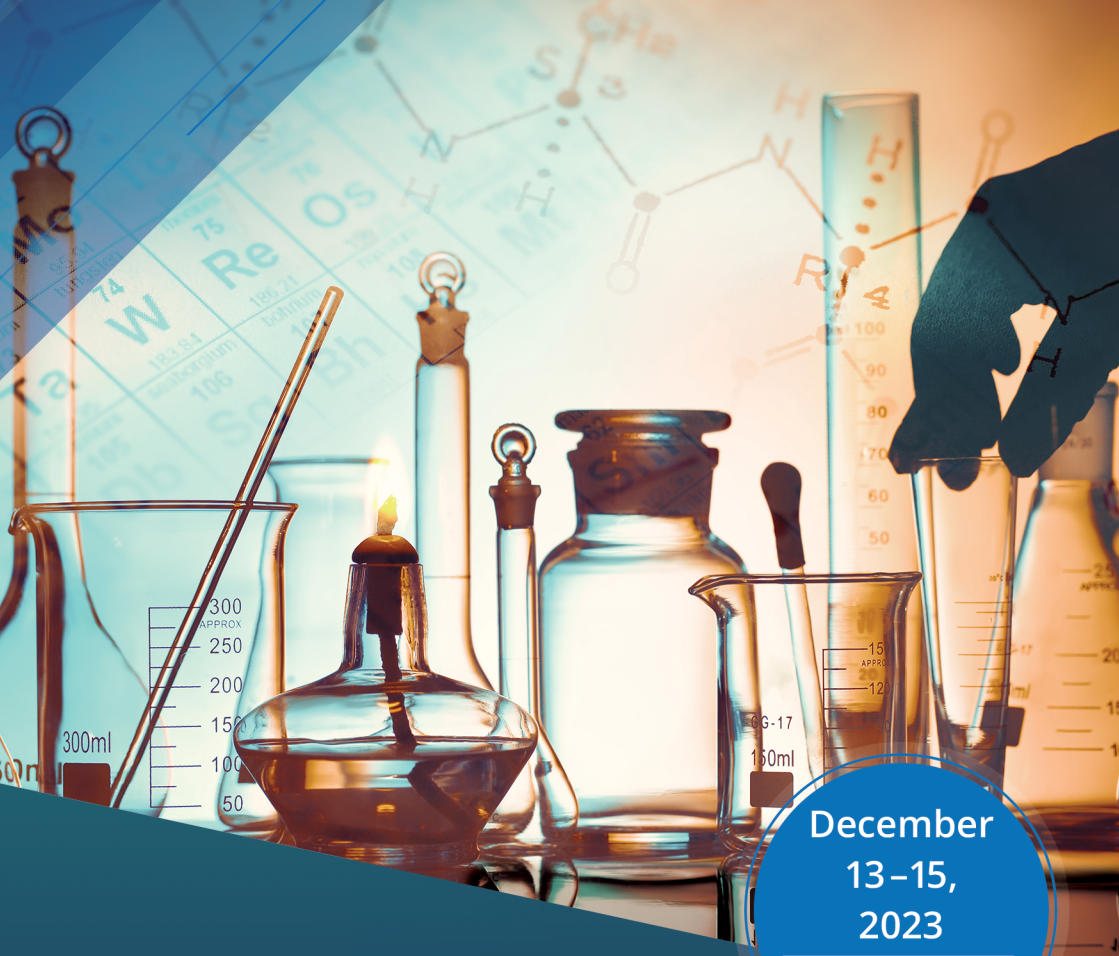

December 13–15, 2023

Szeged

XXI. MEETING OF NOBEL LAUREATES AND TALENTED STUDENTS

An event organized by the National Biomedical Foundation.

NEMZETI TUDÓSKÉPZŐ AKADÉMIA

NATIONAL ACADEMY OF SCIENTIST EDUCATION

10th anniversary of their Noble Prize

Randy W. **Schekman**

Nobel Prize in Physiology or Medicine, 2013

Thomas C. **Südhof**

Nobel Prize in Physiology or Medicine, 2013

Ole **Petersen**

Professor at Cardiff University

80th birthday

Miklós **Sahin-Tóth**

Professor at University of California

60th birthday

VENUES

ACCOMMODATION

1. **Novotel Szeged** *(1 Maros utca, 6721 Szeged)*
2. **Art Hotel Szeged** *(16 Somogyi utca, 6720 Szeged)*
3. **Dóm Hotel** *(6 Bajza utca, 6720 Szeged)*

VISITS TO THE LABORATORIES OF MENTORS

4. **Albert Szent-Györgyi Medical School, University of Szeged** *(Northern Clinical Garden)*
5. **Biological Research Centre** *(62 Temesvári körút, 6726 Szeged)*

PRESS CONFERENCE

6. **Szeged City Hall, Lajos Lechner Hall** *(10 Széchenyi tér, 6720 Szeged)*

OTHER EVENTS

7. **University of Szeged József Attila Study and Information Centre** *(10 Ady tér, 6722 Szeged)*
   - keynote presentations by Nobel Laureates and other distinguished guests
   - presentations by Szent-Györgyi Students
   - presentations by Szent-Györgyi Mentors
   - round-table sessions with the participation of Szent-Györgyi Pupils and Szent-Györgyi Students
   - a closed meeting between the guests of honor and Szent-Györgyi Students
   - round-table sessions with the participation of Szent-Györgyi Pupils and distinguished scientists
   - plenary sessions
   - gala event
   - gala dinner

CONFERENCE PROGRAM

DECEMBER 13, 2023 Wednesday

17.00–21.00 **ARRIVALS AND HOTEL CHECK-INS, REGISTRATION**
Novotel Szeged, Art Hotel Szeged, Dóm Hotel

19.00–21.00 **DINNER** (for hotel guests only)
Novotel Szeged, Art Hotel Szeged

DECEMBER 14, 2023 Thursday

07.00–07.45 **BREAKFAST** (for hotel guests only)

08.00–08.20 **ARRIVAL AT THE UNIVERSITY OF SZEGED JÓZSEF ATTILA STUDY AND INFORMATION CENTRE; CONFERENCE REGISTRATION**

08.30–09.30 **OPENING**
PRESENTATIONS BY SZENT-GYÖRGYI STUDENTS PART 1
Congress Hall
Chairs: Zsuzsanna Helyes and Zoltán Papp

Bálint Szeredás: *MicroRNAs in pancreatic neuroendocrine and adrenocortical tumours: relevance for differential diagnosis and treatment response*

Beáta Várkonyi: *The study of ADP-ribosylation protein modifications in tumour cells*

Inez Bosnyák: *Investigating the consequences of oxygen deficiency in the retina*

Benedek Szathmári: *Evolutionary and genetic aspects of fruiting body formation*

Dorina Debreczeni: *Examining the activity and expression of a background potassium channel with a unique structure*

Simultaneous event:

08.30–09.30 **Round-table sessions Part 1**
(based on a pre-assigned schedule)
With the participation of Szent-Györgyi Pupils and Szent-Györgyi Students
Lecture Halls 1 and 2, Basement

09.30–10.10 **COFFEE BREAK**
Exhibition Space, ground floor & Foyer, 1st floor

10.10–11.10 **PRESENTATIONS BY SZENT-GYÖRGYI STUDENTS PART 2**
Congress Hall
Chairs: Ádám Dénes and Attila Mócsai

Simon Vikár: *Modelling a human autoimmune skin disease under laboratory conditions*

Dorina Kovács: *Can we prevent antibiotic resistance?*

Levente Gulácsi: *Investigation of experimental sepsis*

Sára Vida: *Loss of microglial P2Y12 receptor function alters microglial morphology and contactomics*

Tibor Donka: *Deeper insight into trauma induced coagulopathy*

Simultaneous event:

10.10–11.10 **Round-table sessions Part 2**
(based on a pre-assigned schedule)
With the participation of Szent-Györgyi Pupils and Szent-Györgyi Students
Lecture Halls 1 and 2, Basement

11.10–11.30 **BREAK**

11.30–12.30 **PRESENTATIONS BY SZENT-GYÖRGYI MENTORS**
Congress Hall
Chairs: Mária Deli and Zoltán Rakonczay

Karolina Pircs: *Direct neuronal reprogramming: new possibility to study late onset neurodegenerative diseases*

Bálint Kintses: *The enemy of my enemy is my friend: viruses against antibiotic-resistant superbugs*

Anikó Borbás: *Bicyclic and tricyclic morpholinos: destruction and reconstruction of the furanose ring of nucleosides*

Dóra Reglődi: *Travelling around a peptide*

Simultaneous event:

11.00–11.45 **Honorary guests visit University of Szeged Teacher Training Secondary and Primary School**
Classroom observation of a laboratory practice

12.00–12.45 **Honorary guests visit Radnóti Miklós Experimental Grammar School**
Classroom observation of a laboratory practice

12.30–14.00 **LUNCH**
Atrium hall and exhibition space, ground floor

14.30–17.00 **PLENARY SESSION, CELEBRATING EXCELLENCE**
Congress Hall
Chairs: András Varró and Péter Hegyi

Péter Hegyi: *The National Academy of Scientist Education Program*

Zoltán Rakonczay: *The University Education Program of the National Academy of Scientist Education*

Miklós Sahin-Tóth: *My 25-year quest to decode hereditary pancreatitis*

Randy Schekman: *Genes, cells, and discovery in basic science and disease*

17.00–17.45 **COFFEE BREAK**
Exhibition Space, ground floor & Foyer, 1st floor

17.45–19.30 **PLENARY SESSION, CELEBRATING EXCELLENCE**
Congress Hall
Chairs: András Varró and Péter Hegyi

'Szent-Györgyi Student of Excellence, 2023' - *an award ceremony followed by a presentation by the awardee*

Gábor Orbán: *Sci-fi in cardiology: How we treat cardiac arrhythmias*

Ole Petersen: *The scientific process: how do we get it right?*

Thomas Südhof: *Deconstructing synapse formation, one molecule at a time*

19.45–20.30 **GALA EVENT**
Atrium hall

20.30–22.00 **GALA DINNER**
Atrium hall

DECEMBER 15, 2023 Friday

08.00–09.00 **BREAKFAST**
(for hotel guests only)

08.30–09.30 **A CLOSED MEETING BETWEEN THE GUESTS OF HONOR AND SZENT-GYÖRGYI STUDENTS**
University of Szeged József Attila Study and Information Centre, Grand Lecture Hall

09.30–09.45 **SZENT-GYÖRGYI PUPILS ARRIVE AT THE UNIVERSITY OF SZEGED JÓZSEF ATTILA STUDY AND INFORMATION CENTRE**

09.45–11.00 **MEET THE EXCELLENCE**
ROUND-TABLE SESSIONS WITH THE PARTICIPATION OF SZENT-GYÖRGYI PUPILS AND DISTINGUISHED SCIENTISTS
Atrium hall

Simultaneous event:

09.45–11.15 **Pedagogy session for Szent-Györgyi Teachers and Senior Teachers**
Grand lecture hall
Adrien Lengyel: *Welcome speech*
Sándor Bán: *How to use Artificial Intelligence in Biology teaching*

11.30–12.30 **LUNCH**
Atrium hall

12.30–15.00 **VISITS TO THE LABORATORIES OF SZENT-GYÖRGYI MENTORS**
(on the basis of a pre-assigned schedule)

14.30–15.30 **PRESS CONFERENCE**
(registration required)
Szeged City Hall, Lajos Lechner Hall

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LECTURE ABSTRACTS

In the sequence of performance

BÁLINT SZEREDÁS

MicroRNAs in pancreatic neuroendocrine and adrenocortical tumors: their relevance for differential diagnosis and treatment response

Knowing the grade of pancreatic neuroendocrine tumors (PNETs) is essential for determining the treatment. Currently, its diagnosis is only possible by the examination of biopsy samples, which is a time-consuming method and requires an experienced pathologist. MicroRNAs are short, non-coding RNAs that play a role in the posttranscriptional regulation of gene expression. Their expression is tissue and tumor-specific and may be characteristic of the grade. Since they are stable RNAs, and cells are actively secreting their microRNAs into their environment, they can be detected in archived tissue blocks and peripheral blood samples as well. In this research, we first examined the expression of tissue microRNAs already described to be relevant in PNET to uncover potential differences between G1 and G2 tumors. We have found significant under-expression of the following microRNAs in G2 compared to G1 PNETs: hsa-miR-130b-3p and hsa-miR-194-5p. MicroRNA expression profiling is also underway. Circulating counterparts of tissue microRNAs could later be examined as potential minimally invasive markers that could be of great clinical relevance.

The pharmacological treatment of adrenocortical cancer represents a major problem, as only mitotane is available as a specific drug treatment. The hormone progesterone was shown to have some antitumor activity in adrenocortical cell lines, and in collaboration with an Italian group, we have examined the expression of microRNAs in various adrenocortical cell lines treated by progesterone in vitro.

BEÁTA VÁRKONYI

Study of ADP-ribosylation protein modifications in tumour cells

ADP-ribosylation is a protein modification that regulates a range of cellular processes, such as gene expression, DNA repair and programmed cell death. ADP-ribosylation is mediated by ADP-ribosyltransferases (ARTs), which transfer one or more ADP-ribose (ADPr) units from nicotinamide adenine dinucleotide (NAD^+) to their targets. The cell nuclear ART PARP1 is recognized to perform the bulk of these modifications in the cell. ADPr-binding proteins are pivotal for the biological effects of the protein modification.

In our work, we attempted to use a fluorescently labelled ADPr-binding protein (eAf1521) to visualize ADP-ribosylation. Surprisingly, we could observe cytoplasmic reticular staining in all tumour cell lines examined, which we have colocalized with mitochondrial markers. We believe that the labelling corresponds to singular ADPr because it is not detectable by an anti-ADPr chain antibody. We have also found a correlation between mitochondrial ADP-ribosylation and metabolism through treatment with compounds acting on mitochondrial metabolic pathways. When cells were exposed to oxidative stress, we observed a loss of mitochondrial labelling. Targeted inactivation of the PARP1 gene did not affect mitochondrial labelling.

The correlation of ADP-ribosylation with NAD^+ metabolism suggests that the labelling may be influenced by the varying NAD^+ concentrations depending on the mitochondrial bioenergetics of tumour cells. The unknown ADP-ribosylated molecules identified by eAf1521 could potentially be regulators of mitochondrial activity. We are currently working to identify these molecules and the responsible ARTs.

INEZ BOSNYÁK

Investigating the consequences of oxygen deficiency in the retina

The retina has one of the highest metabolic activities and oxygen consumption, so insufficient blood supply leads to visual impairment. Retinal hypoxia has a key role in the development of age-related macular degeneration, glaucoma, diabetic retinopathy, among others. The occurrence of these diseases is increasing, however, no effective treatment without side-effects is available. Our aim was to investigate the retinal damage in time-dependent manner and to analyze the sensitivity of different cell types.

Retinal ischemia was induced by bilateral common carotid artery occlusion (BCCAO) for 10, 13, 15, or 20 minutes, or by right permanent unilateral common carotid artery occlusion (UCCAO) in mice. Optical coherence tomography (OCT) is a painless method that is widely used in clinical practice. OCT measurements were performed for following the changes in retinal thickness. We evaluated the number and distribution of ganglion cells. Vascular density was analyzed in the different groups and photoreceptors were studied too.

We observed significant changes in almost all retinal layers during the experimental period. The number of ganglion cells decreased in the peripheral region in the 20-minute BCCAO group, and in the central and peripheral regions in the UCCAO group 4 weeks after the intervention. The percentage of vascular density was higher in the 20-minute BCCAO and in the UCCAO groups. In addition, cone loss was also observed in these groups.

Our results suggest that the 20-minute BCCAO is a good model for investigating the consequences of ischemia and reperfusion in the retina in time-dependent manner, while the UCCAO causes more severe damage in a short time, so can be used for testing new drugs.

BENEDEK SZATHMÁRI

Genetic and Evolutionary Aspects of Fruiting Body Formation

Fruiting bodies are complex multicellular sexual reproductive structures of some fungi. During their evolution fungi 'invented' this complex character several times independently. Fruiting bodies reach their final form and size via a well-choreographed developmental programme, however, the evolutionary roots of this developmental programme, as well as the mechanisms of growth and differentiation, are unknown.

In the Fungal Genomics and Evolution Group we also investigate the evolution of fungal development. Based on gene expression data we formulated the following hypothesis: in Basidiomycota the fruiting body cell programmes derive from the basidial develpmental programme, thus fruiting body cells can evolutionarily be considered modified basidia. As the first step of testing this hypothesis I knocked out two RNA binding protein coding genes in our model organism (*Coprinopsis cinerea*) using the CRISPR-Cas9 system. The deletion mutants are deficient in fruiting. Additional experiments based on genetic manipulation (complementation, overexpression), as well as the transcriptome analysis of the mutants, are in progress.

Our results might contribute to the elucidation of the evolutionary and genetic background of fruiting, that can be important from the aspect of biotechnological applicability of this fungal group (e. g. biofuels, mycoremediation, sustainable food-supply, fungal leather alternatives).

DORINA DEBRECZENI

Examining the activity and expression of a background potassium channel with a unique structure

TRESK belongs to the two-pore domain (K2P) family of potassium channels, and it is expressed in some types of human neurons. TRESK has unusual structural properties: it has a much shorter intracellular C-terminal region (iCtr) than the other K2P channels. It is an unanswered question, whether the iCtr of TRESK has any role in determining the activity of the channel.

To find out the function of the iCtr of TRESK, we prepared new mutants: deleted a segment from the end of the sequence, or replaced some amino acids by hydrophobic ones. Our aim was to examine the effect of these mutations on the channel activity.

TRESK was expressed in African clawed frog oocytes and measured by two-electrode voltage clamp technique. Conventional electrophysiological measurements do not allow independent determination of the number and activity of channels. The channel activity can be measured by the newly developed epithelial Na+ current ratio method. The ratio of the potassium and the sodium (Na+ channel) currents is called the ENaR value, which is proportional to the channel activity.

A TRESK mutation (F372L) where phenylalanine is replaced by leucine was identified in a patient with migraine. It was a question if this point mutation has any effect on the channel activity. Both the deletion of the end of the iCtr and the F372L mutation reduced the basal activity. Replacing the original sequence with hydrophobic and positively charged amino acids results in low expression levels, but highly increases TRESK activity compared to the wild type.

In accordance with the conventional measurements and based on the ENaR data, the results support the conclusion that the iCtr of TRESK is an important determinant of the channel expression and activity.

SIMON VIKÁR

Modelling a human autoimmune skin disease under laboratory conditions

Introduction: Bullous pemphigoid (BP) is a rare autoimmune disease associated with blister formation in the skin. It is known that autoantibodies against skin proteins have an important pathogenic role. However, the cellular and molecular mechanisms of the disease development are currently only partially understood and, no targeted therapy is available for the treatment.

Aim: In our work, we aimed to establish a fully human skin separation model to investigate the mechanisms of the blister formation in bullous pemphigoid.

Methods: To set up the skin separation model, frozen sections of healthy human skin were first treated with blood serum from bullous pemphigoid patients. To detect the antibody deposition from the serum, immunofluorescent staining was performed. In the model, tissues treated with patient serum were then coincubated with freshly isolated neutrophils from the blood of healthy volunteers, in the present of fresh blood plasma. Finally, tissue damage was assessed by light microscopy after haematoxylin-eosin staining.

Results: Firstly, we managed to detect the antibody-deposition in the bullous pemphigoid serum treated skin tissues. Subsequently, we successfully set up the skin separation model in our lab. The neutrophils attached to the bullous pemphigoid serum treated skin tissues and caused skin layer separation, which lesions are consistent with the characteristic blisters of the disease.

Conclusion: We successfully established a fully human skin separation model that allows the study of the blister development in bullous pemphigoid.

DORINA KOVÁCS

Can we prevent antibiotic resistance?

Several mechanisms are known, which are responsible for antibiotic resistance, thereby for the evolution and spread of multi-resistant bacterial species, challenging healthcare even more day by day. The development of antibiotic molecules with novel mechanisms of action and with the ability to bypass resistance, seemed to be resultless in the last few decades, which suggests finding new ways to defeat resistance. In my project, I am testing the presence of resistance against beta-lactam antibiotic and beta-lactamase inhibitor combinations (BL-BLI combinations) currently being under development. I conducted the experiments on Klebsiella pneumoniae strains, which is - according to the World Health Organization (WHO) - one of the most resistant bacterial species. I tested nearly 70 genetically diverse strain, against 7 combinations, in a form of modified MIC (minimum inhibitory concentration) measurements. Evaluating the results, we are going to be able to define, against which BL-BLI combinations are already antibiotic resistance genes present in the environment, before they are even used in healthcare. Knowing these genes might be particularly relevant in preventing resistance, they could even contribute to the success of narrow-spectrum therapies. Using our data, we will also predict the future efficacy of these beta-lactam antibiotic and beta-lactamase inhibitor combinations.

LEVENTE GULÁCSI

Investigation of experimental sepsis

Sepsis is a dysregulated host response to infection, which can lead to life-threatening organ failure. Due to the complexity and diverse clinical manifestation, sepsis is one of the most challenging pathologies in medicine. Under septic circumstances multi-organ failure can develop, affecting the central nervous and respiratory systems, circulation, excretion, coagulation and liver function.

In clinical practice, the Sequential Organ Failure Assessment (SOFA) scoring system is used for the quick evaluation the condition of the patient and for monitoring the effectiveness of the therapies.

Among the organ-damaging pathological processes, microcirculatory impairment is prominent, whereas it can lead to an imbalance between oxygen consumption and delivery, and a cellular mitochondrial respiratory failure. In addition, as part of the cellular immune response, neutrophil granulocytes are activated, in which specific cell death, NETosis occurs.

Current therapeutic practice includes organ support treatments such as fluid resuscitation, circulatory and respiratory support, and antibiotic administration. However, there are no approved clinical therapeutic tool to mitigate microcirculatory and mitochondrial dysfunction.

According to international recommendations, our institute has developed a rapidly progressive, reproducible rat sepsis model, in which we investigate organ failure by adapting the human SOFA scoring system, microcirculation by in vivo imaging, mitochondrial respiration by measuring cellular oxygen consumption, and NETosis by fluorescence microscopy. Our aims were to increase the clinical translational potential of experimental sepsis studies and exploring new therapeutic options.

SÁRA VIDA

Loss of microglial P2Y12 receptor function alters microglial morphology and contactomics

Microglia are the main immune cells in the central nervous system that contribute to physiological and pathological brain states. Our laboratory has identified specialised contacts between microglial processes and both neuronal cell bodies and blood vessels through which microglia can shape neuronal activity and cerebral blood flow. The P2Y12 purinergic receptor (P2Y12R) is microglia-specific in the brain and plays a vital role in baseline microglial surveillance, while proper P2Y12R function is essential for the regulatory roles of microglia. In this study, we set out to investigate the effect of P2Y12R loss on microglial physiology and contact with other cell types.

The role of microglial P2Y12R was studied in wild-type mice, P2Y12R knockout mice as well as in mice injected icv with a selective P2Y12R antagonist. For morphological analysis and the contactomics of microglial cells with other cellular elements, we used confocal laser-scanning microscopy.

We found that the absence of P2Y12R altered microglial morphology both in the case of acute pharmacological inhibition and genetic deletion. The loss of function of P2Y12R also selectively changed microglial contactomics with glial cells and dedicated parts of the vasculature in the CNS. The acute and chronic lack of P2Y12R also affected microglia neuron interactions, while genetic deletion of P2Y12R also raised microglial cell numbers.

Our results indicate that P2Y12R function is essential for proper microglial actions even under physiological conditions. Disturbance of P2Y12R signalling has fundamental effects on microglial morphology and function. Since P2Y12R is only expressed by microglia in the brain potentially allowing cell-specific targeting, our results may give way to new therapeutic approaches in a broad range of neurological disorders.

TIBOR DONKA

Deeper insight into trauma induced coagulopathy

Introduction: Trauma-induced coagulopathy (TIC) is a highly complex process that is not yet fully understood. Blood loss, increased use of coagulation factors, increased fibrinolytic activity, and perturbation of platelet activation may all play a role. Our study aims to investigate platelet function and the coagulation cascade in patients with polytrauma.

Methods: We included severely injured (Injury Severity Score (ISS) ≥16), and bleeding patients aged ≥18 years from September 1, 2021. At arrival, platelet aggregometry and rotational viscoelastometry were performed on venous blood samples from injured patients. Baseline respiratory activity, oxidative phosphorylation capacity (OxPhos), mitochondrial electron loss rate, and maximum electron transport capacity of platelet mitochondria isolated from blood samples were assessed.

Results: Baseline mitochondrial respiratory activity of isolated platelets from polytrauma patients (n=57) did not change compared to control patients (n=48). However, OxPhos was significantly impaired compared to the control (37±52 pmol/ml/sec vs 64±18 pmol/ml/sec). Platelet functional tests in all polytrauma patients were below control group values, demonstrating severe coagulation disturbance.

Conclusions: Our results confirm the development of TIC in patients with polytrauma. ADP-induced platelet activation is perturbed, with simultaneous detection of platelet mitochondrial dysfunction, including OxPhos, which is required for platelet activation. A deeper understanding of maladaptive platelet responses after injury may provide the basis for exploring new therapeutic targets in polytrauma care.

KAROLINA PIRCS

Direct neuronal reprogramming: new possibility to study late onset neurodegenerative diseases

Pathological brain aging, leading to various uncurable neurodegenerative disorders, is causing increasingly severe and frequent public health issues in our ageing society. Dementia is one of the most common cognitive disorders that is not part of the 'normal' ageing process. Dementia can be the result of several conditions, including Alzheimer's, Parkinson's, Huntington's disease. New innovative therapeutic options for these conditions are an utmost importance in current neuroscience research to prevent, reverse or halt the adverse effects of neurodegeneration and cognitive impairment.

Based on the most recent cellular reprogramming technologies induced neurons (iNs) uniquely offers an opportunity to study molecular mechanisms of ageing and age-related neurodegenerative diseases in "old" cells directly reprogrammed from human dermal fibroblasts. Through transdifferentiation, these neurons retain the genotype and phenotype specific to the age of the donor. By comparing

iNs from donors of different ages, disease specific, age-related, human-relevant pathological changes can be obtained. iNs can be used in a cost-effective, robust, reliable, reproducible way for preclinical tests of drugs targeting neurodegenerative disorders, thus increasing the efficiency of clinical drug testing and reducing the number of failed trials and the associated costs. At the same time, iNs also offer a unique opportunity for personalized medicine, to optimize drug use and healthcare costs through appropriate, adjusted treatments. In summary, iNs are an extremely promising tool for studying various ageing and age-related neurodegenerative diseases, including Parkinson's, Alzheimer's, Huntington's disease, as well as for understanding the disease mechanisms.

BÁLINT KINTSES

The enemy of my enemy is my friend: viruses against antibiotic-resistant superbugs

Against antibiotic-resistant infections, researchers are developing a promising weapon: bacteriophages, or simply phages - tiny viruses that may have the potential to reverse the battle against bacteria. These microscopic warriors are precisely guided, targeting only harmful bacteria in our body. Envision these phages as microscopic detectives on a mission, seeking and destroying dangerous bacteria with unparalleled precision. Our research delves into the complex dynamics of the spread of epidemic pathogens, where, using large-scale genomics, we attempt to understand their spatial distribution over time. By strategically aligning these patterns of spread with suitable phages, our goal is to harness the full potential of phage therapy against superbugs that have become resistant to traditional antibiotics.

ANIKÓ BORBÁS

Bicyclic and tricyclic morpholinos: destruction and reconstruction of the furanose ring of nucleosides

Nucleoside and nucleotide derivatives are fundamental molecules of life. They play crucial roles in signal transduction and energy metabolism of cells, as well as the storage, transcription and translation of genetic information.

Nucleoside analogues, chemically modified derivatives of nucleosides, are an important class of pharmaceutical agent showing activity towards a range of biological targets. For therapeutic applications, nucleosides are typically modified at a number of different positions, including the ribose sugar, the phosphate backbone, or the nucleobase; the modifications increase in vivo stability and confer desired pharmacological properties. Important representatives of nucleoside analogues are morpholinos (morpoline-ring nucleosides), which contain a morpholine heterocycle instead of the native ribofuranose ring. Morpholino oligomers built up of morpholino monomers are valuable drugs in gene silencing therapy, and are also effective agents

against viruses.

Our research group are dealing with the development of new morpholino-type nucleoside analogues, which contain different bi- and tricyclic ring systems in place of the ribofuranose ring. In this lecture, the principles of chemical synthesis of these special morpholino nucleosides and their potential therapeutic applications will be presented.

DÓRA REGLŐDI

Travelling around a peptide

Pituitary adenylate cyclase activating polypeptide (PACAP) is a protective protein with significant anti-inflammatory anti-oxidant and pro-survival effects, shown in numerous cell culture and animal experiments. Our research group has proven the protective effects of PACAP in several models of neuronal diseases (such as Parkinson's disease, retinal diseases and stroke) and peripheral diseases. In addition, human studies have also pointed out the possible involvement of this protein in human pathological conditions. Mice lacking endogenous PACAP show increased sensitivity to several harmful stimuli and according to some data, they also show earlier aging signs. The aim of the presentation is to show some details from our ongoing studies regarding these above effects.

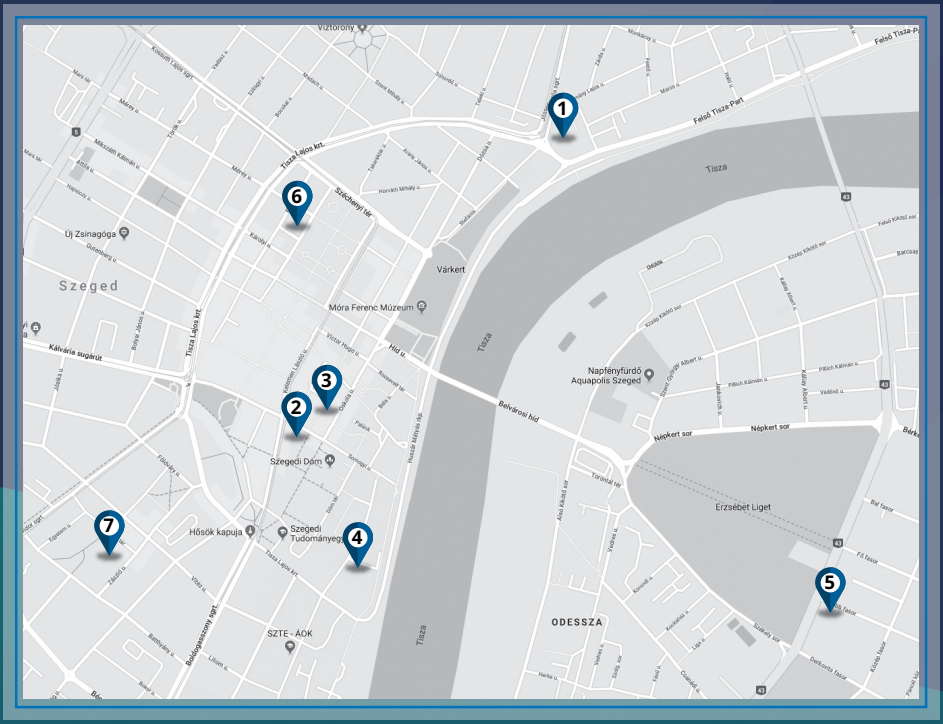
GÁBOR ORBÁN

Sci-fi in cardiology: How we treat cardiac arrhythmias

The heart is the engine of our body. It needs a particular rhythm to function efficiently, achieved by the heart's own electrical impulse generator and conduction system. Proper functioning of this system leads to coordinated electrical activation that causes the heart to contract in an orderly fashion, supplying blood to all parts of the body. Cardiac arrhythmias are conditions in which the normal rhythm 'goes wrong', with the heart pumping blood too fast or too slow, or even irregularly, which can cause a wide range of symptoms and even life-threatening conditions.

Arrhythmia development is like being a musician playing badly in an orchestra. A single musician not keeping the right tempo disrupts the sound of the whole orchestra and, in the case of the heart, the efficient pumping of blood. In treating arrhythmias, these abnormal musicians (myocardial cells) are sought and eliminated to restore the orchestra's sound, i.e., the heart's rhythm. The most effective way to do this is through a surgery called ablation. Ablation involves inserting thin, flexible tubes (catheters) with electrodes into the heart cavity through the blood vessels starting from the patient's femoral vein. The catheters are used to target the areas of the heart responsible for the arrhythmia. To identify these areas precisely, we use so-called electroanatomical mapping systems. Using these is like a conductor wanting to know precisely what instruments in the orchestra are playing and at what rhythm.

Mapping helps us understand exactly what is wrong with the sound of the orchestra, i.e., the functioning of the heart, and we can treat the problem. Mapping is based on creating an electromagnetic field in the operating room. The field is altered slightly by the heart's electrical activity and the movement of catheters we maneuver inside the heart. By detecting this change and then graphing it, we can create a real-time, three-dimensional model of the heart, the catheters inside it, and the propagation of the heart's electrical signals. In my presentation, I will give you an insight into the application of this almost sci-fi technology in cardiology and our research on it.



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2. Art Hotel Szeged
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4. Albert Szent-Györgyi Medical School, University of Szeged (Northern Clinical Gardens)
5. Biological Research Centre
6. Szeged City Hall
7. University of Szeged József Attila Study and Information Centre

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