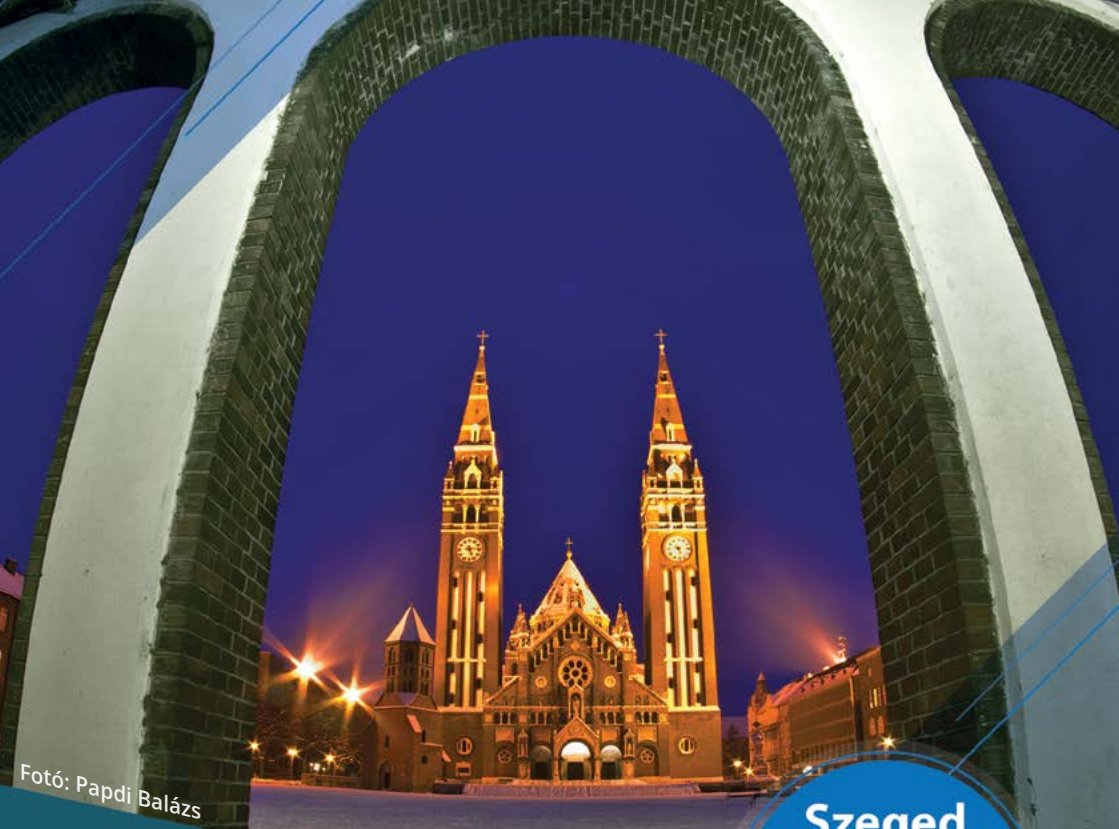

Fotó: Papdi Balázs

Szeged

2020.
december
3–4.

NOBEL-DÍJASOK ÉS TEHETSÉGES DIÁKOK XV. TALÁLKOZÓJA

A Szegedi Orvosbiológiai Kutatások Jövőjéért Alapítvány,
a Szegedi Biológiai Kutatóközpont és a Szegedi
Tudományegyetem közös programja

SZEGEDI TUDÓS AKADÉMIA
SZEGED SCIENTISTS ACADEMY

SZEGEDI TUDÓS AKADÉMIA
SZEGED SCIENTISTS ACADEMY

PROGRAM

2020. DECEMBER 3. csütörtök

14.00–15.00 SZENT-GYÖRGYI MENTOROK ELŐADÁSAI
Üléselnökök: Dr. Varró András, Dr. Hegyi Péter

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

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

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

15.00–15.20 SZÜNET

15.20–16.20 DÍJÁTADÓK

15.20–15.40 **Az év Szent-Györgyi Diákja 2019” díj átadása, a díjazott előadása**
Dobos Dominik: *Analog model of the human cardiovascular system*

15.40–16.00 **„Az év Szent-Györgyi Hallgatója 2020” díj átadása, a díjazott előadása**
Meszlényi Valéria Éva: *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 **A 2020-as év Talentum-díjasának ünnepélyes bejelentése, a díjazott előadása**

16.20–16.30 SZÜNET

16.30–17.00 A SZEGEDI TUDÓS AKADÉMIA LEENDŐ DIÁKOTTHONÁNAK BEMUTATÁSA
(az épület rekonstrukciójáról készített videó vetítése)

17.00–18.40 NEMZETKÖZI KONFERENCIA SZENT-GYÖRGYI ALBERT NOBEL-DÍJÁNAK 75. ÉVFORDULÓJA ALKALMÁBÓL
(a 2012. március 22–25. között megrendezett konferencia összefoglaló filmjének vetítése)

18.40–18.50 SZÜNET

18.50–20.00 GÁLAMŰSOR
(a Nobel-díjasok és tehetséges diákok XIII. találkozójára szervezett gálaműsor közvetítése)

2020. DECEMBER 4. péntek

08.30–10.00 ELŐKÉSZÍTŐ PREZENTÁCIÓ A DÍSZELŐADÁSHOZ
Bán Sándor:
Az antibiotikumok és az antibiotikum rezisztencia története
Teszt feladatsor az előkészítő előadáshoz kapcsolódóann

10.00–10.15 SZÜNET

10.15–11.00 SZENT-GYÖRGYI MENTOROK LABORATÓRIUM BEMUTATÓ VIDEÓINAK MEGTEKINTÉSE
(előzetes beosztás alapján)
Teszt feladatsor a laboratóriumok bemutató videóihoz kapcsolódóan

11.00–12.00 KEREKASZTAL BESZÉLGETÉSEK
a Szent-Györgyi Hallgatók és Diákok között
(előzetes beosztás alapján)
Kötetlen beszélgetés a Szegedi Tudós Akadémiáról

12.00–12.45 EBÉDSZÜNET

12.45–15.30 PLENÁRIS ÜLÉS

Üléselnökök: Dr. Varró András, Dr. Hegyi Péter

12.45–12.50 Megnyitó

12.50–12.55 Szeged bemutatása

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

13.15-13.20 A Szegedi Tudományegyetem bemutatása

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

13.40–13.50 **Dr. Bert Sakmann** köszöntője

13.50–14.00 Szünet

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

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

15.00–15.30 Kérdések

15.30–15.40 SZÜNET

15.40–16.40 SZENT-GYÖRGYI HALLGATÓK ZÁRTKÖRŰ TALÁLKOZÓJA DR. ADA E. YONATH-TAL ÉS DR. BERT SAKMANNAL

Párhuzamos program:

15.40–17.00 Korábbi konferenciákról készített összefoglaló videók vetítése

ELŐADÁS KIVONATOK
az elhangzás sorrendjében

DR. BERÉNYI ANTAL

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. HARACSKA LAJOS

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. HORVÁTH PÉTER

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).

DOBOS DOMINIK

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.

MESZLÉNYI VALÉRIA ÉVA

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 neuro-degenerative 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.

A Nobel-díjasok és
tehetséges diákok XV. találkozója
rendezvény támogatói:

Szeged Megyei Jogú Város

MAGYARORSZÁG
KORMÁNYA

A Szegedi Orvosbiológiai Kutatások Jövőjéért Alapítvány Szegedi Tudós Akadémia Programja az Innovációs és Technológiai Minisztérium támogatásával valósul meg (FEIF/433-4/2020-ITM_SZERZ).