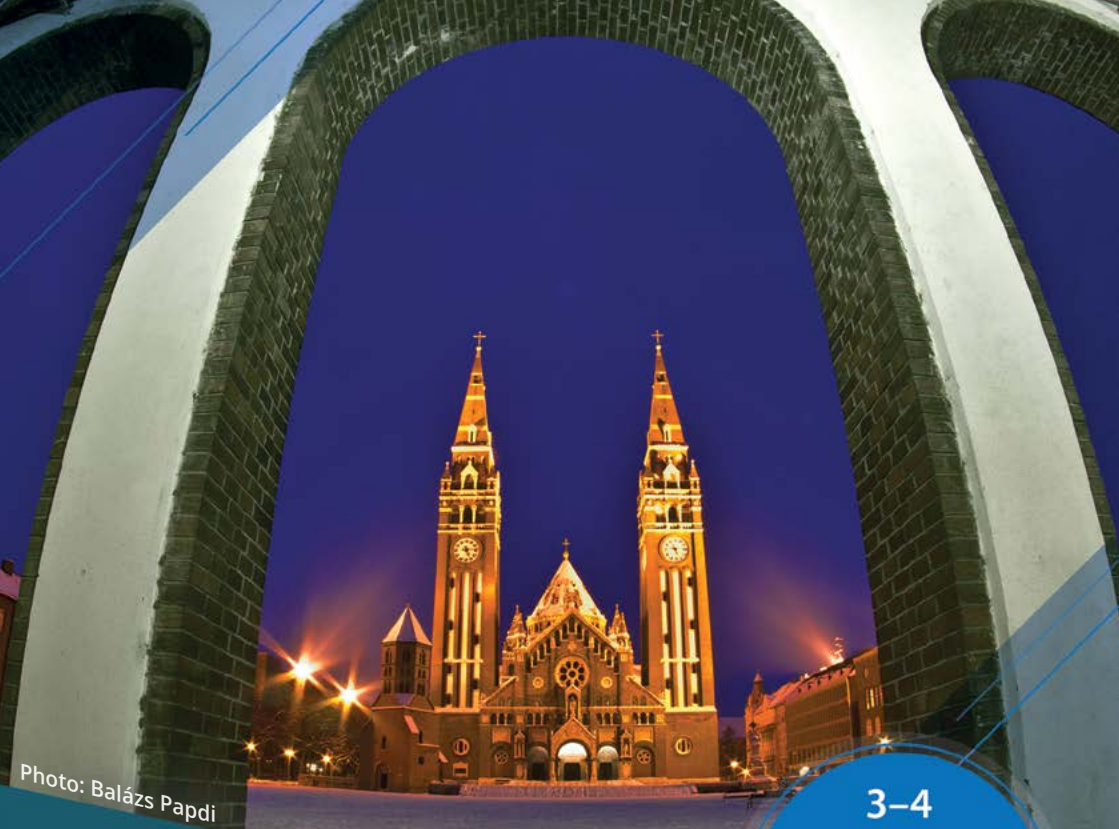

Photo: Balázs Papdi

3–4 December 2020

Szeged

XV. MEETING OF NOBEL LAUREATES AND TALENTED STUDENTS

A joint program of the Foundation for the Future of Biomedical Sciences in Szeged, the University of Szeged and the Biological Research Centre, Szeged

SZEGEDI TUDÓS AKADÉMIA
SZEGED SCIENTISTS ACADEMY

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SZEGED SCIENTISTS ACADEMY

PROGRAM

03 DECEMBER 2020 Thursday

14.00–15.00 PRESENTATION OF SZENT-GYÖRGYI MENTORS
Chairmen: Dr. András Varró, Dr. Péter Hegyi

14.00–14.20 Dr. Antal Berényi (Talentum Prize 2017)
Oscillotherapeutics: Space and time targeted brain stimulation in epilepsy

14.20–14.40 Dr. Lajos Haracska (Talentum Prize 2018)
Molecular mechanism of mutagenesis and carcinogenesis

14.40–15.00 Dr. Péter Horváth (Talentum Prize 2019)
Life beyond the pixels: single-cell analysis using machine learning and image analysis methods

15.00–15.20 BREAK

15.20–16.20 AWARDING CEREMONIES

15.20–15.40 'Szent-Györgyi Pupil 2019 Excellence Award'
Dominik Dobos:
Analog model of the human cardiovascular system

15.40–16.00 'Szent-Györgyi Student 2020 Excellence Award'
Valéria Éva Meszlényi:
Alterations of motor axon terminals in mice due to passive transfer with sera of amyotrophic lateral sclerosis patients with identified mutations

16.00–16.20 'Talentum Prize 2020' Award

16.20–16.30 BREAK

16.30–17.00 CONSTRUCTION WORKS OF THE FUTURE HALL OF RESIDENCE OF THE SZEGED SCIENTISTS ACADEMY

17.00–18.40 THE 75TH ANNIVERSARY OF ALBERT SZENT-GYÖRGYI'S NOBEL PRIZE AWARD CONFERENCE
(summary video of the conference held on 22-25 March 2012, welcoming 9 Nobel laureates in Szeged)

18.40–18.50 BREAK

18.50–20.00 GALA
(video of the gala event at the XIII. Meeting of Nobel laureates and talented students)

04 DECEMBER 2020 Friday

08.30–10.00 PREPARATORY PRESENTATION
Sándor Bán:
History of antibiotics and antibiotic resistance
Test task on the presentation

10.00–10.15 BREAK

10.15–11.00 VIRTUAL VISIT IN THE LABORATORIES OF THE SZENT-GYÖRGYI MENTORS
(upon preliminary assignment)
Test task on the visits in the laboratories

11.00–12.00 ROUND TABLE DISCUSSIONS
between the Szent-Györgyi Students and Pupils
(upon preliminary assignment)
Free discussion on the Szeged Scientists Academy Program

12.00–12.45 LUNCH BREAK

12.45–15.30 PLENARY SESSION

Chairmen: Dr. András Varró, Dr. Péter Hegyi

12.45–12.50 Opening

12.50–12.55 Welcome to Szeged

12.55–13.15 **Dr. Péter Hegyi:**
Introduction of the Szeged Scientists Academy

13.15-13.20 Welcome to the University of Szeged

13.20–13.40 **Dr. Zoltán Rakonczay:**
Introduction of the University Program of the Szeged Scientists Academy

13.40–13.50 **Dr. Bert Sakmann's** welcome speech

13.50–14.00 Break

14.00–14.10 **Dr. Csaba Pál:**
Laudation of Dr. Ada E. Yonath

14.10–15.00 **Dr. Ada E. Yonath:**
Next generation antibiotics

15.00–15.30 Live discussion, questions

15.30–15.40 BREAK

15.40–16.40 CLOSED DISCUSSION OF THE SZENT-GYÖRGYI STUDENTS WITH DR. ADA E. YONATH AND DR. BERT SAKMANN
(upon invitation)

Parallel program:

15.40–17.00 Summary videos of the previous conferences

LECTURE ABSTRACTS

In the sequence of performance

DR. ANTAL BERÉNYI

Oscillotherapeutics: Space and time targeted brain stimulation in epilepsy

Rhythmic brain activity supports many physiological functions from motor control to cognition. Disruptions of the normal oscillatory brain patterns are commonly observed in neurological and psychiatric disorders including epilepsy, anxiety/trauma-related disorders, major depressive disorder, and many others. Therefore, these diseases can be considered also as oscillatory defects ('Oscillopathies') despite having distinct behavioral manifestations and genetic causes. An increasing number of studies suggest that there is a causal link between these disturbed network oscillations and the symptomatic manifestation of the disorders.

Innovative approaches of recording and analysis techniques allowed us to study the pathological oscillations in models of these disorders as possible biomarkers of symptoms. Furthermore, recent advances in brain stimulation technologies enable time- and space-targeted interference with the pathologic oscillations in epilepsy, as possible targets to regulate symptoms.

The overarching goal of our decade-long efforts is to establish an on-demand, non-pharmaceutical approach to prevent the development of epileptic seizures. In my talk, I will present the results of our research in intervening epileptic seizures by promptly disrupting pathologic network oscillations using electrical stimulation. Our translational work aiming the use of this technology on human patients will also be highlighted.

DR. LAJOS HARACSKA

Molecular mechanism of mutagenesis and carcinogenesis

Our research laboratory focuses on the forces driving the molecular mechanisms of mutagenesis and carcinogenesis. DNA is constantly exposed to exogenous and endogenous agents that cause damage, hindering the activity of the replication machinery. Replication fork stalling may cause strand breaks and chromosomal rearrangements, leading to genome instability and resulting in early onset aging and eventually cancer. Several DNA damage tolerance pathways have evolved to rescue the stalled replication fork, which do not necessarily repair the lesion but rather facilitate mechanisms that replicate

across the damaged segment, e.g., translesion synthesis. Translesion synthesis is error prone, which may lead to mutagenesis and cancer. By contrast, error-free lesion bypass mechanisms maintain a low level of mutagenesis and cancers. One of the most intriguing questions is how decision between these different DNA damage tolerance pathways are made. We have recently identified new regulators that act when replication encounters DNA damage. SPARTAN, one of our recent findings, promotes genomic stability by regulating choices of rescuing the stalled replication fork. Association has been found between mutations in SPARTAN and early onset hepatocellular carcinoma and premature aging. Our results demonstrate that Spartan acts as a protease promoting the replication of DNA-protein crosslink-containing DNA, which may highlight its role in preventing carcinogenesis and aging.

DR. PÉTER HORVÁTH

Life beyond the pixels: single-cell analysis using machine learning and image analysis methods

In this talk I will give an overview of the computational steps in the analysis of a single cell-based large-scale microscopy experiments using deep learning techniques. First, I will present a novel microscopic image correction method designed to eliminate illumination and uneven background effects. New single-cell image segmentation methods will be presented using differential geometry, energy minimization and deep learning methods (www.nucleaizer.org). I will discuss the Advanced Cell Classifier (ACC) (www.cellclassifier.org), a machine learning software tool capable of identifying cellular phenotypes based on features extracted from the image. It provides an interface for a user to efficiently train machine learning methods to predict various phenotypes. For cases where discrete cell-based decisions are not suitable, we propose a method to use multi-parametric regression to analyze continuous biological phenomena. To improve the learning speed and accuracy, we propose an active learning scheme that selects the most informative cell samples.

Our recently developed single-cell isolation methods, based on laser-microcapturing and patch clamping, utilize the selection and extraction of specific cell(s) using the above machine learning models. I will show that we successfully performed DNA and RNA sequencing, dPCR, and targeted electrophysiology measurements on the selected cells.

Finally I will show our results in the COVID-19 fight using deep learning methods (Daly etal Science 2020).

DOMINIK DOBOS

Analog model of the human cardiovascular system

The aim of the project was to create a system of a model that demonstrates the human blood circulation and auxilliary software which together display the nature of the circulatory system, the pecularities of its operation, its structure and illnesses. The model serves as a demonstration tool to the study material presented by the software. It also shows information based on data provided by various sensors embedded into the model. This information is very similar to what one should expect from the real circulatory system, and thus carry immense value regarding educational purposes.

VALÉRIA ÉVA MESZLÉNYI

Alterations of motor axon terminals in mice due to passive transfer with sera of amyotrophic lateral sclerosis patients with identified mutations

Introduction: Amyotrophic lateral sclerosis (ALS) is an incurable neurodegenerative disease affecting the motor neurons in the brain and spinal cord, leading to the loss of voluntary muscle function. ALS can be classified as familial or sporadic based on its inheritance. Previously, our research group demonstrated an increase of the calcium level and increase of the number of synaptic vesicles in the motor axon terminals (MATs) of sporadic ALS patients. These alterations could be conferred to mice via passive transfer of the sera from these patients. In the present experiment we aimed to question the similar effect of the sera from another population of patients, which possess identified mutations.

Methods: Patients with 11 different ALS-related mutations participated in our study. Balb/c mice were injected intraperitoneally with sera of ALS patients or healthy individuals for two days. After the passive transfer, calcium histochemistry was performed and MATs in the interosseus muscles were investigated under electron microscope. Intracellular calcium content and synaptic vesicle number was quantified.

Results: Passive transfer resulted in significant increase of intracellular calcium levels and synaptic vesicle number in the MATs of mice due to the passive transfer of sera from ALS patients with identified mutations, similar to sera from sporadic ALS patients. In addition, ultrastructural signs of degeneration were also observed at the neuro-muscular synapses.

Discussion: Our findings support the unifying hypothesis, that the pathomechanism underlying the identical manifestation of the disease with or without identified mutations is based on a common final pathway, in which calcium increase plays a central role.

Sponsors of the XV. Meeting of
Nobel laureates and talented students:

Szeged Megyei Jogú Város

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