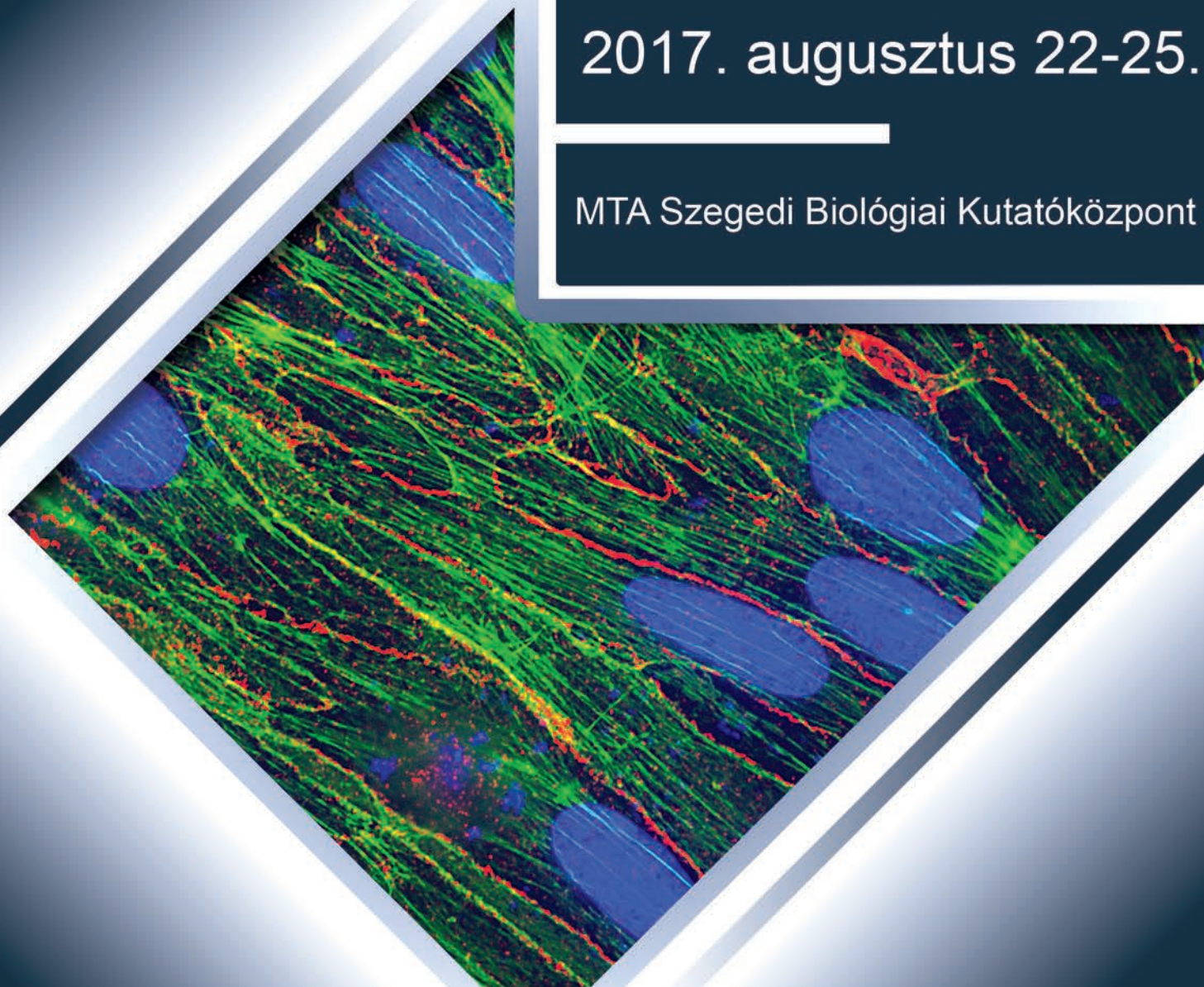


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MTA Szegedi Biológiai Kutatóközpont



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Újgenerációs elektronmikroszkópos technika bevezetése szerkezetkutatási alkalmazásokban

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A sejtbiológiai kutatások feladata az élő anyag működésének, szerkezetének, valamint ezek összefüggéseinek megértése. Ennek érdekében a szekvenálási módszerek hatalmas mennyiségű adatot generálnak —mely „omikai” adatbázisokkal való kombinálás után— alapvető folyamatok megértéséhez járulnak hozzá. A felhalmozódó molekuláris biológiai adatmennyiség strukturális relevanciájának megismerése az eredmények térbeli rekonstruálását igényli. A megcélolni kívánt szubcelluláris tartományban történő analízis nm-es skálán mozgó strukturális vizsgálatokat igényel, mely elektronmikroszkópos módszerekkel érhető el. Ezek közül az elektrontomográfias eljárás biztosítja azt a rugalmasságot, ami a nagyfelbontású mikroszkópiát a térbeli rekonstrukcióval házasítja. Ez az új technológia néhány nm-re javítja a szokványos transzmissziós elektronmikroszkópia 50–100 nm-es z-irányú felbontását, lehetővé téve 150–250 nm mélységű térben is a mikroszkopikus objektumok szerkezeti kapcsolódásainak, a makromolekulák szerveződésének térbeli feltérképezését. A $\pm 70^\circ$ mintadöntési tartományban működő eszköz az MTA SZBK Biofizikai Intézetében 2017-ben kerül beszerzésre, s várhatóan 2018-tól lesz elérhető külső felhasználók számára. A képrögzítés 16 MP felbontású CMOS kamerákkal történik; a felbontás csaknem tetszőlegesen növelhető megfelelően választott nagyítású képek automatikus illesztésével. A képrögzítésen túl a készülék elektrongerjesztéses röntgenanalízis segítségével a 11-es rendszámú elemtől felfelé, a Mn K_α vonalának fél-érték szélességénél mérve 130 eV minimális felbontással kémiai elemvizsgálásra is használható, melynek alapja egy 30 mm² szilikon drift detektor.

Különböző gyártók eszközeinek tomográfias lehetőségeit kipróbálva előzetes eredményként bemutatjuk, hogy 30–40 nm karakterisztikus méretű precipitátumok térbeli sűrűségének meghatározását mennyiben befolyásolja a Holmes effektus, illetve egy atomerő mikroszkópiával standardizált vastagságú mintasorozat segítségével kidolgozott eljárásunkat mutatjuk be, mely segítségével, a Beer-Lambert-Bouguer törvényre alapozva, a jelenség korrigálható. Továbbá, egy idegtudományi probléma során a Rtg-mikroszkópia alkalmazhatóságát is bemutatjuk.

Köszönetnyilvánítás

Köszönjük a Nemzetgazdasági Minisztérium (GINOP-2.3.3.-15-2016-00001) valamint a JEOL és FEI munkatársainak támogatását. Munkánkat a „A Szegedi Orvosbiológiai Kutatások Jövőjéért Alapítvány” támogatta az Emberi Erőforrások Minisztériumának anyagi támogatásával (34232-3/2016/INTFIN).

JEGYZETEK

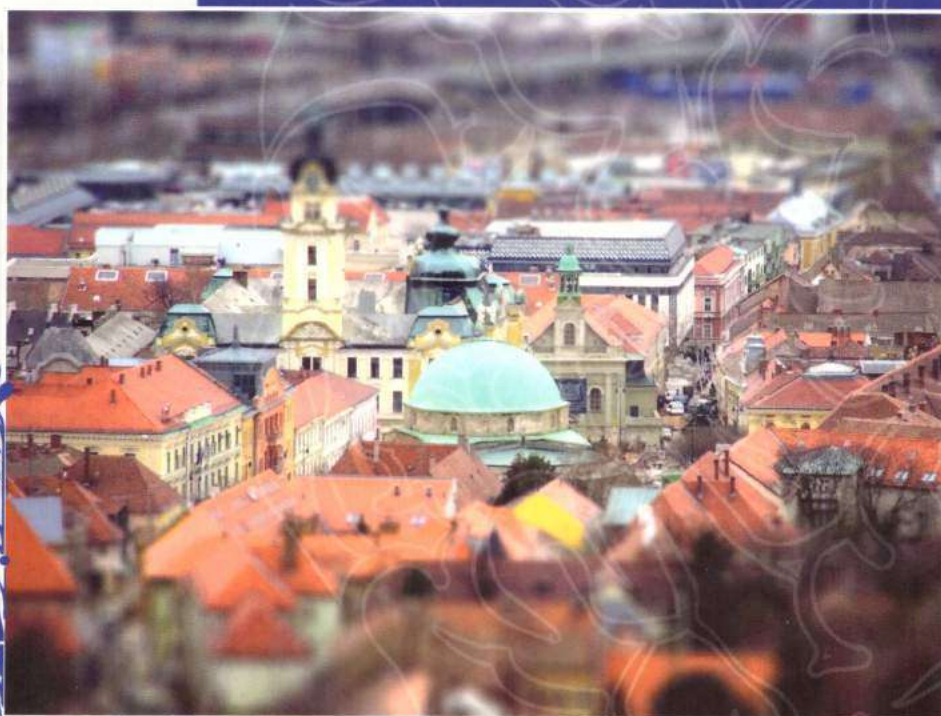


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2018. MÁJUS 10-11.

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MTA SZEGEDI BIOLÓGIAI KUTATÓKÖZPONT
AZ EURÓPAI UNIÓ KIVÁLÓSÁGI KÖZPONTJA

SZEGED, TEMESVÁRI KRT. 62., NAGYELŐADÓ



**Expression and role of pattern recognition receptors and activation of inflammasomes in cerebral endothelial cells and pericytes**

Mihály Kozma, Ádám Nyúl-Tóth, Péter Nagyősi, Krisztina Nagy, Csilla Fazakas, János Haskó, Kinga Molnár, Attila E. Farkas, Attila G. Végh, Péter Galajda, Imola Wilhelm, István A. Krizbai

Institute of Biophysics, BRC HAS, Szeged, Hungary

Integrated optical investigation of the photocycle of dried Photoactive Yellow Protein films in environments of controlled humidity

Szilvia Krekic, Dávid Nagy, László Fábián, László Zimányi, András Dér

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Changes in the motoneuronal calcium level after long-term treatment with blood serum from patients with amyotrophic lateral sclerosis

Valéria Meszlényi^{1,2}, Roland Patai¹, Bernát Nógrádi^{1,2}, Tamás Ferenc Polgár¹, József István Engelhardt³, László Siklós¹

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Biotin and glutathione targeting of solid nanoparticles to cross human brain endothelial cells

Mária Mészáros¹, Lóránd Kiss¹, Zoltán Kóta¹, Tibor Páli¹, Zsófia Hoyk¹, Zsolt Bozsó², Livia Fülöp², András Tóth^{1,3}, Gábor Rákhely^{1,3}, Mária A. Deli¹, Szilvia Veszelka¹

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Interactions of malignant cells with the cerebral endothelium during brain metastasis formation

Kinga Molnár¹, Csilla Fazakas¹, János Haskó¹, Hildegard Herman², Judit Molnár¹, Ádám Nyúl-Tóth¹, Mihály Kozma¹, Attila E. Farkas¹, Anca Hermenean², István A. Krizbai¹, Imola Wilhelm¹

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Improved fluorescence image quality using re-scan confocal microscope (RCM)

Dávid Nagy, Gábor Steinbach, László Zimányi

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Evolution of antibiotic resistance in microstructured environments

Krisztina Nagy, Ágnes Ábrahám, Barbara Dukic, Péter Galajda

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Bernát Nógrádi^{1,2}, Roland Patai¹, Valéria Meszlényi^{1,2}, Tamás Ferenc Polgár¹, László Siklós¹

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Integrated optical Mach-Zehnder interferometer biosensor and its improvement

Dániel Petrovskzi, Sándor Valkai, András Dér

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The effect of lidocaine on the surface charge of brain endothelial cells

Ana Raquel Santa Maria¹, Ana Rita Bras², Dóra Lipka¹, Sándor Valkai¹, András Kincses¹, András Dér¹, Mária A. Deli¹

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Targeted delivery of vesicular nanoparticles across a culture model of the blood-brain barrier

Gergő Porkoláb¹, Mária Mészáros¹, Zoltán Kóta¹, Tibor Páli¹, Ana-Maria Pilbat², Zsolt Török², Zoltán Kupihár³, Piroska Szabó-Révész⁴, Mária A. Deli¹, Szilvia Veszelka¹

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Quantification of intracellular calcium level and neuronal survival after acute blood serum inoculation from patients with different genetic mutations of amyotrophic lateral sclerosis

Tamás Ferenc Polgár¹, Roland Patai¹, Bernát Nógrádi^{1,2}, Valéria Meszlényi^{1,2}, József István Engelhardt³, László Siklós¹

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Membrane tethers in brain metastasis formation

Attila G. Végh¹, Béla Varga^{1,2}, Réka-Anita Domokos^{1,3}, Csilla Fazakas¹, Imola Wilhelm¹, Zsolt Szegletes¹, István A. Krizbai^{1,4}, György Váró¹

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Az impulzus üzemmódú Fourier-transzformációs elektron paramágneses rezonancia spektroszkópia lehetőségei

Sebőkné Nagy Krisztina, Páli Tibor

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P1.51 Az inzulin receptor-pozitív elsődleges érző neuronok fokozott inzulin-függő neurit-növekedési képessége

Pálvolgyi Laura¹, Lázár Bence András^{1,2}, Jancsó Gábor¹, Dobos Ildikó¹, Nagy István³, Sántha Péter¹

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P1.52 Role of ventral pallidal dopamine-neurotensin interactions in the regulation of reward and anxiety

Tamás Ollmann^{1,2}, László Péczely^{1,2}, Veronika Kállai^{1,2}, Daniella Dusa^{1,2}, Kristóf László^{1,2}, Beáta Berta^{1,2}, Anita Kovács^{1,2}, Erika Kertes^{1,2}, Rita Gálosi^{1,2}, Olga Zagorác^{1,2}, Zoltán Karádi^{1,2,3}, László Lénárd^{1,2,3}

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P1.53 A medián ráfe régió glutamaterg sejtjeinek szerepe a szociális érdeklődésben

Fazekas Csilla Lea¹, Török Bibiána^{1,2}, Szöllőssy-Csoma Bálint¹, Horváth Hanga Réka¹, Manon Bellardie¹, Sipos Eszter¹, Zelena Dóra^{1,3}

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Meszlényi Valéria Éva^{1,2}, Polgár Tamás Ferenc¹, Patai Roland¹, Nógrádi Bernát^{1,2}, Engelhardt József István³, Siklós László¹

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P1.55 Szomatosztatin 4 receptor (Sst4) expressziós mintázatának vizsgálata egér központi idegrendszerben

Kecskés Angéla^{1,3}, Pohóczky Krisztina^{1,2,3}, Szentes Nikolett^{1,3}, Bakai Rita¹, Ábrahám István^{3,4,5}, Helyes Zsuzsanna^{1,3,4}

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- [P1.32] Association between childhood adversities and the adult onset of comorbid mood and physical disorders**
J. Oliveira^{*1,2}, V. Paixão², G. Cardoso^{1,3}, M. Xavier¹, J.M. Caldas-de-Almeida^{1,3}, A.J. Oliveira-Maia^{1,2}, ¹NOVA Medical School | Faculdade de Ciências Médicas, Universidade Nova de Lisboa, Portugal, ²Champalimaud Centre for the Unknown, Portugal, ³Lisbon Institute of Global Mental Health, Portugal
- [P1.33] Benserazide, a modulator of down-stream kynurenine pathway, attenuated development of metabolic syndrome induced by antipsychotic medication in mouse model: Potential translation into clinical practice**
G. Oxenkrug^{*}, P. Summergrad, Tufts University, USA
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B. Nógrádi^{1,2}, R. Patai^{*1}, V. Meszlényi^{1,2}, T.F. Polgár¹, L. Siklós¹, ¹Biological Research Centre of the Hungarian Academy of Sciences, Hungary, ²Szeged Scientists Academy, Hungary
- [P1.35] Infections of the central nervous system as a risk factor for mental disorders and cognitive impairment: A nationwide register-based study**
E. Pedersen^{*1}, O. Köhler-Forsberg¹, M. Nordentoft¹, L. Pedersen², M. Benros¹, ¹University of Copenhagen, Denmark, ²Aarhus University, Denmark
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B. Charvet², E. Saba⁶, R. Gorter⁴, J. Pierquin², J. Brunel², S. Khoury⁶, S. Amor^{4,5}, H. Perron^{*1,3}, ¹Geneuro, Switzerland, ²Geneuro, France, ³University of Lyon, France, ⁴VU Medical Center Amsterdam, The Netherlands, ⁵Queen Mary University of London, UK, ⁶American University of Beirut MS Center, Lebanon
- [P1.37] Searching for novel autoantibodies in psychiatric disorders - a case control and follow up study**
A. Zandian^{2,4}, L. Wingård¹, H. Nilsson¹, E. Sjöstedt^{3,4}, D.X.J. Johansson¹, D. Just^{2,4}, C. Hellström^{2,4}, A. Häggmark-Månberg^{2,4}, P. Nilsson^{2,4}, M.A.A. Persson^{*1,4}, ¹Karolinska Institutet, Sweden, ²Royal Institute of Technology, Sweden, ³Uppsala University, Sweden, ⁴Science for Life Laboratory, Sweden
- [P1.38] The effects of systemic inflammation on the trajectory of cognition and structural changes to the brain during nine-year follow-up in the RUN DMC longitudinal cohort study**
A.M. Peters van Ton^{*}, E.M.C. van Leijsen, M.I. Bergkamp, A.M. Tuladhar, F.E. de Leeuw, W.F. Abdo, Radboud University Medical Center, The Netherlands
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M. Pons-Espinal^{*1,2}, L. Blasco-Agell^{1,2}, G. Carola^{1,2}, A. Di Domenico^{1,2}, I. Fernandez-Carasa^{1,2}, E. Tolosa^{3,4}, M. Juan-Otero^{3,4}, A. Raya⁵, A. Consiglio^{1,2}, ¹Institute of Biomedicine of the University of Barcelona, Spain, ²University of Barcelona, Spain, ³Hospital Clinic de Barcelona, Spain, ⁴Institut d'Investigacions Biomediques August Pi i Sunyer, Spain, ⁵Center of Regenerative Medicine in Barcelona, Spain
- [P1.40] Unmutated human antibodies against the NMDA receptor are functional**
N.K. Wenke¹, J. Kreye^{1,2}, E. Andrzejak¹, A. van Casteren¹, F. Ackermann¹, C.G. Garner¹, H. Prüss^{*1,2}, ¹German Center for Neurodegenerative Diseases, Germany, ²Charité - Universitätsmedizin, Germany
- [P1.41] Encephalopathy associated with anti-IgLON5 antibody: Report of three cases**
H.T. Ren^{*}, H.Z. Guan, S.Y. Fan, S. Wang, X.L. Xu, H.L. Hao, C.Y. Liu, L.Y. Cui, Y. Huang, Peking Union Medical College Hospital, China
- [P1.42] Serum inflammatory markers in severe mental illness: Decrease of interleukins 2 and 6 after inpatient treatment in a longitudinal prospective study**
A.A. Schmitt Junior¹, L. Primo de Carvalho Alves^{1,2}, N.S. Rocha^{*1,2}, ¹Hospital de Clinicas de Porto Alegre, Brazil, ²Universidade Federal do Rio Grande do Sul, Brazil
- [P1.43] Premature T helper cell aging in major depression Childhood trauma induces myeloid inflammatory activation only in conjunction with premature aging**
C. Schiweck^{*1}, S. Claes¹, V. Arolt³, N. Müller⁴, E. Vrieze¹, H.A. Drexhage¹, ¹KU Leuven, Belgium, ²Erasmus MC Rotterdam, The Netherlands, ³WWU Muenster, Germany, ⁴LMU Munich, Germany
- [P1.44] Neuro-inflammation and neuro-immune abnormalities in autism spectrum disorders: Targeting the endocannabinoid system for potential pharmacological treatment**
D. Siniscalco^{*1,2}, A.L. Brigida², S. Schultz³, N. Antonucci⁴, ¹University of Campania, Italy,

16TH MEETING OF THE HUNGARIAN NEUROSCIENCE SOCIETY



17-18 JANUARY, 2019 DEBRECEN, HUNGARY

36	Magor Lőrinc	PRONOUNCED HETEROGENEITY AMONG THE ACTIVITY OF VARIOUS NEOCORTICAL NEURONS DURING ABSENCE SEIZURES	c
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38	Ábel Major	EFFECT OF NATURALLY DEPOSITED AMYLOID-BETA ON THE HIPPOCAMPAL NETWORK	c
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44	Kristóf Furuglyás	LAYER-SPECIFIC SINGLE NEURON - LFP CORRELATIONS AND CAUSAL FUNCTIONAL CONNECTIVITY DURING MULTIMODAL MISMATCH DETECTION TASK	d
45	Andrea Gajtkó	CYTOKINE EXPRESSION OF REACTIVE ASTROCYTES IN CHRONIC INFLAMMATORY PAIN	d

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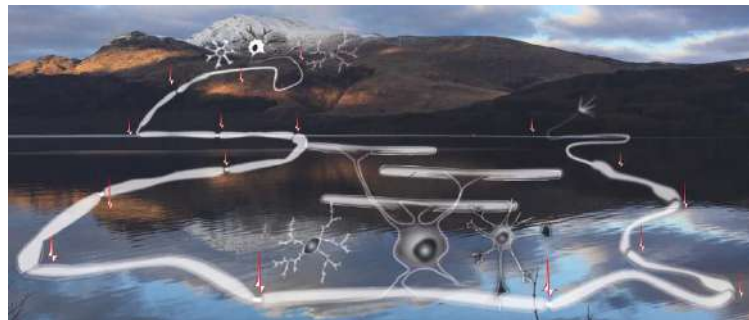


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Poster and short talk abstracts

44. Ultrastructural changes in the motoneuron cell body and motor end-plate induced by acute blood serum inoculation of ALS patients with different genetic mutations

Bernát Nógrádi^{1,2}, Valéria Meszlényi^{1,2}, Roland Patai¹, Tamás Ferenc Polgár¹, József István Engelhardt³, László Siklós¹

¹Hungarian Academy of Sciences, Biological Research Centre, Institute of Biophysics, Szeged, Hungary; ²Foundation for the Future of Biomedical Sciences in Szeged, Szeged Scientists Academy, Szeged, Hungary; ³University of Szeged, Faculty of Medicine, Department of Neurology, Szeged, Hungary

Introduction: Amyotrophic lateral sclerosis (ALS) is a progressive degenerative disorder, primarily affecting motor neurons. The genetic screening of a subpopulation of Hungarian ALS patients revealed mutations on the superoxide dismutase 1 (SOD1) and chromosome 9 open reading frame 72 (C9ORF72) genes. Our aim was to determine the importance of different neuropathological aspects in each of these mutations, such as intracellular calcium level elevation, mitochondrial ultrastructural alterations and survival of motor neurons.

Methods: Balb/c mice were inoculated intraperitoneally (1 ml/day) for 2 days with blood serum of ALS patients with identified mutation or sporadic ALS patients without mutation (n=3-6/group). Non-treated and healthy serum treated groups were used as controls. Lumbar spinal cord and interosseus muscle samples were used for electron microscopic examination of motoneuron somas and neuro-muscular synapses, respectively. Motoneuronal survival was quantified on semi-thin lumbar spinal cord samples, stained with cresyl violet.

Results: Acute passive transfer of the blood serum of ALS patients induced significant decrease in the number of motor neurons in the spinal cord, elevation of intracellular calcium level and increase of lipofuscin vesicle volume. Motoneuronal loss and calcium level changes were most prominent in C9ORF72 mutation. Furthermore, partial mitochondrial volume decreased in SOD1 point mutations.

Conclusions: Our previous experiments demonstrated the central role of intracellular calcium level elevation in ALS. Our current findings proved that different mutations induced similar morphological alterations, but to different extent. The extensive loss of motoneurons and changes in intracellular calcium

Poster and short talk abstracts

level suggest that C9ORF72 mutation might present a more progressive phenotype.

Acknowledgement: The project was supported by the Foundation for the Future of Biomedical Sciences in Szeged Program (13725-2/2018/INITFIN), the UNKP-18-2 New National Excellence Program of the Hungarian Ministry of Human Capacities and the UNICAM Magyarország Ltd.

Changes in motor neuronal calcium level after long-term treatment with serum from patients diagnosed with sporadic amyotrophic lateral sclerosis

Valéria Meszlényi^{1,2}, Roland Patai¹, Bernát Nógrádi^{1,2}, Tamás F. Polgár¹, Izabella Obál^{3,4}, József I. Engelhardt⁴ and László Siklós¹

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Keywords: ALS; motor neuron; calcium homeostasis; stereology; electron microscopy

Introduction: Different pathological processes lead to motor neuronal degeneration in sporadic amyotrophic lateral sclerosis (sALS). Our aim was to understand the effect of chronic treatment with sera that contains anti-motoneuronal antibodies of sALS patients on calcium homeostasis, neuronal survival and motor function.

Methods: Balb/c mice were injected intraperitoneally for 75 days with blood serum from healthy individuals (n=3) and patients diagnosed with sALS (n=6). During the passive transfer isometric muscle strength of their limbs was measured. Number of motor neurons in the spinal cord was measured with the disector method. Intracellular calcium level was quantified with geometrical statistics in the spinal cord and in the axon terminals of their *musculus interosseus* samples.

Results: Isometric muscle strength significantly decreased after long-term inoculation of sera from sALS patients. The number of motor neurons were also significantly decreased in the cervical ($p<0.001$) and lumbar ($p<0.001$) spinal cord. Significant increase of intracellular calcium could be documented in the cervical ($p<0.001$) and lumbar ($p<0.001$) spinal cord and in the axon terminals of their forelimb ($p<0.001$) and hindlimb ($p<0.01$). Ultrastructural alterations of perikarya and neuromuscular synapses were observed.

Discussion and conclusion: Functional regression, motor neuronal loss and elevated intracellular calcium level confirm the motor neuronal degeneration in this model. Such long-term treatment provides a feasible representation of sALS, as no genetic mutations were observed in the patients. However, it is reconcilable with the transgenic animal models widely used in pharmacotherapeutic tests. Therefore this model could be give us further understanding of motor neuronal degeneration in ALS.

Ethical Committee or Institutional Animal Care and Use Committee Approval: Ethical approvals to the study involving animals were given by the Committee for Animal Experiments of the University of Szeged, Szeged, Hungary I. 74-II/2015 and the Biological Research Center of the HAS (approval No. 72-45/b/2001 and No. 03876/0014/2006) and carried out in accordance with the national law (XXVIII. chapter IV. paragraph 31) which conforms to the international law and policies (EEC Council Directive 86/609, OJL 358 1 DEC. 12, 1987; revised 1996). Ethical approvals for obtaining and storing blood samples were approved by The Human Investigation Review Board, University of Szeged, Hungary. (#2557/2009).

Grants and financial support: This work was financially supported by the GINOP-2.3.2.-15-2016-00001, the GINOP-2.3.2.-15-2016-00034 and GINOP-2.3.3-15-2016-00001 programs and the „Foundation for the Future of Biomedical Sciences in Szeged” program with the financial aid of the Ministry of Human Capacities (34232-3/2016/INTFIN). Our work was also supported by the UNKP-18-2 New National Excellence Program of the Ministry of Human Capacities.

Acknowledgements: We thank Erika Rác Bánfiné for her assistance in histochemical and electron microscopic work.

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production

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Fungal evolutionary studies
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Transgenic mice as disease models
Tumor pharmaco- an
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drug testing in insects
Genetic analysis in disease
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Experimental motor neuron disease induced in mice with long-term injections of serum from ALS patients

Valéria Meszlényi^{1,2}, Izabella Obál^{3,4}, Roland Patai¹, Bernát Nógrádi^{1,2}, Tamás F. Polgár¹, Gerda Ricken⁵, Gabor G. Kovacs⁵, Kornélia Tripolszki⁶, Márta Széll⁶, József I. Engelhardt³, László Siklós¹

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Role of a conserved glutamic acid in the catalytic mechanism of type VI sulfide:quinone oxidoreductases

Nikolett Miklovics^{1,2}, Ildikó Dósa², Ágnes Duzs^{1,2}, Gábor Rákhely^{1,2}, András Tóth^{1,2}

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Downregulation of plasma miR-802-5p as an early biomarker for breast cancer brain metastases – possible involvement in the regulation of MEF2C

Kinga Molnár¹, Marta Sereno², János Haskó¹, Tânia Custódio-Santos², Sarah Medina³, Rui Malhó⁴, Mafalda Videira^{1,5}, Zita Reisz⁶, Stephanie Booth³, László Tiszlavicz⁶, Imola Wilhelm¹, István A. Krizbai¹, Maria Alexandra Brito^{2,7}

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Effect of Hofmeister-active salts on the conformation of the NAD⁺ and NADH cofactors: a simulation study

Zoltán Násztor, János Horváth, András Dér

Institute of Biophysics, BRC HAS, Szeged

Computational investigation of Hofmeister-active salts effect on the interfacial properties of the NAD⁺ and NADH cofactors

Zoltán Násztor, János Horváth, András Dér

Institute of Biophysics, BRC HAS, Szeged

NLRP3 activation in motor neurons after acute nerve injury

Bernát Nógrádi^{1,2}, Ádám Nyúl-Tóth^{1,3}, Roland Patai¹, Imola Wilhelm¹, István Krizbai¹, László Siklós¹

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³*Department of Geriatric Medicine, Reynolds Oklahoma Center on Aging, University of Oklahoma, Oklahoma, USA*

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Ultrastructural changes in motor neurons after acute blood serum inoculation of ALS patients with different genetic mutations

Tamás F. Polgár¹, Roland Patai¹, Valéria Meszlényi^{1,2}, Bernát Nógrádi^{1,2}, Kornélia Tripolszki³, Márta Széll³, József I. Engelhardt⁴, László Siklós¹

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³*Department of Medical Genetics, University of Szeged, Szeged*

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Lidocaine turns the surface charge of biological membranes more positive and changes the permeability of blood-brain barrier culture models

Ana R. Santa-Maria^{1,2}, Fruzsina R. Walter¹, Sándor Valkai¹, Ana Rita Brás¹, Mária Mészáros^{1,3}, András Kincses^{1,4}, Adrián Klepe¹, Diana Gaspar⁵, Miguel A. R. B. Castanho⁵, László Zimányi¹, András Dér¹, Mária A. Deli¹

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Phytotoxicity of remediated soils previously contaminated with ULOs

Tibor Sipos¹, Attila Bodor^{1,2}, György Erik Vincze¹, Gábor Feigl⁴, Naila Bounedjoum^{1,2}, Gábor Rákhely^{1,2,3}, Katalin Perei^{1,2}

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Behind the anisotropy imaging using RCM

Gábor Steinbach, Dávid Nagy, László Zimányi

Institute of Biophysics, BRC HAS, Szeged

Utilization of rumen content waste for methane production

Norbert Tolvai¹, Árpád Szilágyi¹, Zoltán Deim¹, Viktória Markó¹, Violetta Tripolszki¹, Gábor Rákhely^{1,2,3}

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²*Institute of Environmental and Technological Sciences, University of Szeged, Szeged*

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Isolation of wood industrial waste degrading fungi

Violetta Tripolszki¹, Árpád Szilágyi¹, Etelka Kovács¹, Norbert Tolvai¹, Viktória Markó¹, Violetta Tripolszki¹, Gábor Rákhely^{1,2,3}

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Microfluidic mixing: on chip synthesis of concentration arrays for cell culturing

Saeed Ur Rahman¹, Krisztina Nagy¹, Ágnes Ábrahám¹, László Dér¹, Péter Horváth², Péter Galajda¹

¹*Institute of Biophysics, BRC HAS, Szeged*

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S100A5 was observed. This secretion was receptor-dependent, as over-expression of S100A5 alone did not result in detectable S100A5 in the media. To dissect the G protein signaling pathway that mediates this secretion, inhibitors of the different G protein signaling pathways were used to treat cells that co-expressed S100A5 with either receptor. These studies suggest that calcium is involved in the secretion downstream of GPR37L1 and GPR37 signaling, as treatment with BAPTA-AM, an intracellular Ca^{2+} chelator, reduces S100A5 secretion from transfected HEK293T cells. These and future studies will help shed light on the functions of the glia-specific and seizure-protective receptors GPR37L1 and GPR37 in the brain.

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POSTER NUMBER: 3002

Diazoxide differentially reduces microglial activation in the oculomotor, facial and hypoglossal nucleus after acute nerve injury in mice

Bernát Nógrádi^{1,2}, Roland Patai¹, Valéria Meszlényi^{1,2}, Tamás F. Polgár¹, László Siklós^{1*}

¹Institute of Biophysics, Biological Research Centre of the Hungarian Academy of Sciences, Hungary; ²Szeged Scientists Academy, Hungary

Microglial cells, the resident immune competent cells of the central nervous system rapidly activate after injury. Our aim was to characterize the magnitude of the microglial activation after acute injury in motor nuclei that are known to be differently susceptible in chronic stress conditions. Furthermore, we wanted to determine the anti-inflammatory effect of a potential neuroprotective substance, diazoxide in these nuclei.

Male Balb/c mice underwent either unilateral eye enucleation, hypoglossal or facial nerve axotomy. Animals were sorted to non-treated (n=4), vehicle treated (n=4, 10 mg/ml dimethyl sulfoxide in distilled water, 0.1 ml/day ip.), prophylactically and postoperatively diazoxide treated (n=5, 7+4 days, 0.25 mg/ml in 0.1 ml/day ip.) and only postoperatively diazoxide treated (n=5, 4 days, 0.25 mg/ml in 0.1 ml/day ip.) groups. Choline acetyltransferase and Iba-1 double immunostaining was used to mark the anatomical borders of the examined motor nuclei and quantify microglial activation, respectively.

Injury of the motor axons resulted in an increase in the density of microglial cells in each observed nucleus (p<0.001), however, microglial activation was less prominent in the oculomotor nucleus compared to the other nuclei (p<0.01). Diazoxide reduced microgliosis in all examined motor nuclei, but this effect was more prominent if the treatment was started prophylactically (p<0.001).

Diazoxide could reduce microglial activation in each examined nucleus either if applied prophylactically, or only postoperatively. The moderate microglial activation in the oculomotor nucleus might be interpreted as the consequence of the reduced release of proinflammatory signal chemokine (C-C motif) ligand 2 in motor neurons, which was demonstrated in our previous experiments.

This work was supported by the Szeged Scientists Academy (34232-3/2018/INTFIN) and the UNKP-18-2 New National Excellence Program of the Hungarian Ministry of Human Capacities.

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POSTER NUMBER: 3037

Validation of a Microglia-Specific NuTRAP Model for Paired Epigenomic and Transcriptomic Analysis of Neuroinflammation

Sarah Ocañas^{1,2}, Ana Chucair-Elliott^{1,2}, Victor Ansere^{1,2}, Kyla Buettner^{1,2}, David Stanford^{1,2,3}, Willard Freeman¹⁻⁵
Department of Physiology¹, Reynolds Oklahoma Center on Aging², Oklahoma Nathan Shock Center for Aging³, Oklahoma Center for Neuroscience⁴ at the University of Oklahoma Health Sciences Center, OKC, OK; Oklahoma City VA Medical Center, OKC, OK⁵

As the sentinel macrophages of the CNS, microglia monitor the CNS for foreign and endogenous substances. Generally, microglia retain a quiescent, surveillance phenotype. Stimulation can cause maladaptive pro-inflammatory phenotypic switching, characteristic of neurological diseases. Regulation of gene expression involves DNA modifications, principally methylation (mC) and hydroxymethylation (hmC) of cytosines. Our hypothesis is that microglial plasticity is regulated by changes in DNA modifications; however, paired epigenomic and transcriptomic studies have not been possible due to technical issues. To address this barrier, we developed a microglial-specific, NuTRAP mouse model that combines Translating Ribosome Affinity Purification (TRAP) and Isolation of Nuclei Tagged in Specific Cell Types (INTACT) methods. The NuTRAP cassette labels nuclei (RanGAP1)

OFFICIAL CERTIFICATE OF PARTICIPATION

Fourteenth - Young European Scientist - Meeting

The Organizing Committee of the 14th YES Meeting declares that

Valéria Éva Meszlényi

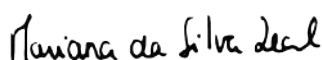
presented the work

Alteration in motor neuronal calcium level induced by acute serum inoculation of
amyotrophic lateral sclerosis patients with genetic mutations

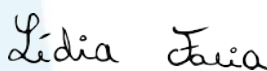
Valéria Éva Meszlényi 1, 2, Roland Patai 1, Bernát Nógrádi 1, 2, Tamás Ferenc Polgár 1, Izabella Obál 3, 4, Márta Széll
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1 - Institute of Biophysics, Biological Research Centre, Hungarian Academy of Sciences, Szeged, Hungary, 2 - Foundation for the Future of Biomedical
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Department of Neurology, University of Szeged, Szeged, Hungary, 5 - Department of Medical Genetics, University of Szeged, Szeged, Hungary

In the Neurosciences Poster Session in the **Fourteenth - Young European Scientist - Meeting**, which took place on September 12th-15th, 2019, at CIM - Centro de Investigação Médica, FMUP - Faculdade de Medicina da Universidade do Porto, Portugal.



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Vice President of the 14th YES Meeting



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Modulation of the inflammasome-mediated crosstalk between motor neurons and microglia using a K_{ATP} channel activator, diazoxide, in differently susceptible brain regions

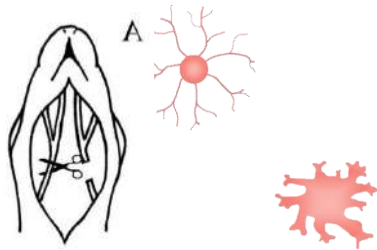
Rebeka Kristóf³, Bernát Nógádi¹, Ádám Nyúl-Tóth², Valéria Meszlényi¹, Tamás F. Polgár³, Roland Patai³, Imola Wilhelm³, István Krizbai³, László Siklós³

1: Biological Research Centre, Department of Biophysics; Szeged Scientist Academy

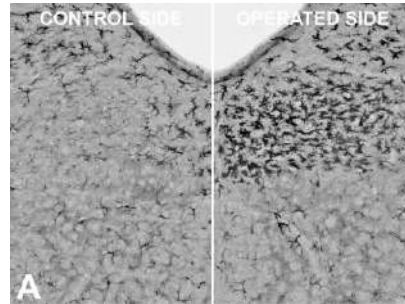
2: Biological Research Centre, Department of Biophysics; Vascular Cognitive Impairment and Neurodegeneration Program, Reynolds Oklahoma Center on Aging/Department of Geriatric Medicine, University of Oklahoma Health Sciences Center

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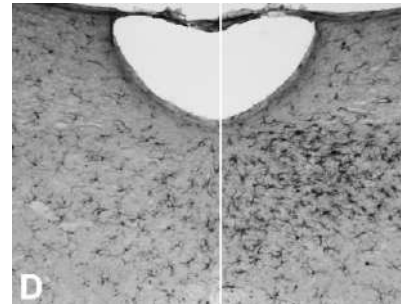
Introduction



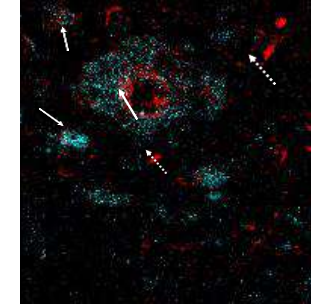
Microglia activation after hypoglossal axotomy



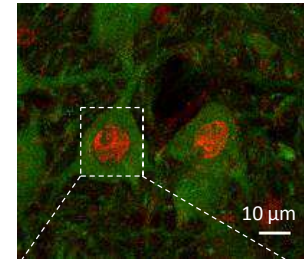
Hypoglossal axotomy + DZX



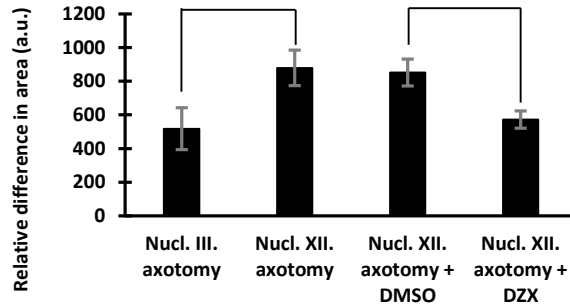
NLRP3/ASC merged



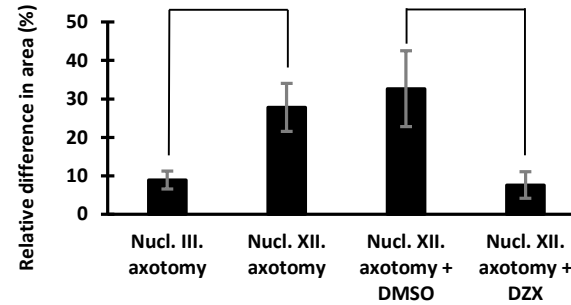
NLRP3/ChAT merged - Unoperated



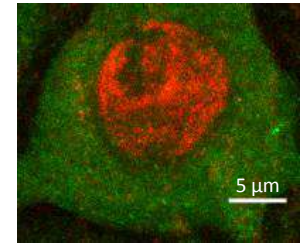
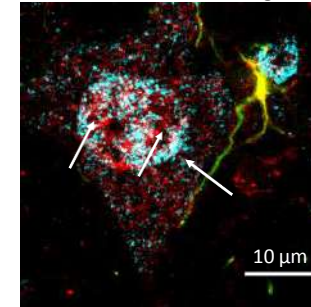
Microglial activation



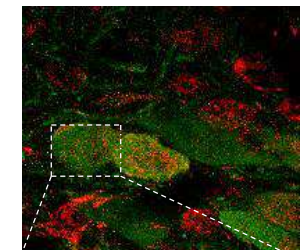
Change in NLRP3 expression



NLRP3/ASC/GFAP merged



NLRP3/ChAT merged - Operated



NLRP3 expression after hypoglossal axotomy



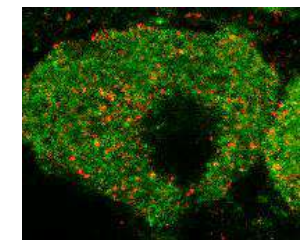
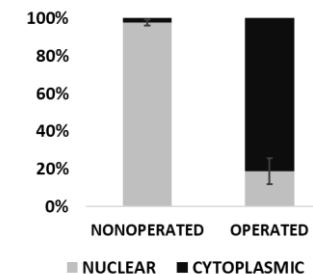
Hypoglossal axotomy + DMSO



Hypoglossal axotomy + DZX



Translocation of NLRP3 in neurons



Spatiotemporal changes of the calcium homeostasis in Schwann cells after sciatic nerve axotomy



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