National Neuroscience Society of Romania (SNN)

UNIVERSITY OF BUCHAREST
VIRTUTE ET SAPIENTIA

26-28 October, Bucharest

ICON4N

# 3rd International Conference on Neuroscience, Neuroinformatics, Neurotechnology and Neuro-Psycho-Pharmacology

# ABSTRACT BOOK

FIZIOLOGIA
physiology
Supplement no. 1/2023

# FIZIOLOGIA
## *physiology*




**ACCREDITED BY CNCSIS - B+CATEGORY ■ CODE 240**

http://www.ebscohost.com/titleLists/a9h-journals.pdf

**Fiziologia (Physiology)** is issued quarterly
Printed at Editura **EUROSTAMPA**
**www.eurostampa.ro**

Bd. Revoluţiei din 1989 nr. 26,
TimişoaraTel/fax: 0256-204816

**ISSN 1223-076**

# Instructions to Authors

**Submission:** Only original papers in English are considered and should be sent to the following address: carmen.tatu@umft.ro

**Manuscripts** should be submitted by e-mail only, written in Microsoft Word 97 or later versions.

**Conditions:** AII manuscripts are subject to editorial review. Manuscripts are received with the explicit understanding that they are not under simultaneous consideration by any other publication. Submission of an article for publication implies the transfer of the Copyright from the author the publisher upon acceptance. Accepted papers become the permanent property of "Fiziologia" (Physiology) and may not be reproduced by any means, in-whole or in part, without the written consent of the publisher. It is the author's responsibility to obtain permission to reproduce illustrations, tables, etc. from other publications.

**Arrangement:**

**Title page:** The first of each paper should indicate the title, the authors' names and their affiliation(s). A short title for use as running head is also required.

**Keywords:** for indexing purposes, a list of 3-5 keywords in English and Romanian is essential.

**Corresponding author:** Indicate the full name, the

email address and the phone number.

**Abstract:** Each paper needs abstract and title in Romanian and English language, fonts size 9, Arial Narrow.

**Body text:** fonts size 10, Arial Narrow.

**Small type:** Paragraphs which can or must be set in smaller type (case histories, test methods, etc.) should be indicated with a „p" (petit) in the margin on the left-hand side.

**Footnotes:** Avoid footnotes. When essential, they are numbered consecutively and typed at the foot of the appropriate page, fonts size 8, Arial Narrow.

**Tables and illustrations:** Tables (numbered in Roman numerals) and illustrations (numbered in Arabic numerals) should be prepared on separate sheets, fonts size 9, Arial Narrow. Tables require a heading, and figures a legend, also prepared on a separate sheet. For the reproduction of illustrations, only good drawings and original photographs can be accepted; negatives or photocopies cannot be used. When possible, group several illustrations on one block for reproduction (max. size 140x188 mm) or provide crop marks. On the back of each illustration indicate its number, the author's name, and article title.

**References:** In the text identify references by

Arabic figures, (in brackets), fonts size 9, Arial Narrow. Material submitted for publication but not yet accepted should be noted as "unpublished data" and not be included in the reference list. The list of references should include only those publications which are cited in the text. The references should be numbered and arranged alphabetically by the authors' names. The surnames of the authors followed by initials should be given. There should be no punctuation signs other than a comma to separate the authors. When there are more than 3 authors, the names of the 3 only are used, followed by "et al" abbreviate journal names according to the Index Medicus system. (also see International Committee of Medical Journal Editors: Uniform Requirements for manuscripts submitted to biomedical journals. Ann Intern Med 1982; 96: 766-771).

Examples:

(a) Papers published in periodicals: Kauffman HF, van der Heide S, Beaumont F, et al: Class-specific antibody determination against Aspergillus fumigatus by mean of the enzyme-linked immunosorbent assay. III. Comparative study: IgG, IgA, IgM, ELISA titers, precipitating antibodies and IGE biding after fractionation of the antigen. Int Arch Allergy Appl Immunol 1986; 80:300 - 306.

(b) Monographs; Matthews DE, Farewell VT: *Using and Understanding Medical Statistics.* Basel, Karger, 1985.

(c) Edited books: Hardy WD Jr, Essex *M:.FeLV-inducted feline acquired immune deficiency syndrome: A model for human AIDS;* in Klein E(ed): Acquired Immunodeficiency Syndrome. Prag Allergy, Busel, Karger, 1986, vol 37,353 - 376.

**Galley proofs:** unless indicated otherwise, galley proofs are sent to the first-named author and should be returned with the least possible delay. Alternations made in galley proofs, other than the corrections of printer's errors, are charged to the author. No page proofs are supplied.

# 3rd International Conference on Neuroscience, Neuroinformatics, Neurotechnology and Neuro-Psycho-Pharmacology

# 3RD INTERNATIONAL CONFERENCE ON NEUROSCIENCE, NEUROINFORMATICS, NEUROTECHNOLOGY AND NEURO-PSYCHO-PHARMACOLOGY

*We kindly invite you to participate in the*

***3rd International Conference on Neuroscience, Neuroinformatics, Neurotechnology and Neuro-Psycho-Pharmacology****,*

*Bucharest, 26-28 October 2023*

*organized by the Romanian Academy – National Center for Brain Research, in collaboration with National Neuroscience Society of Romania, the Romanian Society for Automation and Technical Informatics and University of Bucharest.*

*The aim of the conference is to bring together experts from various areas of brain-related research, including neuroscience, clinical neurology, psychology, and pharmacology, with engineers in systems science, neuroinformatics, and brain-computer interfaces, to present new insights into the brain and mind to identify efficient computational theories, models and tools that will contribute to a better understanding of the brain and its functioning.*

*The opening ceremony and the invited papers will be presented in the Library Aula - "Heliade Radulescu" Hall of the Romanian Academy.*

# ORGANIZERS


Romanian Academy - National Center for Brain Research

National Neuroscience Society of Romania (NNSR)

Romanian Society for Automation and Technical Informatics (SRAIT)

University of Bucharest (UB)

# Honorary Chairman of the Conference

Constantin Popa, Romanian Academy

# Steering Committee

Ioan Dumitrache,

Romanian Academy, National Center for Brain Research, RSATI,
National University of Science and Technology Politehnica Bucharest, Romania

Maria Luiza Flonta,

University of Bucharest, Romanian Academy, Romania

Dafin Muresanu,

Romanian Academy, University of Medicine and Pharmacy Iuliu Hatieganu,
Cluj-Napoca, Romania

Bogdan Ovidiu Popescu,

University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

Leon Zagrean,

University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

# International Program Committee

Chairman: Tudor Badea, Transilvania University, Brasov & National Center for Brain Research - Romanian Academy, Bucharest, Romania

Alexandru Babes, University of Bucharest, Romania

Simona Iuliana Caramihai, SRAIT, University Politehnica of Bucharest, Romania

Daniel David, Universitatea Babes-Bolyai, Cluj-Napoca, Romania

Mircea Dumitru, University of Bucharest, Romania

Florin Filip, Romanian Academy, Romania

Mihai Moldovan, University of Copenhagen, Denmark

Mihnea Moisescu, SRAIT, University Politehnica of Bucharest, Romania

Daniela Popa, Institut de Biologie de l'Ecole Normale Superiore (IBENS), Paris, France

Aurel Popa, University of Medicine and Pharmacy of Craiova, Romania, Essen University Medical School, Germany

Maya Simionescu, Romanian Academy, Romania

Dan Tufis, Romanian Academy, Romania

Victor Voicu, Romanian Academy, Romania

Ana-Maria Zagrean, University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

# Organizing committee

Chairman: Ana-Maria Zagrean, University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

Mihnea Moisescu, SRAIT, University Politehnica of Bucharest, Romania

Violeta Ristoiu, University of Bucharest, Romania

Beatrice Radu, University of Bucharest, Romania

Bogdan Pavel, University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

Carmen Denise Mihaela Zahiu, University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

Ana-Teodora Chirila, SOMS, University of Medicine and Pharmacy Carol Davila, Bucharest, Romania

Nirvana Popescu, SRAIT, University Politehnica of Bucharest, Romania

Ioan Sacala, SRAIT, University Politehnica of Bucharest, Romania

Dragos Popescu, SRAIT, University Politehnica of Bucharest, Romania

# CONFERENCE PROGRAM

## Day 1

## Thursday, October, the 26th

Location:

Romanian Academy Library – "Heliade Radulescu" Hall, Calea Victoriei 125, Bucharest

### 9:00 – 9:15 Opening Ceremony

### 9:15 – 11:30 Plenary Session 1 – Chairs: Maria Luiza Flonta, Beatrice Radu

09:15-09:45 **Dafin Muresanu** - Brain reserve | Towards broadened understanding and concept operationalization

09:45-10:15 **Jurgen Schwarz** - Ether-à-go-go-related gene K channels modulate neuronal excitability and behavior

10:15-10:30 **Mihai Moldovan** - When axonal excitability goes bad and conduction velocity is preserved

10:30-11:00 **Daniela Popa** - Adaptive cerebello-forebrain coupling in normal and pathological conditions

11:00-11:30 **Paolo Fabene** - Epilepsy, inflammation and microbiota

11:30-11:50 Coffee break

## 11:50 – 13:30 Session 1 – Chair: Mihai Moldovan

11:50-12:10 **Mihai Moldovan** - A thought about oscillatory electroencephalographic macrostates

12:10-12:30 **Alexandra Sofonea, Miralena Tomescu** - Personality moderates intra-individual variability in EEG microstate and spontaneous thoughts

12:30-12:50 **Alina Chivu, Miralena Tomescu** - EEG microstates in mood and anxiety disorders: a meta-analysis

12:50-13:10 **Alexandru Ion Berceanu** - Insights from an fMRI study on role play: prosociality relates to lower connectivity in associated neural network

13:10-13:30 **Mihai Stancu** - Adaptive traits for temporal coding in the auditory brainstem

**13:30-14:00 Sponsor presentation**

14:00 – 14:45 Lunch

## 14:45 – 16:30 Session 2 – Chairs: Ana-Maria Zagrean, Tudor Badea

14:45-15:05 **Beatrice Radu** - A new FDA-approved antiepileptic drug, cenobamate, has pro-arrhythmogenic effects

15:05-15:25 **Lucian Hritcu** - Effects of Ceratonia siliqua L. aqueous leaves extract against Aβ1-42-induced cognitive deficit mice: Role of α7-nAChR in modulating Jak2/PI3K/Akt/GSK-3β/β-catenin cascade

15:25-15:45 **Mara Ioana Ionescu, Ana-Maria Zagrean** - The impact of the gut microbiome alteration during pregnancy on both maternal health and offspring neurodevelopment

15:45-16:05 **Raluca Pașcalău, Tudor Badea** - Human retinal cell types anatomy during fetal development

16:05-16:25 **Vladimir Muzyka, Tudor Badea** - Anatomic and Physiologic Characterization of Neurons Expressing Tusc5

16:25-16:45 **Vlad-Alexandru Toma** - Non-determining neuroglial cells in the central nervous system of the crow.

16:45-17:00 Coffee break

## 17:00 – 19:00 Session 3 – Chairs: Aurel Popa, Alexandru Babes

17:00-17:20 **Aurel Popa** - Genetic conversion after stroke. A new therapeutic approach?

17:20-17:40 **Roxana Surugiu** - Extracellular Vesicles (EVs) as crucial predictive factors and therapeutic agents in stroke

17:40-18:00 **Florian Armasescu, Bogdan Amuzescu** - Effects of acute near-infrared laser exposure on primary sensory neuron excitability and gating properties of heterologously expressed voltage-dependent Na+ channels

18:00-18:20 **Alexandru Babes** - Modulation of TRPM8 function by the prostacyclin receptor: involvement of Gq/11 proteins

18:20-18:40 **George Oprita, Alexandru Babes** – The thyroid hormone triiodothyronine potentiates TRPM3 activity in mouse dorsal root ganglia neurons

18:40-19:00 **Razvan Boiangiu** - In silico and in vivo investigation of the 6-hydroxy-L-nicotine and cotinine biological effects

## 19:00-19:30 SNN General Assembly

## 19:30 Welcome cocktail

Location: Romanian Academy Library, Calea Victoriei 125, Bucharest

## Day 2

## Friday, October of 27th

Location:

Romanian Academy Library – "Heliade Radulescu" Hall, Calea Victoriei 125, Sector 1, Bucureşti

### 9:00 – 11:00 Session 4 – Chairs: Dafin Muresanu, Bogdan Ovidiu Popescu

9:00-9:30 **Dafin Muresanu** - The impact of microcirculation on disability prevention

9:30-10:00 **Bogdan Ovidiu Popescu** - Hypertensions and neurodegenerative diseases

10:00-10:30 **Cristina Tiu** – Advances in acute stroke phase treatment in Romania

10:30-11:00 **Amos Korczyn** - Is Alzheimer's disease a disease?

11:00 – 11:20 Coffee break

### 11:20 – 13:20 Plenary Session 2 – Chairs: Ioan Dumitrache, Tudor Badea

11:20-11:50 **Dorin Comaniciu** - Artificial Intelligence and Neuroscience: The Road Ahead

11:50-12:20 **Valentin Dragoi** - Cortical Circuits for Information Processing and Decision Making

12:20-12:50 **George Dragoi** - Neuronal Ensembles Underlying Internally Generated Representations

12:50-13:20 **Samer Hattar** - How Does the Retina Influence the Brain and Behavior

13:20 – 14:20 Lunch

### 14:20 – 16:00 Session 5 – Chairs: Ioan Dumitrache, Simona Caramihai

14:20-14:40 **Ioan Dumitrache, Simona Caramihai, Dragoș Popescu** – Some aspects on Brain modelling as a Complex Adaptive System

14:40-15:00 **Ionuț Predulescu, Ioan Dumitrache, Simona Caramihai** – Automated system for detection of cortical diseases using EEG analysis, virtual reality and artificial intelligence

15:00-15:20 **Stefan Trausan-Matu** - Analysis of Natural Language Generation with Deep Neural Networks

15:20-15:40 **Sorin Grigorescu** - AI-based Operating System for Enhanced Autonomy in Ground and Aerial Robotics

15:40-16:00 **Maria Mernea** - Bioinformatics analysis of natural compounds with anxiolytic effect using target proteins interaction networks

**16:00 – 16:30 Sponsor presentation**

16:30 –16:50 Coffee break

## 16:50 - 18:50 Session 6 – Chair: Andrei Miu

16:50-17:10 **Andrei Miu** - Emotion regulation and psychopathology: Neural and neuroendocrine mechanisms

17:10-17:30 **Alexia Pinoșanu** - How can we assess emotion regulation in the laboratory? A focus on designs with ERP and autonomic measures

17:30-17:50 **Andra Vlaicu** - Emotion regulation and cortisol reactivity: Preliminary data from a prospective study

17:50-18:10 **Simina Pitur** - Childhood maltreatment and emotion regulation: Neural markers of risk for psychopathology?

18:10-18:30 **Gabriela Marcu** - QEEG Neuromarkers of Complex Childhood Trauma - Pilot Study and Preliminary Data

18:30-18:50 **Ramona Ceciu** - Cognitive-Behavioral and Socio-Cultural Correlates of Emotion Regulation

# Day 3

# Saturday, October 28th

Location: Faculty of Biology, Bucharest University, Splaiul Independentei 91-95, Bucharest

## 9:00-10:40 Session 7 **Chairs: Violeta Ristoiu, Bogdan Amuzescu**

9:00-9:20 **Shurooq Shaher, Bogdan Amuzescu** - NMDAR activation, cytotoxicity and oxidative stress induced by aspartame in native and heterologously expressed cell lines

9:20-9:40 **Violeta Maria Caragea** - Goal-directed skilled action in rats is modulated by the dopamine receptors found in the fastigial nucleus

9:40-10:00 **Roberta Stoica** - Effects of two radiotherapy techniques at the cerebrovascular endothelium level

10:00-10:20 **Tudor Selescu** - Role of four thermosensitive TRP ion channels in thermal preference of male and female mice

10:20-10:40 **Sebastian Isac, Ana-Maria Zagrean** - The cumulative detrimental effect of the general anaesthesia on the immature brain after perinatal asphyxia

10:40-11:00 Coffee break

## 11:00 – 12:30 Blitz oral presentations & Posters session

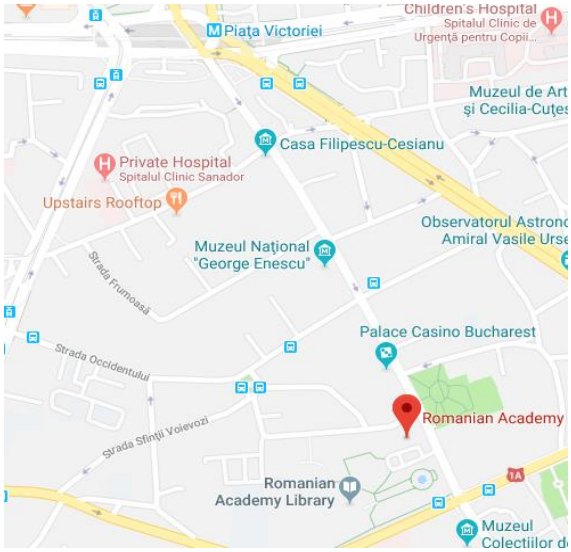
1. Alina Ostafi, Florentina Pluteanu - The effect of nerve growth factor on vascular fibroblasts
2. Ana-Maria Matota - Hyperexcitability in a Rat Model of Absence Epilepsy: Higher EEG Reactivity During Deep Anesthesia
3. Andreea Cercel - Axonal growth in the young adult brain after an ischemic stroke
4. Andreea Violeta Grosu, Violeta Ristoiu - Spared nerve injury model of neuropathic pain induces a specific microglia activation pattern at the spinal cord level
5. Andrei Bordeianu - Burst-Suppression EEG Reactivity in Detecting Post-Ischemic Brain Injury: An Experimental Rat Study
6. Andrei Teodor Bratu - Role of prefrontal cortex in processing aversive auditive and visual stimuli
7. Claudia-Ioana Draghici - Cenobamate upregulates P-glycoprotein, while downregulates tight junction proteins in accordance with blood-brain barrier permeabilization
8. Claudia Moldoveanu - Histological and immunohistochemical aspects of the central nervous system in Alzheimer's disease and the study of antioxidant action as a prophylactic strategy

9. Cristian Ciotei - The trials and errors of studying the impact of gestational gut microbiome disturbance and perinatal asphyxia on the neurodevelopmental reflexes of rat offspring
10. David Ionut Bacioiu - Crosstalk Between TRPA1, TRPM8 and EGF Receptor in Glioblastoma
11. Florin Zamfirache - The impact of sertraline and social rehabilitation on a long-term depression study on rats
12. Ileana Daria Dobre - Retinal Ganglion Cell types of the Mouse Area Centralis
13. Kevin Boboc – How Continuous Use of Ion Channel Blockers Affects Microglial Morphology and Function in a Murine Alzheimer's Model.
14. Laurentiu Tofan - Exploration of the brain connectivity in patients with pharmacoresistant epilepsy and visual hallucinations
15. Melania Magercu - Iba1 down-regulation in primary rat microglia: differential effects induced in defined-medium vs serum-supplemented in vitro systems
16. Stefan Alexandru Tirlea - Cost-Effective Alternative for Long-Term Sleep Analysis in Freely Moving Mice
17. Valentin Ionescu - An Equation for an Impossible Task. Does the Brain-Personality System behave in a Thermodynamic Manner? Hot Brain Hypothesis
18. Vlad Morozan - Investigating the relationship between default EEG macrostate reactivity and anaesthetic depth in response to photic stimulation: a rat model

## 12:30-13:00 Closing ceremony

# Conference Venue

The conference attendance will be organized in the "Heliade Radulescu" hall of the Romanian Academy Library, Romanian Academy - Calea Victoriei 125, Bucharest, Romania




# Sponsors

# Oral Presentations

**O1. The mammalian node of Ranvier: structure and function**

***Schwarz, Juergen R.[1]***

*[1] Hamburg University*

In myelinated nerve fibers, action potentials are generated at nodes of Ranvier located at regularly spaced interruptions of the myelin sheath. They form narrow gaps with small rings of axolemma freely exposed to the extracellular space. The node contains nodal, paranodal and juxtaparanodal parts. The mammalian node contains a high density of Na channels and K-selective leakage channels. Voltage-dependent Kv1 channels are only present in the juxta-paranode. Recently, the leakage channels have been identified as K2P channels (TRAAK, TREK-1). K2P channels are K-selective "background" channels, characterized their ability to be activated, e.g. by temperature, mechanical stretch, or arachidonic acid. I will discuss two functions of the nodal K2P-mediated conductance. First, at body temperature K2P channels have a high open probability, thereby changing the resting potential to about -85 mV. This negative resting potential reduces steady-state Na channel inactivation and ensures a large Na inward current upon a depolarizing stimulus. Second, the involvement of the K2P conductance in nodal action potential repolarization. The identification of nodal K2P channels is exciting since it shows that the nodal K conductance is not a fixed value but can be changed, it can be increased or decreased by a broad range of K2P modulators, thereby modulating e.g. the resting potential. I will exemplify the functional importance of nodal K2P channels by describing in more detail the function of the K2P conductance increase by increasing the temperature to 37°C.

**O2. Personality moderates intra-individual variability in EEG microstate and spontaneous thoughts**

***Tomescu, Miralena I.[1]*; Papasteri, Claudiu[1]; Berceanu, Alexandru I.[1]; Sofonea, Alexandra[1]; Carcea, Ioana[1]***

*[1] CINETic Center, Departement of Research and Development, National University of Theatre and Film "I.L. Caragiale", Bucharest, Romania*

Variability in brain activity that defined behavioral and physiological states cannot resolve is often considered noise and controlled as a covariate in research. However, studying intra-individual variability in brain function can provide valuable insights into the dynamic nature of the brain. To explore this, we conducted a study on 43 participants analyzing the EEG microstate dynamics and self-reported spontaneous mental activity during a five-minute freely mind-wandering state on two separate days. Our results showed that the associations between EEG microstates and spontaneous cognition significantly changed from one day to another. Moreover, microstate changes were associated with changes in spontaneous cognition. Specifically, inter-day changes in Verbal thoughts about Others and future Planning were positively related to bottom-up sensory network-related microstate changes and negatively associated with top-down, attention, and salience network-related microstates. In addition, we find that personality traits are related to inter-day changes in microstates and spontaneous thoughts. Specifically, extraversion, neuroticism, agreeableness, and openness to experience traits moderated the relationship between EEG microstates and spontaneous thoughts' daily changes. Our study provides valuable information on the dynamic changes in the EEG microstate-spontaneous cognition organization, which could be essential for developing interventions and treatments for neuropsychiatric disorders.

**O3. EEG Microstates in Mood and Anxiety Disorders: A Meta-analysis**

***Chivu, Alina[1]; Pascal, Simona A.[2]; Damborska, Alena[3]; Tomescu, Miralena I.[1]****

*[1] CINETic Center, Departement of Research and Development, National University of Theatre and Film "I.L. Caragiale", Bucharest, Romania*
*[2] Faculty of Psychology and Educational Sciences, Department of Applied Psychology and Psychotherapy, University of Bucharest, Bucharest, Romania*
*[3] Department of Psychiatry, Faculty of Medicine, Masaryk University and University Hospital Brno, Brno, Czech Republic*

To reduce the psycho-social burden increasing attention has focused on brain abnormalities in the most prevalent and highly co-occurring neuropsychiatric disorders, such as mood and anxiety. However, high inter-study variability on these patients shows inconsistent and contradictory alterations in the fast temporal dynamics of large-scale networks as measured by EEG microstates. Thus, in this meta-analysis, we aim to investigate the consistency of these changes to better understand possible common neuro-dynamical mechanisms of these disorders. In the systematic search, twelve studies investigating EEG microstate changes in patients with mood and anxiety disorders and subjects with subclinical depression were included in this meta-analysis, adding up to 787 participants. The results suggest that EEG microstates consistently discriminate mood and anxiety impairments from the general population in patients and subclinical states. Specifically, we found a small significant effect size for B microstates in patients compared to healthy controls, with larger effect sizes in unmedicated patients with comorbidity. In a subgroup meta-analysis of ten mood disorder studies, microstate D showed a significant effect size for decreased presence. When investigating only the two anxiety disorder studies, we found a significantly small effect size for the increased microstate A and a medium effect size for decreased microstate E (one study). However, more studies are needed to elucidate whether these findings are diagnostic-specific markers.
Results are discussed about the functional meaning of microstates and possible contribution to an explanatory mechanism of overlapping symptomatology of mood and anxiety disorders.

**O4. Insights from an fMRI study on role play: prosociality relates to lower connectivity in associated neural network**

***Berceanu, Alexandru Ion[1]*; Mihaela, Onu[1]; Papsteri, Claudiu[1]; Sofonea, Alexandra[1]; Boldasi, Romina[1]; Carcea, Ioana[1]***

*[1] National University of Theatre and Film "I.L. Caragiale"*

Role-playing, one of the key processes in acting requires a range of social, cognitive, and affective skills of concern to neuroscience, including complex planning, theory of mind, emotional control, and social cognitive processes like empathy. Together, these skills promote pro-social, cooperative behavior via empathic concern for others. Training in acting was previously reported to increase pro-social attitudes and to improve memory and emotion regulation skills. One key question is what impact does acting training have on brain activity to induce specific changes in behavior? To answer this question, we performed a functional MRI study on 20 subjects without prior training in theatre. A 3T Siemens Skyra-MR scanner was used to perform resting state functional acquisitions after role-play training or control intervention. Independent component analysis tool (ICA) was used to obtain the neural networks maps (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/). Our results showed decreased connectivity in experimental subjects compared to controls, for one of the executive control networks. Moreover, voxel-wise correlation analysis between the strength of network connectivity and behavioral scores on the Adult Prosocialness Scale revealed a strong negative linear relationship between them. In other words, the higher scores on the prosocial attitude scale, the lower the intra-network connectivity of one of the identified networks. In conclusion, a decreased resting functional connectivity in specific neural network connectivity might be a trait for improved pro-social attitude in humans. Our findings suggest that acting training may induce this neural functionality change.

**O5. A new FDA-approved antiepileptic drug, cenobamate, has pro-arrhythmogenic effects.**

***Radu, Beatrice Mihaela[1]; Amuzescu, Bogdan[1]***

*[1] Department of Anatomy, Animal Physiology and Biophysics, Faculty of Biology, University of Bucharest, Bucharest, Romania*

Cenobamate (CBM) is a novel antiepileptic drug approved by US FDA in November 2019 and by EMA in March 2021 that is commercially available as Xcopri® and Ontozry®. The drug is recommended for treatment of adults with focal onset seizures and particularly for epilepsy resistant to other antiepileptic drugs. CBM has two neuronal pharmacological targets, being a positive allosteric modulator of $GABA_A$ receptors and an inhibitor of Na+ channels, particularly of the late persistent Na+ current component. The goal of our study is to test the drug safety of this antiepileptic by evaluating its pro-arrhythmogenic effects. To this purpose, we performed whole-cell patch-clamp recordings on HEK293T cells with persistent/inducible expression of human cardiac ion channel isoforms hNav1.5 ($I_{Na}$), hCav1.2 (α1c+β2+ α2δ1) ($I_{CaL}$), hKv7.1+minK ($I_{Ks}$), and hKv11.1 (hERG) ($I_{Kr}$). We demonstrated that CBM (200 μM) inhibits the peak $I_{Na}$ by 69.5±16.6%, and the peak $I_{CaL}$ by 42.8±15.3, and also reduces IKs and IKr (at 20 μM). These effects might explain the QT shortening effects. This pronounced $I_{Na}$ inhibition exerted by CBM raises the possibility for the drug at clinically relevant concentrations to trigger reentry arrhythmias in preexisting pathological myocardium with conduction heterogeneity in a narrow interval of favorable conditions. Such a possibility, which is common for class I antiarrhythmics, requires increased awareness and careful clinical monitoring of patients with preexisting organic heart pathology during initiation of CBM therapy. *This study was financed by Uefiscdi grant PCE39/2022.*

**O6. Effects of Ceratonia siliqua L. aqueous leaves extract against Aβ1-42-induced cognitive deficit mice: Role of α7-nAChR in modulating Jak2/PI3K/Akt/GSK-3β/β-catenin cascade**

***Hritcu, Lucian[1]*; El Sayed, Nesrine S.[2]; Abidar, Sara[3]; Nhiri, Mohamed[3]; Ibrahim, Weam W.[2]***

*[1] Alexandru Ioan Cuza University of Iasi*
*[2] Cairo University*
*[3] Abdelmalek Essaadi University*

Alzheimer's disease (AD) is a multifactorial neurodegenerative disorder attributed to several etiological factors including cholinergic dysregulation, neuroinflammation, oxidative stress, β-amyloidogenesis, and tauopathy. This demands the search for multitarget drugs, especially of natural sources owing to their pleiotropic activities and low adverse effects. The present study was conducted to investigate the cognitive-improving potential of Ceratonia siliqua L. (Cs) extract compared with donepezil, an acetylcholinesterase inhibitor, on AD-like pathological alterations induced by single intracerebroventricular amyloid-β1-42 (Aβ1-42) injection in mice. Aβ1-42-injected mice were treated with Cs (100 mg/kg/day, po) with or without methyllycaconitine (MLA; 1 mg/kg/day, ip), an α7-nAChR antagonist. Aβ1-42-injected animals demonstrated an elevation of hippocampal Aβ1-42, p-Tau, and acetylcholinesterase. They also showed a decline in phosphorylated levels of Jak2, PI3K, Akt, and GSK-3β, leading to induction of neuroinflammation and oxidative stress. Noteworthy, Cs improved the histopathological and behavioral variables in addition to mitigating AD hallmarks. It also exerted neuroprotection by reducing NF-κBp65 and TNF-α, while elevating Nrf2 and HO-1, along with stabilizing β-catenin under the impact of Jak2/PI3K/Akt/GSK-3β signaling. These beneficial effects of Cs were abrogated by MLA co-administration signifying the α7-nAChR involvement in Cs-mediated effects. Therefore, Cs can ameliorate Aβ42-induced neurodegeneration by modulating Jak2/PI3K/Akt/GSK-3β/β-catenin axis in an α7-nAChR-dependent manner.

Acknowledgment: Lucian Hritcu was supported by a grant of Ministery of Research, Innovation and Digitization, CNCS-UEFISCDI, project number PN-III-P4-PCE-2021-1692, within PNCDI III.

**O7. The impact of the gut microbiome alteration during pregnancy on both maternal health and offspring neurodevelopment**

***Ionescu, Mara I[1]*; Zahiu, Carmen[1]; O'Mahony, Siobhain[2]; Zagrean, Ana-Maria[1]***

*[1] Carol Davila University of Medicine and Pharmacy, Bucharest, Romania*
*[2] University College Cork, Cork, Ireland*

Introduction: In recent years, research has unveiled the remarkable influence of the gut microbiome on various facets of human health. During pregnancy, profound physiological changes occur in both the mother and her developing fetus. Among these transformations, alterations in the gut microbiome have emerged as a pivotal player, impacting maternal health and potentially imprinting long-lasting effects on offspring neurodevelopment.
Aim: Consequently, our research endeavors seek to elucidate the intricate interconnections between the maternal gut microbiome during pregnancy, maternal health, and the developmental trajectories of offspring.
Materials and methods: In pursuit of this aim, we employed female Wistar rats as our experimental subjects and implemented two distinct models to induce prenatal alterations in the gut microbiome: gestational restraint stress and antibiotic administration. Furthermore, we explored the potential beneficial effects of probiotic administration. Recognizing the known adverse consequences of perinatal asphyxia (PA) on offspring, we also investigated the combined impact of maternal antibiotic administration and PA on the neurodevelopmental reflexes of offspring.
Results: Our preliminary findings indicate that antibiotic administration may enhance the vulnerability of the immature brain to PA, as evidenced by alterations in neurodevelopmental reflexes. Prenatal stress led to reduced maternal care and elevated anxiety levels in dams following weaning. Additionally, we observed impaired cognitive function in the offspring. Encouragingly, probiotic treatment demonstrated the capacity to mitigate these effects.
Conclusion: Collectively, these findings suggest that prenatal alterations in the gut microbiome can negatively affect maternal caregiving behavior and influence offspring cognitive function. Probiotic administration during pregnancy may represent a promising and safe strategy for alleviating these effects.

**O8. Retinal cell types development in midgestational human fetuses**

***Pașcalău, Raluca[1]*; Șușman, Sergiu[2]; Badea, Tudor C.[1]***

*[1] Transilvania University of Brașov*
*[2] Iuliu Hatieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania*

Compared to the mouse where the anatomical, molecular and physiological identities of these cell types and their development has been thoroughly investigated, the knowledge of the human retina usually stops at the histological level with a few exceptions mainly based on adult samples. Human retina development differs from the mouse in terms of timeline and macular specialization. Our aim was to characterize human retinal cell types' anatomy and molecular markers expression at different fetal life stages. The study was conducted on human fetal retinas collected 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 staining was performed and images taken at a confocal microscope and processed using ImageJ software. In retinal samples coming from second and third semester cases, the major retinal layers were delineated, except for the outer plexiform layer. The inner plexiform layer developed its sub-lamina. Markers for all the major retinal cell types were positive, with differences in fluorescence intensity across ages and retinal eccentricities. As an orientation for the local development stage of each section we used cell proliferation and synaptic markers as staining for blood vessels and astrocytes. In the retinal ganglion cells, the expression profile of different transcription factors also varied with age and central-peripheral location. In conclusion, human retina intrauterine development evolves through advanced stages corresponding to mouse postnatal development stages.

**O9. MOUSE RETINAL GANGLION CELLS AND SOMATOSENSORY PROJECTION NEURONS EXPRESSING TUSC5/TRARG1**

***Muzyka, Vladimir V.[1]*; Badea, Tudor C.[1]***

*[1] Transilvania University of Brașov*

Recently we have found Tusc5 (tumor suppressor candidate 5, also known as Trarg1) gene and Tusc5 protein expression in retina (Sajgo et al. 2017; Muzyka et al. 2018), projection somatosensory neurons, and neurons of olfactory and auditory systems (unpublished data) in mouse. 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. In mouse retina, Tusc5 is expressed specifically in a subset of Pou4f1+ retinal ganglion cells (RGCs), which are anatomically characterized by small dense "bushy" dendritic arbors laminating exclusively between the characteristic ON and OFF ChAT-positive bands of the inner plexiform layer (IPL) of the retina, and by 'bursty' unsustained responses to the light stimulation with strong surround suppression of the receptive field (Muzyka et al. 2018; Goetz et al. 2022). Our current work will further address the RGC subtype specificity of expression of Tusc5 as well as its cell biological and physiological role in these retinal neurons. In addition, we discovered in terms of cell compartments occupied by Tusc5 protein, its localization in intracellular membrane compartments of endosomal trafficking pathway.
In mouse somatosensory system, Tusc5 is expressed in both dorsal root ganglia (DRG) and trigeminal ganglia (TGG), with more than a half of the neurons being Tusc5+ there. Nociceptor subpopulation markers (Periherin, CGRP, IB4) all demonstrate considerable intersect with Tusc5 expression in terms of individual

cells, while cold receptors (TrpM8+) and proprioceptors (Parvalbumin+) seem to predominantly lack Tusc5 protein expression in the adult mouse DRGs/TGGs. Mechanoreceptors (NF200+) possess a fraction of Tusc5+ neuronal cells. Further studies are performed by our research group to uncover the precise repertoire of Tusc5-expressing neuronal subtypes, as well as the functional significance of that molecule there.
Funding: Unitatea Executiva pentru Finantarea Invatamantului Superior, a Cercetarii, Dezvoltarii si Inovarii Grant PN-III-P4-PCE-2021-0333

**O10. NON-DETERMINING NEUROGLIAL CELLS IN THE CENTRAL NERVOUS SYSTEM OF THE CROW**

***Toma, Vlad-Alexandru[1]*; Nutu, Alexandra Maria[2]; Dume, Bogdan Razvan[1]; Barbu, Lucian[1]; Nicolae, Dragos[3]***

*[1] Babes-Bolyai University*
*[2] Bucharest University, Faculty of Biology*
*[3] Institute of Biological Research, Cluj-Napoca)*

Introduction. Glial origin of the newborn neurons and continuous replacement of them with new cells in adulthood in songbirds as well as mammalians and other vertebrates was demonstrated by immunohistochemistry, confocal microscopy, PET, neuronal tracing, and other various tagging protocols to highlight the line between the origin and final place of the newborn/replaced cell. Here, we depict our recent findings about the cell biology of the presumptive neuronal replacement process in the developing, juvenile, and adult brains of C. frugilegus.
Materials and methods. Different stages of brain development were taken into research and processed for electron microscopy, immunohistochemistry, histology, and confocal microscopy for coronal sections.
Results. Interestingly, the juvenile and adult brains but no newborn brains of the crow had clusters, located in the hyperstriatum ventralis of about 6-12 cells with a large central cell DCX+, PCNA+, delineated by other cells with membrane. Cells around the central cell seem like oligodendrocytes whereas the central cell was estimated to be a migratory neuron-like cell with atypical features such as fatty acids oxidation properties as a prominent metabolic pathway (MCT4+, dark ER, large and dynamic mitochondria, and frequent peroxisomes). The cluster would be connected to other non-determining structures from the hyperstriatum ventralis by a path formed by the extensions of the cells adjacent to the central cell.
Conclusion. Our findings suggest a new pattern of cellular organization in birds' brains and these aspects are supportive data in neuronal replacement theory (Nottebohm) and neurogenesis studies. However, these findings still maintain the reported cells as non-determining cell types with neuron/oligodendrocyte features.
Acknowledgments. This work was supported by a grant of the Ministry of Research, Innovation, and Digitization, CNCS - UEFISCDI, project number PN-III-P1-1.1-TE-2021-0159, within PNCDI III (TE60/2022), by a Young Researcher Grant no. 2939/22.06.2023-Babeș-Bolyai University as well as by „Maya & Nicolae Simionescu" Postdoctoral Fellowship.

**O11. Extracellular Vesicles (EVs) as crucial predictive factors and therapeutic agents in stroke**

***Surugiu, Roxana[1]*; Burdusel, Daiana[1]; Ruscu, Mihai[1]; Aldea, Madalina[1]; Cercel, Andreea[1]; Abuzan, Mihaela[1]; Popa, Aurel[1]; Hermann, Dirk[2]***

*[1] University of Medicine and Pharmacy Craiova*
*[2] University of Duisburg-Essen*

Stroke, a leading cause of death and disability worldwide, demands continued research to enhance both its prediction and treatment. In recent years, extracellular vesicles (EVs) have emerged as pivotal players in the field of stroke, offering promising avenues for both prediction and intervention. EVs are nanosized lipid bilayer vesicles secreted by various cells, including neurons, glial cells, and endothelial cells, and they carry cargo that reflects the physiological state of their parent cells.
EVs have gained significant attention as potential predictive factors for stroke. Several studies have demonstrated that specific miRNAs, proteins, and lipids encapsulated within EVs can serve as biomarkers for stroke risk assessment. By analyzing the composition and abundance of these molecules in circulating EVs, researchers can potentially identify individuals at higher risk of stroke, enabling early preventive interventions.
Moreover, EVs hold immense therapeutic promise in stroke management. Preclinical studies have revealed their neuroprotective and regenerative properties. EVs derived from stem cells, for instance, can modulate neuroinflammation, enhance neuroprotection, and stimulate endogenous repair mechanisms. In conclusion, EVs offer a multifaceted approach to stroke research and management. As our understanding of EVs continues to deepen, their integration into clinical practice may hold the key to improving stroke outcomes and reducing its devastating impact on individuals and healthcare systems.

**O12. Effects of acute near-infrared laser exposure on primary sensory neuron excitability and gating properties of heterologously expressed voltage-dependent Na+ channels**

***Armasescu, Florian[1]; Amuzescu, Bogdan[1]*; Gheorghe, Roxana-Olimpia[1]; Ghenghea, Mihai[1]; Mihailescu, Dan Florin[1]; Ciurea, Jean[2]***

*[1] University of Bucharest*
*[2] "Arseni-Bagdasar" Neurosurgery Hospital Bucharest*

Photobiomodulation represents an interesting alternative physical therapy promoting wound healing, reduction in inflammation, pain, apoptosis, used for over 50 years in different medical applications, including neurological or psychiatric disorders such as stroke, brain trauma, depression, dementia, and neurodegenerative diseases. In advanced stages of Parkinson's disease encouraging results have been obtained via both transcranial or deep fiber-optic based application of near-infrared (NIR) light. Our study assessed the effects of brief exposure to NIR radiation generated by a 808.5 nm diode laser and applied via a multimodal fiber prolonged with a sharp transparent tip to enzyme-dissociated cultured adult rat primary sensory neurons and HEK293 cells stably transfected with human Nav1.5 approached via whole-cell patch-clamp. A series of voltage-clamp protocols were applied initially and repeated after 3 min of laser exposure or control conditions. In a majority of primary

sensory neurons assessed by ruptured patch-clamp laser exposure induced a resting potential depolarization (6.5±1.0 mV) that reverted to initial levels at 10 min after stop of laser beam, while in gramicidin-perforated experiments this effect was not present. Independently, laser exposure increased the number of action potential spikes elicited by external current stimuli. For HEK293 cells stably expressing hNav1.5, groups of n=10 cells were tested in laser/control experiments with either a standard pipette solution (groups A/B – laser/control) or a complex solution containing Na2ATP 4 mM (groups C/D – laser/control). The peak Na+ current amplitude was steady or slightly increased in group A (111.2±14.9% change, mean±SEM) and C (91.2±5.2%), and decreased in control groups D (81.1±6.8%) and B (70.6±10.4%). Remarkably, the slow time constant of inactivation remained steady upon laser exposure – group A (100.4±5.8%) and C (106.0±11.3%), and increased in control groups – group B (130.7±17.8%) and to a lesser extent in group D (112.5±6.2%). A possible interpretation of these results may rely on the fact that NIR exposure facilitates ATP production particularly by activation of the cytochrome oxidase complex, maintaining an adequate state of phosphorylation of Na+ channels.

**O13. Modulation of TRPM8 function by the prostacyclin receptor: involvement of Gq/11 proteins**

***Trif, Cosmin[1]; Banica, Alexandra[1]; Manolache, Alexandra[2]; Anghel, Andreea[1]; Hutanu, Debora[2]; Stratulat, Teodora[2]; Oprita, George[2]; Petrescu, Stefana[1]; Tunaru, Sorin[1]; Babes, Alexandru[2]****

*[1] Institute of Biochemistry of the Romanian Academy*
*[2] University of Bucharest*

The Transient Receptor Potential Melastatin subtype 8 (TRPM8) receptor-channel is involved in innocuous cold sensing and has a potent anti-inflammatory action. Its activation by lower temperature or chemical agonists such as menthol and icilin induces analgesic effects, reversing hypersensitivity and reducing chronic pain. On the other hand, prostacyclin (PGI2) enhances pain and inflammation by activating the prostacyclin receptor (IP-R). Due to the critical roles of TRPM8 and IP-R in the regulation of inflammatory pain, and considering their overlapping expression pattern, we analyzed the functional interaction between human TRPM8 and IP-R.
We employed transient expression of human TRPM8 and IP-R in HEK293T cells and performed intracellular calcium and cAMP measurements. Additionally, we cultured neurons from the dorsal root ganglia of mice and determined the increase in intracellular calcium triggered by TRPM8 agonist, icilin, in the presence of the IP-R agonist, cicaprost, IP-R antagonist, CAY10144 and the Gq/11 inhibitor YM254890.
Our results demonstrate that the activation of IP-R by selective agonists, such as cicaprost, beraprost, and iloprost, inhibits TRPM8 independently of the Gs-cAMP pathway. The potent inhibition of TRPM8 by IP-R involves Gq/11 coupling of IP-R. These effects were also observed in neurons isolated from the dorsal root ganglia (DRGs) of mice.
Our results demonstrate that an unusual signaling pathway of IP-R, namely the coupling to Gq/11 proteins, inhibits TRPM8 which may contribute to a better understanding of the role of TRPM8 and IP-R in the regulation of pain and inflammation.

**O14. The thyroid hormone triiodothyronine potentiates TRPM3 activity in mouse dorsal root ganglia neurons**

***Oprita, George[1]*; Domocos, Dan[2]; Babes, Alexandru[3]***

*[1] Bucharest University, Faculty of Biology*
*[2] Institute of Biochemistry of the Romanian Academy*
*[3] University of Bucharest*

Thyroid hormones, thyroxine (T4) and its active form, triiodothyronine (T3) are essential for metabolism and tissue development, by influencing cellular processes, like mitochondrial activity, protein synthesis and degradation. Some thyroid disorders, including hypothyroidism and autoimmune afflictions have been linked with numerous types of chronic pain, including small fiber neuropathy. Transient receptor potential (TRP) channels are key ion channels involved in peripheric pain transduction in the dorsal root ganglia (DRG) neurons. Although receptors for T3 are present in the DRG, no studies have investigated how these hormones influence peripheric neuronal activity and how alterations may lead to pain. This work focuses on four TRP channels, TRPV1, TRPA1, TRPM8 and TRPM3 and how chronic or acute T3 treatment modifies their function in DRG primary cultures from adult male C57/Bl6 mice. The investigation method used was calcium microfluorimetry and a pharmacological protocol employing specific agonists; capsaicin for TRPV1, allyl isothiocyanate for TRPA1, WS-12 for TRPM8 and CIM0216 for TRPM3. Calcium influx was quantified both as area under the curve and fluorescence modification. The main finding revolves around TRPM3. We have found that 24h, but not 3h treatment with T3 (500nM) leads to significant TRPM3 potentiation. The results suggest that triiodothyronine takes at least 24 hours to exert its effects on dorsal root ganglia neurons. These findings give a starting point in looking for more interactions. Further experiments could focus on electrophysiology, investigating firing frequencies or individual action potentials of neurons treated with T3.

**O15. In silico and in vivo investigation of the 6-hydroxy-L-nicotine and cotinine biological effects**

***Boiangiu, Razvan S.[1]*; Brînză, Ion[1]; Mihășan, Marius[1]; Hritcu, Lucian[2]***

*[1] Department of Biology, Alexandru Ioan Cuza University of Iasi*
*[2] Alexandru Ioan Cuza University of Iasi*

The impact of two structurally related nicotinic intermediates, namely cotinine (COT) and 6-hydroxy-L-nicotine (6HLN), was evaluated on memory deficits, anxious-like behavior and oxidative stress in two animal models of Alzheimer's disease (AD): a rat (Rattus norvegicus) model induced by brain delivery of beta-amyloid fragment 25-35 (Aβ25-35) and a zebrafish (Danio rerio) model induced by scopolamine (SCOP). COT and 6HLN were administered chronically to Aβ25-35-treated rats and acutely to SCOP-treated zebrafish and their performances were assessed using specific in vivo tasks. The oxidative stress parameters and acetylcholinesterase (AChE) activity were measured in the animals' brains. Using in silico tools, we attempted to associate behavioral outcomes with the calculated binding potential of these compounds into two different allosteric binding sites of α4β2 nAChRs. Aβ25-35 and SCOP decreased the memory performances and increased the anxiety-like behavior in the in vivo

assays and increased the oxidative stress and AChE activity in the brain of rats and zebrafish, respectively. As compared to nicotine, the COT and 6HLN ameliorated, more effectively, the memory deficits and anxiety caused by Aβ25-35 or SCOP. Also, the nicotinic derivatives significantly reduced the oxidative stress and AChE activity in the brain of Aβ25-35- or SCOP-treated animals. Furthermore, we showed that COT and 6HLN indeed bind to α4β2 nAChRs with similar or even higher energy than nicotine and that the α4-β2 binding site is preferred over the α4-α4 binding site. COT and 6HLN might improve memory and anxiety-like behavior by modulating cholinergic activity and thus might represent new neuropharmacological agents in AD.

**O16. Analysis of Natural Language Generation with Deep Neural Networks**

***Trausan-Matu, Stefan[1]***

*[1] Polytechnic University of Bucharest*

Deep neural networks achieved remarkable performances for natural language generation (NLG), probably the most well known example being chatGPT. The paper will analyse the achievements and limits of such systems. There are two classes of problems that NLG systems encounter. A first class of problems are specific not only to NLG, they occur also to other applications of machine learning: explainability and ethics. The second class of problems were revealed especially during the use of chatGPT and related systems: hallucinations, repetitions, lack of reasoning.

**O17. Bioinformatics analysis of natural compounds with anxiolytic effect using target proteins interaction networks**

***Mernea, Maria[1]*; Gamazeliuc, George[2]; Gurguţă, Andreea-Mădălina[1]***

*[1] University of Bucharest*
*[2] The National Institute of Research and Development in Mechatronics and Measurement Technique (INCDMTM)*

As classical anxiolytic drugs administered have unpleasant side effects, the identification of a treatment based on plants is very opportune. The literature mentions numerous plants that present anxiolytic effects, but the action mechanisms of their bioactive compounds are not well described. The present study aimed to understand the mechanisms of action of some natural anxiolytic compounds through an approach based on protein-protein interaction networks. We used bioinformatics methods to identify 19 anxiolytic natural compounds. Their molecular targets associated with anxiety were retrieved from literature. Additional molecular targets were predicted using relevant databases and were confirmed based on scientific publications. The resulting list comprised 59 targets. The targets list was enriched with 14 known targets in anxiety according to therapeutic target databases. The targets were used to construct a network of functional interactions. The analysis of the results showed three mechanisms of action of the natural compounds: modulation of synaptic transmission, modulation of inflammation pathways and interference with cancer pathways. All of these pathways are related to anxiety, and modulating them could have beneficial effects. The modulation of synaptic transmission involved modulation of synapses mediated by different neurotransmitters (acetylcholine, dopamine, glutamate, glycine, GABA), as well as serotonin transport or modulation of cannabinoid signaling. Among all the analyzed compounds, apigenin modulates all identified action pathways. Apigenin is a fairly accessible compound, being present in numerous aromatic plants or some vegetables. Other compounds worth mentioning for their complex action and accessibility are caffeic acid, present in chamomile and honokiol present in magnolia bark.

**O18. Emotion regulation and psychopathology: Neural and neuroendocrine mechanisms**

***Miu, Andrei C.[1]****

*[1] Cognitive Neuroscience Laboratory, Department of Psychology, Babeș-Bolyai University, Cluj-Napoca, Romania*

Emotional symptoms are present in virtually all common mental disorders. Work in recent decades has shown that emotion regulation, that is, the processes by which we modulate the intensity, frequency and duration of our emotional responses, plays a major role in the development of emotional symptoms. Not surprisingly, thousands of studies on emotion regulation and psychopathology are published each year, with topics including the neural and neuroendocrine mechanisms of emotion regulation. In this presentation, we will describe the process model of emotion regulation, the leading theoretical framework in current research on this topic, and illustrate problems in the various stages of emotion regulation in multiple mental disorders. We will then focus on describing the neural mechanisms of emotion regulation, by showing complementary work using fMRI and EEG. Finally, we will present emerging evidence on the relation between emotion regulation and the reactivity of the hypothalamic-pituitary-adrenal axis, and its role in chronic stress and mental health. This aim of the presentation is to provide a theoretical background for the accompanying papers in this session, focusing largely on emotion regulation and psychopathology.

**O19. How can we assess emotion regulation in the laboratory? A focus on designs with ERP and autonomic measures**

***Pinoșanu, Alexia[1]; Pițur, Simina[1]; Iacob, Alexandra[1]; Hamadneh, Fatima[1]; Miu, Andrei C.[1]****

*[1] Cognitive Neuroscience Laboratory, Department of Psychology, Babeș-Bolyai University, Cluj-Napoca, Romania*

Emotion regulation has been studied extensively in psychology and cognitive neuroscience in recent decades. Much of this work has involved experimental tasks which can accommodate laboratory measures of the underlying neural mechanisms. However, previous studies have exclusively focused on a single stage of the emotion regulation process (i.e., implementation), comparing ability in using one or two emotion regulation strategies (e.g., reappraisal vs. suppression) to modulate emotional responses to affective stimuli. This presentation will describe a novel experimental task that provides comprehensive assessments of individual differences in three stages of the emotion regulation process, incorporating both behavioral and neural outcomes. We will show recent work from our laboratory, in which emotional responses have been assessed using the Late Positive Potential. We will also describe the results of another study in which this task has been adapted to accommodate heart rate variability measures.

**O20. Emotion regulation and cortisol reactivity: Preliminary data from a prospective study.**

***Vlaicu, Andra[1]; Pițur, Simina[1]; Pocol, Cinzia[1]; Pinoșanu, Alexia[1]; Cîmpean, Sergiu[1]; Goia, Amalia[1]; Lorinți, Larisa[1]; Nicolescu, Irina[1]; Profir, Alex[1]; Hamadneh, Fatima[1]; Miu, Andrei C.[1]****

*[1] Cognitive Neuroscience Laboratory, Department of Psychology, Babeș-Bolyai University*

Emotion regulation problems and dysregulation of the hypothalamic-pituitary-adrenal axis have both been associated with psychopathology. However, the interplay between emotion regulation and cortisol response has not been systematically examined. Interactions between these processes could pave the way for psychological interventions that could help normalize cortisol reactivity, with potential health benefits. The present study set out to investigate the associations between the spontaneous use of multiple emotion regulation strategies (i.e., reappraisal, suppression, distraction, rumination) and cortisol response during social stress induced using a standardized laboratory procedure. Furthermore, this study involves prospective assessments of symptoms of psychopathology at three follow-ups. We will present preliminary data from this study, focusing on the emotion regulation and stress responses.

**O21. Childhood maltreatment and emotion regulation: Neural markers of risk for psychopathology?**

***Pițur, Simina[1]; Crișan, Ștefania[1]; Iacob, Alexandra[1]; Balázsi, Róbert[2]; Szentágotai-Tătar, Aurora[3]; Miu, Andrei C.[1]****

*[1] Cognitive Neuroscience Laboratory, Department of Psychology, Babeș-Bolyai University*
*[2] Department of Psychology, Babeș-Bolyai University*
*[3] Department of Clinical Psychology and Psychotherapy, Babeș-Bolyai University*

Childhood maltreatment is a lifelong risk factor for multiple mental disorders. However, the mechanisms underlying this association are not clear. Emotion regulation stands out among the candidate mechanisms considering its developmental timeline and its transdiagnostic association with psychopathology. The present study examined emotion regulation in a sample of adults with high and low levels of childhood maltreatment. Emotion regulation was assessed using behavioral and ERP measures during an experimental task, reflecting individual differences in the selection, implementation and monitoring of two emotion regulation strategies (i.e., reappraisal and distraction). Symptoms of psychopathology were assessed prospectively, using clinical scales. The presentation will describe the results of this study, shedding light on the association between history of childhood maltreatment, emotion regulation and risk of psychopathology.

**O22. QEEG Neuromarkers of Complex Childhood Trauma - Pilot Study and Preliminary Data**

***Marcu, Gabriela Mariana M.[1]*; Zagrean, Ana-Maria[1]***

*[1] Carol Davila University of Medicine and Pharmacy, Bucharest, Romania*

This pilot study proposes several candidate neuromarkers, such as commonly occurring quantitative electroencephalography (QEEG) patterns or indices, to comprehensively approach the impact of complex childhood trauma (CCT) on institutionalized adolescents' neurobiology. Complex trauma is a relatively new clinical construct that considers traumatic experiences and posttraumatic adaptations to be elements of a singular phenomenon. For institutionalized children, the functional effects of trauma on the developing brain are multiple, like in processing cognitive tasks, processing of emotional/social stimuli, or functional connectivity (Bick & Nelson, 2016). CCT changes adolescents' brain neural proliferation and causes physiological alterations in brain structure and function (Green & Myrick, 2014; Lanius et al., 2010). CCT may impair the development of the areas responsible for executive functions, leading to critical disruption of self-regulation and self-concept and consequently to different disorders (Cook et al., 2005). This supports the idea that CCT is a fear dysregulation disorder (Alexandra Kredlow et al., 2022) and therefore, the search for CCT markers might be guided by previous similar research on posttraumatic stress disorder (PTSD). While the EEG produced by cyclic activation of neuronal assemblies provides a view of the complex processes and interactions between brain locations reflecting functional processing. (Collura, 2009), the QEEG technique analyzes this electrical activity to identify patterns and abnormalities. Mathematical processing is applied to neural electrical signals to obtain specific measurements and indices about how the brain functions. Dysregulation of the nervous system in trauma-exposed individuals was reflected with neuroimaging techniques by neuromarkers of cognitive control (Askovic et al., 2020), altered functioning of three large-scale brain networks (CEN, SN, DMN) (Lanius et al., 2015) or by alterations of P2, P3-family event-related potentials and alpha rhythms (Lobo et al., 2015).
EEG Alpha rhythm is associated with the developmental maturation of neural structures (Valdés-Hernández et al. 2010), typical maturation being reflected by a more significant contribution of α-frequencies to EEG in power (Saby and Marshall 2012) or peak frequency (Cellier et al. 2021). Institutionalized children show decreased α-power compared with never-institutionalized children (Marshall et al. 2004; Sheridan et al. 2012; Nelson et al., 2012), which may be associated with developmental delay in neural functioning (Marshall et al., 2002). Therefore, EEG α-power was proposed as a function of exposure to foster care. (Sheridan et al, 2012). These findings suggest that QEEG may be useful for investigating and assessing CCT through frequency analysis and topographic maps. We hypothesized that CCT symptoms would be associated with specific QEEG patterns in institutionalized adolescents. Those patterns would reflect at least some of the proposed EEG markers for PTSD in literature, such as (1) reduced alpha power in posterior scalp regions (Nooner et al., 2022; Clancy et al., 2020; Nicholson et al., 2023; Howells et al, 2012; Walker, 2009), (2) higher alpha peak frequency (iAPF) (Walbeh & Oven, 2013; Butt et all, 2019), (3) right frontal EEG lateralization (Schore, 2001; Calkins&Fox, 1994; Davidson&Fax, 1989; Davidson et al.,

1990; Inn et al., 2022), (4) total EEG power (root mean square - RMS) low + low voltage (in cases of negligence) (Tarullo et al., 2011). Resting-state EEG in eyes-open and eyes-closed conditions was recorded for five adolescents (12-17 yo) who met the inclusion criteria to collect pilot data for a further extended comparative study. Results will inform which QEEG markers to propose for further validation for CCT.

**O23. Cognitive-Behavioral and Socio-Cultural Correlates of Emotion Regulation**

***Ceciu, Ramona L.*[1]**

[1] *University of Bucharest*

Emotion is one of the main components of human personality and social relations, and emotional regulation is defining for human behaviors. This paper presents some findings of an ongoing research study on correlates of emotion regulation of adults from Romania and their satisfaction with life. The study focuses on few general hypotheses, namely: the better the emotional regulation, the healthier the coping strategies; the healthier the emotional regulation, the higher the level of satisfaction with life; and if the participants belong to different backgrounds, there may be some differences with regard to these variables. We review recent neurobiological and psychological studies on emotion, emotional regulation and coping behaviors so important in people's reaction to stress. Of course, satisfaction with life depends on the individual's perception of oneself and the world, among other aspects. On the other hand, when considering such variables, cultural neuroscience studies suggest that the "neuro-cultural interaction model" is significant because it provides fairly clear answers to debates about cultural differences in attitudes, behaviors and perception biases, demonstrating that culturally relevant differences in neural activation may appear even in the absence of behavioral differences. This paper also presents significant information about some techniques and methods useful in balancing emotions. The preliminary results of this research show that there are significant correlations between emotion regulation and the various coping strategies participants use in their daily life, as well as between these variables and the level of satisfaction with life that they manifest.

**O24. NMDAR activation, cytotoxicity and oxidative stress induced by aspartame in native and heterologously expressed cell lines**

***Shaher, Shurooq*[1]*; Galateanu, Bianca*[2]*; Hudita, Ariana*[2]*; Pop, Cristian-Emilian*[2]*; Mihailescu, Dan Florin*[2]*; Amuzescu, Bogdan*[2]****

[1] *Dept. Medical Laboratories, Babylon Technical Institute, Alfurat Al Awsat Technical University*
[2] *University of Bucharest*

Aspartame, the methyl ester of the dipeptide formed by aspartic acid and phenylalanine, became during the last 40 years a very popular and widely used artificial sweetener, being incorporated in carbonated beverages, dry foods and drugs for oral administration. However, several epidemiology surveys and clinical trials, as well as in vivo and in vitro experiments revealed a number of negative side effects, particularly at the central nervous system level, such as headaches, increased seizure susceptibility, altered mood, irritability, depression, altered spatial orientation and learning, sometimes at doses below the maximum admissible daily intake (40 mg/kg day in Europe), deemed to result from competition between aspartame decomposition product phenylalanine and other large neutral amino-acids for a common transporter across the blood-brain barrier resulting in decreased dopamine and serotonin levels, and also by aspartate-triggered excitotoxicity. Therefore our study aimed to evidence direct activation by aspartame of N-methyl-D-aspartate receptors (NMDARs) induced by transfection of different cells with plasmids containing subunits NR1 and NR2b fused with enhanced green fluorescent protein (eGFP). Thus, via whole-cell patch-clamp experiments in HEK293T cells transfected by electroporation we recorded NMDAR outward transient currents at +40 mV elicited by 200 ms pulses of aspartame or L-glutamate 100 μM, while in transfected CHO-K1 cells 2-4 h exposure to aspartame 10 μM to 10 mM induced increased apoptosis as shown by propidium iodide staining and flow cytometry. Exposure of cultured HeLa cells for 48h to aspartame 100 μM increased production of hydrogen peroxide, detected with a ROS-Glo™ H2O2 chemiluminescence assay (Promega), lipid peroxydation via malondialdehyde levels and catalase activity (assessed with MAK085 and MAK381 kits from Sigma-Aldrich).

**O25. Goal-directed skilled action in rats is modulated by the dopamine receptors found in the fastigial nucleus**

***Caragea, Violeta-Maria*[1]**

[1] *Ruhr University Bochum, Germany*

The cerebellum is mostly known to contribute to implicit memory requiring motor function, such as learning how to ride a bike. However, cognitive and emotional processes affecting memory functions were also recently discovered to recruit cerebellar structures. In the nervous system, dopamine acts as a neurotransmitter and modulates motivation, memory, and body movements. It does that by acting on dopamine receptors found in the pre- and postsynaptic membranes of the neural cells. In our study, we asked if dopamine receptors are also present in the fastigial nucleus (FN) of the cerebellum and, if so, how they modulate skill acquisition and motivation to act towards a goal. To achieve this, we used immunohistochemistry to describe the presence of the D1 and D2 dopamine receptors within the targeted structure, and pharmacology with behavioral testing to look at the influence of these receptors on the acquisition of a skill that requires reaching and retrieving pellets through a narrow slit. Open field, rotarod, and elevated plus maze tests were added to control for motor function and anxiety.
We found that both D1 and D2 receptors are found in the FN, having a similar subfield localization pattern. Our behavioral results showed that the FN is involved in goal-directed action with locally expressed D1 receptors modulating motivation for skilled action and D2 receptors being involved in skill consolidation. Taken together, our results reveal a novel role of the FN in goal-directed skilled action, with this function being tightly regulated by locally expressed dopamine receptors.
Acknowledgements: This work was supported by a grant from the German Research Foundation (Deutsche Forschungsgemeinschaft, SFB 874/B10, project number: 122679504).

**O26. Effects of two radiotherapy techniques at the cerebrovascular endothelium level**

***Stoica, Roberta[1]*; Radu, Mihai[1]; Craciun, Liviu[2]; Patrascu, Stela Rodica Lucia[3]; Radu, Beatrice M.[4]***

*[1] Department of Life and Environmental Physics, Horia Hulubei National Institute for Physics and Nuclear Engineering*
*[2] Department of Applied Nuclear Physics, Horia Hulubei National Institute for Physics and Nuclear Engineering*
*[3] Department of Radioisotopes and Radiation Metrology, Horia Hulubei National Institute for Physics and Nuclear Engineering*
*[4] University of Bucharest*

In the radiotherapy domain, the most used facilities to eradicate the tumor are photon or accelerated ions beams. The main difference between them consists in the possibility to concentrate the maximum energy of the accelerated ions to a certain depth in the tissue (Bragg peak) where the tumor is located and thus, the healthy tissue around the tumor is spared. In the case of cerebral tumors, the oxidative stress induced by the ionizing radiation in the tumor environment can affect the physiological functions of the cerebral cells, including the blood-brain barrier. Due to its significant role in the regulation and protection of the brain homeostasis, it is imposed a characterization of the brain microvasculature after X-rays and accelerated ions (protons in our work) irradiation with relevant clinical doses.
We observed the cytotoxic, functional and genotoxic effects after low-energy accelerated proton irradiation and X-ray irradiation upon 2 in vitro models of murine and human cerebral microvasculature (bEnd.3 cell line and HBEC-5i cell line, ATCC). Using the proton beam line of a TR19 cyclotron, we exposed cells to doses in the range 0-10 Gy, (dose rate: 1 Gy/min). For the X-ray exposure, we used a X-Strahl generator with exposure in the same range (dose rate: 0.125 Gy/min). The cells proliferation and surviving rates in stress-induced conditions, the DNA damage, the reactive oxygen species formation and the calcium ions dynamics at various time intervals post-irradiation were assayed.
As results, we observed an inhibition (up to 90%) of the cellular proliferation for both irradiation types and a strong increase of micronuclei number at doses over 5 Gy, a reduction of the DNA repair capacity by the increased values of the dose (~100%), an increase of the total reactive oxygen species and a significant modification of the intracellular calcium concentration.
In conclusion, we observed a significant modification of the measured parameters with the dose increase, the cell type and the radiation type. Our study presents preliminary results regarding the BBB endothelial cells response to irradiation with the most used radiations in radiotherapy (protons and X-ray).

**O27. Role of four thermosensitive TRP ion channels in thermal preference of male and female mice**

***Selescu, Tudor[1]*; Iodi-Carstens, Mirela[2]; Carstens, Earl[2]***

*[1] Department of Anatomy, Animal Physiology and Biophysics, Faculty of Biology, University of Bucharest*
*[2] Department of Neurobiology, Physiology, and Behavior, University of California, Davis CA*

Many of the Transient Receptor Potential (TRP) ion channels act as molecular thermoreceptors and are expressed in the peripheral nervous system where they play an essential role in transducing external temperature. TRPA1, TRPC5, TRPM2-5, TRPM8 and TRPV1-4 channels have all been reported to be temperature-sensitive. Knock-out (KO) animals for the genes coding these proteins were generated in the last two decades.
Here we assayed the temperature preference of wild-type (WT) and TRP (TRPA1, TRPV1, TRPV4 and TRPM8) KO mice of both sexes, to establish if the absence of any of these genes changes the natural temperature preference of the animals, and if the temperature preference is sex-dependent. The mice were either left to choose their preferred position between two plates, one found at 30 ºC and another at 15, 20, 25, 30, 35, 40, or 45 ºC, or left to explore a near- linear temperature gradient (1.83 m long), between 15 and 45 ºC.
The WT mice, as well as most KO mice, significantly avoided 15-20 ºC and 40-45 ºC temperatures. The TRPM8-KO mice did not significantly avoid 15-35 ºC temperatures, whereas TRPV1-KO females had no significant temperature preference. Mean occupancy temperatures on the gradient, measured in the 90-120 min interval, were significantly higher for females (30-34 ºC) compared to males (26-27 ºC) for all genotypes, except for TRPA1-KO, which exhibited no sex difference.
These result suggest that TRPA1 and TRPV1 may play a role in shaping the sex-dependent temperature preference, whose underlying mechanisms should be further investigated.

**O28. The impact of the general anesthesia during childhood on the immature hippocampus exposed to perinatal asphyxia. An experimental model**

***Isac, Sebastian[1]*; Matache, Irina[1]; Ionescu, Mara Ioana[1]; Pavel, Bogdan[1]; Zagrean Ana-Maria[1]***

*[1]Division of Physiology II - Neurosciences, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania*

Abstract. (1) Introduction: Perinatal asphyxia (PA) is a leading cause of morbidity and mortality in newborns, and its long-term effects on brain development can be devastating. Anesthesia (GA) is commonly used in pediatric care, but its impact on the immature brain already impaired by PA remains poorly understood. The aims of this study were to explore anesthesia-induced biochemical, epigenetic, electrophysiologic and behavioral changes in Wistar rats previously exposed to PA. (2) Material and Methods: we included a number of 40 pups divided into 4 groups with regard to PA and GA exposures: C (control group), PA group, GA group and PA-GA group. In PND 6, the pups were exposed to either PA or normoxia and in PND 15 to either GA or normoxia. After exposures, we assessed the hippocampal IL-1B and S-100B (ELISA) and miR 15a-3p, miR 16-3p, miR 34a-3p, miR146-3p, miR 124-5p, and miR 132-5p (qRT-PCR). At maturity, the pups were exposed to various behavioural (OFT, TMT, FST, and NORT) and electrophysiological tests (visual stimulation under various degrees of anesthesia). (3) Results: We observed an important GA-induced epigenetic modulation, regardless to the PA exposure. A potential cumulative

detrimental effect on the brain of GA in PA exposed pups is limited on minor behavioral and electrophysiological changes. (4) Conclusion: This study offers promising tools in identifying GA-induced brain dysregulations. Further studies are needed to assess if GA could have additional detrimental effects on the immature brain previously exposed to PA. Key words: pediatric anesthesia, perinatal asphyxia, epigenetic, miR, hippocampus.

# Blitz oral presentation & Poster session

**P1. The effect of nerve growth factor on vascular fibroblasts**

***Ostafi, Alina[1]; Bratu, Marina[1]; Pluteanu, Florentina[1]****

*[1] Bucharest University, Faculty of Biology*

The crosstalk between nervous system and vascular system takes place at different levels. The Nerve Growth Factor (NGF) is a neuropeptide primarily related to nervous system development, nociception or nerve regeneration upon injury. High serum levels of NGF were detected in inflammatory states, suggesting effects on cells other than neurons. We hypothesize that NGF may contribute to vascular remodeling associated with chronic inflammation. Studies had shown that vascular fibroblasts (vFb), depending on the activation status, express tropomyosin kinase receptor trkA and the low affinity p75NTR, both being receptors for NGF. However, functional responses of vFb to NGF are not known. Here, we used cultured vFb isolated from normal rat aorta, maintained in 10% serum growth medium, to evaluate the effect of NGF on vFb proliferation, migration or electrical phenotypes. Cells were synchronized for 48-72 h prior to 24-72 h stimulation with 100 ng/mL NGF at different serum concentrations (1%, 3% and 10%) to mimic different activation states of vFb. NGF induced a moderate vFb proliferation in the presence of 1% and 10% serum, while in 3% serum, NGF significantly potentiated both proliferation and migration. Patch-clamp experiments performed in these conditions (NGF and 3% serum) indicated an increase of the sustained outward K+ current IDR and of the inward K+ current IK1 in NGF stimulated vs. control vFb (3% serum only). These results suggest that NGF may contribute to vascular remodeling by enhancing the proliferative and electrical activity of vFb.

**P2. Hyperexcitability in a Rat Model of Absence Epilepsy: Higher EEG Reactivity During Deep Anesthesia**

***Matota, Ana-Maria[1]*; Paslaru, Alexandru-Catalin[1]; Morozan, Vlad-Petru[1]; Stancu, Mihai[2]; Tofan, Laurentiu[1]; Szocs, Dorottya[1]; Mocanu, Alexandra[3]; Pavel, Bogdan[1]; Zagrean, Ana-Maria[4]; Zagrean, Leon[1]; Moldovan, Mihai[5]***

*[1] Carol Davila University of Medicine and Pharmacy*
*[2] Ludwig-Maximilian University*
*[3] University of Bucharest*
*[4] National Neuroscience Society of Romania, Romania*
*[5] University of Copenhagen*

We examined if the burst-suppression (BS) patterns that appear on the electroencephalogram (EEG) during deep anesthesia, which are reactive to external stimuli, exhibit a higher reactivity in the epileptic brain.We used adult Wistar Albino Glaxo Rijswijk (WAG/Rij) rats (a genetic rat model of absence epilepsy, which present characteristic episodes of spike-and-wave discharges (SWDs) on the EEG) as the study group and age-matched Wistar rats as the control group. We recorded two cortical fronto-occipital EEG leads while under isoflurane anesthesia and delivered intermittent photic stimulation (IPS) to one eye at a rate of 0.5 Hz in 1-minute trials. We used offline EEG analysis with the data from the channel ipsilateral to IPS to minimize the effect of visual evoked potentials. The suppression ratio (SR) is the fraction of time spent in suppression, over 1-minute intervals. The baseline SR was 40% - 80%. BS reactivity index (BSRI) is the reduction in SR that occurred during IPS, relative to the baseline SR recorded immediately before IPS. During deep anesthesia, IPS did not trigger SWDs. Controls had a mean BSRI of 0.2 and the WAG/Rij mean BSRI was increased by 55%. Following administration of ethosuximide, the difference in BSRI was reduced, but it paradoxically increased after the administration of carbamazepine, both at doses that had no effect on the controls.Our data suggest that measuring burst-suppression EEG reactivity could be useful to further study the hyperexcitability of the epileptic brain.

**P3. Axonal growth in the young adult brain after an ischemic stroke**

***Cercel, Andreea[1]*; Mihaela, Abuzan[1]; Surugiu, Roxana[1]; Popa, Aurel[1]; Mihai, Ruscu[1]***

*[1] University of Medicine and Pharmacy Craiova*

After a brain impairment, a large number of genes are controlled to promote repair. For cell morphogenesis and function, the kinesin superfamily proteins (KIFs), which include KIF1b, KIF4, KIF21b, and KIF26b, are crucial. However, it is still unclear how they function in axonal regeneration following damage. Here, we investigated whether genes involved in early postnatal brain development may be re-expressed in injured young adult brain tissue. Juvenile rat pups that were 3 days old had significant quantities of KIF4 protein as well as KIF1b, KIF4, KIF21b, and KIF26b transcripts, according to RT-PCR and immunohistochemistry. However, in the subventricular zone and perilesional cortex of young adult rats, only the kif1b and kif4 mRNAs and KIF4 were significantly re-expressed. Only the NPCs in the subventricular zone and the endothelial cells of the inflamed blood arteries have the ability to regenerate. Most intriguingly, NPCs from the leptomeninges are likely responsible for some of the surviving neurons in the peri-lesional area that have KIF4 sporadically re-expressed in them. Conclusion: According to our findings, the young-adult brain's ability to re-express juvenile genes required for axonal growth is limited. In fact, the expression pattern of the kinesins gene and protein seen during early postnatal brain development is partially replicated in the wounded young adult brain.

**P4. Spared nerve injury model of neuropathic pain induces a specific microglia activation pattern at the spinal cord level**

***Grosu, Andreea Violeta[1]*; Pertin, Marie[2]; Kirschmann, Guylene[2]; Filippi, Alexandru[3]; Deftu, Alexandru Florian[2]; Ristoiu, Violeta[4]***

*[1] University of Bucharest*
*[2] Pain Center, Department of Anesthesiology, Lausanne University Hospital and University of Lausanne (CHUV), Lausanne*
*[3] Department of Biophysics, University of Medicine and Pharmacy "Carol Davila", Bucharest, Romania*
*[4] Department of Anatomy, Animal Physiology and Biophysics, Faculty of Biology, University of Bucharest, Romania*

Introduction: Peripheral neuropathic pain pathogenesis involves the activation of immune/glial cells, among which microglia seem to play a key role. These cells' responses are injury-specific, probably because of the different signals sent to the spinal cord by the lesioned DRG neurons. Our aim was to characterize the spatial distribution of microglia activation at the spinal cord (SC) level after the Spared nerve injury (SNI) model. Methods: CX3CR1-GFP mice were assigned to 3 groups: Naive, Sham (sciatic nerve was exposed) and SNI (the tibial/peroneal rami of sciatic nerve were ligated and cut). Seven days after surgery, L3-L5 SC segments were collected, sectioned and immunostained using the neuronal marker Neurotrace Blue Fluorescent Nissl Stain and analyzed for CX3CR1-GFP microglia number and distribution per lamina, which was evaluated by an in-house ImageJ plugin. Results: Seven days after surgery, microglia are mainly activated in the ventral horn in L5 and both in the dorsal and ventral horns in L4 and L3. Moreover, the pattern of microglia activation varies within the same spinal segment, from lamina I to lamina VII, with the highest degree of activation in lamina I and the lowest degree of activation in lamina V. Last but not least, we observed that the number of microglia is higher at the nociceptors' project site (laminas I-II), than in the areas where mechanoreceptors project (laminas III-V). Conclusions: The SNI model of peripheral neuropathic pain determines a specific microglia activation pattern, that varies from one lamina to another, and from one spinal segment to another.

**P5. Burst-Suppression EEG Reactivity in Detecting Post-Ischemic Brain Injury: An Experimental Rat Study**

***Bordeianu, Andrei[1]*; Paslaru, Alexandru-Catalin[1]; Morozan, Vlad-Petru[1]; Tofan, Laurentiu[1]; Stancu, Mihai[2]; Pavel, Bogdan[1]; Zahiu, Carmen[1]; Mocanu, Alexandra[3]; Ilie, Andrei[4]; Calin, Alexandru[4]; Zagrean, Ana-Maria[5]; Zagrean, Leon[5]; Moldovan, Mihai[6]***

*[1] Carol Davila University of Medicine and Pharmacy*
*[2] Ludwig-Maximilian University*
*[3] University of Bucharest*
*[4] University of Newcastle*
*[5] National Neuroscience Society of Romania, Romania*
*[6] University of Copenhagen*

Aim: The electroencephalographic (EEG) assessment of the diffuse ischemic brain injury remains methodologically challenging. The aim of this study was to investigate the impairment in EEG reactivity to intermittent photic stimulation (IPS) following experimental global cerebral ischemia. Rather than focusing on continuous EEG measures, we tested the reactivity of the discontinuous burst-suppression (BS) patterns induced in deep anesthetic coma. Methods: Male Wistar rats were surgically exposed to a mild global cerebral ischemia by electrocauterization of the vertebral arteries and the subsequent clamping of both common carotid arteries for 5 minutes under laser doppler control. A group of rats exposed to sham surgery served as controls. Cortical EEG recordings were carried out at 48 hours after surgery when all rats appeared clinically recovered. The BS patterns, induced by an overdose of chloral hydrate, were quantified by the suppression ratio (SR), measuring the fraction of time spent in suppression, over 1-minute intervals. The IPS was delivered to one eye at 0.5 Hz in 1-minute epochs. The BS reactivity index (BSRI) was defined as the reduction in the ipsilateral SR that occurred during IPS, relative to the baseline SR recorded just prior to IPS. Results: At a baseline SR of 40%-80%, the mean BSRI was 0.27 in controls. In contrast, the mean BSRI was about 3-fold smaller in the rats exposed to GCI. The amplitude of the visual evoked potentials was similar between the groups. Conclusions: Our data suggest that measures of burst-suppression EEG reactivity are sensitive to detect the post-ischemic brain injury.

**P6. Role of prefrontal cortex in processing aversive auditive and visual stimuli**

***Bratu, Andrei Teodor[1]*; Zamfirache, Florin[1]; Radu, Beatrice M.[1]***

*[1] University of Bucharest*

Fear is a physiological state that appears as a response to potentially harmful stimuli. Behaviorally, flight-or-fight or freezing responses can suggest a strong link between fear and executive processing. Anxiety is defined by a highly vigilant state of activation determined by diffused potentially harmful stimuli, that can become another indicator of the link between behavior and cortical activity in relation to stress. This study aims to investigate the effects of aversive auditive and visual stimuli on the prefrontal cortex using a rat model of Pavlovian induced fear. To test this hypothesis, we used Sprague-Dawley male adult rats and we attached to their scalp an external brain computer interface device, without implantation in the vicinity of the prefrontal cortex. For collecting signals we used an OpenBCI 4-channel Ganglion Biosensing Board and we recorded in control conditions and during the visual and auditive stimulation. Behavioral tests, e.g. open field test, forced swim test and elevated plus maze, were also used. Preliminary results showed an increased cortical activity during stimulation. In the left hemisphere was a basal activity of 2.22, that grow up to 2.92 and 4.59 when applying aversive visual and auditive stimuli. In the right hemisphere we recorded an increased activity from the basal value of 1.78 up to 2.80 and 8.00 when stimulation occurred. Changes also occurred at a behavioral level that correlate with electrophysiological data. These findings support the hypothesis of the role of prefrontal cortex in processing aversive stimuli.

**P7. Cenobamate upregulates P-glycoprotein, while downregulates tight junction proteins in accordance with blood brain barrier permeabilization**

***Draghici, Claudia-Ioana[1]*; Stoica, Roberta[2]; Radu, Mihai[2]; Radu, Beatrice M.[1]***

*[1] University of Bucharest*
*[2] Department of Life and Environmental Physics, Horia Hulubei National Institute for Physics and Nuclear Engineering*

Epilepsy is one of the most common neurological diseases that affects more than 50 million people worldwide. It is characterized by seizure episodes provoked by uncontrolled firing action potentials of the neurons. Antiepileptic drugs are used as treatment, and each drug has a different mechanism of action that helps to decrease the number, severity, and duration of seizures. Cenobamate (CBM) received US Food and Drug Administration approval in 2019 for the treatment of epilepsy and since then, numerous experiments were done to determine its precise mechanism of action. Blood brain barrier (BBB) is among the important targets that should be investigated for any new released drug. The aim of our study was to characterize the interaction of CBM with brain microvascular endothelial cells. To this purpose we used the human cerebral microvasculature cell line (HBEC-5i, ATCC) exposed to clinically relevant concentrations of CBM (3 – 300 µM). Next, we analyzed the changes in expression of P-glycoprotein (P-gp), Zona occludens-1 (ZO-1) and claudin-5 by immunofluorescence and qRT-PCR, the effects in cellular viability and the changes in permeability of an endothelial monolayer. We demonstrated that CBM upregulates P-gp, while downregulates ZO-1 and claudin-5. These data correlate with reduction of transendothelial electrical resistance with the CBM dose (10, 100 µM). No significant changes of the cell viability were determined. In conclusion, our results are very relevant from interpreting the clinical effects of CBM at the BBB level. In this context, we speculate that CBM may permeabilize BBB and also induce drug resistance.

**P8. HISTOLOGICAL AND IMMUNOHISTOCHEMICAL ASPECTS OF THE CENTRAL NERVOUS SYSTEM IN ALZHEIMER'S DISEASE AND THE STUDY OF ANTIOXIDANT ACTION AS A PROPHYLACTIC STRATEGY**

***Moldoveanu, Claudia A.[1]***

*[1] Babeș-Bolyai University*

Introduction. The study is a must in addressing neurodegenerative diseases, specifically Alzheimer's disease. The aim of this research was to investigate the correlations between antioxidants and the onset of cerebral amyloidosis, i.e., to analyze the correlation between different methods of detecting the existence of amyloid plaques. Materials and Methods. The experimental part was carried out with female Wistar rats randomly divided into four experimental groups: control, dimethylsulfoxide (DMSO), amyloid and amyloid on Betula pendula folio plant extract administration for 4 weeks. Results. The results obtained confirm the hypotheses of detection of brain amyloidosis by the methods performed, except in the case of Congo Red staining, because the early stage of the subjects' disease was insufficient for the reaction between the dye and the sample and the generation of a signal. As for the administration of the plant extract, the prophylactic role was negative in this batch, with the animals showing up to four times more signs of the disease. Conclusion. Following analysis of brain samples obtained from the four-week study, the conclusion confirms the original objectives and support the occurrence and identification of Alzheimer's disease following injection of individual amyloid and amyloid in combination with a plant extract mixture. However, what remains unexpected from the study and contrary to initial hypotheses is that taking the plant extract induced the same or stronger onset of amyloidosis, compared to previous studies in the field showing that antioxidants limit the onset and proliferation of amyloidosis.
Acknowledgement. This work was supported by a grant of the Ministry of Research, Innovation and Digitization, CNCS - UEFISCDI, project number PN-III-P1-1.1-TE-2021-0159, within PNCDI III (TE60/2022).

**P9. The Trials and Errors of Studying the Impact of Gestational Gut Microbiome Disturbance and Perinatal Asphyxia on the Neurodevelopmental Reflexes of Rat Offspring**

***Ciotei, Cristian[1]*; Barbalata, Didina Catalina[1]; Dogaru, Ioana Alexandra[1]; Zahiu, Carmen[1]; Chitimus, Diana-Maria[1]; Morozan, Vlad-Petru[1]; Chazli, Tasnim[1]; Belcin, Mara-Gabriela[1]; Ieseanu, Mara-Ioana[1]; Zagrean, Ana-Maria[1]***

*[1] Carol Davila University of Medicine and Pharmacy, Bucharest, Romania*

Introduction: Maternal gut microbiota disturbance has been linked to alterations in the offspring's neurodevelopment. Perinatal asphyxia (PA) can induce altering effects on the immature brain. In this study, we aim to discuss the challenges encountered during our research project, which focuses on assessing the impact of gestational antibiotic administration (GAA) and PA on offspring's neurodevelopmental reflexes.
Materials and methods: Starting with the 11th gestational day, we conducted GAA on Wistar rats by orally administering a cocktail of antibacterial and antifungal medications (ampicillin, vancomycin, neomycin, clindamycin, and amphotericin-B) in their drinking water. After birth, pups were divided into four groups: control-normoxy (C-N), antibiotics-normoxy (AB-N), control-perinatal asphyxia (C-PA) and antibiotics-asphyxia (AB-PA). On the 6th postnatal day (PND), we performed the PA by exposing the pups to 90 minutes of asphyxia (9% O2, 20% CO2 in N2). Additionally, we monitored the vital signs of C-PA and AB-PA pups by using a MouseOx monitor. Between PND 7 and 9, we subjected the pups to early-life behavioral tests, including the righting reflex (RR), limb grasping reflex (LGR), cliff avoidance (CA), negative geotactic reaction (NGR), and grip strength response (GSR). Results and Discussion: GAA resulted in a notable increase in miscarriages and a reduction in offspring birth weight. During PA, oxygen saturation levels declined to 45.41%. We observed a consistent extension in the response time across both the perinatal asphyxia and antibiotics groups, with subtle variations between individual tests. The elevated rate of miscarriages prompted a revision of the antibiotic cocktail to mitigate teratogenic and toxic risks. The modified combination now comprises ampicillin, vancomycin, neomycin, and meropenem. These antibiotics are safe to use during pregnancy and most of them have low oral biodisponibility. Furthermore, to gain deeper insights into the microbiota's relevance in the neural development of rat offspring, future investigations will incorporate

probiotics (VIVOMIXX) alongside the new antibiotic cocktail. This will yield data for four distinct groups: control, antibiotics, probiotics, and antibiotics+probiotics. Each of these groups will be further subdivided into normoxia and asphyxia test subgroups. Conclusion: Our preliminary findings underscore the potential early-onset brain impairment resulting from maternal gut disturbance and PA, warranting enhanced prudence in the recommendation of gestational antimicrobial usage. In subsequent research, we aim to further elucidate the potential protective effects of probiotics against perinatal asphyxia and gut microbiome alteration.

Keywords: Maternal gut microbiome; Perinatal asphyxia; Gestational antibiotic administration; Neurodevelopment; Probiotics.

**P10. Crosstalk Between TRPA1, TRPM8 and EGF Receptor in Glioblastoma**

***Băcioiu, David-Ionuț G[1]*; Botezatu, Alina-Ana-Maria[1]; Domocoș, Dan[1]; Cojocaru, Florentina[1]; Cucu, Dana[1]***

*[1] University of Bucharest*

Mutation of Epidermal Growth Factor Receptor (EGFR) and Isocitrate Dehydrogenase (IDH) genes, a highly immunosuppressive tumor microenvironment and an abundance of pro-angiogenic factors are some of the molecular signatures of glioblastoma. Tackling these complex mechanisms requires novel molecular participants to address treatment options for gliomas. Among emerging and underexplored targets, the promiscuous family of TRP channels was found to be related to cancer development, including glioma. Here, we discuss the potential of TRPA1 and TRPM8 as biological markers, being associated with the survival of glioma patients and overall treatment resistance. More specific, we will discuss the role of selected TRP channels in controlling proliferation, cell cycle, apoptotic pathways, as well as migration/invasion. The U87MG glioblastoma cell line and a clone of U87MG cells that express the mutant EGFRvIII receptor have been exposed to TRPA1/TRPM8 agonists (50-100 μM) and inhibitors (10-50 μM), or a mixture of compounds for 24 hours. The wound healing assay was performed to examine cell migration, while flow cytometry was used to seek the effect of TRPA1 and TRPM8 on the cell cycle. Slower migration patterns and a G2/M and sub-G1 blockade is expected in TRPA1-expressing cells. As far as TRPM8 is concerned, we are anticipating crucial functions in cell mitosis and enhancement of cell migration rate. The EGFRvIII cells might also be disproportionately more prone to invasiveness compared to the WT cells, regardless of channel. If so, pharmacological modulation of TRPA1 and TRPM8 could become a notable anti-tumoral option.

**P11. The impact of sertraline and social rehabilitation on a long-term depression study on rats**

***Zamfirache, Florin[1]*; Radu, Beatrice M.[1]; Prundaru, Gabriela[1]***

*[1] University of Bucharest*

Introduction: Recent studies like Moncrieff et al1 found little to no evidence that depression is caused by a chemical imbalance, like lower concentration or activity of serotonin in the brain1. However, the use of SSRIs or other antidepressants have been proven to help patients suffering from long term depression and continue to be used by clinicians. A new approach to understanding depression propose the idea that depression causes changes in neuroplasticity and antidepressants can help reverse some of this neuroanatomical changes2. Our study aims to investigate the impact of long term depression on rats and the way sertraline and social rehabilitation impact the memory, movement and exploring desire of the rodents measured with NOR test as some of key behaviors impacted by depression. Objective 1) Comparing the effect of sertraline v.s. social rehabilitation on the recovery of memory, movement and exploring desire on rodents subjected to a depression inducing protocol.
2) Exploring the rebound effect of drastically stopping the sertraline administration on rats housed in individual cages compared to rats housed in enriched social environment. 3) Exploring long term effects and remission of both approaches on the depression induced rodents.
Methodology: Adult male rats (10, Sprague Dawley) were used for the preliminary research, involving an experimental model of depression on rodents, the chronic retention stress model. This model is presented in prior research as a very well-established source of stress that induces depression, has a strong validity, phenotypic changes over time, both behavioral and physiological, that induces atrophy in hippocampal CA3 pyramidal cells and elevated corticosteroid levels. This involves restraining the rat`s movement for two hours, daily, for 2-3 weeks3. This can be compared with daily human stress induced by stressful jobs or live situations. As antidepressant we used Sertraline hydrochloride 20 mg/ml (Zoloft, Pfizer), that has been shown in numerous controlled studies to have efficacy in the treatment of depression and anxiety disorders, a selective serotonin reuptake inhibitor (SSRI) chosen as a pharmacological product for its proven efficacy and good tolerability4. The delivery method chosen was administered via a biscuit per day, as described in previous research as an easy and noninvasive way. An improved enriched rodent cage was built with the purpose to stimulate rodent colony behavior and promote social rehabilitation, an alternative way of promoting neuroplasticity as a method of treating depression. The NORT (Novel Object Recognition Test) was used to assess memory deficit, one key aspect of cognitive disfunction specific to depression and anxiety6, Open Field and Elevated Maze Plus was used to assess the movement and exploratory activity, and Sucrose preference test to assess anhedonia, a condition specific to depression described as the inability to feel pleasure from things that were prior desired.
Preliminary study results suggest social rehabilitation was as affective as sertraline as an antidepressant method and prevented the relapse when sertraline was discontinued. The rats were tracked with the above described testes over the course of 1 year in 10 distinct stages, from baseline, inducting de depression phenotype, following the evolution of the condition, introducing the antidepressant treatment, the social rehabilitation cage and stopping the sertraline treatment.

**P12. Retinal Ganglion Cell types of the Mouse Area Centralis**

***Dobre, Ileana Daria[1]***

*[1] Transilvania University of Brașov*

Retinal Ganglion Cells (RGCs) transmit parallel streams of information from the eye to the brain. RGC classifications describe between 20 to 40 types, depending on the species. Classification

criteria include dendritic arbor morphologies, axonal projection to specific retino-recipient nuclei of the brain, visual stimulus response properties, function within the visual circuit, and not the least, molecular markers or intersections thereof. Our lab had previously described a combinatorial code of Brn3/Pou4f transcription factors, with distinct expression patterns and functions in RGCs. In contrast to humans, mice do not have a fovea, and RGC densities and dendritic arbor sizes are relatively evenly distributed across the retina. We recently discovered in the retinas of Brn3cCre/WT; Brn3bCKOAP/WT mice a region of high density of RGCs, which we called mouse area centralis. Given the density of RGCs in this area, it was not possible to characterize individual RGC types by dendritic arbor morphologies. We therefore infected Brn3cCre/WT retinas with Cre-dependent AAV-FLEX-eGFP vectors, and double-stained the retinas with anti-eGFP and anti-Brn3b antibodies. Thus we were able to measure and/or trace the dendritic arbors of individual RGCs, and determine whether they were also positive for Brn3b, and found they belong to a limited number of cell types. We will present our data regarding dendritic arbor morphologies for Brn3c+Brn3b- vs. Brn3c+Brn3b+ RGCs. The findings are significant for understanding the visual information transmitted to the brain from this area of high visual acuity in the mouse retina.

**P13. How Continuous Use of Ion Channel Blockers Affects Microglial Morphology and Function in a Murine Alzheimer's Model.**

***Boboc, Ianis K. S.[1,2]; Cojocaru, Alexandru[2,3]; Balseanu, Tudor A.[2,3]; Bogdan, Maria[1]; Calina, Cornelia D.[4]; Catalin, Bogdan[2,3]***

*[1] University of Medicine and Pharmacy of Craiova, Faculty of Pharmacy, Department of Pharmacology, Craiova, Romania*
*[2] Experimental Research Centre for Normal and Pathological Aging, University of Medicine and Pharmacy of Craiova, Craiova, Romania*
*[3] University of Medicine and Pharmacy Craiova, Faculty of Medicine, Department of Functional Sciences, Craiova, Romania*
*[4] University of Medicine and Pharmacy Craiova, Faculty of Pharmacy, Department of Clinical Pharmacy, Craiova, Romania*

Aim: Evaluate the live morphological changes of microglia using in vivo 2P-LSM, in a murine model of AD, and their impact on cognitive and motor function by using behavior tools, under systemic administration of sodium or calcium ion channel blockers.
Methods: 25 transgenic male mice (age 14-16 weeks, weight 20-25 g), positive for amyloid precursor protein (APP) mutations and CX3CR1GFP/WT, were used. These animals were randomly divided into 3 groups (Control, n = 5; Carbamazepine (5 mg/kg), n = 10; Verapamil (3.5mg/kg), n = 10). Furthermore, 5 CX3CR1GFP/WT male mice that were not positive for APP mutations (age 14-16 weeks, weight 20-25 g), were used as wild-type controls. Open Field and Novel Object Recognition tests were performed in order to assess locomotor, anxiety-like behaviour and short term memory. A cranial window was implanted over the right somatosensory area. In order to visualize the cerebral vasculature, a subcutaneous Sulfurodamine 101 injection was made. Image acquisition was made using a Zeiss LSM 7 MP Multi-Photon microscope. Recorded images were processed using the Zen2Blue and ImageJ programs. Individual cells were analyzed using the ''log-log'' method. In order to evaluate the distances to amyloid deposits of the analyzed microglia, we manually measured the distance from clusters of microglia that can be seen in the 2P-LSM of the APP mice.
Conclusion: Verapamil treatment increased memory and exploration behavior, reduced anxiety levels, and enhanced short-term memory. Verapamil and Carbamazepine, reduced the speed of surveillance microglia processes and affected microglia morphology in the cortex.

**P14. Iba1 down-regulation in primary rat microglia: differential effects induced in defined-medium vs serum-supplemented in vitro systems**

***Magercu, Melania[1]*; Chiritoiu, Gabriela[2]; Munteanu, Cristian[2]; Ristoiu, Violeta[1]***

[1] University of Bucharest)
[2] Institute of Biochemistry of the Romanian Academy

Introduction: Previous studies have shown that serum factors strongly perturb microglial morphology and function. To overcome that limitation, serum-free cultures containing pivotal factors were recently optimized to allow primary microglia robust survival (defined-medium conditions). Although Iba1 expression is widely considered only a microglial identifier, the contribution of this actin cross-linking protein in microglia functioning remains unclear. Here we addressed changes in siRNA anti-Iba1 transfection in microglial cells cultured under defined-medium versus serum-supplemented condition.
Methods: Microglia from cerebral cortices of postnatal Sprague-Dawley rats (P14-P21) were positively selected using CD11b-coated-immunopanning dishes. On the fifth day, the purity and cell survival were assessed. To visualize the effects of Iba1 down-regulation at cellular level, a siRNA-A594 molecule was transfected in both defined-medium and serum-supplemented models using Glial-Mag method. Next, we evaluated the efficiency of Iba1 down-regulation through Western blot and LC-MS/MS.
Results: Delivery of siRNA-A594 anti-Iba1 in defined-medium condition showed extensive cell detachment and aggregation. These effects were also observed for the serum-supplemented cultures, but only above 40 nM concentration, allowing us to perform the proteomic analysis. According to Western blot, a twofold reduction was revealed in total Iba1 protein when 20 nM siRNA anti-Iba1 was transfected in serum-supplemented cultures, compared to NT condition. LC-MS/MS results validated these changes, showing up to 30% mean value reduction in Iba1 tryptic peptides assessed, compared to scramble-siRNA-treated cells.
Conclusion: Even though Iba1 expression in rat microglia was effectively down-regulated in serum-supplemented condition, we found severe cell detachment and aggregation in defined-medium cultures.

**P15. Cost-Effective Alternative for Long-Term Sleep Analysis in Freely Moving Mice**

***Tirlea, Stefan-Alexandru[1]*; Nicolicioiu, Armand[2]; Morozan, Vlad-Petru[3]; Iacovache, Andrei[3]; Ilie, Diana-Andreea[3]; Matota, Ana[3]; Gutu, Biana Ioana[3]; Stancu, Mihai[1]; Zăgrean, Leon[3]; Zagrean, Ana-Maria[1]***

*[1] National Neuroscience Society of Romania, Romania*
*[2] University of Bucharest*
*[3] Carol Davila University of Medicine and Pharmacy*

Sleep analysis poses a plethora of technical issues for the researcher. Studying sleep in freely moving animals usually implies wireless data acquisition devices powered by batteries, which in small animals (e.g. mice) have limited capacity due to their size. While there are companies that provide commercial solutions, their services are difficult to support by underfunded institutions. We created a cost-effective alternative for long-term data acquisition in small freely moving animals and used it to assess alterations of sleep patterns in epileptic mice. We used an intrahippocampal kainic acid model on C57Bl/6 mice to induce epilepsy. Concurrently, we surgically implanted a headstage to perform acquisition of electrophysiology recordings (n = 5, 3 epileptic vs 2 control). The setup allowed for wired ECoG, EMG (4+1 channels), and video acquisition for 24 hours. An in-house engineered device tracks the mouse movements and rotates the wires accordingly, preventing wires torsion and allowing free movement. We created a visual interface in Python that allowed for simultaneous EEG and behavioral analysis. We split the data into 30 seconds epochs and plotted the raw signal and its power spectrum, while programmatically playing the corresponding 30 seconds epochs from the video. We manually classified one recording for each lot and used the labels to train a machine-learning mode for sleep analysis. The acquisition was performed successfully for uninterrupted 24 hours in freely moving conditions for each subject. Furthermore, plotting the raw EcoG and power spectrum in parallel enabled us to apply the sleep stage classification criteria used in the Herrero model. The video sequence was used to better differentiate REM and wake stages, which are commonly known to have similar EcoG morphology. Although a promising approach, more research is needed to assess the setup capabilities of analyzing sleep patterns in different animal models.

**P16. An Equation for an Impossible Task. Does the Brain-Personality System behave in a Thermodynamic Manner? Hot Brain Hypothesis**

***Ionescu, Valentin[1]****

*[1] Faculty of Biology, University of Bucharest*

Introduction: Capturing the activity of the whole brain in one equation is almost an impossible task. Extending the domain of observation to the brain-personality system, using our Hot Brain Hypothesis asserting the variation of cognitive functioning according to the emotional temperature, we propose an equation based on our experimental data. Materials and methods: We organized several encounter group sessions characterized by fluid interactions (empathic, sincere and positive) and taken cortisol samples before and after the interaction (N=39, age 15 to 26 years). We administered questionnaires for fluidity, anxiety, depression, empathy, cognitive flexibility, attention, motivation and passionate love. Empathy, anxiety and depression were used to sample the subjects in three groups: supercold, hot and superhot brain-personality subjects and a self-cognition coefficient was calculated to measure the cognitive bias for supercold and superhot brain-personality types. Testing if they validate the concept of emotional temperature requires to find homologues of pressure and volume functions and observe their variation. Results: The emotional variables give a cognitive variation, mediated by HPA axis' behaviour, in line with Hot Brain Hypothesis. We identified a pressure-function (for cortisol values), a volume-function (for cognitive values) and a direct/indirect proportionality with a temperature-function (for anxiety and empathy per cortisol variation). The perfect gas equation thus adapted has a result captured in a small interval (with SD=0.0004). Conclusion: The brain-personality equation presented here explains the therapeutic impact of a fluid interaction, using the flexibility of HPA axis like a pressure indicator approximating the brain-personality system's behaviour under different emotional temperatures.

**P16. Investigating the relationship between default EEG macrostate reactivity and anaesthetic depth in response to photic stimulation: a rat model**

***Morozan, Vlad-Petru[1]*; Mocanu, Alexandra[2]; Bordeianu, Andrei[1]; Stancu, Mihai[3]; Chirilă, Ana-Teodora[1]; Călin, Alexandru[4]; Pâslaru, Alexandru-Cătălin[1]; Tofan, Laurentiu[1]; Zagrean, Ana-Maria[3]; Zăgrean, Leon[1]; Moldovan, Mihai[5]***

[1] Carol Davila University of Medicine and Pharmacy
[2] University of Bucharest, Faculty of Philosophy
[3] National Neuroscience Society of Romania, Romania
[4] University of Newcastle
[5] University of Copenhagen

We recently proposed that the continuous EEG of awake humans can be regarded as an alternating sequence of reoccurring oscillatory patterns, referred to as oscillatory macrostates. The macrostate that showed the largest decrease in occurrence probability during clinical intermittent photic stimulation (IPS) was labelled as the default oscillatory macrostate (DEM) by analogy with the stimulus-negative behaviour of the default mode network. The aim of this study was to experimentally investigate the relationship between DEM and anaesthetic depth. General anaesthesia was induced by either chloral hydrate (CHL) or isoflurane (ISO) in male adult Wistar rats. The fronto-occipital EEG was recorded by means of cortically implanted wired electrodes. Deep anaesthesia was ensured by the occurrence of discontinuous EEG burst-suppression patterns (BS) comprised of high-power oscillations alternating with flat periods. Monocular IPS at 0.5 Hz was delivered in 60-second trials, alternating with a 60-second recovery period. The DEM reactivity (DER) was calculated as the decrease in DEM occurrence probability during IPS on the EEG channel ipsilateral to the stimulated eye. We found that for both CHL and ISO, DER increased monotonously from continuous EEG to BS. With increasing anaesthetic depth, there was an increasing probability that DEM was the macrostate with the lowest oscillatory power among all macrostates. During BS the DEM accounted for most of the flat periods. We introduced the first rat model to assess the dynamics of the oscillatory EEG macrostates. With increasing anaesthetic depth, the default EEG macrostate occurrence was more strongly supressed by photic stimulation. Its increased reactivity was associated with a decrease in its oscillatory power. Our data suggest that the default EEG macrostate is in a competitive balance with the stimulus positive macrostates, which prevail during deep anaesthesia. It is likely that the default EEG macrostate recovery is necessary for consciousness awakening from anaesthesia.