



**SNN
2026**

The Annual Meeting of
the National Neuroscience
Society of Romania

SNN2026 Conference

Anniversary Event - 25 Years Since Launching SNN
28-30 May 2026 - Bucharest, Romania

ABSTRACT BOOK

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SNN2026

The Annual National Neuroscience Society of Romania (SNN) Conference with International Participation

Date: 28 - 30 May, 2026

Venue: Bucharest, Romania

Faculty of Medicine, Carol Davila University of Medicine and Pharmacy

Faculty of Biology, University of Bucharest

The Annual National Neuroscience Society of Romania (SNN) Conference with international participation SNN2026 was a fantastic opportunity to celebrate the 25th years of SNN activity by engaging with cutting-edge neuroscience research, exchanging ideas with leading experts, and exploring the latest advancements in experimental and clinical neuroscience. During the 8 sessions and 6 workshops, SNN2026 conference covered exciting topics such as brain circuits, neurodegeneration, neuroprotection, neurorecovery, vision, neuropathic pain, gut-brain axis, computational neuroscience.

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SNN2026 Conference Program

Thursday, 28 May 2026

09:00 – 15:00 **Workshop Sessions for Young Investigators & Early-Career Researchers**

@ The Faculty of Biology, University of Bucharest

09:00 – 12:00 – **1. Spinal Nerve Ligation Induced Neuropathic Pain Model in Rodents: model design and outcome measures** (Roxana-Olimpia Gheorghe)

This workshop will help participants understand the key components of experimental design for animal models of neuropathic pain, using the core model design proposed by Chung in 1992. The session will present the ligation procedure for the L5 spinal nerve (target species, ligation method, approach, sham operation, type of anesthesia) with critical considerations, variations, and improvements, and outcome measures (mechanical allodynia, thermal hyperalgesia).

09:00 – 11:45 – **2. The shapeshifting microglia: advanced bioimage analysis of the cytoskeleton** (Melania Maria Magercu)

This workshop will present quantitative image analysis concepts and tools for reliable detection of linear and point-like structures individually, and their cross-correlation in cells. The demonstration will explore F-actin and total α -tubulin cytoskeletal rearrangements in microglia via immunofluorescence staining. The participants will learn how to build an automated pipeline for quantitative image analysis using cultured HMC3 cells exposed to IFN γ and TNF α .

12:00 – 14:45 – **3. Application of the patch-clamp technique in the study of electrophysiological properties of nervous system cells** (Bogdan Amuzescu)

This workshop will introduce the participants to the patch-clamp technique and its applications. The practical demonstration will cover the essential steps of a manual patch-clamp experiment: the experimental setup, preparation of a patch pipette, whole-cell recordings in HEK293 cells stably expressing hNaV1.5 channels, and analysis of multi-step recordings to explore voltage-dependent activation and inactivation.

@ The Faculty of Medicine, Carol Davila University of Medicine and Pharmacy, Bucharest

09:00 – 15:00 – **1. EEG in Research Workshop. EEG data processing – an introduction to CARTOOL** (Mihai Moldovan, Miralena Tomescu)

The EEG in Research Workshop – EEG Data Processing: An Introduction to CARTOOL, organized by Prof. Miralena Tomescu in collaboration with Prof. Mihai Moldovan and the Brain Products Team, includes 1) a live hands-on demonstration of EEG data collection using a mobile EEG system provided by Brain Products (SNN2026 sponsor); 2) an introduction to CARTOOL, hands-on pre-processing session covering filtering, re-referencing, interpolation, and downsampling; 3) Microstates Analysis, with further hands-on practice; 4) a round table and discussion of the principles of EEG, data acquisition, and limitations, followed by conclusions and a Q&A session. This workshop aims to familiarize participants with both good practices in EEG data collection and EEG data processing using CARTOOL – a versatile, user-friendly, freely available software developed at the University of Geneva and now internationally used, requiring no coding skills. All welcome, whether you are a newcomer curious about starting with EEG or an experienced user looking to expand your data processing toolkit.

10:00 – 13:00 – **2. EBRAINS Computational Neuroscience Workshop** (Carmen Lupascu)

The workshop will focus on tools and instruments for building and simulating biophysically detailed models of single neurons and for visualization of large, full-scale point-neuron circuits, with the main aim to extend access to this field and related tools to the neuroscientific communities. After the workshop, participants will have a deeper understanding of the techniques and the scientific methods underlying the implementation of detailed computational models.

14:00 – 16:00 **Opening of the onsite registration and poster mounting @ [Faculty of Medicine, Carol Davila University of Medicine and Pharmacy, Bucharest](#)**

16:00 – 17:00 **Session 1 – Chair Ana-Maria Zăgrean**
Opening Ceremony. SNN Anniversary Event – 25th years from launching SNN!

17:00 – 19:15 **Session 2 – Chairs Aurel Popa, Andrei Miu**
Andrei Miu – Neural mechanisms of trauma and psychopathology (Cognitive Neuroscience Laboratory, Department of Psychology, Babeş-Bolyai University, Cluj-Napoca, Romania)
Raul C. Muresan – Waving neurons: How brain oscillations can be used in practical applications and therapy (Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania)
Carmen Lupascu – Multiscale modeling of mouse, rat and human hippocampal CA1 networks (Institute of Biophysics, National Research Council, Palermo, Italy)
Andrei Ilie - Humans 3.0: Brain, Intelligence, and Evolution in the Age of AI (Newcastle Upon Tyne, United Kingdom)
Nicolae Popescu, Aurel Popa - Dr. Ștefan Odobleja's Contribution to the Concept of Artificial Intelligence (Drobeta-Turnu Severin, Romania)

19:30 – 21:30 **Welcome social event @ ["Dimitrie Brândză" Botanical Garden, University of Bucharest](#)**

Friday, 29 May 2026

8:30 – 9:00 **Onsite registration and poster mounting**

09:00 – 10:30 **Session 3 – Chairs Mihai Moldovan, Daniela Popa**
Tibor Szilágyi – Molecular and cellular changes in temporal lobe epilepsy: focus on calcium-binding proteins, glial cells and modulation by antiepileptic drugs (George Emil Palade University of Medicine, Pharmacy, Science, and Technology of Târgu Mureș, Romania)
Irina Oane, Ioana Mîndruță – Functional mapping of orbitofrontal cortex with intracranial electrodes (Epilepsy and Sleep Monitoring Unit, Emergency University Hospital Bucharest; Carol Davila University of Medicine and Pharmacy, Bucharest, Romania)
Mihai Moldovan – The value of EEG reactivity as a translational outcome measure after hypoxic ischemic brain injury (Department of Neuroscience, University of Copenhagen)
Miralena Tomescu – Spontaneous EEG microstates signatures of brain disorders and clinical states risk, diagnostic and predictive biomarkers (Faculty of Educational Sciences, University "Ștefan cel Mare" of Suceava, Suceava, Romania)

10:30 – 10:45 **Coffee break**

10:45 – 11:30 **IBRO Keynote Lecture – Hannah Monyer** - Spatial Coding: Local and Remote Control (Department of Clinical Neurobiology, Heidelberg University Hospital, DKFZ, Germany)

11:30 – 12:30 **Session 4 – Young investigators blitz presentations (posters and oral blitz) – Chair Tibor Szilágyi**
PO1. **Alexandra Mirela Mocanu** - Oxytocin modulates EEG Microstate dynamics during Social Imitation to influence affective states and promote bonding
PO2. **Bella Khlyzova** - Age-Related Variability in EEG Microstates and Their Associations with Cognitive and Socio-Emotional States
PO3. **Tiana Voicu** - Trauma, Resilience, and Creativity: A Mixed-Methods EEG Microstate Study
PO4. **Răzvan-Alexandru Șteopoaie** - Current direction and intensity differentially modulate TMS-evoked EEG responses and corticospinal conduction latency
PO5. **Alexandru George Rusneac** - Empathy and emotion regulation: Linking neural, behavioral, and real-life measures
PO6. **Adelina Păduraru** - Developing a new behavioral task for testing thermal object memory
PO7. **Andra-Maria Dobrin** - A multimodal investigation of emotional interference in working memory
PO8. **Didina-Cătălina Barbălată** - Maternal Dysbiosis and Perinatal Asphyxia: Effects on Maternal Behavior and Neurodevelopmental Reflexes in Rat Offspring

12:30 – 13:30 **Round table – AI in Neuroscience research. How to integrate national and international resources to advance Neuroscience Education and Research in Romania**

Moderators: Andrei Ilie, Andrei Miu, Mihai Moldovan, Hannah Monyer, Aurel Popa, Daniela Popa, Leon Zăgrean

13:30 – 14:00 **Lunch break**

14:00 – 15:10 **Poster & Networking Session**

15:10 – 15:20 **Group Photo**

15:20 – 17:30 **Session 5** – Chair Ana-Maria Zăgrean

Siobhain O'Mahony – Nurturing the Gut-Brain Axis in Menopausal Transition (University College Cork, Ireland)

Grațîela Grădișteanu Pîrcălăbîoru – Microbes and moods: unraveling the gut-depression connection (Bucharest University, Bucharest, Romania)

Daniela Popa – Cerebellum in defensive behaviors (Institut de Biologie de l'École Normale, Paris, France)

Aurel Popa - Genetic conversion of astroglia cells to redress neural balance after stroke (Craiova University of Medicine and Pharmacy, Craiova, Romania)

Mario Scheel-Werner - Microscopy-guided proteomics at organelle-level resolution has the power to reveal unknown proteins in disease or functional-specific regions (Director Business Development EMEA, Syncell Inc., USA - Sponsor presentation)

17:30 – 17:45 **Coffee break**

17:45 – 19:30 **Session 6 – IBRO Early-Career Researchers Session I** – Chair Andrei Ilie

Mara Ioana Ionescu – Prenatal programming of the brain: The role of maternal microbial communities on the neurodevelopment of the offspring (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania)

Sebastian Isac – Pro and anti-apoptotic pathways in the vulnerable brain exposed to anesthesia – an experimental approach (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania)

Milorad Dragić – The GluN2A subunit is necessary for intermittent theta burst stimulation-induced hippocampal plasticity (University of Belgrade, Faculty of Biology, Belgrade, Serbia)

Rosana-Bristena Ionescu – Decoding Neurodegenerative Vulnerability – Multiomics Signatures of Inflammaging and Resilience Loss (University of Cambridge, Cambridge, United Kingdom)

Miruna Rascu – Continuous emotional evidence integration following ketamine (Oxford Centre for Integrative Neuroimaging (OxCIN), Department of Experimental Psychology, University of Oxford, United Kingdom)

Robert Mihai Haret – Modelling spatio-temporal contrast adaptation in retinal ganglion cells (University Medical Centre, Göttingen, Germany)

Mihai Stancu – Noise-induced reduction and early recovery of superior paraolivary nucleus sound-offset responses (Faculty of Biology, Ludwig-Maximilians-Universität, München, Germany)

19:30 – 20:30 **SNN General Assembly**

Saturday, 30 May 2026

09:00 – 10:30 **Session 7** – Chair Violeta Ristoiu

IBRO Keynote Lecture – Marc R. Suter - Hyperexcitability after peripheral nerve injury, which fibers modulate microglia? (Pain Center, CHUV & DNF, Faculty of Biology and Medicine University of Lausanne (UNIL), Lausanne, Switzerland)

Alexandru Babeș – Statins activate temperature-gated Transient Receptor Potential ion channels (University of Bucharest, Bucharest, Romania)

Tudor Șelescu - Probing the limits of TRPM8 multimodality (University of Bucharest, Bucharest, Romania)

10:30 – 10:45 **Coffee break**

10:45 – 12:30 **Session 8 – IBRO Early-Career Researchers Session II** – Chair Alexandru Babeș

Alexandru Deftu – Neuroimmune interactions in neuropathic pain: a focus on neuronal and non-neuronal extracellular vesicles profiling and their effects on DRG neuronal excitability, macrophages and spinal microglia (Pain Center, CHUV & DNF, Faculty of Biology and Medicine University of Lausanne (UNIL), Lausanne, Switzerland)

Violeta Caragea – The functional expression of thermosensitive ion channels in the cerebellum (University of Bucharest, Bucharest, Romania)

Gabriela Marcu – Wired but tired: A resting EEG study of insomnia-related cortical hyperactivation in depression (Lucian Blaga University of Sibiu and Clinical Psychiatric Hospital, Sibiu, Romania)

Andela Šoškic - How does a community-developed reporting standard become standard? The case of ARTEM-IS in EEG research (University of Belgrade, Belgrade, Serbia)

Mihai Simedrea - Can EEG Revolutionize Education? A Neuroadaptive Framework for the First Generation of Digital Natives (Timișoara, Romania)

Michael Bogdan Mărgineanu – Neural characteristics and nerve interplay in small cell lung cancer (Victor Babeș University of Medicine and Pharmacy, Timișoara, Romania)

Ianis Kevyn Ștefan Boboc – Targeted Lipid Nanoparticles Carrying Induced Pluripotent Stem Cell-Derived MicroRNAs as a Novel Therapy for Ischemic Stroke (Craiova University of Medicine and Pharmacy, Craiova, Romania)

12:30 – 12:40 **Coffee break**

12:40 – 14:00 **Session 9** – Chair Beatrice Radu

Delia Gheorghe - *Childhood adversity and resting-state functional connectivity: a population-based discovery and replication study in the UK Biobank* (Transylvanian Institute of Neuroscience, Cluj-Napoca, Romania)

Beatrice Radu – *Blood-brain barrier: the bridge between health and disease* (University of Bucharest, Bucharest, Romania)

Bogdan Pavel – *Clastrum modulation impact on the anesthetic state* (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania)

Bogdan Amuzescu – *Neuronal oscillators: synchrony analysis of a thalamo-cortical network* (University of Bucharest, Bucharest, Romania)

14:00 – 14:30 **Concluding remarks, Awards, and Closing Ceremony**

List of Poster only

P1. **Melisa Asan** - *The influence of aging on color perception and sensibility of contrast*

P2. **Georgiana Balea** - *Emotion regulation in daily life and telomere length: The role of inflammation and autonomic regulation*

P3. **Ana-Maria Catrina** - *The probiotic potential to alleviate offspring brain damage caused by maternal antibiotic exposure – a histological study*

P4. **Cristian Ciotei** - *Is memory affected in adult rats by exposure to perinatal asphyxia and gestational antibiotic-induced dysbiosis?*

P5. **Alexandra Ciubotaru** - *Toward adaptative language-based psychometric assessment*

P6. **Ciprian Constantinescu** - *Neural Tokenisers*

P7. **George Cristian Georgescu** - *Ultrasound guided optic nerve sheath diameter as a proxy for neuroinflammation*

P8. **Monica Giurgiu** - *The effects of the menthol applied on the skin, on the sympathetic - vagal balance in the young adults*

P9. **Melania Magercu** - *Exploring binding interactions in microglial lipid-sensing receptors and CSF lipidome ligands in Multiple Sclerosis: in silico characterization*

P10. **Marius Mihai Pastiu** - *Assessing the Impact of Semaglutide on Anxiety-Like Behavior and Cognitive Function in a Mouse Model of Alzheimer's Disease*

P11. **Laurențiu Tofan** - *Brain networks associated with visual hallucinations evoked by direct cortical stimulation exposure during embryological development*

ORAL PRESENTATIONS

O1. Neural mechanisms of trauma and psychopathology

Andrei C. Miu¹

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Exposure to traumatic events is a risk factor for multiple mental disorders, including posttraumatic stress disorder (PTSD). However, little is known about the mechanisms that drive the onset of PTSD following trauma – or, in other words, about the neural and psychological differences between individuals exposed to trauma who develop PTSD and those who are resilient and maintain their mental health. Two candidate psychological mechanisms are empathy and emotion regulation, which may contribute to vulnerability to PTSD through heightened emotional reactivity and altered capacity to regulate emotional responses to traumatic events. In this talk, I will present recent results from a functional magnetic resonance imaging (fMRI) study that investigated differences in neural activity during empathy and emotion regulation in individuals exposed to traumatic events and controls. I will also illustrate the associations between neural activity during these processes and PTSD symptoms, and outline implications for the development of new interventions to prevent PTSD in individuals exposed to trauma.

O2. Dr. Ștefan Odobleja's Contribution to the Concept of Artificial Intelligence

Nicolae Popescu¹, Aurel Popa²

¹SC Psycho-Soma Med SRL, Drobeta-Turnu Severin

²University of Medicine, Rostock, Germany; University of Medicine, Craiova, România

This article demonstrates that the Romanian scientist Ștefan Odobleja (1902-1978), in his work "Consonantist Psychology", elaborated the principles and laws that are at the basis of artificial thinking. The fascination with artificial intelligence began last century, when humanity was very far from the technologies without which we cannot imagine life today. Artificial intelligence refers to the phenomenon of a machine acting like a human mind. The history of artificial intelligence can include the scientist Ștefan Odobleja as the father of artificial thinking, which is in fact generalized cybernetics, as described in his work.

O3. Molecular and cellular changes in temporal lobe epilepsy: focus on calcium-binding proteins, glial cells and modulation by antiseizure drugs

Tibor Szilágyi¹, Károly Orbán-Kis¹, Ádám Szentes¹, Zsolt András Nagy¹, Ágnes Csüdör¹, Anna Fehér¹, Zsolt Gáll², Eszter Horváth³, István Katona^{3,4}, Krisztina Kelemen^{1,3}

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³Molecular Neurobiology Research Group, HUN-REN Institute of Experimental Medicine, Budapest, Hungary

⁴Department of Psychological and Brain Sciences, Indiana University Bloomington, Bloomington, IN, United States

Temporal lobe epilepsy (TLE) is associated with molecular and cellular remodeling that extends beyond the hippocampus into additional brain regions implicated in seizure generation and propagation. Using the kainic acid rat model of chronic TLE, this study investigates alterations in calcium-binding protein-defined neuronal populations, glial changes, and the effects of the SV2A-targeting antiseizure drugs levetiracetam and brivaracetam. Selected hippocampal and extra-hippocampal regions relevant to the epileptic circuitry were studied, including the amygdala, paraventricular thalamic nucleus (PVT), endopiriform nucleus, piriform and perirhinal cortices, and hippocampal subfields. Beyond established interneuronal markers such as parvalbumin, calretinin, and calbindin, significant epilepsy-associated alterations were observed in the less explored NECAB family proteins. NECAB1-positive neurons were particularly abundant in the PVT, endopiriform nucleus, and amygdala under physiological conditions, while chronic epilepsy induced significant NECAB1 upregulation in the PVT and bilaterally in the amygdala. NECAB2- and calbindin-expressing populations also showed selective region-dependent reorganization, with distinct modulation by levetiracetam and brivaracetam. Significant increase in microglial density was observed in the brivaracetam-treated epileptic group and in some brain-regions of the untreated epileptic animals compared with sham-operated controls and levetiracetam-treated animals. Notably, under epileptic conditions, brivaracetam treatment was associated with increased microglial density across all examined brain regions, in contrast to levetiracetam. Increased astrocyte density was also detected in the vulnerable regions, particularly in the hippocampus, coinciding with a reduction of neuronal density. These findings highlight selective neuronal and glial plasticity in chronic TLE and suggest that calcium-regulatory mechanisms and neuroinflammatory responses may contribute to treatment-specific effects beyond seizure suppression.

O4. Gut Feelings Gone Wrong: Microbiome Dysbiosis in Major Depressive Disorder

Gratiela Gradisteanu Pircalabioru^{1,2,3}

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²Bio-Hub Centre of Excellence in Bioengineering, National University of Science and Technology POLITEHNICA Bucharest,

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Major depressive disorder (MDD) is a complex neuropsychiatric condition increasingly associated with alterations of the gut microbiome and disruption of the microbiota–gut–brain axis. The present study investigated intestinal microbial signatures associated with MDD using sequencing-based microbiome profiling approaches in depressive patients and healthy controls. Microbial community analysis revealed significant dysbiosis in individuals diagnosed with MDD, characterized by alterations in both microbial diversity and taxonomic composition. At the phylum level, depressive patients exhibited an increased abundance of members belonging to *Pseudomonadota* (formerly *Proteobacteria*) and reduced representation of beneficial taxa from the phylum *Bacillota* (formerly *Firmicutes*), particularly butyrate-producing bacteria involved in intestinal homeostasis. Among the most prominent findings was the enrichment of taxa belonging to the family *Enterobacteriaceae* in the MDD group. Expansion of these Gram-negative bacteria is associated with elevated lipopolysaccharide (LPS) production, intestinal barrier dysfunction, and activation of systemic inflammatory pathways. Increased microbial-derived inflammatory signaling may contribute to neuroinflammation through cytokine-mediated mechanisms involving IL-6, TNF- α , and IL-1 β , ultimately influencing neurotransmitter metabolism and neuronal function. In contrast, sequencing analysis demonstrated reduced abundance of *Faecalibacterium prausnitzii*, a key anti-inflammatory commensal bacterium and major producer of short-chain fatty acids, particularly butyrate. Depletion of this species may impair epithelial barrier integrity and reduce anti-inflammatory metabolite production, further contributing to gut permeability and chronic low-grade inflammation frequently associated with depressive disorders.

Beta diversity analysis suggested distinct microbial clustering between depressive individuals and healthy controls, supporting the presence of disease-associated microbial signatures. Additionally, increased interindividual variability observed in MDD patients highlighted the heterogeneity of depressive disorders and suggested the existence of microbiome-defined depressive subtypes. Overall, the results support the hypothesis that major depressive disorder is associated with significant gut microbial dysbiosis involving pro-inflammatory expansion and depletion of beneficial commensal bacteria. These findings reinforce the role of the microbiota–gut–brain axis in depression pathophysiology and highlight the potential of microbiome-based biomarkers and microbiota-targeted therapeutic strategies for precision psychiatry.

O5. Prenatal programming of the brain: The role of maternal microbial communities on the neurodevelopment of the offspring

Mara Ioana Ionescu^{1,2}; Ana-Maria Catrina³; Cerasela Haidoiu³; Vladimir Suhăianu³; Cristian Ciotei²; Didina-Catalina Barbalata²; Ioana Dogaru²; Ana-Maria Zăgrean²

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Introduction: Maternal gut microbiome dysbiosis is increasingly implicated in altered fetal neurodevelopment and heightened susceptibility to neurological injury, particularly in conditions such as perinatal asphyxia (PA). Prenatal antibiotic exposure disrupts maternal microbiota and may adversely affect both maternal behavior and offspring neurodevelopment. This study investigated the effects of gestational antibiotic-induced microbiome disruption on maternal behavior, offspring neurodevelopment, and vulnerability to PA, along the role of probiotic supplementation. Materials and Methods: Pregnant Wistar rats received either an antibiotic cocktail from embryonic day 11 or a multi-strain probiotic throughout gestation. Fecal samples were collected at embryonic day 20 for microbiota analysis. On postnatal day (PND) 6, pups were exposed to PA or normoxia, generating eight experimental groups. Maternal anxiety- and depressive-like behaviors were assessed after weaning. Offspring neurodevelopmental reflexes were evaluated between PND7–9, while cognitive and affective behaviors were assessed from PND35 using the Barnes Maze, Y-Maze, Novel Object Recognition, and Forced Swim Test. Results: Antibiotic exposure reduced beneficial bacterial taxa, including *Bifidobacterium*, *Faecalibacterium*, *Bacteroides*, and *Butyricoccus*, while increasing *Fusobacterium*. Antibiotic-treated dams exhibited anxiety- and depressive-like behaviors that were not prevented by probiotics. Offspring exposed to prenatal antibiotics showed impaired early reflexes, exacerbated by PA and only partially improved by probiotics. Cognitive deficits and depressive-like behaviors were similarly aggravated following PA. Conclusion: Gestational microbiome disruption impaired maternal behavior and offspring neurodevelopment, increasing vulnerability to PA-related neurological injury. Probiotic supplementation provided only partial neuroprotection, highlighting the importance of maintaining maternal microbiome.

Funding. This study was funded by the Sectoral Research and Development Program of the Romanian Ministry of National Defense (PSCD NEURO_Depress).

06. Pro and anti-apoptotic pathways in the vulnerable brain exposed to anesthesia - an experimental approach

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Background: Perinatal asphyxia (PA) remains a major cause of neonatal morbidity and mortality and is associated with adverse neurodevelopmental outcomes. Although general anesthesia (GA) is frequently required in pediatric care, its effects on the immature brain previously exposed to PA are not fully understood. This study investigated epigenetic modulation and apoptosis-related signaling in the immature hippocampus following PA and/or GA exposure. **Methods:** Wistar rats were exposed to either 90 minutes of asphyxia or normoxia on postnatal day (PND) 6, followed by 180 minutes of sevoflurane anesthesia or normoxia on PND-15. Hippocampal tissue was collected from four experimental groups (n=15/group). Total RNA was extracted and analyzed by quantitative reverse transcription polymerase chain reaction (qRT-PCR). Expression levels of selected miRNAs (miR-15a-3p, miR-16-3p, miR-34a-3p, and miR-146-3p) and apoptosis-related genes (MAPK1, MAPK3, TP53, CREB1, and BCL2) were evaluated. **Results:** Exposure to GA significantly increased miRNA expression irrespective of prior PA exposure. GA was also associated with downregulation of the anti-apoptotic genes CREB1 and BCL2, while no significant changes were detected in MAPK1, MAPK3, or TP53 expression. **Conclusions:** Sevoflurane anesthesia induced persistent epigenetic alterations in the immature hippocampus, characterized by CREB-dependent transcriptional dysregulation and impaired neuronal survival signaling rather than activation of DNA damage-mediated apoptotic pathways. Prior PA did not exert additive long-term effects, suggesting that GA alone may disrupt this regulatory axis, whereas PA-induced oxidative stress may contribute minimally to long-term anesthesia-related neurotoxicity.

07. Modelling Contrast Adaptation in Retinal Ganglion Cells

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The visual system adapts, i.e. it adjusts its sensitivity, in order to accurately represent incoming stimuli of varying dynamic ranges. Early in the visual pathway, retinal neurons modulate their activity patterns based on the basic features of the visual input: the mean and variance of the stimulus. In the retina, contrast adaptation (the change in sensitivity as a function of stimulus' variance) is most prevalent in retinal ganglion cells (RGCs). Upon a change in stimulus contrast, the RGCs change their average firing rate, modify their temporal filtering properties and adjust their gain and sensitivity. Using in vitro extracellular multi-electrode array recordings of mouse RGCs, I analyse spiking responses to spatially homogeneous white-noise stimuli of multiple (discrete and continuous) contrast levels. Firstly, I describe changes characterized by contrast adaptation by

employing the linear-nonlinear (LN) model. Under the LN model, each cell is assumed to have a temporal filter, estimated by spike-triggered averaging, that integrates the stimulus linearly. The output is passed through an identified nonlinearity to yield the neuron's instantaneous firing rate. While not accounting for adaptation, the LN model can highlight the differences in sensory encoding under different contrast regimes. Building upon the LN, in order to fully capture the aspects of contrast adaptation within a single model, I fit the nonlinear input model (NIM). The NIM is made up of multiple linear filters with corresponding nonlinearities whose outputs are then pooled in a subsequent downstream nonlinearity that generates the predicted firing rate. Responses predicted by the NIM reproduce the contrast-dependent changes recorded during the recordings of the retina. The NIM proves to be a robust model that, unlike the LN, captures additional nonlinear processing in RGCs that account for contrast adaptation.

08. Complex effects of statins on peripheral nociceptors

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Statins are HMG-CoA reductase inhibitors administered to decrease the levels of cholesterol and lower the risk of cardiovascular disease. Although statins are relatively well tolerated, adverse effects such as myalgia and painful peripheral neuropathy have been reported. Moreover, statins have numerous pleiotropic effects, including anti-inflammatory and analgesic actions in several animal pain models. Here we report that some of the most extensively used statins activate certain temperature-gated transient receptor potential (TRP) ion channels. All tested statins (simvastatin, atorvastatin and rosuvastatin) activate human TRPA1 and, in addition, simvastatin activates human TRPV1 and rosuvastatin activates human TRPM8. The activation of TRPV1 by simvastatin is abolished in a capsaicin-insensitive mutant. Furthermore, the sensitivities of both TRPV1 and TRPA1 to simvastatin are diminished in triple cysteine mutants known to exhibit a reduced redox sensitivity. Rosuvastatin-induced activation of TRPM8 requires an aspartate residue which is essential for sensitivity to the synthetic TRPM8-agonist icilin. In mouse dorsal root ganglion (DRG) neurons, simvastatin activates a subpopulation of neurons which also respond to capsaicin (a TRPV1 agonist) and/or allyl isothiocyanate (a TRPA1 agonist). In addition, TRPM8 plays an important role for the activation of a small population of DRG neurons by rosuvastatin. Taken together, these results indicate that statins activate thermo-sensitive TRP channels expressed in nociceptive sensory neurons, which may explain some of the pleiotropic effects of these widely used drugs.

O9. Probing the limits of TRPM8 multimodality

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The TRPM8 ion channel is an essential receptor in the somatosensory system of animals, from amphibians to mammals, having the special property of cooling-gating. Defensive animal behaviors such as jumping and vigorous shaking are triggered by extreme cold exposure. To investigate the role that TRPM8 plays in these responses, efficacious agonists of this channel are required. One strategy of testing the efficacy of TRPM8 agonists is to make use of the channel's thermal and chemical modalities. Whole-cell currents were recorded in HEK-293T cells expressing mammalian (rat and human) or avian (chicken) TRPM8 orthologs, while superfusing the cells with solutions of TRPM8 agonists, including menthol, WS-12 and Cryosim-1. During chemical stimulation, the extracellular temperature was gradually decreased (60 s ramps from ~32 to 18 °C). At -80 mV, the inward currents showed an increase followed by a local minimum found at the minimum of the temperature ramp. The Q10 coefficients for the decreasing currents ranged from 1.2 to 1.3 in all TRPM8 orthologs, similar to the values for open channel conductance in other ion channels. These results show that at the channel's maximum open probability, as a result of simultaneous agonist and cold stimulation, further cooling decreases the inward currents in a diffusion-limited manner. This allows selecting the most efficacious TRPM8 agonists, namely the agonists that elicit current decreases at the highest temperatures during descending temperature ramps. For the agonist-ortholog pairs tested, Cryosim-1 was the most efficacious at the chicken ortholog, while WS-12 at the rat and human TRPM8 orthologs.

O10. How does a community-developed reporting standard become standard? The case of ARTEM-IS in EEG research

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Underreporting of methodological detail in EEG publications is a well-documented obstacle to reproducibility and meta-analytic synthesis, with key methodological information about study design, preprocessing, analysis models or statistical results often impossible to extract from journal articles. ARTEM-IS is a community-developed tool that addresses this by providing a structured questionnaire/reporting template for EEG methods that can be completed during research and shared alongside the publication, or even retrospectively for published research. The resulting human- and machine-readable reports can serve as a checklist for writers, support completeness assessment by editors and reviewers, and facilitate methods extraction by meta-analysts. This presentation will introduce the problem, describe ARTEM-IS from the user's perspective, and turn to the harder question: once a community-developed tool has been designed and published, how does it become a standard? Three challenges will be addressed: perceived burden, limited motivation under a status quo that journals tolerate while researchers are pressed to move on to the next project, and the difficulty of reaching the EEG community at scale from a grassroots starting point. I will describe ongoing work on each: the COST Action EEG101 (WG1: Reporting Standards) supporting development and outreach, dialogue with journals about endorsement, and tool development that reduces friction and increases usability. Finally, the talk will consider a challenge necessary for long-term success of any standard: how to design transferable governance so that the tool outlives the people who started it.

O11. Targeted Lipid Nanoparticles Carrying Induced Pluripotent Stem Cell-Derived MicroRNAs as a Novel Therapy for Ischemic Stroke

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Current ischemic stroke therapies are mainly limited to vessel recanalization/reperfusion during the first hours after onset, highlighting the need for new therapeutic strategies. Here, we describe the development of bio-inspired lipid nanoparticles (LNPs) designed to deliver miRNAs and target post-stroke inflammation, a major contributor to secondary brain injury. miRNAs were isolated from human induced pluripotent stem cells and encapsulated into LNPs produced using a microfluidic flow technique. This method generated small, uniform nanoparticles with high miRNA encapsulation efficiency. To enhance targeting of the cerebral endothelium, anti-transferrin receptor (TfR) and anti-E-selectin antibodies were incorporated into the lipid layer. We evaluated LNP effects in models of vascular inflammation *in vitro* and brain ischemia *in vivo*. In cultured human brain endothelial cells, empty LNPs (E), dual-targeted empty LNPs (DT-E), and dual-targeted miRNA-loaded LNPs (DT-LNPs) significantly reduced lipopolysaccharide-induced IL-6 production, suggesting an intrinsic anti-inflammatory effect of the lipid carrier. In mice subjected to transient middle cerebral artery occlusion, we assessed empty particles (control) and two DT-LNP doses (DT-Low and DT-Hi). DT-Low reduced infarct volume and tended to improve neurological scores compared to control and DT-Hi. Moreover, DT-Low significantly downregulated IL1 β and E-selectin mRNA expression compared with DT-Hi. Overall, these findings provide proof-of-concept that TfR/E-selectin-targeted LNPs encapsulating iPSC-derived miRNAs represent a promising therapeutic approach for ischemic stroke. Further optimization of lipid composition, dosage, cargo selection, and mechanistic evaluation is required to maximize efficacy.

O12. Childhood adversity and resting-state functional connectivity: a population-based discovery and replication study in the UK Biobank

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Experiences of childhood adversity are major risk factors for later psychopathology. While neurodevelopmental research suggests specific brain networks may be particularly sensitive to adversity exposure, underlying neural mechanisms remain unclear. We addressed this using an epidemiologically inspired population-scale approach with UK Biobank resting-state functional connectivity MRI data (N=62,246; 55% female; M(age)=54.5; SD(age)=7.4). Associations between five childhood adversity domains and 210 resting-state functional connections (21-node ICA parcellation) were tested using linear regression models adjusted for demographic and imaging confounds, with false discovery rate (FDR) correction. Discovery analyses were conducted in first-wave imaging participants (N=58,021), requiring associations to replicate across two adversity assessments before testing in an independent second-wave imaging sample (N=4,225). Sensitivity analyses additionally

adjusted for depressive and anxiety symptoms. Fifty-three adversity-connectivity associations met the predefined robustness criterion in the discovery sample, spanning all adversity domains. Effects were small ($|\beta|=0.01-0.04$) and implicated distributed network-level patterns involving fronto-parietal, default mode, salience and subcortical networks. Among the strongest associations was fronto-parietal-default mode connectivity for emotional neglect ($\beta=0.04$, $p_{\text{corrected}}=8.9 \times 10^{-10}$). Three associations replicated under FDR correction, including emotional neglect with fronto-parietal-default mode connectivity ($\beta=0.07$, $p_{\text{corrected}}=0.005$). This connection also remained robust following additional adjustment for depressive and anxiety symptoms. Childhood adversity was associated with modest but distributed differences in functional connectivity, with a small subset showing reproducibility across adversity assessments, imaging samples and sensitivity analyses, supporting subtle but reproducible network-level correlates of adversity.

O13. Neuronal oscillators: synchrony analysis of a thalamo-cortical network

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Mathematical modeling of neurons and neuronal networks is a rapidly evolving field, due to the impetus provided by large-scale approaches such as the Human Brain Project. Thalamo-cortical (TC) circuits represent a particularly interesting domain with essential roles in sensory information processing and instantaneous memory. The pioneering studies of Mircea Steriade via in-vivo recordings revealed several TC rhythms, and his coworker Alain Destexhe explored by modeling many network configurations including TC and thalamic reticular (RE) neurons. We chose for analysis a network including two TC and two RE single-point neurons described in Destexhe et al. 1996 J. Neurophysiol 76(3). The model, built in C++, included two TC neurons with different densities of hyperpolarization-activated current, leading to spontaneous fast action potentials (AP) bursts on the plateau of T-type calcium "slow spikes" with a frequency of ~4 Hz corresponding to thalamo-cortical theta EEG rhythms. We varied the scaling factors (relative to default) of TC->RE AMPA synapses (scgsyn1) and RE->TC GABA A&B synapses (scgsyn2), and we built several mathematical descriptors of burst synchrony: a function inspired from quantum relative entropy, and a 4x4 synchrony matrix using the discrete Hilbert transform via a python script based on parallel simulations. Qualitatively, when scgsyn1 is small (0-0.03), the TC neurons function independently and RE neurons are hyperpolarized, at 0.04 TC and RE neurons emit quasisynchronous bursts, and at higher values (>0.06) RE neurons feature high-frequency tonic spiking. At scgsyn2>0.35 the first TC neuron is inhibited by RE tonic spiking, resembling the spindle/slow wave-induced TC hyperpolarization in sleep.

Young investigators blitz presentations (posters and oral blitz)

PO1. Oxytocin modulates EEG Microstate dynamics during Social Imitation to influence affective states and promote bonding

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Previously, we showed that resting-state after social Imitation (SI) showed different mind-wandering patterns and large-scale network dynamics that support bonding and relaxed states. In this study, we analyzed 128-channel EEG data from 42 healthy volunteers while performing a 3-minute dyadic SI task and a matched non-SI control task. Salivary oxytocin (OXT) was collected before and after each task, alongside assessments of mind wandering (Amsterdam Resting-State Questionnaire, ARSQ) and perceived stress levels. EEG microstate analysis identified seven canonical microstates (A–G). During SI, microstate D showed a significant decrease across all temporal parameters ($p<0.037$). Microstate G exhibited reduced time coverage ($p=0.032$), global explained variance (GEV; $p=0.012$), and occurrence ($p=0.029$), whereas microstate E showed increased occurrence during SI ($p=0.043$). EEG reactivity analyses during SI relative to baseline $[(\text{PRE}-\text{STIM})/\text{PRE} \times 100]$ revealed increased reactivity of microstate E coverage ($p<0.001$) and microstate C duration ($p=0.008$) and coverage ($p=0.04$), alongside decreased reactivity of microstate A duration ($p<0.001$) during SI. Moderation analyses indicated that OXT significantly shaped the relationship between microstate dynamics and post-SI affective states. Smaller OXT changes were associated with stronger links between increased microstate E activity during SI and elevated stress ($p=0.012$). Additionally, lower post-SI OXT levels were associated with reduced microstate G activity predicting decreased discontinuity of mind-wandering ($p=0.012$). Together, these findings support a hormonally modulated neurocognitive mechanism through which SI alters large-scale brain network dynamics and mind-wandering, promoting bonding.

PO2. Age-Related Variability in EEG Microstates and Their Associations with Cognitive and Socio-Emotional States

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Aging is associated with changes in cognitive functioning, emotional processing, and large-scale brain dynamics. The present study employed EEG microstate analysis to examine resting-state temporal

brain dynamics and characterize global patterns of neural activity linked to cognitive and socio-emotional functioning in healthy older adults. While EEG microstate alterations have been widely studied in clinical populations, their relationship with cognitive performance and subjective experience in healthy aging remains insufficiently explored. Resting-state EEG recordings were obtained from 63 healthy elderly participants and clustered into seven canonical microstates (A–G). Microstate parameters, including duration and occurrence, were analyzed in relation to psychometric measures assessing global cognition (MMSE), spontaneous mentation (ARSQ), affective experience (SAM), and social reward sensitivity (SRQ). Significant sex-specific differences emerged in social sensitivity and microstate dynamics: females showed greater social sensitivity and longer durations of microstates B and F, whereas males exhibited increased occurrence of microstates D and E. In addition, Partial Least Squares correlational analyses identified a significant latent variable ($r = 0.63$, $p = 0.017$) linking microstate dynamics to cognitive and socio-emotional traits. Higher MMSE scores, emotional arousal, dominance, and social sensitivity were positively associated with microstates that exhibited longer durations in females, such as B and F. In contrast, microstates more present in males, such as microstates D and E, showed negative associations with MMSE, emotional experience, and social sensitivity. These findings highlight EEG microstates as sensitive, non-invasive markers of sex differences and their relation to mental status and socio-affective functioning in aging.

PO3. Trauma, Resilience, and Creativity: A Mixed-Methods EEG Microstate Study

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Psychological resilience represents a protective factor against adversity, potentially enhancing creativity. This study examined the interplay among trauma, emotion regulation, resilience, post-traumatic growth, and creativity using a mixed design combining self-report measures in 187 young adults, with repeated EEG recordings across creative (Alternative Uses Task, AUT), cognitive (Serial Subtraction Task, SST), and resting-state (RS) conditions in a subsample of 76 participants. Hierarchical regression identified depression, anxiety, and emotion regulation difficulties as negative predictors of resilience, while well-being, post-traumatic growth, and creative self were positive predictors. Moderated mediation revealed that emotion regulation difficulties indirectly predicted creative self through resilience, moderated by post-traumatic growth across all levels. EEG microstate analyses revealed robust differences between AUT and RS. AUT showed increased segment density in microstate B (auditory/language), with largest effects in microstate C (DMN) and D (DAN), but decreased density in microstate A (visual). Large reductions were found in mean duration across microstates A, B, C, and F (SN). Compared to SST, AUT showed selective effects, with increased microstate C density, decreased microstate F density, and reduced mean duration in microstates E and F. Partial Least Squares analyses linked microstates A, B, and C to trauma, stress and emotion dysregulation, while microstates C, E, and F were associated with resilience, well-being, and creative self. In conclusion, resilience may function as a psychobiological bridge between trauma and creativity, behaviorally through links with post-traumatic growth and neurally through the engagement of microstate C, a neural marker potentially associated with the creative processing of lived adversity.

PO4. Current direction and intensity differentially modulate TMS-evoked EEG responses and corticospinal conduction latency

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Introduction: Transcranial magnetic stimulation (TMS) current direction influences corticospinal recruitment, but its effects on cortical TMS-evoked potentials (TEPs) remain unclear. We examined how anterior–posterior (AP) and posterior–anterior (PA) stimulation, delivered at direction-specific intensities, affect cortical and corticospinal responses. **Methods:** Sixteen participants underwent combined TMS-EEG and EMG recordings over the primary motor cortex. Four conditions were tested in a 2×2 within-subject design: stimulation direction (AP, PA) and intensity calibration, with AP- and PA-specific intensities adjusted to evoke approximately 1 mV motor evoked potentials (MEPs). TEPs were analysed within three predefined time windows (18–35 ms, 35–73 ms, and 73–269 ms) using spatiotemporal cluster-based permutation tests of direction, intensity, and direction $\times$ intensity effects. MEP latency was estimated from rectified EMG using a baseline-threshold method and analysed with repeated-measures ANOVA. **Results:** TEPs showed a significant direction $\times$ intensity interaction in the 18–35 ms and 35–73 ms windows, primarily driven by direction-dependent cortical responses at PA-calibrated intensity. In the 73–269 ms window, no interaction but significant main effects of direction and intensity were observed. MEP latency showed significant independent effects of direction and intensity without interaction: AP stimulation produced longer latencies than PA stimulation. No correlations between stimulation-direction effects in TEP amplitude and corresponding stimulation-direction effects in MEP latency survived correction for multiple comparisons. **Conclusion:** Current direction and intensity differentially influence TMS-evoked cortical responses, while corticospinal latency is predominantly direction-dependent, suggesting partially dissociable cortical and corticospinal effects of AP and PA stimulation.

PO5. Empathy and emotion regulation: Linking neural, behavioral, and real-life measures

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Empathy and emotion regulation are central to social behavior and mental health, shaping how individuals respond to others' distress and manage their own emotional reactions. Both processes have been extensively studied in cognitive neuroscience, using self-report questionnaires, behavioral tasks, and functional magnetic resonance imaging (fMRI). However, it remains unclear how these different levels of assessment relate to one another: do neural and behavioral

responses observed in the laboratory map onto how people experience and regulate emotions in daily life? The present study investigated associations between neural, behavioral, and self-report measures of empathy and emotion regulation. Participants, including individuals exposed to traumatic events and controls, completed two fMRI sessions. During scanning, they performed the EmpaToM, a task assessing empathic responses, and an emotion regulation task assessing the ability to downregulate negative emotion using reappraisal and distraction. In addition, participants reported on empathy and emotion regulation in their daily life over one week, using ecological momentary assessment. This talk will present findings on how laboratory-based neural and behavioral measures relate to real-life emotional functioning. By integrating fMRI, behavioral performance, self-report questionnaires, and ecological momentary assessment, the study highlights the complexity and complementarity of different approaches to measuring empathy and emotion regulation, with implications for understanding vulnerability and resilience following trauma.

PO6. Developing a new behavioral task for testing thermal object memory

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Thermal associative memory reflects the ability to form and recall associations between objects or contexts and their thermal properties. This study aims to investigate the ability of mice to form thermal associative memories by using a new behavioral task based on free exploration of objects found at different temperatures.

One experimental session consisted of two trials: training and test, separated by a defined inter-trial time interval (ITI). For the training, the mice (C57BL/6) were placed in an arena with two objects at different temperatures – either room temperature (RT, ~23°C) and cold (10°C or 15 °C), or warm (32°C) and cold (15°C). During the test, both objects had the same temperature (RT or 32°C). A visual cue was present in the arena during both trials. The effect of various training trial durations, object temperatures, and ITIs were tested, and the exploration time of each object was quantified (object sniffing, leaning and sitting). The thermal preference was quantified as a discrimination index for the warmer object (DI). Preliminary results show that during the training trial, the preference for warmer temperatures is higher (DI>0) when training lasts longer (20+ min), or when the temperature difference between the two objects is higher (>13°C). However, the capacity of the animals to recognize the temperature changes at test was visible when the training duration was shorter (10 min).

Altogether, these results support that mouse can form associative memories using the temperature of objects as a basis for learning.

PO7. A multimodal investigation of emotional interference in working memory

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Air Working memory ensures the temporary storage and active manipulation of information necessary for ongoing cognitive operations. Emotional content competes for its limited attentional resources, and this competition is further amplified in individuals with elevated anxiety. According to attentional control theory, anxiety impairs the filtering task-irrelevant distractors, with interference effects mostly pronounced in the presence of emotionally negative stimuli. However, positive emotional distractors have also been reported to bias working memory under certain conditions. A multimodal approach investigating individual factors linked to anxiety could support a better understanding of these complex mechanisms. A preliminary sample of 14 healthy volunteers (18-25 years) completed a 2-back computerized memory task with letters superimposed on standardized emotional images (neutral, pleasant, unpleasant), while eye movements and electrodermal activity (EDA) were simultaneously recorded. Participants subsequently rated image arousal and valence and completed standardized anxiety questionnaires. EDA increased substantially above the resting baseline in 12 out of 14 participants ($\Delta\text{EDA} = +22.16 \pm 3.48 \mu\text{S}$, $p < 0.001$), with homogenous baseline levels across participants ($32.38 \pm 1.41 \mu\text{S}$). Emotional stability significantly moderated the effect of emotional valence on letter fixation duration ($\beta = -0.36$, $p = 0.021$), with less stable participants showing greater attentional disruption caused by unpleasant stimuli ($r = -0.52$, $p = 0.041$). These preliminary findings suggest that individual personality traits, rather than state anxiety alone, modulate attentional and physiological responses to emotional interference in working memory, supporting the validity of the proposed multimodal protocol.

PO8. Modulatory effect of Tumor Suppressor Candidate 5 on Transient Potential Receptor Channels

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Tusc5 (Tumor suppressor candidate 5) is a cold suppressed gene expressed in adipose tissue (AT) involved in adipogenesis. Tusc5 is also present in dorsal root ganglion (DRG) neurons, which receive information from the AT and are involved in thermosensation. Considering the cold sensitivity of Tusc5 and its presence in both AT and DRGs, it is possible that it could be involved in shared regulatory functions of these tissues. The transient potential receptor (TRP) family includes thermosensitive ion channels expressed in DRGs and are essential for detecting changes in ambient temperature. Investigating the effect of Tusc5 on these channels could shed new light on interactions between the adipose tissue and peripheral temperature-sensing neurons. To assess the modulatory effect of Tusc5 on TRP channels, we used primary DRG neuron cultures from knock-in Tusc5eGFP ♂/♀ heterozygote and homozygote mice, fed

with a normal or high fat diet. Using ratiometric fura-2 calcium imaging, we measured the activation of endogenous TRPM8, TRPA1, TRPM3 and TRPV1 channels when challenged with specific agonists. Experimental data suggests that Tusc5 is differentially expressed in distinct populations of temperature-sensitive DRG neurons: Tusc 5 is absent from DRG neurons that express TRPM8, thus involved in cold sensing, while it is abundant in DRG neurons sensitive to TRPA1, TRPV1 and TRPM3 agonists, involved in heat sensing. Moreover, a high fat diet leads to amplified activation of all TRP channels. Taken together, our data indicates that Tusc5 expression identifies different populations of DRG neurons involved in detecting changes in ambient temperature.

PO9. Maternal Dysbiosis and Perinatal Asphyxia: Effects on Maternal Behavior and Neurodevelopmental Reflexes in Rat Offspring

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Introduction

A growing body of evidence indicates that the maternal gut microbiota plays an important role in the neurodevelopment process of the offspring and influences early behavioral outcomes. Furthermore, perinatal asphyxia represents a significant cause of long-term neurological impairment. The current study explores the impact of maternal gut dysbiosis through gestational antibiotic administration (GAA) on maternal and offspring behavior, respectively the impact of perinatal asphyxia (PA) on early-life reflexes. Materials and methods Wistar rat females (n=52) were divided into 8 study groups depending on the exposure to antibiotics, probiotics and offspring PA. The GAA was initiated on the 11th gestational day using vancomycin, ampicillin, neomycin and meropenem. Probiotics were administered throughout the gestation period. PA exposure was performed on the sixth postnatal day (PND). The offsprings' neurodevelopment was assessed through early-life behavioral tests in PND 7-9: righting reflex, negative geotactic reaction, and grip strength response. The impact of microbiota interventions on maternal behaviour was analyzed through Elevated Plus Maze (EPM) and Forced Swim Test (FST). Results GAA determined impaired offsprings' reflexes in both normoxia and PA groups, compared to Control groups ($p < 0.05$). Groups that received both antibiotics and probiotics showed a slight performance improvement. Antibiotic-treated dams exhibited signs of anxiety and depressive-like behaviors, which probiotic supplementation could not alleviate. Conclusion. These preliminary results highlight the link between maternal microbiota and offsprings' neurodevelopment. Although probiotics show potential in mitigating dysbiosis- and PA-related neurological issues, more research is needed to elucidate the mechanisms both on a systemic and cellular level.

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POSTER SESSION

P1. The influence of aging on color perception and sensibility of contrast

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The loss of visual acuity primarily characterizes the aging process of the eye. However, in everyday practice, there are many other specific aspects that influence the quality of life and autonomy of patients. The purpose of this study is to evaluate the effect of aging on factors that influence vision quality, such as contrast sensitivity and color perception, for subjects who do not have visible ocular conditions. This observational study was conducted on a cohort of 66 participants aged between 20 and 80 years. The clinical evaluation included standardized tests to measure contrast sensitivity and color vision. The data were analyzed to highlight functional dynamics. Using Pearson and Spearman tests. Correlations were identified between age and the decline of visual parameters. To confirm significant differences between age groups, the ANOVA test was applied. Results showed a progressive decline in contrast sensitivity as age groups advanced, with contrast sensitivity being the most vulnerable parameter. Color perception was also affected and became problematic after the age of 60, while age groups 20-40 years and 41-60 years maintained an excellent ability to distinguish shades. Parameters such as high blood pressure and diabetes were also monitored, as they showed a negative impact. The study concluded that physiological aging leads to a significant decline in vision quality. The use of statistical tests emphasizes the need for a personalized approach in ophthalmological screening for the older patients. Integrating these evaluations into current clinical practice can allow for early diagnosis of functional deficit.

P2. Preparation Emotion regulation in daily life and telomere length: The role of inflammation and autonomic regulation

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Exposure to increased levels of stress has been associated with accelerated telomere erosion. However, little is known about the role of emotion regulation, that is, the processes through which individuals modulate the experience and expression of emotion. Emotion regulation has been shown to modulate neuroendocrine responses to stress, and we hypothesized that it could also influence telomere erosion. In the present study, we aimed to investigate the association between emotion regulation in daily life and telomere length. The sample includes a homogeneous group of young adults who do not smoke or report frequent alcohol use, as these behaviors are known to accelerate telomere erosion and could confound the effects of stress and emotion regulation. Emotional experience and the use of multiple emotion regulation strategies were assessed using ecological momentary assessment (EMA) over seven days. Telomere length will be assessed from genomic DNA extracted from

peripheral blood samples using quantitative real-time PCR (qPCR), both at baseline, before the start of the EMA period, and after one year. This relatively long follow-up period is necessary to allow sufficient time for changes in telomere length to occur. We also assessed interleukin-6 (IL-6), a proinflammatory cytokine that has been linked to telomere erosion. Furthermore, we assessed heart rate variability (HRV) during rest and emotional challenge in order to characterize autonomic regulation, which may be related to emotion regulation. We will present the design of this study and preliminary results focusing on associations between individual differences in emotion regulation, symptoms of psychopathology, IL-6, and HRV.

P3. The Probiotic Potential to Alleviate Offspring Brain Damage Caused by Maternal Antibiotic Exposure – a Histological study

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Introduction. Maternal exposure to broad-spectrum antibiotics during gestation has been increasingly associated with adverse neurodevelopmental outcomes in offspring, potentially mediated through alterations in maternal and neonatal gut microbiota composition and disruption of the microbiota–gut–brain axis. The present study aimed to evaluate the neuroprotective effects of probiotic supplementation in preventing and attenuating brain structural damage induced by maternal antibiotic exposure and perinatal asphyxia in rat offspring. **Materials and methods.** Experiments were performed on Wistar rat offspring obtained from dams assigned to four gestational treatment groups: control, antibiotics, antibiotics combined with probiotics, and probiotics alone. All animals were maintained under standard laboratory conditions with unrestricted access to food and water. Maternal dysbiosis was induced beginning on embryonic day 11 through administration of an antibiotic cocktail consisting of ampicillin, vancomycin, neomycin, and meropenem. A multi-strain probiotic formulation was administered throughout pregnancy. On postnatal day 6, pups were subjected either to perinatal asphyxia or normoxic conditions, resulting in eight experimental groups. Brain samples collected on postnatal day 75 were processed for histopathological analysis using hematoxylin and eosin staining. **Results.** Histological examination revealed that offspring exposed to perinatal asphyxia exhibited vascular congestion, edema, neuronal pyknosis, and hippocampal and cerebellar neuronal degeneration. Maternal antibiotic exposure exacerbated these neuropathological alterations, even under normoxic conditions. In contrast, probiotic administration markedly reduced the severity of brain lesions. **Conclusion.** Probiotics provide protective roles mediated through modulation of the microbiota–gut–brain axis and restoration of neurodevelopmental homeostasis.

P4. Is memory affected in adult rats by exposure to perinatal asphyxia and gestational antibiotic-induced dysbiosis?

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Chronic exposure to stressors during gestation is linked to different neurodevelopmental problems. We employed a rodent model of maternal dysbiosis and mild perinatal asphyxia to evaluate whether the offspring's spatial memory is affected in adulthood. Furthermore, the use of probiotics was investigated as a putative protective factor. **Materials and Methods:** Four experimental groups were formed: a control, an antibiotic group (receiving a cocktail of antibiotics beginning on gestational day 11), a probiotic group, and an antibiotic and probiotic group. Post-natal day 6 pups were exposed to either normoxia or asphyxia, and the groups were further divided into a perinatal asphyxia group and a normoxia group. The Y-Maze Spontaneous Alternation (YMSA) test and a 5-day Barnes Maze Test (BMT) were used to evaluate memory in adult offspring. The process of scoring the behaviour was automatised using DeepLabCut and Simba (Simple Behavioral Analysis) programs. **Results:** Our preliminary results indicate that memory, as evaluated by the YMSA test and BMT, is not affected by maternal dysbiosis, by mild perinatal asphyxia, or the combination of these two conditions. **Conclusions:** Under our experimental conditions, gestational dysbiosis and mild perinatal asphyxia do not appear to impair spatial or short-term memory performance in adulthood. However, more severe perinatal hypoxia or dysbiosis conditions should be explored, as these could overcome the early developmental recovery potential and trigger deleterious effects on memory. **Funding.** This study was funded by the Sectoral Research and Development Program of the Romanian Ministry of National Defense (PSCD NEURO_Depress).

P5. Toward adaptative language-based psychometric assessment

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Advances in large language modelling have created new opportunities for psychometric assessment based on naturalistic language use. Conventional psychometric instruments rely on fixed items and standardized norms, which may limit sensitivity to subtle cognitive changes and introduce bias related to educational level, literacy, and professional background. Language modeling approaches enable the analysis of spontaneous speech and free-text responses, offering rich markers of cognitive functioning across semantic, lexical, syntactic, and discourse levels. In the context of neurocognitive disorders, such methods show particular promise for the early detection of mild cognitive impairment and prodromal dementia, where alterations in lexical diversity, semantic coherence, narrative structure, and ease of lexical and conceptual evocation often precede overt functional decline. Additionally, impairments in the retrieval and contextualization of landmark events embedded in an individual's sociocultural space may provide sensitive indicators of disruption in autobiographical and semantic memory systems. Furthermore, language model-driven adaptive testing allows assessment tasks to be dynamically adjusted to an individual's educational attainment and to vocabulary domains associated with specific professions, reducing construct-irrelevant variance and improving diagnostic accuracy. By tailoring prompts, difficulty, and semantic content while preserving underlying cognitive targets, language-based assessments may enhance both sensitivity and validity. This work reviews emerging psychometric frameworks integrating language modelling for cognitive assessment, discusses validation and ethical challenges, and outlines pathways toward personalized, longitudinal, and clinically relevant measurement systems for cognitive decline.

P6. Neural Tokenisers

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Fixed-grain tokenisation in large language models conflicts with evidence that biological cognitive systems parse continuous input into variable, semantically coherent chunks driven by prediction error. Standard approaches such as Byte Pair Encoding determine segment boundaries by subword co-occurrence frequency statistics computed over a training corpus, producing units that reflect distributional regularities rather than semantic structure. We present a learned encoder-decoder that detects concept boundaries via prediction-error-like dissimilarity signals in a state-space model, compressing 100–200 byte spans into dense concept vectors from which original content is recovered at median BLEU 0.93 and median edit distance 0.59%. These results establish the feasibility of semantically coherent concept-level compression at biologically plausible chunk sizes, and motivate evaluation frameworks beyond surface reconstruction fidelity toward metrics capturing the semantic quality of learned boundaries.

P7. Ultrasound guided optic nerve sheath diameter as a proxy for neuroinflammation

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Background: Monitoring of intracranial pressure (ICP) and its relation to neuroinflammation remain major challenges in neurocritical care. The optic nerve sheath, an extension of the subarachnoid space, undergoes morphometric changes in response to cerebrospinal fluid (CSF) pressure fluctuations. This study evaluates the longitudinal utility of point-of-care ultrasound (POCUS)-derived optic nerve sheath diameter (ONSD) as a dynamic marker of intracranial compliance across acute cerebral pathologies. Methods: In this prospective observational cohort study, adult patients with acute neurological disorders, including ischemic stroke, meningitis, encephalitis, and metabolic encephalopathy, underwent serial bilateral ONSD assessments during hospitalization. Measurements were standardized at 3 mm posterior to the globe using a 9–10 MHz linear transducer. Correlative analyses were performed between ONSD and transcranial Doppler-derived middle cerebral artery pulsatility index (PI), neurological severity scores (GCS, RASS), CSF pleocytosis, and serum C-reactive protein (CRP) levels. Results: Interim analysis demonstrated significant associations between ONSD and neurological status. ONSD inversely correlated with GCS scores and positively correlated with TCD-derived PI, suggesting an association between optic nerve sheath distension and altered cerebral hemodynamics. In infectious pathologies, increased ONSD paralleled elevations in CSF leukocytosis, supporting a potential relationship with neuroinflammatory edema. Conclusions: Serial ONSD monitoring provides a reproducible, non-invasive bedside assessment of intracranial dynamics. Integration of longitudinal ONSD measurements into multimodal neuromonitoring may facilitate earlier detection of secondary brain injury and support individualized neuroprotective strategies.

P8. The effects of the menthol applied on the skin on the sympathetic-vagal balance in young adults

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During daily activities, minor or mild mentally stressful events may stimulate the autonomic nervous system, changing the sympathetic-vagal balance and influencing the cardiovascular physiology. Heart rate variability analysis is a non-invasive approach used to evaluate the autonomic cardiac regulation. The parasympathetic cardiac depressant effects are beneficial in stressful situations and involve stimulation of the motor nucleus of the vagus nerve. In the present study, we used menthol, an agonist of the cold receptor TRPM8, to trigger parasympathetic effects in a group of 10 healthy young male adults. A menthol solution (16%) was applied to the skin of the forehead and posterior cervical regions. The electrocardiogram was recorded weekly in two different sessions, with/without menthol applied before the mental mathematical stress test, and the short-term heart rate variability was analyzed. The recording was organized in two parts: the first part, as a control, with the subject relaxed with the eyes closed, and the second as a test, consisting of some mathematical calculus, started 10 min after menthol exposure. The data acquisition and analysis were performed by means of Biopac Student Lab 4.1 software. Our results show a significant drop in the sympathetic activity, associated with an increase in the vagal activity following menthol application. In conclusion, our data indicate that menthol has a positive impact on the subjects' stress task management.

CaragP9. Exploring binding interactions in microglial lipid-sensing receptors and CSF lipidome ligands in Multiple Sclerosis: in silico characterization

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Microglia are highly sensitive to lipid disturbances, mediating cell debris clearance towards repair in demyelinating disorders, such as Multiple Sclerosis (MS). Yet, specific lipid-protein binding interactions and ligand recognition on the microglial surface remain largely unexplored in MS pathophysiology. Here, we undertook a dual in silico approach to study the binding interactions of cell membrane receptors in microglia and lipid ligands identified in cerebrospinal fluid (CSF) samples from MS patients, with regard to the underlying mechanisms of inflammation and demyelination. Firstly, molecular docking scores (binding affinity and inhibition constant) for the CSF lipidome ligands and lipid-sensing receptors with validated biological function in microglia were simulated for each target-ligand complex in AutoDock Vina, using literature-known active sites for docking analysis. Secondly, other lipid-protein assemblies using the CSF-selected lipid ligands were collected from BioDolphin database; proteins with experimentally validated role in microglia for demyelination or inflammatory responses were selected via Human Protein Atlas and systematic literature search. A network of protein-protein interactions (PPI) was further built and clustered stochastically using MCL algorithm. PPI network analysis has revealed 2 main protein clusters from a lipid-centric perspective. First cluster features inflammatory response modulation in MS microglia via Prostaglandin D2 and E2 receptor subtypes in interaction with complement pathway receptor C5a. The second cluster involves interactions that regulate demyelination via lipid peroxidation in microglia (PPAR α and PPAR γ), alongside with suppression of microglia-induced inflammatory responses by lipid-sensor FFAR4 via

GLP-1R axis. Understanding microglial lipidome may hold the key to addressing the progressive nature of MS pathophysiology.

P10. Assessing the Impact of Semaglutide on Anxiety-Like Behavior and Cognitive Function in a Mouse Model of Alzheimer's Disease

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Background: Alzheimer's disease (AD), the predominant cause of dementia globally, is characterized by progressive cognitive deterioration and behavioral dysregulation, with prevalence projected to rise alongside demographic aging. This study examined whether semaglutide (SEM), a GLP-1 receptor agonist, could mitigate anxiety-like behavior and restore cognitive function in a transgenic mouse model of AD. **Materials and Methods:** Twenty mice were distributed across four groups (n = 5): wild-type vehicle-treated (WT + VEH), wild-type semaglutide-treated (WT + SEM), AD vehicle-treated (AD + VEH), and AD semaglutide-treated (AD + SEM). Over four weeks, glycemic levels and body weight were monitored. Behavioral profiling employed the Open Field Test, Novel Object Recognition Test, Social Chamber Test, and Elevated O-Maze Test. **Results:** SEM significantly reduced blood glucose in AD mice, normalizing toward wild-type levels. Recognition memory improved in SEM-treated AD animals, reflected by an increased discrimination index. Locomotor activity and exploratory behavior did not differ significantly across groups; however, SEM demonstrated a probable anxiolytic effect, most evident in the composite anxiety index, with meaningful improvement in the AD + SEM group following treatment. **Conclusions:** These findings suggest semaglutide may confer dual benefits in AD management, targeting both metabolic dysregulation and cognitive impairment. Given evidence linking metabolic disturbances to neurodegeneration, SEM represents a promising therapeutic candidate. Larger, mechanistically focused studies are warranted to validate these observations and clarify neuroprotective pathways involved.

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