7th Conference of Lithuanian Neuroscience Association

Program and Abstracts

Organizer

Lithuanian Neuroscience Association

Sponsors

Lietuvos mokslo taryba

interlux

LaboChema

Werfen

Istituto Italiano di Cultura Vilnius

Bitsens

Organizing committee:

Prof. OSVALDAS RUKŠĖNAS
Prof. AIDAS ALABURDA
Dr. RAMUNĖ GRIKŠIENĖ
Dr. INGA GRIŠKOVA-BULANOVA
Dr. VILMA KISNIERIENĖ
Dr. RIMA NAGINIENĖ
Dr. ALEKSANDRAS PLESKAČIAUSKAS

27 November 2015
National Open Access Scholarly Communication and Information Center
Saulėtekio al. 5, Vilnius

PROGRAM

9.15–10.00	**Registration.**
10.00–10.10	**Opening. President of LNA Osvaldas Rukšėnas.**
	I session (Chair – Kastytis Dapšys)
10.10–10.40	**Gintaras Kaubrys.** Healthy ageing and Alzheimer's disease: neurobiological and cognitive differences and connections. *Vilnius University, Vilnius, Lithuania.*
10.40–11.10	**Vidmantas Alekna, Asta Mastavičiūtė**. Healthy ageing: a holistic approach. *Vilnius University, Vilnius, Lithuania.*
11.10–11.30	**Coffee/Tea.**
11.30–12.00	**Mihai Petrovici.** The neuromorphic challenge: theory, circuits and applications. *The Human Brain Project, Kirchhoff-Institute for Physics, University of Heidelberg, Germany.*
12.00–12.30	**Vilmantė Borutaitė**. Ageing and mitochondrial sensitivity to ischaemia. *Lithuanian University of Health Sciences, Kaunas, Lithuania.*
12.30–13.00	**Olga Zamalijeva, Vytautas Jurkuvėnas, Sigita Girdzijauskienė.** Trajectories of cognitive aging: findings from adult cognition studies. *Vilnius University, Vilnius, Lithuania.*
13.00–14.00	**Lunch. Coffee/Tea.**
14.00–15. 30	**Poster session.**
	II session (Chair – Gytis Svirskis)
15.30–16.30	**Plenary lecture. Artūras Petronis.** Epigenetics of mental disorders. *The Campbell family Mental Health Research Institute, University of Toronto, Toronto, Canada.*
16.30–17.00	**Gaetano Tieri**. Wearing my virtual body: behavioral, physiological and neural reactivity to the physical appearance and to the actions of an embodied avatar. *Social and Cognitive Neurosciences Laboratory, IRCCS Fondazione Santa Lucia, Rome, Italy, Department of Psychology, La Sapienza University, Rome, Italy.*
17.00	**Concluding remarks. Awards.**
17.20–19.00	**Party.**

ORAL PRESENTATIONS

HEALTHY AGEING AND ALZHEIMER'S DISEASE: NEUROBIOLOGICAL AND COGNITIVE DIFFERENCES AND CONNECTIONS

G. Kaubrys[1, 2]

[1] Clinic of Neurology and Neurosurgery, Faculty of Medicine, Vilnius University
[2] Centre of Neurology, Vilnius University Hospital Santariskiu Klinikos
E-mail: gintaras.kaubrys@santa.lt

Currently, there is no medication to prevent or modify the progression of Alzheimer's disease (AD). Age is the greatest risk factor for late-onset sporadic Alzheimer's disease (LOAD). Certain aspects of ageing may play crucial role in developing of LOAD. An integrative approach should be used to investigate cognition, gene expression, protein and metabolite changes and levels in cognitively healthy elderly persons and AD patients. Qualitative and quantitative changes in cognitive performance, some molecular markers, vascular pathology, oxidative stress, impaired synaptic function, inflammation – all are associated both with normal ageing and AD. Involution of brain in ageing and neurodegeneration in LOAD may have numerous common aspects, while these seemingly similar cognitive and neurobiological features sometimes lead to outright progressive neurodegeneration, caused or accompanied by Abeta accumulation and tau hyperphosphorylation, and progressive brain atrophy and dementia, while in other cases a person continues to accumulate the mild cognitive and neuropathological changes, but still retains normal general cognitive functioning level far beyond the mean life expectancy in overall population. Investigation and understanding of the cognitive decline patterns, physiological and molecular mechanisms that are shared and those, which are different in normal ageing and AD, may help to delineate an understanding of the etiopathogenesis of AD. These insights could lead to novel medications for late-onset sporadic AD.

The modern data about the similarities and differences of cognitive, genetic, and other features of persons in normal ageing and AD are delineated and analyzed in the presentation. Possible connections between normal ageing and AD, and corresponding consequences for the understanding of AD mechanisms are highlighted and discussed.

HEALTHY AGING: A HOLISTIC APPROACH

V. Alekna, A. Mastavičiūtė, M. Tamulaitienė

Vilnius University, Faculty of Medicine
E-mail: asta.mastaviciute@vu.mf.lt

Lithuanian population is ageing rapidly. The proportion of population aged 60 or over doubled since 1960 up to 24 percent. Eurostat has predicted that in 2040, one in three Lithuanian residents will be aged 65 or older compared to one in five nowadays. It is critical to meet the challenge of various social, medical and economic problems common in older people with the aim to increase the years of healthy life. Healthy ageing allows people to implement their potential for physical, social and mental well-being. According to World Health Organisation healthy ageing starts at birth with our genetic inheritance. As people age, they experience a gradual decrease in physiological reserves and can be affected the development of other health characteristics, such as diseases, and geriatric syndromes (frailty, dementia, gait problems, falls and fractures, malnutrition, and increased frequency of infection). Most diseases and conditions causing disability usually develop during a relatively short time before death. Personal characteristics which include gender, ethnicity, social norms, occupation, education and wealth, are also important determinants of healthy ageing. There are few ways of healthy ageing dependently on lifestyle. Healthy lifestyle behaviours increase the chance of life expectancy, but with the different chances of morbidity. The interaction of these intrinsic factors and environments makes a foundation for the individual's functional ability. The environments include home, age-friendly communities and the broader society. The environments play rather a critical role in determining the way of person's ageing and responding to different experiences throughout life.

In conclusion, the holistic approach to healthy ageing requires active efforts to the wider determinants of health, promoting healthy lifestyles and wellness, ensuring getting housing right for older people and preventing social isolation.

THE NEUROMORPHIC CHALLENGE: THEORY, CIRCUITS AND APPLICATIONS

M. A. Petrovici[1, 2]

[1] Kirchhoff-Institute for Physics, University of Heidelberg, Germany
[2] The Human Brain Project
E-mail: mpedro@kip.uni-heidelberg.de

Due to the vast complexity of the human brain and the manifest nonlinearity of the equations describing the dynamics and interaction of its components, a purely analytical understanding of cognition and behavior appears difficult to attain. Therefore, modern theoretical neuroscience relies heavily on combining the rigorousness of mathematical analysis with the number-crunching power of computer simulations.

Over the past decades, the increase of available computational power in accordance to Moore's law has driven the evolution of the field of computational neuroscience. However, it has become increasingly clear that brain-scale simulations on modern supercomputers (scaled up to an appropriate size) will be extremely - if not prohibitively - power-hungry. The power problem is a consequence of the conventional architecture of general-purpose processors, which is obviously not optimized towards neural network simulation. Neuromorphic hardware is the product of an entirely different approach, which aims at implementing various aspects of brain architecture and dynamics directly in silico.

Starting from biological data, I will discuss abstract neuron and synapse models and how they can be realized in VLSI circuits. These single unit models need to then be connected in an architecture that allows the flexible definition of network models. In this context, I will use the Heidelberg single chip and wafer-scale systems as an exemplary case. A particular focus will lie on the configurability of neuron and synapse parameters and the problems that arise from spatial and temporal parameter noise. Since calibration of the system can only overcome these effects to a limited extent, I will discuss how remaining network-level distortions can be handled by modelers using these machines. As an outlook, I will outline several models that are currently being developed which are expected to draw major benefits from their implementation on analog neuromorphic hardware.

TRAJECTORIES OF COGNITIVE AGING: FINDINGS FROM ADULT COGNITION STUDIES

V. Jurkuvėnas[1, 2], S. Girdzijauskienė[1], O. Zamalijeva[1, 2]

[1] Vilnius University, Department of General Psychology
[2] Vilnius University, Laboratory of Special Psychology
E-mail: olga.zamalijeva@fsf.vu.lt

Lithuania along with other countries in the West is experiencing demographic aging. These demographical changes have important socio-economic and health consequences for societies. Age is not only a risk factor for physical and mental illness, but is also associated with cognitive decline. Cognitive aging is a process of gradual and progressive weakening of several cognitive functions. It has long been assumed by many researchers in psychology and medicine that cognitive aging, unrelated to pathology, only has a small effect on a person's general cognitive functioning. However, mounting evidence suggest that cognitive aging is a multidimensional process and that at least in some areas of cognition differences between age groups are quite significant and different trajectories of various cognitive functions can be observed. The most common distinction is made between crystallized and fluid abilities. It seems that crystallized abilities tend to increase or at least not to decline with age. On the other hand, the decline of fluid abilities can be shown even at the age of 30 year or earlier. Both, biological and psychological theories explaining cognitive decline are still being developed and are only partially supported with empirical data. Although cognition declines with age, there are great individual differences between people of the same age. It is hypothesized that at least partially age-cognition relationship is mediated by psychosocial variables. Our studies show that social support, neuroticism, subjective physical health, socioeconomic status and education play a role in age-cognition relationship. Further research should investigate the role of age-cognition mediators in longitudinal design studies and focus on identifying modifiable protective factors that might help insure best possible cognitive functioning of aging population.

Plenary lecture

EPIGENOMICS OF PSYCHIATRIC DISEASE

A. Petronis[1, 2]

[1] Campbell Family Mental Health Research Institute
Centre for Addiction and Mental Health
[2] University of Toronto, Canda
E-mail: art.petronis@camh.ca

Putative epigenetic (epiG) misregulation of genes sheds a new light on numerous epidemiological, clinical, and molecular complexities of non-Mendelian diseases. Perfect DNA sequences may be useless or even harmful if not expressed in the appropriate amount, at the right time of the cell cycle, or in the right compartment of the nucleus. EpiG modifications of DNA and histones, more so than DNA sequence-based ones, can explain a series of general non-Mendelian features of the three major psychiatric diseases: schizophrenia, bipolar disorder, and major depression. Such features include: discordance of identical twins; relatively late age of onset and coincidence of the first symptoms with changes in the hormonal status in the organism; sexual dimorphism; parental origin effects; fluctuating course and sometimes partial or even full recovery. Apart from the general epiG aspects of non-Mendelian irregularities, epiG mechanisms may also provide a new perspective on the neurochemical and neurodevelopmental findings as well as identification of the molecular effects of environmental and stochastic factors. The epiG theory does not reject the role of DNA sequence variation but rather suggests that in complex psychiatric diseases the contribution of epiG factors may be substantial, and that DNA sequence variation should be investigated in parallel with epiG regulation. I will discuss the key theoretical principles of psychiatric epigenomics and review recent experimental activities dedicated to identification of inherited and acquired epigenetic changes in common psychiatric diseases.

WEARING MY VIRTUAL BODY: BEHAVIORAL, PHYSIOLOGICAL AND NEURAL REACTIVITY TO THE PHYSICAL APPEARANCE AND TO THE ACTIONS OF AN EMBODIED AVATAR

G. Tieri[1, 2, 3]

[1] Department of Psychology, Sapienza University of Rome, Rome, Italy
[2] Fondazione Santa Lucia, IRCCS, Rome, Italy
[3] Braintrends Ltd, Applied Neuroscience, Rome, Italy
E-mail: gaetano.tieri@uniroma1.it

The feeling that our body belongs to us (Ownership) and that we are responsible for its actions (Agency) represent the two basic aspects of the Embodiment and are fundamental for bodily self-consciousness and for interacting optimally with objects and other individuals. Over the last two decades neurological and psychological studies have shown that the Embodiment is a plastic construct that relies on brain regions involved in multisensory integration. In this talk, I will present a series of studies where Embodiment and its changes are explored by means of Immersive Virtual Reality. More specifically, we recorded behavioral, physiological and neural responses underlying changes of embodiment obtained through a mere passive observation of a virtual body in a first-person perspective. I will show how using either Head Mounted Display or CAVE automatic virtual environment system, it is possible to induce a clear illusory feeling of Embodiment over a virtual body and to modify the inducing feeling by manipulating the visual appearance of the virtual body itself and the appropriateness of its actions. Moreover, I will provide evidences that perceptual and motor properties of the virtual body, modulate behavioral, autonomic (skin conductance and skin temperature) and neural (EEG) responses of the people who embody it. Finally, I will discuss possible applications of these results to improve the current Brain-Body Computer Interface systems and thus help people with Spinal Cord Injury to re-interact with objects and other individuals.

POSTER PRESENTATIONS

P1. INFLUENCE OF 9-METHYL-2[-3-(4-PHENYL-1--PIPERAZINYLPROPYL)]-1,2,3,4-TETRAHYDRO-β--CARBOLIN-1-ONE ON NOREPINEPHRINE LEVELS IN PREGNANT WISTAR RAT BRAINS AND DEVELOPMENT FETUSES

A. Anasiewicz, U. Cieślak, E. Kuśmierczuk, J. Jarosz-Czapnik, W. Biel

Department of Human Anatomy, Medical University in Lublin, Poland
E-mail: agnes.anas@gmail.com

The chemical compound 9-methyl-2[-3-(4-phenyl-1-piperazinylpropyl)]-1,2,3,4-tetrahydro-b-carbolin-1-one has been synthesized at the Institute of Pharmacology, Polish Academy of Sciences in Cracow. This chemical compound has a potent antiserotonin action.

The studies were performed as recommended by WHO. Macroscopic external evaluation of fetuses, both sectional and skeletal according to Dawson's and Peter's methods were employed. The evaluation of birth defects of internal organs was carried out according to Wilson's technique in Barrow's and Taylor's modification.

Pregnant females (10-12 rats in each group) were treated with 1/50, 1/100, 1/250, 1/500, 1/1000 of DL_{50} (650 mg/kg body mass) of 9-methyl-2[-3-(4-phenyl-1-piperazinylpropyl)]-1,2,3,4-tetrahydro-b-carbolin-1-one by gavage on each day 7-14 of gestation. Controls were performed on rats: UC – untreated, TC – treated with H_2O by gavage in equal volume, TCc- treated with carboxymethylocellulose by gavage in equal volume and ST-chlormethine hydrochloride.

Females were euthanized and caesarean sections were performed on last day of gestation and malformations of fetuses were determinate by gross examination and Alcian Blue with Alizarin Red double skeletal staining. Obtained brains of pregnant females were fixed in liquid nitrogen homogenised and tested.

On the basis of these studies it has been found out that 9-methyl-2[-3-(4-phenyl-1-piperazinylpropyl)]-1,2,3,4-tetrahydro-b-carbolin-1-one in all doses has teratogenic effects. A significant differences of norepinephrine levels in pregnant females brains after 1/50 of DL_{50} and 1/100 of DL_{50} of 9-methyl-2[-3-(4-phenyl-1-piperazinylpropyl)]-1,2,3,4-tetrahydro-b-carbolin-1-one was noted.

P2. INFLUENCE OF CADMIUM AND GREEN TEA EXTRACT ON LIPID PEROXIDATION IN BRAIN AND RED BLOOD CELLS OF MICE

R. Bernotienė, A. Simanaitienė

Neurosciences Institute and Department of Biochemistry,
Medical Academy, Lithuanian University of Health Sciences, Kaunas, Lithuania
E-mail: rasa.bernotiene@lsmuni.lt

Cadmium (Cd) is a toxic, nonessential transition metal, classified as a human carcinogen by the National Toxicology Program. Cd can enter into the brain parenchyma and neurons causing neurological alterations in humans and animal models, leading to lower attention, hyper nociception, olfactory dysfunction and memory deficits. However, mechanisms underlying Cd neurotoxicity remain not completely understood.

It is known that green tea extract (GTE) is characterized as one of natural antioxidants source, which can be used to protect cells from oxidative stress damage. So, the present study was conducted to investigate whether the GTE could play any protective role against the potent neurotoxin Cd-induced oxidative impairment in mice brain and red blood cells.

Experiments were done on outbred white laboratory mice using intraperitoneal injections of $CdCl_2$ (0,16 mg Cd^{2+}/kg body weight) and/or GTE (50 mg GTE/ kg body weight) solutions. The exposure-time was 6 weeks. Lipid peroxides were estimated by measuring thiobarbituric-acid-reactive substances and were expressed as malondialdehyde (MDA).

Our experiments showed that Cd increased lipid peroxidation in mice brain by 30% in comparison with control mice group. Meanwhile, in GTE and Cd+GTE mice groups, the content of MDA was decreased by 12% and 22%, respectively, as compared to control mice group. Our results showed that GTE can reduce the effects of Cd on mice brain.

Further experiments carried out in order to examine influence of Cd and/or GTE on the content of MDA in mice red blood cells. The results indicated that in all groups MDA content was at the control level.

Our results showed that $CdCl_2$ increased lipid peroxidation in mice brain, meanwhile GTE decreased lipid peroxidation in mice brain. Moreover, GTE is capable to reduce lipid peroxidation caused by $CdCl_2$ in mice brain. Besides, our results indicated that in all groups lipid peroxidation in mice red blood cells was at the control level.

P3. MITOCHONDRIA DERIVED ROS MEDIATE AB_{1-42} INDUCED NEUROTOXICITY IN PRIMARY NEURONAL CULTURES

A. Bružas, S. Jankevičiūtė, P. Čižas, V. Borutaitė, R. Morkūnienė

Institute of Neurosciences, Lithuanian University of Health Sciences, Kaunas, Lithuania
E-mail: arturasbruz@gmail.com

Oxidative stress is involved in neurodegenerative diseases including Alzheimer's disease. Mitochondria may produce large amount of reactive oxygen species (mtROS), resulting in mitochondrial dysfunction and neuronal death. Recent evidence suggests that mtROS may be important regulator of inflammatory response, however, the role of mtROS in Aβ-induced neuronal death and microglial activation is not entirely understood. In our study, we investigated whether various Aβ aggregates alone or in complexes with specific antibodies are capable to induce mtROS in rat primary neuronal-glial cultures. Results with MitoSOX Red, a mitochondrial superoxide indicator, showed that only small $Aβ_{1-42}$ oligomers (<5 nm) were capable to induce superoxide production in neurons and microglial cells. Larger $Aβ_{1-42}$ oligomers (>5 nm) and $Aβ_{1-42}$ fibrils did not cause mtROS production. MitoTEMPO, a scavenger of mitochondrial superoxide, significantly decreased superoxide level in both, neurons and microglia after 1 h incubation with small $Aβ_{1-42}$ oligomers. In addition, Aβ-induced mtROS in microglia was also prevented by antioxidant N-acetyl-L-cysteine and Ca^{2+} chelator BAPTA, indicating that Ca^{2+} may be involved in Aβ-caused generation of mtROS in microglia. Large Aβ oligomers (> 5nm) became neurotoxic in complexes with specific antibodies and stimulated acute production of mitochondrial superoxide in microglia, which was significantly decreased by MitoTEMPO. Effect of immune complexes on mtROS in neurons was mild and insensitive to MitoTEMPO. Importantly, immune complex- mediated neuronal loss and microglia proliferation was significantly decreased by MitoTEMPO after 24 h. Our findings demonstrate that small Aβ oligomers cause production of mitochondrial superoxide in neurons and microglia. MtROS may mediate activation of microglia cells and phagocytosis in immune complex- induced neurotoxicity, which can be rescued by selective mitochondrial antioxidant MitoTEMPO.

P4. STATIONARY STIMULUS SIZE AFFECTS RECEPTIVE FIELD AREA IN THE VISUAL NEURONS OF THE RAT SUPERIOR COLLICULUS

E. Dakneviciute, G. Svirskis, G. Baranauskas

Neurophysiology laboratory, Neuroscience Institute,
Lithuanian University of Health Sciences, Kaunas, Lithuania
E-mail: ciauskutia0@gmail.com

The concept of receptive fields is widely used in vision research. A receptive field is defined as the region of visual field over which one can influence the firing of the recorded neuron. Outside this region no visual stimulus can evoke a response. Typically receptive fields are determined by probing with a small, less than 5º stimuli. It has been shown that in the rodent superior colliculus neurons typical receptive field diameter ranges from 5º to 40º. Here we show that in the neurons of the rat superior colliculus stationary small 1.5º and 3.75º in diameter stimuli evoked responses from an area of 486 deg^2 – 1293 deg^2, corresponding to 22º – 36º diameter, while large 15º stimuli evoked responses from 2882 deg^2 – 4008 deg^2, corresponding to 54º – 63º diameter, in many cases the response area was limited by the size of the monitor, used for stimulus presentation. Similar result was obtained both with multi-unit recordings and with single units and with the gamma-frequency (40 Hz – 120 Hz) local field potential power signal. These data show that in the rat the superficial layer superior colliculus neurons can receive direct and/or indirect retinal ganglion cell inputs from most retinal areas.

Acknowledgments: This work was funded in part by the European Social Fund under the Global Grant measure.

P5. AN ASSESSMENT OF DIFFERENT MODALITIES COVERING A CONSTRUCT OF INTEROCEPTIVE AWARENESS

A. Grabauskaitė, M. Baranauskas, I. Griškova-Bulanova

Department of Neurobiology and Biophysics, Vilnius University, Lithuania
E-mail: aida.grabauskaite@gf.stud.vu.lt

The interoceptive awareness may be defined as a set of a person's beliefs in his or her own abilities and tendencies in concentrating on his or her senses originating from inside the body. The concentration on one's body senses may lead either to maladaptive or adaptive behavior, e.g. exaggerating one's symptoms and getting too anxious or focusing one's attention to breathing and getting calmer.

For better understanding of interoceptive awareness, Wolf Mehling and his colleagues (2012) created a psychological assessment tool, called the "Multidimensional Assessment of Interoceptive Awareness" (MAIA). The aim of our study was to adapt the MAIA for the Lithuanian population and for the first time to compare performance between genders. We conducted a systematic forward-back translation of the MAIA, a 32-item, 8-scale tool, and gave it to a total number of 376 participants (192 males), with age ranging from 17 to 30 years (median age 21).

The Cronbach's alpha of Lithuanian version of MAIA's different scales ranged from 0,402 to 0,820, the 5 of 8 scales appeared to be reliable (*Noticing, Not-Distracting and Not-Worrying* did not*)*, similarly to the MAIA in other languages. The median scores for all participants for the each scale were as follows: 3.5 for *Noticing*, 2 for *Not-Distracting*, 2.33 for *Not-Worrying*, 2.86 for *Attention regulation*, 3.4 for *Emotional Awareness*, 2.5 for *Self-Regulation*, 2.33 for *Body Listening* and 4 for *Trusting.* Women had higher median scores in the scales of *Noticing* (i.e. 3.5>3.25, p=0.021) and *Emotional Awareness* (3.4>3.2, p=0.003), while men had higher scores in *Not-Worrying* (2.5>2.0, p<0.001) and *Trusting* (4.0>3.67, p<0.001).

Our results confirmed the multidimensional nature of the interoceptive awareness construct, showing differences between the genders.

P6. COMPUTATIONAL MODEL OF PERIODIC SPIKING IN NEURAL NETWORKS: IMPLICATION FOR SCHIZOPHRENIA

R. Jackevičius[1], I. Griškova-Bulanova[2], B.P. Graham[3], A. Saudargienė[1]

[1] Department of Informatics, Vytautas Magnus University, Kaunas, Lithuania
[2] Department of Neurobiology and Biophysics, Vilnius University, Vilnius, Lithuania
[3] Division of Computing Science & Mathematics, School of Natural Sciences, University of Stirling, Stirling, UK
E-mail: a.saudargiene@if.vdu.lt

Schizophrenia is a mental disorder that affects about 1% of the population. It is characterized by symptoms, affecting thoughts, behavior and social aspects. Major neuronal degeneration is not observed in schizophrenic patients, but abnormalities in cortical circuits are present. These abnormalities are reflected in impaired EEG g frequency (30-80Hz), being crucial for many processes including sensation, perception, working memory, attention, etc. Reduced synaptic connectivity and NMDA receptor hypofunction are proposed as a mechanism underlying the decreased g frequency oscillations in schizophrenia.

In this study we investigate the effect of reduced network connectivity on g frequency oscillations applying a computational modeling approach. A model of a spiking neural network of cortical area is composed of 800 pyramidal neurons, 150 regular-spiking interneurons and 50 fast-spiking interneurons (Spencer in Frontiers in Human Neuroscience 3, 2009). All cells are randomly interconnected and have recurrent connections within the populations of pyramidal neurons, fast-spiking and regular-spiking interneurons. The input to the network consists of random spikes at 100 Hz frequency to each cell. The populations of pyramidal neurons, regular-spiking and fast spiking interneurons generate synchronous oscillations at 40Hz. We reduce the recurrent connectivity between the pyramidal neurons and the total connectivity between all cells. The strongest effect is observed for the impaired recurrent

connectivity: reduction by 20% of synaptic connectivity between the pyramidal neurons reduces the excitability of the network and abolishes g frequency oscillations in all three cell populations. This effect is dependent on the synaptic weight strength: g frequency oscillations are impaired only for strong recurrent connections. Decrease in total connectivity increases network excitability due to disinhibition and again prevents g frequency oscillations.

In this way network resembles reduced connectivity similarly to observations made in schizophrenia, suggesting that computational modeling is a powerful tool to facilitate understanding of healthy and pathological functioning of neuronal networks in the brain.

P7. ALZHEIMER'S DISEASE PATIENTS' CEREBROSPINAL FLUID INDUCES NEURONAL DEATH

S. Jankevičiūtė[1], G. Pšemeneckienė[2], E. Grušauskienė[2], K. Petrikonis[2], R. Morkūnienė[1], V. Borutaitė[1]

[1] Institute of Neurosciences, Lithuanian University of Health Sciences, Kaunas, Lithuania
[2] Department of Neurology, Lithuanian University of Health Sciences, Kaunas, Lithuania
E-mail: silvija.jank@gmail.com

Alzheimer's disease (AD) is the most common form of dementia. The biomarker studies based on cerebrospinal fluid (CSF) and blood serum (BS) nowadays represent the most direct and convenient means to study disease progression. Unfortunately reliable biomarkers for early diagnostics of AD are not determined yet. The use of model systems, such as primary cell cultures, and biomarkers in BS and CSF may serve as a new method of early AD diagnostics. The aim of this study was to investigate effects of AD and other dementia patients' blood sera and cerebrospinal fluids on primary cerebellar granule cell cultures (CGC).

Experiments were carried on rat primary mixed neuronal-glial CGC cultures. BS and CSF were donated by healthy, age-matched individuals (with zero degree of dementia, HP, n=21), patients with Alzheimer's disease (DAT, n=24) and other dementia patients n=9. AD group consisted of individuals with early (E-DAT, n=14) or middle-stage (M-DAT, n=10) of dementia. CGCs were incubated with BS and CSF for 24 hours.

We found that sera of all groups have no effect on cell viability. BS and CSF from AD groups reduced density of neurons in cultures. It is important that only M-DAT patients' CSF caused neurotoxic effects and reduced the viability of neurons by 16-27% compared to the control and HP groups. HP biological fluids stimulated microglia proliferation, but AD (E and M) and other dementias BS and CSF samples inhibited the proliferation of microglia. The highest inhibitory effect was noted with M-DAT patients' BS, which reduced the number of microglia by about 200%.

Our study revealed that BS and CSF from patients with AD but not HP and other dementia patients reduced density of neurons in primary neuronal-glial cultures. The results show that only M-DAT patients CSF significantly reduced viability of neurons. This can be used as a basis for development of diagnostic methods for early diagnostics of Alzheimer's disease.

P8. INITIAL CHARACTERIZATION OF THE BEHAVIOURAL PHENOTYPE OF NTM-DEFICIENT MICE

T. Mazitov, J. Innos, E. Vasar

University of Tartu
E-mail: timur.mazitov@yahoo.com

Neurotrimin protein (Ntm) belongs to the IgLON family of proteins (with Lsamp, Obcam, Kilon and IgLON5) and might be both a homo- and a heterophilic cell adhesion molecule. In human studies the members of the IgLON family have been implicated in psychiatric disorders, obesity and certain types of cancer. The study provides an initial characterization of the behavioural phenotype of Ntm-deficient mice.

Ntm gene deficient male mice (received from University of California, Davis) and their wild-type littermates were tested in behavioral tests. Also some preliminary pharmacological experiments with amphetamine and ethanol were performed.

In vision, hearing and olfaction tests, Ntm-deficient animals had normal sensory abilities. In three anxiety tests (hyponeophagia, elevated plus maze and light/dark box) and in the locomotor activity test (motility box) the behaviour of Ntm-deficient mice did not differ from that of their wild-type littermates. There were no differences in the Morris water maze test between the genotypes. In a memory test that has a strong emotional component (the active avoidance test) Ntm-deficient mice tended to learn slower. In the loss of righting reflex test with 4 g/kg of ethanol, there was no difference between the genotypes; in the locomotor activity test with 5 mg/kg of amphetamine Ntm-deficient mice were, similarly to Lsamp-deficient mice, less sensitive to the locomotor stimulating effect of amphetamine.

In this study, the initial phenotyping of Ntm gene mice was performed. Ntm-deficient mice have learning impairment and altered sensitivity to amphetamine, possibly reflecting changes in the dopaminergic system. The results of this study show that, although Lsamp and Ntm are close interaction partners, forming heterodimers, the deletion of Lsamp induces more significant changes in the phenotype at both behavioural and pharmacological level than the deletion of Ntm.

P9. THE RELATIONSHIP BETWEEN MEN AND WOMEN RESTING CORTICAL ALPHA ASYMMETRY AND PERSONALITY TRAITS ASSESSED USING NEO PI-R QUESTIONNAIRE

R. Mončiunskaitė, R. Grikšienė, I. Griškova-Bulanova, L. Jarutytė, O. Rukšėnas

Vilnius University, Department of Neurobiology and Biophysics
rasa.monciunskaite@gf.stud.vu.lt

Resting frontal alpha asymmetry is considered to be a reliable physiological index of the trait and state reactivity that represents person's positive versus negative affective styles or approach and withdrawal motivation. Abundant literature about alpha asymmetry and the main personality domains data is inconsistent. The aim of the present study was to evaluate the relationship between resting cortical $alpha_1$ (8-10 Hz), $alpha_2$ (10-13 Hz) and $alpha_{Total}$ (8-13Hz) asymmetry and the main personality domains or facets (empathy) assessed using NEO PI-R questionnaire based on Big Five Model of personality in men and women.

Resting state EEG and personality profiles of 51 men (23.5 ± 3.6 SD years) and 83 women (22.6 ± 2.7 SD years) were assessed. Three minutes artifact free resting state EEG recordings were Fast Fourier Transform processed. Asymmetry indexes of $alpha_1$, $alpha_2$ and $alpha_{Total}$ power, for selected frontal, central and parietal electrodes pairs were calculated. Higher values of the asymmetry index indicate relatively greater activation of the left hemisphere.

We found that Neuroticism manifests significantly stronger in men while Extraversion and Consciousness - in women. Openness to experience and Agreeableness do not differ between genders. The statistical analyses of alpha power demonstrated significant gender differences in $alpha_1$, $alpha_2$ and $alpha_{Total}$ asymmetry in frontal and frontocentral areas – right hemisphere was more active in men versus women. Higher level of Neuroticism in women was inversely related to $alpha_2$ asymmetry score over anteriofrontal ($r = -0.24$, $p = 0.04$), frontal ($r = -0.23$, $p = 0.04$) and parietal ($r = -0.29$, $p = 0.01$) areas. Stronger Extraversion manifestation was positively related to frontal $alpha_1$ asymmetry ($r = 0.40$, $p = 0.001$) in men, $alpha_2$ ($r = 0.25$, $p = 0.04$) in

women which indicates relatively stronger left frontal activity. Whereas men demonstrated inverse Extraversion and $alpha_1$ asymmetry ($r = -0.35$, $p=0.02$) index relationship in parietal electrodes. Negative correlation between men Openness to experience score and frontal $alpha_1$ asymmetry index ($r = -0.43$, $p=0.003$) suggests this personality dimension is related to a relatively greater right frontal activity. Correlations between Agreeableness and $alpha_2$ asymmetry index revealed relatively higher left frontal activity in men ($r=0.3$, $p<0.05$) as well as women ($r = 0.25$, $p<0.05$). Empathy was related to right anteriofrontal ($alpha_1$ $r = -0.32$, $p=0.03$) and left central ($alpha_1$, $alpha_2$ all $r \geq 0.3$, all $p \leq 0.04$) and parietal ($alpha_1$ $r = 0.4$, $p=0.01$) activity only among men. No correlations were found between Consciousness score and resting alpha asymmetry index among both genders.

In conclusion, anteriofrontal and frontal cortical activity tends to be more related to the main personality domains in both men and women: relatively higher right cortical activity relates more to Neuroticism, Openness to experience and Empathy, while left – to Extraversion, Agreeableness. Correlations between asymmetry index and personality domains tend to be more focused in $alpha_1$ band among men whereas in women it is distributed in both $alpha_1$ and $alpha_2$.

P10. KARHUNEN–LOÈVE TRANSFORM BASED MORPHOLOGICAL EVALUATION OF SINGLE SWEEP VISUALLY EVOKED POTENTIALS

A. Kybartaitė-Žilienė[1], A. Gelžinis[2], A. Kriščiukaitis[1]

[1] Neuroscience Institute, Lithuanian University of Health Sciences, Lithuania
[2] Ophthalmology Department, Hospital of Lithuanian University of Health Sciences, Lithuania
E-mail: asta.kybartaite@lsmuni.lt

Visually evoked potentials (VEPs) are signals evoked by a visual stimulus. They consist of brief discrete deflections embedded in background electroencephalographic (EEG) activity, which often has larger amplitude. Background EEG cancellation is a major part of VEPs analysis algorithms often realized by coherent averaging or other methods requiring large minimal amount of registered sweeps. In some cases, especially for pediatric patients, or in poor patient compliance cases, long procedure duration and fatigue might cause impaired attention and non-steady target fixation, affecting the quality of recorded VEPs. The possibility to reconstruct VEPs in every single sweep from limited size ensembles opens new diagnostic possibilities and shortens registration procedure improving its quality. Also analysis of single sweeps is a valuable tool for studying induced plastic modifications in visually evoked responses and possible signal habituation effect. Multivariate analysis methods are known as powerful tool for quasi-periodic signal morphological analysis. In this study we demonstrate a possibility of single-sweep VEP reconstruction by means of truncated expansion (Karhunen–Loève transform) using optimally time adjusted generalized basis functions.

We perform Karhunen–Loève transform of every single sweep containing VEP using generalized universal basis functions (eigenvectors of covariation matrix) from learning set of sweeps, i.e., an ensemble of collected typical recordings. For calculation of basis functions we used 740 sweeps from high quality recordings from 8 different subjects. Certain pathologies evoke varying latency in recorded VEPs, so we propose to use optimal shift in time of generalized basis functions when processing each individual sweep.

The synthetic signal containing VEPs with known jitter and amplitude variation was created for testing and evaluation of the method. Also known amplitude background EEG was added to the testing signal. An acceptable quality for visual examination and diagnostics of reconstructed signal was achieved when ratio of VEP amplitude to background EEG reached -10 dB. The difference between the modelled latency shift and the one estimated from the reconstructed signal was 8.38 ± 0.74 ms at the same -10 dB VEP to background EEG ratio.

Application of adaptively time-shifted basis functions enables optimal reconstruction of single sweep VEP signal with latency shift or jitter and opens qualitatively new diagnostic possibilities.

P11. SUPEROXIDE DISMUTASES ACTIVITY IN MICE BRAIN UNDER SELENIUM AND / OR ALIUMINIUM IONS TREATMENT

I. Sadauskienė[1, 2], I. Stanevičienė[1, 2], K. Adomaitytė[3], A. Liekis[1], R. Naginienė[1], V. Šimakauskienė[1], D. Baranauskienė[1, 2]

[1] Neuroscience Institute, Lithuanian University of Health Sciences
[2] Department of Biochemistry, Medical Academy, Lithuanian University of Health Sciences
[3] Department of Neurobiology and Biophysics, Faculty of Natural Sciences, Vilnius University
E-mail: ilona.sadauskiene@lsmuni.lt

Within a cell superoxide dismutases (SOD) are the first line of defence against reactive oxygen species. The present study was conducted to investigate the influence of Se and/or Al ions on the SOD activity in brain as well as distribution of these elements in the blood and the brain of laboratory mice.

Experiments were done on 4-6 weeks old outbreed mice. SOD activity was determined in brain after 24 h and 14 d. Al and/or Se solution i.p. injections. SOD activity was determined spectrofotometrically. The concentration of protein was measured by the Warburg-Christian method. Se and Al concentrations were determined by inductively coupled plasma mass spectrometer.

It was evaluated the effect of Al on SOD activity in mouse brain after a single i.p. Al injection. The results showed that SOD activity was the same value in control and experimental groups. After a single Se dose injection SOD activity decreased by 28.6% (compared to the control group). Subsequently, were evaluated changes in SOD activity following a single Se+Al mixture injection. It was observed a significant decrease in SOD activity (17.8%).

In further experiments, there was evaluated the effects of Al and/or Se on SOD activity after 14 d. i.p. injections. The results showed that injections of these elements alone did not cause changes of SOD activity. The data of the effect of both elements showed that SOD activity decreased by 41.5% (compared to the control group).

Estimation of the element distribution in brain homogenates showed that after 24 h and 14 d. concentrations did not differ from controls. While in blood: after 24 h exposure levels of Se increased in Se and Se+Al group (22% and 41% respectively) and after 14 d. increased Se and Al concentrations. It is also established in Se+Al group, but Al concentrations are significantly lower.

Our studies revealed that the Se and Al total effect in the mice brain reduce the enzymatic SOD activity after 24 h and 14 d. repeated exposure.

P12. 5-HYDROXY-1,4-NAPHTHALENEDIONE ALTERS ENERGY METABOLISM IN GLIOMA CELLS

K. Sidlauskas, R. Bagdonaviciute, D. Majiene, J. Liobikas

Lithuanian University of Health Sciences,
Neuroscience Institute, Kaunas, Lithuania
E-mail: kastytis.sidlauskas.11@ucl.ac.uk

5-hydroxy-1,4-naphthalenedione (juglone) belongs to a class of naphthoquinones. Recently, the attention has been drawn to the anticancer properties of this substance. However, the effect of juglone has not been evaluated on gliomas - one of the most common and deadliest brain tumours. Not only do these tumours show high rates of cell proliferations, they are also highly infiltrative. In this study, we chose cultured C6 glioma cells as our experimental *in vitro* model system. The obtained data revealed that juglone induced cell death in a concentration dependent manner. On the contrary, the cytotoxicity of juglone did not depend on the incubation time. The non-cytotoxic concentrations of juglone reduced cell proliferation and invasion rates. In order to obtain more details on the mechanisms of cytotoxicity, we investigated the changes induced by juglone in oxidative phosphorylation system of C6 glioma cells. We found that juglone reduces the basal and ADP-stimulated mitochondrial respiration rates with pyruvate/malate. Moreover, C6 cell treatment with amytal, an inhibitor of mitochondrial complex I, reduced the cytotoxic effect of juglone. Overall, this study shows that juglone might be a potential anticancer agent. However, further *in vivo* studies are needed.

P13. MOLECULAR PATHOLOGY OF MONOGENIC EPILEPSIES

B.Tumienė[1, 2], A. Maver[3, 4], B. Peterlin[3, 4], V. Kučinskas[1, 2], A. Utkus[1, 2]

[1] Department of Human and Medical Genetics, Faculty of Medicine, Vilnius University, Vilnius, Lithuania
[2] Centre for Medical Genetics, Vilnius University Hospital Santariškių Clinics, Vilnius, Lithuania
[3] Clinical Institute for Medical Genetics, Division of Gynecology, University of Ljubljana Medical Centre, Ljubljana, Slovenia
[4] Faculty of Medicine, University of Ljubljana, Slovenia
E-mail: tumbir@gmail.com

The aim of the study was the evaluation of molecular pathology of monogenic epilepsies through bioinformatic analyses and exome sequencing studies.

Gene catalog of 798 epilepsy-associated genes was comprised through extensive bioinformatic analyses. Exome sequencing was applied to 51 patients with epilepsy. Molecular pathology of monogenic epilepsies was evaluated in the gene catalog according to variant inheritance patterns and gene functional annotation and compared to the results of exome sequencing.

Through application of exome sequencing, pathogenic, likely pathogenic or strong candidate variants were found in 29 epilepsy-associated genes and one novel, not previously associated with human diseases gene. Autosomal dominant, mostly *de novo* variants dominated in a cohort of patients whereas genes with autosomal recessive pattern of inheritance comprised more than a half of epilepsy gene catalog. Two patients had dual diagnoses. Classification of gene catalog's genes according to functional annotation revealed that almost half (44%) of genes were involved in (1) metabolic pathways while others had functions in (2) signaling/receptors (18%), cell cycle/cytoskeleton (11%), transport/channels (10%), gene expression regulation (7%), neurodevelopment (6%), DNA and RNA maintenance (2,5%) and unknown (1,5%).

Bioinformatic analyses and exome sequencing studies allow for elucidation of previously largely unknown molecular pathology of monogenic epilepsies.

P14. INFUSION OF ROTENONE PROTECTS RAT BRAIN MITOCHONDRIA AGAIST ISCHEMIC DAMAGE

E. Rekuviene[1], R. Jonikas, L. Ivanoviene[1], V. Borutaite[1,2], R. Morkūniene[1,2]

[1]Department of Biochemistry, Lithuanian University of Health Science, Eiveniu 4, Kaunas, Lietuva
[2]Institute of Neurosciences, Lithuanian University of Health Science, Eiveniu 4, Kaunas, Lietuva
E-mail: e.rekuviene@gmail.com

Mitochondrial damage, particularly opening of mitochondrial permeability transition pore (mPTP), is thought to be critical in ischemic insults. The opening of mPTP can induce mitochondrial depolarization and inhibition of ATP synthesis leading to cell death. Mitochondria isolated from various brain regions were shown to have different sensitivity to mPTP and this may account for different vulnerability of brain cells to ischemic stress. Therefore, inhibition of mPTP by pharmacological compounds may be efficient strategy to improve brain tissue functions in pathological conditions. Our recent results have shown that inhibitors of respiratory chain complex I are involved in regulation of Ca^{2+}–induced mPTP of isolated rat brain mitochondria. In this study we aimed to investigate whether the infusion of respiratory chain complex I inhibitor rotenone (Rot) to *vena cava* is protective on mPTP opening, mitochondrial respiration, generation of reactive oxygen species (ROS) in rat brain cortex and cerebellum mitochondria and necrosis level during 120 min. brain ischemia.

We obtained that brain ischemia significantly decreased resistance of cortex and cerebellum mitochondria to Ca^{2+}-induced mPTP opening, activated ROS generation, affected respiration functions with substrates pyruvate plus malate and succinate and increased the activity of lactate dehydrogenase (LDH) in brain medium. Rot infusion before ischemia re-established resistance to Ca^{2+}-induced mPTP opening of both, cortex and cerebellum mitochondria to control level. In addition, Rot significantly decreased ROS production in cortical mitochondria and protected state 3 respiration with succinate of cerebellum mitochondria. However, Rot infusion did not changed LDH activity as necrosis marker in medium of both brain regions. In conclusion, pre-treatment with rotenone before ischemia significantly reduced brain mitochondria injury, providing neuroprotective benefit of complex I inhibitors against ischemia caused damage.

NOTES

ISBN 978-609-459-605-6

7th Conference
of Lithuanian Neuroscience Association

Program and Abstracts

1,1 aut. l., 2 sp. l. Tiražas 130 egz.
Išleido Vilniaus universitetas,
Vilniaus universiteto leidykla
Universiteto g. 3, LT-01513 Vilnius

Spausdino UAB „BMK LEIDYKLA“
J. Jasinskio g. 16, LT-01112 Vilnius