SNN 2025

The Annual Meeting of the National Neuroscience Society of Romania

# The Conference of the National Neuroscience Society of Romania 2025 - SNN2025

**Brașov, May 29-31, 2025**

# ABSTRACT BOOK

FIZIOLOGIA
physiology

**Supplement I/2025**

# FIZIOLOGIA
## *physiology*




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## SNN2025 Conference – International Speakers

*Hannah Monyer* – Department of Clinical Neurobiology, Heidelberg University Hospital, DKFZ (German Cancer Research Centre)
*Kapil Bharti* - Scientific Director, National Eye Institute – NIH
*Jeffrey S. Diamond* - Scientific Director, National Institutes of Neurological Disease and Stroke - NIH.
*Luca Leonardo Bologna* - Institute of Biophysics (IBF) of the Italian National Research Council (CNR)
*Michele Migliore* - SUNY Dept. of Neurology, Downstate Health Science University, Brooklyn, USA ; Institute of Biophysics, National Research Council, Palermo, Italy
*Florin Amzica* - University of Montreal, Canada
*Mihai Moldovan* - Department of Neuroscience, University of Copenhagen
*Daniela Popa* - Institut de Biologie de l'École Normale, Paris, France
*Clément Léna* - Institut de Biologie de l'École Normale, Paris, France
*Andrei Ilie* – Newcastle Upon Tyne, UK
*Ioana Florentina Grigoraș* - Nuffield Department of Clinical Neurosciences, University of Oxford
*Mihai Stancu* - Faculty of Biology, Ludwig-Maximilians-Universität München
*Dirk Hermann* - University Hospital Essen, Essen, Germany

## SNN2025 Conference – Program

### Thursday, May 29, 2025

14:00 - 17:00 **Onsite registration and poster mounting**
14:00 - 15:30 **Workshop Session 1 -** Stereotactic injection of tracer substances in mouse tectum. *Organisers*: *Edwin Humberto Hodelin Maynard, Tudor Constantin Badea*
15:30 - 16:50 **Workshop Session 2 -** Live imaging of cellular organelles in tissue culture. *Organisers*: ***Vladimir Muzyka, Tudor Constantin Badea***

17:00 – 17:30 **Opening Ceremony**

17:30 – 19:30 ***Session 1 -*** *Chair, Ana-Maria Zăgrean*
**Luca Leonardo Bologna** (CNR-IBF, Palermo, Italy), *The EBRAINS-Italy Infrastructure ecosystem for Neuroscience Research*
**Michele Migliore** (CNR-IBF, Palermo, Italy), *Computational Modelling of neurons and networks: how it is done and how it can support experimental and clinical studies*
**Andrei Ilie** (James Cook University Hospital, Newcastle Upon Tyne, UK) - *Science 3.0*
**Susan Wu** (RWD Life Science, Sponsor Presentation) – *RWD Overall Solutions for Neuroscience Research*

19:45 – Welcome social event

### Friday, May 30, 2025

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

09:00 - 10:35 ***Session 2 -*** *Chair, Tudor Constantin Badea*
**Kapil Bharti** (National Eye Institute, Maryland, USA) - *Developing Cell Therapy for age-related*

*macular degeneration: from Bench to Bedside*

**Raluca Pascalau** (Transylvania University, Brasov, Romania) - *Gene expression profiles of human retinal ganglion cell types*

**Mihai Stancu** (Ludwig-Maximilians-Universität München, München, Germany) - *Trading speed for accuracy: the role of stress peptides at the giant synapse of the Calyx of Held*

**Bernd Müller-Zülow** (Miltenyi Biotec - Sponsor presentation) - *From Whole Organs to Spatial Biology – Total Tissue Characterization using Imaging Techniques*

10:35 - 10:50 Coffee break

10:50 – 12:40 ***Session 3 -*** *Chairs: Daniela Popa, Tibor Szilágyi*

**Hannah Monyer** (Heidelberg University, Heidelberg, Germany) - *From inhibition to neurodegenerative diseases*

**Daniela Popa** (IBENS, Paris, France) - *The cerebellum: a new player in the emotional brain*

**Clément Léna** (IBENS, Paris, France) - *Taking breaks when acquiring a motor skill is not a lazy choice*

12:40 - 12:50 Coffee break

12:50 – 14:30 – ***Session 4*** - **Young investigators blitz presentations**
*Chairs: Violeta Ristoiu, Andrei Ilie*

1. **Florian-Vintilă Armăşescu** (University of Bucharest, Bucharest, Romania) - *Mechanisms of NIR laser photobiomodulation: in vitro studies on primary sensory neurons and voltage-dependent Na+ channels*
2. **Alexandra Ciubotaru** (Alexandru Obregia Psychiatry Hospital, Bucharest, Romania) - *Towards general decision support systems in medicine*
3. **Constantin-Iulian Chiță** (University of Central Lancashire, Lancashire, UK) - *Short-term face familiarity displays greater modulatory effects for neutral expressions than for emotional ones – Findings from a mixed behavioural-electrophysiological study*
4. **Claudia-Ioana Drăghici** (Horia Hulubei National Institute for Physics and Nuclear Engineering, Măgurele, Romania) - *The neuroprotective effect of naringenin on endothelial inflammation induced by lipopolysaccharide on a BBB microvasculature model*
5. **Edwin Humberto Hodelin Maynard** (Transylvania University, Research and Development Institute, Brasov, Romania) - *Mapping Brn3b+ Cell populations of the Superior Colliculus using Brn3bCre mice*
6. **Melania Magercu** (University of Bucharest, Bucharest, Romania) - *Investigating microglial migratory properties that underlie their accumulation in multiple sclerosis*
7. **Michael Bogdan Margineanu** *(The Francis Crick Institute, London, UK) - Metabolic lactate shuttle powers electrical activity of small cell lung cancer*
8. **Alexandra Mocanu** (University of Bucharest, Bucharest, Romania) – *EEG Microstates Reactivity during Social Imitation*
9. **Maria Alexandra Nuțu** (University of Bucharest, Bucharest, Romania) - *Fine Particles, Fine Damage: Cytotoxic and metabolic effects of Diesel nanoparticles on different cell components of the central nervous system*
10. **Adelina Paduraru** (University of Bucharest, Bucharest, Romania) - *Modulatory effects of Tumor suppressor candidate 5 on Transient Receptor Potential ion channels*
11. **Gisselle Fernandez Pena** (Transylvania University, Research and Development Institute, Brasov, Romania) **-** *Exploration of the specific role of RGC-32 gene in an Experimental Autoimmune Encephalomyelitis murine model.*

12. **Aloyma Veliz Perez** (Transylvania University, Research and Development Institute, Brasov, Romania) - *Analysis of Ret gene expression in retinal ganglion cells and their brain projections using Immunofluorescence in CFP-Reporter Mice*
13. **Diana Petre** (Transylvania University, Research and Development Institute, Brasov, Romania) - *Distribution of Retinal Ganglion Cells expressing Brn3b and Brn3c in the Area Centralis of the mouse retina and their axonal projections in the brain*
14. **Miruna Rascu** (University of Oxford, Oxford, UK) - *Transcranial Focused Ultrasound Stimulation of the Human Basolateral Amygdala Modulates Emotional Biases*
15. **Matei-Alexandru Stamate** (University of Bucharest, Bucharest, Romania) - *Attractiveness-Driven Bias in Cooperative Decision-Making: An ERP Study on Neural Signatures in Social Exchange*
16. **Matei Serban** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) - *From Stability to Collapse: Rethinking Cognitive Decline as a Functional Transition in Brain Network Dynamics*

*14:30 – 15:30 Lunch break*

*15:30 - 17:00 -* ***Poster & Networking Session***

17:00 - 19:00 ***Session 5*** *- Chair, Beatrice Radu*

**Jeffrey Diamond** (National Institute of Neurological Disorders and Stroke, Maryland, USA) - *Synaptic mechanisms in the retina that shape visual signaling at night*

**Robert Haret** (University Medical Center, Göttingen, Germany) - *Modelling Contrast Adaptation in Retinal Ganglion Cells*

**George Opriță** (University of Bucharest, Bucharest, Romania) – *TRPV1-TRPV2 interactions. Less than the sum of their parts.*

**Dan Domocos** (University of Bucharest, Bucharest, Romania) - *The effects of statins on temperature-gated Transient Receptor Potential ion channels*

**Bogdan Amuzescu** (University of Bucharest, Bucharest, Romania) - *Effects of hypothermia on brain serotonergic circuits – expression levels of serotonin transporter (SERT) and receptors 5-HTR1A and 5-HTR2A*

19:00 – 19:45 **SNN General Assembly**

## Saturday, May 31, 2025

09:00 – 11:15 ***Session 6*** *– Chairs, Mihai Moldovan, Bogdan Pavel*

**Mihai Moldovan** (Copenhagen Univ., Copenhagen, Denmark) - *To burst or not to burst – an EEG reactivity model*

**Florin Amzica** (University of Montreal, Montreal, Canada) - *Coma: How deep can it get?*

**Beatrice Radu** (University of Bucharest, Bucharest, Romania) - *X-rays or protons? Unraveling their impact on brain microvascular cells for smarter brain cancer therapy*

**Ioana Florentina Grigoras** (University of Oxford, Oxford, UK) - *Non-invasive neuromodulation of deep brain circuits using a novel ultrasound stimulation system in healthy people*

**Gabriela Marcu** (Lucian Blaga University, Sibiu, Romania) - *The Role of ERP Components in Assessing Cognitive Control Dysfunctions in Adolescents with Complex Trauma*

11:15 – 11:30 Coffee break

11:30 – 14:00 ***Session 7*** *– Chairs, Bogdan Ovidiu Popescu, Aurel Popa*

**Bogdan Ovidiu Popescu** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) – *Brain cleansing system and neurodegeneration*

**Aurel Popa** (Craiova University of Medicine and Pharmacy, Craiova, Romania) - *Post-stroke genetic conversion of astrocytes into new neurons. Facts and Fantasies*
**Dirk Hermann** (University Hospital Essen, Essen, Germany) - *Role of the extracellular matrix in regulating post-ischemic brain plasticity*
**Daniel Pirici** (Craiova University of Medicine and Pharmacy, Craiova, Romania) - *Aquaporin 4 facilitation decreases amyloid burden in a mouse model of Alzheimer's disease*

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

## Poster only presentations

1. **Andrei Bordeianu** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) - *Heart Rate Variability Under Conditions of Intermittent Photic Stimulation in Anesthetized Rats*
2. **Ana Maria Catrina** (Cantacuzino National Military Medical Institute for Research and Development, Bucharest, Romania) - *Preparation and Assessment of Primary Microglia Isolated from Mixed Glia Cell Culture of Newborn Rat Pups*
3. **Cristian Ciotei** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) - *The Probiotic Usage Potential to Alleviate Depressive Behavioural Features and Anxiety in an Animal Model of Gestational Antibiotic Exposure*
4. **Vlad-Petru Morozan** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) - *Antibiotic exposure during gestation increases perinatal asphyxia severity in rodents*
5. **Vladimir Muzyka** (Transylvania University of Brasov, Research and Development Institute, Brasov, Romania) - *Neural sub-cellular localization of TUSC5/TRARG1 protein in TUSC5eGFP novel mouse line*
6. **Davide Ortolan** (National Eye Institute, Maryland, USA) **-** *Determination of non-stochastic changes of RPE organelles during cell polarization using volumetric image analysis*
7. **Ruchi Sharma** (National Eye Institute, Maryland, USA) - *Region specific iPSC-Retinal Pigment Epithelium Models Reveal Differential Sensitivity to AMD*
8. **Gontea Adina Teodora** (Transylvania University of Brasov, Research and Development Institute, Brasov, Romania) - *Retina-specific ablation of the transcription factor Pou4f1/Brn3a does not significantly impact Ipsi vs. Contralateral projection of Retinal Ganglion Cells in mice*
9. **Laurentiu Tofan** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) - *Multimodal analysis of the networks involved in producing visual symptoms*
10. **Stefan-Alexandru Tirlea** (Carol Davila University of Medicine and Pharmacy, Bucharest, Romania) - *Democratizing Sleep Research: A Cost-Effective System for Long-Term Rodent Monitoring*
11. **Florin Zamfirache** (University of Bucharest, Romania) - *Frontal transcranial direct current stimulation in moderate to severe depression: Clinical and neurophysiological findings from a pilot study*

# ABSTRACTS
## *for oral presentation*

### O1. Gene expression profiles of human retinal ganglion cell types.

***Raluca Pașcalău[1,2], Sergiu Șușman[3], Tudor Constantin Badea[1,4]***

*[1]Research and Development Institute, Transylvania University of Brașov, Romania*
*[2]Ophthalmology Clinic, Cluj County Emergency Hospital, Romania*
*[3]Department of Pathology, IMOGEN Centre of Advanced Research Studies, Cluj County Emergency Hospital, Romania*
*[4]National Center for Brain Research, Romanian Academy, Bucharest, Romania*

The molecular mechanisms involved in the specification of the retinal ganglion cell (RGC) types have been studied thoroughly in animal models, especially mice. In comparison, human RGCs are much less studied. Our aim was to identify molecular markers for human RGCs cell classes at different fetal life stages. We started with a bioinformatic screening for candidate genes using the Single Cell Portal from Broad Institute and the Seurat package in R. We then collected human fetal retinas from therapeutic abortion specimens under a written informed consent. The retina was dissected out of the eye, cut in anatomically oriented squares and vertically sectioned using a cryostat. Immunofluorescence was performed and images taken at a confocal microscope and processed using ImageJ software. Transcriptomic data show that human RGCs comprise four major types, considered to be orthologues of the four mouse alpha cell types, namely midget ON and OFF, parasol ON and OFF cells and 3 to 10 sparse cell types. We identified pan-alpha-orthologue markers such as IRX, and SCRT2 and single type markers: TBR2/EOMES for Midget ON, TBR1 for Midget OFF, CHRNA2 for Parasol ON and TLE1 for Parasol OFF cells. In addition to that, the four alpha-orthologue types could be defined by combinatorial expression of transcription factor and neurotrophin receptor families such as POU4Fs, IRXs and NTRKs. We studied their expression dynamic across fetal ages and retina eccentricities The results of this preliminary study offer a perspective on human RGC types specification, with implications in future visual function restoration therapies.

### O2. Trading accuracy for speed: Stress peptide signaling at the calyx of Held

***Mihai Stancu[1,2,3], Sara Pagella[1,2], Sebastian Bias[1], Benedikt Grothe[1,3], Jan M. Deussing[4], Ian D. Forsythe[5] and Conny Kopp-Scheinpflug1***

*[1]Division of Neurobiology, Faculty of Biology, Ludwig-Maximilians-University Munich, Großhaderner Straße 2, 82152, Planegg-Martinsried, Germany*
*[2]Graduate School of Systemic Neurosciences (GSN), Munich, Germany*
*[3]Munich Cluster for Systems Neurology (SyNergy), Munich, Germany*
*[4]Molecular Neurogenetics Laboratory, Max Planck Institute of Psychiatry, Munich, Germany*
*[5]Auditory Neurophysiology Laboratory, Department of Neuroscience, Psychology & Behaviour, College of Life Sciences, University of Leicester, Leicester, LE1 7RH, UK*

Stress significantly alters sensory perception by modulating neural circuits via the hypothalamic-pituitary-adrenal (HPA) axis. Neuropeptides such as corticotropin-releasing factor (CRF) and urocortins (UCN1, UCN2, UCN3) regulate this axis through differential activation of CRF receptors. While CRFR1 typically drives stress responses, CRFR2, activated by UCN2 and UCN3, may support stress recovery and anxiolysis. The auditory system, particularly the calyx of Held synapse in the brainstem, is a critical site for rapid threat detection and thus ideal for studying stress-related neuromodulation. Here, we show that UCN3 enhances synaptic efficacy via CRFR2 activation, increasing EPSC amplitude, transmission speed, and reliability. Pharmacological CRFR2 blockade produces inverse effects. In vivo, UCN3 knockout mice exhibit reduced auditory dynamic range, poorer spectral tuning, and faster but less efficient synaptic responses. These findings establish a functional role for UCN3-CRFR2 signaling in shaping auditory synaptic transmission and highlight its contribution to sensory adaptation towards faster, yet less accurate encoding of sound during stress.

### O3. Modelling Contrast Adaptation in Retinal Ganglion Cells

***Robert M. Haret[1,2,3], Tim Gollisch[1,2,4]***

*[1]Department of Ophthalmology, University Medical Center Göttingen, Göttingen, Germany*
*[2]Bernstein Center for Computational Neuroscience, Göttingen, Germany*
*[3]International Max Planck Research School for Neurosciences, Göttingen, Germany*
*[4]Cluster of Excellence "Multiscale Bioimaging: from Molecular Machines to Networks of Excitable Cells" (MBExC), University of Göttingen, Göttingen, Germany*

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.

## O4. The effects of statins on temperature-gated Transient Receptor Potential ion channels

***Dan T. Domocos[1], George Opriţă[1,2], Adelina Păduraru[1], Tudor Selescu[1], Andreas Leffler[4], Alexandru Babes[1], Ramona-Madalina Babes[3]***

*[1]Dep. of Anatomy, Physiology and Biophysics, Faculty of Biology, Univ. of Bucharest, Romania*
*[2]National Center for Brain Research, Research Institute for Artificial Intelligence, Romanian Academy, Bucharest, Romania*
*[3]Dep. of Biophysics, "Carol Davila" Univ. of Medicine and Pharmacy, Bucharest, Romania*
*[4]Dep. of Anesthesiology and Intensive Care, Hannover Medical School, Germany*

Statins represent a class of drugs that inhibit HMG-CoA reductase, prescribed to lower LDL cholesterol levels and reduce the risk of cardiovascular disease. Although generally well tolerated, statins have been associated with side effects such as myalgia and painful peripheral neuropathy. On the other hand, increasing evidence suggests that statins exert pleiotropic actions beyond lipid lowering, including anti-inflammatory and analgesic effects observed in animal pain models. The mechanisms underlying these off-target effects remain poorly understood. In the present study, we show that several widely used statins can activate members of the temperature-sensitive Transient Receptor Potential (TRP) ion channel family. Specifically, simvastatin and atorvastatin activate human TRPV1 and TRPA1, while rosuvastatin activates human TRPM8 in heterologous expression systems. Simvastatin's activation of TRPV1 is abolished in a capsaicin-insensitive mutant, and its effects on both TRPV1 and TRPA1 are reduced in triple cysteine mutants with diminished sensitivity to reactive oxygen species. Rosuvastatin appears to activate TRPM8 via a critical aspartate residue also required for sensitivity to the synthetic TRPM8 agonist icilin. In mouse dorsal root ganglion (DRG) neurons, simvastatin stimulates a subset of neurons responsive to TRPV1 and TRPA1 agonists, while rosuvastatin activates a smaller neuronal population that expresses TRPM8. Collectively, these findings suggest that statins activate thermosensitive TRP channels in nociceptive sensory neurons, which may contribute to their diverse pleiotropic effects.

## O5. Effects of hypothermia on brain serotonergic circuits – expression levels of serotonin transporter (SERT) and receptors 5-HTR1A and 5-HTR2A

***Andrei Negoiţă[1,2], Adela Banciu[3], Daniel Dumitru Banciu[3], Bogdan Amuzescu[1]***

*[1]Dept. Biophysics & Physiology, Faculty of Biology, University of Bucharest, Romania*
*[2]Institute for Diagnosis and Animal Health, Bucharest, Romania*
*[3]Dept. Biomaterials & Medical Devices, Faculty of Medical Engineering, Politehnica University of Bucharest, Bucharest, Romania*

The role of anterior hypothalamus and preoptic area in thermoregulation has been studied since the late 19th century (Charles Richet 1884, Arohnson&Sachs 1885), and thermosensitive neurons have been recorded in these areas by Jack Boulant et al. in the 1980s. Numerous animal studies with injection of serotonergic agonists or antagonists proved activation of thermoregulatory responses, and complex serotonergic circuits with origin in raphe nuclei (RN) and projection in hypothalamus and preoptic area (POA), nucleus accumbens (Acc), central amygdala (CeA) were identified. In SERT-/- rats stress-induced hyperthermia was absent (Olivier et al. 2008). We assessed the expression of SERT, 5-HTR1A and 5-HTR2A at mRNA and protein level in several brain regions: brainstem raphe, hypothalamus, Acc and POA, CeA, pyriform cortex (Pyr) in adult male CD1 mice kept 4h/day at 4°C or normal temperature (control) for 1-2 months. For qRT-PCR total RNA was extracted from fresh samples with a GenElute™ kit (Sigma), followed by RT and qPCR with hydrolysis probes (Applied Biosystems). For immunofluorescence 200 µm slices were cut from fixed brains and stained with primary antibodies ASR-021, ASR-033, AMT-004 (Alomone) and secondary antibody N2404-Ab635P-L (NanoTag), co-stained with phalloidin-AlexaFluor488 and DAPI. We obtained statistically significant (two-tailed t test) increases in mRNA expression in hypothermia vs. control for all 3 markers in raphe, for SERT and 5-HT2A in hypothalamus. Fluorescence intensities (averaged over small relevant regions) were higher in hypothermia for all 3 markers in dorsal RN, CeA, Acc, and similar to control levels in Pyr, proving activation

of central serotonergic thermoregulatory circuits.

## O6 Frontal transcranial direct current stimulation in moderate to severe depression: Clinical and neurophysiological findings from a pilot study

***Florin Zamfirache[1], Gabriela Prundaru[1], Cristina Dumitru[2], Beatrice Mihaela Radu[1]***

[1]Department of Anatomy, Animal Physiology, and Biophysics, Faculty of Biology, University of Bucharest, Splaiul Independentei, no. 91-95, Bucharest, Romania
[2]Department of Educational Sciences, Faculty of Educational Sciences, Social Sciences and Psychology, The National University of Science and Technology POLITEHNICA Bucharest

Transcranial Direct Current Stimulation (tDCS) is a promising intervention for major depressive disorder (MDD), yet its underlying neurophysiological mechanisms remain unclear. This pilot study examined both clinical and EEG-based effects of frontal tDCS in individuals with mild to severe depression. Thirty-one participants underwent the Flow Neuroscience tDCS protocol, targeting the dorsolateral prefrontal cortex via a bilateral F3/F4 montage. The protocol included an Active Phase (5 sessions/week for 3 weeks) and a Strengthening Phase (2 sessions/week thereafter). Depression severity was assessed using the Montgomery–Åsberg Depression Rating Scale (MADRS), while neurophysiological markers—Frontal Alpha Asymmetry (FAA), Beta Symmetry, and Theta/Alpha Ratio—were recorded using quantitative EEG before and after the intervention. Fourteen participants had baseline MADRS scores ≥20, indicating moderate to severe depression. Post-treatment, significant MADRS score reductions were observed, especially in the moderate/severe group (response rate: 71.4%) versus the mild group (20.0%). While FAA and Beta Symmetry showed no significant changes, a notable decrease in the Theta/Alpha Ratio at F4 ($p = 0.018$, Cohen's $d = –0.72$) was found in the moderate/severe subgroup, suggesting improved frontal cortical activation. These results support the clinical efficacy of tDCS, particularly in more severe cases. The Theta/Alpha Ratio may be a promising biomarker of tDCS response, whereas other EEG measures might reflect more stable, trait-like features. Further studies should explore personalized stimulation protocols and include advanced EEG metrics to clarify the neurophysiological basis of tDCS efficacy in depression.

## O7. Non-invasive neuromodulation of deep brain circuits using a novel ultrasound stimulation system in healthy people

***Ioana-Florentina Grigoras[1], Eleanor Martin[2], Morgan Roberts[2], Olivia Wright[2], Tulika Nandi[1], Sebastian Rieger[1], Jon Campbell[1], Tim den Boer[1], Ben Cox[2], Bradley Treeby[2] and Charlotte Stagg[1]***

*[1]Nuffield Department for Clinical Neurosciences, University of Oxford, Oxford, UK*
*[2]Department of Medical Physics and Biomedical Engineering, University College London, London, UK*

Transcranial ultrasound stimulation (TUS) is an emerging non-invasive brain stimulation technique that can, unlike TMS or tDCS, target deep brain structures. However, current systems have so far lacked the precision to target distinct deep brain nuclei. Here, we introduce an innovative MRI-compatible TUS system that can achieve unprecedented nucleus-specific neuromodulation. Our advanced system integrates a 256-element, helmet-shaped transducer array operating at 555 kHz with stereotactic positioning, personalized treatment planning, and real-time monitoring using functional MRI. In a seven-session study, we investigated how sonicating the lateral geniculate nucleus (LGN) impacted brain activity in the LGN itself and downstream pathways in seven young healthy volunteers. In the first three sessions, participants had MRI scans and a low-dose CT head to identify the LGN location and conduct TUS wave simulations. In sessions 4-5, participants received online LGN TUS (continuous 300ms, every 3 seconds, 5 times in each of the 10 stimulation blocks per run, 6 runs per session) during a visual checkerboard task and concurring fMRI acquisition. In sessions 6-7, we acquired resting-state, task-fMRI and visual behavioural data before and after offline TUS (80-second theta-burst stimulation) of either LGN or active control site. We found a significant increase in activity in the ipsilateral visual cortex (V1) during online stimulation (n=7). However, no significant effect was detected in the stimulated LGN, likely due to the large fMRI voxel size and nucleus depth. Interim analysis showed a significant decrease in task-related ipsilateral V1 activity (n=4) at 10 minutes, but not 2 hours, only after offline LGN stimulation. Our results show that our advanced TUS system can non-invasively modulate deep brain circuits, providing new avenues to develop future targeted therapies for neurological and psychiatric disorders.

## O8. ERP Markers of Cognitive Control Dysfunction in Adolescents with Complex Childhood Trauma

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Complex childhood trauma, characterized by chronic exposure to abuse, neglect, or instability, may disrupt neurodevelopment, particularly in brain systems related to executive control. Event-related potentials (ERPs) provide a non-invasive means to explore potential neural correlates of trauma-related cognitive difficulties, even in the absence of overt behavioral symptoms.
This study employed two electrophysiological approaches, a controlled group ERP study and a case analysis of maltreated adolescent siblings, to examine potential effects

of trauma on cognitive control. The aim was to explore whether ERP patterns could reflect neurodevelopmental vulnerabilities relevant to early identification and intervention. Twenty trauma-exposed adolescents and forty healthy controls completed a cued Go/NoGo task during 19-channel EEG recording. ERP components were analyzed using cluster-based permutation testing and latent component analysis. Additionally, two sibling pairs in foster care were assessed using the same task and compared with normative data and each other.
The group analysis identified ERP differences that may reflect difficulties in proactive and reactive control (e.g., reduced CNV, NoGo-N2, NoGo-P3) and predictive processing (diminished VN). These were associated with occipital and frontal latent components. Case-level findings complement these results: half-siblings showed heightened early sensory responses (P1/N1), while twins exhibited reduced N2 and posterior alpha desynchronization - patterns possibly linked to hypervigilance or impulsivity.
Although preliminary, these converging results suggest that ERP measures could be informative for identifying at-risk adolescents and tailoring neurodevelopmentally informed interventions. The results add to the utility of electrophysiological endophenotypes in clinical neuroscience approaches to childhood trauma.

## O9. Aquaporin 4 facilitation decreases amyloid burden in a mouse model of Alzheimer's disease

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Preclinical studies have implicated the Aquaporin 4 (AQP4) water channel in the accumulation and inadequate clearance of amyloid-β (Aβ) in Alzheimer's Disease (AD), however, a head-to-head comparison of AQP4 facilitator/inhibitor has not been yet performed.
METHODS: Two-months-old APPPS1 mice were treated daily for 28 days with either TGN-020-AQP4-inhibitor or the TGN-073-facilitator (200mg/kg), with vehicle-treated APPPS1 and wild type C57BL/6J mice as controls, and with extensive neuropathological / behavioral characterization.
RESULTS: AQP4-inhibitor-treated mice showed a robust increase in total Aneta while the facilitator led to a massive reduction in brain Aβ. AQP4-facilitator-treated mice also showed reduction in Aβ40 and Aβ40/Aβ42 ratio, while the inhibitor-treated animals showed an increase of both Aβ40 and Aβ42. Furthermore, AQP4-faciltator led to significant reduction in anxiety and increased memory performance.
DISCUSSION: Our data strongly suggest that AQP4 modulation is a powerful tool for facilitating Aβ clearance in AD and supports the development of AQP4-targeted therapies in the treatment of AD.

## O10. Complex neuro-biophysical approach for simultaneous investigation of neuronal-electrical activity and structural-functional changes in the area of the Caudate Putamen of the rat brain - in an animal model of Parkinsonian disease in vivo.

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Our research implemented the neurobiophysical approach using an animal model system with specially designed ECoG electrodes. Under the action of 6-hydroxydopamine (6-OHDA), a neurotoxin that selectively destroys catecholaminergic neurons, this method allows the reproduction of the main pathophysiological features of the disease in animals, such as the loss of dopamine in the striatum and the associated motor and cognitive impairments. This work analyzes the structural and functional changes in rats' Caudate-Putamen (CP) area under local inhibition of dopaminergic receptors. Using the electro-corticography-ECoG method, we use micro-electrodes to record the electrical processes occurring in the cortex in response to sensory stimulation (external or internal). When the electrode is placed on the primary sensory area receiving signals from a certain sense organ, a superficial positive wave with a latency period of 5 12 ms can be observed. The dendrites of cortical neurons are a dense array of uniformly oriented processes, especially in the superficial layers of the cortex. Dendrites can generate spreading potentials, which are essential in transmitting excitation. In addition, neuronal axons form collaterals that terminate on dendrites in the superficial layers. Upon activating the excitatory and inhibitory synapses on the dendrites, currents flow to and from these sources, creating a dipole electric field between the cell body and the dendrite. These dynamic changes in the dipole lead to wave-like fluctuations of the potential in the bulk conductor. Local structural changes caused by inflammatory (and/or degenerative) processes affect the amplitude of the recorded ECoG signal. Electrocorticography (ECoG) is often used to assess brain bioelectrical activity in small laboratory animals, allowing the recording of signals directly from the cerebral cortex. Direct recording of cortical activity using (ECoG) can be used to assess the functional state of the cortex.

# ABSTRACTS

## *for Young Investigator Blitz Presentations*

### BO1. Mechanisms of NIR laser photobiomodulation: in vitro studies on primary sensory neurons and voltage-dependent Na+ channels

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Photobiomodulation, a therapeutic method discovered by Endre Mester almost 60 years ago, was widely applied for treating neuropsychiatric disorders, including Parkinson's and Alzheimer's disease. Extensive studies have proved that red or near-infrared (NIR) radiation enhances electron flow via the mitochondrial respiratory chain, increasing production of ATP and reactive oxygen species that further trigger multiple signaling pathways. We assessed the effects of NIR stimulation with a 808.5nm diode laser applied via a multimode optic fiber with a sharp tip placed over the cell on enzyme-dissociated cultured adult rat primary sensory neurons and human embryo kidney (HEK293) cells stably expressing human voltage-dependent Na+ channels (hNav1.5) approached via patch-clamp. For each type of cell, specific series of voltage- or current-clamp protocols were applied initially and after 3 min of laser exposure or control conditions. Laser exposure induced in neurons a resting potential depolarization (6.6±1.8 mV vs. 2.4±1.8 mV in control, mean±SEM, p=0.0594). In Nav1.5-expressing cells, peak INa amplitude slightly increased after laser application (111.2±14.9% vs. 70.6±10.4% in control experiments), and in outside-out patches the differences were larger (96.64±5.25%-laser vs. 37.95±9.14%-control). Via chemiluminometry we evidenced a delayed increase in ATP production in laser-exposed HEK293 cells: 1.06±0.06 μM for a 6-min exposure followed by a 6-min pause vs. 0.90±0.06 μM in controls. An explanation of these effects is that NIR exposure facilitates ATP production, maintaining an adequate state of Na+ channels phosphorylation, but we cannot exclude direct polarization effects on macromolecules including ion channels produced by the intense oriented electric field of the laser beam.

### BO2. The neuroprotective effect of naringenin on endothelial inflammation induced by lipopolysaccharide on a BBB microvasculature model

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Neuroinflammation, triggered by systemic immune stimuli such as lipopolysaccharide (LPS), can impair the structural integrity and homeostatic function of the blood–brain barrier (BBB), contributing to the progression of neurodegenerative diseases. We investigated the neuroprotective effects of naringenin, a flavonoid with antioxidant and anti-inflammatory properties, against LPS-induced endothelial dysfunction. We used an in vitro BBB microvasculature model based on bEnd.3 mouse brain endothelial cells.
We performed a series of assays to evaluate the endothelial responses to LPS exposure and naringenin treatment. Cell viability was assessed using the MTT assay: LPS significantly reduced viability, while naringenin exhibited minimal cytotoxicity at concentrations up to 40 μM. However, higher concentrations determined a dose-dependent cytotoxic effect. Based on these findings, minimally cytotoxic concentrations of naringenin were chosen for subsequent experiments involving ZO-1 expression and cytoskeletal analysis to evaluate its protective potential without affecting cell health. Oxidative stress was measured using a reactive oxygen species (ROS) detection assay, where naringenin reduced LPS-induced oxidative stress dose-dependently. We performed immunocytochemistry for the tight junction protein ZO-1 to evaluate BBB integrity. The exposure to LPS decreased ZO-1 expression, while naringenin treatment partially restored it and improved junctional continuity. Additionally, we analyzed cytoskeletal organization through actin staining, which showed that naringenin preserved the cytoskeletal structure and mitigated LPS-induced disruption. These findings suggest that naringenin, at lower concentrations, can protect against LPS-induced oxidative stress, tight junction disruption, and cytoskeletal disorganization, highlighting its potential as a therapeutic agent for protecting BBB integrity in neuroinflammatory conditions that can lead to neurodegenerative diseases.

### BO3. Mapping Brn3b+ Cell populations of the Superior Colliculus using Brn3bCre mice.

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Aims: To understand the neuroanatomy of Superior Colliculus Neurons expressing Brn3bCre using Brn3bCre mice. Methods: We targeted different dorsoventral and rostrocaudal positions of the superior colliculus using stereotactic injections of Cre-dependent AAV-FLEX-tdTomato virus in Brn3bCre mice. Intracardiac paraformaldehyde fixation was performed on day 30 after injection, followed by retina and brain dissection/sectioning, and imaging at low (X5) and high-resolution (X20). Results: Neurons expressing Brn3bCre are present ipsilateral to the injection site within the optic and inner grey layers of the superior colliculus, with their dendrites projecting towards the superficial grey and zonal layers of the superior colliculus. Furthermore, we observed that these cells project in the ipsilateral dorsal terminal nucleus of the accessory optic tract and the bilateral lateral posterior thalamic nucleus. Additionally, we found axonal projections, that originated in the superior colliculus ipsilateral to the injection, followed the ipsilateral optic brachium and tract, through the optic chiasm, backed up the contralateral optic tract, and ended into the contralateral lateral posterior thalamic nucleus, suggesting a communication between superior colliculus and contralateral lateral posterior thalamic nucleus via the optic tract. Conclusions: AAVFLEX-tdTomato injections in Brn3bCre mice reveal Brn3bCre+ cell bodies in several layers of the ipsilateral superior colliculus. The ipsilateral midbrain, superior colliculus, and bilateral thalamus contained target nuclei encompassing projections of Brn3bCre+ neurons. We identify a novel path for contralateral signal spread from the superior colliculus via the optic chiasm without spreading into the retinas and the visual cortex.

## BO4. Investigating microglial migratory properties that underlie their accumulation in multiple sclerosis

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In multiple sclerosis (MS), microglia accumulate and form small clusters that are prone to MS lesion formation. However, microglia are associated as well with remyelination, which can be observed across all types of lesion. Considering the opposing capabilities of microglia, this work aims to further characterize the role of microglia in MS pathogenesis from a migratory perspective.
We exposed defined-medium cultures of microglial cells (serum-free) to cerebrospinal fluid (CSF) collected from five drug-naive female patients after the first attack of clinical symptoms, with relapsing-remitting MS. Here, we analysed the migration pattern of microglia, coupled with bioimage analysis of microglial actin cytoskeleton, at 4% and 10% CSF-MS exposure, relative to 10% serum exposure (serum control, SC) and non-treated control (NC).
Microglia exposed to 10% CSF-MS showed significantly increased migration compared to 4% CSF-MS and NC microglia; there was no significant difference when compared to SC-exposed microglia. In both SC and CSF-MS groups, microglia exhibited shorter but more numerous actin filaments compared to unstimulated NC; the longest actin filaments were identified in NC condition. Additionally, we demonstrated that microglia exposed to 4% CSF-MS and NC microglia relied on a constant migration dynamic, while 10% CSF-MS showed a peak between 4.5 and 6h after exposure. Interestingly, the SC-exposed microglia were characterized by a fast migration pattern in the first 3 hours and remained constant only beyond this timepoint. To conclude, the exposure of cerebrospinal fluid from MS patients altered microglial migration in a concentration and time-dependent manner.

## BO5. Metabolic lactate shuttle powers electrical activity of small cell lung cancer

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Small-cell lung cancer is one of the most aggressive types of cancer, comprising two distinct cell type subpopulations, neuroendocrine (NE) and non-neuroendocrine (non-NE) cells, that cooperate to promote proliferation, invasion and metastasis. Neuroendocrine cells, the predominant metastatic cell type, are electrically excitable and resemble neurons while non-NE cells have a supportive role and resemble astrocytes. In this study we aimed to characterize the metabolic cooperation between NE and non-NE cells in relation to electrical activity that we show to promote malignancy. Based on evidence indicating that NE cells show strong dependency on oxidative phosphorylation and non-NE cells secrete large amounts of lactate, we hypothesized that a mechanism analogous to the astrocyte-neuron lactate shuttle sustains the high ATP-demanding electrical activity in NE cells. We show that astrocyte-like non-NE cells express MCT4 transporter favoring lactate export and neuronal-like NE cells express MCT1 transporter involved in lactate import. Co-culturing NE cells with non-NE cells and non-NE conditioned medium increased the calcium activity of NE cells which could be suppressed by an MCT4 inhibitor, diclofenac. A strong reduction in calcium activity upon diclofenac treatment was

also observed ex vivo in precision-cut lung slices with SCLC tumors expressing calcium indicator GCaMP6f. In addition, overnight incubation with MCT1 inhibitor SR-13800 led to a more depolarized resting membrane potential and decreased excitability in NE cells in the presence of lactate. Overall, we identify a metabolic cooperation mechanism analogous to the astrocyte-neuron lactate shuttle that supports electrical activity of SCLC.

## BO6. EEG Microstates Reactivity during Social Imitation

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It was previously reported that global patterns of neuronal activity predicted changes in spontaneous thoughts induced by social imitation. Specifically, social imitation was linked to an increase in the duration of microstate C, famously associated with the default mode network (DMN)[1]. The current study aims to further assess changes in microstate dynamics that occur during dyadic social imitation tasks (SI) as opposed to control (CTRL) tasks. In addition to the 5-minute baseline periods before (PRE) and after the imitation task[1], we included 128-channel EEG recordings from 32 healthy young volunteers, subjected to a 3-minute SI task, and a CTRL task of non-social imitation of geometrical figures displayed on a computer screen. After K-Means group clustering of individual cluster groups, we found 5 microstate classes conventionally labelled as A-E. We calculated the mean duration of each class during SI and CTRL and found that microstates C and D (Wilcoxon $p<0,0001$) show a significant decrease during SI. Furthermore, we compared the changes during SI and CTRL to the PRE baseline period by calculating the reactivity (=(PRE-STIM)/PRE*100)) of each class duration. We found that microstate C, having a baseline mean duration of 128,4ms (mean;SEM), showed the largest reactivity during imitation. Its reactivity during SI was 23,3% and only 18,3% during CTRL ($p=0,03$). Overall, our data suggests that DMN-associated microstate C, and attention-related microstate D, show the largest decrease during the SI as opposed to CTRL. This might predict the neurocognitive beneficial effects of social imitation.

## BO7. Fine Particles, Fine Damage: Cytotoxic and metabolic effects of Diesel nanoparticles on different cell components of the central nervous system

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Air pollution, particularly traffic-related air pollution (TRAP), represents a growing threat to public health, with fine and ultrafine particulate matter (PM2.5-10) linked not only to cardiovascular and respiratory diseases, but also increasing the risk for developing neurodegenerative disorders, according to some studies. We investigated the cellular and neurotoxic effects of unfiltered diesel particulate matter (DPM) using a multimodal in vitro and ex vivo approach. Four cell lines representing neuronal (SH-SY5Y), endothelial (bEnd3), epithelial (HEK293), and microglia (BV2) populations were exposed to DPM, and assessed for oxidative stress (ROS-Glo $H_2O_2$) and viability (Resazurin assay). Exposure to DPM led to increases in oxidative stress in SH-SY5Y cells and significant reductions in viability for all cell lines, partly attenuated by prior filtration of DPM. Complementary ex vivo experiments on adult rat hippocampal slices cultured for 10 days revealed that exposure to unfiltered DPM induced a small accumulation of phosphorylated tau, as indicated by AT8 immunoreactivity in the CA3 region, paralleling patterns seen in β-amyloid-treated controls. Acridine orange staining further revealed increased intensity and density of fluorescent granules in DPM-treated slices compared to controls, suggesting elevated cellular stress, lysosomal activity, or particle uptake. These alterations were absent in slices maintained in control medium. Taken together, our results demonstrate that Diesel-derived nanoparticles induce oxidative stress in neuronal-like cells and reduce viability in a cell-type dependent manner.

## BO8. 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.

## BO9. Exploration of the specific role of RGC-32 gene in an Experimental Autoimmune Encephalomyelitis murine model.

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Introduction: Multiple sclerosis (MS) is an autoimmune, inflammatory, and neurodegenerative disease of the central nervous system (CNS), determined by a complex combination of genetic and environmental factors. MS is primarily characterized by inflammation, demyelination, and oligodendrocyte cell death. RGC-32 is a cell cycle regulatory gene expressed by oligodendrocytes, CD4+ T lymphocytes, endothelial cells, and astrocytes, all of which play a fundamental role in MS. RGC-32 is modulating disease progression in experimental autoimmune encephalomyelitis (EAE), a mouse model of MS. Aims: To determine whether RGC-32 plays a role in the optic neuritis and visual function in EAE. Methods: We induced EAE in C57BL/6 mice (n = 2 RGC-32 WT; n = 3 RGC-32 KO) by MOG 35–55 peptide immunization. Mice were weighed and scored daily for three weeks using a 5-point clinical scale (Stromnes et al, 2006). Pathological changes were histologically assessed in the lumbar spinal cord, brain, and optic nerve. Results: RGC-32 KO mice developed EAE symptoms later than RGC-32 WT mice, while the final clinical score was comparable in KO and WT mice. Moreover, we noticed additional symptoms previously not reported during EAE, such as ptosis and difficulty breathing. At the top of disease progression, KO mice exhibited increased inflammatory infiltrates in the spinal cord and optic nerve compared to wild-type. Histological signs of optic neuritis only partially correlated with spinal cord inflammation. Optomotor responses of KO animals were significantly better than wild-type responses when compared at 12 days post-immunization. Conclusions: RGC-32 KO mice exhibit somewhat milder and delayed EAE progression compared to wild types, but disease phenotypes exhibit large intra-group variabilities, requiring larger experimental groups.

## BO10. Analysis of Ret gene expression in retinal ganglion cells and their brain projections using Immunofluorescence in CFP-Reporter Mice

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Ret gene is a proto-oncogene that encodes a receptor tyrosine kinase for the glial cell line-derived neurotrophic factor (GDNF) family ligands. It is known that Ret signaling plays a crucial role in the development, survival, and connectivity of neuronal populations in the peripheral and central nervous systems. Nevertheless, the precise role of Ret signalling in the visual system remains poorly understood. We used immunostaining to examine the expression pattern of Ret in genetically modified mice (Rax: Cre; Ret CKCFP/WT = RetHet and Rax: Cre; Ret CKCFP/CKCFP= RetKO), using a conditional knock-in CFP reporter allele. In the retina, Ret expression was observed in retinal ganglion cells (RGCs), amacrine cells, and horizontal cells in both genotypes, with stronger staining intensity in RetKO mice. In coronal brain sections, RetCFP RGC projections were detected in retinorecipient areas, including the lateral geniculate nucleus, superior colliculus, pretectal area (olivary pretectal nucleus), optic tract and optic chiasm. Conversely, no Ret expression was detected in the medial terminal nucleus or the suprachiasmatic nucleus. Retina-specific ablation of Ret did not significantly affect RetCFP RGC numbers, suggesting that Ret is not required for RGC survival. Interestingly, RetCFP, cell bodies were observed in the amygdala and several hypothalamic nuclei. To our knowledge, this is the first study mapping Ret expression in RGCs brain projections. These results provide insight into the spatial distribution and relative expression levels of Ret in retina and brain projections and lay the groundwork for future studies on Ret-dependent signaling in visual processing, with potential implications for understanding visual system development and related neurodegenerative diseases. Keywords: ret gene, retina, retinal ganglion cell, brain, retinal ganglion cells projections.

## BO11. Distribution of Retinal Ganglion Cells expressing Brn3b and Brn3c in the Area Centralis of the mouse retina and their axonal projections in the brain

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Retinal Ganglion Cells (RGCs) constitute critical components of the visual system, functioning as the primary output neurons of the retina relaying visual information to the brain. High visual acuity is predominantly achieved within specialized retinal regions, such as the fovea or Area Centralis (ArCe), which are distinguished by a high density of RGCs. Within the retina, Brn3b and Brn3c, two members of the Pou4f family of transcription factors are expressed exclusively in RGCs and co-localize in a RGC subpopulation highly enriched in the ArCe. To explore the developmental timing of ArCe formation and axonal projections in specific areas of the brain, we analyzed retina, SC and LGN of Brn3cCre; Brn3bCKOAP mice at embryonic day E15, postnatal days P0, P7, P15, and adults. We also investigated the roles of Brn3c and Brn3b in ArCe formation, by specifically ablating the two transcription factors from Brn3cCre; Brn3bCKOAP RGCs, using either Brn3cKO/Cre;Brn3bCKOAP/WT or Brn3cCre/WT;Brn3bCKOAP/KO mice, and appropriate controls. Using sparse AAVFLEXeGFP virus infections in Brn3cCre; Brn3bCKOAP retinas combined with anti-Brn3b and anti AP (Alkaline Phosphatase) imunostainings, we found that the majority of Brn3b+Brn3c+ RGCs belong to the OFF-DS (JamB) and OFF-delta (OFF-sustained) types, with a few instances of midget-like and small bistratified cells. Preliminary data suggests postnatal refinement of the ArCe density gradient relative to the rest of the retina, and no obvious involvement of either Brn3b or Brn3c in ArCe formation. Albinism does not impact the ArCe, since the high density of Brn3c+Brn3b+ RGCs is not affected in TyrCj/Cj;Brn3cCre; Brn3bCKOAP mice (a common albinism model).

## BO12. Transcranial Focused Ultrasound Stimulation of the Human Basolateral Amygdala Modulates Approach-Avoidance Behaviour Towards Emotionally Ambiguous Faces

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The amygdala plays a central role in evaluating the emotional salience and ambiguity of social cues, such as facial expressions, as well as in shaping automatic emotional biases. Through its interactions with prefrontal regions, it supports flexible behaviour by integrating these biases with contextual information. While these mechanisms are well supported by animal and correlational studies, causal evidence in humans has remained limited due to the challenge of non-invasively targeting deep brain structures.

Here, we used transcranial ultrasound stimulation (TUS), a novel, non-invasive neurostimulation technique, to test the causal role of the human basolateral amygdala (BLA) in steering emotional approach/avoidance biases in healthy volunteers. Participants (n=30) underwent three TUS-fMRI sessions, receiving offline TUS targeting the bilateral BLA, insula (active control), or sham (counterbalanced order). Following TUS, participants engaged in a cognitive task designed to probe social-emotional flexibility in learning and overwriting automatic responses, while 7T-fMRI data were collected. BLA TUS increased the probability of approaching neutral faces compared to sham, and led to slower reaction times for these stimuli, suggesting a reduced disambiguation of emotional content. Resting-state fMRI provided proof of target engagement, showing reduced BLA connectivity with anatomically connected cortical and subcortical regions following TUS. Preliminary analyses of task-fMRI data suggest altered BOLD responses to neutral faces in the amygdala and ventral prefrontal cortex (vPFC).

In conclusion, by employing TUS, we provide novel causal evidence for the involvement of amygdala-prefrontal circuits in guiding emotional approach/avoidance biases and resolving emotional ambiguity.

## BO13. Attractiveness-Driven Bias in Cooperative Decision-Making: An ERP Study on Neural Signatures in Social Exchange

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Human social interactions are strongly shaped by both physical and emotional perceptions of others. Facial attractiveness, in particular, has been shown to influence empathy—a core cognitive mechanism underpinning social behavior. Individuals tend to display a positive behavioral bias toward those they perceive as attractive. Given that empathy has been identified as a key driver of cooperative behavior, this suggests a potential pathway through which attractiveness-based biases can impact pro-social decision-making. This study investigates how perceived facial attractiveness influences cooperation by using a socio-economic decision-making paradigm combined with EEG recording to examine neural responses. Event-Related Potentials (ERPs), specifically the N170, P300, and LPP components, were analyzed in relation to the moment of cooperative choice. A novel aspect of the study involved presenting participants with facial images that were partially obscured by surgical masks or ski goggles prior to decision-making, to examine how limited facial visibility might influence attractiveness perception and subsequent empathy-driven responses. We hypothesized that the brain's predictive mechanisms would infer attractiveness from partially covered faces, eliciting more empathetic and cooperative behavior compared to fully visible faces. This research contributes to a deeper understanding of the neural mechanisms underlying social behavior and implicit biases in human interactions.

## BO14. From Stability to Collapse: Rethinking Cognitive Decline as a Functional Transition in Brain Network Dynamics

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Cognitive decline is commonly conceptualized as the hallmark of progressive structural brain injury. However, recent data suggest that functional networks may developing states of instability long before pathology shows anatomical change. This gradual reduction in synchrony, entropy, and adaptability may further represent an emergent instability in a dynamic system that eventually yields neurodegeneration (ND) as the chronic system response. We intend to put forward a conceptual model in which we frame cognitive decline as a phase transition emerging from instability in dynamic networks, based on original clinical and computed data.

Using real-world data from both neurovascular and oncologic patients, we deconstructed machine learning based models of outcome prediction and functional markers of network behavior (entropy compression, loss of controllability, anticipatory failure, hub failure). To unpack early phase functional disorganization and correlate them to clinical events we used principles of nonlinear systems theory and explainable AI. In all cases and in different clinical contexts, models exhibited good explanatory performance to forecast degrees of conscious change and severity of outcome. Explanatory tools also described signs of functional collapse, even in patients who were apparently clinically stable. Inflammatory and molecular signatures of compromised neuroplasticity also support an emergent mechanistic rational for dynamic network fragility, suggesting cognitive decline may be more emergent from destabilization of systemic resilience rather than progressing along a cascade or series of injury. We presented a systems level model of cognitive decline as a transition through states of critical instability based on original data collected across clinical, molecular, and computational realms. This approach may support attempts for improvement in early screening, monitoring, and preventing progressive ND.

# ABSTRACTS
## *for Poster Presentations*

### P1. Heart Rate Variability Under Conditions of Intermittent Photic Stimulation in Anesthetized Rats

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This study aimed to investigate HRV under IPS in rats subjected to deep or superficial anesthesia, characterized by Burst-Suppression (BS) or Slow-Waves (SW) EEG patterns. ECG and EEG recordings were performed in male Wistar rats from two groups, deep anesthesia BS group (n=10) and superficial anesthesia SW group (n=10), anesthetized with 0.4 g/kg body-weight i.p. and 0.1 g/kg body-weight i.p. chloral-hydrate, respectively. IPS at a frequency of 0.5 Hz was delivered to one eye in 3-minute trials, as follows: 1 minute without stimulation, 1 minute with stimulation, and 1 minute without stimulation. The sympathetic (S) and vagal (V) components and the ratio between them (R=S/V) were analyzed from the ECG recordings using BIOPAC Systems Inc. software. In the BS group, the S mean was 0.78, the V mean was 0.21, and the R mean was 4.26 (p=0.00018). In the SW group, the S mean was 0.23, the V mean was 0.76, and the R mean was 0.31 (p=0.00018). For the BS group, sympathetic activity was more pronounced, which led to a higher ratio. In contrast, in the SW group, vagal activity was more pronounced, which led to a lower ratio. Our data suggest that IPS influences the sympathetic and vagal components of the ECG signal for various anesthetic levels.

### P2. Preparation and Assessment of Primary Microglia Isolated from Mixed Glia Cell Culture of Newborn Rat Pups

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Microglia, the resident immune cells of the central nervous system, are key regulators of brain homeostasis and play crucial roles in neuroinflammation and neurodegeneration. This study outlines a protocol for isolating primary microglia with high purity from mixed glial cultures derived from neonatal Wistar rat brains and describes methods for assessing their functional properties. Brains were harvested from neonatal rat pups and meninges carefully removed under a microscope. Cortices and hippocampi were isolated, finely minced, and enzymatically digested in trypsin at 37°C. Digestion was halted using a trypsin inhibitor and DNase. Following cell resuspension and counting, cells were plated into T-75 coated flasks (density of 500.000 cells/cm2). Culture medium was changed the following day and every 5 days afterwards. Primary microglia or an astrocyte-microglia-oligodendrocyte mixture were plated on 24-well dishes. For the functional assessment, lipopolysaccharide (LPS) was added in both types of cell cultures for glial activation. ELISA tests, immunostaining (IBA1, GFAP) and morphological evaluation by fluorescence microscopy were conducted. After 7 days in vitro, the mixed glial cultures consisting in ~75% astrocytes (GFAP+) and ~25% microglia (IBA1+) reached confluency. Microglia, along with a minority of oligodendrocytes grew on top of the astrocytic layer. LPS activated microglia in a dose-dependent manner, shifting their morphology to amoeboid. The method used in this study enables reliable isolation and culture of functional primary microglia from neonatal rat brain tissue. These cultures, maintained either alone or in a combination with astrocytes, represent a valuable in vitro model for investigating neuroinflammatory mechanisms and related neuropathologies.

### P3. The Probiotic Usage Potential to Alleviate Depressive Behavioural Features and Anxiety in an Animal Model of Gestational Antibiotic Exposure

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Introduction: Chronic exposure to stressors during gestation has been linked to the development of depression and anxiety. These conditions may cause detrimental changes in maternal care with downstream consequences on offspring neurodevelopment. We employed a rodent model of gestational antibiotic administration to evaluate the behavioural outcomes associated with maternal microbiome disruption, and to investigate whether

probiotics could serve as a protective intervention. Materials and Methods: Our study involved 4 experimental groups. One group of pregnant rats received an antibiotic cocktail (ampicillin+vancomycin+neomycin+meropenem) daily (4 pm-8 am) starting with embryonic day (E) 11. Another group additionally received a multi-strain probiotic daily (8 am-4 pm). A third group received just the probiotic mix from E1 and a fourth, control group consisted in untreated rats. Maternal behaviour was assessed using Forced Swim Test (FST) and Elevated Plus Maze (EPM).
Results: The antibiotic group was associated with diminished exploratory behaviour in the EPM, indicating elevated anxiety-like behaviour. In FST, the antibiotic group was less prone to climbing and swimming with increased time of immobility ($p<0.05$), exhibiting features of depressive-like behaviour, compared to control. Co-administration of probiotics diminished these changes, and was correlated with increased time spent in open arms in the EPM, suggesting attenuated anxiety and depressive-like responses.
Conclusions: These preliminary results support the hypothesis that probiotic supplementation during gestational antibiotic exposure may exert neuroprotective effects, partially mitigating behavioural manifestations of anxiety and depression in mothers. Further studies with different probiotics and additional tests for anxiety and depression-related behavioural changes should be explored.

## P4. Antibiotic exposure during gestation increases perinatal asphyxia severity in rodents

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Antibiotic use in gestation is essential for treating infections. Nevertheless, the antibiotic-induced disruption in the maternal gut microbiota has been associated with various adverse long-term health outcomes in children. We investigated the effect of antibiotic exposure during gestation, on the outcome of perinatal asphyxia (PA), a severe birth complication. Pregnant Wistar rats were treated from gestation day 11 to term with antibiotics (AB), antibiotics combined with probiotics (AB-P) or probiotics alone (P). All pups underwent a standardized experimental PA protocol after birth (P6). We assessed the extent of the Hypoxic-Ischemic Brain Injury (HIBI) by the EEG reactivity to photic stimulation after 10 weeks.
Burst-suppression EEG patterns were induced by deep sedation with chloral hydrate. For each rat, we recorded multiple trials consisting of 1 minute of photic stimulation (0.5 Hz) interleaved with a 1-minute recovery period. The EEG bursts were manually counted. A burst count reactivity index (BCRi) was derived as the increase in burst count during stimulation, relative to baseline burst count. When compared to the P group (Mann-Whitney U), the antibiotic group showed a decreased BCRi ($U = 1.00$, $p = 0.003$), whereas the BCRi remained unchanged in the AB+P group ($U = 20.50$, $p = 0.654$). These data suggest that antibiotic exposure during gestation increased the severity of HIBI via microbiome disruption. The coadministration of probiotics could emerge as a clinical strategy to counteract the detrimental effect of antibiotic treatment during pregnancy.

## P5. NEURAL SUB-CELLULAR LOCALIZATION OF TUSC5/TRARG1 PROTEIN IN TUSC5eGFP NOVEL MOUSE LINE

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Recently we have found Tusc5 (tumor suppressor candidate 5, also known as Trarg1) gene and Tusc5 protein to be expressed in postnatal mouse retina (Sajgo et al. 2017; Muzyka et al. 2018), projection somatosensory neurons, and neurons of olfactory and auditory systems (unpublished data). To study neuronal subtype-specific expression in the above sensory systems, we generated a conditional Tusc5 knock-in mouse line with the Cre-dependent expression of eGFP fluorescent reporter (unpublished). Using genetic crosses to express retina-specific Cre enzyme, we demonstrated that eGFP reporter expression coincides with Tusc5-expressing neurons in the eyes of our mutant heterozygote mice. Tusc5 retina-specific knock-out does not express Tusc5 in the retina, while eGFP expression is present in the cells, under the Tusc5 gene locus endogenous regulatory elements. This novel mouse allele enables us to identify Tusc5-positive neuronal subpopulations and study their anatomy and physiology.
We analyzed cellular compartments to be occupied by Tusc5 protein in transfected HEK293, we found its pronounced co-localization with the markers of endosomal trafficking pathway – EEA1 and Transferrin. We also evaluated the results of our previous mass spectroscopy analysis performed in the intracellular vesicular fractions from wild-type mouse retina samples this time using confocal microscopy and colocalization image analysis. We assessed eight potential candidates for colocalization with Tusc5 in RGCs, and confirmed it for at least four of the molecules (colocalization coefficient above 0.4). This data requires further protein biochemical validation using

coimmunoprecipitation and western blot techniques to elucidate the protein-protein interactors of Tusc5 in RGCs.

## P6. Retina-specific ablation of the transcription factor Pou4f1/Brn3a does not significantly impact Ipsi vs. Contralateral projection of Retinal Ganglion Cells in mice

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The Pou4f transcription factor Brn3a domain, is required for the development of Retinal Ganglion Cell (RGCs) types, which convey visual information from the eye to the brain. RGC axons reach the midline at the optic chiasm and either cross to the contralateral hemisphere or turn away from the midline and project ipsilaterally (only 3-5% of all mouse RGCs). It was previously reported that Brn3a is enriched in contralaterally projecting RGCs, but its role in contra vs. ipsi decisions remains unclear, since germline Brn3a knockout mice die at birth. We therefore specifically deleted Brn3a from RGCs using our Cre-dependent Brn3a conditional allele (Brn3aCKOAP) and a retina - specific Cre driver (Rax:Cre). We labelled RGC projections by injecting the fluorescent tracers ChTB-A555 in the right eye and ChTB-A488 in the left eye of retinal Brn3a KO (Rax:Cre; Brn3aCKOAP/KO), heterozygote (Rax:Cre, Brn3aCKOAP/WT), or wild type control (Brn3aCKOAP/WT) adult mice. We then quantitated ipsi and contra A488 and A555 fluorescence intensities in the Lateral Geniculate Nucleus, the Olivary Pretectal Nucleus, the Suprachiasmatic Nucleus and the Superior Colliculus). No significant differences between ipsi and contra projections to the investigated retinorecipient areas were observed across the three genotypes.We also determined axonal projections of Brn3aAP RGCs in monocularly enucleated heterozygote (Rax:Cre, Brn3aCKOAP/WT) and KO (Rax:Cre, Brn3aCKOAP/KO) mice, using alkaline phosphatase staining. Brn3aAP RGCs project to the characteristic ipsilateral dLGN position,in both Brn3a heterozygote and KOmice. In conclusion, Brn3a RGCs project both ipsi and contralaterally and Brn3a does not play a role in ipsi vs. contra specification.

## P7. Democratizing Sleep Research: A Cost-Effective System for Long-Term Rodent Monitoringacters

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Long-term electrophysiological recordings in freely moving small animals are critical for sleep and epilepsy research but often limited by the cost, fragility, and energy constraints of wireless commercial systems. We designed an in-house, cost-efficient setup combining a wired ECoG/EMG/video acquisition platform with a mechanical untwisting mechanism to address wire torsion—a major limitation in traditional tethered systems. Using a headstage linked via a guitar string to a horizontal "wing," movement-induced torsion activates a contact-based feedback loop connected to an Arduino-controlled motor, realigning the tether in real time. We employed an intrahippocampal kainic acid model in C57Bl/6 mice (n=5; 3 epileptic, 2 control) and recorded 24-hour sessions across 5+1 channels. Data were segmented into 30-second epochs, manually labeled using synchronized EcoG, spectral density, and video, following Herrero et al.'s criteria. Labels included REM/NREM/Wake and epileptic spike presence. A 1D convolutional neural network trained on a single 24h dataset per group achieved 64% accuracy in sleep stage classification. Results confirmed a significant reduction of epileptic spikes during REM sleep and a corresponding decrease in REM duration in epileptic animals—consistent with REM's known protective effects. The video component proved essential in REM vs. Wake differentiation, while NREM was reliably detected via EcoG alone. This system offers a reliable, low-cost alternative for extended sleep studies in mice, particularly valuable for underfunded labs. While promising, limitations include small sample size and reduced REM epochs in adult C57Bl/6 mice. Further refinements in both hardware and machine learning models are needed for broader application.