6th Conference of Lithuanian Neuroscience Association

PROGRAM AND ABSTRACTS

Organizer

Lithuanian Neuroscience Association

Sponsors

Organizing committee:
Prof. Osvaldas Rukšėnas, Prof. Aidas Alaburda, Dr. Rima Naginienė,
Dr. Inga Griškova-Bulanova, Dr. Vilma Kisnierienė, Dr. Ramunė Grikšienė

5 December, 2014
Hotel Panorama, Sodų str. 14, Vilnius

PROGRAM

9.00 – 9.50 **Registration.**

9.50 – 10.00 **Opening. President of LNA Osvaldas Rukšėnas.**

10.00 – 10.40 **Plenary lecture. Linas Bieliauskas.** Changes in visuospatial processing with aging. *Neuropsychology Section, University of Michigan, Michigan, USA.*

I session (Chair – A. Bulatov)

10.40 – 11.10 **Aurelija Jučaitė**. Neurochemical – neurovisual investigation: news and clinical application. *Karolinska Institute, Stokholm, Sweden*

11.10 – 11.40 **Valentina Vengelienė.** Alcoholism: cannot be cured but can it be treated? *Institute of Psychopharmacology, Central Institute of Mental Health, University of Heidelberg, Mannheim, Germany; Vilnius University, Vilnius, Lithuania.*

11.40 – 12.00 **Coffee/Tea.**

12.00 – 12.20 **Inga Griškova-Bulanova.** Electrophysiological markers of neuropsychiatric diseases. *Vilnius University, Vilnius, Lithuania.*

12.20 – 12.40 **Jolita Janušauskaitė.** Preclinical diagnostics of Parkinson's disease. *Lithuanian University of Health Sciences, Kaunas, Lithuania.*

12.40 – 13.00 **Agnė Mikailionytė.** The role of video game play in enhancing visual function in healthy individuals and those who have visual impairments. *Department of Life Science, Kingston University, London, UK.*

13.00 – 14.00 **Lunch. Coffee/Tea.**

14.00 – 14.30 **Annual meeting of LNA.** ***(for LNA members).***

II session (Chair – A. Alaburda)

14.30 – 14.50 **Aušra Saudargienė, Marja-Leena Linne.** The Human Brain Project: advancing future neuroscience, medicine and computing. *Vytautas Magnus University, Kaunas, Lithuania; Tampere University of Technology, Tampere, Finland.*

14.50 – 15.20 **Bruce Graham.** Computational modeling of hippocampal circuits in health and disease. *University of Stirling, Scotland.*

15.20 – 15.50 **Gytis Baranauskas.** Can Brain-Computer interface improve the lives of disabled people already today? *Department of Physiology and Cell Biology, Faculty of Health Sciences and Zlotowski Center for Neuroscience, Ben-Gurion University, Beer-Sheva, Israel; Laboratory of Neurphysiology, Neuroscience Institute, Lithuanian University of Health Science, Kaunas, Lithuania*.

15.50 – 16.20 **Adomas Bunevičius** Behavioral Neurosurgery: From Patient to Data. *Lithuanian University of Health Sciences, Kaunas, Lithuania.*

16.20 – 19.00 **Poster session. Cheese & wine.**

18.00 **Poster Awards.**

ORAL PRESENTATIONS

CHANGES IN VISUOSPATIAL PROCESSING WITH AGING

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Visuospatial abilities are particularly sensitive to age-related decline, although the neural basis for this decline (and its everyday behavioral correlates) is as yet poorly understood. We used fMRI to examine age-related differences in patterns of functional activation that may underlie changes in visuospatial processing associated. All participants completed a brief neuropsychological battery and also a figure ground task (FGT) assessing visuospatial processing while in the fMRI scanner. Participants included sixteen healthy older adults (OA; aged 69-82 years) and 16 healthy younger adults (YA; aged 20-35 years). We examined age-related differences in behavioral performance on the FGT in relation to patterns of fMRI activation. OA demonstrated reduced performance on the FGT task and showed increased activation of supramarginal parietal cortex as well as increased activation of frontal and temporal regions compared to their younger counterparts. Decreased performance on the FGT was related to increased supramarginal gyrus activity and increased medial prefrontal activity in OAs, but not YAs. These frontal and parietal activations were intercorrelated in the YA but not OA, suggesting an age-related pattern of discordance in functional activations in these areas. Our results are consistent with an anterior-posterior compensation model in that frontal activity likely reflects compensatory activation for decreased posterior efficiency in OA. Furthermore, successful FGT performance requires the perception and integration of multiple, piecemeal stimuli and thus it is plausible that healthy aging may be accompanied by changes in visuospatial processing that mimic a more subtle form of dorsal simultanagnosia. Ultimately, decreased visuospatial processing in OA was found to have a frontoparietal neurobiological signature that may be a contributor to the general phenomenon of increased piece-meal execution of behavior associated with normal aging. OA's poorer ability to perform tasks that tap neural processes implicated in simultanagnosia may be an early marker useful for distinguishing healthy aging from unhealthy aging.

MOLECULAR IMAGING: SCIENCE AND CLINICAL APPLICATIONS

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Brain imaging using positron emission tomography (PET) dates back to 1878. The last 60 years have produced an evolution and revolution of technological advancements in brain metabolism and radioligand development. These advancements facilitated development of modern cerebral molecular imaging, with increasing number of receptors, enzymes and larger protein aggregates being visualized and available for quantitative analysis. PET application in the diagnosis of neurological disorders is increasing pace. Imaging of dopamine transporter is becoming routine clinical practice for patients with Parkinsonian syndromes, imaging of GABA receptors and brain metabolism is critical in the management of epilepsy. Advances in the radiolabelling of beta-amyloid peptide has shown the time course of amyloid deposition in dementia and Alzheimer‘s disease. Further developments of such biomarkers as Tau protein and alfa-synuclein are in development and will open new possibilities for patient management as well as development of novel therapies. PET is also increasingly applied in drug industry for science driven drug development, as a part of early clinical studies in understanding mechanism of action of drug, basis for dose choice or as a biomarker for patient diagnosis and therapy monitoring. PET continues to be at the frontiers of the scientific studies aimed to understand cognitive brain functions, pathophysiology of neurological and psychiatric disorders. Recent research in Attention deficit hyperactivity disorder (ADHD) will be presented.

ALCOHOLISM: CANNOT BE CURED BUT CAN IT BE TREATED?

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Continuous alcohol use affects a wide range of brain neurotransmitter/neuropeptide systems, such as glutamatergic, GABAergic, dopaminergic, serotonergic, opioidergic, cholinergic, glycinergic, cannabinoidergic, as well as corticotropin-releasing factor and neuropeptide Y systems. Thus, following long-term, chronic alcohol consumption virtually all brain neurotransmission seems to be affected. At least some of those changes are causing the transition between controlled and compulsive alcohol use, however it is difficult to determine which of the systems contribute the most to the development of alcohol addiction, as it is likely to be caused by the interplay between different brain systems.

Relapse to alcohol use is known to occur in the majority of clinical cases if no appropriate treatment is provided, indicating that it is a fundamental part of this disease and should be considered as one of the central features of an addicted behavior. In animals that had long-term voluntary access to alcohol and became withdrawn from it for several days/weeks re-presentation of alcohol leads to robust but temporal increase in alcohol intake over baseline drinking. This robust phenomenon is called the alcohol deprivation effect (ADE) and is observed across several species, including rats, mice and monkeys. The mechanisms underlying the temporary increase in alcohol consumption following periods of abstinence are not clear, although it seems to be a rather complex behavioral phenomenon. The rat ADE model has been used in the last decade to map the neurochemical substrates underlying relapse-like drinking behavior. Data of several studies performed in our institute demonstrated that the levels of alcohol consumed during ADE can be modified by manipulating glutamatergic, dopaminergic and glycinergic systems suggesting new promising targets for the pharmacological intervention of alcohol-related problems.

ELECTROPHYSIOLOGICAL BIOMARKERS OF NEUROPSYCHIATRIC DISEASES

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A *biomarker* is defined as "a *characteristics* that is objectively measured and evaluated as an *indicator* of normal biological or pathogenic processes, or pharmacologic responses to a therapeutic intervention". In this way biomarkers can provide an objective basis for diagnosis, treatment selection, and outcome measures in neuropsychiatric disorders. Electrophysiological biomarkers, i.e. obtained through recording and interpretation of electrophysiological signal from the body, are particularly promising, as they are non-invasive and relatively inexpensive. When considering neuropsychiatric disorders, main attention is drawn to the brain-derived electrical responses – electroencephalogram and evoked/event-related potentials and/or oscillations. In this talk I will illustrate the properties of currently investigated electrophysiological biomarkers for neuropsychiatric disorders and discuss strengths and weaknesses of this approach.

PRECLINICAL DIAGNOSTICS OF PARKINSON'S DISEASE

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It is well known that Parkinson's disease (PD) - specific pathological features antedate the onset of classical clinical features by many years. At the time of clinical diagnosis no possible pathogenetic treatment would be effective in significantly modifying the disease progression. In light of current research aimed at development of efficient neuroprotective therapy, it is essential to identify „healthy" subjects with higher risk of developing neurodegenerative diseases such as Parkinson's disease.

As PD pathology is rather diffuse than restricted to substantia nigra, a brainstem region which is well known to be affected in case of PD, ongoing research is attempting to indentify set of markers which would reflect broad spectrum of disease pathology and together serve as a preclinical diagnostic tool for PD. To date, the most promising markers seem to be REM sleep behavior disorder, smell impairments and changes in substantia nigra echogeneicity as observed on transcranial sonography. In this presentation possible targets for preclinical PD diagnostics will be discussed.

COMPARING DIFFERENT STYLES OF VIDEO GAME PLAY AS SUITABLE VISUAL TRAINING TASKS OVER A 120 HOUR TRAINING PERIOD

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Video gaming has been shown to improve visual function in individuals with no visual problems, in amblyopia, and developmental dyslexia. This pilot study investigates whether longer training periods improve function. Four participants, with an average age of 28, played either an action or a turn – based strategy (TBS) game for 120 hours over a three month period. The games selected were Doom 3 and Civilization 4. Visual function was assessed before and after training by a series of psychophysical experiments (spatial contrast sensitivity (CS), temporal and peripheral CS for 20 Hz and 24 Hz, and motion detection and discrimination) programmed on MATLAB using Psychophysics Toolbox extensions. The spatial frequencies tested were 0.5, 2, 10, and 20 cycles/degree (cpd).

Game training indicated a significant improvement in both temporal CS and peripheral CS, both at 24 Hz ($p = <.05$). Action gamers had a higher CS at the peripheral CS of 24 Hz in 2 cpd compared to TBS gamers ($p = <0.05$). Additionally, in the peripheral CS of 20 Hz, action gamers had a higher CS at 0.5 cpd ($p = <0.5$). All of the CS values were higher after the training for both game genres, aside from central CS which is expected. The present study is the first of its kind examining training over 120 hours using two different game genres. Additionally, there are indications that TBS games provide a similar level of visual improvement as in action games. This is particularly important as non – action and alternative games are popular (an estimated 200 million worldwide players) and studies into the genre are limited in their amount.

THE HUMAN BRAIN PROJECT: ADVANCING FUTURE NEUROSCIENCE, MEDICINE AND COMPUTING

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The Human Brain Project (HBP) is a European Commission Future and Emerging Technologies (FET) Flagship that aims to achieve a multi-level, integrated understanding of brain structure and function with the help of novel information and communication technology. Information and communication technologies (ICT) for neurosciences, developed by HBP, are expected to enable large-scale collaboration and data sharing, reconstruction of the brain at different biological scales, federated analysis of clinical data to map diseases of the brain, and the development of brain-inspired computing systems.

HBP research is organised into twelve Subprojects, each divided into Work Packages and Tasks, with well-defined goals and milestones. Six Subprojects are building the so called ICT Platforms, while the other six are gathering data, clarifying theory and controlling ethical aspects. The results of HBP are expected to accelerate progress towards effective diagnosis and treatment of brain diseases and the development of novel robotics and information technology that mimics the human brain. The HBP is led by Professor of Neuroscience Henry Markram of the Swiss Federal Institute of Technology and co-directed by Professor of Physics Karlheinz Meier from Germany. In this presentation we will describe the goals of the HBP and discuss its potential impact on the neuroscience and society in general.

COMPUTATIONAL MODELLING OF HIPPOCAMPAL CIRCUITS IN HEALTH AND DISEASE

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A subfield of Neuroinformatics, Computational Neuroscience uses mathematical modelling, analysis and computer simulation to study form and function in the nervous system. Modelling has become a vital tool in cellular biological research, particularly for the endeavour of trying to understand the operation of networks of neurons, in both healthy conditions and in different disease states. Such computational modelling is used to untangle the myriad intracellular signalling pathways, particularly those underlying plasticity, the electrical properties of neurons and how neural networks may learn and process information. A particular difficulty for clinical and experimental neuroscience is to relate pathological changes in brain diseases to behavioural symptoms and cognitive deficits. A computational model of a healthy brain network allows precise intervention to mimic the pathology of a disease and to then read out the impact on network activity and even information processing underpinning cognition. In this talk I will illustrate this by describing models of the mammalian hippocampus that achieve declarative memory formation and recall. Various pathologies of Alzheimer's disease have been applied to such models to investigate their impact on short-term memory formation, which is a major symptom of the disease. A significant result from these modelling studies is to demonstrate the robustness of the hippocampal neural networks, so that evidence of memory decline only emerges after significant damage to the networks, corresponding to advanced stages of disease.

CAN BRAIN-COMPUTER INTERFACE IMPROVE THE LIVES OF DISABLED PEOPLE ALREADY TODAY?

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Brain-Computer interfaces provide a direct communication pathway between human brain and a device; thus these interfaces could help to control various devices such as computers or wheelchairs for disabled people, who lost their ability to move any limbs. However, so far their use was limited to laboratory tests. This lecture will demonstrate that the main problem is limited information transfer rates enabled by Brain-Computer interfaces of all types. Two types of Brain-Computer interfaces exist, non-invasive, mostly EEG-based and invasive ones with electrodes, inserted into the brain of a patient. While in EEG-based interfaces the nature of the signal seems to be the limiting factor for information transfer rates, it is less clear what limits these rates in invasive devices, in which multiple electrodes provide very rich neural data. I will argue that our limited knowledge about parameters that are encoded in these signals is the main reason for limited information transfer rates in invasive Brain-Computer interfaces.

BEHAVIORAL NEUROSURGERY: FROM PATIENT TO DATA

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Depression is a common complication in patients with established brain tumour (BT) diagnosis affecting approximately up to 50% of patients. Depression carries numerous adverse health effects in BT patients that include shorter survival, worse health-related quality of life and cognitive impairment. However, depression often remains under-diagnosed and untreated in BT patients mainly due to numerous patient-related, physician-related and system-related barriers, such as time-constraints and paucity of evidence-based depression screening algorithms. Towards this end, routine use of depressive symptoms self-rating scales for depression screening is expected to improve identification of patients suffering from mental disorders. The Hospital Anxiety and Depression Scale – Depression subscale has demonstrated superior diagnostic properties for major depressive screening purposes in BT patients, followed by Patient Health Questionnaire-9 and Distress Thermometer. However, positive predictive value of depression screening is low indicating that a significant proportion of BT patients with positive screening results will not meet the diagnostic criteria of depressive disorder. Consequentially, depression treatment should not be initiated based on the screening results and all high-risk patients should be referred for psychiatric assessment. Further studies investigating diagnostic accuracy of depression screening in BT patients are strongly encouraged in order to define optimal depression screening algorithm. Integration of biomarkers can contribute towards more accurate identification of distressed BT patients and help guiding treatment. Finally, it is important to learn if depression screening intervention can improve functional and patient-oriented outcomes and what is the optimal management strategy of depressed BT patients.

There is a rising worldwide movement encouraging sharing of scholarly research data with investigators and other interested parties. Data sharing has well-acknowledged advantages to society and research community, such as accelerated research progress and optimized use of available research funding. Advantages for researchers include potential to improve citation metrics, research/publication quality and visibility/collaboration opportunities. Important hurdles that prevent from more universal implementation of data sharing culture include but are not limited to ethical issues (for studies in human subjects), lack of universally implemented data sharing guidelines, risks for data mis-use and limited number of data sharing outlets. Towards this end, there is a growing number of specialized database journals that publish papers describing datasets providing authors with needed credits.

POSTERS

P1. IMITATIVE AND COMPLEMENTARY RESPONSE PREPARATION IN SCHIZOPHRENIA

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Different processes associated with future prediction such as foresight, planning and anticipation are impaired in schizophrenia. We hypothesize that schizophrenia can influence another brain function associated with future prediction processes – response preparation.

Response preparation is usually explored using two types of stimuli. Precue stimulus provides advanced information about a subsequent imperative stimulus which requires the response. The type of information we used in our study was the probability of imperative stimuli. In natural life, a relationship between a signaling event and upcoming event is probabilistic rather than deterministic, so using such type of information has an advantage of making experiment closer to real environment conditions.

The higher probability of imperative stimulus, the faster is response to it. This response time reduction shows that subjects use probabilistic information to make a higher level preparation of response to the higher probability imperative stimulus.

Previous studies showed that preparation of neutral responses (which have no common features with stimuli) is not impaired in schizophrenia patients. There are also evidences, that schizophrenia patients have tendency of inappropriate copying of behavior of other people that causes problems in their social life.

The purpose of our study was to investigate preparation of imitative responses (copying action showed in stimulus) and complementary responses (making different action, than stimulus shows). We investigated 15 schizophrenia patients and 19 participants of control group. Subjects had to make preparation using probabilistic information of two imperative stimuli of 75% and 25%. Our results showed that despite both imitative and complementary responses being slower in schizophrenia patients than in control group, schizophrenia patients are able to use probabilistic pre-cueing information and make adequate preparation of imitative and complementary responses.

P2. CATEGORIZATION MODE OF SIMULTANEOUSLY PRESENTED VISUAL OBJECTS

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We investigated the manner of perception and categorization of several visual objects presented simultaneously with the aim to answer if the process is parallel or successive. Our previous experiments showed that categorization duration and accuracy depended on the number of categories but not on the number of objects in visual stimulus. Such results could suggest simultaneous processing of category features of multiple objects presented in the visual field.

Our present psychophysical experiment was designed to answer whether global or local object features have greater impact on parallel categorization. Twenty five participants took part in the investigation. Ten categories of artificial items pictures were used as stimuli. Pictures of objects were presented either intact or distorted globally or locally. A visual stimulus containing pictures of one, two or three objects was presented on a computer display for 200 ms and then followed by a probe-word. Subjects were asked to respond whether they saw the object defined by the probe-word. Altogether, 480 stimuli were presented for each participant.

The results of the experiment repeated our previous findings: categorization duration and error rate directly depend on the number of categories but not on the number of objects in visual stimulus. This tendency was neither influenced by global nor local picture distortions.

P3. EFFECT OF GENDER AND COGNITIVE LOAD ON THE NEURAL CORRELATES OF MENTAL ROTATION

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Mental rotation abilities consistently present gender differences which emerge particularly with growing task difficulty. One of possible explanations of this phenomenon is unequal efficiency of brain resource usage, however mechanisms responsible for gender differences in neural efficiency during visuo-spatial task performance are not well determined. The aim of presented study was to investigate cognitive load dependent changes in male and female brain activity with increasing mental rotation complexity.

In this study 21 males and 32 females were tested using modified mental rotation paradigm. The selected paradigm is known to be sensitive to factors related with subject's capabilities (e.g. intelligence, talent or expertise) and allows the investigation of mental load integration during the task performance as well as comparison between active cognitive processes (rotation) and retention of visual image in short-term memory. In order to assess efficiency of brain resource usage with high temporal resolution, multichannel electroencephalography (EEG) method was used.

Behavioral results confirm the initial hypothesis of gender specificity: in comparison to females, males are significantly faster (1013 ms versus 1280 ms) and more accurate (84 % versus 75 %). Results obtained applying global field power (GFP), topographical and microstate analysis methods on EEG data, highlight statistically significant influence of active cognitive processes (rotation versus retention), gender and task complexity. Higher global field power values obtained during active rotation as compared to visual retention may be associated with quantitatively higher level of brain activity. Furthermore, differences in GFP between genders point towards relatively more resource-intensive mental rotation processes in female group in comparison to males. Meanwhile, disparities emerging in activation topographies during the growth of cognitive load reflect qualitative neural differences and suggest that distinct stages of the task recruited at least partly separate brain areas.

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P4. SEX DIFFERENCES IN MENTAL ROTATION OF SEQUENTIALLY PRESENTED 3D FIGURES

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Sex differences in rotation of a visual object in mind are associated with dissimilarities in brain activity during the task. Event related potentials (ERP) allow to assess the neural processing underlying mental rotation with high temporal resolution. Shepard and Metzler (S/M) paradigm is one of the most commonly used in studies aimed to test visuospatial abilities. However, combining ERP method with original S/M paradigm, where two figures are presented in parallel, highlights the problem related to large effect of saccades on EEG signal in each trial. The aim of the present study was to evaluate sex and sex steroids effects on brain activity while subjects perform modified S/M task in which figures are presented sequentially.

High density EEG recording was used to assess changes of brain activity during the task. Accuracy and response time were measured to evaluate performance quality. Only data of subjects (15 men, 19 women) who demonstrated significant increase of reaction time with increasing angular disparity of paired 3D figures were used for behavioral and ERP analysis.

Men in comparison with woman were more accurate (84.0 % ± 3.23SE vs 77.6 % ± 2.63SE), but slightly slower (1267 ms ± 50SE vs 1232 ms ± 54SE). Sex steroids analysis revealed significant negative 17ß-estradiol effect on women accuracy ($r=-0.34$, $p=0.046$) and significant increase of women response time with increasing progesterone level ($r=0.34$, $p=0.042$). However, testosterone has no effect on men or women performance.

Global field power (GFP), topographical ANOVA (TANOVA) and ERP wave analysis methods were applied on ERP data. In agreement with previously demonstrated mental rotation effects on ERP's the positivity of slow late ERP wave (peak amplitude located over parietal scalp, latency - about 400 ms after onset of stimulus) decreased with increasing angular disparity between figures. Significantly higher GFP values in woman as compared with men 330 – 1000 ms after onset of the stimulus may indicate quantitatively higher level of women brain activity during the rotation. However, significant topographic differences (TANOVA analysis) between groups during the similar time period (330 – 670 ms after onset of the stimulus) allow estimation of differently active sources on the scalp of men and women during rotation.

P5. GENDER DIFFERENCES IN SENSORY GATING

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Sensory gating is a concept describing how well brain is able to filter stimulus and switch involuntary attention. Abnormal sensory gating is a candidate of being a biomarker for psychiatric disorders such as schizophrenia, also it has been investigated in attention-deficit hyperactivity disorder, traumatic brain injury, migraine, autism, as well as that it is a valuable tool to study normative psychological conditions.

Sensory gating could be evaluated in two ways:

(a) objectively, or by neurophysiological method; it involves the use of early event-related potentials: P50 (P1) and mismatch negativity (MMN);

(b) subjectively, or by phenomenological way by which a self-reported Sensory Gating Inventory (SGI) is being evaluated; SGI is made of 36 items of perceptual anomalies that are divided into 4 dimensions: Perceptual Modulation (PM), Over-inclusion (OI), Distractibility (D) and Fatigue-Stress Modulation (FS).

This poster presents a literature review regarding gender differences in neurophysiological and phenomenological sensory gating. We performed a search in PubMed database for auditory P50, MMN and gender differences and selected original studies using non-verbal and non-emotional stimuli. In addition, SGI studies were also reviewed. All papers included healthy adult samples. The final number of articles meeting the criteria was as follows: 4 articles for SGI, 5 for MMN and 13 for P50.

As a result, some studies revealed poorer sensory gating performance in women: lower P50 suppression, also higher scores in D and FS dimensions of SGI. On the other hand, other studies found no gender differences in early ERPs or in SGI scores. Summing up, the amount of studies, considering gender as a factor is limited and it is not possible to confirm or discard gender effects. Wider scale studies should be conducted taking into account gender as a factor.

P6. PASSENGER FERRY SEAFARERS 24-HOUR HEART RATE STUDY

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Introduction: Safety of sea transportation significantly depends on human activities, in particular crew team must consist of qualified and healthy professionals. Work-related fatigue may cause a mental or physical health problems resulting in increased accident and injury risk. Relatively little is known about fatigue among seafarers, especially during short-distance trip. One of the ways to explore the physical condition of the sailors is a persistent heart rate recording during the trip.

The aim of this study: Was to investigate the sailor's heart rate variation during short trip.

Methods: Recordings were made from 8 healthy sailors, men aged 32 ± 4 years. The heart rate was recorded with „eMotionLAB" system during short-distance trip. Subjects filled activity reports, which represent the three states - work, rest and sleep. The obtained data was reviewed and the most suitable episodes, lasting not less than 24 hours, were selected for analysis. Discretization of RR intervals was 0.5 s. RR interval filtering, separately distinguishing the high-frequency components within range from 0.15 Hz to 0.4 Hz was used. We also conducted an analysis of the histogram to check the distribution of rhythm during 24 hours.

Results and Conclusions: Subjects were divided into three groups. For the first group heart rate (HR) dynamics were corresponding to the normal rhythm of life. HR slowed down during resting periods and slowed down even more during the sleep. Heart rate variability (HRV) was similar in all three states. The second group of subjects had converse HR behavior, the conclusion was that some test subjects couldn't relax and because of that HR didn't changed during sleep or rest states. The third group of subjects during the rest state was engaged in physical activity (sports) so HR increased, but no meaningful reduction during the sleep time was detected. We argue that a longer recording time is required to make the precise conclusions.

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P7. SPATIAL AND TEMPORAL ASPECTS OF THE OPPEL-KUNDT PHENOMENON

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The visual overestimation of the length of the filled extent is under consideration in the present communication: a space interval filled with uniform and evenly distributed elements (stripes or spots) is perceptually longer than unfilled interval of the same physical length. Since the German physicist J.J. Oppel reported the phenomenon in 1855, there was not decisive progress in theoretical explanations of the illusion, nevertheless, the spatial-temporal factors determining the illusion magnitude have been intensely studied and positive regularities in the illusion manifestation have been established. The longer stimuli induce the stronger illusion. The illusion magnitude increases with the increasing number of filling elements but reaches a maximum and slightly decreases afterwards with further growth of density. The maximum illusion strength is present with 7 to13 fillers, and the higher numbers of elements or just a solid bar become less effective illusory patterns. The illusion magnitude increases in a monotonous manner with an increase of the stimulus exposure duration within approximately the 100−1000 ms interval. Unequal spacing and non-uniformity of the filling elements in all respects (height, thickness, tilting, curvature, luminance, and color) provide the lower illusion values. Removal of filling elements from any position of the regular sequence reduces the illusion magnitude as well. Movement of the sequence within the filled window weakens the illusory effect also. The difference between two stimulus parts in brightness contrast, again, generates a less prominent illusion.

The converging evidence from the data collected in various studies leads to an assumption that an overestimation of the filled extent in the Oppel-Kundt stimulus can be associated with the spatial-temporal integration along the continuous path of excitations evoked either by the real contour of a solid bar or by the illusory contour of a regular sequence of the filling elements.

P8. MÜLLER-LYER ILLUSION MANIFESTATION FOR PATIENTS WITH PARANOID SCHIZOPHRENIA

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Aim of the study was to assess drugs' effect on Müller-Lyer illusion manifestation for people with paranoid schizophrenia (PS).

Methodology: PS group were patients hospitalized at Hospital of Lithuanian University of Health Sciences Kaunas Clinics Psychiatry Clinic. Control group was gathered from Dermatological and Venereal Disease Clinic. Study protocol was approved by LUHS bioethics centre. The stimuli consisted of white Müller-Lyer figures. Inner angles of figures changed from 10^{o} to 350^{o}. 40 figures were presented during one experiment and each patient carried out 10 experiments. Subject's aim was to place the central element in the exact middle between outer figures. We measured distance (arc min) between subjects' chosen position and geometrical center of figure. Antipsychotic drug doses were converted to chlorpromazine equivalents and benzodiazepines were converted to diazepam equivalents. Drugs' pharmacological properties were evaluated on each experiment day individually. ANOVA and T-test were used for statistical analysis.

Results: Illusion had tendency to manifest stronger for PS group, when evaluating more acute ($< 79^{o}$) angles. When the figure's inner angles approached 180^{o}, manifestation of the illusion diminished. Results for subject group demonstrated higher standard errors of means (0.93 ± 0.02 vs. 0.79 ± 0.03) with significant difference ($p < 0.01$). Illusion manifested stronger when patients received higher than 300 mg of chlorpromazine equivalent dose of antipsychotics. Illusion manifestation was stronger for patients who received higher doses of benzodiazepines (> 10 mg of diazepam equivalents per day) compared to patients who received lower doses, patients who received no benzodiazepines and control group ($p < 0,05$).

Conclusion: Müller-Lyer illusion tended to manifest stronger for patients with schizophrenia and for those who received higher doses of antipsychotics and benzodiazepines when evaluating more acute and obtuse corners.

P9. CLOCK TASK – EFFECT OF DIFFICULTY LEVEL: TIMING, HEMISPHERIC SPECIALIZATION AND REACTION TIME

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Background: Different Clock tasks are commonly used to explore the visuospatial processing. Effects of these tasks on visuospatial processing have been evaluated using fMRI only. However, understanding the timing of a processing is also important. This study aimed to establish clock task paradigm and evaluate task difficulty effects using ERP technique.

Methods: 8 healthy participants performed Clock task. Participants were instructed to press one of the two buttons whenever a target or non-target stimulus appeared. EEG was recorded from 70 channels. Eye-movements were monitored with two additional EOG channels placed below the left and the right eye.

Clock task paradigm: Stimuli were schematic analog clocks with five angular disparities: 30º, 45º, 60º, 75º, and 90º and three different lengths of the hands: long, medium, and short. A target was an angle of 60º. Difficulty of the task was manipulated by the length of the hands, resulting in 3 difficulty levels.

Spatially defined transiently stable states of the ERP (microstates) associated with brain activation were identified and t-maps were quantified. Waveshapes of ERPs were created for each electrode.

Results: Behavioral results showed that the mean reaction time increased with an increasing difficulty level in each angle condition.

Waveshapes analysis revealed stronger right hemisphere activation over parietal electrode sites in both targets and non-targets. This is in line with the parietal cortex spatial functions and the right hemisphere dominance in a visuospatial processing. The P1, the N1, and the P300 components were observed in all difficulty levels for both targets and non-targets.

In microstate analysis, significant differences were observed between all 3 difficulty levels from 200 to 500 ms. T-maps analysis showed topographically different maps for different levels of difficulty in this time window.

Conclusion: Difficulty level affects reaction times and ERPs in 200-500 ms time window.

P10. POSSIBILITIES OF DETAILED ANALYSIS OF VISUAL EVOKED POTENTIALS' SHAPE IN NORM AND OPTIC NEURITIS CASES

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Registration of visually evoked potentials (VEPs) as responses to eye stimulations with an alternating checkerboard pattern is a neurophysiological test that provides diagnostic information about conductivity in visual pathways in the central nervous system, when neural impulses are transmitted from the retina through the optic nerve to the visual cortex, where thousands or millions of similarly oriented cortical pyramidal neurons fire in synchrony while processing information. VEPs may provide good prognostic information particularly when evaluating optic neuritis and other demyelinating diseases. Three of the evoked response peaks (N75, P100, N145) are often used in clinical practice.

Patients with optic neuritis have quite extended VEP latency for several years even if their vision becomes normalized; patients with pronounced myelitis have reduced VEP amplitude and normal latency; patients due to other conditions that affect the optic nerve, e.g., tumour, have delayed and/or reduced VEP amplitudes.

In general, evaluation of VEPs is difficult because they are embedded in a background spontaneous electroencephalographic activity (EEG) with much larger amplitude. Majority of techniques applied for evoked potentials analysis are based on signal averaging with the aim to cancel background EEG. Our idea is to test the possibilities of advanced multivariate analysis techniques for detailed VEP shape evaluation in every single sweep. We expect that this detailed information will reveal new diagnostic possibilities.

We analyzed VEP waveforms registered from 10 optic neuritis patients and 10 healthy persons. Preliminary results showed possibility for qualitative evaluation of VEP shape dynamics related to habituation, latency jittering or other effects. Qualitative estimates could be used as candidates for diagnostic features in future investigations.

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P11. INFLUENCE OF SLEEP AND RELATIVE REWARD SIZE ON MEMORY CONSOLIDATION: AN fMRI STUDY

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Introduction: Memory is the brain's capability of saving and retrieving information. Concurrent activation of the brain dopaminergic system (e.g. presentation of a reward) has been shown to improve the recall of a memory item. Furthermore, reward presentation context influences the extent of activation elicited in brain dopaminergic system (effect known as "adaptive scaling") thus affecting memory encoding. Little is known about the interaction of reward-related effects with other memory consolidation promoting factors, such as sleep.

We hypothesize that the relative reward size elicits larger activity in brain dopaminergic system, detectable via functional magnetic resonance imaging (fMRI), which in turn promotes memory retrieval. We also hypothesize that sleep enhances the reward-related effects on memory consolidation.

Materials and methods: The fMRI data were acquired using a 3T Philips Achieva scanner. Data were analyzed using Statistical Parametric Mapping (SPM8). 39 healthy participants were included in the final analysis (mean age 24.95 ± 2.86; 20 female).

The experimental design consisted of two scanning sessions 38 hours apart, with a sleep/no-sleep treatment in between. A single trial encoding session consisted of a word stimulus, associated with one of three possible rewards (low/medium/high) presented in one of two possible contexts (low/medium or medium/high). In the retrieval session, participants had to discriminate words presented in the encoding session from new distractor words.

Results and conclusions: In the encoding session, relatively higher rewards (i.e. medium reward in a low/medium reward context) elicited significantly higher activity in hippocampal and parahippocampal areas than relatively lower rewards. This is in line with the "adaptive scaling" model. Yet, our findings from the retrieval session suggest that recall of memory items is promoted by a higher expected reward value (i.e. medium reward in a medium/high reward context).

P12. SYNCHRONIZING AND DESYNCHRONIZING VISUAL BRAIN REGIONS USING TACS MODULATES VISUAL PERCEPTION

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It is becoming increasingly clear that brain oscillatory activity prior to the presentation of a stimulus plays an important role for its subsequent processing. This relationship between oscillations and perception is underlined by several behavioural and electrophysiological studies demonstrating that perception fluctuates rhythmically at a frequency of around 5-10 Hz. In a previous EEG-fMRI study we investigated the impact of prestimulus oscillations on the detection of briefly presented contour stimuli and found that a 7 Hz oscillation impacted on visual perception via rhythmically modulating the information flow between a left occipital and right parietal area. However, these results are based on correlating brain oscillatory activity with perception and we therefore do not know whether this relationship is going beyond correlation, i.e. is of a causal nature. Here we addressed this issue by applying transcranial electric stimulation (tACS), which allows controlling externally the phase of cortical oscillations by applying weak electrical current. To this end we separately modulated the prestimulus phase in the left occipital (lOcc) and the right parietal cortex (rPC) such that they were either synchronized (0 deg. phase shift) or desynchronized (180 deg. phase shift). Additionally, three control conditions were carried out where stimulation was applied (i) only for a very short period of time (sham), (ii) only at lOcc, or (iii) only at rPC. We hypothesized that synchronizing lOcc and rPC increases perception performance whereas desynchronizing lOcc and rPC decreases perception performance compared to sham stimulation. Our results indeed showed higher perception performance for the synchronized compared to the sham stimulation condition ($p<0.01$). Furthermore, perception performance was modulated by the phase of stimulation. Together, these results support and go beyond our previous finding in suggesting that brain oscillations causally impact on visual perception by dynamically regulating the flow of information between visual processing regions.

P13. DOPAMINE AND WORKING MEMORY – FROM ELECTROPHYSIOLOGY TO NEUROIMAGING

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The prefrontal cortex, which is playing a critical role in planning complex cognitive behavior, decision making, control of emotions and social behavior, is a dynamic region which undergoes many plastic changes. Neuroplasticity can occur on many different levels, from cellular changes of synaptic efficacy and its cytoarchitecture, to large-scale changes in neural networks. Long-term depression and long-term potentiation are the two most studied forms of long-term plasticity. Dopamine (DA) neurons from ventral tegmental area, which widely innervate prefrontal cortex, can modulate functioning of pyramidal neurons and interneurons in cortex. Many physiological studies have shown strong modulatory effect of dopamine on long-term depression and long-term potentiation. However, these plastic changes depend strongly on the environment of cells and basal concentration of dopamine. Thus, through modulation of the pyramidal neurons, dopamine can change the state of the prefrontal network and affect many executive and cognition functions, especially working memory. Using computational model of prefrontal network, based on electrophysiological studies and exhibiting similar to in-vivo properties, we can modulate dopaminergic effects on Working Memory. It seems that DA can modulate focus, resistance to distractors and context updating.

P14. NEUROBIOLOGICAL CORRELATES OF INTELLIGENCE

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Perceived intelligence, as the ability to reason, plan, solve complex problems and think abstractly is a trait that undoubtedly distinguishes humans from other animals. The essence of human intelligence for centuries was the subject of interest to scientists and philosophers, but only relatively recent, we have appropriately advanced techniques, like fMRI or DTI, that have enabled us to more fully explore this topic and answer the question of what exactly constitutes a biological substrate of intelligence. Currently, the best factors correlating with the level of intelligence are considered to be the thickness of the cerebral cortex, the number of neurons and connections between them and the integrity of the white matter, which are responsible for the efficient processing of information and enable a rapid response. A good model of the anatomical structure of the brain showing the various levels of intelligence is associated with the fronto-parietal integration theory of intelligence, which has been proposed based on the analysis of several dozen different studies using neuroimaging techniques. The scientists are looking for the genes that affect intelligence. From among the potential candidates, four genes appear to be particularly interesting and are the genes encoding: ApoE4, CHRM2, cathepsin D, and COMT.

P15. INNERVATION OF RABBIT HEART VENTRICLES: AN IMMUNOHISTOCHEMICAL APPROACH

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Introduction: In some cases of myocardial infarction, afferent nerve fibres induce sympathetic hyperactivation, which causes the onset of ventricular arrhythmias or even sudden cardiac death. The present study analysed immunohistochemically the distribution of the intracardiac nerve plexus of the rabbit heart.

Materials and methods: Rabbit hearts (N = 10) were stained immunohistochemically for protein gene product 9.5 (PGP9.5), tyrosine hydroxylase (TH), choline acetyltransferase (ChAT), nitric oxide synthase (nNOS), calcitonin gene related peptide (CGRP) and substance P (SP). The area, density and distribution of positively stained neuronal structures within the epicardium, myocardium and endocardium were analysed.

Results: The largest nerves were found in the epicardium; a nerve fibre plexus was observed in the myocardium and the endocardium. In epicardial nerves, TH positive fibres were dominant (64%), while other phenotypes were less pronounced (5-7%). The heterogeneous nerve fibre plexus of the myocardium mainly contained TH positive fibres (1%), while nNOS fibres were more dense than ChAT fibres ($p < 0.05$). A negative correlation was found between the density of nerve fibres and the distance from the epicardium ($p < 0.01$). In the endocardium, the nerve fibre density of all fibre phenotypes was 10 times higher than in the myocardium ($p < 0.05$), with SP and CGRP positive fibres exceptionally pronounced (3% and 4%).

Conclusions:

- The largest nerves were found in the epicardium; the nerve plexus observed in the myocardium was scarce; in the endocardium, it was dense.
- TH positive fibres are dominant in the epicardium, myocardium and the endocardium.
- The cholinergic innervation in all parts of the ventricles was scarce, with nNOS positive fibres dominating over ChAT positive fibres in the myocardium.
- The endocardium contains a dense nerve fibre plexus with pronounced CGRP and SP positive fibres. The fibres were relatively abundant in comparis]on to the myocardium.

P16. ANIMAL MODELS OF TUBEROUS SCLEROSIS COMPLEX

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Tuberous sclerosis is an autosomal dominant disease caused by a mutation in Tsc1 or Tsc2 genes. The birth incidence of TSC is 1:6000. Disease manifestations are observed in number of organs: brain, heart, skin, liver. Severity varies among patients. There is a triad of TSC-related symptoms in the brain: lesions, epileptic seizures, intellectual disability. Three main types of lesions may be distinguished: cortical tubers, sub-ependymal nodules (SEN), sub-ependymal giant cells tumours (SCGT). They may develop during embryogenesis or form in childhood or adolescence. Tumours consist of heteromorphic cells, showing abnormal morphology as GC – giant cells and DN – dysplastic neurons. The phenotype is linked to molecular basis. Proteins encoded Tsc1 and Tsc2- hamartin and tuberin respectively - form a complex (TSC1/2) inhibiting the mammalian target of rapamycin (mTOR) which is involved in cell growth, proliferation and differentiation. Mutation in Tsc1 or Tsc2 resulting in impaired formation of the complex leads to hyperactivation of mTOR signaling pathway. Referring to Knudson hypothesis, "the second hit" (loss of the second intact allele) is required to develop certain pathological features associated with TSC. Molecular mechanisms and cellular phenotypes of TSC are studied using transgenic animal models and patients' samples. This review presents a wide range of possible models from Eker rats, conventional KO mices and conditional KO mices (considering different subsets of neurons, glial cells and neural progenitors) to human cells. Although they give some insights into mechanism of the disease, there are still many limitations. First of all, null mutations of Tsc genes are lethal and heterozygous mice do not fully recapitulate the features of TSC. Secondly, human cells obtained post mortem do not serve for understanding disease progression and development, which seems to be crucial for TSC.

P17. IMMUNOHISTOCHEMICAL ANALYSIS OF NEURONS LOCATED IN CARDIAC CONDUCTIVE SYSTEM

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Introduction: Heart rate is regulated by cardiac conductive system (CCS). Its work is regulated by the autonomic nerve system and in part by neurons residing closest to CCS. Therefore distribution and immunohistochemistry of neurons located in CCS were studied in the rabbit and pig hearts.

Aim of the study: 1) to determine location and immunohistochemistry of neurons within CCS of rabbit and pig hearts, 2) to compare neurons located in rabbit and pig CCS.

Materials and methods: The localization and innervation of CCS components – sinoatrial node (SAN) and atrioventricular node (AVN) was identified by a multiple immunohistochemical labelling of hyper-polarization activated cyclic nucleotide-gated potassium channel 4 (HCN4, marker to cardiac conductive myocytes), protein gene product 9.5 (PGP9.5, general neural marker), choline acetyltransferase (ChAT, cholinergic marker), tyrosine hydroxylase (TH, adrenergic marker), nNOS (nitriergic marker).

Results: Rabbit SAN contained neurons that were distributed solitary or gathered in small clusters. 3 types of cells were observed – positive for ChAT, nNOS and both ChAT and nNOS (biphenotypic). There was no TH (+) neurons or SIF cells in rabbit SAN observed. We did not observe any neural cells in rabbit AVN.

Distribution and immunohistochemistry of neurons located in pig CCS were more complicated. Solitary neurons or small ganglia were observed in both SAN, as well as in AVN. 4 types of neurons ChAT (+), nNOS (+) and 2 types of biphenotypic: positive for ChAT and nNOS or ChAT and TH were found in pig CCS. Also SIF cells were observed in SAN.

In general, most neural cells, found in CCS, were smaller compared to intracardiac ganglia neurons (14.5 ± 0.3 μm vs, 25.4 ± 0.4 μm in rabbit and 12.5 ± 0.5 μm vs, 26.6 ± 0.8 μm in pig).

Conclusions: 1) rabbit heart contained 3 types of neurons only in SAN; 2) pig heart contained 4 types of neurons in both SAN and AVN; 3) neurons of CCS were smaller compared to located in ganglia.

P18. INFLUENCE OF CADMIUM, NICKEL AND ZINC IONS ON LIPID PEROXIDATION IN BRAIN AND RED BLOOD CELLS OF MICE

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Heavy metals – cadmium (Cd) and nickel (Ni) are acknowledged as human carcinogens. They 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. Meanwhile, zinc (Zn) may be substituted by toxic metals in some of its important enzymatic and organ functions. However, there is little information about intracellular mechanisms implicated in Zn-dependent protection of an organism against deleterious effects of Cd or Ni.

The present study was conducted to evaluate the effects of Cd, Ni and Zn ions on lipid peroxidation in brain and in red blood cells of mice and a possible Zn antioxidant effect against Cd and Ni toxicity.

Experiments were done on outbreed white laboratory mice using intraperitoneal injections of $CdCl_2$ (14 µmol Cd/kg body weight), $NiCl_2$ (19 µmol Ni/kg body weight) and/or $ZnSO_4$ (24 µmol Zn/kg body weight) solutions. The exposure-time was 14 days. Lipid peroxides were estimated by measuring thiobarbituric-acid-reactive substances and were expressed as malondialdehyde (MDA).

In our experiments we determined that after 14 days of $CdCl_2$ or $ZnSO_4$ injections, the content of MDA in mice brain was significantly increased by 16% and 73%, respectively, as compared to control mice group. Meanwhile, in Ni treated mice group MDA content was decreased by 19%. Also, our results indicated that in Zn+Cd treated mice group the content of MDA in mice brain was decreased by 27%, but in Zn+Ni treated group this parameter increased by 38% as compared to control. Further experiments carried out in order to examine influence of Cd, Ni and Zn ions on the content of MDA in mice red blood cells. The results indicated that in all groups MDA content was at the control level. Moreover, we did not find the proof that Zn has antioxidant effect in mice brain and in red blood cells after 14 days of exposure to Cd and Ni.

P19. EFFECT OF SELENIUM ON ANTIOXIDANT ABILITY AGAINST ALUMINIUM INDUCED OXIDATIVE STRESS IN MICE BRAIN AND BLOOD

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Aluminum (Al) can induce oxidative stress and is linked to an increased risk of neurodegenerative disorders. Neural tissue is susceptible to oxidative damage due to high content of unsaturated lipids that may be peroxidized. Antioxidants such as glutathione (GSH) have a wide scope to sequester metal ions involved in oxidative stress. Selenium (Se) can increase the antioxidant capacity as a cofactor of glutathione peroxidase.

In the present study we investigateda possible protective effect of Se on the redox status in mice brain and bloodunder short term exposure to Al. Balb C mice were injected i.p. with $AlCl_3$ (7.5 mg Al^{3+}/kg (0.15 LD_{50})) and/or Na_2SeO_3 (0.125 mg SeO_3^{2-}/ kg (0.025 LD_{50})) for 14 days. Control mice received the same volume of 0.9% NaCl. (License of State Veterinary Service for Working with Laboratory Animals No. 0221).

Al ions reduced GSH concentration in the blood by 9% ($p>0.05$). The treatment with Na_2SeO_3 had no effect on GSH concentration in the blood. However, after treatment with Al+Se, the GSH concentration decreased by 24% ($p<0.05$) as compared to control. After 14 days exposure to Al and/or Se there were no statistically significant changes in GSH concentration in the brain in any experimental group.

Al or selenite ions did not cause any significant changes in the concentration of malondialdehyde (MDA), the final product of lipid peroxidation (LPO), in the blood. However, the treatment with both effectors induced significant increase in the MDA concentration by 26%. After exposure to $AlCl_3$ or Na_2SeO_3,MDA concentration in the brain increasedby 17% and 13% ($p<0.05$) respectively, as compared to control. Co-exposure to Al and selenite ions resulted in the increase in brain MDA concentration by 16% as compared to control. Thus, the pretreatment with Na_2SeO_3 20 min before $AlCl_3$ injections did not reduce Al induced brain LPO. Moreover, SeO_3^{2-} alone induced increase in MDA concentration in mice brain.

Our results indicated that administration of Se did not protect mice brain from Al induced oxidative damage. Increase in MDA concentration in the blood after co-exposure to Al and Se may be due to GSH depletion.

P20. PROTECTIVE EFFECT OF MK-801 AGAINST SMALL Aβ1-42 OLIGOMER-INDUCED NEURONAL DEATH

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Soluble Aβ1-42 oligomers are thought to play a central role in pathogenesis of Alzheimer's disease. However, the molecular mechanisms how Aβ1-42 oligomers trigger neurotoxic cascades are not fully elucidated. It has been suggested that oligomeric forms of Aβ may depolarize neurons by direct activation of membrane receptors such as NMDA. The aim of this study was to investigate the toxic effects of small Aβ1-42 oligomers in primary neuronal/glial cerebellar granule and pure microglia cell cultures, evaluating whether these neurotoxic effects are sensitive to NMDA receptor antagonist MK-801.

We found Aβ1-42 oligomers caused plasma membrane depolarization in both neurons and microglia, but by different mechanisms. During 0.5–1 h incubation small Aβ1-42 oligomers induced neuronal membrane depolarization that was insensitive to MK-801 and NBQX (AMPA receptor antagonist), indicating that NMDA and AMPA receptors are not involved in neuronal depolarization. Meanwhile, in CGC's MK-801 (but not NBQX) protected only the microglia cells from Aβ1-42 -induced membrane depolarization. Protective effect of MK-801 was also seen in pure microglia cultures indicating that microglia depolarization may be associated with activation of NMDA but not AMPA receptors.

Measuring glutamate levels in pure microglial culture media we found that 0.5–4 h incubation of cells with Aβ1-42 oligomers did not cause release of glutamate, while in CGC cultures Aβ1-42 oligomers increased glutamate level and this was prevented by MK-801. We also found that MK-801 preserved neuronal viability and protected CGC from $A\beta_{1\text{-}42}$ -induced neurotoxicity during 24 h incubation.

In conclusion, Aβ1-42 oligomers induced rapid NMDA receptor–independent neuronal depolarization and NMDA receptor–dependent release of glutamate in CGC cultures leading to excitotoxic neuronal death. At the same time, Aβ1-42 oligomers induced rapid and NMDA receptor–dependent microglial plasma membrane depolarization.

P21. GLUTAMATE AND NMDA EFFECT ON PLANT CELL ACTION POTENTIAL CHARACTERISTICS

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Plant cells, not being specialized for electrical signaling, use electrical excitability to transmit action potentials from cell to cell over long distances, thus regulating series of physiological features. During plant action potential, influx of Ca^{2+} through membrane induces rapid efflux of Cl^- resulting in instantaneous membrane depolarization. It is known, that glutamate – neurotransmitter in mammalian nervous system, applied to some higher plants tissues, induce electrical signaling. In variety of plant species glutamate increases cytosolic Ca^{2+} concentration and therefore affects action potential generation. Since iGluRs (ionotropic glutamate receptors) homologs in plants – GLR - were discovered, variety of research suggests that glutamate in plants acts via glutamate receptor-like channels which are closest to mammalian NMDA type glutamate receptors. Most data regarding glutamate signaling in higher plants was obtained from entire plant organism or specific complex tissues investigations. Using intermodal characeaen cell model system we were able to investigate glutamate effect on single cell level.

Applying intracellular microelectrodes and voltage-clamp method we investigated influence of glutamate and NMDA (specific synthetic agonist) on action potential mediated ion currents. Experimental results show that characeaen cells are sensitive to 1 mM of glutamate and NMDA. Exogenously applied glutamate and NMDA significantly increases maximum value of Cl^- efflux during action potential generation, reduces action potential threshold and increases action potential amplitude. The effects of NMDA are proportional to applied concentration and significantly higher than effects of the same dose of glutamate. This research demonstrates effect of glutamate on cell membrane transport systems, electrical excitability and supports concept that NMDA type receptors are involved in these processes.

P22. ANTICANCER ACTIVITY OF JUGLONE ON GLIOBLASTOMA CELLS

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Glioblastoma causes approximately 50% of all brain tumours. Despite radical treatment, median survival of the patients is still around one year. The current conventional treatment is limited because of its severe side effects, chemoresistance and tumour recurrence. Moreover, most of the recent clinical trials of the newly discovered agents failed to show any beneficial effects in glioma treatment. Therefore, the search of new chemotherapeutic agents is of high importance.

In this study we investigated the anti-tumour effect of 1,4-naphtoquinones. We found that juglone (5-hydroxy-1,4-naphtoquinone) has a profound antiproliferative and antimigratory effects on glioblastoma cells at low concentrations. In order to delineate the mechanism of action of this substance, we determined the levels of reactive oxygen species (ROS) extra- and intracellularly in treated cells. The results revealed that juglone induced the generation of ROS significantly. However, catalase in the culture medium did not change the antiproliferative properties of juglone. As mitochondrion is one of the main sources of ROS, we determined the complex metabolic changes that juglone induces in glioma cells. The data showed that juglone reduces basal cell respiration rate by directly changing the activity of the components of the electron transport chain.

These findings suggest that juglone is a potential antitumour agent because of its properties to reduce cell proliferation, migration as well as induce metabolic changes in glioblastoma cells.

P23. SYNERGISTIC PROTECTIVE EFFECT OF ROTENONE AND CYCLOSPORINE A ON CALCIUM-INDUCED MITOCHONDRIAL PERMEABILITY TRANSITION IN RAT BRAIN MITOCHONDRIA

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Mitochondrial permeability transition (MPT) is implicated in a number of brain disorders such as ischemic insult and neurodegeneration. The activation of MPT leads to mitochondrial depolarization and cessation of ATP production leading to cell death. Therefore, the inhibition of MPT by pharmacological compounds may be efficient strategy to improve brain tissue functions under pathological conditions such as brain ischemia/reperfusion injury. Mitochondria isolated from various brain regions were shown to have different sensitivity to calcium-induced MPT and this may account for different vulnerability to ischemic stress. In this study we investigated mitochondria isolated from different brain areas - cerebral cortex and cerebellum and tested effects of selective inhibitor of MPT cyclosporin A (CsA) and inhibitors of respiratory chain complex I, rotenone and amytal, to calcium-induced MPT measured as calcium retention capacity (CRC). Mitochondrial respiration function using substrates pyruvate plus malate and succinate as well as CRC was similar in both types of mitochondria. The CsA (0.5 μM) caused significant increase of CRC (up to 70%) of cortical mitochondria, however, ten times higher CsA concentration (5 μM) was required to increase CRC of cerebellum mitochondria. We also found that rotenone (1 μM) was even more potent than CsA and increased CRC by 100% and 115% in cerebral cortex and cerebellum mitochondria, respectively. Importantly, CsA and rotenone acted synergistically and substantially increased CRC of both, cortical and cerebellum mitochondria (up to 170% and 180%, respectively). The second inhibitor of complex I, amytal, neither alone nor together with CsA significantly changed CRC of both mitochondria. These results demonstrate that complex I inhibitor rotenone may regulate MPT and that CsA and rotenone can act synergistically and protect isolated rat cerebral cortex and cerebellum mitochondria against calcium -induced opening of MPT.

P24. CATALASE ACTIVITY IN MICE BRAIN: THE EFFECTS OF SELENIUM AND ALIUMINIUM IONS

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Neurodegenerative diseases are chronic diseases, which damage the front basal cluster and cortex of brain, produce a significant brain cholinergic dysfunction and manifest themselves as memory, language and behavioral abnormalities. To reduce the risks of these diseases and to treat them successfully it is important to determine the mechanisms of their development. It is believed that those diseases are associated with oxidative damage of brain cells. Very often the cause of oxidative stress is the effect of heavy metals on the functioning of body cells. Metal ions not only cause oxidative stress, but also affect functions of cell enzymes and other important compounds directly. Antioxidant system in the human body protects cells/tissues against damages induced by reactive oxygen species.

Catalase is a common enzyme, which catalyzes decomposition of hydrogen peroxide to water and oxygen. It is a very important enzyme in protecting the cell from oxidative damage caused by reactive oxygen species. The aim of this work was to evaluate the effects of selenium and aluminum ions on the catalase activity in brain cells of laboratory mice.

Catalase activity was determined in mice brain after a single and 14 days injection of Al^{3+} , SeO_3^{2-} and (Al^{3+}+SeO_3^{2-}) saline solution in the abdominal cavity.

The results showed that 24 h after i.p. injection of Al^{3+} (0.5 LD_{50}), $SeO_3$2- (0.025 LD_{50}) or their mix the catalase activity was the same in control and experimental groups. As 0.5 LD_{50} Al^{3+} dose for 14 days is lethal, we used these Al^{3+} doses: 0.1 LD_{50}, 0.15 LD_{50} and 0.25 LD_{50}. We found that Al^{3+} caused a statistically significant increase of brain catalase activity, as compared to the control group of mice. Meanwhile, the SeO_3^{2-} and (Al^{3+}+SeO_3^{2-}) saline solution injection for 14 days, as well as after 24 h exposure, did not affect brain catalase activity.

P25. THE ROLE OF miR-449 IN BRAIN TUMOURS

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Combinations of initial genetic mutations determine the phenotype of brain tumours. Our previous results showed that deletion of Pten/p53 genes in the stem cells of subventricular zone give rise to gliomas, whereas deletion of Rb/p53 generated primitive neuroectodermal tumours (PNET). In order to find the mediators of this phenomenon, we performed microRNA expression profiling identifying up-regulated microRNA-449 in Rb-/-; p53-/- tumours. The further analysis revealed that Rb-/-; p53-/- cells with down regulated microRNA-449 had similar phenotypic features to Pten-/-; p53-/- cells in in vitro and in vivo experimental models. Moreover, the role key downstream targets of microRNA-449 were identified in these tumours. Our results suggest a crucial role of microRNA-449 in the formation of different brain tumour phenotypes.

P26. PROTECTIVE EFFECT OF ZN AGAINST PB AND NI INDUCED BRAIN δ-ALAD INHIBITION

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δ-aminolevulinic acid dehydratase (δ-ALAD) – cytosolic, Zn dependentenzyme, catalysing second step of heme biosynthetic pathway. δ-ALAD is a sulfhydryl-containing enzyme that is a target for heavy metals and sulfhydryl agents. Inhibition of this δ-ALAD leads to accumulation of δ-aminolevulinic acid and heme depletion that finally cause overproduction of reactive oxygen species and neurotoxicity. This study was conducted to estimate the ability of Zn to protect heme synthesis system from Ni^{2+} and Pb^{2+} toxicity.

The obtained results showed that single exposure to Pb^{2+} decreased brain δ-ALAD activity by 31 %, while enzyme activity in liver and blood was suppressed much more drastically – by 74 % and 99 % respectively. Zn pre-treatment insignificantly increased δ-ALAD activity in the brain and partly diminished suppressing effect of Pb^{2+} in mice liver and brain. Single dose of $NiCl_2$ reduced brain δ-ALAD activity by 21%,whereas liver and blood enzyme activity was suppressed greater - by 38 % and 53 % respectively. Zn pre-treatment protected brain as well as blood enzyme activity from the inhibitory effect of Ni^{2+} and partly restored enzyme activity in the liver of mice.

Repeated exposure to Pb^{2+} had no effect on brain δ-ALAD activity, yet drastically by 73 % and 92 % suppressing liver and blood enzyme activity. Repeated pre-treatment by Zn diminished the suppressing effect of Pb in liver and blood, but didnot restore δ-ALAD activity to the control level. Repeated administration by Ni^{2+} surprisingly increased brain enzyme activity by 26 %, however inhibited δ-ALAD activity in liver by 32 % and had no effect on enzyme activity in the blood. After repeated exposure to both metals, Zn^{2+} restored brain δ-ALAD activity to the control level, but didn't provide any protection on liver δ-ALAD activity.

Pre-treatment by Zn provided protective effect against Pb induced δ-ALAD inhibition in the brain, liver and red blood cells after single and repeated both metal administration, and protected δ-ALAD activity from the inhibitory effect of Ni only after single administration of both metal.

P27. THE ROLE OF MOLECULAR KARYOTYPING IN THE INVESTIGATION OF EPILEPSY PATIENTS

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Introduction: Molecular karyotyping methods enabled huge progress in epilepsy diagnostics and genetic etiology uncovering. According to ISCA consensus statement 2010, it is an established first-tier diagnostics for patients with developmental disabilities or congenital anomalies, giving a 7-20% additional diagnostic yield in epilepsy patients depending on epilepsy forms and comorbidities. Tens of genes were affirmed as being a single cause or giving a predisposition to epilepsy by thorough investigation of genomic imbalances found in these patients.

Case reports: we present four cases with genomic imbalances found by molecular karyotyping in patients with epilepsy. The first one is a 7-years-old female with severe intellectual disability, dysmorphic features, corpus callosum hypoplasia, congenital heart defect, bilateral clubfoot, dolichosigma and pyelectasy. Molecular karyotyping revealed a terminal chromosome 1q deletion [del(1)(q43)].

The second patient, a 3-years-old female, presented with congenital heart defect, severe developmental retardation, dysmorphic features and stridor, interstitial deletion of chromosome 6 [del(6)(q16.1q22.31)] was diagnosed.

The third patient is a female of 2.5-years with lissencephaly, severe developmental retardation and mild dysmorphism. A balanced translocation t(5;19)(p10;q10) inherited from a healthy father was found by conventional karyotyping while array-CGH disclosed an interstitial deletion of chromosome 17 [del(17)p13.3p13.2].

The fourth female patient presented at the age of 35 years with a profound intellectual disability, dysmorphism and diabetes mellitus. Unbalanced translocation [der(9)t(9;19)(q34.3;q13.43)] was revealed by array-CGH. These genomic imbalancies were inherited from a healthy father who is a balanced translocation carrier.

Conclusion: molecular karyotyping has a high practical and scientific value in the investigation of epilepsy patients.

P28. THE ROLE OF COX-2, PPAR AND MMP-9 ON BRAIN PLASTICITY AFTER STROKE

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Stroke is one of the most common brain injuries. It may result in huge impairment of cognition and motor function in patients. Repair process is related to neuroplastic changes but physical therapy alone is not effective, so there is a need to take a look in molecular aspects of that process and maybe use pharmacological treatment. In our work we focused on 3 proteins- COX-2, PPAR and MMP-9. We observed upregulation of proinflammatory factor COX-2 in stroke area, moreover chronic treatment of ibuprofen, anti-inflammatory factor- increases neuroplasticity in that region. Also PPAR-gamma receptors are involved in inflammation development and administration of antagonists of PPAR-gamma reduces recurrent stroke. MMP-9 is an enzyme engaged in both maturation of synapses (which is part of neuroplasticity process) and angiogenesis in repair process after that injury. Determination of the role and interactions of these proteins could play a key role in potential pharmacological therapy and will help us understand more about the process of neuroplasticity.