session resulted in elevated heart rate and notable changes in blood glucose concentration. Participants also demonstrated measurable declines in cognitive performance, particularly in tasks involving attention control and executive processing.
Conclusion: Extended nighttime exposure to violent video games can acutely disrupt cognitive performance and physiological homeostasis in young adults. These findings underscore the potential health risks associated with excessive late-night gaming and highlight the need for greater awareness and further longitudinal research into its long-term effects and possible interventions.

P.08

The Isoxazole Derivative of Usnic Acid Impairs Mitochondrial Function and Metabolism in Breast Cancer Cells

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Background: Usnic acid (UA), a lichen-derived secondary metabolite, exhibits anticancer properties but is limited by its cytotoxicity to normal cells at therapeutically effective concentrations. To enhance its selectivity and pharmacological potential, the isoxazole derivative ISOXUS has been synthesized. ISOXUS has been shown to induce paraptosis-like cell death in breast cancer models. This study investigates its differential metabolic effects in malignant and non-malignant breast cell lines to elucidate its selective mechanism of action.
Methods: MCF-7 breast cancer cells and HB2 non-tumorigenic breast epithelial cells were employed in the study. The cells were treated with the ISOXUS (3 μg/mL) for either 6 or 24 hours. Cellular ATP levels were quantified spectrophotometrically, while bioenergetic profiling was conducted using the Seahorse XFp extracellular flux analyzer. Mitochondrial functionality was further assessed through the BIOLOG Phenotype MicroArray system. The inhibitory effect of ISOXUS on ETC complex II were investigated using molecular docking and confirmed experimentally. Finally, the detection of ROS after ISOXUS treatment was assessed by fluorescence microscopy.
Results: ISOXUS markedly impaired the utilization of key metabolic substrates involved in the production of NADH and FADH□, leading to reduced mitochondrial electron flow and oxygen consumption rate (OCR) specifically in MCF-7 breast cancer cells, with minimal effects observed in HB2 normal epithelial cells. Molecular docking studies identified mitochondrial respiratory chain complex II as the primary target of ISOXUS, a finding that was subsequently validated through experimental assay. The disruption of electron transport in MCF-7 cells was associated with a significant increase in reactive oxygen species (ROS) production.
Conclusions: ISOXUS functions as a metabolic inhibitor by targeting mitochondrial complex II in breast cancer cells, leading to impaired ATP synthesis and elevated reactive oxygen species (ROS) production, ultimately resulting in decreased cell viability.

P.09

Application of stabilized dried blood spot (DBS) sampling for the simultaneous determination of retinol, α-tocopherol, and coenzyme Q10 in monitoring of antioxidant micronutrients

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Oxidative stress, driven by excessive production of free radicals and reactive oxygen species (ROS), is commonly involved in the pathogenesis of cancer, cardiovascular disease and atherosclerosis. Therefore, antioxidants are of increasing interest as molecules that can safely interact with free radicals and terminate the chain reactions before vital molecules are damaged. Endogenous antioxidant defenses, such as retinol (vitamin A), α-tocopherol (vitamin E), and coenzyme Q10 (CoQ10), are critical in neutralizing these free radicals and preventing cellular damage. Monitoring the status of these lipophilic antioxidants is therefore of great importance in both clinical practice and research focused on redox balance and oxidative stress-related conditions.

Traditionally, the assessment of antioxidant levels has relied on serum or plasma samples obtained through venous blood collection, which requires trained personnel and controlled logistics. As an alternative, dried blood spots (DBS) offer the advantages of minimally invasive, at-home sample collection and simplified transport. However, lipophilic antioxidants are highly sensitive to environmental degradation on DBS cards, particularly due to oxidation during drying and storage.

To overcome this challenge, we implemented a novel stabilized DBS cards (Lipid Saver™, Ahlstrom) designed specifically to protect lipophilic compounds from oxidative degradation. These cards are chemically pre-treated to create a microenvironment that reduces the impact of environmental oxidative stress on analytes. When combined with a validated one-step methanol extraction and LC-MS/MS isotope dilution method, Lipid Saver™ cards enable accurate and simultaneous quantification of retinol, α-tocopherol, and CoQ10 from a single DBS sample.

Comparative analyses were conducted on patients, with paired DBS and serum samples. The DBS method exhibited slightly higher variability compared to serum, yet remained within the acceptable ranges defined by clinical method validation guidelines. Passing-Bablok regression and Bland-Altman analysis were used to assess agreement. High correlation coefficients confirmed strong linear relationships between DBS and serum measurements. Moreover, Bland-Altman plots showed no proportional bias, indicating consistent agreement across the full concentration range. These results support the use of established serum reference intervals for DBS-based measurements.

By addressing a key limitation in antioxidants determination which is a sample degradation, our method offers a robust tool for exploring antioxidant defenses in the fight against oxidative damage. This innovative method offers a reliable, non-invasive solution for monitoring lipophilic antioxidants outside traditional clinical settings. It is particularly suited for large-scale epidemiological studies, personalized nutrition programs, and longitudinal research in free radical biology.

P.10

Short-term effects of a fish-based ketogenic diet on TMAO and metabolic hormones in healthy adults

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Background: Ketogenic diets (KD) are increasingly studied for their metabolic effects, yet little is known about their short-term impact on gut-derived metabolites such as trimethylamine N-oxide (TMAO) and associated biomarkers. This study evaluated how a short-term, fish-rich KD affects metabolic hormones and choline-related compounds in healthy adults.

Methods: Fourteen healthy participants followed a 14-day KD based on fish as the primary source of protein and fat. Energy intake was reduced by approximately 500 kcal/day from estimated requirements. Participants received individualized dietary plans, nutritional education, and continuous support from a registered dietitian. Blood samples were collected at baseline and after the intervention following a 12-hour overnight fast. Analyzed biomarkers included insulin, leptin, TMAO, betaine and choline.

Results: After the intervention, significant decreases were observed in insulin and leptin concentrations ($p < 0.05$). TMAO and betaine levels significantly increased. No significant changes were found in choline concentration.

Conclusion: A two-week, fish-based KD induced measurable metabolic changes, including increased TMAO and decreased insulin-related markers in healthy adults. These results highlight the potential influence of short-term ketogenic interventions on metabolic and gut-derived biomarkers. Further research is required to better understand the long-term effects and the influence of specific dietary fat and protein sources on the observed results.

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P.11

HIITing Inflammation, Boosting Cognition: A 12-Week Interval Training Intervention

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Background: Aging affects immune function, altering immunoglobulin and cytokine levels. Chronic low-grade inflammation is increasingly linked to cognitive decline. Circulating cytokines may cross the blood–brain barrier, triggering neuroinflammation that disrupts neuroendocrine and neurotransmitter systems. Elevated pro-inflammatory cytokines are associated with impairments in executive function, likely due to neurotoxic effects on the prefrontal cortex, while the hippocampus appears particularly susceptible to inflammationrelated neurodegeneration. Physical activity may counteract these effects by reducing proinflammatory and increasing anti-inflammatory cytokines, promoting neuroplasticity and protecting key brain regions.
Aim: This study aimed to assess the effects of aerobic and resistance interval training on cytokine profiles and cognitive performance in older adults.
Methods: Eighty-seven participants (mean age: 69.3 ± 3.4 years) were randomly assigned to Aerobic High-Intensity Interval Training (AHIIT), Resistance Circuit High-Intensity Interval Training (HICT) or a control group (CON). Over 12 weeks, participants trained three times per week. AHIIT included eight 60-second cycling bouts at 80–90% HRmax. HICT involved eight bodyweight exercises, each followed by 30 seconds of rest. Cognitive tests (Stroop, TMT A and B, VPT) and blood cytokine levels (via ELISA) were assessed pre- and post-intervention.
Results: HICT significantly reduced TMT-B time and proinflammatory cytokines (TNF, IL5, IL-2, IL-7, IL-1β). AHIIT improved Stroop performance. Both HICT and AHIIT enhanced short-term memory (VPT).
Conclusions: A 12-week interval training program improved executive function and memory.
Findings confirm the effectiveness of HICT and/or AHIIT as practical tools for supporting brain health among older adults.

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P.12

The Impact of Time-Restricted Eating on Body Composition and the Kynurenine Pathway in Women 60 +

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Research background: In women over the age of 60, obesity and depression are becoming increasingly prevalent problems due to hormonal changes and aging process. Research suggests that time-restricted eating (TRE) may help prevent and manage these conditions.
Objectives: The aim of the study was to demonstrate the impact of a 12-week intervention involving a 14-hour time-restricted eating window on body composition and the kynurenine pathway.
Methodology: The study included 15 women aged 65 ± 6.5 with overweight or obesity. Body composition was assessed using bioelectrical impedance analysis (BIA) before and after intervention. LC-MS/MS was used to examine the concentration of metabolites in the kynurenine pathway.
Results: After 12 weeks, changes in body composition were observed. Total body weight decreased (by 1.01 kg; $p<0.05$), BMI decreased (by □ 0.38 kg/m²; $p<0.05$), total fat mass decreased (by □ 1.20 kg; $p<0.05$), and visceral fat area was reduced (by □ 3.1 cm²; $p<0.05$). Parameters related to muscle mass, mineral content, and body hydration remained unchanged.
The results of kynurenine pathway metabolite concentrations showed a significant decrease in tryptophan levels (by 7643.856 nmol/L; $p<0,05$), 3-hydroxyanthranilic acid (by 13.14 nmol/L; $p<0.05$), and 3-hydroxykynurenine (by 2,028 nmol/L; $p<0.05$) as well as an increase in kynurenic acid (by 5.65 nmol/L; $p<0.05$), and quinolinic acid (by 76.66 nmol/L; $p<0.05$).
The results of the study show that a 12-week TRE intervention can be an effective method of preventing obesity because it reduces excess fat. TRE can also prevent depression by increasing the levels of metabolites associated with neuroprotective processes.

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Time efficient training with substantial effects: mitochondrial protein response to highintensity interval training

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Background
High-intensity interval training (HIIT) is known as a time-efficient method to improve
aerobic fitness. It is therefore worth investigating whether as little as two weeks (six sessions) of HIIT can impact physical capacity, including effects at the cellular level.
Objectives
This study aimed to examine the adaptive response to six sessions of HIIT, specifically
focusing on changes in the levels of mitochondrial proteins.
Methodology
Twenty students (age 21.2 ± 0.9) were assigned to either HIIT (n = 10) group or the control
(CON, n = 10) group. The HIIT group completed six training sessions over a 14-day period.
Each session consisted of a 5-minute warm-up and 6 × 90-second intervals performed at 80% of maximal aerobic power (MAP), with 180-second rest periods between exercise bouts. Before and after the training intervention, we conducted anthropometric measurements asl well as evaluations of aerobic and anaerobic capacity. In addition, we investigated the effects of HIIT on proteins involved in oxidative phosphorylation in skeletal muscle using proteomic analysis. A total of 89 subunits from the mitochondrial respiratory chain complexes and the ATP synthase complex were considered. Muscle biopsy samples were obtained from three representative participants from both the HIIT and CON groups before and after the intervention for proteomic analysis.
Results
Training induced an increase in aerobic capacity. The HIIT intervention caused an increase in proteins level involved in oxidative phosphorylation.
Conclusion
HIIT training can be a good strategy for improving exercise capacity in a short period of time.

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The effect of swim training on lifespan and tibialis anterior muscle mass in female mice with amyotrophic lateral sclerosis

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Background
Swim training applied to mice before the onset of disease symptoms prolonged the lifespan of mice with amyotrophic lateral sclerosis (ALS) (Flis et al., 2018). It also had a beneficial effect on muscle mass and strength, bioenergetics, and oxidative stress (Cieminski et al. 2021; Flis et al. 2019).
Objectives
This study aimed to investigate the effect of swim training initiated in ALS mice after the first symptoms appeared on their lifespan and the mass of the tibialis anterior (TA) muscle.
Methodology
We used transgenic hmSOD1 G93A (ALS model) female mice, divided into three groups: ONSET (with the first symptoms of disease, 108±5 days of age) and TERMINAL (untrained (UT) and trained (TT), n=6 per group). The training was conducted five times per week, beginning at the symptomatic stage and continuing until the mice were unable to continue training or died.
Results
Swim training extended the life of ALS mice by about 15%. Body weights of terminally ill mice were not significantly different on the day of death, but TA weights were lower in trained mice.
Conclusion
Swimming training applied to mice with the onset of disease increased the lifespan of female ALS mice, which was accompanied by a significant reduction in TA muscle mass at the time of death.

References

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Metabolomic Profiles of A549 Cells Exposed to Nicotine and Nicotine Benzoate

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Background: Nicotine and its protonated salt forms, such as nicotine benzoate, are key active components of electronic cigarette (e-cigarette) aerosols, widely used for their improved palatability and bioavailability. While the systemic pharmacological effects of nicotine are well documented, the cellular and metabolic impact of its salt formulations remains insufficiently understood. This knowledge gap is particularly relevant given chronic inhalation exposure and potential interactions with pulmonary epithelial cells, including malignant ones. Investigating these effects provides additional context for evaluating potential risks associated with nicotine salt exposure.
Objective: This study aimed to investigate the cytotoxic and metabolic effects of nicotine and nicotine benzoate on A549 human lung adenocarcinoma cells.
Methods: A549 cells were exposed to increasing concentrations (0.1 nM–1000 μM) of nicotine or nicotine benzoate for 24 hours. Cell viability was assessed using the MTT assay. Metabolic changes were evaluated through untargeted liquid chromatography–mass spectrometry (LC-MS)-based metabolomics.
Results: Nicotine exposure had no significant effect on cell viability. In contrast, nicotine benzoate markedly increased cell viability, with the strongest effect observed at the highest tested concentration. Metabolomic profiling revealed distinct alterations in metabolic pathways. Nicotine benzoate triggered pronounced changes in redox-associated metabolites, consistent with oxidative stress adaptation. Simultaneously, the accumulation of biosynthetic intermediates, points to enhanced anabolic activity and metabolic reprogramming. These shifts suggest the activation of redox-protective and proliferative pathways that may support cancer cell survival and growth.

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